

2ND EDITION

THE HONG KONG GERIATRICS SOCIETY

Curriculum in Geriatric Medicine

Chief Editor : Tak-kwan KONG

The Hong Kong Geriatrics Society
香港老人科醫學會





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Front Cover: painting by Mr. Wing-ki KONG (1921-2005) in his seventies.
Inside of Front Cover, Upper: Chinese calligraphy "*People treasure eventide for ever*" （人間重晚晴）, an extract from Chinese poem of Shanyin LI (813-858), by Mr. Shun-kai TSE (1941-) in his sixties.
Inside of Front Cover, Lower: painting "*Eventide*" (A scene in the Westminster Union, depicting inmates of a workhouse infirmary), 1878, by Hubert von Herkomer (1849-1914). Reproduced by courtesy of the Walker Art Gallery, National Museums Liverpool.

CURRICULUM

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香港醫學專科學院出版社
HONG KONG ACADEMY OF MEDICINE PRESS

Dedicated to my parents and all elders who have taught us how to be better geriatricians through their illnesses

The Hong Kong Geriatrics Society Curriculum in Geriatric Medicine

Second Edition

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ISBN 978-988-78347-0-0
First edition 2005
Second edition 2017
A record for this book is available from the Hong Kong Central Library

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Publisher: Hong Kong Academy of Medicine Press, 10/F, Hong Kong Academy of Medicine Jockey Club Building, 99 Wong Chuk Hang Road, Aberdeen, Hong Kong SAR, China; tel: (852) 2871 8822; fax: (852) 2515 9061; email: hkampress@hkam.org.hk; website: http://www.hkampress.org

E-book: An electronic version of the book and its companion *The Hong Kong Geriatrics Society Learning Manual (Second edition)* is available at The Hong Kong Geriatrics Society website: http://www.hkgs.org. Details of subscription to the printed and electronic versions are available at the Hong Kong Geriatrics Society website.

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Printed and bound in Hong Kong
First printing December 2017

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FOREWORD 1 to the Second Edition

In the early 1970s, I was fortunate enough to be one of the pioneers in the field of Geriatric Medicine of Hong Kong. We started the first local geriatric unit in Princess Margaret Hospital and I served as the consultant geriatrician.

Thirty-six years after leaving Princess Margaret Hospital, I returned to visit the Consultant Geriatrician in-charge, Dr Tak-kwan Kong, the Chief Editor of this book. This time, I was a geriatric patient. Yes, everything in life comes full circle. Under Dr Kong's care, my health gets better and I get to know him better too. Although he is very knowledgeable and competent, he is always modest and always a gentleman keeping a low profile. I must praise The Hong Kong Geriatrics Society for their wise choice of Dr Kong as the Chief Editor of this book.

This year I wholeheartedly welcome Dr Kong's second edition of *Hong Kong Geriatrics Society Curriculum in Geriatric Medicine*. The first edition, published in 2005, has been well received across the medical fraternity. This comprehensive textbook of geriatric medicine is highly recommended for doctors who have to deal with the medical problems of elderly patients. It is also a valuable reference for candidates who study courses in Geriatric Medicine and Gerontology. The first textbook has been improved and updated. This second edition is aimed at not only primary care doctors but also geriatric specialists.

As we read this book let us be thankful to all the co-authors who have made their invaluable contributions. A good job well done. Thanks are especially due to the Chief Editor, Dr Tak-kwan Kong who had the inspiration, capability, and determination to see it developed and produced.

Sik CHAN
Founding President (1981–1984), The Hong Kong Geriatrics Society
2016

FOREWORD 2 to the Second Edition

According to our cultural ideals, our elders are the group we most respect and cherish. This, however, has not always been the reality. Older people in Hong Kong do not always receive the level of medical care and social support that enables them to live in the comfort and dignity that they deserve.

This textbook, first published in 2005, is an important step towards righting this wrong. Providing essential information about not only medical care but also ethical, legal and social issues, this book is a rich collection of experience and knowledge needed by all who care for older people. This second edition could not come at a better time, as it is becoming more apparent that meeting the needs of elderly people is an urgent issue that Hong Kong must address.

Hong Kong's oldest people will soon be one of the largest and most significant groups in society. In 2015, 14.6% of the population (1.08 million people) were aged 65 years or above. This number is projected to burgeon to 30%, nearly one-third of the population, by 2041. Even more crucial are the numbers of the very old. It is estimated that Hong Kong will be home to almost half a million people aged over 85 years by 2041. All medical practitioners, students, and other health workers, whatever their specialty, need to be well-versed in the management of the health of older people.

While current debate is framing the rapid rise on numbers of older people as a problem, this textbook is offering all the elements of a solution. Older people do not have to live out their last years in ill health and be considered a burden by their family and the community in which they live. An improved understanding of their needs, the challenges, and approaches to improving their health will enable older people to live their last years in the best health possible, and thus continue to enrich our society with their much-needed experience, skills, and wisdom.

Early diagnosis and prompt management of conditions likely to cause or exacerbate chronic illness can enable older people to live happier, healthier, and more productive lives. In recent years there has been a revolution in thinking about the health of older people. The aim is not just to live longer but to live a healthier, more active life. The last few years of life should, and can, be the very best years.

To achieve great age was once considered one of the greatest human achievements. The very old should not be condemned to a life of pain, loss of dignity, and social isolation. With the help of the excellent information contained in the chapters of this book, we can work together to make those last years, truly 'golden'.

Donald LI
President (2012–2016), Hong Kong Academy of Medicine
2016

PREFACE to the Second Edition

The first edition of this book, published in 2005, arose from the need for a locally relevant textbook for the Postgraduate Diploma in Community Geriatrics Programme, organised by The Hong Kong Geriatrics Society (HKGS) and the University of Hong Kong, and awarded to 247 doctors since 2000. In this second edition, the HKGS has enriched the contents of this book to meet the demands of those in specialist practice in geriatric medicine, a number that has grown to 191 since 1975. Thus, the original chapters have been expanded and new chapters added on comprehensive geriatric assessment, acute geriatric care, long-term hospital care, transitional care and discharge support, geriatric day hospital, telemedicine, orthogeriatrics, oncogeriatrics, end-of-life care, thyroid disorders, and pressure ulcers. This second edition has been expanded from 33 to 53 contributing authors, and from 359 pages to over 450 pages.

I would not have accomplished this mammoth mission without the dedication of our authors, the excellent publication editorial support of Ms Yvonne Kwok and Ms Betty Lau at the Hong Kong Academy of Medicine Press, and the professionalism of our four associate editors: Drs Francis Chun-keung Mok and Christopher Chor-ming Lum, past and current Chairmen of the Specialty Board in Geriatric Medicine respectively, and Drs Felix Hon-wai Chan and James Ka-hay Luk, past and current Presidents of The Hong Kong Geriatrics Society respectively.

The editorial work started in 2013 when I was in Princess Margaret Hospital (PMH). I retired from PMH in August 2016, and was called upon to the Prince of Wales Hospital (PWH) in September 2016. This enabled me to experience PWH's fine academic geriatric centre that has prospered since its planning by our forbearers in PMH. It is here in PWH that I encountered some of the authors with whom I have been collaborating. It is also where I experienced the geriatric giant of falls when descending stairs and can now appreciate the meaning of fear-of-fall in geriatric patients under my care.

It is often said that textbooks cannot keep abreast of advances in knowledge, given the time they take to produce. The team effort of authors, associate editors, and editor of this book have ensured the text is as up-to-date as possible, evidenced by the number of recently published articles in the references cited. The decision to have this edition accessible online in electronic format also opens the way for more rapid updating. The true value of this book, however, lies not in its being a mere collection of the latest information but rather how such knowledge can be usefully applied in the care of elderly people in our daily clinical practice. Clinical relevance is ensured by summarising practical tips in key points and the use of case scenarios in this book and its companion Learning Manual. Geriatric relevance is ensured by sections that describe how the special qualities and needs of frail elderly patients alter their presentation and management. Chapters that describe a geriatric approach and geriatric syndromes provide guidance to readers and ensure a problem-based approach in geriatric medicine. Local relevance is ensured by the inclusion of local resources and services in patient care as well as literature relevant to Chinese patients.

Ignatz Nascher, who created the word "geriatrics" to cover a special branch of medicine dealing with old age, wrote in his preface to the first American 500-page textbook on *Geriatrics* in 1914, "The spirit of veneration of ancestors and the aged, such as exists in China, does not exist among us. The sentimental interest in the aged is confined to the immediate family of the individual and there the interest is often less sentimental than dutiful." Most of the increase in elderly persons in the next century will be in Asia, in particular China. It is timely, therefore, that the HKGS has embarked on the production of a textbook of geriatric medicine relevant to Chinese, as a tribute to our elderly people so treasured among our Chinese culture. All the authors are Chinese with expertise in the provision of medical, psychiatric, social, and psychological care of Chinese elderly patients.

I believe that this textbook will contribute to quality care of Chinese elderly people by equipping doctors with the knowledge they need, and that it provides a vital resource for trainees and trainers in geriatric medicine and gerontology.

Tak-kwan KONG
Chief Editor
2017

FOREWORD 1 to the First Edition

Old age is the fulfillment of every individual's birthright. Thus, an ageing population will be an universal phenomenon affecting all nations. With life expectancy in Hong Kong among the world's highest (in 2003 male 78.6; female 84.3) and birth rate among the lowest (1.2 children born per woman), our population is expected to follow this ageing trend. The percentage of people aged 65 plus is expected to rise markedly from 11.7% in 2003 to 27% in 2033. It is a really daunting prospect because the projected figures also reveal a more rapid rise in the "old" old. Therefore, we especially the medical profession should think seriously about this and most importantly should take a more positive step to ensure that our elders can lead a more meaningful and independent life in our society.

Over the years, the policy of the Hong Kong Government in the provision of care for elderly people is "care in the community and by the community". With the emphasis on community care, the general practitioners will be facing an increasing load of elderly patients in their daily practice. I can still recall that in the late 70's when I was teaching in the University of Hong Kong, I always made the remark that by the year 2000, most of the doctors will have to be "geriatricians" whether they like it or not, unless they opt to practice paediatrics or obstetrics.

Up to now, teaching in geriatric medicine for medical students in the two universities is still inadequate for them to face the challenges brought about by this ageing population. On the post-graduate scene, for doctors working in public institutions under the Hospital Authority, training is a major component in their daily routine. In general, they usually have adequate supervision, plenty opportunities and reasonable resources to complete and attain their higher professional training. But for doctors in the private sector, especially for general practitioners, the situation is just the opposite.

I am indeed pleased to see that The Hong Kong Geriatrics Society and the University of Hong Kong have taken a major step forward in 2000 and started the Post-graduate Diploma in Community Geriatrics course. This really is the first proper and well-structured training course for doctors in the private sector. However, the Programme is always heavily over-subscribed in the last four years. In order to satisfy the need and demand for a local reference book as a teaching and training manual, Dr. Kong and his colleagues have put together their expertise in the publication of the book "Hong Kong Geriatrics Society Curriculum in Geriatric Medicine". This practical book will definitely help most doctors to improve their knowledge, confidence and competence to cope with the many common problems encountered in our daily practice. Finally, Dr. Kong and all the contributing authors are to be congratulated for their concerted effort and I sincerely think this really sets a milestone in the development of geriatric medicine in Hong Kong.

Yau-yung NG
JP, FRCP(Glasg)
Past President,
The Hong Kong Geriatrics Society
2004

FOREWORD 2 to the First Edition

Hong Kong was early on the world stage in recognizing that medical problems of an ageing population were so complex and unique as to require a specialist approach. Following the lead of Great Britain where many of the Hong Kong pioneers in geriatric medicine received their specialist training; the medical speciality of geriatric medicine has become firmly established in Hong Kong. It has developed to a high state clinically, academically and in structure and organization.

It is fitting therefore that this important textbook has now emerged providing a full spectrum of world knowledge on the subject, as well as considering its special application to Hong Kong. As a comprehensive textbook of geriatric medicine, it is relevant to all doctors whose practice includes elderly patients. They will find that it contains practical advice in relation to the special clinical problems of older people as well as local information in relation to the structural aspects of their care. It also provides the core knowledge for those studying for the Postgraduate Diploma in Community Geriatrics.

The book is the work of 33 authors, most of whom live and work in Hong Kong. That such a galaxy of talent and expertise in geriatrics should be available in a reactively small population, is itself a remarkable accomplishment which would be hard to match elsewhere in the world. The editor, Dr. TK Kong is to be congratulated on bringing about this considerable achievement. It reflects his 21 years of full time work in geriatric medicine, Presidency of The Hong Kong Geriatrics Society as well as his considerable expertise in teaching the subject and in research.

I am delighted to have this opportunity to commend the work of Dr. Kong and The Hong Kong Geriatrics Society to a wide readership.

John C. BROCKLEHURST
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University of Manchester
2004

PREFACE to the First Edition

When I graduated in 1980 from the University of Hong Kong, geriatric medicine was not taught and I had no idea of what geriatric medicine was like. In 1983, I had the chance to join the geriatric unit of Princess Margaret Hospital. I frankly told Dr. YY Ng, the consultant geriatrician in-charge of the unit then, I was just waiting for a post in internal medicine. To this, Dr. Ng replied, "Never mind, just stay on and see if you like the specialty. Some doctors may not like working with elderly patients who are incontinent of urine and faeces… We see and care elderly patients as a multidisciplinary team." To acquaint myself quickly with the subject of geriatric medicine when I first started working in the geriatric unit, I brushed through the slim book "Lecture Notes on Geriatric Medicine." Over the years, I witnessed how geriatric medicine was practised and how elderly patients were cared and helped in the geriatric unit. I stay on, finding the specialty both intellectually challenging and emotionally rewarding. In 1988, I had the fortune to have a year of Commonwealth Medical Fellowship for training in geriatric medicine under Professor John Brocklehurst in Manchester. I admired how Professor Brocklehurst could turn the neglected topic of incontinence into a serious subject of study, and was impressed by their serious undergraduate and postgraduate training in geriatric medicine, something that I would like to see to happen in Hong Kong.

Although the medical faculties of both universities in Hong Kong now have geriatric medicine included in their undergraduate curriculum, the knowledge and skills taught to medical students are inadequate to equip doctors facing a growing elderly population, and a growing knowledge base in geriatric medicine. This has become more pressing with the increasing emphasis on community care and the involvement of general practitioners and family doctors in the care of elderly people. It is with this in mind that The Hong Kong Geriatrics Society, together with the University of Hong Kong, started the Postgraduate Diploma in Community Geriatrics Programme in 2000. Now that the Programme has been running for four years, we realize the need for a local instruction and learning manual and thus the publication of this book "Hong Kong Geriatrics Society Curriculum in Geriatric Medicine". The book is targeted also at a wider readership and aims to provide locally relevant and practical information to doctors involved in the care of elders in the community in Hong Kong and to equip them with the necessary knowledge, skills and attitude to practise geriatric medicine.

The curriculum contents are based on the Hong Kong Geriatrics Society Guidelines for Postgraduate Training in Community Geriatrics 2003, as well as taking reference the core competencies required in the Diploma in Geriatric Medicine examinations in the United Kingdom. This book is presented in three main sections: geriatric services and geriatric approach, geriatric syndromes, and common geriatric diseases. Emphasis is placed on clinical and local relevance. As editor, I am pleased to gather a team of committed authors, and to them, I must express my deep appreciation for their contributions and hard work. The majority of the contributing authors are local geriatricians, but psychogeriatricians, a family physician and a social gerontologist have also written on relevant subjects.

I do hope that this book will be a useful local resource to doctors caring for elderly people.

Tak-kwan KONG
Editor
2004

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Acknowledgements

The authors of Chapter 4 would like to thank Dr Derrick Kit-sing Au, Director of Quality and Safety, Hospital Authority, Hong Kong, for permission to use the chapter authored by him in the first edition; Ms Irene Chan, Occupational Therapist I, Ms Dora Chan, Senior Occupational Therapist, Mr Kenneth Leung, Senior Physiotherapist and Ms Lai-ming Leung, Nursing Officer, Department of Rehabilitation, Kowloon Hospital for professional advice and photos of Figures 4.1b-f and 4.2; and Dr Tak-kwan Kong, Consultant Geriatrician, Department of Medicine and Geriatrics, Princess Margaret Hospital for providing Figure 4.1a.

The author of Chapter 13 would like to thank Dr Leung-sing Ho, Consultant Surgeon, Department of Surgery, Princess Margaret Hospital, for providing the photo of the surgical specimen of Plate 13.2.

The author of Chapter 33 would like to thank Dr Kam-cheong Lee, Consultant Pathologist, Department of Pathology, Princess Margaret Hospital, for providing Plates 33.5c, 33.6c, and 33.8d.

The authors of Chapter 37 would like to thank Dr Bernard Bun-lap Wong, specialist in cardiology in private practice, for his valuable comments on the section of ischaemic heart disease, heart failure, and hypertension in this chapter. Plates 37.1, 37.2, and 37.3 are provided by Dr Tak-kwan Kong, Consultant Geriatrician, Department of Medicine and Geriatrics, Princess Margaret Hospital.

The author of Chapter 40 would like to thank Prof Henry Chan, Honorary Clinical Professor, Department of Medicine, The University of Hong Kong, for reviewing and providing valuable comments on this chapter. Plates 40.1 and 40.2 are provided by Ms Naomi Yeung, APN (ICN), Kwong Wah Hospital.

The author of Chapter 44 would like to thank the Working Group on Pressure Ulcer Care, Acute Care for the Elderly Project, Geriatric Subcommittee, Hospital Authority, Hong Kong (Dr Ian Chin-pang Chan, Dr Kuen Lam, Dr Sze-ting Lau, Dr Kwun-man Lo, Dr Chung-tai Sy, Dr Wai-ming Wong, Ms Gloria Aboo, Ms Joyce Cheung, Ms Wan-tong Lai, Ms Wai-kuen Lee, Ms Tammi Lo, Ms Helen Shiao, Ms Yin-ping Wan, Ms Yuet-han Yu, Mr Charles Cheung, and Mr Paul Chui) for preparing the guideline on pressure ulcer prevention and management. Plate 44.1 is provided by the Department of Nursing, TWGHs Fung Yiu King Hospital.

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CHAPTER 1

Development and Practice of Geriatric Medicine

Tak-kwan KONG

OBJECTIVE

To learn the "what", "why", and "how" of geriatric medicine; the origin of geriatric medicine and its development in Hong Kong; global and local ageing demographics; biological ageing and frailty; the unique aspects of the history, physical examination, and diagnosis in elderly people including the altered / atypical presentation of disease, the multifactorial nature of illness, and multiple pathology; the need for a comprehensive geriatric assessment and holistic approach, as well as a model of geriatric practice.

KEY POINTS

- Elderly people are heterogeneous and encompass a broad spectrum from fit to frail.
- Biologically aged patients are characterised by frailty and complexity.
- When stressed by illness, drugs, and adverse social factors, frail elderly persons readily present with geriatric syndromes (including the classic "geriatric giants": intellectual decline, instability, immobility, and incontinence) with functional decline.
- An apparent "social problem" may represent a social crisis precipitated by disease(s) yet to be diagnosed.
- A comprehensive geriatric assessment means knowing an elderly person in all dimensions; it is the cornerstone of geriatric medicine.
- Make a problem list instead of a diagnosis in a frail elderly individual.
- Do not ignore functional assessment.
- Question both the elderly individual and his / her caregiver.
- The essence of geriatric medicine is to assess and treat the medical and rehabilitative needs of elderly people. This is accomplished by establishing the problems, identifying their causes, treating conditions that are treatable, correcting deficiencies that can be corrected, and compensating for what cannot be corrected.
- The aims of a geriatric service are: to maintain health in old age by continuing social engagement and avoiding disease; enabling early detection and appropriate treatment of disease; maintaining maximum independence in the presence of irreversible disease and disability; and providing sympathetic care and support during terminal illness.

1.1 Historical Background

1.1.1 Origin of Geriatric Medicine

The history of geriatric medicine can be traced to the Industrial Revolution of the 19th century, when the poor and the aged sick were kept in workhouses, leading miserable and monotonous lives in gloomy environments. This is depicted vividly in a painting by Hubert von Herkomer titled "Eventide" (see inside of front cover). "Eventide" depicts inmates of the St. James's Workhouse in Soho, London in 1878. Workhouses were the last resort of those — often the old — who through extreme poverty were unable to support themselves. von Herkomer was drawn to the subject because of his sympathy for what he called "the sorrowful side of humanity".[1]

Many workhouse inmates were stricken by illnesses but the prevailing attitude of the time was that nothing further could be done. A turning point occurred in 1935 when Marjory Warren was given the medical responsibility for one such workhouse infirmary in London where primarily elderly patients were labelled as "incurable".[2] With enthusiasm, optimism, and hope, she showed the medical profession that something could be done, and published her work in the 1940s.[3,4] She noted that elderly patients were heterogeneous and thus assessed and classified them according to their mobility, continence and mental state, as well as addressing their underlying

problems. In this way, she introduced comprehensive geriatric assessment (CGA) and rehabilitation, and managed to discharge some of the institutionalised elderly people back to the community. Thus geriatric medicine arose in the UK 80 years ago in reaction to an attitude of neglect and apathy towards elderly patients who were thought to be incurable and thus abandoned in chronic infirmaries where they were obliged to spend their remaining years. The need for a positive attitude in the medical care of elderly people was expressed by Ignatz Nascher when he coined the name "Geriatrics" in 1909[5]: "Geriatrics, from geras, old age, and iatrikos, relating to the physician, is a term I would suggest… to emphasise the necessity of considering senility and its diseases apart from maturity and to assign it a separate place in medicine." Since Marjory Warren showed that many elderly patients had conditions from which they could often be cured or rehabilitated, the specialty of geriatric medicine has continued to grow and an impressive knowledge base has been accumulated.[6,7] This, combined with increasing expertise in the interdisciplinary treatment and assessment of elderly patients and in ensuring that health and social services work in a coordinated fashion, has improved the care of ill elderly people.

1.1.2 Development of Geriatric Medicine in Hong Kong

Although the origin of geriatric medicine has traditionally been associated with the work of Marjory Warren in London from the late 1930s, a parallel development of geriatric medicine was emerging simultaneously in Glasgow led by Noah Morris, Professor of Materia Medica and Therapeutics at Glasgow University, with its clinical unit at Stobhill Hospital.[8] From this cradle of geriatrics in Glasgow emerged three of the first Professorial Chairs of Geriatric Medicine in the UK, and two Presidencies of the British Geriatrics Society — Ferguson Anderson, Bernard Isaacs and John Brocklehurst, who in turn, have served respectively as the mentors of three Presidents of the Hong Kong Geriatrics Society (HKGS) — Sik Chan, Ngai-sing Ng, and Tak-kwan Kong. The development of geriatric medicine in Hong Kong is thus closely tied to that of Glasgow.[9]

To plan the new establishment of geriatric services in Hong Kong, Sik Chan was sent by the Hong Kong Government to the UK from late 1973 to early 1974 to see how British geriatric services were organised.[9,10] He stayed mainly at Stobhill Hospital with Ferguson Anderson and returned to establish the first geriatric unit in Hong Kong at Princess Margaret Hospital (PMH) in 1975 following the model of geriatrics in Glasgow,[9] himself becoming the first consultant geriatrician in Hong Kong. Sik Chan, together with his followers, founded the HKGS in 1981 as a professional association in the field of geriatric medical care, and was elected the first President of the HKGS. The Glasgow connection was further strengthened by Yun-cheong Lee, who visited Stobhill Hospital in 1979. In his subsequent role as the first Honorary Secretary of HKGS, he invited the Glaswegian professors in geriatric medicine to share their experience and expertise at the annual scientific meetings of the HKGS in the early 1980s. Yau-yung Ng, the second consultant geriatrician at PMH, visited Francis Caird and Brian Williams at Stobhill Hospital in 1982, and paved the way for future specialist training in geriatric medicine for medical staff of the new geriatric unit. Yau-yung Ng succeeded Sik Chan as the second president of HKGS in 1984, and became the first Honorary Clinical Lecturer in geriatric medicine at the University of Hong Kong (HKU) in 1979. Since then, geriatric medicine has been included in the undergraduate curriculum. Clinical bedside teaching in geriatrics for HKU medical students commenced at PMH in 1981 and in 1988 for the Chinese University of Hong Kong that appointed its first professor in geriatric medicine in 1994, Jean Woo. The HKU appointed its first clinical associate professor in geriatric medicine in 2014, Joseph Shiu-kwong Kwan, mentee of Professor Steven Allen of Bournemouth University who lectured under Professor John Brocklehurst of the University of Manchester in the mid 1980s.

Over the years, a steady stream of medical staff from Hong Kong has received higher training in geriatrics both locally and from overseas geriatric departments, mainly in the UK, but also in the US, Australia, New Zealand, and Canada. For the UK, centres of excellence in geriatric medicine in Glasgow[11] (under Dr Brian Williams, Professors Francis Caird and David Stott) and Manchester[12,13] (under Professors John Brocklehurst, Raymond Tallis, and Michael Horan) have nurtured a significant number of geriatricians in Hong Kong. Other training centres in geriatric medicine in the UK to whom local geriatricians have been attached include those in Birmingham, Bournemouth, Cambridge, Cardiff, Edinburgh, Leeds, London, Newcastle, and Oxford.

Since the inauguration of the Hong Kong Academy of Medicine in 1993 to oversee structured specialist training in Hong Kong, the HKGS has been recognised by the Hong Kong College of Physicians (HKCP) as the professional society to advise on higher specialist training in geriatric medicine. In 1993, Ngai-sing Ng, the then President of the HKGS and Jean Woo, the Chairman of the HKCP Subspecialty Advisory Committee (renamed Specialty Board in 1996) in Geriatric Medicine, contributed the first framework for training guidelines in geriatric medicine in Hong Kong.[14,15] The number of specialists in geriatric

medicine has grown from one in 1975 to 191 in 2017. In 2000, the HKGS and the HKU introduced the Postgraduate Diploma in Community Geriatrics to further the knowledge and skills of general practitioners and family physicians.[16] The HKGS also facilitated the establishment of the first overseas centre for the Diploma in Geriatric Medicine (Glasgow) examination in Hong Kong in June 2005.

Hospital-based community geriatric assessment teams have been set up since 1994 to provide timely assessment of frail elderly people before residential placement, as well as to provide outreach support to residential care homes. Although most of the older geriatric units in Hong Kong developed separately from medical departments, integration of geriatric medicine with internal medicine has been the direction of the Hospital Authority so that new geriatric services operate under internal medicine. Structural reorganisation towards integration has occurred for the older geriatric units since 1994. Integrated discharge support programmes with close collaboration between the hospital geriatric team and non-governmental organisation home support team have been rolled out since 2008. Their role is to enhance the function and quality of life of elderly individuals living at home after hospital discharge through early service coordination to address post-discharge support and provision of timely home care and geriatric services.

1.2 Epidemiology of Ageing and Its Impact

1.2.1 Global Ageing

The global elderly population (≥65 years) is projected to grow considerably in both absolute and relative terms in the coming three decades, primarily because of a historical decline in fertility and mortality.[17] The gain in life expectancy since the early 1800s has

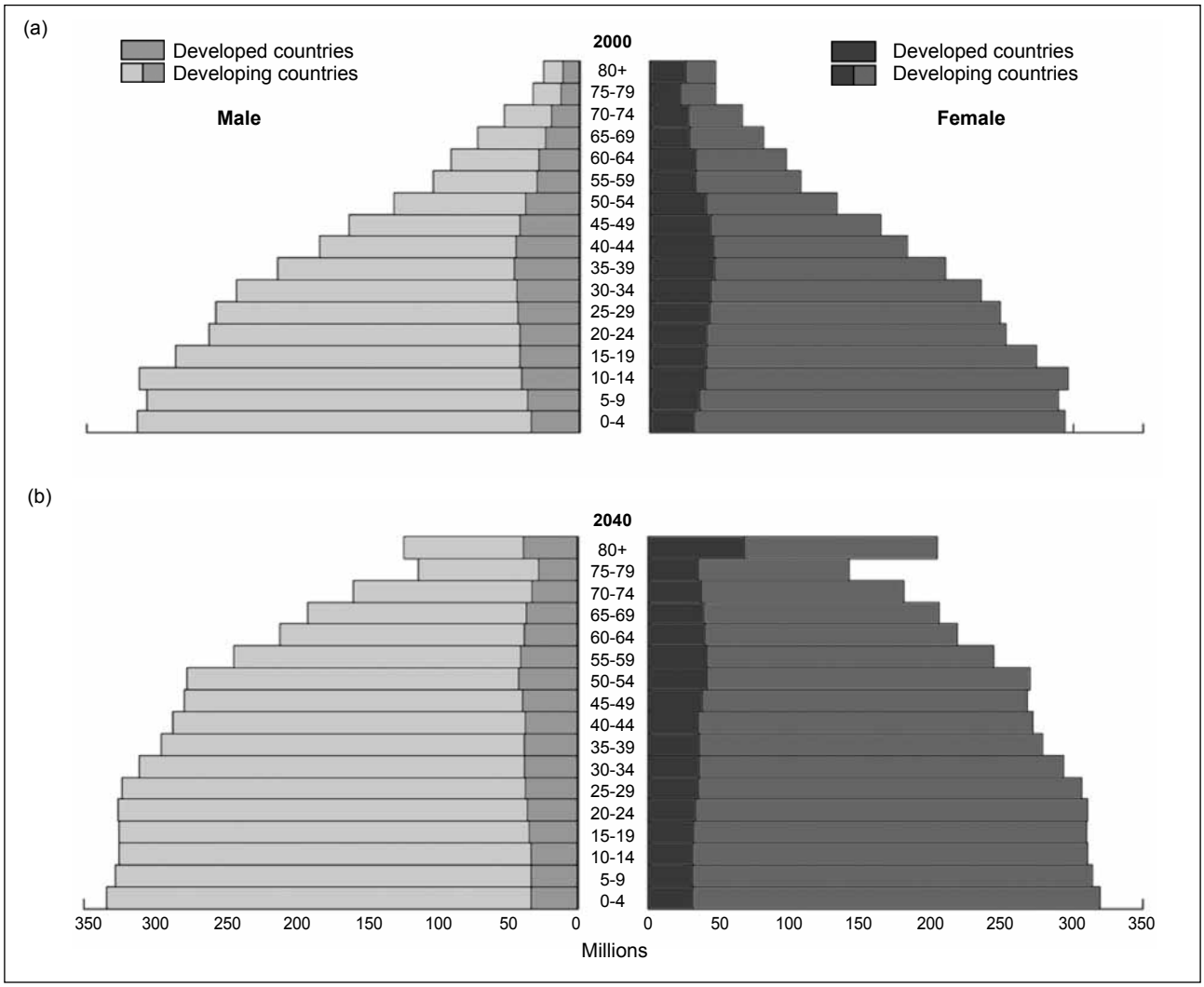

Figure 1.1 Pyramids of global population: (a) 2000 and (b) 2040, showing rapid growth for developing countries and higher growth rates at higher ages[17]

been attributed to advancements in nutrition, public health, medicine, socioeconomics, and education.

In 2008, the global elderly population was 506 million people aged ≥65 years, but is projected to increase rapidly to 1.3 billion by 2040. Similarly, the relative size of the elderly population is expected to double from 7% of the global population in 2008 to 14% by 2040.[17] Growth of the elderly population is especially marked in developing countries (Figure 1.1[17]) and among the older age-groups (Figure 1.2[17]). Ageing impacts everything from a microscopic to a macroscopic level: genes, cells, organ systems, medicine, society, economics, and ethics.

1.2.2 Ageing in Hong Kong and Its Impact

In 2010-15, the global total fertility rate was 2.52 births per woman and the overall life expectancy at birth in 2015 was 71.4 years (73.7 years for female and 69.1 years for male)[18]; the corresponding figures for Hong Kong in 2016 revealed a much lower fertility rate of 1.205 births per woman, but a higher life expectancy at birth of 87.3 years for females and 81.3 years for males.[19] The elderly population (aged ≥65 years) in Hong Kong has risen from 4.5% of the total population in 1971 to 14.6% in 2015 with a number of one million, and this is expected to rise further to 24% by 2031 and a population of two million (Figure 1.3[19]).

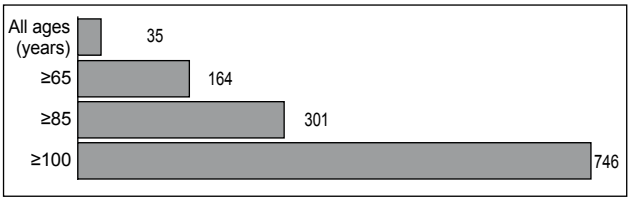

Figure 1.2 Percentage change in the world's population: 2005 to 2040[17]

Contrary to a somewhat ageist misconception, those who have reached their birth life expectancy can still live for a further significant number of years; the life expectancy of a woman in 2016 at the age of 80 years was 12.25 years in Hong Kong, while the life expectancy of an 80-year-old man was, on average, 9.40 years.[20] Since the prevalence of illness and disability rises with age, the growth of elderly population, particularly those in the older-old group (≥75 years) has important implications for health and social services. As demonstrated in the figures for hospital utilisation in Hong Kong, elderly people are over-represented in hospital populations. In the year 2006, the ≥65 age-group, which constituted 12.4% of the Hong Kong population (Figure 1.4a[19]), accounted for 49.0% of patient-days utilised in public hospitals

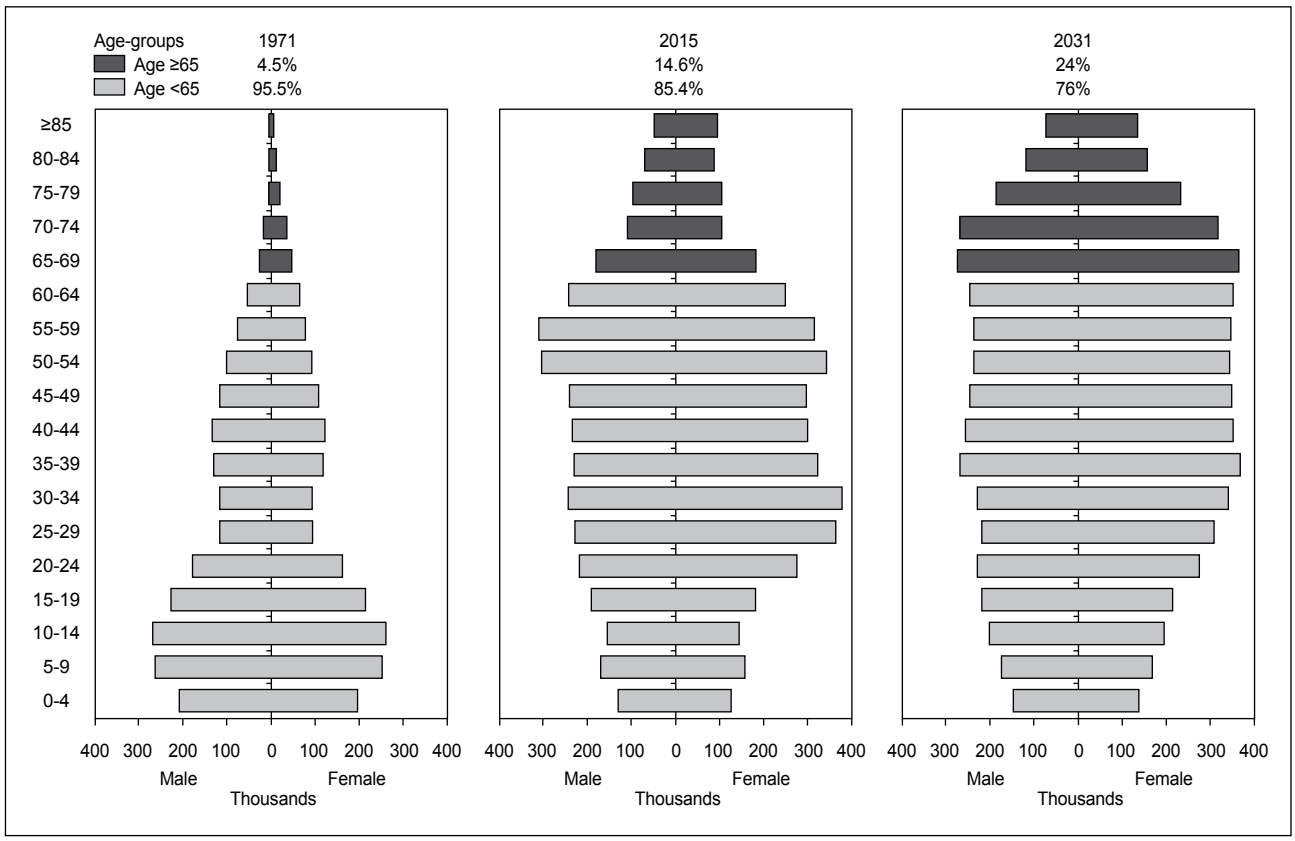

Figure 1.3 Hong Kong population age pyramids: 1971-2031[19]

(Figure 1.4b[21]). Within the ≥65 age-group, the hospital consumption per person per year rises rapidly with increasing age: those aged ≥85 years spend on average 10 days a year in hospital, compared with 4-5 days a year for those aged 75-79 years, and 2 days a year for those aged 65-69 years (Figure 1.4c). This contrasts sharply with the low hospital utilisation rate of under 1 day a year for adults aged <45 years.

Knowledge of these differential hospital service requirements of the age-sex distribution of elderly population is important when planning hospital utilisation, and draws attention to the rapidly expanding ≥75 age-group in future hospital planning for elderly services.[22,23] The hospital bed-days consumed by the ≥65 age-group is estimated to rise from 4.79 million to 8.78 million (an increase of 83%) between 2015 and 2031, with a corresponding rise for the ≥75 age-group from 3.43 million to 5.85 million (an increase of 71%). By 2031, the ≥65 age-group is projected to consume 69.1% of patient-days in public hospitals while the ≥75 age-group will consume 44.1% (Figure 1.5), a challenge to the hospital services in Hong Kong.[21]

1.2.3 Ageing in Asia

As shown in Plate 1.1, the relative size of the elderly population (aged ≥65 years) was largest in the developed countries in 2008, but by 2040, high proportions of elderly people are expected to be found throughout much of the less-developed world.[17] Growth of the elderly population from 2015 to 2030 is projected to be faster in Asian countries, at an annual growth rate of 3.5-4%, more than double that in developed countries (1.5-2%).[17] Ageing in Asia is particularly dramatic for China: between 2000 and 2050, the proportion of people aged ≥65 years is expected to rise from 7% to 23%. This grand demographic change poses a challenge to the healthcare of elderly people, who will number more than 332 million in 2050 in China, far greater than the combined elderly populations of North America, Europe, and Japan.[24,25] In 2008, Japan became demographically the oldest of the world's major nations: 21.6% of all Japanese were aged ≥65 years. The top two countries in terms of the absolute size of the 65 years-and-over population in 2008 were China and India at 106.1 and 59.6 million, respectively.[17]

Although societies in geriatric medicine have been established in a number of countries in Asia, the development of geriatric medicine as a specialty is lagging behind, and out of pace with the ageing population in the region. In 1998, China, Hong Kong,

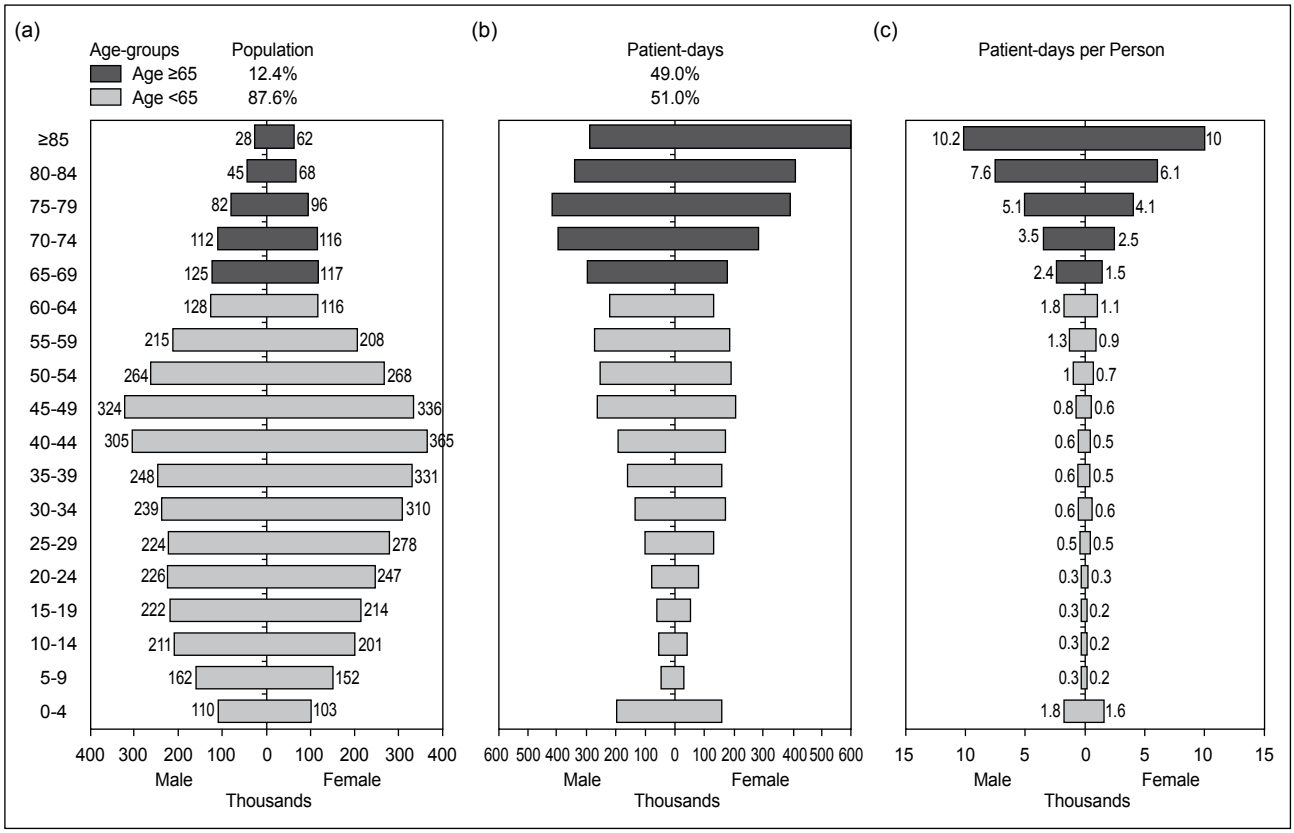

Figure 1.4 Hospital utilisation by Hong Kong population in 2006 by age-groups and sex. (a) Hong Kong population age pyramid 2006.[19] (b) Patient-days consumed in public hospitals by Hong Kong population in 2006.[21] (c) Patient-days consumed in public hospitals by each person in Hong Kong in 2006 (derived by dividing figures in Fig 1.4b by figures in Fig 1.4a)

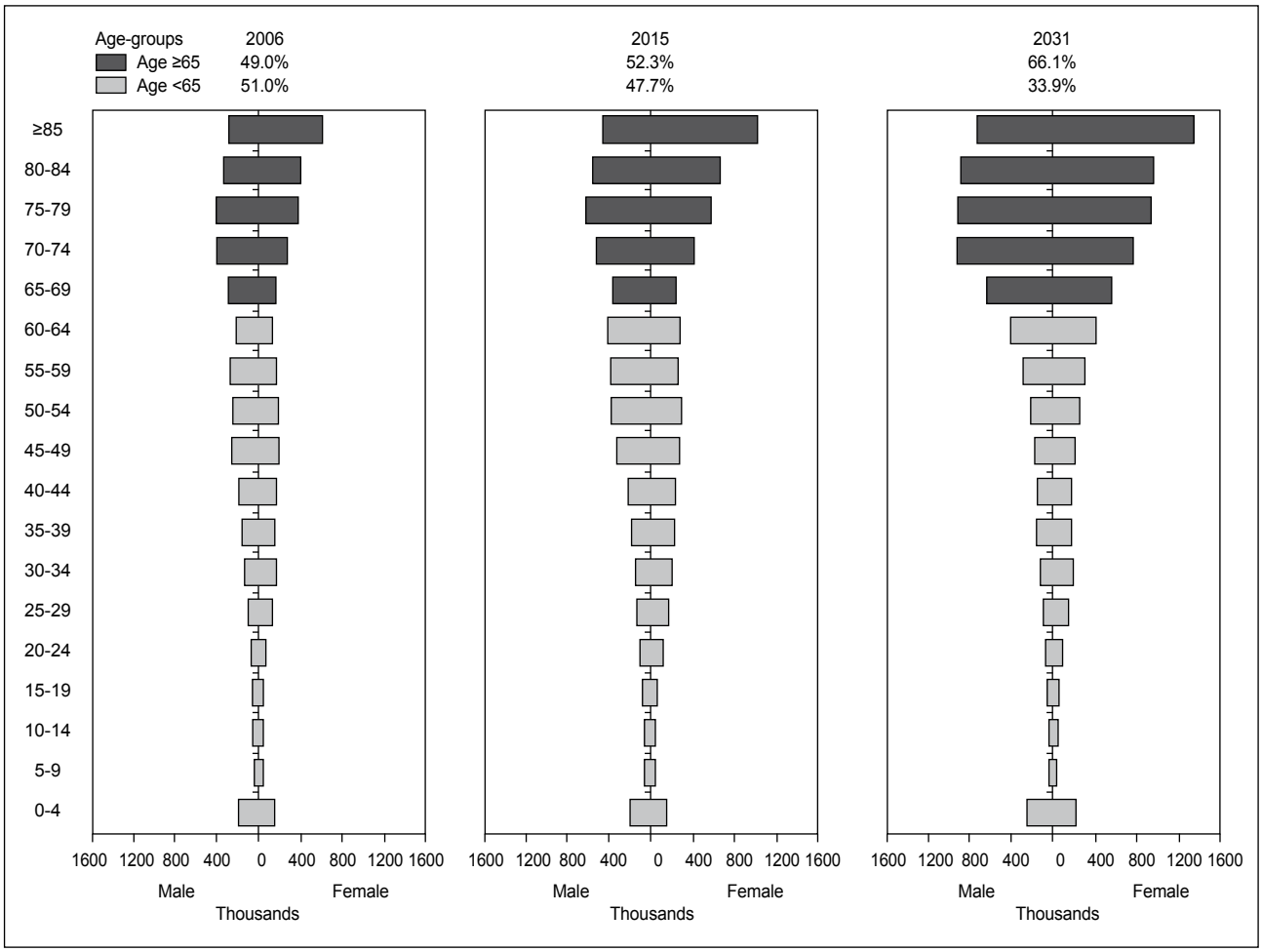

Figure 1.5 Hospital utilisation (patient-days) by Hong Kong population in 2006, and projected for 2015 and 2031[21]

India, Indonesia, Japan, the Philippines, Singapore, South Korea, Taiwan, and Thailand collaborated in a research study on the status of geriatric medicine in Asia. During the course of the study, the collaborators formed the Geriatric Medicine in Asia Working Group, with the objective to initiate and stimulate academic and collaborative activities among geriatricians / gerontologists in Asia, thereby promoting the development of geriatric medicine in Asia. The first informal meeting was held during the 6th Asia / Oceania Regional Congress of Gerontology in Seoul, South Korea in June 1999.[26] The collaborative study revealed that, except for a few countries, geriatric medicine was relatively underdeveloped in Asia. Important barriers to its development were identified: low perception, economic issues, and training issues. In 2005, geriatricians from Hong Kong, Malaysia, and Australia held an informal meeting in Hong Kong to prepare for the establishment of a network of geriatricians in the Asia-Pacific region. In 2006, the first Asia-Pacific Geriatric Conference was held in Kuala Lumpur, Malaysia. During the Conference, a network of geriatricians in the Asia-Pacific region was set up with the aim of promoting development of geriatric medicine in the region. Australia, China, Hong Kong, Indonesia, Malaysia, the Philippines, Singapore, South Korea, Taiwan, and Thailand have joined the network and regular Asia-Pacific Geriatric Conferences have been held ever since.

1.3 Biological Ageing and Its Clinical Implications

1.3.1 Frailty and Geriatric Syndromes

Demographic trends and the age distribution of illness make it unrealistic for one specialty, geriatric medicine, to care for all elderly people. The hallmark of ageing is heterogeneity, aptly described by Bernard Isaacs, "Most old people are young people in old bodies. Some young people are old people in young bodies." The spectrum of elders from the fit to the frail is illustrated in Figure 1.6, and the challenge of health and social care in the face of an ageing population is to minimise the conversion from fitness to frailty and

hence the proportion of frail elders.

What kind of elderly people, therefore, should geriatricians ideally manage? The best definition of a geriatric patient is one who exhibits the features of biological ageing.[27] The term "biological ageing", in contrast to "chronologically ageing", is emphasised because not all patients age at the same rate. Such biologically aged patients are characterised by frailty and complexity.

Frailty[28] is a failure to integrate responses in the face of stress, so that functions that require integration of higher brain processing, such as maintaining balance, walking, and intellect, are more likely to fail when stressed by disease. This results in the four "I's" of geriatrics — immobility, instability (falls), intellectual impairment (confusion), and incontinence — the "giants of geriatrics", a term coined by Bernard Isaacs[29] to embrace the "common final pathway" of the clusters of acute and chronic diseases that frequently afflict very old people, with a gigantic impact and yet often ignored. These "I's" of geriatrics have subsequently been expanded by geriatricians to include the geriatric syndromes[30-33] commonly encountered in clinical practice; they share common social risk factors and clinical characteristics (Table 1.1). Early recognition of these geriatric syndromes by geriatric assessment will reduce the development of disability in elderly persons (see Chapter 2).[33]

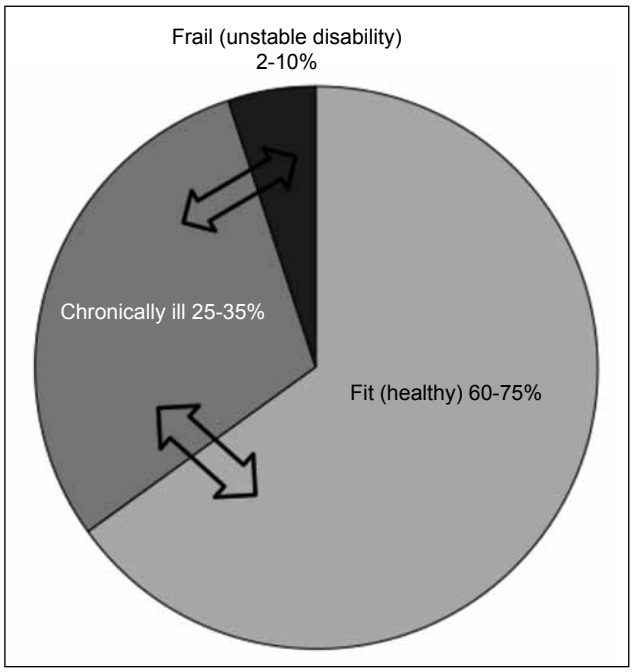

Figure 1.6 Heterogeneity in an elderly population

Because of declining homeostatic mechanisms and adaptive capacity, frail elderly persons readily lose equilibrium when stressed by illness, drugs, or adverse social factors and present with functional decline (Figure 1.7). The consequences of biological ageing are complex but the most important clinically relevant aspects are: atypical presentation of disease, multiple pathology and multiple aetiologies, disabilities, and adverse social factors.

1.3.2 Atypical Presentation of Disease

Although diseases in fit elders may present with classic features of a single system disease, similar to those of their younger counterparts and described in textbooks for younger adults, altered presentations are common among frail elders, often described as "atypical" when referenced to younger adults.

1.3.2.1 Geriatric Presentation

A disease in a frail elder may result in decompensation with onset of one or more of the major geriatric

Table 1.1 Geriatric syndromes: their social risk factors and common features[29-33]

Geriatric syndrome	Social risk factor	Common feature
Original geriatric giants[29] • Immobility • Instability (falls and fractures) • Incontinence • Intellectual impairment (cognitive dysfunction, delirium, dementia)	• Isolation • Ignorance • Impecunity (poverty) • Informal care with inadequate support	• Gigantic number • Gigantic impact (loss of independence) • Complex (multiple causes, no simple care) • Inter-related and interacting • Ignored (inverse care law)
Subsequent additions[30-33] • Iatrogenesis • Impaired vision / hearing • Immune deficiency • Ill-treatment (inadequate care, elder abuse) • Irritable colon • Insomnia • Impotence • Frailty • Sarcopenia (muscle dysfunction) • Failure to thrive (malnutrition, anorexia, weight loss) • Depression		

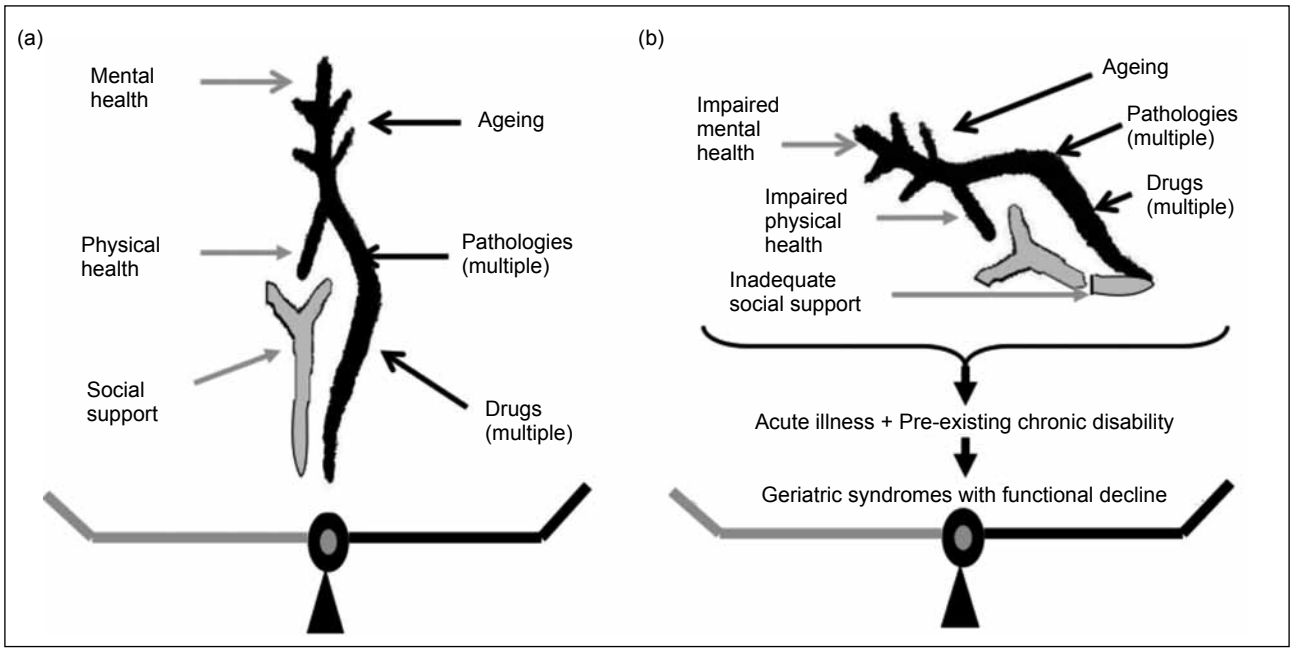

Figure 1.7 The frail elderly figureperson, represented by (a) the ancient Chinese character for "old", is in unstable equilibrium, converting to (b) the biologically aged patient when stressed

syndromes (Table 1.1). The global manifestation, e.g. confusion, falls, may overshadow features of the focal disease, e.g. pneumonia, myocardial infarction, and divert the focus of the attending doctor from the probable diseases that underlie these global manifestations, e.g. ordering a computed tomographic brain for confusion, X-rays for injuries after a fall.

1.3.2.2 Silent Presentation

A depressed inflammatory response and impaired visceral pain perception — secondary to ageing, disease and drugs — may mean that infections in old age are associated with an absent, blunted, or delayed fever response; myocardial infarction and peptic ulcer can be painless. Nonetheless, Roy Fox has cautioned that "Illness is silent only because the physician is not listening. The patient may be screaming, 'I have pneumonia', but the physician is deaf. It is an acquired deafness from the influence of formal training and the society in which we live."[34]

1.3.2.3 Pseudo-silent Presentation

In the pseudo-silent presentation,[35] an elderly patient's complaints and change in status are falsely attributed to ageing or social problems. Problems that require medical treatment are not entered into a careful diagnostic approach but managed with social measures, such as referral for institutional care. The fallacy of the prejudicial label "social problem" is best expressed in Bernard Isaacs' words: "If a patient falls and breaks his femur, that is a medical problem. If a patient falls and does not break his femur, is that a social problem? Elderly patients are admitted to hospital not because of social problems, but because of medical problems with social consequences, or social problems with medical consequences."[29] Research has revealed the high prevalence of undiagnosed medical conditions among elderly people prior to entry to care homes.[36,37]

1.3.2.4 Masked Presentation

Presentations of diseases may also be masked by the common occurrence of multiple pathologies in old age (e.g. diagnosis of tuberculosis masked by pre-existing lung cancer or heart failure), and the tendency to prescribe symptomatic treatment without an exact diagnosis, e.g. non-steroidal anti-inflammatory drugs–masked gout, culture-negative infective endocarditis, B_{12} deficiency masked by symptomatic blood transfusions.

1.3.2.5 Missed Presentation

The altered presentation of diseases in old age accounts partly for the literature reports of very late diagnosis of common diseases in old age,[38] at autopsy,[39] or retrospectively after an infectious outbreak had occurred with disastrous consequences.[40,41]

1.3.3 Multiple Pathologies and Multiple Aetiologies

Elderly people often have multiple pathologies, accumulated from a young age or developing in old age. Apparent inactive past medical problems can be relevant to an elder's present illness, e.g. an elder

who adapted well to his past poliomyelitis would have coping problems after a stroke, intolerance to aspirin may result from the development of a stomal ulcer from previous gastrectomy. While multiple pathologies can result in multiple complaints, a single complaint may well be due to multiple aetiologies, e.g. dyspnoea due to the coexistence of anaemia, pulmonary tuberculosis, and heart failure. Ultimate improvement in dyspnoea thus requires diagnosis and treatment of the underlying multiple aetiologies. Organ system specialisation may mean that an elder with several pathologies is attended by several specialists, and prescribed multiple medications; the consequent drug-drug and drug-disease interactions may have unfavourable outcomes for a frail elder.

1.3.4 Disability

In addition to common disabling illnesses in old age such as arthritis, strokes, and fractured hips, the consequent geriatric syndromes often interact and result in further deprivation of independence and functional decline. Multiple pathologies and the global impact of focal disease add to the burden of chronic disabling disease in old age, while the presence of adverse social factors further increases the handicap.

1.3.5 Adverse Social Factors

Research by medical sociologists has shown that the presence of any one of isolation, ignorance, and poverty has an adverse effect on ageing. Geriatric syndromes may arise from the interaction of medical diseases with breakdown of social support systems. With the trend towards community care for elders, Challis et al[37] have warned that an emerging geriatric giant is "informal care". Proper preparedness is much needed in community care support so that "informal care" is not replaced by another "new" geriatric giant, "inadequate care" or elder abuse.[42]

1.4 Geriatric Approach

1.4.1 Limitations of the Single Organ-system Approach in the Care of Biologically Aged Patients

In contrast to the younger adult in whom presenting problems can often be explained by one single disease (the dictum "one patient, one disease"), the biologically aged patient characteristically has multiple causes for their presenting problems. Ageing, disease, and treatment interact with one another to modify the pattern of illness (Figure 1.8). Economy of diagnosis or trying to link all problems to a single aetiology cannot be the guiding principle when treating an elderly individual. Because of their complexity, geriatric problems are seldom amenable to a simple solution, and are often frustrating to those tuned to singular presentations capable of a single diagnosis. Thus, a biologically aged patient may have multiple complaints without a single major complaint, and even a major complaint may not relate to the most overt or most serious identifiable disease. Pitfalls in diagnosis[29] (Table 1.2[43]) and management are prone to occur if a geriatric perspective is not taken, resulting in abusive diagnostic labels like "senility" and "social problem" instead of exact diagnoses, as well as the tendency to prescribe symptomatic treatment instead of treatment targeted at underlying causes. Such symptomatic treatment may even carry the risk of iatrogenesis in acutely hospitalised elderly patients (Figure 1.9).

1.4.2 Model of Care for Biologically Aged Patients

The response to the complex situation of the biologically aged ill person (with multiple diseases, multiple needs, and a multifactorially determined state) must be correspondingly complex. It is not sufficient to identify acute illnesses and deal with them medically in a narrowly defined sense. The essence of geriatric medicine as a specialty is to assess and treat the medical and rehabilitative needs of older people. Every ill person deserves a diagnosis. This is carried out through a process known as CGA. In simple terms, CGA is the process of knowing a frail elderly person, whose health is a delicate balance (Figure 1.7) of factors that impair well-being (ageing,

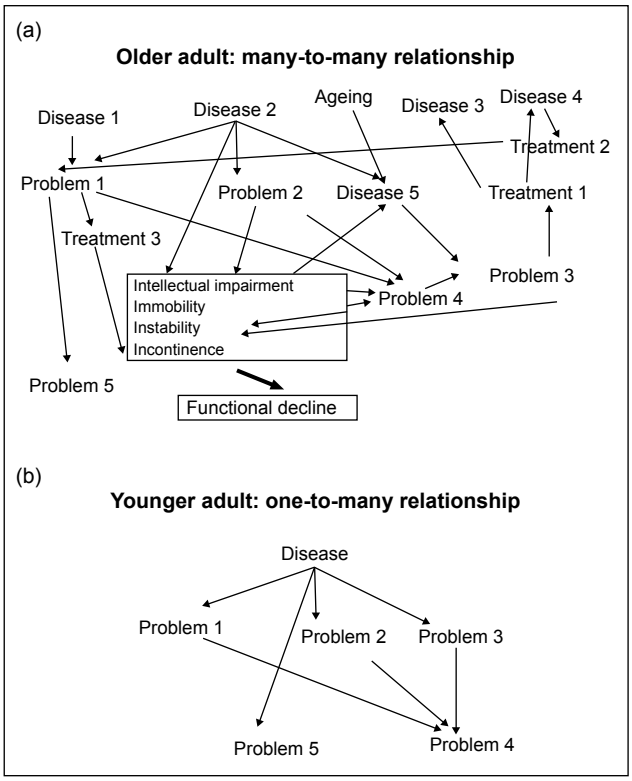

Figure 1.8 The complexity of illness in (a) the older adult compared with (b) the younger adult

Table 1.2 Diagnostic pitfalls in old age[43]

History taking	• "Poor historians"[43] • Multiple "vague" complaints • Attempting to get a "typical history" • Ignoring geriatric syndromes • "Atrophy of the question mark" (doubt converted to certainty) • Ignoring old notes • Neglecting medication history • Ignoring functional assessment • "Social / placement" problem • Neglecting relatives / carers interview • Ignoring the elder and asking relatives / carers only
Physical examination	• Over-interpretation of normative changes of ageing (e.g. crepitations) • Areas infrequently inspected (e.g. pressure sores, napkin rash, toe nails, soles, mouth / dentures, eyes and ears, palpable bladder, rectal examination [for faecal impaction], scars) • Not examining the patient out of bed (gait)
Investigation	• Under-investigation (e.g. under-use of biopsy / histological confirmation) • Over-investigation ◦ Over-reliance on investigations (especially diagnostic imaging) ◦ Use of "screening" biochemical test battery • Interpreting tests in old age ◦ ? Normal for population ◦ ? Normal for individual ("comfort range") ◦ False-negative finding may be due to faulty procedure ◦ Abnormal finding may not be clinically significant / relevant ◦ Age-related changes

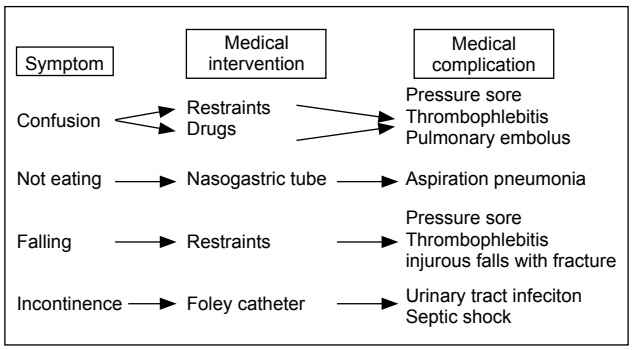

Figure 1.9 Complications of acute hospitalisation

multiple pathologies, multiple drugs) and those that promote well-being (mental health, physical health, and social support). CGA requires an assessment of the four domains of functional ability: physical health, cognitive and mental health, and socio-environmental factors (see Chapter 2).

Although geriatric medicine originated from the long-term care setting, its success in tackling the problems of frail elders led to the earlier involvement of geriatricians in the acute phase of their illness. In the progressive care model, separate acute / rehabilitation / long-term settings and facilities are made available for patients at different stages of their illness and treatment programme.[44]

The acute geriatric patient can be defined on a need-related basis as[27]: residents of residential care homes for elders (signifying a degree of physical and / or mental frailty); acute presentation with confusion, poor mobility, falls or incontinence; long-standing confusion, poor mobility, falls or incontinence; presenting with specific conditions that require intensive rehabilitation, e.g. stroke; multiple pathologies and thus multiple medications; and those previously under the care of a geriatric specialist.

Effective CGA programmes in the acute care of a geriatric patient will require a multidisciplinary team, involving not only doctors and nurses but also therapists and social workers, working closely together, and tight coordination of hospital-based and community-based resources. This kind of organisation of services is rarely found in acute medical or surgical wards where the emphasis is, understandably, on the management of acute illness based on single organ resuscitative treatments and single interventions. The integrated medicine and geriatrics model, as practised in many local hospitals, will not work properly without a supportive ward environment and multidisciplinary team. Pooled or scattered beds are not the answer, as this will fragment the multidisciplinary team and undermine the close working relationships that are essential to the care required by geriatric patients.[21,45]

1.5 Geriatric Medicine and Geriatric Services

Geriatric medicine is the branch of general medicine concerned with the healthcare of older people.[46] The Royal College of Physicians has defined geriatric

medicine as follows: "It is in the nature of illness in elderly people that several diseases may co-exist, interact, and manifest in non-specific and atypical ways, social and psychological problems may be prominent, functional handicap readily appears, and recovery may take longer than in younger people. The essence of geriatric medicine is to establish what the problems are, identify their causes, treat conditions that are treatable, correct deficiencies that can be corrected, and compensate for what cannot be corrected."[47] As evident from above, the major reason for the establishment of the geriatrics specialty is not simply an increase in numbers of old people, but rather an increase in knowledge of old people, and the need for a special attitude and organisational skills.[48] Geriatric medicine emphasises a problem-based, rehabilitative, and holistic approach (Table 1.3); by caring for a whole person (not just his / her parts) interacting with his / her environment (physical as well as psychosocial) over a period of time (not just an acute episode in the hospital). The model of care in geriatric medicine shifts from diagnosis to functional assessment; cure to care; foretelling prognosis to evaluating goals; and from technology to the human touch with interdisciplinary team management. The expertise of geriatricians is therefore 3-fold: first, awareness of the different presentation, course, and management of the illness in the biologically aged person; second, the organisation of hospital-based services appropriate to such patients; and third, the coordination of health and social services. Because the model of care in geriatric medicine differs from that of general medicine, the audit mechanisms of a geriatrician should differ from those of a general physician.[49,50] In addition to an extra emphasis on the geriatric problems of falls, incontinence, memory impairment, and mobility, geriatricians must also focus on specific areas: failed discharge; death audit including identification of unexpected problems and sympathetic terminal care; long-term care and nursing home care; use of standardised assessment scales; and geriatric day hospital care.[49-51] A multidisciplinary team approach is required in the audit of these special areas of concern, since elderly patients have complex medical and social problems that demand long-term care and commitment.[50,51]

The aims of a geriatric service are: maintenance of health in old age by continuing social engagement and avoidance of disease; early detection and appropriate treatment of disease; maintenance of maximum independence consistent with irreversible disease and disability; sympathetic care and support during terminal illness. The spectrum of geriatric service is outlined in Table 1.4 and the service components further discussed in other chapters. To match the principle of progressive care (acute / rehabilitation / long-stay) in geriatric medicine, Peter Millard has proposed a multi-compartment model to evaluate geriatric hospital services,[52,53] by taking into consideration the dynamic interactions of the three compartments of geriatric care (acute, rehabilitation, and long-stay). It has also been emphasised that outcome evaluation should also take account of the different case-mix and functional levels of elderly patients, as heterogeneity is their hallmark.[54] Relevant outcome indicators for geriatric medicine and geriatric services include improved or

Table 1.3 Holistic approach in geriatric medicine

Wholeness in		Interaction of	Special approach
• Person	• whole body	organ-system (not just her parts)	Need for a generalist approach / specialist knowledge
	• whole person	body (physical), mind (psychological), and soul (spiritual)	Need for special attitude, humanistic / maternalistic approach
• Space	• whole environment	person-environment (physical [spatial], social [human relationships], chemical [drugs])	Need for CGA / multidisciplinary approach Continuing care: from hospital to community
• Time	• whole life history	life events and illnesses	Progressive care: acute / rehabilitation / long-term care

Abbreviation: CGA = comprehensive geriatric assessment

Table 1.4 Spectrum of geriatric service

- Acute assessment and care (need-related, age-related, integrated)
- Rehabilitation (e.g. stroke, Parkinson's disease, falls, arthritis, chronic lung disease, complex frail elders)
- Long-term care (infirmary)
- Liaison geriatrics (psychogeriatrics, orthogeriatrics, oncogeriatrics, liaison with emergency department)
- Geriatric specialist outpatient clinic
- Special services / clinics (e.g. memory, falls, continence, stroke)
- Geriatric day hospital
- Community geriatric services (community geriatric assessment service, integrated discharge support programme)

preserved function, home as the discharge destination (previous accommodation), and quality of care from the patients' perspective.

1.6 Issues and the Future of Geriatric Practice

The following issues must be addressed if optimal healthcare of the ageing population is to be provided: attitudes, capacity, needs matching, collaboration, and integration.

The prevailing reaction to ageing demographics has been negative, often seen as a burden or as a problem of intergenerational tension and resource competition rather than a blessing of intergenerational harmony. Negative attitudes and behaviour towards elderly patients in health and social care sectors have been exposed by the mass media as scandalous care and received much publicity. As geriatric medicine originates from neglect and a compassion for the "unfittest,"[55-58] geriatricians should take the lead as role models to give loving care to elderly people and imbue all our staff and colleagues with a positive attitude towards their care.[59] Treating people as individual human beings and responding to their needs with compassion and sensitivity will enhance patient dignity.[60] Nonetheless, dignified care of elderly people in hospital is frequently compromised by systemic and organisational factors, including an intense focus on efficiency and timely discharge; continually moving elderly patients with multiple pathologies around multiple specialist wards; risk aversion without considering an appropriate balance between risk-taking and promoting individual autonomy.[60] From an elderly person's perspective, person-centred care for elderly people needs to be specifically related to communication, privacy, personal identity, and feelings of vulnerability.[61]

The results of good treatment such as reduced mortality from fractured hip, acute myocardial infarction, stroke and pneumonia, means more surviving patients with chronic problems such as heart failure, stroke, and chronic obstructive pulmonary disease. This, together with the ageing demographics, will increase the demands made on healthcare and hospital capacity (Figure 1.5), and is most felt during winter crises and flu outbreaks. Rockwood and Hubbard have criticised modern healthcare for a common error in addressing the needs of complex frail elderly patients who are acutely ill: instead of getting to grips with how service is provided, they want the frail old people to go away, to some more appropriate place.[28] The response to dealing with elderly people with complex problems is not to pack them into emergency rooms, or admit them straight to nursing homes, or to call them names, but to recognise that they have complex problems and to understand and promulgate how to treat these problems,[62] to embrace the complexity of the patients and their need for care.[28] Moving frail elderly patients quickly through the system without addressing their problems and needs will result in chaotic flow, the revolving-door phenomenon, and overcrowding.[53,63] Peter Millard[63,64] has advocated appropriate assessment and rehabilitation at critical time points to maintain a regulated and goal-directed flow between the acute, rehabilitative, and long-term care of complex elderly patients with the needs and goals of patients matched to appropriate staff-mix and environment (Figure 1.10[64]). This science of tending the aged (Gerocomy) has been grounded on mathematical modelling of elderly patients flowing through acute, rehabilitation, and long-term care compartments.[63,64] Getting the total bed capacity right will depend on appropriate division and allocation of beds to the three compartments.

The fact that younger and fitter patients have short stays in hospital and older, frailer and complex patients have longer hospital stays results in the predominance of older frail patients in the modern hospital inpatient population. A high proportion of these complex elderly patients have the geriatric giants of falls, immobility, confusion, impaired cognition, incontinence, and iatrogenesis, have multi-pathology and multi-morbidity illnesses, and are physically dependent with complex social problems.[65,66] These geriatric conditions often contribute to their need for acute admission, and influence hospital course and discharge plans; but organ failure (e.g. heart failure, respiratory failure) is more readily diagnosed than high-order-system failure (e.g. balance failure [falls], acute brain failure [delirium]). The needs of these complex frail elderly patients can hardly be matched by a hospital system and environment based on specialties of organs and diseases.[28,67] To achieve optimal hospital service provision for acutely ill frail elderly patients with

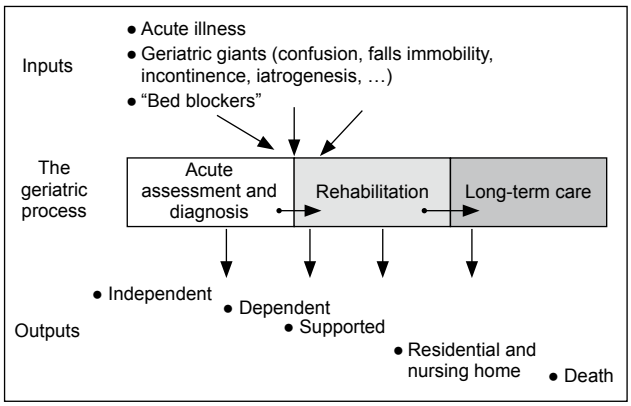

Figure 1.10 Regulated and goal-directed flow in geriatric service (adapted from Millard et al[64])
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multiple conditions, a right balance needs to be struck between narrow specialism and broad-based geriatric holistic care. This translates to the practical issue of access to the expertise and care of a geriatrician and interdisciplinary team.[45] Future generations of doctors, nurses, and allied health professionals need to be prepared by adequate exposure and training in the core elements of geriatric medicine.

For elderly patients to have timely access to the expertise of a geriatrician, healthcare need to be geared towards close collaboration between geriatricians and family physicians in the community setting, and in the hospital setting, a joint working relationship between geriatricians and other hospital specialists, with liaison between geriatrics and orthopaedics, surgery and its subspecialties (e.g. urology, neurosurgery), psychiatry, oncology, and the emergency department.

Elderly patients are at high risk of receiving fragmented and disorganised medical and social care from multiple care providers because of their multiple medical conditions, social precariousness, and frailty. An optimal geriatric service should be able to connect and organise service provision across different service providers in an integrated manner, especially during the hospital-community transition, breaking down health-social barriers with seamless sharing of information about patient care.

In Hong Kong, a proposal paper for enhancement of future acute geriatric care to meet the needs of an ageing population was presented by the Geriatric Subcommittee representative and discussed in the Medical Service Development Committee of the Hospital Authority in 2004.[21] In 2011, the Hospital Authority published its Elderly People Service Plan[68] and in 2012, the Strategic Service Framework for Elderly Patients[69]: the directions for healthcare services for elderly people have been formulated from single specialty to cross-specialty / multidisciplinary; from variable quality care to agreed standards; from fragmented services in acute and community care to integrated services for patient transitions; from institutional episodic care to continuous care appropriate to individual patient needs; and from dependent patient to empowered patient.

In their lectures in memory of the early pioneers of geriatric medicine, Colin Powel and John Grimley Evans shared respectively their views on the challenges facing future geriatrics.[70,71] To the first principles of geriatrics (adequate assessment, accurate diagnosis, appropriate treatment, aftercare, advocacy for patient and specialty) laid down by Marjory Warren, Colin Powel added two further principles for future geriatrics: alliance and accountability.[70] Alliance refers to evolution from multidisciplinary teams to interdisciplinary teams, from solo practice to professional joint practice, and ultimately partnership with patients and an effective relationship with others, both inside and outside of geriatric medicine. Accountability refers to serving frail elderly people, moving towards a frail-friendly healthcare system, and ultimately being accountable to society.[70] John Grimley Evans concluded in his Ignatius Leo Nascher Lecture, "The first century of the specialty saw principles of clinical practice and service design for the specialty established throughout the developed world. The challenges for the second century are of a different order, and dominated by the ethics, politics and economics of ageing societies in which ageist prejudice is still firmly entrenched. In the tradition of advocacy for older people established by Nascher, geriatricians have a duty to encourage their nations to respond humanely and rationally to the new demography."[71]

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CHAPTER 2

Comprehensive Geriatric Assessment

Tak-kwan KONG

OBJECTIVE

To learn the meaning of comprehensive geriatric assessment (CGA), its domains and commonly used assessment instruments, as well as its application in clinical programmes.

KEY POINTS

- CGA is a diagnostic process of getting to know an elderly individual in all dimensions; it is the cornerstone of geriatric medicine.
- Key domains in the assessment of elderly patients include physical health, functional capacity, mental function, socioeconomic resources, and environmental resources.
- Useful information can be acquired from both elderly patients and their family / friend / caregiver.
- When caring for a frail old person with multiple illnesses, multiple medications and complex needs, we have to ask the right questions. Instead of asking just where they should go and who is responsible, we should work together and ask who are they, and how we can help satisfy their needs.
- Dual sensory impairment in vision and hearing is associated with cognitive and functional decline and increased mortality.
- To identify early cognitive impairment, a combination of patient-based and informant-based screens are the most appropriate approach.

2.1 Introduction

The simultaneous presence of multiple interacting problems (physical, psychological, and social) and the unmet needs of a frail elderly patient require an assessment more complex than that provided by a routine medical diagnosis. Comprehensive geriatric assessment (CGA) is a multidimensional interdisciplinary diagnostic process intended to determine a frail elderly person's medical, psychosocial, and functional capabilities, and problems and aid in the development of a coordinated and integrated plan for management and long-term follow-up.[1] CGA forms the basis of a frail elderly person's treatment and rehabilitation plan. The goals are to reduce pain, improve function, delay death, and ultimately improve quality of life. In contrast to high technology, CGA emphasises high touch. It extends beyond the patient to the individual, from diagnosis to assessment, and from treatment to management. CGA, coupled with multidisciplinary care, has become the cornerstone of geriatric medicine and geriatric care systems.

2.2 Evidence of the Effectiveness of CGA

Early pioneers of geriatric medicine such as Marjory Warren and Lionel Cosin introduced the concept of CGA in the 1930s on the basis of faith and optimism. Today, CGA programmes have evolved and been the subject of scientific scrutiny in the era of evidence-based medicine. Since the late 1970s, controlled trials have evaluated the effectiveness of CGA with consequent publications of positive results in the 1980s.[2] A 1993 meta-analysis by Stuck et al[3] of 28 controlled trials of CGA programmes that involved hospital units, hospital consultation teams, in-home assessment services, outpatient assessment services, and hospital-home assessment programmes demonstrated the benefit of CGA in terms of reduced mortality risk, improved likelihood of living at home, reduced hospital readmissions, greater chance of cognitive improvement, and greater chance of physical function improvement. Since then, major international conferences have been held to discuss this new concept of CGA,[4] and further controlled studies and meta-analyses have confirmed the favourable outcomes.[5-10]

2.3 Complications and Costs of CGA

Although beneficial, there are concerns about CGA. Complications can occur when it is overused or abused. An example is delirium as a result of fragmented and duplicate assessment by different

professions.[11] CGA should be performed in the clinical setting with due regard to the patient's tolerance and well-being with appropriate clinical interpretation of the assessment results. Another concern, notably from hospital management, is the costs associated with the involvement of multiple disciplines. Wieland[12] reviewed 19 randomised controlled trials that reported the cost endpoints in CGA, and concluded that CGA was cost-efficient (less cost for same outcome or same cost for better outcome) for the majority, and cost-effective for a few. A related issue is not to invest in programmes that are not effective. It is therefore important to observe the organisational elements of CGA that are associated with effective programmes, as concluded from previous meta-analysis and reviews of CGA (Table 2.1).[2,12]

CGA programmes can fail if they target individuals who are too healthy to derive benefit or if they fail to identify the frail. To be effective, assessment must be linked with management, called Geriatric Evaluation and Management in the US. Returning elderly patient to a previous care environment may not be feasible if iatrogenic problems are identified during assessment. The three essential components of an effective CGA programme are therefore:

- identifying or targeting a frail elderly individual;
- assessment of the individual and consequent recommendations for care; and
- implementation of the recommendations by a geriatrician and / or interdisciplinary team

2.4 CGA Programmes

2.4.1 CGA within the Hospital

In-patient CGA programmes have been developed for both the acute and the post-acute setting. An acute care for the elderly unit is designed to cater for the special needs of acutely ill elderly inpatients, combining CGA with interventions such as interdisciplinary care for geriatric syndromes in an age-friendly environment, as well as discharge planning and support. CGA programmes in the post-acute environment focus on the continuation of post-acute care and rehabilitation of elderly inpatients by an interdisciplinary team. The interdisciplinary team conference is crucial to the success of an inpatient CGA programme. Meta-analyses have demonstrated the effectiveness of such programmes in both the acute and post-acute rehabilitation setting.[5-7] A 2011 Cochrane meta-analysis of 22 trials involving 10315 participants in six countries concluded that inpatient CGA programmes increased a patient's likelihood of being alive and resident in their own home following an emergency admission to hospital, especially if they were cared for on a ward that performed CGA, and were also associated with a potential cost reduction compared with general medical care.[8]

Table 2.1 Organisational elements of comprehensive geriatric assessment associated with effective programmes[2,12]

- Targeting the frail
- Interdisciplinary team structure
- Comprehensive / multidimensional geriatric assessment
- Management with clinical control of treatments and care
- Long-term follow-up

2.4.2 CGA and the Hospital-home Transition

CGA that targets elderly patients at high risk of recurrent heart failure after hospital discharge, combined with post-discharge follow-up at the hospital and at home has been shown to reduce hospital readmissions, improve quality of life, and reduce net cost.[9,13]

2.4.3 CGA in the Community

In Hong Kong, CGA in the community is performed by geriatric day hospitals (GDHs) and community geriatric assessment teams (CGATs). Stroke patients are the major users of GDHs. Physical function in terms of self-care, mobility, and household function has been shown to improve following discharge from a GDH.[14] CGATs were established in Hong Kong in 1994 to enhance and preserve the health and quality of life of elderly persons in the community by timely assessment and appropriate management. At its inception, CGAT services included assessment prior to admission to subvented care homes or hospitals, outreach geriatric clinics for care homes, hospital discharge support services, and geriatric home care. The effectiveness of CGAT in supporting frail elders in the community is evidenced by the lower hospital utilisation by care homes with CGAT support in terms of accident and emergency (A&E) department attendance, total hospital admissions, and total hospital bed-days, with relative reductions of 24%, 24%, and 43%, respectively (Figure 2.1).[15]

2.4.4 CGA at the Hospital Interface: Gate-keeping Versus Goal-keeping in Caring for the Frail Old

For the frail old with multiple problems, the question is often asked, "where should s/he go, whose responsibility is this?" In a time of rationing and rationalisation, modern healthcare tends to answer with gate-keeping that is primarily resource-driven, leaving less room for goal-keeping that is needs-led (Figure 2.2a). This was aptly described by the Canadian geriatrician, Professor Kenneth Rockwood,[16] "Modern health care needs to reconcile itself to complex patients. There are many wrong ways to address this, each of which has the following

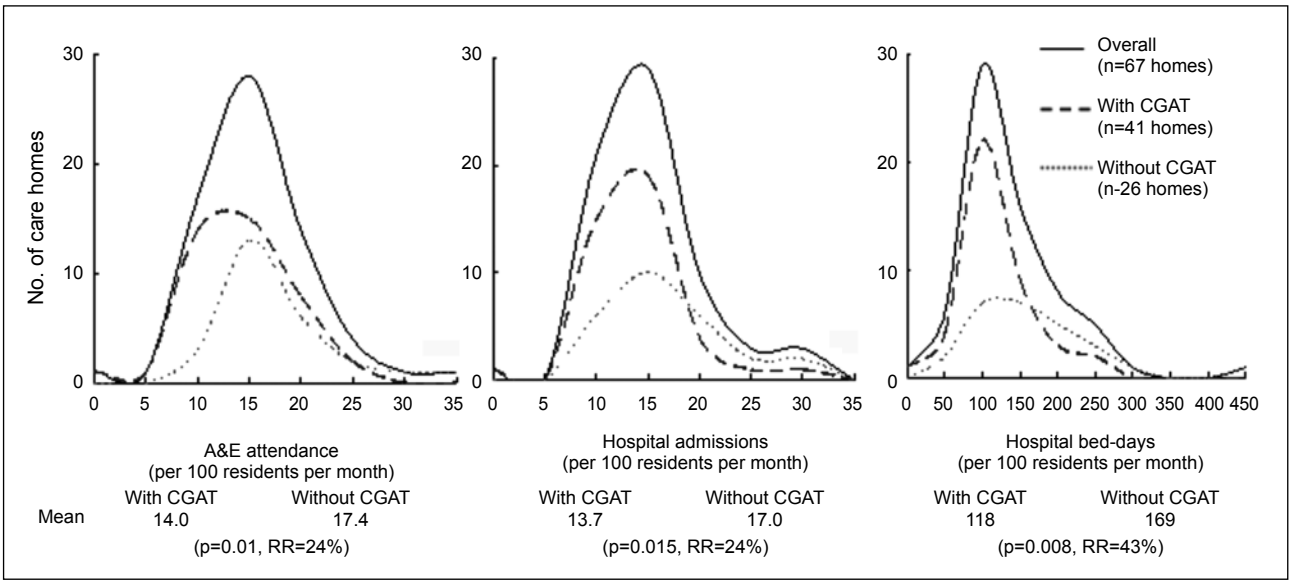


Figure 2.1 Effectiveness of community geriatric assessment team (CGAT) in reducing hospital utilisation by care home residents, based on survey data (November 2004 to January 2005) for Princess Margaret Hospital from Hospital Authority, Hong Kong[15]
Abbreviations: A&E = accident and emergency department; RR = relative risk

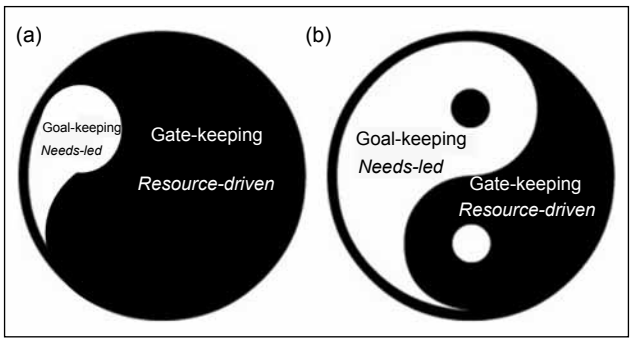


Figure 2.2 (a) Gate-keeping and goal-keeping dichotomy and (b) their harmonisation through comprehensive geriatric assessment

in common: instead of getting to grips with how service is provided, they want the frail old people to go away, to some more appropriate place."

Nevertheless there is evidence that with CGA and effective organisational elements mentioned above, goal-keeping can be harmonised with gate-keeping, whereby goal-keeping can lead to gate-keeping with a reduction in resource utilisation, and gate-keeping can result in goal-keeping with attention to the needs of elders (Figure 2.2b). Evidence to support such cost-effective CGA programmes has been shown in a number of settings. First, CGA prior to entry into a care home has enabled detection of treatable previously undiagnosed illnesses, improved physical function, alleviation of the need for care home placement, and reduced total health and social costs.[17,18] As of 2014, 6.1% of elders aged >65 years in Hong Kong were resident in a care home. Timely introduction of specialist assessment prior to care home entry locally may help to reduce this relatively high institutional rate with important implications for quality and costs of elderly healthcare. Second, the benefit of CGA has been shown at A&E departments. Attention has been drawn to the problems of A&E in managing frail elders.[19,20] Studies of CGA at A&E have shown enhanced function and reduced use of care homes without increased cost.[21-23] Studies of patients who present to A&E following fall also highlight the importance of CGA in this area, with reduced serious injury and subsequent bed-day utilisation.[24-26] Third, it has been shown that CGA reduces serious adverse drug reactions while reducing suboptimal prescribing.[27] Inappropriate medication and adverse drug reactions are important causes of hospital admission of elders.[28,29] Thus CGA that targets polypharmacy can be both goal-keeping in improving the medical care of elders and gate-keeping in reducing iatrogenic hospitalisations.

2.5 CGA Domains and Instruments

The World Health Organization and the British Geriatrics Society have recommended the following domains for comprehensive assessment of elderly patients: physical health; functional capacity; mental function; social resources; economic resources; and environmental resources.[30,31] A number of validated instruments for geriatric assessment have been developed and published since the 1960s on these various domains (Table 2.2[32-91]).

Table 2.2 Domains and instruments of comprehensive geriatric assessment[32-91]

Domain	Instrument / test
Physical health	
Vision	Snellen Visual Acuity Test, visual function assessment instruments[32-36], Melbourne Edge Test,[37] visual field, Albert's line cancellation test for visual neglect[38,39]
Hearing	Screening Version of Hearing Handicap Inventory for the Elderly (HHIE-S),[40-42] Whispered voice test[43]
Swallowing	Water swallow test[44,45] (see Chapter 43)
Nutrition	Mini Nutritional Assessment (MNA)[46,47] [see Chapter 30], Simplified Nutritional Assessment Questionnaire (SNAQ)[48]
Medication	GerontoNet ADR risk score,[49] Medication appropriateness index,[50] Morisky 8-item medication adherence scale[51] (see Chapter 15)
Comorbid conditions and disease severity	Charlson Comorbidity Index,[52] Cumulative Illness Rating Scale–Geriatrics (CIRS-G)[53,54]
Functional capacity	
Basic activities of daily living (ADL)	Katz index, Barthel index, Functional independence measure (FIM) (see Chapter 4)
Instrumental activities of daily living (IADL)	Lawton's IADL[55]
Balance and mobility	Get-up and go,[56] Timed Up & Go,[57] Berg Balance Scale,[58] Functional Reach Test[59] (see Chapter 20)
Frailty	"FRAIL" Questionnaire Screening Tool,[60] Canadian Study of Health and Aging Clinical Frailty Scale[61,62]
Sarcopenia	SARC-F screen for sarcopenia[63]
Mental function	
Cognition	Clock drawing test,[64-66] Executive clock drawing task (CLOX),[67-69] Mini-Cog,[70] Saint Louis University Mental Status (SLUMS) examination,[71] Rapid Cognitive Screen (RCS)[72] Mini-Mental State Examination (MMSE),[73] Chinese version of the Mini-Mental State Examination (CMMSE)[74,75] Abbreviated Mental Test (AMT)[76,77] Addenbrooke's Cognitive Examination Revised (ACE-R)[78] Montreal Cognitive Assessment (MoCA)[79-83] Ascertain Dementia 8 (AD8),[84,85] Chinese AD8 (cAD8)[86] Informant Questionnaire on Cognitive Decline in the Elderly (IQCODE)[87]
Confusion	Confusion assessment method (CAM)[88] (see Chapter 24)
Mood / anxiety / fears	Geriatric Depression Scale (GDS)[89] (see Chapter 25)
Socioeconomic resources	Social network and support checklist (see Chapter 16) Eligibility for care resources / disability allowances
Environmental resources	Checklist for positive environment (age-friendly,[90] supportive [therapeutic, prosthetic], accessible, adaptable, comfortable, safe[91]), negative environment (iatrogenic, barriers, hazards), transport facilities, accessibility to local resources

The Royal College of Physicians and the British Geriatrics Society have recommended standardised assessment scales for use in geriatric practice to facilitate 75-plus screening, clinical and research communication, and case-mix comparison: the Barthel ADL index, the Abbreviated Mental Test (AMT) score, the Geriatric Depression Scale, the Philadelphia Geriatric Center Morale Scale, and a social support checklist.[92]

Over the years, CGA has evolved from a selection of single-domain, individually validated measures (the 'first generation' of assessment instruments) to the 'second generation' omni-comprehensive assessment instruments developed for a specific healthcare environment (e.g. Minimum Data Set–Nursing Home) in the 1990s to the 'third generation' of standardised communication system in transitional care in the 2000s based on a common set of standardised assessment items in addition to a limited number of setting-specific items, e.g. interRAI HC (home care), interRAI LTCF (long-term care facility), and interRAI AC (acute care).[93,94] The third-generation instruments share core elements of information and are intended for elderly patients in all healthcare environments and to improve information transfer in transitional care. These instruments use the same philosophy of assessment to facilitate clinical communication both between different caregiving professionals and across acute, post-acute, and long-term care settings. They aim to enhance interdisciplinary care planning and continuity of care with effective use of information technology.[93]

2.6 Visual Assessment

Assessment of vision is especially important in old age because impairment is common, strongly associated with falls and fractures, adversely affects communication and medication handling; and yet is often remediable through surgery or prescription of spectacles.

Assessment of vision begins with taking a history: questions about function (can you feed yourself? dress? handle / inject medication? read? watch television? cook? sew? shopping? drive?); use of aids (spectacles and why? magnifying glass?); accident, falls, and near-falls (bump to one side?); and risk factors (diabetes, stroke, hypertension, steroid, smoking, alcohol). Examination includes response to hand shaking, test of newspaper reading, inspecting spectacles, visual acuity and contrast sensitivity testing, fundoscopy, visual field examination, and tests of perceptual neglect.

Visual acuity is the ability to see detail at a distance. This is recorded as a Snellen fraction when the person being assessed reads down the Snellen chart of a series of letters or letters and numbers that reduce in size from the top to the bottom. The Snellen fraction records the ability to identify a letter of a certain size at a specified distance. The first number or numerator of the Snellen fraction is the testing distance (standard is 20 feet or 6 m). During the visual acuity test, one eye is covered and the vision of each eye is recorded separately, as well as both eyes together. When the person is unable to correctly identify more than half the letters on a line, the previous line will be recorded as the visual acuity. The second number or denominator of the Snellen fraction represents the distance that the average eye can see the letters on a certain line of the eye chart. A Snellen fraction of 20/40 or its metric equivalent 6/12 means that the eye being tested can see at 20 feet (6 m) the smallest letter that can be seen by the average eye at 40 feet (12 m). Visual acuity worse than 20/400 or 6/120 is recorded as count fingers (at a certain number of feet), hand motion (at a certain number of feet), light perception, or no light perception.

Impaired vision is considered present when the level of vision is below that which the individual requires for his or her everyday tasks. A common cut-off point is binocular visual acuity of 6/12 or 6/18 as used in the Medical Research Council study. Legal blindness is taken as 6/60. The common causes of impaired vision are refractive errors, cataract, glaucoma, macular degeneration, and stroke.

Visual function assessment instruments have been developed to measure a patient's vision capability that may not be reflected by testing visual acuity alone.[32] These consist of questions to assess visual perception (activity limitation, near vision, intermediate vision, and distance vision), sensory adaptation (light / dark adaptation, visual search, colour discrimination, and glare disability), peripheral vision, depth perception, social functioning, role limitations, dependency, and mental health. Chinese versions of such visual function assessment instruments that deal with daily vision-dependent activities and associated everyday problems relevant to Chinese culture have been developed and used.[33-36]

Edges in the environment, such as steps and pavement, are naturally occurring visual stimuli of functional significance. An impaired ability to perceive edges will disadvantage elderly persons, especially those with slow reaction times, muscle weakness, or reduced peripheral sensation, and increase their fall risk.[95] Edge contrast sensitivity can be assessed by the Melbourne Edge Test that contains 20 circular test patches of 25 mm diameter, with a series of edges of reducing contrast and variable orientation.[37]

Visual field is assessed by confrontation and perimetry. Visual field loss is present in 1 in every 20 community-dwelling elderly people, increased in incidence 5-fold between 55-80 years of age, and is associated with impaired daily functioning and fall risk.[96,97] Glaucoma, stroke, cataract, and age-related macula degeneration are common causes of visual field loss in old age.[52,53,98]

Perceptual neglect or hemi-neglect is a defect in the detection, orientation, or response to stimuli (visual, auditory, or tactile) from spatial regions on the side contralateral to the side of cerebral damage, and the deficit cannot be attributable to malfunction in more basic sensory or motor systems. Perceptual neglect has been found in 49% of non-dominant hemisphere strokes and 25% of dominant hemisphere strokes in the early stages.[32] It can readily be diagnosed from the characteristic sitting posture of slumping to one side diagonally, leaving behind an exposed triangle ('inverted triangle sign') on the back of the chair, with the trunk and head rotated away from the side of weakness, and conjugate eye deviation away from the side of weakness (Plate 2.1). Visual neglect can be assessed by the Albert's line cancellation test,[38,39] in which the patient is asked to cross out lines ruled on a sheet of paper; the central line being crossed by the examiner as a demonstration (see Learning Manual). The Albert's test score is the percentage of lines left uncrossed and is an important prognostic factor in determining both mortality and functional recovery from stroke.[32] Patients with hemi-neglect are prone to fall and injuries on the side of neglect and may ignore half a plateful of food (Plate 2.1). Caregivers need to be taught to recognise and appreciate the consequences and distress for patients with this perceptual deficit.

2.7 Hearing Assessment

Assessment of hearing in old age is important because impairment is common and disabling; it adversely affects communication; impairs physical, cognitive, and social function; is associated with mood disturbances and behaviour disorders; and

yet often can be improved by amplification, use of appropriate hearing aids, and aural rehabilitation (see Chapter 28).

Assessment of hearing starts with taking a history: questions about function (hear door / telephone bell? hear telephone conversation? visit friends? accused of turning radio / television up too loud? accused of being stupid?); use of aids (hearing aids / amplifiers?); and risk factors (chronic noise exposure, diabetes, hypertension, ototoxic drugs, alcohol). The Screening Version of Hearing Handicap Inventory for the Elderly (HHIE-S)[40,41] is a validated self-administered 10-item questionnaire designed to detect emotional and social problems associated with impaired hearing (Table 2.3). A HHIE-S score of >8 has a sensitivity of 72%, specificity of 77%, positive predictive value of 58%, and negative predictive value of 86% for detecting impaired hearing in elderly persons. With a HHIE-S score of >24, the corresponding values become 41%, 92%, 67%, and 78%.[41] The HHIE-S has been translated and adapted for other languages, including Chinese[42] (Table 2.3).

Examination includes screening hearing tests, otoscopy to identify impacted ear wax, eardrum perforation or other abnormalities, and audiometry. Tuning fork tests are used to distinguish conductive from sensorineural hearing loss. Vision should also be assessed since hearing and vision impairment often coexist in elderly persons. Loss of visual clues in a hearing-impaired elderly person will further impair speech, and dual sensory impairment is associated with cognitive and functional decline and increased mortality.[99,100]

A simple hearing test is the whispered voice test.[43] A tester stands behind (to prevent lip reading) and to the side of the seated patient, at arm's length (0.6 m) from the patient's non-test ear that is masked by gently occluding and rubbing the external auditory canal, and whispers sets of three random numbers (e.g. 6, 1, 9). The patient is asked to repeat the sequence. If the patient cannot repeat back over 50%

Table 2.3 Screening Version of the Hearing Handicap Inventory for the Elderly (HHIE-S)[40,41] and its Chinese translation[42]

Question	Score*
1. [E] Does a hearing problem cause you to feel embarrassed when you meet new people? 聽力問題使您在遇見陌生人時感到窘迫嗎？	
2. [E] Does a hearing problem cause you to feel frustrated when talking to members of your family? 聽力問題使您在與家人交談時感到沮喪嗎？	
3. [S] Do you have difficulty hearing when someone speaks in a whisper? 有人跟您細聲說話時是否會感到費勁？	
4. [E] Do you feel handicapped by a hearing problem? 聽力問題會使您感到障礙或不方便嗎？	
5. [S] Does a hearing problem cause you difficulty when visiting friends, relatives or neighbours? 聽力問題會使您在拜訪親朋好友時遇到困難嗎？	
6. [S] Does a hearing problem cause you to attend religious services less often than you would like? 聽力問題會使您參加活動的次數比以前少嗎？	
7. [E] Does a hearing problem cause you to have arguments with your family? 聽力問題會導致您與家人爭吵嗎?	
8. [S] Does a hearing problem cause you difficulty when listening to television or radio? 聽力問題會使您在看電視或聽廣播時感到困難嗎？	
9. [E] Do you feel that any difficulty with your hearing limits or hampers your personal or social life? 您是否感到聽力問題影響您個人或社會生活？	
10. [S] Does a hearing problem cause you difficulty when in a restaurant with relatives or friends? 聽力問題使您在餐館與親友交談時遇到困難嗎？	
Total score:	

Abbreviations: [E] = emotional handicap question（情緒問題）; [S] = social handicap question（情景問題）
* Scores (range, 0-40): 0 = "no（不是）" response; 2 = "sometimes（有時）"; 4 = "yes（是）"

of the test items over a minimum of two sets, s/he is assumed to have hearing impairment that requires further audiometric assessment.

An audioscope is an instrument that serves as both an otoscope and simplified audiometer. It delivers pure tone frequencies at 20, 25, and 40 decibels at 500, 1000, 2000, and 4000 Hz. Critical frequencies for speech reception are 500, 1000, 2000, 3000 Hz. In the American Speech-Language-Hearing Association guidelines, 25 dB is the standard screening level used for adults. To use the audioscope, the tester selects the largest ear speculum needed to achieve a seal within the external auditory canal, obtains a clear view of the tympanic membrane, and removes any impacted ear wax before testing. The tonal sequence is then initiated with the patient indicating by raising a finger that s/he has heard the tone. The tester records whether the tone is heard at each frequency for each ear. When tested in the physicians' offices and a hearing centre, the sensitivity of the audioscope was 94% in both locations, while its specificity was 90% in the hearing centre and 72% in the physicians' offices.[41] A better test accuracy was obtained when the HHIE-S test was combined with an audioscope test.[41]

2.8 Cognitive Assessment

Cognitive impairment occurs along a continuum from ageing-related cognitive decline to mild cognitive impairment (MCI) with intact daily function to dementia that affects daily function. Cognitive impairment is a geriatric giant with significant impact on the patient, their family and friends, and clinicians. Early detection of impaired cognition allows for diagnosis, appropriate treatment, and support. Cognitive assessment is commonly used to screen for cognitive impairment; obtain differential diagnoses of its cause; rate its severity; monitor change; and make decisions about competency, management, and placement.

With a view towards better care from geriatricians for patients with cognitive impairment, the British Geriatrics Society produced a consensus document in 2005 that recommended use of Mini-Mental State Examination (MMSE)[73] and an executive clock drawing task (CLOX1)[67] as the two initial screening tests in a cognitive screening algorithm.[101] Patients with abnormalities in either one of these tests were further evaluated by two additional cognitive tests: the Confusion Assessment Method[88] and the Informant Questionnaire on Cognitive Decline in the Elderly (IQCODE)[87] to screen for delirium and dementia, respectively.

In recognition of the importance of the maintenance of cognitive or brain health to both the individual and society, the International Association of Gerontology and Geriatrics and its Global Aging Research Network convened an expert consensus panel in 2015 and published a consensus paper that recommended a combination of validated, brief (3-7 mins) patient-based and informant-based screens as the most appropriate approach to the identification of early cognitive impairment.[102]

2.8.1 Clock Drawing Test

The clock drawing test (CDT) is a simple and quick test designed originally as a measure of visuospatial ability and attention in hemi-neglect patients, and to screen for constructional apraxia.[103] The CDT is now also recognised as a test for executive and other cognitive functions.[64] The test requires the patient to draw a clock face on a piece of paper with or without the arms set at a specified time. The CDT is commonly used together with the MMSE. These two cognitive tests are complementary to each other, with the CDT more suited for screening executive and visuo-constructional functions, and the MMSE more suited for orientation, memory, and language functions.[64,104] There are many versions of the CDT that differ in instruction, scoring, and ease of use. Despite the different scoring protocols, study has shown that they all correlate well with the severity of global cognitive impairment, although particular scoring methods may be better suited to assess vascular dementia than Alzheimer's disease and vice versa.[74] A comprehensive review of the multiple clock drawing scoring systems revealed that no CDT was consistently superior in terms of predictive validity for dementia screening, and concluded that a qualitative assessment of 'normal' versus 'abnormal' by 'eyeballing' the clocks may be sufficient for use of CDT as a dementia screening instrument in a primary / general medicine / community setting.[105] A CDT adapted for use among elderly Chinese in Hong Kong has been designed (fill inside a pre-drawn circle of 2.5" diameter the numbers of a clock face with arms indicating the 3 o'clock position) and proved to be a valid measure to screen for dementia, even for illiterate individuals traditionally thought to be non-compliant with tests that required writing or drawing.[65,66]

The CLOX designed by Royall et al[67] is used to elicit executive impairment and discriminate it from non-executive constructional failure (Table 2.4). The CLOX is divided into an unprompted drawing task (with specified time of 1:45) that is sensitive to executive control (CLOX1) and a copied version that is not (CLOX2). Points are awarded based on the answers to a set of 15 questions (e.g. does the figure resemble a clock?). Maximum scores for both the drawing task (CLOX 1) and the copying task (CLOX 2) are 15 points each. A lower score indicates

Table 2.4 The executive clock drawing task (CLOX)[67] and its Chinese translation[68]

Executive clock drawing task (CLOX1 and CLOX2)

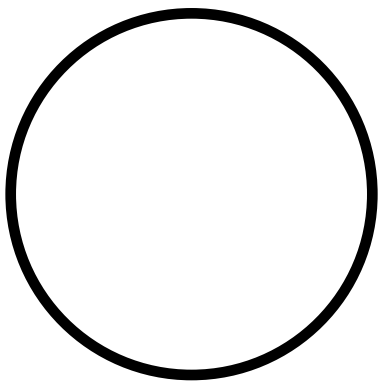
STEP 1 (CLOX1 unprompted executive clock drawing task):
Turn this form over on a light-coloured surface so that the circle below is visible. Have the patient draw a clock on the back. Instruct him / her to "請你畫一個鐘，顯示1:45。將手臂和數目字放在面上邊，以至連一個細路都識睇。" "draw me a clock that says 1:45. Set the hands and numbers on the face so that a child could read them." Repeat the instructions until they are clearly understood. Once the subject begins to draw, no further assistance is allowed. Score this clock in the CLOX1 column.

STEP 2 (CLOX2 non-executive clock copying task):
Return to this side and let the subject observe you draw a clock in the circle below. Place 12, 6, 3, and 9 first. Set the hands again to "1:45". Make the hands into arrows. Invite the subject to copy your clock in the lower right corner. Score this clock (CLOX 2).

	Organisational element	**CLOX1**	**CLOX2**
Q1	Does figure resemble a clock?	Q1=1 point	Q1=1 point
Q2	Outer circle present?	Q2=1 point	Q2=1 point
Q3	Diameter >1 inch?	Q3=1 point	Q3=1 point
Q4	All numbers inside the circle?	Q4=1 point	Q4=1 point
Q5	12, 6, 3 and 9 placed first?	Q5=1 point	Q5=1 point
Q6	Spacing intact? (symmetry on either side of the 12-6 axis?) If yes, skip next.	Q6=2 point	Q6=2 point
Q7	If spacing errors are present, are there signs of correction or erasure?	Q7=1 point	Q7=1 point
Q8	Only Arabic numerals?	Q8=1 point	Q8=1 point
Q9	Only numbers 1-12 among the Arabic numerals?	Q9=1 point	Q9=1 point
Q10	Sequence 1-12 intact? No omissions or intrusions	Q10=1 point	Q10=1 point
Q11	Only two hands present?	Q11=1 point	Q11=1 point
Q12	All hands represented as arrows?	Q12=1 point	Q12=1 point
Q13	Hour hand between 1 and 2 o'clock?	Q13=1 point	Q13=1 point
Q14	Minute hand longer than hour?	Q14=1 point	Q14=1 point
Q15	None of the following: (1) hand pointing to 4 or 5 o'clock? (2) '1:45' present? (3) intrusions from 'hand' or 'face' present? (4) any letters, words or pictures? (5) any intrusion from circle below?	Q15=1 point	Q15=1 point
	Total	/ 15	/ 15

For Caucasians, CLOX1 score of <10 indicates executive dysfunction ± constructional dyspraxia, CLOX2 score of <12 indicates constructional / visuospatial dyspraxia (Royall 1998[67])

For Chinese in Hong Kong, CLOX1 score of <7 (1 standard deviation below mean) or CLOX2 score of <11 (1 standard deviation below mean) indicates cognitive dysfunction (Wong 2004[68])



impairment, with a cut-off score of 10/15 for the drawing task and 12/15 for the copying task. The CLOX has been translated into Chinese (Table 2.4) and was found to correlate strongly with MMSE when tested among elderly Chinese in Hong Kong, although performance depended on education with a lower cut-off score of 7/15 and 11/15 for CLOX1 and CLOX2, respectively among Chinese subjects.[68] Nonetheless when the Chinese version of CLOX was tested in Chinese elderly patients with subcortical ischaemic vascular disease against more formal executive measures than that used by Royall et al,[67] it performed poorly as a screening test for executive dysfunction.[68] A study of the same Chinese version of CLOX among Singaporean Chinese, however, supported CLOX as a valid cognitive screen with adequate psychometric properties, and its use as an adjunct in differentiating Alzheimer's disease from dementia with a vascular element, in which deficits in executive control function are more prominent.[69]

The CDT has been incorporated as a component of other cognitive screening tests, e.g. Mini-Cog,[70] Saint Louis University Mental Status examination (http://aging.slu.edu/index.php?page=multi-language-slums),[71] and Rapid Cognitive Screen.[72] The Mini-Cog was developed as a brief 5-point cognitive test to discriminate dementia from non-dementia among multilingual elderly persons with diverse educational status by combining a delayed three-item recall (0-3 points) with a clock drawing test (0 or 2 points) as recall distractor.[70] A score of 0-2 indicated a positive screen for dementia.

2.8.2 MMSE

The MMSE was first described by Folstein et al in 1975 as a "practical method for grading the cognitive state".[73] It was called "mini" because it "concentrates only on the cognitive aspects of mental functions, and excludes questions concerning mood, abnormal mental experiences and the form of thinking." The MMSE consists of 19 tests of 11 domains covering orientation to time and place (10 points), registration of three words (3 points), attention or calculation tested by serial sevens or spelling (5 points), recall of three words (3 points), verbal and written language including naming, repetition, comprehension (8 points), and visual construction (1 point). Folstein et al[73] suggested a cut-off score of ≤23 (out of a maximum score of 30) for the presence of dementia in persons with at least 8 years of education. Numerous other cut-offs have been calculated from receiver operating characteristic curve analysis of specific populations together with adjustments for age and education.[106] The norms declined with advancing age, especially for less educated women. Given any age and gender, the norms were higher for individuals with a higher education level.[106]

A meta-analysis of 34 dementia studies and five MCI studies was conducted to evaluate the accuracy and clinical utility of MMSE as a cognitive test in high and low prevalence settings (Table 2.5).[107] The study concluded that MMSE has some value both in specialist and non-specialist settings but in two different capacities. In specialist settings such as memory clinics it was reasonably effective in identifying dementia but could not be relied upon alone if a result was negative (patient scored above threshold) and should not be used alone for diagnosing MCI. Conversely, in non-specialist settings such as primary care, the only value of the MMSE was in excluding dementia in someone worried about their memory, while a positive result (scoring under threshold) could be explained mostly by non-dementia conditions.[107] MMSE cannot serve as a substitute for systematic evaluation that includes history taking, examination, and laboratory tests.

The MMSE is affected by educational background, language and communication disorders, and sensory loss. Limitations of the MMSE include its non-linearity, a floor effect in advanced dementia (a score of 0 does not mean an absolute absence of cognition), a ceiling effect in very mild disease (a score of 30 does not always mean normal cognitive function), and a lack of sensitivity for frontal / executive or visuospatial functions. The pentagon task of the MMSE does not assess executive function as it simply requires the patient to copy the image.[108] Thus MMSE may have a limited ability to detect early stage of dementia with executive dysfunction, e.g. vascular dementia.[108] The MMSE has been standardised with clear guidance for its administration, scoring and time allowed for each of the components, with resultant reduction in inter-rater variability and administration time.[109]

A Cantonese version of MMSE has been validated

Table 2.5 Accuracy of the Mini-Mental State Examination as a cognitive test for dementia in various prevalence settings[107]

Setting	Sensitivity (%)	Specificity (%)	Positive predictive value (%)	Negative predictive value (%)
Memory clinic	79.8	81.3	86.3	73.0
Mixed specialist hospital	71.1	95.6	94.2	76.4
Non-clinical community	85.1	85.5	34.5	98.5
Primary care	78.4	87.8	53.6	95.7

as an instrument to detect cognitive impairment in a local Chinese population in Hong Kong; a cut-off score of 19-20 is recommended as an indication for further evaluation of cognitive impairment[74]; further studies suggested that the optimal cut-off point was ≤18 for illiterate subjects, ≤20 for those with 1-2 years of schooling, and ≤22 for those with >2 years of schooling.[75]

The second edition of the MMSE (MMSE-2) was developed in 2012 under the copyright of the Psychological Assessment Resources (http://www.minimental.com/), with a 30-point standard version equivalent to the original MMSE; a 16-point brief version for rapid cognitive screening; and a 90-point expanded version enhanced with two new tasks (story memory and processing speed) to increase sensitivity for milder forms of cognitive impairment, including subcortical dementia. A simplified Chinese translation of MMSE-2 is available.

2.8.3 AMT

Based on a study in 21 geriatric departments in the UK sponsored by the Royal College of Physicians of London, Hodkinson[76] shortened the Blessed, Tomlinson, and Roth's mental test[110] from 26 questions to 10. He noted that the AMT gave comparable results to the full mental test and could replace it where the test was used to recognise cognitive impairment due to delirium or dementia.[76] The shorter test also achieved far more consistent cooperation by patients being tested. Validation study of the AMT in the UK recommended a cut-off score of 8 (<8 suggesting abnormal cognitive function) with a sensitivity of 91% and specificity of 75%.[111] A modified local Chinese version of AMT (Table 2.6), which was validated in a local Chinese population, is commonly used in Hong Kong.[77] The cut-off of 6 yielded a sensitivity of 96% and specificity of 94% in differentiating normal versus abnormal cognitive function (e.g. delirium, dementia) in local elderly patients.[77] The lower AMT cut-off of 6 for the local Chinese population has been attributed to a lower education level.

2.8.4 Montreal Cognitive Assessment

The Montreal Cognitive Assessment (MoCA) is a cognitive test of 10 items covering the domains of short-term memory, visuospatial skills, executive function, phonemic verbal fluency, abstraction, attention, concentration and working memory, language function, and time orientation (http://www.mocatest.org). Its final English version is a 1-page 30-point screening test, with a cut-off score of <26 considered abnormal and 1 point added for persons educated ≤12 years.[79] Compared with the MMSE, the MoCA is significantly better for detecting MCI, with a sensitivity and specificity of 90% and 87%, respectively.[79] Validated Chinese versions with adjusted cut-offs for use in Hong Kong,[80,81] Taiwan,[82] and China[83] are available. In the Cantonese Chinese version[81] of MoCA, with 2-point added for illiterate and 1-point for persons educated 1-6 years, a cut-off of <22/23 has a sensitivity of 78% and specificity of 73% in detecting amnestic MCI, while a cut-off of 19/20 has a sensitivity of 94% and specificity of 92% in detecting Alzheimer's dementia. In the original 2009 Hong Kong MoCA (HK-MoCA),[80] with 1-point added for persons educated ≤6 years, a cut-off of 21/22 has a sensitivity of 73% and specificity of 75% in differentiating patients with cerebral small vessel disease (the most common cause of vascular cognitive impairment) from controls. The HK-MoCA was subsequently updated to the 2015 version with age and education corrected normative data of total score of MoCA (http://www.mocatest.org/wp-content/uploads/2015/03/HK-MoCA_20151030.pdf). Cognitive impairment is determined when the score ≤ age and education corrected percentile cut-offs at 7th and 2nd percentiles for MCI and dementia, respectively. A brief version of HK-MOCA, the Hong Kong version of Montreal Cognitive Assessment — 5-minute protocol (HK-MoCA 5-min), is available for cognitive screening via telephone.

2.8.5 Informant-based Cognitive Assessment

In contrast to patient-based mental status tests that

Table 2.6 Abbreviated Mental Test (Hong Kong version)[77]

Question	Score
Age (± 5 years)	0/1
Time (to the nearest hour, or am, pm, night)	0/1
(Ask to memorise address for recall at the end of the test: 42 Shanghai Street)	
Year (± 1 year)	0/1
Place name	0/1
Recognition of two persons (doctor, nurse)	0/1
Date of birth (date and month)	0/1
Date of Mid-Autumn festival	0/1
Name of present Chief Executive of the Hong Kong Special Administrative Region or Chinese leader	0/1
Count from 20 to 1 backwards	0/1
Recall address: 42 Shanghai Street	0/1

directly assess the patient's performance, informant-based cognitive assessment indirectly assesses the patient by gathering information about everyday behaviour and activities from informants (family or friends). The strengths of this approach are: change in functioning and time course of decline can be assessed; less influenced by education and premorbid intelligence; less artificial and reflects capacity in a natural setting; applicable even when the patient is non-communicable or uncooperative.[112] A weakness is that the indirect assessment is more influenced by reporting factors such as bias in interpretation of the patient's behaviour; informants' motivation to provide information; and the quality of the relationship between the informant and the patient.[112]

The Ascertain Dementia 8 (AD8) is a brief (3-min administration time) informant-based cognitive screening tool with eight questions that assess change in memory, temporal orientation, judgement, and function (Table 2.7).[84,85] An AD8 score of ≥2 in a memory clinic setting (dementia prevalence 89%) has a sensitivity of 92%, specificity of 46%, positive predictive value of 93%, and negative predictive value of 43% for detecting dementia even at a very mild stage. The AD8 has been translated into Chinese in Taiwan and validated.[86] At a cut-off of 2, the Chinese AD8 has a sensitivity of 97.6%, specificity of 78.1%, positive likelihood ratio of 4.5, and negative likelihood ratio of 0.03 in detecting dementia.[86]

The IQCODE is a self-administered tool that comprises 26 items completed by an informant familiar with the patient. The informant rates the patient's cognitive function as better or worse than 10 years ago based on a 5-point scale.[87] The short form of IQCODE contains 16 items and takes 10-15 minutes to complete. IQCODE is scored by adding up the score for each question and then dividing by the number of questions, 26 for the long IQCODE and 16 for the short IQCODE. The result is a score that ranges from 1-5; a score of 3 means 'no change', 4 'a bit worse', and 5 'much worse'. Balancing sensitivity and specificity for screening for dementia, the cut-off for the long IQCODE is 3.27/3.30, while the cut-off for the short IQCODE is 3.31/3.38. The IQCODE has been translated into other languages, including Chinese (http://crahw.anu.edu.au/risk-assessment-tools/informant-questionnaire-cognitive-decline-elderly.)

Table 2.7 Ascertain Dementia 8 (AD8) questions asked of informants[84,85] and its Chinese version[86]*

AD8	Chinese AD8	Cognitive abilities asked
Instructions to informant: "Remember, 'Yes, a change' indicates that you think there has been a change in the last few years caused by cognitive (thinking and memory) problems."	填表説明：若您以前無下列問題，但在過去幾年中有以下的『改變』，請勾選；若無或不確定，請繼續下一題。一共有八題。	
1. Problems with judgement (e.g. falls for scams, bad financial decisions, buys gifts inappropriate for recipient)	判斷力上的困難：例如落入圈套或騙局、財路上不好的決定、買了對受禮者不合宜的禮物。	Judgement
2. Reduced interest in hobbies / activities	對活動和嗜好的興趣降低。	Function
3. Repeats questions, stories, or statements	重複相同問題、故事和陳述。	Memory
4. Trouble learning how to use a tool, appliance, or gadget (e.g. VCR, computer, microwave, remote control)	在學習如何使用工具、設備和小器具上有困難。例如：電視、音響、冷氣機、洗衣機、熱水爐（器）、微波爐、遙控器。	Function
5. Forgets correct month or year	忘記正確的月份和年份。	Temporal orientation
6. Difficulty handling complicated financial affairs (e.g. balancing checkbook, income taxes, paying bills)	處理複雜的財物上有困難。例如：個人或家庭的收支平衡、所得税、繳費單。	Judgment
7. Difficulty remembering appointments	記住約會的時間有困難。	Memory
8. Consistent problems with thinking and / or memory	有持續的思考和記憶方面的問題。	Memory

* The above AD8 questions are asked of an informant. Items endorsed as "Yes, a change" are summed to yield the total AD8 score

Table 2.8 Clinical Frailty Scale[62]

1. Very fit — robust, active, energetic, well-motivated and fit; these people commonly exercise regularly and are in the fittest group for their age. Self-rate health as 'excellent'
2. Well — without active disease, but less fit than people in category 1
3. Well, with treated comorbid disease — disease symptoms are well controlled compared with those in category 4
4. Apparently vulnerable — although not frankly dependent, these people commonly complain of being 'slowed up' or have disease symptoms or self-rate health as "fair", at best. If cognitively impaired, do not meet dementia criteria
5. Mildly frail — with limited dependence on others for instrumental activities of daily living
6. Moderately frail — help is needed with both instrumental and non-instrumental activities of daily living
7. Severely frail — completely dependent on others for the activities of daily living
8. Terminally ill

Table 2.9 The "FRAIL" Questionnaire Screening Tool[60]

≥3 = Frailty; 1 or 2 = pre-frail
Fatigue: Are you fatigued?
Resistance: Cannot walk up one flight of stairs?
Aerobic: Cannot walk one block?
Illnesses: Do you have more than five illnesses?
Loss of weight: Have you lost more than 5% of your weight in the past 6 months?

Table 2.10 SARC-F Screen for Sarcopenia[63]

Component	Question	Scoring*
Strength	How much difficulty do you have in lifting and carrying 10 pounds?	None = 0 Some = 1 A lot or unable = 2
Assistance in walking	How much difficulty do you have walking across a room?	None = 0 Some = 1 A lot, use aids, or unable = 2
Rise from a chair	How much difficulty do you have transferring from a chair or bed?	None = 0 Some = 1 A lot or unable without help = 2
Climb stairs	How much difficulty do you have climbing a flight of 10 steps?	None = 0 Some = 1 A lot or unable = 2
Falls	How many times have you fallen in the past year?	None = 0 1-3 Falls = 1 ≥4 Falls = 2

* Total score of ≥4 is predictive of sarcopenia and poor outcomes

Table 2.11 Simplified Nutritional Assessment Questionnaire[48]*

My appetite is	Food tastes	When I eat	Normally I eat
1. very poor	1. very bad	1. I feel full after eating only a few mouthfuls	1. less than one meal a day
2. poor	2. bad	2. I feel full after eating about a third of a meal	2. one meal a day
3. average	3. average	3. I feel full after eating over half a meal	3. two meals a day
4. good	4. good	4. I feel full after eating most of the meal	4. three meals a day
5. very good	5. very good	5. I hardly ever feel full	5. more than three meals a day

* A score of <14 may identify persons with anorexia and at risk of significant weight loss

2.8.6 Choice of Cognitive Tests

The appropriate choice of a cognitive test depends both on the time available and the purpose of assessment. An ideal cognitive screening tool should be brief and have no copyrighted costs.[102,113] Surveys in the West have shown the MMSE to be the most commonly used. Nonetheless the MMSE takes on average 8 (range, 4-21) minutes to perform and the acquisition of copyright restriction of MMSE by the Psychological Assessment Resources in 2001 has increased the need to identify briefer and effective cognitive tests for use in clinical practice.[113,114] A systematic review and meta-analysis of dementia screening tests up to 2014 evaluated the diagnostic performance of all cognitive tests for the detection of dementia,[114] and concluded that the Mini-Cog test[70] and the Addenbrooke's Cognitive Examination Revised[78] are the best alternative screening tests for dementia, and the MoCA[79] is the best alternative for MCI.[114]

2.9 Geriatric Assessment for Geriatric Syndromes

The Royal College of Physicians advocated early CGA for frail elderly people.[115] Commonly used validated assessment tools for screening frailty in elderly persons include the Canadian Study of Health and Aging (CSHA) Clinical Frailty Scale (Table 2.8)[61,62] and the "FRAIL" Questionnaire Screening Tool (Table 2.9).[60] Clinical Frailty Scale is a measure of frailty based on clinical judgement when interpreting the results of history taking and clinical examination, developed in the CSHA. Each one-category increment of the scale significantly increased the medium-term risks of death (21.2% within 70 months) and entry into an institution (23.9%). Sarcopenia and falls can be identified through the SARC-F questionnaire (Table 2.10).[63] The assessment tools FRAIL (for frailty) and SARC-F (for sarcopenia and falls) have been combined with other screening assessment tools Simplified Nutrition Assessment Questionnaire

for anorexia and undernutrition (Table 2.11)[48] and Rapid Cognitive Screen for cognitive dysfunction[72] into a Rapid Geriatric Assessment (RGA)[116,117] toolkit to quickly screen for the common geriatric syndromes of frailty, sarcopenia and falls, anorexia and undernutrition, and intellectual impairment. The RGA can be completed quickly within 4 minutes, facilitating early recognition of geriatric syndromes, diagnosis of underlying causes, and implementation of intervention and a care plan to reduce disability in elderly persons.[116,117]

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CHAPTER 3

Acute Geriatric Care

Chun-por WONG

OBJECTIVE

To identify the common acute conditions of which an older patient presents to a primary care and hospital setting, and the principles of a multidisciplinary approach to management of conditions.

KEY POINTS

- Acute illness is more common in elderly patients and associated with an increased risk (4.2-fold) of hospital admission.
- Infection remains the most common cause of acute illness.
- Different models of hospital acute care exist with various benefits and strengths. Collaboration of geriatricians with primary care physicians and other hospital specialists is important.
- There are inherent differences between an elderly patient with an acute condition and a younger patient with the same condition.
- Comprehensive geriatric assessment is the key in geriatric medicine.
- Treating an elderly patient with acute illness requires specialist knowledge and skills.

3.1 Acute Illnesses in Elderly Patients

An older patient is more at risk of an acute illness and consequent admission to an acute service of a hospital.[1] In Hong Kong, the risk of hospitalisation among elderly persons is 4.2 times higher than that of the total population, a rate that increases rapidly after the age of 60 (Figure 3.1[2]). Although those over the age of 65 constitute only 13% of the Hong Kong population, they account for more than 60% of hospital bed days and healthcare budget for public services.

Treatment of frail elderly patients poses many challenges: atypical presentation of diseases and delirium, in particular, are a challenge to correct history taking and physical examination. Nonetheless, if the primary care physician can provide holistic care, many admissions can be avoided, and with it, the risk of acquiring hospital-related complications.

3.1.1 Common Acute Conditions in Elderly Patients

About half of all acute conditions in elderly patients are related to infection.[1] Viral and bacterial infections — especially of the lungs, urinary tract, and biliary tree — are most prevalent. Gastrointestinal conditions such as gastroenteritis, constipation, and diarrhoea are also common. A study in the Netherlands[3] on 639 acutely hospitalised elderly persons aged ≥65 years (mean age, 78 years) showed that the common medical reasons for admission were infectious disease (40.9%), gastrointestinal disease (22.8%), malignancy (6.2%), cardiovascular disease (4.3%), and fluid and electrolyte imbalance (10.5%).

3.1.2 Atypical Presentations

Atypical presenting symptoms and signs are the rule rather than the exception in elderly patients. In the case of infectious disease, which is the highest cause of acute presentation in elderly patients, there may be no fever as well as failure to mount a high white cell count response in the blood test. 'Silent' acute abdominal conditions often present with no symptoms other than mild discomfort and constipation. 'Silent' malignancy may present as back pain secondary to metastases from breast or lung cancer, or as a silent mass in the abdomen. 'Silent' myocardial infarction may present with no chest pain.

Hence, meticulous care must be exercised before an accurate diagnosis can be reached. A high index of suspicion of acute ailments must be maintained for any elderly patient with vague symptoms.

3.2 The Acute Geriatric Service

An older patient, who is vulnerable to many diseases after the ageing process, may become acutely ill and

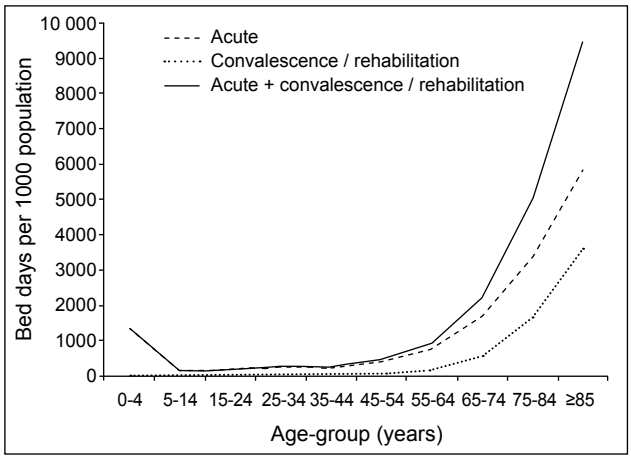

Figure 3.1 Age-specific in-patient utilisation rates[2]

require professional management at both primary and secondary care levels.

3.2.1 Consultation Service, Geriatric Evaluation and Management Unit, and Acute Care for Elders Units

In general, three models of in-hospital geriatric intervention have been described for high-risk elderly patients:

(a) A multidisciplinary geriatric consultation team who carry out a systematic assessment of the patient and recommend a plan of treatment in a typical general acute medical ward.[4]

(b) Treatment of patients in a geriatric evaluation and management unit (GEMU).[5] A GEMU is a ward that specifically admits frail elderly patients for systematic multidisciplinary assessment, review, and therapy.[6] GEMUs have been crucial in developing basic concepts of comprehensive geriatric assessment (CGA).

(c) During the last 10 years, another kind of in-patient ward has been introduced, the Acute Care for Elders units (ACE). These units are designed to prevent general functional decline in hospitalised elderly patients. The ACE model is characterised by a prepared environment, patient-centred care by a primary nurse, and interdisciplinary team rounds and medical care reviews.

3.2.1.1 Consultative Service

Randomised clinical studies of the effect of geriatric consultation teams working in a general acute medical ward to care for elderly patients have shown conflicting results, but generally no or only small additional beneficial effects have been found. In Hong Kong, most acute geriatric services are based on this model.

3.2.1.2 The Designated Geriatric Ward with GEMU

Acutely hospitalised older people require access to a service that can appropriately diagnose and treat acute illness in an environment that minimises the risks of hospitalisation. These include functional decline, falls, delirium, and loss of morale. They also recognise the impact of psychosocial issues on an older person's quality of life.

To provide effective acute care services to older people, an acute geriatric ward should possess at least:

(a) A multidisciplinary geriatric consultancy service, led by a trained geriatrician working closely with nursing and allied health staff, within a process that facilitates early referral. This process should begin in the emergency department.

(b) A ward design and culture in keeping with age-friendly principles, with a focus on maintaining independence in a safe physical environment.

(c) Risk screening, prevention, and management protocols for common problems that affect older people such as falls, delirium, incontinence, pressure areas, malnutrition, and medication errors.

In practice, there is significant variation in the delivery of these basic models of care that depends on the hospital setting, resources, and patient needs. For example, a major teaching hospital may offer both a dedicated geriatric unit and an integrated geriatric consultancy service. Admission policies also vary depending on service model and are based on age, patient needs, or completion of an acute care episode.

In Hong Kong, most of the admission criteria for direct admission to a designated geriatric ward are based on need, not age, although most of those who fulfil these criteria will be aged >65 or even >75 years. The need-based criteria include many features: multiple pathologies; multiple functional disabilities such as immobility, incontinence, and pressure sores; living in institutions such as residential care homes; impaired cognition such as dementia or acute confusional state; and poor social support.

3.2.1.3 Acute Care for Elders

In the US, a randomised controlled trial of an ACE demonstrated improved mobility and activities of daily living function, along with reduced nursing home placement. This model of care has also demonstrated improved processes of care and patient satisfaction. A mortality benefit was demonstrated in one study of frail acutely ill older patients admitted to a designated geriatric unit, and a similar study demonstrated a reduced acute length of stay.[7] In an Australian teaching hospital, an enhanced multidisciplinary approach implemented in general medical wards that cared for acutely ill older patients demonstrated a reduction in hospital mortality and functional decline.[8]

The common features of these inpatient models of care in both acute and subacute settings and that are associated with improved outcome are a multidisciplinary, comprehensive, patient-centred approach to care led by geriatricians in an appropriate setting with the ability to follow up as necessary. Interventions were commonly targeted to patients most likely to benefit, particularly for subacute care.

3.3 Atypical Presentation of Acute Conditions in Elderly Patients and Their Management

There are significant differences in the acute management of a medical condition in an elderly individual compared with a young individual. For example, a 20-year-old man admitted with acute streptococcal lobar pneumonia may present typically with high swinging fever and chills, cough, purulent sputum production, shortness of breath, malaise, and pain in the chest wall. He will require typically less than 5 days of hospital stay with initial therapy of intravenous antibiotics. A short course of breathing and coughing exercises by the physiotherapist is also usually required. The young man will then often be discharged before day 5 and continue with a few days of oral antibiotics, before making a full recovery at around 1 week to 10 days. He will then be fully recovered and resume his previous schooling or work with few ill effects.

On the contrary, an elderly person of 80 years old with a similar acute condition of streptococcal lobar pneumonia may present atypically with excessive drowsiness, easy falling, confusion, low-grade fever, incontinence, and some shortness of breath. Cough may be present with copious sputum although chest pain is rare. He will often require a prolonged hospital stay. After a similar few days of intravenous antibiotics, he will not improve dramatically although fever, cough, malaise, and confused mental state might improve. There may be weakness in his legs, and a longer period of chest physiotherapy combined with limb strengthening exercises will be required. He may then be prone to complications of prolonged bed rest, such as pressure ulcers and urinary tract infection. His mental function may continue to deteriorate with increased confusion. He ultimately requires a longer stay in hospital. His family members may not have the confidence or resources to take care of him at home and may consider residential care home placement for him on discharge. If this occurs, he may become depressed and withdrawn, further hindering his chance of full recovery and mobility. If he is still in work, he may lose his job.

It can be seen that many significant differences exist in the presentation, management, course, and prognosis of the two patients with the same acute condition of streptococcal pneumonia. Typically, the younger patient will have experienced an acute medical condition, with no concomitant or subsequent functional, social, or psychological problems. The acute treatment of the younger patient is thus simple and has involved only management of the medical aspect of disease. Meanwhile the older patient has required multidisciplinary management for medical, functional, psychological, and social elements of the same disease.

In the management of elderly patients with acute medical conditions, the following aspects should be addressed.

3.3.1 Medical Problems

The medical management of acute conditions in an elderly patient requires attention to comorbidities and careful use of medication with dosages appropriate to the kidney and liver function (see Chapter 15). Particular attention should also be paid to fluid and electrolyte balance when drugs or fluids are given intravenously, as the cardiovascular system may have been compromised.

3.3.2 Functional Problems

As mentioned above, associated functional problems are common in an elderly patient with acute illness.[9] Muscle weakness, unsteady gait due to dizziness or vertigo, coordination problems due to muscle weakness or cerebellar dysfunction or even visual impairment, joint control due to arthritis, and tremors due to neurological lesions are all often more pronounced in an elderly patient. Hence, the risk of falls is also higher. In an acute setting that is environmentally friendly to an elderly patient, beds are often designed to be low to the ground to facilitate access for patients with mobility and muscle problems. Physical restraints may be inappropriately applied and increase the chance of injurious falls. A course of physiotherapy and occupational therapy are often required to improve and restore the functional level in an elderly patient with acute illness.

3.3.3 Psychological Problems

An elderly patient with any acute illness is prone to an acute confusional state, or a transient worsening of mental function in a patient with established dementia. Depression is also often associated with acute illness in an elderly patient, as multiple chronic disabling conditions often lead to undue worries, disabilities, and handicaps.

3.3.4 Social Problems

The onset of acute illness in an elderly individual may precipitate a significant change in working capability. Alternatively, relatives may be concerned about the need for additional care after hospital discharge.

Financially, the patient may be unable to afford daily essentials or any nutritional supplements that might be needed. The onset of disability may also obstruct normal social activity.

3.4 CGA in Acute Setting

In view of the multifaceted problems faced by an elderly individual who develops an acute medical condition, CGA is the key to successful management[10-14] (see Chapter 2 for details). Below is a list of the areas a geriatrician may choose to assess:

(a) Chief complaints, current symptoms and history of present illnesses and their functional impact
(b) Current drugs, their indications and effects
(c) Relevant history of past illnesses
(d) Recent and impending significant life changes
(e) An objective measure of overall personal and social functionality
(f) Current and future living environment and its appropriateness to function and prognosis
(g) Family background, support, and availability
(h) Current caregiver network including its deficiencies and potential
(i) An objective measure of cognitive status
(j) An objective assessment of mobility and balance
(k) Rehabilitative status and prognosis
(l) Current emotional health and substance abuse
(m) Smoking and alcohol consumption
(n) Nutritional status and needs
(o) Disease risk factors and screening status
(p) Services required and received

3.5 Special Consideration in Treating Elderly Patients

The treatment of older patients often requires a different skill set. There will be differences due to the patient, treatment, or patient-treatment interaction.

3.5.1 Patient Characteristics

Because of the ageing process and the presence of comorbidities, the potential benefits and the enhanced risk of medical treatment in an older patient can be much altered. For some conditions, such as deep venous thrombosis and atrial fibrillation, anticoagulation treatment can have enhanced treatment effects in elderly patients compared with young patients. The ageing brain also makes an elderly patient more susceptible to side-effects of drugs such as psychotropic agents and anticholinergics.

3.5.2 Treatment Characteristics

Older patients may have problems with drug compliance owing to multiple factors, such as polypharmacy, motivation, memory decline, and social and financial conditions. The most significant difference in drug treatment in elderly patients is their susceptibility to adverse drug reactions and drug-drug interactions (see Chapter 15).

3.6 Summary

Acute illnesses are more common in elderly patients than those of a younger age-group, and they consume a greater proportion of hospital services. Infection remains the most common cause of acute illnesses. Different models of acute hospital care exist with various benefits and strengths. Collaboration of geriatricians with primary care physicians and other hospital specialists is important. There are inherent differences between an elderly patient with an acute condition and a younger patient with the same complaint. CGA is the key in geriatrics. Treating an elderly patient with acute illness requires specialist knowledge and skills.

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CHAPTER 4

Geriatric Rehabilitation

Mandy Pui-man FUNG, Jennifer Ma Wai-wai MYINT

OBJECTIVE

To learn the World Health Organization classification of functioning, disability and health, goal setting and motivation, role of the multi-professional team, the interdisciplinary team approach, as well as the use and interpretation of measures to assess functional status and outcome for activities of daily living (e.g. Barthel Index, Functional Independence Measure) and special geriatric considerations in rehabilitation (e.g. risk of immobilisation and deconditioning, exercise intensity and precautions, hip fractures, interplay between medical conditions, functional limitations, and psychosocial factors). Commonly used aids and adaptations in the local setting will also be discussed.

KEY POINTS

Practical tips

- Assess (objective observations) and ask (personal goals and family concerns).
- Strike a balance between safety and independence.
- Do not decide lightly on an institutional placement.
- Think in terms of what is do-able.
- Agree rehabilitation goals among team members.

Practical advice to the patient

- Exercise and training are useful regardless of age.
- Exercise programmes in elderly people may need to be modified due to cognitive, sensorineural impairment.
- Simple persistent exercise is better than infrequent therapy.
- Use appropriate aids and devices.
- Seek help and support if feeling demoralised, of if you notice deterioration of function.

4.1 Introduction

The challenge of geriatric rehabilitation lies in bringing together the knowledge and insight of geriatric medicine and rehabilitation medicine to benefit elderly persons with significant disability. Assessment of an elderly person with disability should be based on sound rehabilitation concepts and framework, and on adequate understanding of how the medical condition(s) and impairment affect an individual's function and quality of life.

4.2 The World Health Organization Framework for Rehabilitation — International Classification of Functioning, Disability and Health

According to the World Health Organization (WHO), the aim of rehabilitation is to enable people with disabilities to reach and maintain their optimal physical, sensory, intellectual, psychological, and social functional levels. Rehabilitation provides disabled people with the tools they need to attain independence and self-determination. It aims to maximise function and minimise limitation of activity and restricted participation as a result of an underlying impairment or disease.

The WHO's International Classification of Functioning, Disability and Health framework[1] was established as a standard to describe health and disabilities. It defines health and some health-related components of well-being in two parts. Part 1 classifies functioning and disability whereas part 2 expresses environmental and personal contextual factors.

- Body functions and structures refer to physiological functions of body systems and anatomical parts of the body. Impairments are problems in body

function or structure as a significant deviation or loss that may be temporary or permanent, progressive, regressive, static, intermittent, or continuous.
- Activity and participation involve execution of a task or action by an individual and involvement in a life situation. The extent or magnitude of difficulty are measured as capacity and performance, respectively. Capacity is measured in a standardised environment (e.g. Barthel Index or Timed Up and Go test). The domains that can be measured are learning, applying knowledge, communication, self-care, mobility, and domestic life. Limitations in activities are evaluated against the norm of a person without similar impairments. Performance is measured in the person's living environment and involves interpersonal interactions, community, social, and civic life. Examples of performance measures are quality-of-life measurements and stroke-adapted Sickness Impact Profile[2] for stroke sufferers.
- Contextual factors are personal or environmental factors or backgrounds that are believed to interact with the individual health conditions, forming either facilitators or barriers. These factors are described from the perspective of the person being described and include products and technology, local physical surroundings, social support and relationships, attitudes, customs, religions, and the services and systems in place in the community.

Taking into account information available about the impairments in body functions or structures, activity limitations, restrictions in participation and contextual factors, comprehensive assessment, follow-up, service provision, and academic research may be planned for the individual as well as similar groups of patients.

4.3 Principles of Geriatric Rehabilitation

Older people are more likely to have disabilities and are more likely to require assessment for rehabilitative services.[3] Therefore, geriatricians should have a working knowledge of rehabilitation principles in a variety of clinical settings (acute, subacute, outpatients, community, and in institutions). Adopting a multidisciplinary or interdisciplinary team approach is required with close involvement of the patient and formal or informal carers.

Geriatric rehabilitation involves simultaneous management of multiple conditions and understanding the concepts of frailty, variability, and inactivity. Maintenance of good health and residual physical and mental functions need to be complemented with environmental management and carer support.

Frailty is considered highly prevalent in old age and to confer high risk for falls, disability, hospitalisation, institutionalisation, and mortality. It is described as a status of global impairment of physiological reserves involving multiple organ systems.[4] The clinical correlate of frailty manifests as increased vulnerability, impaired capability to withstand intrinsic and environmental stressors, and limited capacity to maintain physiological and psychosocial homeostasis. Depending on criteria and cohort, geriatric frailty is found in 20-30% of elderly population aged >75 years and increases with advancing age. As it is a multisystem failure, no single cause may be found and a multisystem perspective is required in trying to reverse the process. Exercise and nutrition are important aspects of treatment for frailty.

Clinically, frailty can be defined as three or more variables of a phenotype[5] consisting of unintentional weight loss, self-reported exhaustion, low energy expenditure, slow gait speed, and weak grip strength.

Sarcopenia is a key feature of frailty and is defined as the age-related loss of muscle mass, strength, power, quality, and function. Age-related changes in the neurological and endocrine systems, low-grade inflammation, and loss of muscle homeostasis are thought to give rise to sarcopenia.

Variability is another factor that needs to be considered during treatment or service planning. An important concept is that the degree of activity limitation or restriction in participation is not always directly proportional to the impairment. This is why it is inadequate to infer (or guess at) what an elderly person can or cannot do simply based on medical diagnosis or even physical examination (impairment level). Assessments of functional performance (activity level) and social aspects (participation level) are important.

Inactivity is the most common reason for loss in functional capabilities in the aged. Immobilisation due to disease or environmental factors (e.g. acute hospitalisation) results in deconditioning even after a few days. Deconditioning is described as multiple changes to organ system and physiology that are induced by inactivity and reversed by activity. Complications of bed rest include increased risk of falls, impaired mobility, bed sores, loss of muscle mass and strength (loss of strength at a rate of 2-3% per day) as well as other multiorgan involvement. Functional loss easily occurs when threshold values for physical performance are crossed. Therefore bed rest should be prescribed judiciously with an ongoing and appropriate activity programme.

Rehabilitation should be aimed at preventing

functional decline and optimising independence, preventing inactivity and frailty from progressing. The perceived level of health, anxiety, depression, and other psychosocial factors play an important role in how much rehabilitation the patient receives. Special considerations should be made to the cognitive status of older people who may be unable to learn new tasks and comply with regimented exercise programmes. Changes in the sensory system, neuromuscular, and skeletal systems prohibit correct posture, a safe gait pattern, and exercise capacity. Changes in the pulmonary and cardiovascular systems prevent full physical work capability. Thus, motivating older people to adhere to exercise programmes needs to take account of these physiological changes and requires specific instructions such as "do this exercise for as long as you feel comfortable, then stop and rest". The level of prescribed exercise should also be tailor-made.

4.4 Goal Setting in Geriatric Rehabilitation

Goal setting in geriatric rehabilitation focuses on maximising functional independence and quality of life. The wish and choices of an elderly person should be respected. In practice, it is often constrained by a number of parameters:

- Patient's functional level prior to the episode of illness or disability
- Severity of concomitant diseases and impairments
- Whether support from family members and other caregivers is present, and to what extent
- Safety considerations

Some elderly patients and family members may have excessively high expectations of functional recovery. Unrealistic expectations require management and advice by healthcare professionals. Often the opposite is the case — low self-esteem and low expectations of an elderly person (sometimes also from the healthcare professionals) may lead to suboptimal rehabilitation and outcome.

Elderly persons may display a sense of helplessness related to their impairment and disability. Before labelling them as 'poorly motivated', it is important to look for reversible factors that may mimic lack of volition or 'poor motivation':

- Pain that may impair a patient's training and cause sleep disturbance, e.g. osteoarthritis may be exacerbated in a stroke patient during standing and walking training
- Fear of fall: common in post-fracture elderly people who do not 'want' to train because they are frightened of falling again
- Depression: reactive depression or adjustment disorder is common after stroke and other acute causes of disability. It responds to supportive counselling and if necessary, antidepressants
- Side-effects of medications: Is the patient lethargic due to electrolyte disturbances? Dull due to anticholinergics or sedatives?

Goal setting should be based on an understanding of the underlying pathological process causing the disability, and knowledge of prognosis and the course of the recovery. In practice, setting rehabilitation goals often presumes prognosticating subgroups. For instance, an elderly person with significant underlying cardiopulmonary conditions is unlikely to be able to meet the energy demand of prosthetic walking after above-knee amputation and is likely to remain chair-bound; a stroke patient with a combination of motor, sensory, and visual deficits cannot expect to walk outdoors unassisted; an elderly individual who requires a stick for walking before a hip fracture is likely to need a quadripod or walking frame on discharge.

Living at home is a major rehabilitation goal for many elderly persons with a moderate level of disability. The decision to place an elderly individual in residential care should not be made lightly and without first exploring the possibility of living at home with social support and adaptations. Sometimes assessment in the form of a home visit (domiciliary visit) by the rehabilitation professionals is necessary. Appropriate aids and devices will enable an elderly person to engage in functional ambulation and self-care activities.

Services provided by non-governmental organisations and post-discharge support services of many public hospitals can now enable older people to be returned to the community with support such as meals on wheels, home-help service, and outreach nursing or allied health services. These services are invaluable in ensuring a safe return to home and also enable follow-up for any unresolved health or environmental issues for those living alone.

4.5 Assessment of Functional Status and Outcome

Assessment refers to the process of evaluating a patient's impairment, activity, and participation level, including recognition and measurement and determining the cause of functional problems.[6]

Standardised assessment scales are of help for the following purposes.

- Diagnosis: Is a specific impairment present?
- Prognosis: What outcome may be expected?
- Quantification: How severe is the problem?
- Measuring outcome: How much change has occurred?
- Audit: Are the desired process and outcome of

rehabilitation achieved?
- Research into a specific disability
- Population screening: How prevalent is a specific disability in the population?

Functional measures should be relevant, validated, and reliable with high sensitivity and specificity. Measures used in different settings should be suitably chosen and training may be required to use the tool appropriately. For example, a busy family physician may apply the Abbreviated Mental Test for screening whereas an experienced occupational therapist may use the Dementia Rating Scale that is a more detailed multidimensional assessment.

In the context of geriatric rehabilitation, the focus is often on self-care functions although this narrow focus is not always adequate. Many aspects of the patient's function (and functional limitations) can be measured, but it is important to remember that assessment is not synonymous with routine application of standardised assessment scales. Useful assessment can also be made qualitatively, e.g. by careful observation of the gait pattern of a neurological patient.

4.5.1 Self-care Activities, or Basic Activities of Daily Living

There are various ways to categorise activities of daily living (ADLs). Most models and assessment scales group personal self-care (e.g. bowel, bladder management, grooming, dressing, feeding, transfer and mobility, personal hygiene, and toileting) into one category (called personal or basic activities of daily living [BADLs]), and other homemaking and interpersonal items (e.g. home maintenance, housekeeping and managing personal finance, recreational, community participation) in another (called instrumental [IADL] or extended activities of daily living).

The most popular BADL scales are the Barthel Index (BI) and the Functional Independence Measure (FIM). These are global functional scales widely used in different contexts including stroke and fracture rehabilitation. BI is more popular in the UK, where it is the most widely studied of all self-care measures. Many centres in Hong Kong are still using the BI or modified BI because it is simple and easy to use.

The original BI (Table 4.1) was developed in 1965 by Mahoney and Barthel.[7] It has 10 items of physical functioning and none for cognitive functions. The maximum total score is 20, hence abbreviated as BI-20. Over the years there have been numerous attempts to modify it, including a version by Shah et al,[8] who tried to improve its sensitivity to functional changes by making the total score 100 ('BI-100' or modified BI). A Chinese version of the Modified Barthel Index (MBI-C) with item content and rating criteria adapted to Chinese culture has been validated in Hong Kong for elderly stroke patients.[9]

Table 4.1 Items and scores in Barthel Index BI-20[7] and BI-100[8]

BI-20		BI-100	
Item	**Score**	**Item**	**Score**
Bowel	0,1,2	Bowel control	0,2,5,8,10
Bladder	0,1,2	Bladder control	0,2,5,8,10
Grooming	0,1	Personal hygiene	0,1,3,4,5
Toilet use	0,1,2	Toilet	0,2,5,8,10
Feeding	0,1,2	Feeding	0,2,5,8,10
Transfer	0,1,2,3	Bed / chair transfer	0,3,8,12,15
Mobility	0,1,2,3	Ambulation	0,3,8,12,15
		Or wheelchair	0,1,3,4,5
Dressing	0,1,2	Dressing	0,2,5,8,10
Stairs	0,1,2	Stairs	0,2,5,8,10
Bathing	0,1	Bathing	0,1,3,4,5
Total	**0-20**	**Total**	**0-100**

The FIM[10,11] is a more complex scale. It comprises 18 items organised into six categories, each item having a 7-point scale. FIM was developed in the US and is being increasingly adopted by rehabilitation centres in Europe, Australia, and China including Hong Kong. Its strength lies in the more exact specifications for scoring. Rehabilitation personnel, however, need appropriate training before using FIM.

4.6 Rehabilitation Therapy for Older People

Exercise should be considered and encouraged by all healthcare professionals for all elderly persons with deconditioning problems and physical disability. Inpatient rehabilitation specifically designed for geriatric patients has the potential to improve outcomes related to function, admission to nursing homes, and mortality.[12] Group- and home-based exercise programmes, and home safety interventions reduce the rate of fall and risk of falling[13] as well as having many other positive health outcomes. Prescription of therapeutic exercise in elderly people should take account of physical (particularly neuro-muscular-skeletal) and cardiopulmonary limitations.
- Gentle active stretching exercises increase flexibility but have little effect on strengthening and endurance.
- Resistive exercise has a strengthening effect (e.g. quadriceps exercise with appropriate ankle weights) and can reverse muscle atrophy.
- Weight-bearing exercises maintain or increase bone mass. Swimming is a good aerobic exercise but it is not weight-bearing and has little benefit for osteoporosis.
- Elderly patients with cardiac problems or severe

chronic obstructive pulmonary disease should be specially assessed (e.g. with a sub-maximal stress test) and advice given before embarking on exercise training.

Aerobic exercises are those that involve rhythmic movements of large muscle groups. Stationary cycling, daily walking, swimming, and Tai Chi exercises are suitable aerobic activities. An exercise programme of 20-30 minutes 3-5 times a week that does not exceed 60-80% of maximal heart rate may be optimal. Warm-up and cool-down periods render the exercise programme safer.

In a re-conditioning programme, it is prudent to start with low intensity, short exercise duration. Interval breaks allow some rest, but the session time should be kept at 30 minutes or longer. In addition, resistive exercises can be used (e.g. ankle weights) for strengthening.

The intensity of the exercise can be guided by either the percentage of maximal heart rate (HR_{max}), or rate of perceived exertion. One way of calculating target heart rate is by an aged-adjusted maximal heart rate formula, HR_{max} is (220 minus age). Target heart rate is set at 60-80% x HR_{max} for the relatively fit elderly individual. A lower range (45-55%) is selected for the significantly deconditioned or sedentary elderly. Although this method is easier to calculate, it is not commonly recommended because it yields a significantly lower target heart rate, especially in the very old. For example, an 80-year-old patient will have a predicted target heart rate of 84/min that may also be their baseline heart rate if we use this formula: (220-80) x 0.6 = 84. Also, there may be confounders such as beta-blockers or beta-agonist medication intake.

A useful concept of the intensity of exercise, and the capacity for it, is the metabolic equivalents (METs). 1 MET is equivalent to the consumption of 3.5 ml O_2/kg/min. Some examples of MET levels are as follows.

- A healthy older person should be able to engage in activities of 8 METs or more.
- The minimal aerobic power compatible with full independence is around 3.5 METs.
- Performance of very light self-care ADL, e.g. washing, shaving, writing at desk, washing dishes and clothes, requires about 1.5-3 METs.
- Performance of light household work, e.g. mopping floor, cleaning windows, vacuuming, carrying 15-30 lb objects requires about 3-5 METs.
- Use of walking aids and prosthesis in amputee patients increases the demand significantly.

The use of METs can guide aerobic exercise endurance but it should be used cautiously in exercise prescription. This is partly because most of the experiments and data used in constructing the table showing the MET for different activities are derived from normal subjects who were healthy and young, hence their values tend to be overestimated for elderly patients with cardiopulmonary problems. When the MET is used for exercise prescription, it is advisable to begin the exercise programme with an initial target range of 50-60% of the 'maximal' MET level, estimated from the cardiovascular or cardiopulmonary graded exercise stress test. For elderly patients who may have cardiopulmonary disease, the initial training intensity may be as low as 40% of the 'maximal' MET level with gradual escalation if tolerated.

Rate of perceived exertion is a simple scale developed by Borg[14] in 1973 (revised 1982), also known as the Borg's scale (Table 4.2). For reconditioning of an elderly person, levels 3 to 5 are appropriate.

4.6.1 Elderly Persons after Operative Treatment of Hip Fracture

Early operation and early postoperative mobilisation are now proven to be important for good outcome. Postoperative weight-bearing exercises are usually prescribed by orthopaedic surgeons and carried out by physiotherapists. Different surgeons may have different preferences for the pace of progress in weight bearing. Factors to consider include the type of fracture, the type of internal fixation, any intra-operative complications, and the general physique of an elderly person before fracture. The common types of weight-bearing instructions include:

- non–weight bearing (NWB): 0% body weight placed on postoperative limb
- touch-down weight bearing (TDWB): 10-15% body weight placed on postoperative limb
- partial weight bearing: 30% body weight placed on postoperative limb
- full weight bearing: 75-100% body weight placed on postoperative limb

Table 4.2 Rate of perceived exertion

Rating	Description
0	Nothing at all
1	Very weak
2	Weak
3	Moderate
4	Somewhat strong
5-6	Strong
7-9	Very strong
10	Very, very strong
*	Maximal

Few elderly persons can comprehend partial weight bearing in percentage terms. In practice, careful physiotherapist instructions and supervision using appropriate walking aids for training ensure safety.

There is only approximate and incomplete consensus for guiding progression in ambulation. Progression is fastest in cases with a cemented type of hemiarthroplasty. A fractured neck of femur with internal fixation by compression hip screw ('dynamic hip screw') is also generally stable. More cautious initial ambulation by TDWB is necessary in most subtrochanteric fracture cases. Comminuted fracture with loss of cortical stability on the medial side ('medial buttress') and subtrochanteric fractures may require a period of NWB and TDWB.

Apart from the orthopaedic considerations, the early postoperative period may be complicated by medical problems (deep vein thrombosis, pulmonary embolism, retention of urine, hospital acquired sepsis, delirium, constipation). Nutritional status also needs monitoring as many are at risk of malnutrition and poor intake with associated weight loss. The role of the geriatrician or rehabilitation physician becomes invaluable in managing such frail patients.

Advices to be given to an elderly person post–hip fracture rehabilitation (also to caregiver) are shown below.

- Sitting: do not cross legs / do not reach for objects on the floor by bending trunk / level of knees should be below level of hips (i.e. hip flexion post-discharge <90 degrees; postoperative in-hospital <60 degrees)
- Walking: do not turn suddenly on the operated leg
- Up-stairs with walking aid: 'good' leg first, then operated leg, then walking aid
- Down-stairs: reverse order ('good' leg last)
- Exercise: besides walking, strengthening abduction exercises for the operated leg will reduce lurching (Trendelenburg) limp. Lie on the 'good' side, abduction (side-raising) of operated leg for 20-30 repetitions. May add ankle weight as tolerated.
- Encourage resumption of life activities after full progression of the training period. Avoid social withdrawal and immobilisation.
- Secondary prevention with specific treatment for osteoporosis.

4.7 Gait Aids and Wheelchair

To become safely independent in functional ambulation, the patient must have adequate strength, balance, range of motion, sensory perception, and cognitive skills. Gait aids are considered to be an extension of the upper limbs. Determining the gait aid best suited to an individual depends on the amount of weight bearing permitted on the lower limb, patient's balance, and upper limb and trunk function. The use of a gait aid helps to transmit body weight away from the affected lower limb. Nonetheless compared with normal unassisted gait, the energy expenditure per unit distance will be increased when weight-bearing proportion decreases on the affected side. All patients should be trained in the proper use of the gait aid. Some commonly prescribed aids[15] and their current local price range are listed in Table 4.3.

4.7.1 Cane / Walking Stick (Figures 4.1a, 4.1b)

As a walking aid, it is held in the hand opposite the affected side. It widens the base of support and reduces stress on the involved lower limb that may be injured, inflamed, or weakened by shifting the centre of gravity towards the contralateral arm. With the use of a cane, the upper limb exerts force on the cane that minimises pelvic drop on the side opposite the weight-bearing lower limb. When ascending stairs, the good leg is advanced first, but when descending, the cane and the affected leg lead. Nowadays, canes are commonly made of aluminium that is lightweight with adjustable notches at its shaft. The height of a cane should be adjusted so that the highest point is at the level of the greater trochanter when the patient is in an upright position. This will position the elbow in 20-30 degrees of flexion. Too long a cane will force the elbow into too much flexion and put excessive demands on triceps and shoulder muscles. A too

Table 4.3 Price range of basic model of commonly prescribed aids in Hong Kong (as at May 2017)

Aids	Price (HK$)
Standard wheelchair	1300 - 3380
Tilt-in-space wheelchair	6500 - 15,000 100,000 - 140,000
Power wheelchair	17,000 - 40,000
Mobile hoist	18,000 - 30,000
Ceiling hoist	36,000 - 78,000
Commode chair	Non-wheel: 500 Wheel: 1500 - 2200
Tilt-in-space commode	13,000 - 25,000
Raised toilet seat	280 - 480
Shower chair	260 - 1300
Bath board	280 - 380
Handrails	300 - 500 Foldable: 1000 - 1900
Ripple mattress	1450 -2600
Sitting cushion	800 - 4000
Geriatric chair	2200 - 4000
Hospital bed	6500 - 16,000
Cane (stick)	80 - 250
Quad	100 - 450
Walker (frame)	225 - 450
Rollator	500 - 900

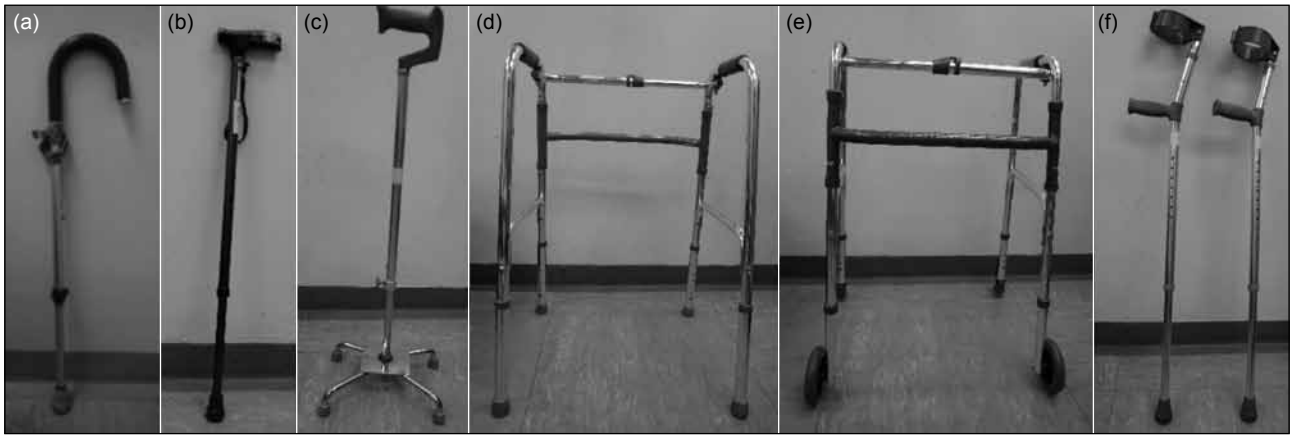

Figure 4.1 Commonly prescribed walking aids: (a) walking stick with C-handle; (b) walking stick with functional grip handle; (c) quadripod; (d) walker (frame); (e) rollator; and (f) crutches

short cane will tend to hold the elbow in complete extension and hence reduce support during the stance phase.

There are several varieties of cane handle. The C-handle (Figure 4.1a) is commonly used but may be uncomfortable and difficult to grasp, especially for those with hand problems. Moreover, the weight-bearing line falls behind the shaft of the cane, reducing its supportive value. A functional grip handle (Figure 4.1b) fits the grip and being more comfortable and more centred over the shaft provides better support. Some canes have a plastic palm grip at the top to provide better grip. Canes are usually rubber tipped to absorb shock and prevent slippage. Clinicians, users, and carers should check these regularly for wear and tear.

4.7.2 Quadripod (Figure 4.1c)

As canes have only a single point of contact with the body, they provide less support than other gait aids and are not advisable for patients who need more than partial weight relief. Stability can be increased by adding extra legs to the base of the cane to make it a quad. The gait of the patient is slower than that using a cane. The quad base of support can be either narrow or wide. In using the quad, the lateral two prongs should be directed away from the body.

4.7.3 Walker / Frame (Figure 4.1d)

Walkers (Figure 4.1d) have a large base of support, and are very stable and easy to use, especially when compared with canes, quads, and crutches. It often serves as the first walking aid especially for patients with poor balance, strength, and coordination. Sometimes weights (e.g. sandbags) can be added to the crosspieces of the frame for greater stability in patients with lower limb incoordination but good upper limb strength. The disadvantages are its larger size and lack of manoeuvrability that may restrict it to indoor activities and patients are unlikely to be able to climb stairs using it. Patients can often progress to a less cumbersome walking aid after further training and functional recovery. To use the walker, a patient needs good standing balance and upper limb strength to lift it and advance it forward.

Walkers usually have adjustable legs and can often be folded for easy transportation. The proper height of the walking frame is waist level when the patient is standing upright. This should place the handgrips so the elbows are in 15-20 degrees of flexion. A walker is fitted by placing it about 10-12 inches in front and partially surrounding the patient.

4.7.4 Rollator (Figure 4.1e)

The rollator is a metal frame walker with two rubber wheels at the front and two rubber tips at the back. The handgrips are adjustable and should be at waist height when the patient is standing upright. The rollator provides less stability than the walker but is more manoeuvrable. It helps to facilitate movement of the walker for patients who lack coordination or strength in the upper limbs or trunk that creates difficulty in lifting up and advancing other walkers. The front wheels, however, may provide some instability and patients usually require supervision.

4.7.5 Crutches (Figure 4.1f)

Crutches provide better stability than canes. Crutches should only be prescribed to patients with enough strength in their upper limbs, especially the key muscle groups of shoulder flexors and depressors, elbow and wrist extensors and finger flexor so that the elbows can be extended and raise the body 1 to 2 inches off the floor. As using crutches needs more

skill than other gait aids, it is less commonly used in elderly people.

4.7.6 Wheelchair

Wheeled mobility systems can be manually propelled or attendant propelled. Because a wheelchair is often used during all waking hours and during all activities, having the right one can make the difference between independence and dependence. Associated important considerations are reach dimension of the wheelchair user and space required for wheelchair passage and turning. Details of the dimensions can be accessed through the Design Manual — Barrier Free Access 2008 by the Buildings Department of Hong Kong SAR Government (http://www.bd.gov.hk/english/documents/code/BFA2008_e.pdf).

4.7.6.1 Manual Wheelchair

- Indications: Wheelchair to be manually propelled by patients with sufficient strength in upper limbs or at least one upper or lower limb. They are usually prescribed for patients in whom prolonged walking is contraindicated such as poor endurance, functionally impossible, inadvisable due to cardiopulmonary, neuromusculoskeletal, or musculoskeletal conditions.
- Contraindications: Absolute contraindications include blindness and poor judgement. Relative contraindications include ischial pressure ulcers, postoperative conditions of the pelvis or proximal femur, vertebral fractures, disc and nerve root compression, truncal weakness, and postural defects.
- Prevention of complications: Back pain and deformities can be prevented by proper positioning or seating. Pressure ulcers can be prevented by frequent weight shifting. Lower limb contracture may be prevented by frequent stretching. Dependent lower limb oedema may be prevented by elevating the legs and using compression stockings.
- Components: Please refer to Figure 4.2 for wheelchair terminology.
- Checklist: The SEAT x 2 checklist as listed in Table 4.4 serves as a very useful guide and memory aid for the clinician as well as a comprehensive communication tool for the team.

4.7.6.2 Attendant-propelled Wheelchair

These wheelchairs are to be pushed by another person. Common long-term indications are cognitively impaired patients unable to walk due to various medical conditions and for elderly people who are unable to cope with long distance ambulation. Factors to consider when ordering an attendant-propelled wheelchair include not only the fitness and comfort of the patient but also that of the person pushing the wheelchair.

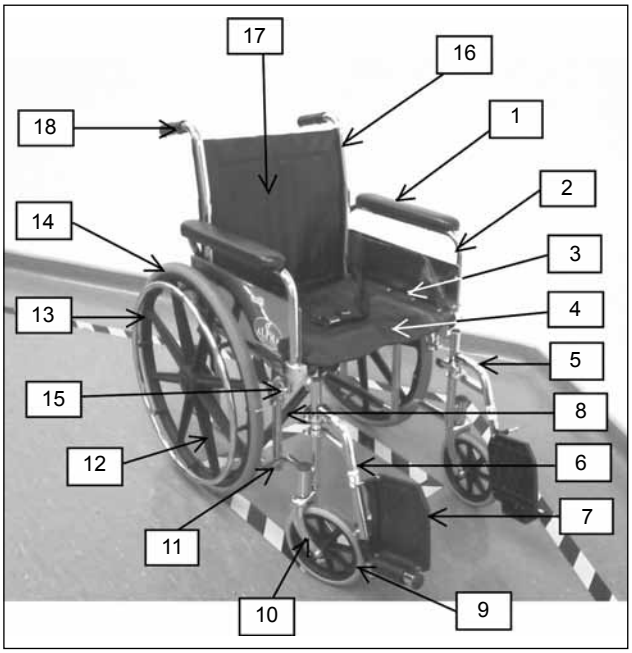

Figure 4.2 Wheelchair terminology
1: Arm pad; 2: Arm rest; 3: Clothes guard; 4: Sling seat; 5: Down tube; 6: Foot rest; 7: Footplate; 8: Cross brace or X-frame; 9: Caster; 10: Caster fork; 11: Tipper lever; 12: Axle; 13: Handrim; 14: Wheel; 15: Brake; 16: Back post; 17: Sling Back; 18: Push handle.

Table 4.4 The SEAT x 2 checklist for wheelchair

Support (***SCALPS***)
Safety (restraints and stability)
Comfort and Cosmesis
Arms
Legs
Pelvis
Spine and head
Skin — Pressure distribution and relief
Easy propulsion
Easy transfers
Alternation of tone
Accommodation (***GrOW FAST***)
Of ***G***rowth (more relevant in paediatric cases)
Of ***O***ther (Individualise)
Of ***W***orsening (progressive disease)
Of ***F***unctional ***A***ctivities (ADL needs)
Of ***S***tructural deformities and contractures
Of ***T***echnology
Transportability
Terrain

Abbreviation: ADL = activities of daily living

4.7.6.3 Complex Medical Condition Requiring Special Attention

Some patients may require passive positioning to inhibit gastroesophageal reflux and facilitate gastric emptying. Seating may then require both reclined and upright positions following meals.

People with restrictive lung disease, aspiration, and other pulmonary conditions may require varied therapeutic positioning for good pulmonary toilet. They may require proper trunk support without restriction of rib movement. Ageing and dementia patients usually lose their depth perception which creates a strong fear of movement. Seating addresses this fear by providing security through more surface contact. Cushioning to aid comfort can usually help. For an elderly person with severe kyphosis, sitting in a slightly reclined position may be helpful to move the trachea out of the dependent position and prevent aspiration.

4.8 Adaptations to ADL

Functional independence in performing ADL can enhance positive self-image and quality of life. Its attainment depends on a large range of coordinated body functions including range of motion, strength, endurance, coordination, dexterity, balance, sensation, vision, cognition, and perception. The general principle of ADL adaptation includes restoration of body function to accompany the task, or modification of the task and environment so that the task can be accomplished safely.

One study found that simply facilitating the patient's participation during ADL, rather than performing the activity for the patient (e.g. having the patient help dress themselves rather than being dressed), increased independence and reduced disruptive behaviour in nursing home patients with dementia.[16] In the process of assisting people, families and significant others should be included. Family-centred care includes recognising family strengths, respecting different methods of coping, and facilitating family and professional collaboration at all levels of healthcare. The process should be continuously supported by the team. Family members also play an important role in evaluation and advocacy for the person in need of rehabilitation services.

The detailed evaluation of these functional limitations as well as their specific ADL adaptations and functional ambulation are typically carried out by an occupational therapist and physiotherapist with input from other members of the interdisciplinary rehabilitation team. Commonly prescribed aids for elderly people and the cost of the basic models are summarised in Table 4.3.

Many aspects of design for persons with a disability applied to both the interior and exterior of all structures and buildings with specific chapters on elderly with frailty have already been covered in the Design Manual – Barrier Free Access 2008 by the Buildings Department of Hong Kong SAR Government.

4.8.1 Specific Principles for ADL Adaptation[17-19]

4.8.1.1 Limited Range of Movement

Using adaptations to increase reach as well as storing frequently used items within reach is helpful. Joint protection techniques for arthritis patients and compensation for limited grasp are commonly used strategies.

4.8.1.2 Weakness

Commonly used techniques include use of two hands, increasing friction to decrease the power needed for pinch or grasp, allowing gravity to assist, and using lightweight or powered tools.

4.8.1.3 Low Endurance

Energy conservation techniques are commonly used to overcome the problem. These include good planning and organisation of work ahead with elimination of extra or unnecessary tasks; having a good setup and preparation before starting a task; pacing work with rests to prevent fatigue; using lightweight or powered tools and working with gravity assistance; avoiding stressful positions and environmental stressors such as a hot and humid environment.

4.8.1.4 Incoordination or Poor Dexterity

Using anti-slip materials to stabilise the object that is to be used, stabilising the proximal limb segments so that control can be concentrated on the distal limb segments. Adding weight to distal segments or using heavy utensils may also be helpful in selected situations.

4.8.1.5 Poor Vision

Magnifying words or images with high contrast is common. Appropriate organisation of tools so everything is kept in its proper place to reduce unnecessary searching. Sometimes auditory or tactile feedback may also be used to compensate for low vision.

4.8.1.6 Decreased Sensation

To protect the less-sensitive part from injury, attention should be directed to the affected part on a regular basis such as using a long-handled mirror to inspect skin or using a timer with regular reminders to perform pressure relief.

4.8.1.7 Impaired Cognition

Auditory and visual aids are commonly used as compensatory strategies. Memory aids include an alarm clock, mobile phone, calendar, auditory reminder, camera, diary, pill box, and posted reminders. They are commonly recommended to our elders and carers as compensatory aids. Electrical

Table 4.5 Territory-wide transport network for the disabled

Service provider	Destination			
	Hospital Authority institutions	General outpatient clinic	Private hospital	Others
Hospital Authority				
• Non-Emergency Ambulance Transfer Service (非緊急救護車)	√			
Public				
• Hong Kong St. John Ambulance Services			√	
• Non-Emergency Ambulance Transfer Service provided by Auxiliary Medical Service		√	√	
Non-governmental organisation or private				
• Easy-Access Bus (易達巴士)	√			
• Rehabus Feeder Service (復康穿梭巴士)	Six feeder routes			
• Accessible Hire Car Service (易達轎車)	√	√	√	√
• Diamond Cab (鑽的)	√	√	√	√

appliances with a timer can also help minimise risk of fire for those cognitively impaired. Mobile link service (隨身寶) is an outdoor support service for elders. It is a one button mobile device equipped with a hands-free speaker, location tracking, and remote buzzer features. Personal Emergency Link Service (平安鐘) provides 24-hour support to elderly people: whenever the link device is pressed, the user is connected directly to the call centre. Safety Phone Service (平安手機) is designed for the socially active elderly people with a large screen and keypad, one button solution for assistance call and GSM tracking. These services may be rented on a monthly service plan from commercial telecommunication companies.

4.8.2 Some Common Adaptations

The difference between BADL and IADL has been mentioned above in Chapter 4.5. BADLs involve personal care while IADLs refer to more advanced problem-solving skills and more complex environmental interactions. It also includes environmental adaptations to facilitate home and community access as well as transportation adaptations.

4.8.3 Seating Systems

In elderly people, seating is important for general care. A proper seating system allows them to be placed in an upright position to improve respiration, digestion, and urinary function. It also allows individuals to be transported more easily and helps others to perform their care and hygiene. Moreover, it offers projection of an attractive and healthy body image.

Some common ergonomic considerations in prescription of seating devices include seat height which is the distance from the bottom of the heel to the popliteal area; seat depth that should be 1-2 inches shorter than the distance from the popliteal area to the back of the buttocks, and armrest height which is the distance from bottom of the buttocks to elbow. From clinical experience with reference to the standard chair dimensions commonly available, it is common to prescribe chairs with seat height of 14-17 inches and seat depth of 15-16 inches. Special attention should be paid to avoid excessively soft seating cushions, especially for sofas, as they actually lower the seat height and make standing more difficult for elderly people.

4.8.4 Community Transportation

Community transportation is important to promote reintegration of disabled people into society. It includes public transportation or self-driving. Driving is a complex task that requires continuous integration of visual, motor, cognitive, and perceptual skills at a high level of functioning. Driving by an elderly person is relatively rare in Hong Kong when compared with utilisation of public transportation.

Public transportation adaptations include tactile guide paths, escalator audible signals, colour-contrast grab poles, braille plates, and a buzzer sound system for those visually impaired. Induction loops to assist hearing-aid users, information display and in-train information panels are helpful for those passengers with hearing impairment. Facilities to help those with impaired mobility include multi-use spaces in trains, wide gates, portable or movable ramps, front kneeling capability of bus, wheelchair space with back rest and restraining lap belt. Table 4.5 provides further information about the territory-wide transport network. The 'Guide to public transport for people with disabilities', published in 2014 by the Transport Department of Hong Kong SAR Government, offers information about facilities for the disabled on public transport, the Rehabus services, and public transport operators (http://www.td.gov.hk/mini_site/people_with_disabilities/2014/index.htm).

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CHAPTER 5

Long-term Hospital Care: Infirmary Care

Tak-kwan KONG

OBJECTIVE

To learn the organisation of long-term hospital care, what determines high-quality long-term care and how it can be achieved, and assessment specific to a long-term care environment.

KEY POINTS

- Long-term hospital care or infirmary care focuses on providing chronic care for individuals who are unable to manage independently in the community.
- An infirmary is a low-technology environment, functionally the home of the resident.
- Patients in long-term care hospitals have chronic illnesses with losses and isolation; multiple comorbidities (e.g. stroke, dementia); and may become increasingly frail, often marked by multiple hospitalisations; and their terminal phase may span over a longer period than those with incurable cancer.
- High-quality long-term care requires attention to autonomy, urinary continence, faecal continence, drug use, falls, pressure sores, disability, dementia, and depression.
- High-quality long-term care is hindered by heroic medicine, intense protection (safety concerns), legal ethics (defensive medicine), task-orientated staff, a medical-social divide, and strict infection control measures.
- Specialist clinical assessment prior to institutional placement is beneficial for elders and their carers, and enables identification of previously unreported yet treatable and remediable clinical conditions and disabilities.
- Pre-admission assessment in Hong Kong is performed by Community Geriatric Assessment Teams using a standardised Infirmary Core Assessment Form.
- The international interRAI Long-Term Care Facilities Assessment System (interRAI LTCF) consists of assessment items specific to long-term care settings as well as core elements common to other settings.

5.1 Introduction

Long-term hospital care or infirmary care may be defined as the institutional provision of healthcare for individuals who are unable to manage independently in the community. This care may be chronic care management or short-term rehabilitation. Acute hospitals and long-term hospital care differ with regard to patient acuity and resource provision in terms of diagnostic (availability of laboratory and radiology / imaging investigations) and staffing (nurse-to-patient ratios) support. While acute hospital care focuses on providing intensive care, often using high technology, to a patient who is generally expected to recover or improve, infirmary care focuses on providing chronic care to those unable to manage independently in the community. An infirmary is a low-technology environment, functionally the home for the resident who is usually an elderly person with deteriorating health and who often stays for years, hence comfort, dignity, and rights are paramount.

Historically, geriatric medicine arose as a specialty in the 1940s in the UK in reaction to an attitude of apathy towards elderly patients who were thought to be incurable and consequently dumped in chronic institutions or workhouse infirmaries (see Chapter 1.1.1). A note recorded in 1806 in a British Infirmary stated, "doctors are tending to refer to the infirmary people who are not proper objects to be admitted and who should rightly be referred to the workhouse and almshouse,"[1] and reflected the lack of medical interest or special care for frail elderly people at that time. A traditional geriatric department deploys its beds in a system of progressive care with three main types of bed provision: beds for acute illness and assessment, beds for rehabilitation, and beds for long-term care.[2] In the 1990s, geriatric medicine in the UK had developed towards acute medicine and

rehabilitation. Long-stay or continuing care beds in hospital had declined and been replaced largely by long-term care in the community with varying degrees of input from geriatricians.[3,4] The most dependent frail elderly people with the greatest nursing needs remained in hospital beds. In Scotland, frail elderly patients are admitted to hospital long-term geriatric medical beds after assessment by geriatricians. The reasons for entry to long-stay hospital care include complex medical conditions or special nursing needs; slow rehabilitation; need for terminal nursing care; carer respite; or awaiting placement elsewhere e.g. residential care or nursing home beds.[5] Geriatric medicine in North America follows a different path towards the care of chronic complex frail elderly people with institutional long-term care for the most dependent within nursing homes.[6,7]

5.2 Infirmary Care in Hong Kong

In Hong Kong, infirmary hospitals were established in the public sector to provide care for elderly and chronically ill patients and reduce pressure on acute hospital beds. Historically, such infirmary hospitals had a poor image, were located in less accessible sites, lacked on-site medical support, and were regarded as a dumping ground for patients.[8] Introduction of the geriatric principles of assessment, rehabilitation, and continuing care at these infirmary hospitals has transformed the service provision and enabled some patients to become sufficiently fit through rehabilitation to be discharged back to the community. Those who remain severely dependent continue to be cared for in the infirmary.[8] In 1981, for the purpose of estimating demand, the Medical Development Advisory Committee adopted a planning ratio of five general infirmary beds per 1000 elderly persons aged ≥65 years. In 1989, a Central Infirmary Waiting List (CIWL) was established for applicants in need of infirmary care in public hospitals. To enable ageing in place for frail elderly residents of Care and Attention Homes who had declined in health to an infirm status, 300 infirmary unit places were set up in 1992 in 10 Care and Attention Homes run by voluntary agencies and overseen by the Social Welfare Department. As of the end of June 2014, 19 such residential care homes were running 29 infirmary units providing a total of 580 infirmary unit beds. Since the setup in 1994 of district-based Community Geriatric Assessment Teams (CGATs) led by geriatricians, those registered on the CIWL are assessed by CGAT to ensure that only those who require long-term hospital care will receive it. Those in need of imminent intensive nursing care and frequent medical attention are given priority placement with a quota set aside each year for this purpose. As at December 2015, there were 2041 infirmary beds in public hospitals providing rehabilitation and / or long-term care, designated either as central infirmary beds (1234 in number) for admission from the CIWL, or local infirmary beds (807 in number) for slow-stream rehabilitation or the continuing care of patients recovering from an acute medical episode.

A 2002 survey by geriatricians of CIWL applicants and patients occupying infirmary beds revealed that the two principal diagnoses leading to infirmary status were stroke (58.8%) and dementia (26.2%).[9] The principal reasons for recommending hospital infirmary care were dependency on hospital-based technological support and / or complex professional care to prevent excess mortality. Analysis of the current residence of applicants on the CIWL showed that 80% lived in residential care homes (50% private, 30% subvented) while 20% lived in their own homes.[9] Many of these frail elderly people in the community were supported by outreach service provision by CGAT and Community Nurses.

In 2013, a Task Group of the Hospital Authority (HA) General Infirmary Service was set up under the Geriatric Subcommittee of the HA to give advice on the development, coordination, and provision of a General Infirmary Service in public hospitals; on issues related to the administration of central infirmary applications and placement; and on interface issues to foster collaboration with relevant external stakeholders. A website that describes the HA General Infirmary Service, its application procedures, and infirmary eligibility criteria is available to concerned professions and the public at: <http://www.ha.org.hk/visitor/ha_visitor_index.asp?Parent_ID=10085&Content_ID=10100&Ver=HTML>.

In September 2016, in line with the Government's policy direction, the HA piloted an Infirmary Service Public-Private Partnership Programme by contracting with a non-governmental organisation, Po Leung Kuk, to operate infirmary services at the Wong Chuk Hang Hospital. The Hospital was converted to the Po Leung Kuk Wong Chuk Hang Extended Care Home with a maximum capacity of 64 beds. It is envisaged that the Care Home will provide a home environment in a non-hospital setting without compromising quality of care. Medically stable CIWL applicants are selected for the pilot by CGATs and the district-based CGAT provides professional advice about the provision of infirmary care in this pilot Care Home. Information about this Infirmary Service through Public-Private Partnership is available on the website <http://www3.ha.org.hk/ppp/infirmppp.aspx?lang=eng>. With a trend towards more and more relatively stable frail elderly patients being cared for in the community outside of a hospital setting, the complexity and frailty of patients who remain in hospital infirmaries will grow, posing a challenge to

the staff and support required. A survey in October 2014 of the central infirmary units of the HA revealed that the overall mean percentage of central infirmary patients with tracheostomies was 14.4% although this varied from 0% to 44% among different public hospitals with infirmary units. The predominance of patients with a tracheostomy in some infirmary units, not necessarily chronologically old but biologically aged, often with multiple comorbidities and disabilities, has implications for manpower and facilities support, as well as infection control. In the second half of 2016, realising the importance of oral hygiene and dental health in frail elderly patients, the Government piloted an outreach dental service to two public hospital central infirmary units.

5.3 Goals, Standards, and Quality of Long-term Care

5.3.1 Long-term Care Patients

Patients in long-term care hospitals have chronic illnesses with losses and isolation; often with multiple comorbidities (e.g. stroke, dementia, fractures, chronic obstructive pulmonary disease, heart failure); and may progress to increasing frailty, often marked by multiple hospitalisations. Their terminal phase may span a longer period of time than those with incurable cancer.

5.3.2 Need for a Home in a Long-term Care Hospital

The goal of long-term care is to provide a good quality of life for patients[10] and is related to the physical environment, the social environment, and attitudes and behaviour of care staff. It is often assumed that care in the community outside of a hospital is more homely. However, Millard[11] argued that "the hospital and even the institution is part of the community, and irrespective of how good care at home is there will always be some people who require to be tended within institutions. Hospital care is not always bad, and home care always good, for some the alternative is the truth. For some the hospital and the residential home represent the right choice." For those who have become infirm and end up in a long-term hospital (infirmary) for the rest of their life, the hospital becomes home. In the Bolingbroke Hospital long-term care project developed for multiply handicapped elderly people in London, Millard created a home within a hospital, with a homely design of furniture and furnishing, and rooms furnished with residents' own possessions.[11] He showed that the elderly person surrounded by personal belongings was perceived in a less negative way than the same person in bare surroundings.[12] In place of a traditional custodial approach to dementia patients in long-term care institutions, a socially interactive scheme of activities has been advocated: a people-oriented and programme-based approach in the presence of a supportive and therapeutic environment, and with a focus on physical space as a human living space rather than a staff working space.[13] Participating in artwork (e.g. painting, calligraphy) and music also enhances the quality of life of patients in hospital.[14-16]

5.3.3 Guidance for Quality Long-term Care

The quality of long-stay care shows how a society values the aged who cannot be rehabilitated. The need for standards for long-term institutional care for elderly people has been widely recognised internationally and locally. The procedures ensure standards are both mandatory (regulation and inspection) and voluntary (quality assurance). While regulations have tended to focus more on physical structure, space and staffing levels, more attention should be focused on quality of care for the individual resident (privacy, dignity, independence, choice, and fulfilment of residents). Quality assurance has been defined as the process whereby service providers and those receiving care can assess the services to check that acceptable standards are being met. Standards of good practice should include all three elements of structure (number and calibre of staff, physical environment, patients' personal environment), process (nursing procedures, flexibility in the patient's day, patient activities, access to visitors), and most importantly outcomes (benefits to the patient: mobility, continence, social integration, quality of life).[17]

The International Association of Gerontology and Geriatrics (IAGG) has published a textbook of practical knowledge required for quality long-term care in care homes,[18] that can lead to IAGG certification on completion of additional online learning activities <http://www.garn-network.org>. The Royal College of Physicians and the British Geriatrics Society have published guidance and an audit scheme on quality long-term care under the nine headings of autonomy, urinary continence, faecal continence, drug use, falls, pressure sores, disability, dementia, and depression.[10,19,20]

5.3.3.1 Preserving Autonomy

Personal needs assessment identifies personal goals, individual waking and bed times, preferences about clothing, activities, exercise, food, and form of address. Personalised care plans are supported by a key worker. Personal environmental factors should be attended to, e.g. bedroom access, personal possessions; personal equipment (e.g. aids, chair); personal storage and cupboards; variable lighting, heating and ventilation. For those unable to articulate choice, an independent advocate may be sought.

5.3.3.2 Promoting Urinary Continence

Reversible causes of urinary incontinence should be identified. It is important to be sensitive to toileting needs of residents that may manifest as distress, restlessness, or agitation. Pads and pants should be changed regularly.

5.3.3.3 Promoting Faecal Continence

Constipation / faecal impaction is a common cause of faecal incontinence and can present as spurious diarrhoea or reduced appetite / intake. It is unlikely that one suppository will solve the problem, and daily enemas for up to a week may be necessary. Hydration and mobilisation can help reduce the problem.

5.3.3.4 Optimising Medication

The appropriate medication should be prescribed at the minimum effective dose. Use of psychoactive drugs should be regularly reviewed and polypharmacy reduced. Opportunities for non-drug treatments should be sought.

5.3.3.5 Preventing and Managing Falls

Improving muscle strength and functional status by exercise, especially balance training, can prevent falls. Reducing environmental hazards with appropriate lighting, furnishings, and floor surfaces is beneficial. High-risk residents should be identified and monitored. Vitamin D / calcium supplementation may help. Psychotropic medication and use of restraints, which result in more injurious falls, should be avoided. Alternatives to restraint should be applied with attention paid to the human needs of residents (e.g. toileting needs, food, water, warmth, comfort, companionship).

5.3.3.6 Preventing and Managing Pressure Sores

Assessment should determine risk of pressure sore formation, severity and nature of pressure sores, and underlying causes / predisposing factors. A personal care plan should be formulated for skin care, appropriate mattress / support surface and seating, promotion of mobility and activity, and ensuring balanced nutrition.

5.3.3.7 Overcoming Disability by Optimising Aids / Adaptations and Environment / Equipment

Appropriate chairs of different heights should be available for different needs. Beds should be of appropriate height and firmness for different needs. Attention should be paid to the availability of dentures for eating, clean spectacles, access to large print, and properly functioning hearing aids.

5.3.3.8 Positive Care for People with Dementia

The physical environment should provide for safe wandering, including access to gardens and sunshine, provide clues to finding one's way, avoid noxious stimuli (e.g. noise, glare, crowding), and enhance visual, tactile, musical and other sensory stimulation. Staff approaches to care should be individualised, minimise the use of physical and chemical restraints, emphasise the patient's dignity, and involve and encourage family participation in activities and care. Therapeutic programmes and activities should be appropriate for residents' cognitive and functional status, and focus on their strengths, interests, and familiar activities.

5.3.3.9 Detecting and Managing Depression

Depression should be suspected in those with risk factors (pre-existing depressive illness, physical musculoskeletal symptoms, lack of visits from friends / relatives), unusual behaviour (tearing or removing clothes, eating faeces, screaming) and in dementia patients who have weight loss, altered appetite, or increased agitation and restlessness. For managing depression, reversible problems should be identified (e.g. anaemia, heart failure, painful arthritis, hypo- or hyper-thyroidism), voicing of feelings encouraged, activities and social stimulation encouraged, and antidepressants considered if biological symptoms (e.g. weight loss, poor fluid intake) are prominent.

5.3.4 Interdisciplinary, Multi-professional, Interagency Approach to Quality Long-term Care

In 2000, the Royal College of Physicians, the Royal College of Nursing, and the British Geriatrics Society formed a joint working party on a comprehensive interdisciplinary approach to quality care for care home residents.[21] Ten major recommendations were made together with the resources needed for implementation. These are shown as follows.[21]

- An agreed comprehensive assessment tool should be adopted for each resident that records diagnoses and disabilities to facilitate care planning, commissioning, and governance.
- An individualised care plan that addresses the assessed needs and wishes of the individual
- A population-based approach for the planning and provision of services
- The responsibility for identification and integration of healthcare support to the care home should lie with a specialist gerontology nurse.
- Geriatricians and old age psychiatrists, the two principal specialists relating to the health problems of a care home population, should be engaged in structured planning of care.

- General practitioners' role and responsibilities in relation to care homes should be defined; training and qualifications should be encouraged and recognised.
- The management of medication and the role of the pharmacist may be enhanced through an institutional approach.
- The organisation, application, and governance of all the professions allied to medicine (therapists, podiatrists, etc.) may be enhanced through integration with other professionals and through core institutional-based care rather than individual contracting.
- The concept of 'a teaching nursing home' should be developed.
- Research to inform further developments that aim to improve health and care outcomes.

The role of geriatricians in long-term care has been stated as below.[21]

- Complex assessments and development of disease management programmes
  - o The assessment and clinical formulation of people with multiple pathologies and disabilities
  - o The assessment of risks, benefits, and in particular clinical determination of and advocacy for opportunities and limits to treatment
  - o Liaison and communication, in particular with residents' and significant others' wishes
- Specialist opinion and advice to general practitioners about the management of care home residents
- Clinical governance, in particular engagement in standards setting and regulation, and clinical complaints arbitration
- Education and training
- Research and development

Old age psychiatrists, if adequately resourced, could address the needs of older people with mental health needs, in particular the following groups of residents in care homes[21]:

- those with dementia;
- those with psychosis presenting in late life;
- people with long-standing mental illness who have become aged;
- individuals with learning disabilities; and
- older people with personality disorders

In 2011, the British Geriatrics Society, in partnership with clinicians experienced in care home work, academic researchers, and the care homes sector published a report highlighting the need to build joint professional leadership from the health, social, and care home sectors, statutory regulators, and patient advocacy groups to find solution that would improve the quality of support to care homes.[22]

5.3.5 Barriers to Quality Long-term Care

The following may hinder high-quality long-term care:

- Practice of heroic medicine, in which death is viewed as an ultimate failure and aggressive interventions are performed to save life at all costs.
- Intense protection out of 'safety' and regulatory concerns, e.g. use of bed-side rails and physical restraints for fear of falls. Excessive concern with safety may lead to infringement of personal rights. 'Responsible risk-taking' should be regarded as normal, and residents should not be discouraged from undertaking certain activities solely on the grounds that there is an element of risk.
- A legal model of ethics that emphasises competency, consent, and confidentiality (in contrast to social ethics that values communication, clarification, and consensus building)
- Task-orientated staff: superficial, routine-orientated, and task-related staff-resident interactions constitute a barrier to open and meaningful communication between nurses and patients.[23]
- Medical-social divide: Fragmentation of service organisation and between health and social care is another obstacle to high-quality long-term care. Integration of specialist clinical assessments with those of social service care managers has resulted in reduced physical deterioration of elderly people and delayed care home admission.[24] Continuity of care and provision of resources for living will be much facilitated by integration of health and social care at organisational and professional levels.[25]
- Strict infection control measures: For long-term care residents, the hospital is functionally their home, and residents share common eating and living areas and participate in various activities. Too restrictive measures to decrease transmission of organisms or infection among residents may undesirably interfere with resident socialisation or rehabilitation. Thus, the psychosocial consequences of isolation measures must be carefully balanced against the infection control benefits.[26] It has been suggested that universal glove use may be preferable to contact isolation because it reduces social isolation for long-term care residents.[27]

5.4 Assessment in Long-term Care

5.4.1 Assessment can Guide Care at Different Points of Time[21]

5.4.1.1 Pre-admission: the Need for Care or Remedy

The transition to long-term institutional care

represents a major change in the lives of elderly people. To ensure the correct match between clinical needs and services, many countries have found value in standardised pre-admission assessments. Studies have demonstrated that a specialist clinical assessment prior to institutional placement led to benefits for elders and their carers, less contact with nursing homes and emergency services, and cost savings.[28,29] Pre-admission specialist clinical assessment identified previously unreported yet treatable and remediable clinical conditions and disabilities.[28,29] Treatment and rehabilitation rather than institutionalisation may produce a better outcome in some individuals and be cost-effective. Assessment of needs and specialist care requirements can help resource allocation and actual care provision to the individual awaiting infirmary care.

5.4.1.2 Admission: the Development of an Effective Care Plan

Admission assessment can help with formulation of a care plan individualised to assessed needs, and indicate issues that will require support from health and care services.

5.4.1.3 Review

Assessment should occur on a regular basis as indicated by the care plan developed on admission. Any significant changes in medical, physical, or mental function should trigger review. Review may:

- confirm the appropriateness of the existing care plan;
- identify opportunities to improve the health of an individual through treatment, avoiding iatrogenesis, and rehabilitation; and
- identify a need for care to become palliative in the presence of progressive clinical and functional deterioration.

5.4.2 Assessment Tool and Instruments

In Hong Kong, pre-admission assessment for infirmary applicants by CGATs is with a standardised Infirmary Core Assessment Form designed by local geriatricians specifically for frail elderly people. The assessment covers areas of mental status and cognition (abbreviated mental test), communication and comprehension, vision, ambulation (Modified Functional Ambulatory Category), basic self-care (Barthel-20), continence, skin, nutrition, psychosocial status, hospital utilisation, current medication, need for special care and procedures (tracheostomy, non-invasive ventilation, oxygen therapy, frequent suction, feeding tube, urinary catheter, peritoneal dialysis, stoma / wound care, carrier of antibiotic-resistant bacteria); and these together with clinical findings and diagnosis lead to formulation of an individualised problem list, action, and placement decision.

Internationally, the Minimum Data Set–Resident Assessment Instrument (MDS-RAI), later known as the Minimum Data Set–Nursing Home (MDS-NH), was used in the 1990s for the specific long-term care setting in care homes,[30] originally developed and mandated in the US. This evolved into the interRAI Long-Term Care Facilities Assessment System (interRAI LTCF) in the 2000s with assessment items specific to long-term care settings and core elements common to other settings <http://www.interrai.org/long-term-care-facilities.html>. The interRAI LTCF has been translated into multiple languages, including Hong Kong Chinese.

The areas covered by MDS 3.0 <https://downloads.cms.gov/files/MDS-30-RAI-Manual-V114-October-2016.pdf> include:

- identification information
- hearing, speech, and vision
- cognitive problems
- mood
- behaviour
- preferences for customary routine and activities
- functional status, functional abilities, and goals
- bladder and bowel
- active diagnoses
- health conditions
- swallowing and nutritional status
- oral and dental status
- skin conditions
- medications
- special treatments, procedures, and programmes
- restraints
- participation in assessment and goal setting

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CHAPTER 6

Community Geriatrics — Residential Care Home Medicine

Francis Chun-keung MOK, Alfred Si-yan AU, Bernard Ming-hei KONG

OBJECTIVE

To understand the medical and healthcare provided to institutionalised elderly persons in Hong Kong and recognise the increased vulnerability and dependency of this group of individuals.

KEY POINTS

- Institutionalised elderly persons constitute a special group with vulnerability and dependency.
- A comprehensive, multidisciplinary assessment is needed for an elderly person's placement decision.
- Different types of residential care home services are available in Hong Kong to meet the needs of elderly persons.
- Community Geriatric Assessment Service is the prototype of residential care home medicine in Hong Kong.

6.1 Introduction

Most elderly people would like to live in the community, if possible. However, because of various medical or psychosocial reasons, some live in institutions. According to Hong Kong census data in 2011, about 8.6% of those aged ≥65 years lived in an institutional setting and this increased to 24.9% for those aged ≥85 years.[1] The health service should facilitate alternative community support for those who could still live in the community. A comprehensive multidisciplinary assessment should be performed when an elderly person plans to move into residential care. A good interface between hospital services and community services is crucial for the proper placement decision. Those living in residential care homes for the elderly (RCHEs) have high vulnerability and dependency because of their old age and poor health or physical / mental disabilities. Communal living of these vulnerable persons in an enclosed environment also requires good infection outbreak precautions. The standard of care, both from medical and social perspectives, should be closely monitored and continuously upheld.

6.2 Community Institutional Care for Elderly Persons

Different countries have developed different systems of care for elderly people who have to move into institutions from their homes. A cross-national study has revealed wide variability in institutional rates and care provision in community institutionalised settings.[2] In this study, a nursing home is defined as an institution providing nursing care 24 hours a day, assistance with activities of daily living and mobility, psychosocial and personal care, paramedical care such as physiotherapy and occupational therapy, as well as room and board. A residential home for elderly people is defined as an institution providing living conditions adjusted to the needs of residents usually requiring no more nursing care than can be given by a visiting nurse.[2]

Different countries, however, have used different nomenclatures for community institutionalised settings for elderly people without adhering to the above definitions. Thus, the term 'nursing home' as used in the US may refer to skilled, unskilled, or specialised facilities. In the UK, 'care home' generally refers to a home registered for provision of personal and residential care for elderly people, but also includes homes providing nursing care. Guidance has been developed by professional geriatric and gerontological associations to promote quality care of frail, elderly people in nursing and residential care homes.[3,4]

6.2.1 Types of RCHEs in Hong Kong

The Social Welfare Department (SWD) administers a licensing system to regulate all RCHEs in Hong

Kong. The aim is to provide residential care and facilities for elderly persons aged ≥65 years who have caring needs. Persons aged between 60 and 64 years may apply if there is a proven need.[5] These RCHEs are subvented / contract homes, or self-financed / private homes.

Varying types of RCHEs are set up to meet the different care needs of elderly persons. From the lowest level of care to highest level of care, these include Hostel for the Elderly, Home for the Aged, Care and Attention Home, and Nursing Home. The first two categories have been being phased out since 2003. For elders who are chronically ill or disabled to a degree of total dependence beyond nursing home level, infirmary care has to be provided. There are hospital infirmaries run by the Hospital Authority (HA) and infirmary units set up in some subvented RCHEs. In addition, temporary accommodation, including Emergency Placement Service and Residential Respite Service, provides short-term accommodation for elderly persons with urgent or temporary care needs.

In 2013, the number of elderly persons aged ≥65 years in Hong Kong was one million (14% of total population). As at March 2014, some 61 200 elderly persons were residing in the 75 000 places provided by 748 RCHEs in Hong Kong.[6]

6.2.2 Assessment of Elderly Persons for RCHE Placement

Since 2000, the SWD has implemented the Standardized Care Need Assessment Mechanism for Elderly Services. Under the Mechanism, an internationally recognised assessment tool called the Minimum Data Set–Home Care (MDS-HC) was adopted to ascertain the care needs of elderly persons and match them with appropriate long-term care services, including both residential and community care. Accredited professionals including social workers, nurses, or therapists (occupational and physiotherapy) conduct the assessment for an individual elderly person upon their application for institutional care.[7] Elderly persons assessed to have care needs (i.e. impairment of moderate level and above) are matched with the appropriate services in accordance with the assessment results. The eligibility of an elderly person for residential care home services is also assessed.

The MDS-HC was designed to capture multidimensional information of an elderly person. Domains assessed include:

- cognitive patterns
- communication / hearing patterns
- vision patterns
- mood and behaviour patterns
- social functioning
- physical functioning including
  - o instrumental activities of daily living
  - o activities of daily living
  - o continence (bowel and bladder)
- disease diagnoses
- health conditions and preventive health measures (fall and lifestyle)
- nutrition and hydration status
- dental status
- skin conditions
- environmental assessment
- service utilisation (formal care, treatment programmes)
- medication use

6.2.3 Community Geriatric Assessment Service

Community Geriatric Assessment Service (CGAS) is the prototype of residential care home medicine in Hong Kong. In 1994, CGAS was set up by the HA as a community-based geriatric specialist–led service providing multidisciplinary care to enable assessment and management of elderly people living in RCHEs.

The objectives of CGAS at its inception were:

- to provide timely assessment and appropriate management of medical and health problems of elderly persons at risk;
- to improve the interfacing between medical and social services for elderly persons at district level;
- to develop and provide community-based rehabilitation programmes for elderly persons;
- to ensure that placement decisions / arrangements for elderly persons in need of residential care are appropriate and avoid inappropriate institutionalisation; and
- to promote the quality of care for elderly persons through training and education of caregivers and advise on the delivery of care in RCHEs.

Starting with eight teams to serve subvented RCHEs, the service was extended to the private RCHEs in 1997. In 2004, Clinical Management System access was available in RCHEs which unified the clinical and health information platform with other HA services. As at 31 March 2014, there were 13 Community Geriatric Assessment Teams (CGATs) providing service to 638 (89%) of the 715 RCHEs licensed and regulated by the SWD under the RCHE Ordinance in Hong Kong. The number of CGAS attendances in RCHEs has increased from around 52 500 in 1998 to around 744 700 in 2010.[7]

Through the multidisciplinary assessment, advice on the overall care programme and appropriate residential placement for geriatric patients based on their care needs were provided. A typical

CGAT comprises geriatricians, nurses, therapists (occupational and physiotherapy), medical social workers, and clerical support staff. These teams provide specialist geriatric service to RCHEs through regular visits and consultations, pre-admission assessment for elderly persons on the Central Infirmary Waiting List and multifaceted assessment for those entering RCHEs, coordination of community domiciliary services, and referrals to specialist outpatient departments and Geriatric Day Hospitals, according to an elderly person's need.[8] Continuity of care was established as elderly persons were both treated and followed up by the same team of geriatric service both in hospitals and after discharge. CGATs enhanced the interface between medical and social services for elderly persons in the community through outreach service to RCHEs and the provision of comprehensive multidisciplinary assessment and management services to elderly inmates. Rehabilitation programmes and training of staff in institutions were also important provisions of CGAS to RCHEs. Anticipatory care like high-risk screening (e.g. for fall, malnutrition, etc.) was also practised.

Reports showed that CGAS reduced the admission rate, unplanned readmission rate, accident and emergency (A&E) attendance rate, and the incidence of adverse events among RCHE inmates covered by the service. In addition, CGAS played an important role in the management of infection outbreaks. CGAS provided professional advice on infection control and triage in suspected cases during the 2003 severe acute respiratory syndrome (SARS) epidemic. Knowledge and support were also provided to the staff of the RCHEs during influenza and gastroenteritis outbreaks. During hospital admission surge periods of winter months, CGAS supported early discharge of patients (including those directly from A&E departments), increased frequency of visits to RCHEs, monitored infectious disease surveillance, and provided early medical intervention. CGAS built a good interface between the traditional hospital-based geriatric services and the welfare-focused community services for frail and disabled elderly persons living in RCHEs. The caring standards of the RCHEs were improved in parallel with the service development of CGAS throughout these years.

6.2.4 Important Domains in Caring for Elderly Persons in RCHEs

The following are important domains in the care of an elderly person in RCHEs:

- nutrition and body weight monitoring;
- swallowing difficulties and feeding skills;
- oral hygiene and dental care;
- tube feeding;
- urinary catheter care and continence care;
- pressure sore prevention and management;
- foot care;
- fall prevention and management;
- blood pressure monitoring and postural hypotension;
- hypothermia prevention and management;
- medication storage and administration;
- prevention of infection outbreak and transmission — scabies, influenza, gastroenteritis, hepatitis, myiasis, tuberculosis (TB), infections due to multidrug-resistant organisms (MDRO) such as methicillin-resistant *Staphylococcus aureus* (MRSA) and vancomycin-resistant enterococci (VRE);
- environmental hygiene and cleansing;
- cognitive impairment and dementia, behavioural problems;
- correct technique of handling disabled persons;
- exercise and rehabilitation maintenance; and
- end-of-life care

6.2.5 Common Medical Problems of RCHE Inmates

According to the 2009 census, hypertension was the most common chronic illness in institutionalised elderly persons (59.1%), with stroke and Alzheimer's disease / dementia being the second and third common illnesses (32.2% and 31.6%, respectively).[9] Other common illnesses included diabetes mellitus (21.7%) and arthritis (19.9%). Other chronic conditions affecting institutionalised elderly persons include malnutrition and polypharmacy. Incontinence was a distressing condition affecting many living in RCHEs. Local census data of 2010 revealed that around 54% reported urinary incontinence and 52% reported faecal incontinence in the week prior to enumeration among institutionalised elderly persons.[10]

These medical conditions and geriatric syndromes are discussed in relevant chapters and sections of this book.

6.3 Infection Control in RCHEs

RCHEs are more vulnerable to outbreaks of communicable diseases, with inmates having a high risk of hospitalisation and death related to infection.[11,12] They are usually frail, dependent, and have multiple comorbidities such as diabetes mellitus, being bed bound, malnutrition, cancer, etc. They are more vulnerable to infections as the body resistance is lowered due to immunologic senescence, malnutrition, and medications such as immunosuppressants. The environment of many RCHEs in Hong Kong is rather congested with shared facilities (e.g. toilets, bathing facilities), and hygiene practices may be substandard. These also favour the transmission of infection. A recent study in Hong Kong showed that transmission rate of MRSA in RCHEs was high and was related to congested living

environment.[13] The source of infection could be from elderly inmates, staff, or visitors. The movement of inmates between hospitals and RCHEs also increases the risk of infection. Early discharge of patients from hospitals to RCHEs because of inpatient bed congestion increases the risk of infection transmission.

Respiratory tract infections, urinary tract infections, skin infections, and gastroenteritis are common infections in RCHEs. These inmates having invasive medical devices in situ such as urinary catheter, nasogastric tube, peritoneal dialysis catheter, etc. make them more prone to infection.

One should remember that frail, elderly patients may not present with typical signs of infection including fever.[14] Sometimes very subtle signs like unexplained tachycardia, poor appetite, incontinence, instability, confusion, or just malaise and lethargy should alert one to search for ongoing infection in an elderly person.

6.3.1 Strategies of Infection Control

6.3.1.1 Prevention

General health of elderly inmates should be up kept. This may be achieved with good nutrition, regular exercise, smoking cessation, and good control of diseases.

Maintaining good personal, environmental, and food hygiene can stop the transmission chain. Proper hand hygiene, both for inmates and staff, is particularly emphasised by the HA and the World Health Organization (WHO) for infection control. With bed-bound inmates, staff is usually the transmission vector for infections. It has been shown that good promotion programme and education are effective in improving hand hygiene among staff of RCHEs.[15] Skin care and good oral hygiene of inmates are important in preventing infections like scabies and myiasis. Good environment with good ventilation, regular cleansing of soiled areas especially toilets, proper spacing between beds, repellents for flies, mosquitos, rodents, etc. are also important for infection control.

Vaccination should be provided to those who have the requisite indications. The Department of Health freely offers two kinds of vaccination to RCHE inmates. Seasonal influenza vaccine is administered annually according to the WHO recommendation. RCHE staff is also eligible for the vaccination. Free pneumococcal vaccine is also offered for eligible RCHE inmates aged ≥65 years who have not received the vaccine previously (see Chapter 32.5.1). Both vaccination programmes have solid proof of efficacy in reducing the morbidity, hospital admission, and mortality.[16] These would help to reduce inappropriate antibiotic usage in RCHEs. It has been shown that staff vaccination for influenza could also benefit the RCHE inmates.[17]

6.3.1.2 Surveillance of Outbreaks

General condition of every RCHE inmate should be monitored daily with proper record keeping. Daily temperature check is considered a simple but useful way to detect early disease in inmates. Clustering of febrile illness or other disease symptoms (e.g. upper respiratory tract infection or diarrhoea) warrants special attention. Close liaison with CGAT, early medical attention and reporting to Centre for Health Protection (CHP) and Licensing Office of RCHEs are all important steps for diagnosing an outbreak and enabling implementation of early control measures including early medical treatment, cohorting of cases, prophylaxis for nearby inmates, etc.

Some outbreaks are seasonal, e.g. influenza outbreak during winter times (January to March) and summer times (July and August). One should be more alert for outbreaks during these seasonal times. At least one trained staff of every RCHE should be appointed as Infection Control Officer (ICO) to pay special attention to monitor the health state of the inmates and staff regularly, and maintain alert for any possible outbreak. ICO posting in RCHE was introduced after the SARS outbreak in 2003 in Hong Kong. Since then, protocols and action plans were set up for suspected infectious disease outbreaks in RCHEs which included: (1) implementing fever registry and sentinel surveillance in RCHE, (2) visiting medical officer programmes in RCHE to increase medical support, (3) setting up and assessing infection control standards in RCHE, and (4) advising RCHE on screening, cohorting, and isolation of infected inmates.

6.3.1.3 Common Infections

The common infections in RCHE settings and their characteristics are summarised in Table 6.1.[18] For more details, clinical microbiological literature should be consulted.

Pulmonary TB is worthy of special mentioning in our local setting. It is an air-borne disease. The notification rate is higher for the elderly population in Hong Kong.[19] Both local and world data suggest that the notification rate is even higher in RCHE population.[20] Tuberculin skin test is recommended for admission screening of old age home residents in many countries. This is not applicable in Hong Kong because of the high incidence of past infection in the elderly population, rendering its specificity too low for the purpose. The practice of chest X-ray screening on admission to RCHE might help to detect asymptomatic patients but this is neither sensitive nor specific. It has been shown that TB could spread

Table 6.1 Common infections in residential care homes for the elderly (RCHE) settings[18]

Infection	Description
Urinary tract infections	Among the most common bacterial infections in RCHE. Asymptomatic bacteriuria and pyuria are common. Infection is more common in inmates with indwelling urinary catheter. Antibiotic treatment should only be given when the patient is symptomatic or exhibits functional decline.
Respiratory tract infections	Respiratory tract infection outbreaks in RCHE are common and can occur year around. Most common pathogens are influenza, parainfluenza, respiratory syncytial viruses, adenoviruses or rhinoviruses. For influenza outbreaks, early identification and oseltamivir usage (for treatment and prophylaxis) and enhancement of infection control measures would help.
	Streptococcus pneumoniae is an important cause of invasive pneumococcal diseases. Both influenza and pneumococcal vaccination could prevent outbreaks. Legionnaire's disease could be the cause of outbreaks in RCHE associated with contaminated water systems.
Gastrointestinal infections	Diarrheal outbreaks are usually caused by norovirus or rotavirus. Both inmates and staff could be affected through contact transmission. The attack rates could be high. Elderly patients may have severe dehydration and even death.
	Occasional foodborne outbreaks could be due to *Salmonella* or *Clostridium perfringens*.
	Clostridium difficile infection after antibiotic treatment should also be considered for unexplained diarrhoeal disease.
Skin infections	Usually fungal or bacterial and are self-limiting. However, in the presence of malnutrition, pressure sores or wounds and incontinence, severe infections could occur. Usual aetiological bacteria include *Staphylococcus pyogenes* and *Streptococcus*. Deep ulcers (stages 3 and 4) generally contain anaerobic and aerobic bacteria. Associated osteomyelitis can be diagnosed accurately only by pathological examination.
	Scabies infections can cause outbreaks through personal contacts and mite-contaminated vehicles, affecting both inmates and staff. Treatment by permethrin or benzyl benzoate should be effective. Disinfection of bedding, clothing, and the environment simultaneously is important to treat the disease and stop the transmission.
Pulmonary tuberculosis	Routine chest X-ray screening on RCHE admission. The presence of old tuberculosis scar and atypical presentation in elderly persons lead to difficulty in diagnosis. High clinical suspicion is needed. Treatment requires specialist care.
Hepatitis	Infection with hepatitis B virus and hepatitis C virus occurs in inmates. Staff who handle blood / body fluids or who are at risk of needlestick injury should receive the hepatitis B vaccine.

in a RCHE environment without notice.[21] Thus, clinical alertness is most important for early detection of active cases. The management of TB in an elderly person requires prolonged treatment which is associated with high incidence of medication-related adverse effects.[22] Management of tuberculosis in frail elderly patients is complex and requires specialist care and sometimes even inpatient management.[23]

Inmates with air-borne infection (pulmonary TB, disseminated herpes zoster) should receive inpatient treatment in a hospital with standard isolation facilities until the patient is not infectious.

6.3.1.4 Issues on MDRO

The ample usage of antibiotics, sometimes inappropriately, has led to the emergence of many MDRO in the recent years. The common ones are MRSA, VRE, carbapenem-resistant Enterobacteriaceae, and methicillin-resistant *Acinetobacter*. Risk factors for the emergence of MDRO included exposure to antibiotics, poor hand washing facilities, and poor staff standard.[24] All these infections are transmitted through personal contacts. As many of these elderly patients are just carriers of these MDRO, they do not develop clinical disease related to these organisms. However, it takes a long time (weeks to months, or up to years) to have the carrier state conversion. As such, many of these MDRO carriers have to return to their homes after their acute problems have settled. It is important for the RCHE staff to take stringent contact precaution measures to prevent these MDRO from spreading to other people (both staff and inmates) in the RCHE. Good personal hygiene, especially hand hygiene, is of paramount importance. CHP offers training to RCHE staff when in need — the control measures (isolation and cohorting), advice to visitors, environmental cleansing and disinfection, and proper personal protection measures. Besides attending to the isolation need, the inmate's physical and psychological aspects should also be taken care of.[25]

Geriatricians should take a leading role in helping RCHE staff to monitor the process of infectious disease control. The CHP has issued guidelines on prevention of communicable diseases in RCHEs[26] which are useful for RCHE staff.

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CHAPTER 7

Community Geriatrics — Transitional Care and Discharge Support Programmes

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OBJECTIVE

To learn about transitional care in western countries and in Hong Kong, as well as the importance of discharge planning and post-discharge support in the outcome for at-risk elders admitted to hospital.

KEY POINTS

- Hospitalisation of elderly people often results in adverse outcomes.
- Some unplanned hospital readmissions are avoidable.
- Transitional care in the form of discharge planning and post-discharge support for at-risk elders can improve outcomes and reduce hospital readmissions.
- Discharge planning as a process begins at hospital admission and involves ongoing assessment throughout hospital stay focusing on the patient's post-discharge needs.
- Medication safety is of pivotal importance in every discharge planning procedure.
- A variety of discharge support programmes has been developed over the past 10 years to bridge the gaps in care transition systems as well as to meet the needs of patients and caregivers.

7.1 Introduction

Hospitalisation of elderly people often results in adverse outcomes.[1-3] Up to 30% of older persons have a decline in functional status after a hospital stay for acute illness.[4,5] Moreover, older patients continue to experience adverse events after hospital discharge.[6] Up to one-quarter of hospital discharges in the US had complicated post-hospital care patterns[7] where the patient had to be transferred from a lower- to higher-intensity care environment within 30 days of initial hospital discharge. Unfortunately, a shortage of acute hospital beds creates a demand for shortened inpatient stay and many older patients will be consequently discharged before they can achieve a reasonable recovery from the acute illness. In Hong Kong, where a structured primary care system is unavailable, up to 16-20% of patients discharged from a medical specialty have an unplanned readmission within 28 days. It had been estimated that approximately 40% of these unplanned readmissions are due to clinician, patient, system, and social factors that could be avoided (Table 7.1).[8] Hence, care transition after an acute hospitalisation is a vulnerable time for the patient. The development of safe care transition has grown in importance with the recognition of a discontinuity of care related to multiple clinicians and divided responsibility.

Transitional care in a broad sense is focused on care that is transferred between different healthcare settings. In the UK, where the service is termed intermediate care, and Australia, the focus is on out-of-hospital support. Table 7.2 shows the different definitions in different countries.[9-12]

7.2 Evolution of Transitional Care in the US

In the US, there are multiple healthcare settings that hand over patients to and admit patients from a structured primary care system. Barriers to smooth care transition in earlier trials have been identified at the delivery, clinical, and patient levels (Table 7.3).[13] The main components of effective care transition rely on effective transfer of information and communication between different stakeholders and the patient and their family as well as medication reconciliation (Table 7.4).[13]

Because of an awareness of the huge costs related to hospital readmissions, there has been increasing demand to implement measures that can reduce their occurrence. The Medicare's Hospital Readmission Reductions Program, resulting from the 2010 Affordable Care Act signed by President Obama, proposes penalties in the form of reducing Medicare

payment for those hospitals with excessively high readmission rates.[14] The Centers for Medicare and Medicaid Services has started to fund community care transitional programmes. This, in contrast to the era with barriers listed in Table 7.3,[13] provided a strong financial incentive to implement transitional care. There has since been a dramatic increase in the provision of programmes for transitional care, research in the area, and provision of post-discharge support.[15]

7.3 The Ideal Transitional Care

Ten domains have been proposed as the key components of an ideal transition in care of a patient from hospital to community.[16] The 10 domains that take place in descending order of their proximity from hospital to community are[16]:

- discharge planning
- information communication
- information timeliness and organisation
- medication safety
- patient education
- social and community supports
- advance care planning
- coordinating care among team members
- monitoring and managing symptoms post-discharge
- outpatient follow-up

Table 7.1 Reasons for avoidable readmission[8]

Factor	Reason
Clinician (42.3%)	• Low threshold for admission (10.6%) • Premature discharge (9.3%) • Drug-related adverse event (7.7%)
Patient (41.9%)	• Relapse or progress of previous complaint (28.9%) • Compliance problems (8.1%)
System (14.6%)	• Inadequate discharge planning (5.7%) • Inadequate palliative care / terminal care (4.1%)
Social (1.2%)	• Carer system, lack of support and community services (0.8%)

Discharge planning is the essential part of any transitional care. This will be discussed in detail in Section 7.4. Complete communication of information and availability, timeliness, clarity, and organisation of that information refer to timely accessibility of a discharge summary and other means of information transfer from hospital to post-discharge care. As adverse drug events are the most common type of adverse event (accounting for 66% of incidents),[6] reducing medication errors and medication safety are of pivotal importance. This includes taking an

Table 7.2 Different definitions of transitional care programme in the US, UK, and Australia[9-12]

US

American Geriatrics Society 2003[9]

Transitional care has been defined as a set of actions designed to ensure the coordination and continuity of healthcare as patients transfer between different locations or different levels of care within the same location. Representative locations include (but are not limited to) hospitals, subacute and postacute nursing facilities, the patient's home, primary and specialty care offices, and assisted living and long-term care facilities.

Naylor et al, 2011[10]

A broad range of time limited services designed to ensure healthcare continuity, avoid preventable poor outcomes among at-risk populations, and promote the safe and timely transfer of patients from one level of care to another or from one type of setting to another.

UK

Department of Health[11]

Intermediate care is defined as:

- services targeted at people who would otherwise face unnecessarily prolonged hospital stays or inappropriate admission to acute inpatient care, long-term residential care, or continuing National Health Services inpatient care
- services provided on the basis of a comprehensive assessment, resulting in a structured individual care plan that involves active therapy, treatment, and opportunity for recovery
- services which have a planned outcome of maximising independence and typically enabling patients / users to resume living at home
- services which are time-limited, normally no longer than 6 weeks, and frequently as little as 1-2 weeks or less
- services which involve cross-professional working, with a single assessment framework, single professional records, and shared protocols

Australia

Department of Health and Aging[12]

Transition care provides short-term care that seeks to optimise the functioning and independence of older people after a hospital stay. Transition care is goal-oriented, time-limited, and therapy-focused. It provides older people with a package of services that includes low-intensity therapy such as physiotherapy and occupational therapy, as well as social work, nursing support, or personal care. It seeks to enable older people to return home after a hospital stay rather than enter residential care prematurely.

The programme facilitates a continuum of care for older people who have completed their hospital episode, including acute and subacute care (e.g. rehabilitation, geriatric evaluation and management), and who need more time and support to make a decision on their long-term aged care options.

Table 7.3 Barriers to smooth care transition in the US before 2010[13] (reproduced with permission)

System level
- Organisation of the healthcare delivery system into distinct, independent institutions resulting in lack of formal relationships between care settings which create barrier to cross-site communication and collaboration
- Lack of financial incentives promoting transitional care
- Different financing and contractual relationships facilities with various pharmaceuticals companies: each facility has incentives to prescribe or substitute medications according to its own medication formulary. This constant changing of medications creates confusion for the patient, caregiver, and receiving clinicians
- Poor communication between inpatient and outpatient clinicians
- Lack of information systems designed to facilitate the timely transfer of essential information across settings
- Lack of a structured approach to relay information during admission and discharge and absence of reliable systems to ensure the transmission of such information
- Transitions of care at admission and discharge are fraught with fragmented and incomplete information transfer
  - Inefficient and unstructured systems for communication of important clinical data, such as medication changes or tests that are pending
  - Lack of a longitudinal patient relationship and standardised follow-up procedures
  - The infrequency of hospitalist–primary care physician communication
  - Omission of details of patients' hospital course

Clinician level
- Growing reliance on designated institution-based physicians (i.e. "hospitalists" and "SNFists") and productivity pressures: difficult for primary care physicians to follow their patients when they require hospitalisation or short-term rehabilitation
- Nursing staff shortages forcing an increasing number of acute hospitals to divert patients to other facilities where a completely new set of clinicians, who often do not have timely access to the patients' prior medical records, manages them
- Care managers and social workers predominantly assigned to specific care settings: older patients with multiple problems may be assigned to more than one care manager, including disease-specific and general care managers

Patient level
- Little advocacy or outcry from patients for improving transitional care until they or a family member is confronted with the problem firsthand
- Older patients and their caregivers are not well prepared or equipped to optimise the care they will receive in the next setting
  - Unrealistic expectations
  - Disempowered to express their preferences or provide input for their care plan
- Medication changes during hospitalisations
- Inadequate understanding of diagnoses, medications, and follow-up needs
- Discharging patients with incomplete diagnostic work-up

Table 7.4 Components of effective care transition 2003[13] (reproduced with permission)

Before any decision to transfer, the potential for harm from imposing an additional transfer to a new setting needs to be weighed against the potential for benefit. The appropriateness of the match between the proposed care setting and the patient's medical, nursing, and functional needs should determine the decision to transfer

In general, effective care transitions include
- Communication between the sending and receiving clinicians regarding:
  - a common plan of care;
  - a summary of care provided by the sending institution;
  - the patient's goals and preferences (including advance directives);
  - an updated list of problems, baseline physical and cognitive functional status, medications, and allergies; and
  - contact information for the patient's caregiver(s) and primary care practitioner
- Preparation of the patient and caregiver for what to expect at the next site of care
- Reconciliation of the patient's medication prescribed before the initial transfer with the current regimen
- A follow-up plan for how outstanding tests and follow-up appointments will be completed
- An explicit discussion with the patient and caregiver regarding warning symptoms or signs to monitor that may indicate that the condition has worsened and the name and phone number of who to contact if this occurs

accurate medication history, reconciling changes throughout the hospitalisation, and communicating the reconciled medication regimen to patients and providers across transitions of care. Educating patients and promoting self-management involves teaching patients and their caregivers about the main hospital diagnoses and instructions for self-care, including medication changes, appointments, and who to contact if there is a problem. Enlisting the help of social and community services is of particular importance when a patient experiences functional decline after hospitalisation. This is the rationale for the development of community-based care-transition programmes. Discharge support programmes will be discussed in Section 7.5. Advance care planning involves establishing goals of care and healthcare proxies, as well as engaging with palliative care or hospice services if appropriate. Care transition across different healthcare settings is bound to be discrete and fragmented; therefore a patient-centred coordinated care among team members is required to synchronise care across settings and providers.

Monitoring and managing symptoms after discharge is crucial to prevent avoidable hospital readmission. Looking out for new or worsening symptoms; medication side-effects, discrepancies, or non-adherence; and other self-management challenges will allow problems to be detected and addressed early, before they result in unplanned healthcare utilisation. Finally, optimal outpatient follow-up with appropriate post-discharge providers is crucial for an ideal transition. These appointments need to be prompt and with providers who have a longitudinal relationship to the patient.

7.4 Discharge Planning

Discharge planning is the pivotal starting point for effective transitional care. It has been defined as the process of activities that involves the patient and a team of individuals from various disciplines working together to facilitate the transition of that patient from one environment to another. It includes a systematic process of (1) assessing the assets and limitations of the patient during hospitalisation, (2) planning for continuity in his / her healthcare upon discharge from the hospital, and (3) coordinating the required individual, family, hospital, and community resources to implement the discharge plan.[17] From the healthcare system perspective, the development of an effective discharge planning system is crucial and can achieve multifaceted contributions (Table 7.5).[8] It is recommended that every patient should have a discharge plan from the start of their hospital admission.[18]

There are five components of the discharge planning system:
(1) screening and assessment
(2) discharge planning process
(3) coordination of discharge
(4) implementation of discharge
(5) evaluation of post-discharge follow-up

7.4.1 Screening and Assessment in Discharge Planning

It has been estimated that at least 80% of patients have a straightforward hospital stay and thus a 'simple discharge' from hospital. The remaining 20% have more complex needs and require a multidisciplinary team to coordinate the services and design a care plan

Table 7.5 The significance of an effective discharge planning system in healthcare delivery[8]

An effective discharge planning system is a critical component of a delivery system for healthcare which will ensure: (1) quality of patient care, improving patient outcomes, and enhancing patient satisfaction; (2) avoidance of delay in discharge, minimising inappropriate length of stay and unnecessary hospitalisation; (3) safeguard against premature discharge and reduce the prevalence of avoidable readmissions; and (4) utilisation of resources to maximise organisational effectiveness and optimise clinical outcomes

Table 7.6 Simple discharge versus complex discharge[18]

Simple discharge	At least 80% of patients are simple discharges, who: • will usually be discharged to their own home or place of residence • have simple ongoing care needs that do not require complex planning and delivery • are identified on assessment with length of stay predicted • no longer require acute care • can be discharged directly from accident and emergency department, ward areas or assessment units
Complex discharge	The remaining 20% patients belong to complex discharges, who/whose: • will be discharged home or to a carer's home, or to intermediate care, or to a nursing or residential care home • have complex ongoing health and social care needs that require detailed assessment, planning, and delivery by the multi-professional team and multi-agency working • length of stay in hospital is more difficult to predict

Table 7.7 Risk factors for adverse health outcome in older patients after discharge home[19]

• Length of hospital stay • Prior hospital admission • Advance age • Depression • Cognitive impairment • Malnutrition and obesity • Functional status: premorbid functional dependence, poor preadmission functioning in instrumental activities of daily living, dependence with activity of daily living, and reduced mobility status • Comorbidities • Multiple medications • Lack of social support: living alone, caregiver depression, caregiver stress, caregiver difficulty, and unrealistic caregiver • Poor socioeconomic environment: receipt of social assistance, financial and insurance concerns, and suboptimal living conditions in the home after discharge

with all the parties concerned. The hospital discharge is then classified as a 'complex discharge'.[18] The characteristics of both types of patients are listed in Table 7.6.[18]

The majority of patients are expected to have a smooth course of hospitalisation and uncomplicated post-discharge events. It is therefore necessary to be able to identify at-risk patients who are likely to have adverse outcomes after hospital discharge for targeted multidisciplinary assessment and intervention. Risk factors for adverse outcome (Table 7.7),[19] functional decline[20] as well as hospital readmission[8] have been identified. It is important to note that although these factors can be separated into discrete categories, many are interrelated and some factors intuitively can be seen to enhance the degree of risk associated with other factors. For example, cognitive instability can interfere with a patient's management of their medication. Various screening tools have been developed but none has been identified as the gold standard for general use.[21,22] It seems that individual healthcare settings will have to develop their own screening instrument specific to their local setting.

In Hong Kong, the Hospital Authority (HA) has developed a validated prediction model named "Hospital Admission Risk Reduction Programme for the Elderly" (HARRPE) to identify older people at high risk of readmission within 28 days.[23,24] The HARRPE score comprises 14 predictors that are categorised into patient sociodemographics, prior utilisation of accident and emergency department and medical ward admission in the past 1 year, comorbidity, and current index admission. The higher the HARRPE score (range, 0-1), the higher the readmission risk (e.g. 0.2 = 20% chance of readmission). This score has been widely used as a screening tool in various post-discharge support models in Hong Kong since 2008.

All at-risk patients identified by the initial screening should undergo a timely standardised comprehensive geriatric assessment shortly after admission. The principles and operation of a comprehensive geriatric assessment have been described in detail in Chapter 2.

7.4.2 Discharge Planning Process

The discharge planning process involves ongoing assessment throughout the hospital stay, focusing on the patient's post-discharge needs. Early involvement of a multidisciplinary team is mandatory. This whole system has the patient at the centre of service provision and responds to their needs. Effective multidisciplinary teamwork should be facilitated by regular team meetings to discuss the patient's progress and the current obstacles to discharge. The engagement and active participation of patients and their carers as equal partners is essential to the delivery of care and in the planning of successful discharge. The joint decision about the discharge plan should then be documented clearly in the hospital record to facilitate communication between different parties. It should be linked to an action plan and goals and should include the triggers for referral on other disciplines or agencies. As there is a high probability that the patient's condition will change during their hospital stay, the plan will be subject to continuous review and modification.

7.4.3 Coordination of Discharge

From the patient's perspective, the journey of hospitalisation is unpredictable, mysterious, and out of their control. The patient usually will not have the necessary vocabulary or skills to speak with healthcare workers. Communication with healthcare workers is further limited in a busy ward setting. At the point of discharge, patients will often be unable to obtain adequate discharge information and post-discharge care. This was evident from the results of a hospital-based patient experience and satisfaction survey carried out by the HA. Comparatively low scores were obtained in the following five areas: opportunity for a family or someone else close to the patient to talk to a doctor; healthcare workers discuss / comfort you about your condition; provision of information on medication side-effects; provision of information on danger signals to watch for after discharge; and provision of information needed in patients' care and recovery.[25] In order to guide our patients on this journey, it is important for the development and implementation of a discharge plan to be supervised by appropriately qualified personnel, who understand the patient's condition, are eager to explore their needs and concerns, and know how to communicate with them effectively.

Medication management is of utmost importance. It is vital the patient takes the correct medications at the correct dose at the correct time after discharge. Effort should be made to assess the patient's ability to take medications as instructed. If the patient is unable to do so, then it is important to ensure that there is reliable supervision at home, sometimes with the aid of community resources.

Provision of equipment is another area where healthcare workers should be involved. Mobility aids, appliances, and any other equipment such as tube feeding appliances or catheter care devices should be available for the patient before they leave hospital.

7.4.4 Implementation of Discharge

Upon hospital discharge, in order to ensure that the patient has adequate information, the hospital must

transfer the comprehensive discharge summary and medical information to the appropriate facilities or outpatient services that will provide follow-up or ancillary care. A copy of the discharge summary should also be available to the patient. It should state the tentative diagnosis, procedures, or operations performed during hospitalisation, an up-to-date list of medication (including drug allergy and adverse drug reaction) and a follow-up appointment. A call-back telephone number in case of enquiry is also required. If the patient requires ongoing nursing care, a nursing discharge summary, written in the patient's usual language, should be provided.

From the perspective of the ward, a discharge checklist should be made available. This checklist for transition of elderly patients should include the following items: home consideration, provision of community services, carers, medications, equipment, and transport.

7.4.5 Evaluation of Post-discharge Follow-up

Ongoing communication and coordination between hospitals and community service providers is required after a patient is discharged from hospital. It is important to provide an informative and comprehensive discharge summary in time to facilitate the evaluation of post-discharge follow-up. Telephone follow-up is recommended for high-risk patients after discharge, and the first contact should be within 24 hours, an important transition period between hospital and home. For the patient with complex needs, a case management approach should be adopted in which a liaison nurse coordinates and communicates with targeted clients and other disciplines. After developing a good rapport with the client, the nurse serves as a bridge between the client and other disciplines, facilitating timely referral of the client to community nurses for home visits or a multidisciplinary team for assessment and intervention if necessary; and communicates with doctors for ad-hoc medical support if required. He or she also reminds the client about follow-up appointments and is a source of health education and promotion. Every involved discipline should be authorised to access the consultation notes of other disciplines through the Clinical Management System so that the clinical information is shared with better coordination and achievement of the common goal. A close collaboration with the community and social services is vital to ensure continuity of patient healthcare and social support after discharge.

7.5 Discharge Support Programmes

Discharge planning should be coupled with post-discharge support for transition care interventions to be effective. Risk of readmission to hospital is significantly reduced by interventions provided both in the hospital and in the patient's home,[26] i.e. across the hospital-community interface. The majority of programmes are disease-specific and target patients with moderate and severe ill-health. There are mainly four types of intervention: (1) home-based, (2) intensive education / counselling, (3) multidisciplinary care approaches, and (4) telephone follow-up.[27] The majority of interventions involve a combination of these four approaches. Clinical efficacy has been demonstrated according to systematic review of discharge support interventions.[28,29]

Disease-specific post-discharge support interventions are suitable for complex cases when the indexed diseases are identifiable. Common examples include chronic obstructive pulmonary disease, chronic heart failure, chronic renal failure, malignancy, and stroke. Favourable outcomes reported in early supported discharge for stroke patients include reductions in length of hospital stay and deaths, institutionalisation and dependency, improvement in extended activities of daily living scores, and satisfaction with services. The greatest benefits were seen in stroke patients with mild-to-moderate disability.[30] Non-disease specific, but tailored, post-discharge support intervention for complex cases is suitable when diseases are non-identifiable in high-risk geriatric patients.

In the UK, a more sophisticated concept is the hospital-at-home (HaH), a service that provides active treatment by healthcare professions in the patient's home for a condition that otherwise would require acute hospital inpatient care.[31] The service offers a mixture of supportive care provided by healthcare assistants (sometimes for 24 hours, but more usually 8 am-8 pm) and rehabilitation care.[32] HaH services have been reviewed most extensively. It has been found that admission avoidance HaH care can achieve outcomes comparable with inpatient hospital care.[33]

7.6 Transitional Care in Hong Kong

Transitional care is an important phase of care for hospitalised elderly patients. Inappropriate support results in frequent return of frail older adults to hospital shortly after discharge. Although the population of Hong Kong is rapidly ageing, transitional services have been relatively underdeveloped. HA Community Geriatric Assessment Teams were established in 1994.[34] The teams provide timely on-site assessment and specialised geriatric care to subvented or private nursing home residents. They enable efficient transition between hospitals and welfare institutions.

The Social Welfare Department and non-governmental organisations have also provided social care services

to support discharged patients in the community. However, increasing population demand, long waiting time for social support services, and social services not matching patient needs are common shortfalls of the service.

In 2008, the Labour and Welfare Bureau of the Hong Kong SAR Government collaborated with the HA to pilot an Integrated Discharge Support Programme (IDSP) for frail elderly patients. The programme aimed to enhance the support to frail home-dwelling elderly people discharged from hospital and their caregivers, reduce unplanned hospital readmissions, and facilitate their stay in the community.[35] The programme was piloted in three hospitals: United Christian Hospital, Princess Margaret Hospital, and Tuen Mun Hospital.

IDSP adopted a geriatrician-led multidisciplinary approach to enhance discharge planning as well as post-discharge support. The Discharge Planning Team was set up to perform a multidimensional assessment of high-risk frail elderly patients admitted to hospital and to formulate discharge care plans. The target groups included patients with high rehabilitation needs (e.g. patients with acute stroke), high social needs (e.g. patients with poor social support), and patients with high medical risk as identified by the HARRPE score.[24] Patients recruited to the IDSP had a case manager assigned for provision / coordination of ambulatory and community and rehabilitative services. A Home Support Team operated by a non-governmental organisation provided transitional home care services as well as caregiver training and support. If needed, continuation of rehabilitation in a geriatric day hospital or fast-track medical review could be offered. Regular case conferences were held to monitor the progress of individual frail elders recruited to the IDSP.

After 3 years of pilot, the IDSP was found to effectively improve frail elders' functional outcomes, improving their health-related quality of life, reducing caregiver stress, as well as reducing attendance at the emergency department and unplanned readmissions to medical wards.[35] In 2011, the programme was extended to a territory-wide service. The new service, renamed Integrated Care Model, adopts a case management approach and includes transitional residential care for short-term residence in designated care homes prior to settlement back home. Clinical efficacy of this new model has been demonstrated.[36]

The HA Patient Support Call Centre (PSCC) was established in 2009.[37] The call centre's nursing staff proactively contact high-risk elderly patients as identified by their HARRPE score on discharge from hospital and provide advice on disease and medication management. Supporting teams comprising geriatricians and nurses are formed in each cluster to provide on-site support to PSCC. The service has also demonstrated effectiveness in reducing attendance at emergency departments and emergency hospital readmissions.

In 2012, the virtual ward service was also piloted. The pilot delivered HaH services by community nurses and geriatricians to frail home-dwelling elderly people discharged from hospital with high HARRPE score and major functional disabilities. Early results are encouraging.[38]

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CHAPTER 8

Geriatric Day Hospital

James Ka-hay LUK

OBJECTIVE

To understand the history, benefits, function, operation, outcome measurements, effectiveness, and evidence base of a geriatric day hospital (GDH), with reference to Hong Kong.

KEY POINTS

- The first GDH was opened in Hong Kong in 1975. There are now 19 GDHs in Hong Kong.
- The benefits of GDHs include reduced reliance on hospitalisation, making maximum use of multidisciplinary expertise and equipment, promoting independence and integration into the community, engaging home carers in rehabilitation, and providing a one-stop unit for comprehensive geriatric assessment.
- The function of a GDH includes rehabilitation, maintenance, assessment, medical management, nursing care, and medico-social care.
- GDHs are usually staffed by a geriatrician-led multidisciplinary team.
- The availability of adequate transport is crucial to bring patients with varying degrees of mobility problems to the GDH.
- Patients can be referred from the hospital or the community.
- The New Patient Index, Corrected New Patient Index, and New Referral Rate can be used to assess GDH outcomes.
- Despite controversy between overseas studies of GDH effectiveness, local studies support their role as an integral part of the geriatric service in Hong Kong.

8.1 History of Geriatric Day Hospital

The first geriatric day hospital (GDH) was opened in the UK in 1958.[1] Their existence in the UK expanded rapidly in the 1960s and GDHs now operate in a similar form in Australia, Canada, New Zealand, the US, and some European countries. They are an important adjunct to inpatient geriatric care and accepted as an integral part of geriatric services in these countries. In Hong Kong, the first GDH became operational at Princess Margaret Hospital in 1975.[2] As at January 2017, there are 19 GDHs in Hong Kong providing 919 places and serving different regions of the territory (Table 8.1).

8.2 Benefits of GDHs

Hospital admission for elderly patients can be disabling and shorter hospital stays can leave insufficient time for their functional recovery from a medical illness. For suitable elderly patients, GDHs provide further rehabilitation following discharge from hospital. They allow patients to be treated while remaining in a familiar environment, promoting independence and integration into the community, and engaging home carers in rehabilitation. In addition, GDHs allow simultaneous intervention in large numbers of patients in the same location, making effective use of multidisciplinary professional staff and equipment. By providing a place to continue treatment, length of hospital stay can be shortened and hospital discharge facilitated. GDHs also provide a one-stop unit for comprehensive geriatric assessment and management of complex elderly patients that is difficult, if not impossible, to arrange in the usual outpatient setting. As a whole, GDHs offer easy access to on-site medical staff, a higher level of rehabilitation therapy and a chance for social interaction that may be important for elderly patients.

8.3 Functions of GDHs

The functions of GDHs have been described in Brocklehurst's review as rehabilitation, maintenance, assessment, medical management, nursing care, and social care.[3] Of these five functions, rehabilitation — which aims to restore the patient to an optimal level

Table 8.1 Geriatric day hospitals in Hong Kong

Cluster	Hospital
Hong Kong East Cluster	Pamela Youde Nethersole Eastern Hospital Ruttonjee Hospital Wong Chuk Hang Hospital
Hong Kong West Cluster	TWGHs Fung Yiu King Hospital Tung Wah Hospital
Kowloon Central Cluster	Kowloon Hospital Kwong Wah Hospital Queen Elizabeth Hospital TWGHs Wong Tai Sin Hospital
Kowloon East Cluster	Haven of Hope Hospital Yung Fung Shee Geriatric Day Hospital (United Christian Hospital)
Kowloon West Cluster	Caritas Medical Centre Princess Margaret Hospital (Lai King Building) Yan Chai Hospital
New Territories East Cluster	Alice Ho Miu Ling Nethersole Hospital North District Hospital Shatin Hospital
New Territories West Cluster	Pok Oi Hospital Tuen Mun Hospital

of ability — has been ranked by geriatricians as the most important.[4] For maintenance, their objective is to maintain the level of independence achieved by rehabilitation. Comprehensive geriatric assessment is the cornerstone of geriatrics and can be performed efficiently at a GDH that offers multidisciplinary team expertise.[5] Medical care can be provided and allows specialist investigations, prolonged observation, and monitoring of treatment. Nursing care such as wound dressings, administration of medications, and changing of tubes such as nasogastric tubes and indwelling urinary catheters can also be carried out at GDHs. Although social care is provided for elderly patients, it is not regarded as the main function of GDHs. Patients with predominantly social problems should instead attend a day centre operated by the Social Welfare Department or non-governmental organisation.

Other specialist services may also be provided by some GDHs in the UK and Hong Kong. These usually include a comprehensive falls services, memory clinics, continence clinics, Parkinson's disease clinics, and various fast track clinics.[6] In the UK, a number of GDHs have also developed services that allow subacute problems to be reviewed by a multidisciplinary team on the same day, or the day following Accident and Emergency Department attendance.[7]

8.4 Staffing, Size, and Location of GDHs

As the main role of a GDH is to provide rehabilitation for patients with chronic disability, they are usually staffed by a geriatrician-led multidisciplinary team that includes nurses, a physiotherapist, an occupational therapist, and social workers. Dietitians, a speech therapist, clinical psychologists, and other allied healthcare professionals are also frequently active members of GDHs. A study in the UK showed that a GDH led by a geriatric consultant is more effective than one led by a general practitioner.[8] Similarly in Hong Kong, most GDHs are geriatrician led and staffed by a multidisciplinary team.

There are variations in size of GDHs in Hong Kong, ranging from a capacity of 13 to 100 day places. Most are located in hospitals, be they acute or extended care hospitals. Nonetheless, some GDHs in Hong Kong (e.g. Yung Fung Shee Memorial Centre in Kowloon East Cluster) have a separate location.

8.5 GDH Operation

In the UK, patients usually attend a GDH 1.2-2.5 times per week on average according to previous reports. Attendance at a GDH twice per week seems to be optimal and most cost-effective.[9] More frequent treatments can be more expensive than inpatient or residential care. In Hong Kong, similar to the UK model, most elderly patients will be allocated 2 or 3 whole-day sessions per week. Nonetheless, GDHs usually provide a certain flexibility and some patients may have half-day sessions with a different frequency depending on individual need.

The modern GDH has adopted a more active rehabilitative rather than maintenance role in the care of elderly patients. Patient progress is discussed at a regular multidisciplinary case conference where the rehabilitation goals are reviewed and amended when necessary. Patients are discharged from the GDH if the multidisciplinary team agrees at the case conference that the patient's progress has reached the

set target. The duration of treatment varies among GDHs and individual patients. In the UK, the average duration is around 13 weeks, with a mean of 19 visits per patient. Morales Martinez et al[10] classified GDH attenders as once only (for assessment), short-term (2-6 visits, for investigation), medium-term (≥7 visits, for rehabilitation), and chronic (for medical or social support care). A survey of GDHs by the Geriatric Subcommittee of the Hong Kong Hospital Authority in 2012 revealed that 8% of attendances were new attendances and that the mean and median attendances per GDH patient were 10.7 and 11.7, respectively. A similar duration and number of attendances per patient has been reported in GDHs in New Zealand.[11] In some local GDHs, patients are scheduled to return for assessment 6 months following discharge. If deterioration in function or mobility is evident, patients can be recalled for a further course of rehabilitation.

The availability of adequate transport is crucial to bring patients with varying degrees of mobility problems to the GDH. The majority of patients rely on Non-emergency Ambulance Transfer Service or Elderly Transport Service. Other means of transport include the Rehabus operated by the Hong Kong Society for Rehabilitation, taxi, and occasionally patient self-transport.

8.6 Patient Source

In Hong Kong, GDHs usually accept elderly patients following discharge from hospital, although they may also be referred from outpatient clinics, a Community Geriatric Assessment Team, a Visiting Medical Officer, the Integrated Care and Discharge Support Program, or a Patient Support Call Centre.[12] The patient characteristics in GDHs are different to those of hospital inpatients. Patients referred to a GDH are usually more medically stable and living in the community, either at their own home or in a residential care home. They are a selected group of patients fit enough to travel to the GDH several times a week and are required to be fairly cooperative and at least moderately motivated if they are to benefit from the rehabilitation training offered by the GDH. Thus, before accepting them for treatment, many GDHs either require patients to be referred by geriatricians or will screen patients to confirm their suitability for GDH training or willingness to regularly attend the GDH. An elderly patient who is aggressive may not be suitable for GDH treatment as they will disrupt smooth operation of the GDH and pose a danger to other attendees, staff, and themselves.

8.7 Factors That Affect the Performance of GDHs

The following factors may affect the performance of a GDH.

(1) The staffing of GDHs, especially by multidisciplinary therapists
(2) The presence of medical support services, including geriatric bed availability for GDH patients if inpatient treatment is required
(3) The availability of adequate transport to bring patients on time to the GDH and return them home afterwards
(4) The size of the GDH[13]
(5) The presence of social supportive services so that isolated patients can be given appropriate social support following discharge from the GDH
(6) The availability of equipment for more efficient rehabilitation and pain control, that in turn depends on the space of GDHs and the funds available to purchase suitable equipment

8.8 Evaluation of Activity of GDHs

Many parameters have been suggested to evaluate the outcomes of GDHs. These include the use of activity indicators as well as effectiveness measurements.[14]

8.8.1 Activity Indicators for GDH

8.8.1.1 New Patient Index

Martin and Millard[15] showed that most active GDHs had an average stay of 9.5 visits for new patients. By taking 10 days of attendance as the optimum length of treatment for new patients at GDHs, they devised a New Patient Index (NPI) to measure the activity of a GDH. An active GDH will have a NPI approaching 1, while an inactive GDH will have a NPI approaching 0.

NPI = (Number of new patients expected x 10) / (Size of unit x days worked)

For example, assume a GDH has 380 new cases per year, with a capacity of 22, and 246 working days that year. Then the NPI will be: (380 x 10) / (22 x 246) = 0.7

8.8.1.2 Corrected New Patient Index

The limitation of the NPI is that it is affected by the occupancy rate: units that do not work at full capacity will have an apparent reduction in activity when assessed by the NPI. Professor Millard, in his reply letter to TK Kong's enquiry, provided information on his revision of the NPI to the Corrected New Patient Index (CNPI) to correct for variations arising from differences in occupancy rate (Millard PH, personal communication, March 1990). The revised formula for the CNPI[14] is:

CNPI = (Number of new patients x 10) / (Size of unit x occupancy x days worked)

8.8.1.3 New Referral Rate

The New Referral Rate (NRR) assesses the activity

of a day hospital by measuring the proportion of attendances that represent new patient referrals[16]:

NRR = (Number of new patients x 100%) / Total attendance

8.8.2 Other Activity Indicator Outcome Assessments

Occupancy is an easy reflection of GDH activity. However, a high occupancy, while indicating full use of capacity, may also occur when there are inactive units with long length of stay and low turnover. Total attendance in a year is a crude index, as it is affected by the number of working days, the size of the GDH, and the occupancy. Average frequency of attendance in terms of number of visits per week can also be another indicator of GDH activity. A high number may reflect intensive rehabilitation, but has to be interpreted relative to the length of stay. Other indices that have been reported include proportion of chronic cases, readmission number, discharge destination, percentage of inactivity, and default rates.[14]

8.9 Effectiveness Measurement for GDH

The outcome of GDHs can be assessed by measuring improvement in motor, functional, social, cognitive, and psychological states as well as quality of life. Various validated measurements for these are available although it is beyond the scope of this chapter to describe these in detail. Readers are encouraged to read the relevant chapters and references for more detailed information.

8.10 Effectiveness of GDH

Although the model of GDH has become quite universal in the UK, Australia, New Zealand, and Hong Kong, in other parts of the world such as the US, the model has not been widely implemented, in part because of a controversial evidence base. In 1994, Siu et al[17] reported failure of GDHs to demonstrate better outcomes from comprehensive assessment compared with assessment in a clinic setting without GDH. In 1999, Foster et al[18] failed to find a significant benefit of GDHs versus alternative services. The Huntingdon Day Hospital Trial compared GDH with day centre rehabilitation by measuring mobility and activities of daily living.[19] After 3 months, they could demonstrate no difference between a GDH and day care centre in outcome measures. The Cochrane review (2008 version) of 13 trials that involved 3007 participants showed that compared with no treatment, attendance at a GDH offered benefits including improved activities of daily living and decreased use of hospital beds. Nonetheless, there appears to be little advantage of GDHs compared with other forms of comprehensive elderly services including those in day care centres as well as domiciliary treatment.[20] In a more recent 2011 UK study, patients who underwent home-based rehabilitation were not disadvantaged compared with those who attended day hospital rehabilitation.[21]

Despite these controversies, the GDH continues to be an integral part of geriatric care in Hong Kong. The effectiveness of GDHs has been demonstrated by various local studies. In 1995, a local GDH study showed that it was effective in improving the self-care ability and mobility of discharged patients and the improvements also related significantly to the three major diagnostic groups: stoke, Parkinsonism, and hip fractures.[22] Another local study in the same year compared conventional medical management and GDH for stroke patients: care in the GDH hastened functional recovery and reduced outpatient visits for elderly stroke patients without additional cost.[23] Another study in Hong Kong has also proven that a GDH is effective in improving the rehabilitation of stroke patients.[24]

One important question about GDHs concerns whether they are suitable for patients with poor cognitive function. Patients need to be fairly cooperative and motivated before they can benefit from the rehabilitation training at a GDH. To address this question, a local study was performed in 2011 to examine the effect of cognition on gain in functional and motor performance in 547 older patients undergoing rehabilitation at a GDH.[25] There was a strong association between Cantonese version of Mini-Mental State Examination admission and Functional Independence Measure (FIM) on discharge. Cognitive function was not associated with FIM efficacy and efficiency suggesting that selected patients with impaired cognitive function could benefit from GDH rehabilitation. This study supports the provision of GDH services to selected older patients with varying degrees of cognitive function since even severely demented patients could demonstrate significant improvement in FIM, with efficacy and efficiency comparable with that of patients with higher cognitive function.

Another important controversy is whether the improvement gained in a GDH can be maintained after discharge. One study performed in Canada of community-dwelling older patients reported that there was no sustained improvement in mobility or functional status 3 months following discharge from a GDH.[26] In order to address this controversy and examine whether the same situation applies in Hong Kong, a study was carried out in 2011 of the FIM score 6 months after discharge from a GDH. It showed that although there was a significant drop in FIM 6 months following discharge from a GDH, patients could maintain a significant degree of their

rehabilitation.[27] This suggested that GDH training can have a long-lasting effect.

In summary, although the benefit of GDHs in overseas studies is controversial, they remain crucial to the geriatric services in Hong Kong. Further local study is recommended to demonstrate the benefits of GDH in the local setting.

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CHAPTER 9

TELEMEDICINE

Teresa Kim-kam YU

OBJECTIVES

- To understand the use of telemedicine in different areas of geriatric care services and in specific diseases of older age.
- To know the benefits and drawbacks of telemedicine.

KEY POINTS

- There is ambiguity in the terms telemedicine, telehealth, telecare, and eHealth.
- The main categories of telemedicine include store-and-forward, real-time interactive telemedicine, and remote monitoring.
- The use of telemedicine in different areas of geriatric care (inpatient, post-discharge follow-up, nursing home care, home care, and electronic patient records) and in specific diseases (dementia, congestive heart failure, diabetes mellitus, and stroke) of older age are discussed.
- The major benefits of telemedicine are to improve access to care (especially for remote and rural areas), reduce healthcare costs, and facilitate medical education. The major drawbacks include cost (especially the setup cost and telecommunication cost), decreased human interaction, and organisational and bureaucratic difficulties.
- Although telemedicine has been suggested to be feasible and useful, the general standard of research in this field is poor and more randomised controlled studies are required, especially with regard to its efficacy and cost-effectiveness in the care of older persons.

9.1 Definition and Technical Issues

The population in Hong Kong is ageing. The proportion of the population aged ≥65 years is projected to rise markedly from 13% in 2011 to 30% in 2041.[1] The ageing population has imposed stress on our healthcare system and innovation is needed to overcome this global challenge. Advances in telecommunication technology may provide a new way of caring for older persons.

The World Health Organization gives a very broad definition of telemedicine — the delivery of healthcare services, where distance is a critical factor, by all healthcare professionals using information and communication technologies for the exchange of valid information for diagnosis, treatment and prevention of disease and injuries, research and evaluation and for the continuing education of healthcare providers, all in the interests of advancing the health of individuals and their communities.[2] While others restrict telemedicine to the use of information and communication technologies to provide clinical services, some use the term telehealth, telecare, or eHealth as a broad definition that encompasses clinical and non-clinical services such as medical education, administration, and research.

The telecommunication technology used in telemedicine includes simple telephone, fax machine, email, internet with broadband transmission, real-time video conferencing, fibre optics, smartphones, wireless communication channels, and satellites. Telemedicine began in the mid 20th century and widespread interest in the healthcare field has escalated in recent decades with the availability of more sophisticated technology.

Major categories of telemedicine in the literature are as follows.[3]

- Store and forward — store-and-forward telemedicine refers to the use of asynchronous (not real-time) computer-based communication between a patient and a consulting provider, or a referring healthcare provider and a medical specialist at a distant site for the purpose of diagnostic and therapeutic assistance in the care

of patients who otherwise have no timely access to specialty care.[4]

- Real-time interactive telemedicine — provides synchronous real-time interaction between a patient and healthcare provider, usually via a videoconferencing device or wireless tools.
- Home healthcare and remote monitoring systems — provides care to individuals and families in their place of residence to promote, maintain, restore health, or to minimise the effects of disability and illness, including terminal illness. Use of remote monitoring and interactive devices allows the patient to remit information about vital signs on a regular basis to a provider without the need for travel.[5] Monitoring technology is classified into two broad categories, a portable or wearable device (e.g. blood pressure monitor, glucose monitor) and fixed or environmental devices (e.g. motion sensor, acute fall detector).[6]

Predominant themes of telemedicine are shown in Table 9.1.[3]

9.2 Use of Telemedicine in Different Areas of Geriatric Services

Geriatric care services range from hospital inpatient care to community care. A search of the literature revealed that telemedicine may be more feasible for some areas of geriatric care than others, and research into the application of telemedicine has focused on these areas.

9.2.1 Inpatient Management

A major theme in the telemedicine literature is teleconsultation with telecommunication linkage of major hospitals to peripheral hospitals. Most small hospitals do not have 24-hour coverage of specialist services and telemedicine may help to reduce this disparity, e.g. teleradiology. As geriatric specialists may not be available in all hospitals, especially in small peripheral hospitals, linkage will bring geriatric specialty care to peripheral hospitals. The use of telemedicine in inpatient management has been beautifully demonstrated in the area of acute stroke care, where a peripheral hospital (spoke hospital) was connected to a major hospital (hub hospital) with real-time video conferencing. This enabled cutting-edge management of acute stroke to be provided to patients in the peripheral hospital.[7] A Cochrane review on telemedicine versus face-to-face patient care found that there were no detrimental effects on patient care and that the use of telecommunications technology was feasible despite little evidence of clinical benefits.[8] The technological aspects appeared to be reliable and well accepted by patients but no formal economic analysis was included.

Table 9.1 Predominant themes of telemedicine[3]

• Teleconsultation
• Home care support and patient self-monitoring of chronic disease
• Diagnostic accuracy of systems for teleradiology and telepathology
• Technical standards
• The medical needs of underserved populations
• Acceptability of telemedicine to providers and patients
• Diagnostic test interpretation
• Chronic disease management
• Post-hospitalisation or postoperative follow-up
• Emergency room triage
• Real-time video conferencing by a specialist

9.2.2 Post-discharge Follow-up

The early few weeks following discharge from hospital are the most vulnerable period for patients as they may be deconditioned, develop complications, or have problems with changes to medication. The Community Health Call Centre of the Hospital Authority targets patients who have been discharged and who are considered high risk with the use of the validated Hospital Admission Risk Reduction Program for the Elderly score,[9] and proactively telephones these patients to follow up within 48 hours of discharge. Results have been encouraging with a 25-50% reduction in frequent and unnecessary readmissions to hospital.[10] Nonetheless, a Cochrane review on telephone follow-up for post-discharge problems in patients discharged from hospital to home found that the 33 studies included were of low methodological quality. Although some studies reported favourable effects of the intervention for some outcomes, overall the studies showed clinically equivalent results for the telephone follow-up and control groups.[11]

9.2.3 Nursing Home Care

It is more cost-effective to provide telemedicine in facilities for older persons such as elderly care centres and nursing homes. Many studies have examined the feasibility of telemedicine for outreach care in a nursing home. A Hong Kong study showed that telemedicine was adequate for most of the service delivery and for the geriatricians; 97% of routine follow-ups and 90% of urgent referrals could be managed by telemedicine.[12] The study also showed that the telemedicine service was cheaper with a 9% reduction in accident and emergency department (AED) visits and 11% fewer admissions to acute hospitals.

Another local randomised controlled study of telemedicine in an elderly nursing home also showed that 95.4% of follow-ups conducted through real-time videoconferencing were considered

feasible.[13] Healthcare professionals, nursing home staff, and patients were generally satisfied with the telemedicine service. In addition, clinical outcomes were not compromised, as there was a 44.4% decrease in total AED attendances and a 46.6% decrease in AED admissions in the telemedicine group, along with some cost savings. Nonetheless, the results did not reach statistical significance.

9.2.4 Home Care

With the increasing availability of computers and internet connection in general households, provision of telemedicine at home for the older person may be feasible and cost-effective. Home care for an older person via telemedicine includes monitoring of chronic disease and installation of sensors / detectors in the home for health support.

For chronic disease monitoring at home, much research has been done in the areas of congestive heart disease,[14-18] post cardiac surgery,[19,20] chronic obstructive airway disease,[18] diabetes mellitus,[18,21-23] and renal disease.[24] Telecommunication methods have included telephone follow-up, store and forward of vital signs data, internet-based education, and real-time videoconferencing. Most studies showed equal or slightly positive results compared with routine care. Telemedicine monitoring was feasible with satisfactory patient acceptance. One tele-home care study showed that patients with heart failure, chronic lung disease, and / or diabetes who used a standard phone line to communicate with the hospital had a significant decrease in bed days of care, emergency room visits, and glycated haemoglobin (HbA1c) level at 6 months compared with controls.[14] A systematic review of 29 peer-reviewed articles on home telehealth showed that it had a moderately significant positive effect on clinical outcomes.[25] In addition, subanalyses revealed a significant positive effect of telehealth for some disease categories such as heart disease and psychiatric conditions, but not diabetes.[25] Recent electronic advances with biosensors on microchips have made monitoring technology possible in the form of clothing, watches, and jewellery. Advances in communication have also enabled wireless transfer of data from sensors.[26]

In addition to portable monitors or those that can be worn, there are also fixed or environmental devices.[6] The term "smart home" applies to a home equipped with technology that allows monitoring of its residents and / or encourages independence and the maintenance of good health.[27] Recent studies have evaluated smart homes with regard to emergency assistance, fall prevention / detection, reminder systems, administration of medication, and assistance for those with cognitive or sensory impairment.[28] The technologies used included infra-red sensors,[29] dual-axis accelerometer with microcomputer,[30,31] smart clothing integrating sensors in textile construction,[32] floor vibration and acoustic sensing,[33] and a global speech / sound recognition system.[34] Some of the technologies are experimental however, and ethical concerns have been raised about personal privacy.

A UK review of telecare and older people gives a comprehensive review of the types of telecare equipment available and various UK pilot studies.[35] Telecare equipment and services available included telephone or web-based information systems, electronic assistive technology (e.g. automatic doors, automatic beds, stair lifts, and electronic prompts to ensure medication intake), safety monitoring (e.g. monitors for bath floods, gas leaks, unlocked doors, and fires), personal monitoring (e.g. fall detectors, and 'wander' monitors), and vital signs monitoring (e.g. blood pressure, body weight, and temperature).[35]

In a large systematic review of 68 randomised controlled trials and 30 observational studies of home telecare for frail elderly patients with chronic conditions, the most effective telecare interventions appeared to be automated vital signs monitoring and telephone follow-up by nurses.[36] The cost-effectiveness of these interventions, however, was less certain and there was a lack of evidence for their effectiveness in home safety and security alert systems. Another Cochrane review of the interactive health communication applications for people with chronic disease comprised computer web-based information packages with health information and at least one of social support, decision support, or behaviour change support.[37] The review showed that use of interactive health communication had a significant positive effect on knowledge. Users felt better socially supported and behavioural and clinical outcomes may have improved.[37]

9.2.5 Electronic Patient Record

Older persons often have multiple diseases and may require multispecialty follow-up. An electronic patient record (ePR) can provide a comprehensive health profile of older persons and will facilitate their healthcare. Information about a patient's follow-up and drugs will facilitate medication review, recognition and prevention of iatrogenesis, attention to and avoidance of medication duplication, and potential drug-drug interactions and drug-disease interactions.

In Hong Kong, the ePR of the Hong Kong Hospital Authority can provide clinical information about a patient's diagnosis, surgical / endoscopic / diagnostic procedures, clinical notes (including general outpatient records, specialist outpatient records, accident and emergency records, and

inpatient discharge records), radiology reports and actual radiological images, laboratory results, medications from all clinical specialties, and dates of future appointments.

9.3 Specific Diseases in Geriatric Care That may Benefit from Telemedicine

9.3.1 Dementia

Telemedicine has a long history of use in psychogeriatrics. Structured interview by videoconferencing using the Cambridge Cognitive Examination has been proven reliable.[38] Comparison of face-to-face, fax, and videoconferencing scoring on the Mini-Mental State Examination (MMSE) has also shown that sentences, but not pentagrams, can be reliably scored over videoconferencing.[39] Another study also showed that MMSE and the Geriatrics Depression Score could be determined through telehealth methods and yielded similar results to direct assessment.[40] Moreover, another study of telemedicine diagnosis of dementia in older persons at remote sites revealed the results to be in full agreement with in-person clinical examinations. Subjects also reported a high degree of satisfaction.[41]

Information technology can also help to identify the location of a demented older person. Integrating the technologies of radiofrequency identification, global positioning system, global system for mobile communications and geographic information system, it is possible to locate a demented older person within 8 metres of average deviation.[42] Alerting systems ('wander' monitors) could alert carers if a patient left a certain area. A low-power mobile phone (PHS) was carried by the subjects and their location was identified within 100 metres of the PHS antenna. Carers could set a patient's movement zone with specialised computer software and when the subject left that zone, alert signals would be sent to the carers and the patient's location map sent via email.[43] Another study used radiofrequency identification technology to develop a safety monitoring mechanism that could alert carers when subjects carrying the light tags approached a dangerous area or strayed too far.[44]

9.3.2 Congestive Heart Failure

Congestive heart failure (CHF) is prevalent in older persons and many studies have evaluated the usefulness of telemedicine, with the use of telephone support and / or telemonitoring in this disease. A recent Cochrane review on telemonitoring programmes for CHF patients included 25 studies and five published abstracts with over 8000 participants: 16 studies evaluated structured telephone support, 11 evaluated telemonitoring, and two evaluated both.[45] It found that telemonitoring significantly reduced all-cause mortality and structured telephone support demonstrated a non-significant positive effect. Both telemonitoring and structured telephone support reduced CHF-related hospitalisations. Quality of life, evidence-based prescribing, patient knowledge, and New York Heart Association functional class were also improved. Healthcare costs were reduced and patients found the interventions acceptable.[45]

9.3.3 Diabetes Mellitus

The IDEATel (Informatics for Diabetes Education and Telemedicine) project — which included the home telemedicine unit providing videoconferencing, electronic transmission for finger prick glucose and blood pressure readings, secure web-based messaging, clinical data review, and access to educational materials — has been shown to be a feasible means to care for elderly diabetic patients at home.[21] A more recent study of case management intervention with nurse and dietitian teleconferencing via a computer, upload of blood glucose and blood pressure data, and access to educational material showed that patients valued the emphasis on monitoring of health outcomes and the supportive contact with healthcare staff.[22] Daily monitoring and education of elderly diabetic veterans of different racial / ethnic groups by a low-tech telephone-based in-home messaging device showed improvement in HbA1c (especially in the African-Americans), decreased hospital admissions, and reduced bed days of care.[23] A promising approach using handheld communicating devices — such as smartphones loaded with software to apply physician algorithms for insulin dose calculation, food choice optimisation, remote monitoring of blood glucose, and even teleconsultation — has also been shown to improve glycaemic control.[46]

9.3.4 Stroke

Telemedicine can facilitate the acute care of stroke patients at hospitals that lack the specialists of major stroke centres with a hub-and-spoke telestroke network. Hubs were generally located in urban areas with the spokes in rural regions or in urban hospitals with no stroke specialists. With this networking, the specialists in the hubs could order timely state-of-the-art treatment (e.g. tissue plasminogen activator) to stroke patients at the spoke sites.[7]

In the rehabilitation phase, educational talks, exercise, and psychosocial support conducted by a physiotherapist using videoconferencing to a sample of stroke patients in a community centre was proven to be feasible.[47] This community-based stroke rehabilitation through videoconferencing showed improvements in Berg Balance Scale, State Self-Esteem Scale, Medical Outcomes Study 36-item Short Form, and stroke knowledge test scores. A

Table 9.2 Benefits and drawbacks of telemedicine[49]

Benefits	• Provision of care not previously deliverable, e.g. in rural and underserved areas • Improved access to information — both for healthcare professionals and patients • Improved access to services and increased care delivery — decreased waiting time, decreased travel time, and improved equity of access • Improved professional education • Reduced variability of diagnoses and improved clinical management • Reduced healthcare cost
Drawbacks	• Cost, especially the initial setup cost and the cost of telecommunication • Decreased human interaction between healthcare professionals and the patient — particularly for physical examination which is difficult to carry out through telemedicine • Decreased human interaction between healthcare professionals • Issues concerning the quality of health information — especially those for the general public, produced by individual, charitable organisations or special interest groups. The content may be biased, insufficient, or even inaccurate. • Organisational and bureaucratic difficulties — high costs, underdeveloped infrastructure, lack of technical expertise, lack of legal policies or guidelines on privacy and confidentiality of patient information, difficulty of organisation and / or human culture change, competing priorities within the health systems, and a perceived lack of demand for telemedicine.[50]

recent Cochrane review of telerehabilitation services for stroke patients included 10 trials with a total of 933 participants and concluded that the studies were generally small with inadequate reporting quality. Interventional approaches included upper limb training, lower limb and mobility retraining, case management, and caregiver support. There was insufficient evidence to draw a conclusion about the effectiveness and no studies evaluated the cost-effectiveness of telerehabilitation after stroke.[48]

9.4 Benefits and Drawbacks of Telemedicine

Health authorities have high expectations for telemedicine but the general standard of research on telemedicine is poor and there is limited objective information about its benefits and drawbacks. Table 9.2 summarises the benefits and drawbacks of telemedicine, mostly based on a study by Hjelm.[49]

9.5 Summary

With advances in technology, increasingly widespread availability of home computers with an internet connection, the unprecedented spread of mobile technologies, and the increasing emphasis of care in the community, telemedicine may be the way forward for the care of older persons. Telemedicine may be feasible and useful in some areas of geriatric care such as inpatient care, post-discharge follow-up, nursing homes and home care, and in certain geriatric diseases such as dementia, CHF, diabetes mellitus, and stroke. It is important to know that the general standard of research in this field is poor and has often excluded patients with typical impairments related to old age (e.g. cognitive and visual impairment, hearing problems, and communication barriers).[51] More good-quality randomised controlled studies are required to understand telemedicine, especially its efficacy and cost-effectiveness in the care of older persons.

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CHAPTER 10

PREVENTIVE GERIATRICS

Tai-pong LAM, Felix Hon-wai CHAN

OBJECTIVES

To learn the general principles and specific issues in relation to health promotion and illness prevention among elderly patients, to review the barriers in the practice of prevention, and discuss the way forward in enhancing primary care.

KEY POINTS

Learning points

- Primary care plays a crucial role in promoting health awareness and tackling the causes of ill health among older people.
- Biological, pathological, and psychosocial factors may contribute towards poor health in elderly people.
- Preventive measures for elderly persons are not limited to improving physical health, but should also extend to improving overall functioning, and psychological and social health so as to achieve overall 'healthy ageing'.
- The knowledge, skills, and attitudes of treating doctors are important elements in the provision of preventive care for elderly people.

Practical tips for primary care physicians

- Physical exercise is of tremendous benefit to elderly patients.
- Iatrogenic diseases are the most preventable conditions in older persons.
- All patient populations should be viewed as populations at risk.
- The structure and organisation of the practice affects the delivery of preventive care.
- Reference framework published by the Primary Care Office containing summary sheets with up-to-date information will facilitate the practice of prevention.

Practical advice to patients

- Asymptomatic diseases such as hypertension and non–insulin-dependent diabetes mellitus are worthy of medical attention.
- Doctor shopping can be detrimental to patients' health.
- Controlling blood pressure and maintaining healthy blood glucose and cholesterol levels are important in preventive care.

10.1 Introduction and Terminology

As the Hong Kong population is ageing rapidly, the consumption of healthcare services by elderly people is also increasing disproportionally. Since the majority of elderly people are still living in the community setting, primary care plays a crucial role in promoting health awareness and tackling the causes of ill health among older people.[1] The following shows the common terms used in preventive geriatrics.

- 'Ageing' is a process of gradual and spontaneous change, resulting in maturation through childhood, puberty, and young adulthood, and followed by decline in general health through middle and late age. Ageing, therefore, has positive elements of development and the negative elements of decline.
- 'Health' is not the mere absence of disease, but rather the individual's ability to realise aspirations, satisfy needs, and respond positively to the challenges of the environment — a resource for everyday life, not the object of living.
- 'Health promotion' is the process of enabling people to increase control over, and to improve, their health.[2] Health promotion is any planned

combination of educational, political, regulatory, and organisational support for actions and conditions of living that are conducive to the health of individuals, groups, or communities.[3]

- 'Primary prevention' aims to reduce the incidence of disease by personal and communal efforts, such as by decreasing environmental risks, enhancing nutritional status, immunising against communicable diseases, or improving water supplies. It is a core task of public health, including health promotion.[4]
- 'Secondary prevention' aims to reduce the prevalence of disease by shortening its duration. If the disease has no cure, secondary prevention may increase survival and quality of life. It seldom prevents disease occurrence; it does so only when early detection of a precursor lesion leads to complete removal of all such lesions. It comprises a set of measures available to individuals and communities for early detection of disease conditions and prompt intervention to control disease and minimise disability, such as by using screening programmes.[4]
- 'Tertiary prevention' consists of measures aimed at softening the impact of long-term disease and disability by eliminating or reducing impairment, disability, and handicap; minimising suffering; and maximising potential years or useful life.[4]

10.2 Contributors to Poor Health

The contributors to poor health in elderly people can be broadly categorised under either biological or psychosocial factors.[5-7] Resourceful information can be obtained in the Hong Kong Reference Framework for Preventive Care for Older Adults in Primary Care Settings[4] and its Module on Health Assessment. Detailed information is not repeated here but important areas are listed in Table 10.1.

Table 10.1 Factors contributing to poor health in elderly people

Type	Factor
Biological	• Cardiovascular diseases • Falls and fractures • Hearing and visual impairment • Cancers • Smoking-related illnesses • Iatrogenic diseases • Poor oral hygiene • Lack of exercise • Alcohol consumption
Psychosocial	• Inadequate social roles (post-retirement) • Unsuitable housing • Poverty • Mental health problems • Bereavement • Relationship problems • Unmet need of carer support • Elder abuse

Table 10.2 Important preventive measures

Type	Measure
Biological	• Blood pressure measurement • Height and weight measurement • Nutritional assessment • Cholesterol measurement • Faecal occult blood testing • Breast examination by a physician • Pap smear / pelvic examination • Visual acuity testing • Hearing test • Influenza and pneumococcal vaccination • Mobility and balance assessment • Activities of daily living
Behavioural	• Smoking cessation • Alcohol consumption screening • Dietary advice, including improving calcium intake • Physical exercise • Oral and dental hygiene

10.3 Important Preventive Measures

You will again find a lot of useful information in Hong Kong Reference Framework (refer to the Annex).[4] It is important to note that preventive measures for elderly people are not limited to improving physical health, but also extend to improving the overall functioning, and psychological and social health so as to achieve 'overall healthy ageing'.[5-7] Examples are shown in Table 10.2.

Physical exercise is worth a special mention as it brings tremendous benefits to elderly patients. Please refer to Chapter 4.6 of this textbook and Chapter 24 of "Oxford textbook of geriatric medicine (2nd edition)" for more details.[8]

A useful strategy described in the Hong Kong Reference Framework is to take into consideration elderly patients' functional capacities to select appropriate preventive measures.[4] Elderly patients are, therefore, broadly categorised into the following.

10.3.1 Independent with No Known Chronic Diseases

Primary preventive measures such as vaccination, following healthy lifestyles, and screening for chronic illness are adopted to maintaining optimal functional capacity and extending a healthy active life.

10.3.2 Independent with Chronic Diseases

Primary and secondary preventive measures, and appropriate management of chronic diseases are adopted in order to maintain functional independence.

10.3.3 Older Adults with Disabilities

These are elderly patients with multiple comorbidities and functional deficits. Appropriate primary,

secondary, and tertiary preventive measures should also be adopted, together with functional assessment by reviewing two major divisions of functional ability: basic activities of daily living and instrumental activities of daily living. The aims of these prevention strategies are to prevent further loss of function.

10.4 Barriers to the Practice of Prevention

10.4.1 The Healthcare System

In Hong Kong, 15% of primary care is provided by the government in heavily subsidised public outpatient clinics. However, over 70% of care is provided by private practitioners with little or no government payments involved. The majority of these private patients pay their medical expenses out of their own pockets. Many elderly patients may not see the immediate benefits of paying for preventive consultations or investigations while the doctors fear being seen as soliciting 'new business' by their patients if any laboratory investigations suggested are not directly related to their current illness.

In 2009, the government introduced the Elderly Health Care Voucher Scheme to subsidise primary healthcare services in the private sector, including dental and other preventive care services, for local residents aged ≥70 years. Public Private Partnership (PPP) programmes in chronic disease management have been piloted in selected clusters in the Hospital Authority.

10.4.2 The Structure and Organisation of the Practice

In the government general outpatient clinics, each patient is allowed an average of 5 to 10 minutes for each consultation. Time is, therefore, limited for preventive care. Lack of time is also a major barrier in private practice as some busy practitioners may see over 50 patients per day. However, electronic health record and enhanced PPP schemes with appropriate timely alerts to the doctors should help facilitate preventive care. A summary of preventive care will greatly enhance its delivery.

10.4.3 The Patient

Doctor and patient factors are probably far more significant than the factors of healthcare system and the structure and organisation of practice in the development / obstruction of prevention practices in primary care.

Local elderly Chinese patients in Hong Kong are strongly influenced by the Chinese culture in their health belief model. Patients often visit their primary care physicians (PCPs) for self-limiting illnesses and tend to neglect any asymptomatic diseases such as hypertension and non–insulin-dependent diabetes mellitus. This, to a certain extent, was reflected in a morbidity survey done by PCPs in 2008, with nearly 28% of patient encounters being, primarily, for upper respiratory tract illnesses.

Another major patient factor is doctor shopping. Many elderly patients visit several doctors concurrently including traditional Chinese medicine practitioners, and they decide for themselves who they want to see for a particular illness. It must be most disturbing to many PCPs that they are never certain which physicians the patients will visit in future. This is detrimental to the establishment of an effective doctor-patient relationship, which is central to the provision of care by the doctor.

Lack of financial resources may also make it difficult for an elderly person to seek preventive services on a regular basis.

10.4.4 The Doctor

The last, and probably the most important, barriers to the practice of prevention, are the doctor factors. PCPs often concentrate on the major presenting complaint and the curative aspect of a consultation. Some may not see the roles and responsibilities in the area of prevention. It is generally accepted that doctors have a medicolegal responsibility to provide the correct diagnosis and treatment and, yet, there is no defined boundary as to what constitutes the appropriate level of preventive care expected from doctors. In addition, there is little financial incentive for doctors to practise preventive care.

The doctor factors that hinder the practice of prevention can be divided into three types as described below.

10.4.4.1 Knowledge Factor

The lack of knowledge of preventive care may be a problem for some PCPs, but, probably only in a minority of the PCPs. Lack of clear guidelines for the local population might have caused confusion among some doctors in the past. This has now been overcome, to some extent, with the provision of the Hong Kong Reference Framework.

10.4.4.2 Skill Factor

Skills in communication are central to all forms of preventive care. It requires special skills to convince elderly people, particularly the asymptomatic ones, to agree to undertake important preventive measures.

10.4.4.3 Attitude Factor

The attitude of the doctor is, by far, the most important factor in determining the extent of

preventive medicine provided. Some doctors do not provide any prevention at all as they do not perceive any benefits of such practice. This may be due to the fact that preventive measures often do not provide immediate results. Another reason is that doctors often set an unrealistic target for themselves and give up altogether after a short while since they fail to achieve the target. For example, many PCPs may not realise that 5-10% is the average success rate for smokers to quit in 12 months after being identified by PCPs as individuals for behaviour modification. Furthermore, PCPs often underestimate or simply fail to recognise the interest patients may have in the advice being given to them. They also fail to recognise the significant influence they may have on their elderly patients.

10.4.5 How to Overcome These Barriers?

Having identified the major barriers to practising prevention, let us see how some of these may be overcome.

Firstly, PCPs should be made aware of their great contributions to health. They are respected by their elderly patients who, in fact, treasure the health advice being given to them. The concept of viewing our patient populations as populations at risk should be emphasised. It is our responsibility to maintain our elderly patient population as healthy as possible, and preventive care is one of the essential ingredients in achieving this goal. Summary sheets with up-to-date information will facilitate the practice of prevention. Patient-held records with information on preventive care are also very useful.

In the wider community context, the government and its health agencies should provide education to the public on the need to have a regular 'family doctor' for every family and individual so that there is continuity in the care provided; this will serve to facilitate the practice of preventive care. The general public should also be educated on the importance of preventive care such as regular checking of blood pressure, and cholesterol and blood glucose levels. In addition, cervical and colorectal cancer screening as well as annual vaccination for influenza should be provided. For elders aged ≥65 years, the Centre for Health Protection in Hong Kong recommends influenza and pneumococcal vaccination (see Chapter 32.5.1). PCPs must realise that it is not easy to change the health belief model of elderly patients, and the change in their attitude can only be achieved over a long period of time with combined efforts from individual doctors and public health agencies.

The need to restructure the consultation time is a matter of consideration for individual private practitioners. It may be less financially rewarding in the short term, but is certainly, professionally, more satisfying. One would argue that one's professional life is probably extended if one is more satisfied with one's own work, thus, leading to better overall professional income in the long term.

10.5 The Way Forward

Healthcare is undergoing reforms in Hong Kong. Enhancing primary care is one of the priorities of the government. Primary Care Office has also been set up within the Department of Health and charged with the responsibility of promoting the importance of primary care. The Hong Kong Reference Framework for Preventive Care for Older Adults in Primary Care Settings provides a locally relevant reference for preventive care of our elderly patients. PCPs are now in a strong position to deliver these services to enhance the health of our elderly patients.

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CHAPTER 11

PSYCHOGERIATRIC SERVICES

Pey-chyou PAN

OBJECTIVE

To learn the scope of psychogeriatrics, its history and current developments both internationally and locally, as well as the different psychogeriatric services available in Hong Kong.

KEY POINTS

- Psychogeriatrics is a subspecialty in psychiatry that cares for patients above the age of 65 years.
- A comprehensive mental healthcare service for older people consists of a wide range of social institutions and facilities among which the specialist psychogeriatric team is only one of the components.
- Systematic development of a psychogeriatric service in Hong Kong only began in 1994.
- Different types of services in the public sector include an outpatient service, an outreach service, a consultation liaison service, inpatient care, an Elderly Suicide Prevention Programme, a clinical psychology service, a psychogeriatric day hospital / day patient service, and memory clinics.
- Future developments in psychogeriatrics will be shaped by a number of forces that include limited resources, advances in new technology, and changing health priorities.

11.1 Scope of Psychogeriatrics

Psychiatry is the branch of medicine that treats clinical conditions characterised by a prominent disturbance in mental activity and behaviour. Mental activity relates to consciousness, emotion, perception, thought, cognition, and insight.

Psychogeriatrics is a subspecialty of psychiatry, the scope of which is defined by age, with a lower age limit of its clientele being set at 65 years. Some clinicians favour a definition based on age of onset of the clinical condition in question. Thus disorders that begin at an early age are excluded. The advantage of such a definition is that it allows continuity of care for patients with a long-term psychiatric disorder by a clinical team who originally diagnosed and managed the patient. Nonetheless there are disadvantages to this approach. With increasing age, patients will develop other illnesses. The interaction between illnesses, disabilities, and altered response to medications demand alteration to the treatment approach. Changes in the social context of patients as they age also introduce new stresses and support systems. These developments are better handled by a clinical team that deals specifically with elderly patients.

Sadavoy[1] believes psychogeriatrics has five defining elements:

- complexity of the factors causing or perpetuating the disorders and intervention strategies;
- chronicity of the disorders and their associating factors;
- comorbidity and hence multiple interacting concurrent illnesses and treatment modalities;
- continuity of environmental, psychological, and physiological development that constitutes the person as an individual; and
- context in which a psychiatric disorder emerges will have very important implications for the manifestation, management, and outcome of the disorder

11.2 History of Psychogeriatric Services

Like geriatric medicine, psychogeriatrics as a psychiatric subspecialty has a rather recent origin. Although most psychiatric hospitals have a significant proportion of long-stay elderly patients who have historically often been grouped in chronic wards, they have usually received inadequate care for their psychiatric problems. Patients with dementia have been avoided by staff or even banned from psychiatric wards. Functional disorders in elderly people were often undertreated.

In the early 1970s, a number of joint psychogeriatric

assessment units were set up in the UK, offering assessment and placement advice to elderly patients in the community. This marked the birth of psychogeriatrics in Great Britain. Under the influence of several pioneers, notably T. Arie,[2] J. A. Whitehead,[3] and B. Pitt,[4] a number of highly effective comprehensive psychogeriatric services were set up, adopting an enthusiastic community-oriented approach coupled with effective use of beds.

Over the next two decades, a psychogeriatric service grew steadily in the UK. Multidisciplinary old age psychiatric teams proliferated, serving defined catchment areas covering the entire nation. Guidelines were established for manpower requirements, standards of practice, and collaboration between psychogeriatricians and geriatricians.[5] On an academic level, important advances were made in neuroscience, epidemiology, diagnosis, and treatment of psychogeriatric conditions in the primary care setting and social aspects of psychogeriatrics.[6]

On the other side of the Atlantic, development of geriatric psychiatry took a different course. Pioneering geriatric psychiatrists came on the scene in the 1970s, notably C. Eisdorfer, P. McHugh, M. Folstein, L. Jarvik, and M. Blumenthal as representative figures. They established some of the most influential centres in the US. In 1975, the National Institute of Mental Health established its Mental Disorders of Aging Branch. This was followed by the establishment of the American Association for Geriatric Psychiatry in the late 1970s. Another important milestone in the establishment of geriatric psychiatry in the US was the institution of board certification in 1981. In 1999, 2425 individuals were board certified in geriatric psychiatry.[7]

When it comes to service provision, the US system of care is shaped by two major forces: the patchwork of funding sources and their peculiar incentives; and a long tradition of entrepreneurialism by both individuals and institutions that provide services. Within the constraints of the funding system, institutional and individual providers find niches where they can deliver a service and be adequately paid for it. Unlike the British system that has a strong 'central planning' element, the US system sees links between components of the care system to arise 'from the bottom up'.[8]

The European Union does not recognise psychogeriatrics as an independent specialty. A survey of 30 European countries showed that only 11 (37%) had training programmes for geriatric psychiatrists. Only 10 (33%) required continuing professional development for psychogeriatricians at consultant level.[9]

Due to the common language, and often the pattern of medical training, Commonwealth countries such as Australia and New Zealand have generally been most enthusiastic in creating specialist old age psychiatry services. In South-East Asia, various regions and countries are at different stages of development of psychogeriatric service. Many have commenced development of such services in response to their rapidly ageing population over the last one to two decades.[10]

11.3 Current Trends for the Development of Mental Health Services for Elderly People

In 1996, the Division of Mental Health and Prevention of Substance Abuse of the World Health Organization, in collaboration with the World Psychiatric Association Section of Geriatric Psychiatry, issued a consensus statement on psychiatry in elderly people.[11] They agreed that a mental health service for elderly people should consist of the following components:

- family and social support;
- a primary healthcare team;
- a specialist old age psychiatry team;
- an inpatient unit;
- rehabilitation;
- day care;
- respite facilities;
- a range of residential care for people unable to live in their own homes;
- liaison with general medicine, or where available, geriatric medicine facilities;
- education of healthcare providers in all disciplines, particularly in the primary healthcare sector; and
- research addressing the prevalence of mental illness among elderly people, and evaluation of innovative projects

It can be seen that a comprehensive mental healthcare service for older people consists of a wide range of social institutions and facilities among which the specialist psychogeriatric team is only one component.

11.4 Psychogeriatric Services in Hong Kong

11.4.1 Historical Development

Psychogeriatrics as a psychiatric subspecialty is a recent concept. In the past, elderly people with psychiatric illnesses received treatment at facilities serving the general population. A study by Lo[12] showed that the government Mental Health Services had a very low utilisation rate by elderly people in the 1960s and 1970s. This was unlikely to be due to a generally low prevalence of major psychiatric disorders among the elderly population. A number of epidemiological studies showed that the prevalence

of two principal psychiatric conditions to affect older people, namely dementia and depression, was comparable with that of western studies.[13,14] The true reason for such a low utilisation rate must therefore lie elsewhere. It is quite likely that the uniform service offered to patients across all age-groups failed to address the needs of a large proportion of elderly patients with mental disorders. Furthermore, the stigma of psychiatric disorders was a deterrent to potential service users.

Psychogeriatrics started its development in the mental hospitals. Prior to the advent of modern psychiatric medication, people with mental illness were often confined in mental hospitals for very long periods, often for life. This led to considerable over-crowding and an increasing population of elderly patients who had spent much of their adult life in institutions. The introduction of effective medication has greatly improved the prognosis of patients with serious mental disorders, but the trend has not been entirely reversed. To facilitate care and management, these elderly patients were grouped in the same wards. Thus in the late 70s, two psychogeriatric wards were first established in Kowloon Hospital to provide care for these long-stay elderly patients. In 1981, three psychogeriatric wards were established in the then newly opened Kwai Chung Hospital.[15]

The first attempt to establish a community-based psychogeriatric service was made by the Psychiatric Department of the University of Hong Kong in 1984. In collaboration with the St. James' Settlement that provided supporting nursing staff and space, the Department established a pilot psychogeriatric assessment centre. This was a walk-in clinic that accepted patients from all over the territory. Patients were required to purchase their medication from nearby private dispensaries. Most patients were followed up at the clinic but a proportion was referred either to Queen Mary Hospital or to other clinics under the government Mental Health Services.[16]

In July 1991, the Chinese University of Hong Kong set up the first psychogeriatric team in Hong Kong to look after the Shatin catchment area. The team has since expanded and now provides a full range of psychogeriatric services to elderly people living in New Territories East and North District.

Systematic development of a psychogeriatric service in Hong Kong only began in 1994 when a Working Group on the Care for the Elderly recommended the formation of eight multidisciplinary Psychogeriatric Teams, each with a psychogeriatrician, psychiatric nurses, social worker, and clinical psychologists. This recommendation was adopted by the government and became the cornerstone of a territory-wide public-funded psychogeriatric service.

11.4.2 Current Situation

Although there exists private psychiatrists with a special interest in psychogeriatrics, systematic psychogeriatric care in Hong Kong remains largely a public-funded service. Currently, there are psychogeriatric teams based at all the Hospital Authority clusters, covering the whole of Hong Kong. These clusters all provide a range of psychogeriatric services to suit the needs of elderly patients. Nonetheless due to historical reasons, significant variation exists in the resources and magnitude of such cluster-based services. Psychogeriatric services available in the public sector will be further elaborated in the next section.

Psychogeriatrics is recognised as a psychiatric subspecialty for training purposes by the Hong Kong College of Psychiatrists. Current training comprises a 6-month module with one of the psychogeriatric teams. The College has also established a Clinical Division of Old Age Psychiatry; though at present it does not offer a distinct professional qualification specific for psychogeriatrics.

Within the Hospital Authority, psychogeriatrics is represented by a subject officer. Representatives from the various psychogeriatric teams constitute the Psychogeriatric Working Group under the Hospital Authority. It meets regularly to discuss service development and provision that is of common interest to all the psychogeriatric teams.[10]

Formed in 1998, the Hong Kong Psychogeriatric Association has rapidly developed into an organisation representing the various professions and individuals who are dedicated to improving the mental health of senior citizens in Hong Kong. It has become an important driving force that promotes inter-disciplinary / inter-sector collaboration, development of new service models, local research activities, and sharing of new knowledge on a regional basis.

11.4.3 The Multidisciplinary Team

Like geriatric medicine, psychogeriatrics is very much team work in which a number of disciplines contribute their skills towards enhancing the well-being of patients and their carers. Members of the multidisciplinary team include psychogeriatricians; psychiatric nurses working in inpatient, day-patient and community settings; occupational therapists; physiotherapists; medical social workers as well as clinical psychologists.

The specialist psychogeriatrician often serves as

coordinator, who has to facilitate constructive communication and resolve conflict. In this way, the team can work synergistically, towards common goals and offer the best to its clients.

11.5 Different Types of Psychogeriatric Services in the Public Sector in Hong Kong

11.5.1 Outpatient Service

The psychogeriatric outpatient service is similar to other specialist outpatient services provided by the Hospital Authority. Access to this service requires a medical referral. This is then followed by a triage process so that patients with more urgent problems receive higher priority and hence earlier review.

Common clinical conditions presenting to psychogeriatric outpatient clinics include depression, dementia, sleep disorders, and benzodiazepine dependence (often in the form of long-term use for past insomnia and anxiety symptoms).

11.5.2 Outreach Service

All the psychogeriatric teams provide an outreach service to public-funded elderly residential homes, and more recently, to private homes. Essentially, this is an outpatient service delivered to the home. Although different teams have slightly different protocols, the basic pattern is similar. Under this service, psychogeriatricians based at hospitals and psychiatric centres pay regular visits to designated residential homes. Other healthcare professionals also have an input to this programme with varying degrees of involvement. Hospitals and psychiatric centres provide clerical support, clinical investigations, and dispensary services.

The psychogeriatric outreach programme is very popular among patients, relatives, and residential home staff because it allows better communication and hence understanding by the different parties concerned. This results in better care of certain categories of patients, especially those who have cognitive impairment or communication difficulties. Patients who are disabled benefit as they are able to attend clinical appointments in a comfortable and familiar environment. This programme is also cost-effective for the Hospital Authority with a reduced burden on outpatient clinics, and for residential homes that have a reduced need for manpower for transportation and escort.[17]

11.5.3 Consultation Liaison Service

Elderly people often have multiple morbidities and many will have concurrent physical and mental disorders. In addition, many physical conditions and medications produce psychiatric symptoms. Psychiatric conditions such as affective disorders, anxiety disorders, and somatoform disorder can have physical symptoms that may conceal psychiatric manifestations. Many forms of physical illness, especially chronic conditions that cause great suffering and pain, often produce secondary psychiatric disorders such as depression. The latter may exert a negative effect on rehabilitation and recovery. Elderly patients with dementia or other forms of cognitive impairment are often unable to consent to medical / dental treatments and residential placement. It is often necessary to determine their ability to make such decisions. Finally, certain forms of behaviour — such as attempted suicide, elder abuse and abnormal illness behaviour — are complicated phenomena with biopsychosocial components that require systematic assessment. All these create a fertile ground for close collaboration between psychogeriatricians and geriatricians, as well as the development of consultation-liaison work in old age psychiatry. Thus, psychogeriatricians are often involved in the assessment of patients in medical and surgical wards of acute and extended care hospitals.

Studies have demonstrated the usefulness of psychogeriatric intervention in a medical setting. A randomised controlled trial of psychiatric liaison intervention for medical inpatients aged over 75 years showed that those in the intervention group had improved physical function, fewer readmissions to hospital or nursing home placement within 12 months of discharge, and a shorter length of stay.[18] A similar intervention was shown to be effective in bringing about recovery from depression in frail, older people living at home.[19]

11.5.4 Inpatient Care

Despite the emphasis on community-based care, there are times when patients become unmanageable in the community and thus require hospital admission. An inpatient psychogeriatric service thus constitutes an indispensable link in the entire spectrum of care for elderly people with psychiatric disorders.

Most patients require only a relatively short stay, in the range of weeks. Patients with depression, functional psychosis, and even behavioural and psychiatric symptoms of dementia mostly require acute admission for assessment and treatment. Patients who impose a high level of stress on carers may be admitted for respite care. Finally, there are patients with more complicated clinical problems who require a more intensive work-up in the inpatient setting. They too require short-term elective admission.

The Mental Health Ordinance[20] allows people with mental illnesses to be admitted to psychiatric hospitals or observation units for observation and

treatment either voluntarily or compulsorily. These psychiatric hospitals and observation units are designated in law for such purposes (the 'gazetted wards'). Admissions under the Mental Health Ordinance are called formal admissions. Meanwhile, people with mental illness can also be admitted to hospital just like any other patients. These are called informal admissions. Whenever possible, patients will be admitted informally, or formally but voluntarily to gazetted wards, so as to be treated in the least restrictive way possible. Nonetheless there are occasions when patients refuse admission, not knowing that they constitute a risk to themselves or other people. For their own well-being, they have to be admitted compulsorily to a gazetted ward.

Although most patients can be treated and discharged in a reasonably short period of time, some continue to stay for reasons that are not always clinical. These chronic patients, together with patients who have aged in the psychiatric hospital, constitute the main bulk of long-stay patients in the psychogeriatric setting. With the development of a community-based service, more and more residential homes are ready to admit long-stay psychiatric patients, enabling more to be discharged from the hospitals.

11.5.5 Elderly Suicide Prevention Programme

The high suicide rate of senior citizens has long been an issue of concern. In October 2002, the Hospital Authority launched a new service to provide a timely and user-friendly psychogeriatric service to elderly people who are depressed and at risk of committing suicide. This is a territory-wide service and accepts referral from a number of sources. These include private medical practitioners, general outpatient clinics, hotlines, community elderly services operated by non-governmental organisations, and the Social Welfare Department. It also accepts internal referrals from various clinical departments of the hospital and community nursing services. The service has a number of components.

The first component is a psychogeriatric fast track clinic (FTC) that offers initial assessment of elderly people referred to the Elderly Suicide Prevention Programme (ESPP) with minimal delay, so that the usual triage and waiting list of ordinary psychiatric outpatient service is by-passed.

For those referred who are reluctant to attend the FTC, due to worries about psychiatric stigma or the fees incurred, the ESPP provides a psychogeriatric nursing service known as early intervention service under which nurses will contact the referred elderly person and visit the latter if appropriate, to establish contact, assess the condition of the person in more detail, and try to motivate the patient to attend FTC.

Another important component of the ESPP is called telecheck. This is modelled after the successful experience in Italy of De Leo et al[21] that regular telephone contacts by healthcare professionals ('telecheck') and a hotline service to elderly people living alone in the community was associated with a marked decrease in suicide rate, not only on a short-term basis, but in the long term as well, as shown in a recent 10-year study.[22] Patients who are receiving service under the ESPP will receive regular phone calls from psychogeriatric nurses to monitor their condition, to provide support, and to arrange earlier appointments if necessary. This serves as an important adjunct to the conventional community psychiatric nursing service that provides regular home visits to these patients.

Apart from direct clinical work, the ESPP also has a strong educational component. A joint effort by the Hospital Authority and associations that represent private medical practitioners resulted in a programme of talks to medical practitioners in Hong Kong about suicide in elderly people, and the detection and management of depression. Additional educational programmes targeting healthcare and social service workers, as well as the general public are run at both cluster and central level. This educational component is based on the Gotland study[23] in which general practitioners on the Gotland Island in Sweden were trained in the detection and treatment of depression (for all age-groups). This was followed by a reduction in suicide rate. The reduction was nonetheless only temporary, indicating that a persistent effort is needed for a more lasting effect.

Compared with the rest of the world, the suicide rate among elderly people is relatively high among East Asian countries. As a large-scale comprehensive project to tackle the problem of suicide in elderly people, the ESPP is a pioneer in this region.[24]

11.5.6 Clinical Psychology Service

Clinical psychologists serve many important roles in a psychogeriatric context. They provide detailed neuropsychological assessment that is vital to the early and correct diagnosis of dementia and related conditions. They offer specific treatment and intervention for individuals, couples, families, and groups in relation to mood disorders and adjustment problems. Apart from older adults themselves, informal carers can also benefit from a psychotherapeutic input. Psychological expertise can be applied in behavioural management and care planning for an older adult and family and also for professional carers. Finally, psychologists also have an indispensable role in public education, promoting

a more balanced view towards ageing and the associated mental health problems.[25]

11.5.7 Other Services Provided in Some Clusters

11.5.7.1 Psychogeriatric Day Hospital / Day Patient Service

Although the psychogeriatric day hospital plays a major role in the assessment process[26] and some have used it for treatment of acute illness,[3] it is most usually used as a longer-term support for the most vulnerable and to offer respite for carers.[27] To fulfil such diverse roles, a psychogeriatric day hospital / service requires a team of experienced professionals, offering physical and mental rehabilitative work in the form of comprehensive disability and ability assessment, treatment planning, psychotherapy, behavioural intervention, skills training, physiotherapy, and community orientation.

There has been debate over the usefulness of the psychogeriatric day hospital.[28,29] Although some of their functions may be shared by day centres, other functions such as clinical assessment and the care of patients with serious functional psychiatric disorders (such as resistant depression, residual psychotic symptoms in patients with chronic schizophrenia or delusional disorder) are clearly beyond the scope of the latter. In the face of challenge from alternative services that are less capital expensive, further research is needed to clarify the role of the psychogeriatric day hospital and how they may be most effectively used.[30]

11.5.7.2 Memory Clinics

Compared with conventional psychiatric and geriatric clinics, memory clinics offer a number of advantages in the management of people with dementia and related disorders. First, they do not carry the clinical stigma associated with psychiatry and the ageist stigma associated with geriatric medicine. Second, the name itself clearly spells out the purpose of the clinic, thus facilitating accurate referral.

Memory clinics now operate in a number of clusters. Some have been established by geriatric services, others by psychogeriatric services and some jointly operated by psychogeriatric and geriatric services.[31] Even though there are marked differences in the models adopted, there are certain similarities. These include:

- thorough and systematic assessment, often using standardised and validated tools;
- use of more vigorous diagnostic and staging criteria;
- use of established management protocols and guidelines;
- multidisciplinary participation; and
- systematic evaluation of the effects of intervention

Memory clinics have been shown to significantly improve the quality of life for carers of people with dementia because of the treatment and advice they offer.[32] For the staff, memory clinics offer opportunities for sharing of skills, knowledge, and perspectives among the different disciplines involved. They also provide useful experience for medical, psychiatric, and family medicine trainees.

11.6 Future Trends

To meet the challenge of a rapidly ageing population and increasing demand for better quality of life, Jeste and Palmer[33] have advocated a new positive psychiatry of ageing. This model would focus not simply on symptom relief, but on recovery and promotion of successful ageing. It would view old age not as a period of inevitable biopsychosocial decline, but as one with continued potential for regeneration. In addition, this new approach would not only incorporate principles of prevention but also employ interventions to enhance positive psychological traits including resilience, optimism, social engagement, and wisdom. They argue that this new model will not only improve patient outcome, but reduce healthcare costs.[33]

As a young subspecialty in psychiatry, psychogeriatrics has a lot of potential for change and growth. New technologies in diagnosing and treating psychiatric disorders, public expectations as well as financial constraints due to a rapidly expanding elderly patient population and a shrinking workforce will be the forces that determine the future service model.

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CHAPTER 12

Orthogeriatrics and Fragility Fracture in Old Age

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OBJECTIVE

To elucidate the role of geriatrics in the management of fragility fracture in elderly people. Pre- and post-operative care, rehabilitation and discharge planning, aftercare, and secondary prevention of fractures are discussed.

KEY POINTS

- Fragility fracture in older people can be considered a prototype of geriatric syndrome. It is often associated with neurocardiac instability, frailty, sarcopenia, malnutrition, dementia, and inappropriate medication. Despite surgery for hip fracture, half of all patients are unable to regain their pre-fracture functional state. Hip fracture is associated with rates of high mortality and institutionalisation in older adults.
- Falls and fractures occur in older adults with different risk profiles and care needs — more active older adults with compromised health experience outdoor falls, frail community-dwelling adults experience indoor falls. Long-term care facility-dwelling patients who have multiple comorbidities usually have a complicated hospital course and poorer rehabilitation potential.
- Orthogeriatric collaborative care is associated with a significant reduction in in-hospital mortality, long-term mortality, length of hospital stay, and shorter time to surgery. It is also cost-effective in some countries. Thus patients with hip fracture should be offered a comprehensive orthogeriatric hip programme from admission. The organisation of the orthogeriatric model should be based on the availability of resources.
- Orthogeriatric care should offer:
  - orthogeriatric assessment for rapid optimisation of fitness for surgery on the day of, or the day after, admission;
  - early identification of individual goals for multidisciplinary rehabilitation to recover mobility and independence, and to facilitate return to pre-fracture residence and long-term well-being;
  - continued coordinated orthogeriatric and multidisciplinary review, including adequate analgesia, monitoring of postoperative complications, nutritional augmentation, early mobilisation and rehabilitation;
  - multifactorial risk assessment to identify and address future fall risk and bone health; and
  - early supported discharge planning with integration of related services, particularly mental health, falls prevention, bone health, primary care, and social services.

12.1 Introduction

In western countries, the incidence rate of fragility fractures increased with population ageing over the last decade and then declined from a plateau observed around the year 2000. A similar trend has been observed from the Hong Kong Hospital Authority data that could account for 98% of all local hospital admissions for hip fracture.[1] The age-adjusted incidence rate of hip fracture among the population aged ≥65 years in Hong Kong (per 100 000 population) decreased from 381.6 for men and 853.3 for women in 2001 to 341.7 and 703.1, respectively, in 2009.[1] Such a decline in this trend of incidence rate could be related to increasing use of bisphosphonates, provision of fall services, and / or promotion of physical exercise or Tai Chi.[1] The decline in incidence, however, pre-dated the increasing use of osteoporosis drugs and implementation of falls prevention programmes. Thus other factors to account for the incidence decline have yet to be explored, including reduced exposure to unfavourable early life environment (e.g. smoking, inactivity), that impact on subsequent bone health.[1]

Osteoporosis, the underlying condition that

precipitates fragility fracture, is a lifelong process but asymptomatic at its onset. It progresses from Colles' fracture and vertebral fractures to hip fracture with consequent major distress, dependency, and disability.[2] This notion is supported by data from a population-based retrospective cohort study in which patients with newly diagnosed distal radius fracture had a 5.6-fold increased risk of subsequent hip fracture within 1 year.[3] Subsequent fractures may occur within a short time of the first fracture.[4] In a study of 184 nursing home residents with hip fracture, 6% of residents experienced a subsequent fracture (leg, arm, or second hip) within 6 months, 12% within 1 year, and 21% within 5 years.[5] Fractured hip in older adults is associated with significant morbidity, functional decline, and mortality. From the National Hip Fracture Database 2013 Report that provides information on 61 508 cases with hip fracture from 180 hospitals across England, Wales, and Northern Ireland, 3.5% of patients developed pressure ulcers, 46.2% returned home at 30 days, and 8.2% died within 30 days.[6] Another research shows that 50% of patients recover their pre-fracture walking ability by 6 months, but after 1 year, less than 50% of patients are able to walk unaided, and only 40% are able to perform all activities of daily living independently.[7] Mortality from a second fracture is high in nursing home residents, with 31% of residents dying within 1 year and 60.1% within 5 years of a baseline hip fracture.[5]

Fragility fracture can be considered a prototype geriatric illness. Falls and fractures occur in older adults with different risk profiles and care needs — active adults with compromised health status experience outdoor falls, frail community-dwelling adults experience indoor falls, and patients from long-term care institutions have multiple comorbidities and a complicated hospital course.[8,9] Like other geriatric syndromes, the presentation of hip fracture is sometimes atypical, the diagnosis may be missed or delayed when the patient cannot report the fall incident or pain, or in a minority of cases, hip X-ray does not reveal a fracture. It frequently leads to pain, immobilisation, and other medical and surgical complications such as thromboembolism, infection, gastrointestinal bleeding, and delirium in the perioperative period. Any delay in the care process will likely harm the patient and add to the cost of care. Patients with hip fracture require high-quality care that requires a systematic, well-coordinated multidisciplinary approach. Key elements of a strategy for osteoporotic fractures aim to optimise the patients' condition to facilitate early surgical treatment, early mobilisation and rehabilitation, and prompt detection and treatment of any postoperative complications. In addition, the higher risk of developing a second fracture after a fragility fracture provides the basis of aftercare — a falls prevention programme and treatment for osteoporosis.

12.2 The Evolution of Orthogeriatric Care

Geriatrician Dr Bobby Irvine and orthopaedic surgeon Dr Michael Devas were the pioneers of orthogeriatrics in Hastings in 1959.[10] They emphasised the postoperative rehabilitation phase that addressed the needs of older people with hip fracture, and day-to-day care was provided by a geriatrician, supported by nurses and therapists. The orthopaedic consultant contributed to goal setting at a weekly postoperative multidisciplinary meeting. Since then different models of orthogeriatric care have been described (Table 12.1).[11,12] Models vary considerably in terms of site of care (orthopaedic or geriatric / rehabilitation or dedicated orthogeriatric ward), leadership (orthopaedic surgeon or geriatrician; multidisciplinary or shared responsibility by orthopaedics and geriatrics), and timing of care (preoperative or postoperative; early rehabilitation or late rehabilitation). Such variations depend on manpower availability and funding. The benefits of orthogeriatric collaboration in patients with hip fracture have been supported by systematic review and meta-analysis. Orthogeriatric collaboration is

Table 12.1 Models of orthogeriatric care[11,12]

Model	Description
Reactive consultation	Patients referred to a geriatrician or other physician on an ad-hoc basis. Medical management and rehabilitation is managed primarily by the orthopaedic team. Patients are not necessarily reviewed by a geriatrician. No integration or shared responsibility.
Regular geriatrician liaison visits to orthopaedic ward	Regular liaison visit to orthopaedic ward by a geriatrician or nursing specialist (hip fracture nurse) for ward rounds and multidisciplinary rehabilitation meetings. The geriatrician or specialist nurse is accompanied by a member of the orthopaedic team to ensure continuity of care. Some patients are reviewed preoperatively.
Shared orthopaedic and geriatric care on a dedicated orthogeriatric ward	It enables easy collaboration and communication between specialists and minimises disruptive ward moves for patients. Preoperative assessment by geriatricians, specialist staff such as hip fracture nurses, early rehabilitation, postoperative multidisciplinary care are all high-quality features.
Rehabilitation in a geriatric ward postoperatively	Perioperative care is provided by the orthopaedic team. Patients are transferred to a geriatric ward for rehabilitation. The break in continuity of care and change in ward environment are potentially disadvantageous, especially for patients susceptible to postoperative delirium. Geriatric input may be delayed until transfer to the rehabilitation ward. The orthopaedic consultant gives advice on postoperative rehabilitation.

associated with significantly reduced in-hospital mortality, long-term mortality, length of stay, and shorter time to surgery.[13,14] The shared care model appears to be more effective in reducing the length of stay and time to surgery. In a cost-utility analysis in Israel, the comprehensive orthogeriatric care model was more cost-effective (23% fewer resources per patient: US$14 919 vs. US$19 363), and provided additional quality-adjusted life years (avert 0.226 additional disability-adjusted life years per patient as a result of a lower 1-year mortality rate in patients who received orthogeriatric care) compared with a standard care model.[15]

While the effective components of an orthogeriatric care have not been defined, different professionals working in close cooperation and adopting a comprehensive geriatric assessment (CGA) has been shown to have beneficial outcome over usual care in the management of elderly patients with fragility hip fractures in terms of reducing the related complications, mortality, and disability.[16] CGA of an elderly patient with hip fragility fracture should include the domains of pre-fracture functional status, clinical assessment, comorbidities, nutritional status, cognition and behaviour, and social support. Preoperatively, CGA should identify the comorbidities that need to be treated and stabilised to reduce surgical risk. Postoperatively, CGA should detect and manage medical complications, initiate early mobilisation to reduce early mortality and prevent cognitive and functional decline. Before discharge, the orthogeriatric team should draw up an individualised discharge and rehabilitation plan to promote the patient's functional recovery and to prevent further fragility fractures through fall risk assessment and rehabilitation and measures to improve bone quality and reduce fall risk. Nutritional support and pain treatment are important throughout the different stages of an orthogeriatric care.[16]

In 2011, the National Institute for Health and Care Excellence (NICE) published 'The management of hip fracture in adults' along with a series of implementation tools and resources, with evidence updated in 2013.[17] In 2012, they published a set of quality standards for hip fracture (NICE QS16).[18] They suggest that patients admitted with hip fracture should be offered a formal hip fracture programme that includes:

- preoperative orthogeriatric assessment;
- prompt and effective pain management throughout hospital stay;
- rapid optimisation of fitness for surgery on the day of, or the day after, admission;
- optimisation of comorbidities and complications immediately so that surgery is not delayed by anaemia, anticoagulation, volume depletion, electrolyte imbalance, uncontrolled diabetes, uncontrolled heart failure, correctable cardiac arrhythmia or ischaemia, chest infection, or exacerbation of chronic chest conditions;
- schedule surgery for hip fracture on a planned trauma list, with supervision by a consultant or senior staff member;
- cemented arthroplasty for displaced intracapsular fracture, with the offer of total hip replacement if clinically eligible;
- extramedullary implants such as a sliding hip screw in preference to an intramedullary nail for trochanteric fractures above and including the lesser trochanter (AO classification types A1 and A2);
- early identification of individual goals for multidisciplinary rehabilitation to recover mobility and independence, and to facilitate return to pre-fracture residence and long-term well-being;
- continued coordinated orthogeriatric and multidisciplinary review to detect and manage postoperative complications and optimise the rehabilitation progress;
- early physiotherapy assessment and, unless medically or surgically contraindicated, mobilisation on the day after surgery;
- secondary prevention of fracture through a multifactorial risk assessment to identify and address future fall risk, and a bone health assessment to identify and manage osteoporosis before discharge from hospital;
- early supported discharge through liaison with or integration of related services, particularly mental health, falls prevention, bone health, primary care, and social services; and
- the hip fracture programme team retains a comprehensive and continuing clinical and service governance lead for all stages of the pathway of care, including the policies and criteria for both immediate care and early supported discharge

12.3 Preoperative Assessment and Care

An assessment by the orthogeriatrician-led hip fracture programme will identify individual goals early and guide the appropriate overall management approach, identify patients at risk of perioperative complications, and provide targeted interventions. Since patients with hip fracture vary in their baseline functional and comorbid status, it is important to assess the patients' pre-fracture functional and mobility states. Professionals with substantial involvement in the preoperative assessment include orthopaedic surgeons, nurses, orthogeriatricians, and anaesthetists. The goal of surgery for hip fracture is not just pain relief, but also for patients to regain their pre-fracture state and long-term well-being. Three categories of hip fracture patients can be identified:
(1) patient with good premorbid state — surgery

should be performed on the day of admission if their medical condition permits, and early postoperative mobilisation should begin within 48 hours of surgery;

(2) patient with poor premorbid status or unstable perioperative conditions — an orthogeriatrician should attempt to optimise the patient for surgery within 24-36 hours and early postoperative mobilisation should begin within 48 hours of surgery; and

(3) patient with poor premorbid status or who is unfit for surgery — the goal is to provide supportive and / or palliative care

Any delay in surgery will increase the risk of the complications of immobility, length of stay, and the often-repeated starvation of the patient. The goal of preoperative orthogeriatric assessment is to identify comorbidities and risk factors for perioperative complications, and to prepare and optimise elderly patients for early surgery (targeting door-to-theatre time within 24-48 hours). Consultation with other medical specialists may delay surgery and can be reduced. A preoperative comprehensive medical assessment comprises an evaluation of the causes of fall (circumstances and symptoms), a review of medication, pre-fracture mobility and functional states, and the level of social support. Physical examination may identify causes of falls such as Parkinsonism, stroke, or postural hypotension. Mental state examination may identify cognitive impairment with an associated risk of postoperative delirium and / or poor rehabilitation potential. The orthogeriatrician, in close collaboration with the anaesthetist, should ensure an accurate diagnosis and management of all conditions concurrent with the hip fracture, such as uncontrolled diabetes, chronic lung disease, congestive heart failure, arrhythmia, sepsis, anaemia, electrolyte disturbance, and stroke. For patients who have already developed complications, surgery should be postponed until their medical conditions are optimised. Other preoperative interventions that have been shown to be effective are listed as below.[19-21]

- A standardised tool to estimate perioperative cardiovascular risk — the revised Goldman Cardiac Risk Index.[22] For the patient with a cardiac murmur that is asymptomatic but haemodynamically significant, preoperative echocardiogram is not necessary for non-cardiac surgery. The absence of echocardiography should not lead to delay in fracture surgery. Patients with a high cardiovascular risk will benefit from beta-blocker therapy.
- Preparation of the patient to achieve a target international normalised ratio of ≤1.5 in those prescribed anticoagulation therapy. Use of oral low-dose vitamin K can safely and effectively reverse the effect of anticoagulation therapy.
- Pressure-gradient stockings as soon as possible after admission.
- A heel pad or heel elevation device to prevent pressure ulcers at the heel.
- Venous thromboembolism prophylaxis with low-molecular-weight heparin, or mechanical devices for those in whom anticoagulants are contraindicated.
- Lung expansion manoeuvres, such as incentive spirometry, to reduce postoperative pulmonary complications such as atelectasis and / or pneumonia.
- Nutritional augmentation.
- Transfusion to maintain haemoglobin level up to 10 g/dL.
- Pulse oximetry assessment from the time of emergency admission to 48 hours after surgery, and administration of oxygen as necessary to maintain oxygen saturation at ≥90%.
- Adequate analgesia to allow early mobilisation.
- Identification of the type of diabetes, previous complications, and the patient's usual anti-diabetic medication. Excessive hyperglycaemia or hypoglycaemia should be avoided prior to surgery.
- Antibiotic prophylaxis, given at induction of anaesthesia, to prevent superficial and deep wound infection.
- Regional anaesthesia for most patients as it may lower the risk of acute postoperative confusion.
- Avoid indwelling urinary catheterisation where possible as it increases the risk of urinary tract infection.
- Delirium prevention using a structured protocol.

12.4 Postoperative Assessment and Care

After surgery, continuous, coordinated, orthogeriatric, and multidisciplinary review is important. The rehabilitation progress of the patient and their care plan should be regularly reappraised through multidisciplinary meetings. Older adults are prone to exacerbation of pre-existing chronic illnesses, such as retention of urine in benign prostatic hyperplasia, fluid and electrolyte disturbances in renal or heart failure, metabolic complications in diabetes, flare of gouty arthritis, and nosocomial pneumonia in chronic lung disease. An orthogeriatrician should focus on early detection and appropriate management of these complications to allow early rehabilitation. Regular medication should be scrutinised to identify inappropriate medications that will increase the risk of fall (antihypertensives, antipsychotics, antidepressants, benzodiazepines) or delirium, and drug-drug interactions. Examples of complications and their management approach are listed in Table 12.2.[23-27]

Table 12.2 Postoperative complications and their management[23-27]

Complication	Management approach
Pain	• Early use of intramuscular or oral opiates in the postoperative period. • Regular use of paracetamol, with or without codeine phosphate or tramadol in all patients. • Avoid use of non-steroidal anti-inflammatory drugs because of their gastrointestinal toxicity in older adults and their adverse effects on fracture healing.
Wound infection	• Close monitoring for wound infection. Superficial wound infection refers to infection of the wound that does not extend below the deep fascia layer. It can be treated with antibiotics, and if indicated, surgical debridement. • Deep wound infection often involves the implant and necessitates removal (Girdlestone excision arthroplasty).
Pressure ulcer	• One-third of hip fracture patients will develop pressure sores. • Rest all patients on pressure-reducing surfaces — from hospital admission, in transit, in theatre, and in the ward. • Assess pressure areas at risk regularly on admission and at least twice a day while patients remain immobile. • Address the risk factors for pressure sore development such as pressure, shearing forces, friction, incontinence, pain, and malnutrition. • Refer to wound care nursing expert for management of early or superficial skin damage. • Encourage early mobilisation if the patient's condition allows.
Venous thromboembolism	• Clinically apparent incidences of deep vein thrombosis and pulmonary embolism are 3% and 1%, respectively. • Anti-embolic stockings for all patients unless contraindicated (gross oedema, leg deformity, peripheral vascular disease, peripheral neuropathy). • Balance the risks of bleeding and thromboembolism. • Pharmacological thromboprophylaxis (fondaparinux or low-molecular-weight heparin) if no contraindications — allergy to heparin, on oral anticoagulation with international normalised ratio of >2.0, thrombocytopenia (platelet <50 x 10^9 /L), known bleeding disorder, evidence of active bleeding, uncontrolled hypertension (blood pressure >230/120 mm Hg), lumbar puncture / epidural spinal analgesia expected within next 12 hours or performed within last 4 hours, new stroke (ischaemic or haemorrhagic). • Intermittent pneumatic compression device if pharmacological thromboprophylaxis is contraindicated. • Avoid over-transfusion.
Malnutrition	• Nutritional assessment as part of the routine nursing care. • Early referral to a dietician for specialist advice for patients with suboptimal nutritional intake. • Simple practical measures such as providing additional carers to assist in nutrition and feeding.
Delirium	• Delirium after surgery for hip fracture is common (up to 35%).[23] The risk is higher in patients with trauma than elective surgery.[24] • Identify risk factors for postoperative delirium[25,26]: o Non-modifiable (age, cognitive impairment, dementia, depression, comorbid diseases such as renal failure, type / severity of surgery). o Modifiable (general anaesthesia, infection and inflammation, metabolic disturbances such as anaemia, hypoxaemia, hypotension, medications such as anticholinergic / neuroleptics, pain and discomfort such as urinary catheter, sleep disruption). • Prevent postoperative delirium based on structured protocol[25,27]: o Ensure adequate central nervous system oxygen delivery (ensure oxygen saturation 90-95%, transfuse to keep haematocrit >30%, maintain systolic blood pressure >90 mm Hg) o Maintain fluid / electrolyte balance o Ensure adequate nutritional intake o Adequate pain control o Reduce or stop unnecessary medications (benzodiazepines, anticholinergics, antihistamines, or drugs with significant interactions or adverse effects) o Monitor bladder / bowel function (remove urinary catheter by postoperative day 2 and screen for retention of urine) o Encourage early mobilisation and rehabilitation on postoperative day 1 o Provide appropriate environmental stimuli (access to spectacles and hearing aids for those dependent on them, good nursing care with regular patient orientation to time, place, and person)
Anaemia	• There is insufficient clinical evidence to guide the blood transfusion after hip fracture surgery. • Overtransfusion may increase the risk of thromboembolism.

12.5 Rehabilitation

An early discharge to a rehabilitation environment should be considered once the patient is medically stable, able to transfer and mobilise for a short distance, and has the mental ability to participate in continued rehabilitation but not yet achieved his / her full potential. Although evidence supporting a specific type or venue for rehabilitation after hip fracture is lacking, rehabilitation can improve mobility and balance, increase independence, and reduce the need for a care home and / or social care. Multidisciplinary care is central to the management of frail older people with multiple medical, psychological, and social needs. The team may comprise an orthogeriatrician, nurses, physiotherapists, occupational therapists, and a social worker. The principles of rehabilitation rely on coordinated, formalised integration of all members of the multidisciplinary team and involvement of the patient and carer in goal setting. Regular multidisciplinary team meetings are essential for updating patient rehabilitation progress and reviewing goals and discharge planning. For patients with hip fracture complicating a life-threatening illness, the care goals could include minimising pain and relieving other symptoms with attention paid to the patients' wishes regarding end-of-life care. The orthogeriatrician remains the leader of the

team throughout the rehabilitation process until the patient is discharged. It is important to know the key carer's wishes for provision of ongoing post-discharge support for the patient. Strong liaison between hospital and community services that provide rehabilitation and social support is in the best interests of the patient to maintain his / her functional state and reduce the chance of inappropriate hospital re-admission. The key to early supported discharge is continuity of care across various care services and settings. Hurdles, such as fragmented social support systems and access to transportation, should be minimised. Further outpatient rehabilitation can take place in a geriatric day hospital, outpatient department, or in the form of home physiotherapy based on the patient's goals and social situation.

12.6 Aftercare and Secondary Prevention of Fragility Fracture

The occurrence of the first fragility fracture reveals underlying osteoporosis, functional decline, and a higher risk of recurrent fall, fractures, and fatality. It provides an opportunity to prevent further fractures. Standards and tools are suggested for implementation of secondary fracture prevention[28,29], including:

- identification of patients with fractures;
- post-fracture evaluation — identification of secondary causes of osteoporosis, multifaceted health and lifestyle risk assessment, falls risk assessment, medication review, risk assessment with dual-energy X-ray absorptiometry (DEXA), and vertebral fracture risk assessment;
- interventions — calcium and vitamin D supplementation, specific pharmacotherapy for osteoporosis, long-term medication adherence, education, and falls prevention; and
- implementation of the Fracture Liaison Services that aim to enhance communication between the hospital, primary care physicians, and other community services through a care pathway

The effect of medication review on prevention of secondary fracture is lacking. In a randomised controlled trial of 199 patients aged >65 years with hip fracture, suggestions for medication change conveyed orally and in written form to hospital physicians during hospital stay, and to general practitioners after discharge, were shown to increase treatment with fracture-preventing drugs (calcium, vitamin D; 77% vs. 58%; P=0.01) and bone-active drugs (bisphosphonates, selective oestrogen receptor modulator [SERM], strontium ranelate, and parathyroid hormone [PTH]; 29% vs. 15%, P=0.04) after 12 months.[30] Nonetheless, such interventions did not affect the number of fall risk-increasing drugs (e.g. antihypertensives, diuretics, antipsychotics, antidepressants, and sedatives) or other secondary endpoints such as falls, fractures, and death.

12.6.1 Lifestyle Measures

All postmenopausal women with osteoporosis should receive adequate calcium and vitamin D. Other important lifestyle measures include exercise, smoking cessation, counselling on falls prevention, and avoidance of heavy alcohol consumption. Postmenopausal women should take adequate supplemental elemental calcium (500-1000 mg/day), such that their total calcium intake (diet plus supplements) approximates 1200 mg/day. Supplemental 800 IU vitamin D daily is recommended. Vitamin D and calcium supplements have been used as concomitant therapy in nearly all osteoporosis clinical trials. The RECORD trial that randomised 5292 previously mobile elderly people (aged ≥70 years) with recent clinical fracture to vitamin D alone, calcium plus vitamin D or placebo, showed no difference in secondary fracture rates or mortality.[31] Nonetheless, compliance with medication was only moderate (dropped to 63% after 2 years) in the RECORD trial and vitamin D status was assessed in only a small sample (1.1%) of the study population.[32] For patients in long-term care facilities, a meta-analysis of pooled data from 12 875 patients in seven clinical trials revealed that vitamin D supplementation was the intervention for which there was the strongest evidence for reduction of hip fractures (odds ratio=0.86), compared with other interventions such as hip protectors, exercise or multimodal interventions, fluoride, or sunlight exposure.[33] In a systematic review of 29 randomised trials (with a sample of almost 64 000 participants) that assessed all fractures and changes in bone mineral density in individuals aged ≥50 years, calcium supplementation, or calcium with vitamin D, effectively prevented osteoporotic fractures.[34] The authors showed that, in a subpopulation with a compliance rate of at least 80%, the level of risk reduction was doubled compared with those with poor compliance.[34] The recommended minimum daily dose of 1200 mg calcium or 800 IU vitamin D proposed in this systematic review is consistent with previous reports that 400 IU per day vitamin D is insufficient for fracture prevention, and that doses of 700-800 IU per day are needed to reduce the risk of hip and non-vertebral fracture in ambulatory or institutionalised women.[34,35]

12.6.2 Pharmacologic Therapy

Patients with fragility fracture are regarded as having severe osteoporosis and at high risk of subsequent fracture. The indications for pharmacologic treatment for secondary prevention of fracture include[36]:

- aged ≥75 years without the need for a DEXA scan;
- aged 65-74 years if osteoporosis is confirmed by DEXA scan (T-score of ≤ –2.5); and
- aged <65 years if T-score is ≤ –3.0, or T-score is -2.5 with one or more additional age-independent risk factors

Table 12.3 Strength of evidence that pharmacotherapy can reduce the risk of different types of fracture in women with postmenopausal osteoporosis*[38]

Agent	Fracture skeletal site			
	Vertebral	Non-vertebral	Hip	Wrist
Raloxifene	H	I	I	I
Alendronate	H	H	H	L
Risedronate	H	H	H	L
Zoledronic acid	H	H	H	I
Ibandronate	H	M	L	I
Denosumab	H	H	H	I
Teriparatide	H	M	L	I

* Strength of evidence: I = insufficient strength; L = low strength of evidence; M = moderate strength of evidence; H = high strength of evidence

A comprehensive meta-analysis of therapies for postmenopausal osteoporosis suggests that a 50% reduction in all skeletal sites can be achieved during 3 years of pharmacotherapy (e.g. alendronate).[37] In the absence of high-quality head-to-head drug comparison trials to determine the relative efficacy of the individual drugs, choice of therapy should be individualised and based upon efficacy, safety, cost, convenience, and other patient-related factors. The effectiveness and safety profiles of each treatment for osteoporosis (bisphosphonates, PTH, denosumab, oestrogen or oestrogen plus progestin, SERM) are updated in a systematic review (Table 12.3).[38] Bisphosphonates should be considered as first-line therapy because of their efficacy, favourable cost, and the availability of long-term safety data. There is strong evidence that bisphosphonates achieve a 35% reduction in secondary fractures after hip fracture regardless of bone density and a 20-28% reduction in mortality.[39] Oral alendronate is available as a generic preparation and is inexpensive. For those who cannot tolerate oral alendronate because of its gastrointestinal adverse effects, intravenous zoledronic acid can be used as an alternative and has been shown to reduce vertebral and hip fractures. Ibandronate is also available in an intravenous form although no direct hip fracture prevention data are available.

PTH is an anabolic agent. In contrast to antiresorptive agents, PTH stimulates bone formation and activates bone remodelling. Teriparatide reduces the risk of vertebral and non-vertebral fractures. It is used as second-line therapy in postmenopausal women or men with severe osteoporosis and fragility fracture or in patients in whom other osteoporosis therapies have failed. There is no consensus on the optimal use of denosumab. It can be used as initial therapy in certain patients at high risk of fracture, such as older patients who have difficulty with dosing requirements of oral bisphosphonates or who are intolerant of or unresponsive to other therapies, and in those with impaired renal function. The antiresorptive effects of SERM (e.g. raloxifene) are less than those of bisphosphonates. SERM should be reserved for women who cannot tolerate bisphosphonates or for women with osteoporosis and increased risk of invasive breast cancer. Strontium ranelate has been approved in Europe since 2004, but is not approved by the US Food and Drug Administration for the treatment of postmenopausal osteoporosis. In 2013, the European Medicines Agency recommended that use of strontium ranelate be restricted because of concerns about the risk of adverse cardiac events.[40]

12.6.3 Falls Prevention

Multifactorial falls prevention programmes have been shown by systematic reviews to be effective in reducing the incidence and severity of falls in the community,[41] and in care facilities and in a hospital setting.[42] Effective falls prevention interventions may include periodic medication reviews to eliminate unnecessary medications (especially psychoactive drugs), treatment of postural hypotension and other cardiovascular disorders, correction of visual problems, provision of appropriate assistive devices and physical therapies such as gait, strength, and balance training. The prevention of falls should also include assessment and management of environmental hazards.

12.7 Orthogeriatrics Moving Forward

The role of the orthogeriatrician has grown over the last few years. Their role has expanded from one of primarily involvement in the care and management of fragility hip fractures, to provision of specialist care for patients admitted with fracture at other sites such as spine, pelvis, appendicular and those who have major trauma. There is also an increasing role for the orthogeriatrician to optimise the preoperative care of patients undergoing elective joint and spine surgery.[43]

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CHAPTER 13

ONCOGERIATRICS

Tak-kwan KONG

OBJECTIVE

To review the clinical features and management issues unique to elderly persons with cancers, and update the current model of care in clinical oncology for geriatric patients.

KEY POINTS

- There are multiple challenges in managing cancer in old age: diagnostic uncertainty because of multiple pathologies, multiple aetiologies, and conditions that mimic cancer; treatment uncertainty because of a lack of evidence base on risk-benefit; ethical issues related to 'undertreatment' or 'overtreatment'; and the need for sensitive and compassionate communication with elderly patients and their families.
- Cancer treatment decisions should be based on biological age that takes into account the individual's functioning and reserve, rather than chronological age. An elderly person's biological age is best estimated by comprehensive geriatric assessment (CGA).
- Clinical trials of CGA in oncology have demonstrated benefits in recognising unsuspected geriatric problems and syndromes, predicting treatment outcome and survival, influencing treatment choice and intensity, and in implementing a multidisciplinary intervention plan.
- The goals of treatment of cancer in an elderly person should include cure if possible, prolongation of survival, prolongation of active life expectancy, symptom management, sympathetic care and support, and preservation of quality of life.
- An elderly person with cancer will benefit from individualised care based on a CGA and the input of a geriatrician.
- Collaboration between oncologists and geriatricians has emerged in the past decade as a clinical model that integrates geriatric principles and assessment into oncology care of elderly persons with cancer.

13.1 Introduction

Cancer has become a disease of elderly people because of population ageing and the higher risk of cancer in old age. It is a leading cause of mortality and morbidity in elderly persons. Cancer statistics from 2013 in Hong Kong revealed that over 60% of cancers occurred in people over the age of 60; gender-specific incidence of all cancers increased with age (Figure 13.1).[1] Most common cancers show an age-related rise in incidence rate (Figure 13.2).[1,2] In Hong Kong, one-third of all deaths in 2013 were due to cancer; and three-quarters of cancer deaths occurred in persons aged ≥60 years.[1] The median age at death due to cancer was 73 years in men and 74 years in women.[1]

Despite the growing importance and impact of cancer in old age, studies have shown that elderly persons with cancer are under-represented in clinical trials and undertreated.[3-5] Thus, breast cancers, small cell carcinoma of lung, and non-Hodgkin's lymphoma remain untreated or undertreated, despite the documented benefits of cancer treatment among elderly patients.[6-8] There is a great need to deliver qualitative cancer care for older persons, as recommended by international professional societies.[9-11]

A 60-year-old man or woman in Hong Kong in 2016 can expect to live another 23.9 and 29.0 years, respectively.[12] Contrary to the common myth that an 80-year-old person has 'fulfilled one's life expectancy', a man or woman who survives to age 80 can expect to live another average 9.4 and 12.3 years, respectively.[12] Many elderly patients prefer early diagnosis and more complete treatment of their cancer so that their remaining years of life can be well spent. Study has shown that the majority of chronically ill older

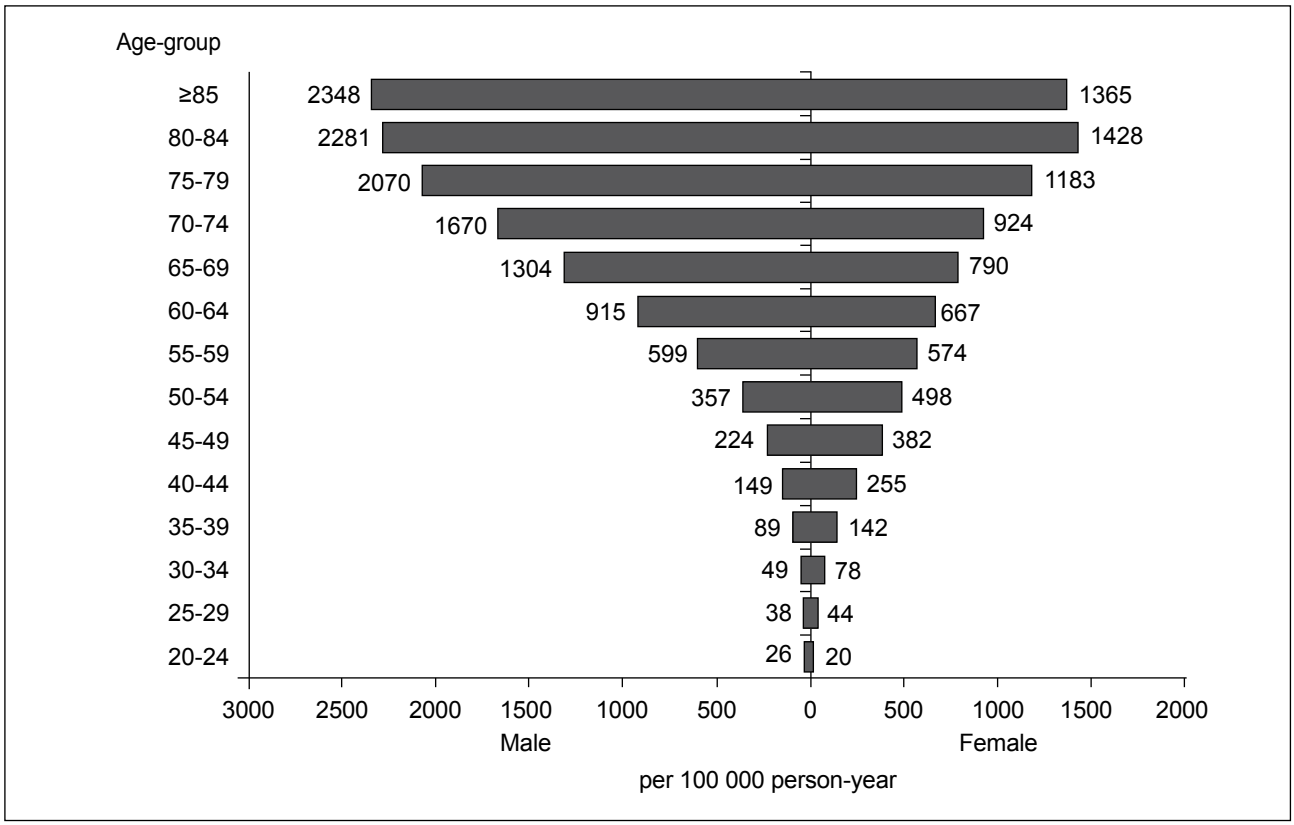

Figure 13.1 Age- and gender-specific incidence rate of cancers in 2013 for Hong Kong[1]

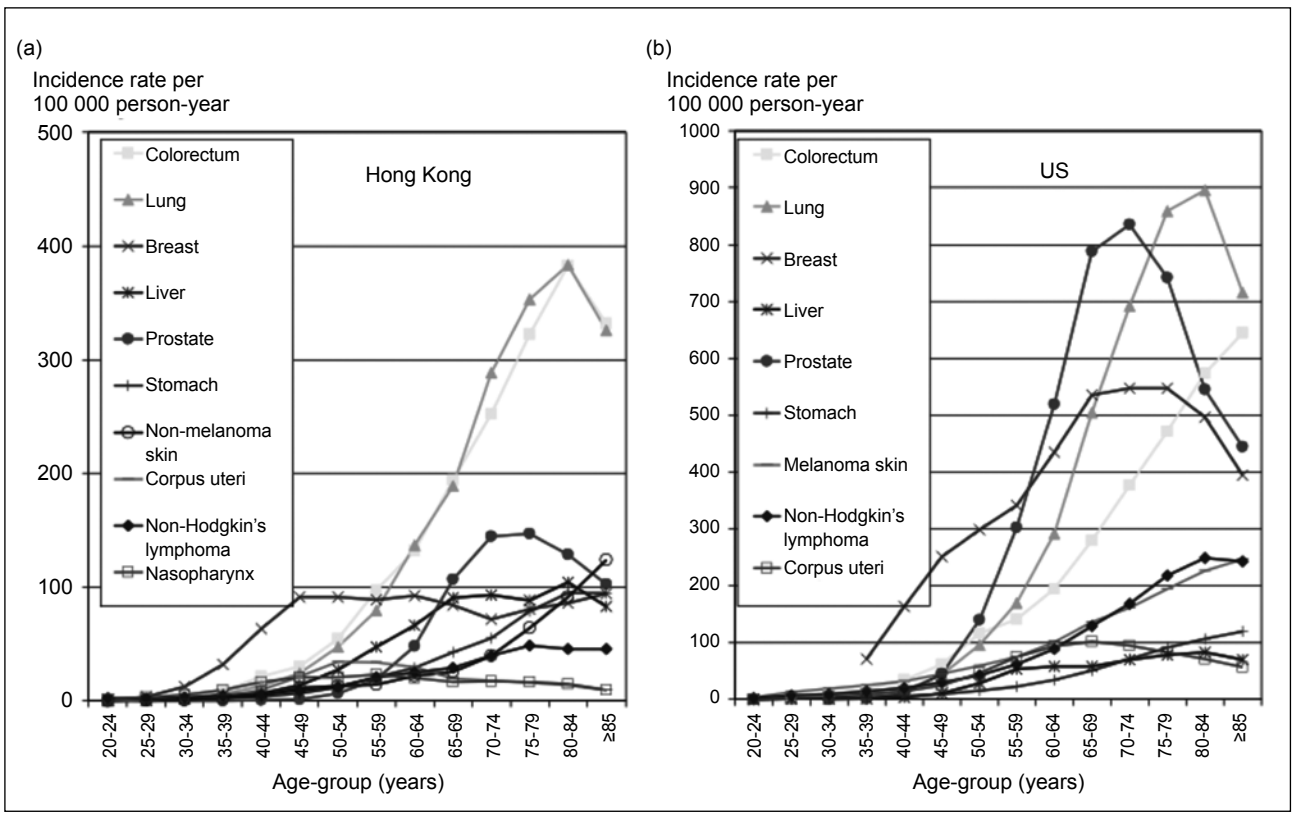

Figure 13.2 Increasing incidence of common cancers with age for Hong Kong (2013)[1] and the US (2008-2012)[2]

adults value improving or maintaining function more than prolonging life.[13] Since elderly patients are heterogeneous in terms of their fitness and frailty, it is essential to identify those who are potentially likely to benefit from aggressive treatment versus those who are more vulnerable to adverse outcomes. To meet the multiple challenges in managing cancer in old age, organised collaboration between geriatricians and oncologists, or oncogeriatrics, has emerged as the recommended model of care.[10]

13.2 Special Features and Needs of Elderly Cancer Patients

Compared with younger adults, the presentations of cancer among elderly patients are often non-specific and may be masked by concurrent chronic conditions and comorbidities, present in 40-90% of older cancer patients.[14] Stroke may be the first presentation of cancer in old age and its co-occurrence increases disability and mortality.[15,16] Cancers in elderly persons are underdiagnosed because the symptoms may be attributed to ageing, chronic diseases, or social and psychological problems.[7] Cancers in elderly persons tend to be diagnosed at a more advanced stage. Multiple malignancies are common at presentation, occurring in up to 20% of those aged >70 years.[17] Decreased functioning is common among elderly cancer patients, 20% of whom have impaired activities of daily living (ADL), and 70% have impaired instrumental activities of daily living (IADL).[18,19]

The ready availability of radiological imaging and 'tumour' markers may lead to overdiagnosis of cancers without histological confirmation. An incidental finding of raised serum alkaline phosphatase, sclerotic bone lesions, and active uptake on a bone scan in an asymptomatic elderly person may be due to non-malignant causes such as Paget's disease of bone rather than bone secondaries. Extremely elevated CA 19-9 serum concentration, thought to be a marker for pancreatic or biliary cancer, has been reported in cholestasis caused by choledocholithiasis.[20] Increased serum CA-125 concentration has been reported in a number of non-cancer conditions other than ovarian cancer.[21] In countries where tuberculosis is prevalent, pulmonary, pelvic or peritoneal tuberculosis are important diagnoses to consider other than ovarian cancer.[21-24] Tuberculous or nontuberculous mycobacterial infections, infective endocarditis, masked hyperthyroidism, and giant cell arteritis may result in cachexia, ill health, or lytic bone lesions in old age and simulate cancer (Plate 13.1).[25-27] To complicate the diagnosis further, these conditions can coexist with cancer or occur after treatment of cancer (Plate 13.2).[28-30] Early and accurate diagnosis of these cancer mimics will lead to correct and appropriate treatment.

The clinical behaviour of some tumours changes with age. Some become more aggressive, e.g. acute myelogenous leukaemia, due to a higher prevalence of unfavourable genomic changes or resistance to chemotherapy in older persons.[31] Other cancers become more indolent, e.g. breast cancer, due to a higher prevalence of hormone receptor–rich tumours and endocrine senescence.[32]

Surgery forms the basis of most curative treatments, but elderly patients have more perioperative risk factors that can be barriers to surgical treatment such as multiple comorbidities, polypharmacy, malnutrition, frailty, cognitive dysfunction, and a higher anaesthetic risk. The availability of laparoscopic techniques may nonetheless reduce surgical risk even in high-risk elderly patients. Concerns have been raised that treatment of older patients with systemic cytotoxic therapy often relies on evidence extrapolated from younger patients. Nonetheless more recent studies that included older patients have shown that treatment regimens previously considered potentially too toxic for older patients were well tolerated if patients were carefully selected. Radiotherapy may be chosen as a curative option in patients with an operable tumour who are unfit for surgery but there may be practical problems. An elderly patient may find it difficult to make multiple frequent visits to a hospital for radiotherapy treatment.

The hallmarks of ageing are biological changes that lead to decreased homeostatic reserve. Those that occur in the bone marrow, kidneys, immune, and cardiovascular systems are of particular relevance to cancer treatment. The management of cancers in elderly persons, in contrast to younger adults, has to take into account differences in drug metabolism, pharmacodynamics, and pharmacokinetics; polypharmacy and adverse drug reactions, the potential for secondary complications of disease and treatment; potential for development of unrelated conditions during cancer therapy; comprehension deficits, diminished social support, limited financial resources (financial toxicity) that impacts compliance and access to care; and competing concurrent chronic conditions.[33-35]

The geriatric syndromes of incontinence, instability and falls, intellectual decline, impaired hearing and vision, iatrogenesis, isolation, and impaired nutrition and immune deficiency not only affect cancer therapy but also impact more on the patient and the family than the cancer. A rehabilitative approach is required for elderly patients with cancer to maintain or restore function and autonomy. There is a need to palliate symptoms from cancer and / or its treatment; and for sensitive and compassionate communication with an

elderly patient, who may have sensory or cognitive deficits, and with the family, who may request non-disclosure to the patient.

13.3 Comprehensive Geriatric Assessment in the Management of Cancers in Old Age

Elderly population is heterogeneous and can be subclassified according to chronological age as young-old (65-74 years), older-old (75-84 years), and oldest-old (≥85 years). Nonetheless there is great variability in the health and functioning of elderly persons of the same chronological age. Biological age, which takes into account an elderly individual's functioning and reserve, is more important than chronological age when making individual decisions about cancer treatment. The estimate of biological age is best performed by carrying out a comprehensive geriatric assessment (CGA, see Chapter 2). The International Society of Geriatric Oncology recommends that the following domains be evaluated in a geriatric assessment: functional status, comorbidity, cognition, mental health status, fatigue, social status and support, nutrition, and presence of geriatric syndromes.[9,10] The InterRAI Consortium (http://www.interrai.org/) is developing a specific assessment tool for elderly patients with cancer.

In the past decade, clinical trials of CGA in oncology have demonstrated benefits in recognising unsuspected geriatric problems and syndromes,[36-40] predicting treatment outcome and survival,[41-44] influencing treatment choice and intensity,[45,46] and lastly in implementing a multidisciplinary intervention plan.[47,48]

Cancer patients, rated as functionally normal by Karnofsky Performance Scale that is commonly used in oncology, have been identified by CGA to have important deficits that may affect treatment tolerance and outcome.[36] The application of CGA in elderly cancer patients revealed unsuspected geriatric syndromes, including cognitive impairment (dementia and / or delirium), depression, weight loss, nutrition, use of high-risk medications, and ADL dependence; and cancer treatments were changed in 21-49% of patients consequent to CGA.[37-40] A common geriatric syndrome revealed by CGA is delirium that can readily be reversed by early recognition of potential underlying causes, including fluid imbalance (dehydration or overload); infection (e.g. urinary tract, skin, respiratory infection); electrolyte abnormalities (e.g. hyponatraemia, hypercalcaemia); uncontrolled pain; hypoxia; hypoglycaemia; hyperglycaemia; nutritional deficiencies; constipation or diarrhoea; hypothermia or hyperthermia; unfamiliar environment or isolation; sensory deprivation or overload; sleep deprivation; and adverse drug reactions.[49] Impaired cholinergic neurotransmission has been implicated in the pathogenesis of delirium.[50] Anticholinergic effects have been identified in many drugs other than those classically thought of as having major anticholinergic actions.[50,51] Examples of such drugs include furosemide, digoxin, ranitidine, codeine, prednisolone, nifedipine, and warfarin.[51] The cumulative anticholinergic burden of multiple medications prescribed to an elderly patient may place stress on the reduced brain reserve and induce delirium.[50-52]

CGA trials in elderly cancer patients have demonstrated benefits in predicting the risk of complications of surgery and chemotherapy, as well as treatment outcome. Dependence in IADL has been found to predict surgical outcome.[41] A score that incorporates hearing loss, fall within 6 months, IADL (ability to take medications), difficulty walking one block, and reduced social activity predicted chemotherapy toxicity; whereas the Karnofsky Performance Scale failed to identify older adults at increased risk of chemotherapy toxicity.[42] CGA is more effective than clinical judgement in identifying elderly patients with diffuse, large cell lymphoma who will benefit from aggressive immunochemotherapy.[43]

CGA influences cancer treatment choice and decisions. A study has revealed that knowledge of CGA lowered the number of prescribed drugs, reduced chemotherapy intensity, or initiated supportive care in 44.9% of patients. Another study showed that CGA allowed pretreatment patient optimisation when remediable problems were detected.[46]

Studies have shown that CGA was helpful in the implementation of a multidisciplinary intervention plan in elderly cancer patients, with improved ADL, pain control, quality of life, and mental health scores; as well as reduction of inappropriate medications or unmet community needs, and provision of information about the socioeconomic status of the patient, and caregiver support.[47,48]

13.4 Geriatrician-oncologist Collaboration in the Individualised Care of an Elderly Cancer Patient

Consensus guidelines recommend the routine use of CGA for elderly persons with cancer.[9,10] In the comprehensive geriatric model for cancer in elderly persons, specific interventions (surgery, chemotherapy, radiotherapy, hormonal treatment) are individualised according to the functional reserve compressed by ageing and disease; as well as the biological and physical functioning and sociopsychological needs of an elderly person revealed by a CGA.[7] A geriatrician, supported

by an interdisciplinary team of nurses, therapists and social workers, assesses the physical, mental and social functional status of an elderly cancer patient; as well as the presence of comorbidities and geriatric syndromes (immobility, incontinence, instability, intellectual decline, iatrogenesis, immune deficiency, impaired sight / hearing, insomnia, isolation, impecunity, irritable colon, impotence). Management-related decisions have to be guided by an elderly patient's functional level along the fitness-frailty continuum; tolerance to and benefit-to-risk ratio of diagnostic and therapeutic interventions; socioeconomic support; long-term consequences; life expectancy, and their wish and value. One such clinical algorithm of an individualised approach to an elderly cancer patient based on CGA is outlined in Figure 13.3[53] (see Chapter 13L case scenarios for illustrations). The goals of treatment of cancer in an elderly person include cure if possible, prolongation of survival, prolongation of active life expectancy (number of years of life remaining in an independent state free from significant disability), symptom management, prevention of adverse drug reactions, sympathetic care, and support and preservation of quality of life. The provision of supportive care for an elderly patient is important as it enhances quality of life, and includes recognising and managing pain effectively; relieving constipation; providing nutritional and hydration support; preventing and treating nausea and vomiting; treating fatigue and insomnia; providing psychological support to an elderly person and family; discussing end-of-life care; and providing palliative care.[7] The need for human interactions in palliating distress is best illustrated by the story recounted by Archie Cochrane who served as a camp doctor during the Second World War:

> "Another event at Elsterhorst (prisoner of war camp) had a marked effect on me. The Germans dumped a young Soviet prisoner in my ward late one night. The ward was full, so I put him in my room as he was moribund and screaming and I did not want to wake the ward. I examined him. He had obvious gross bilateral crepitations and a severe pleural rub. I thought the latter was the cause of the pain and the screaming. I had no morphia, just aspirin, which had no effect. I felt desperate. I knew very little Russian then and there was no one in the ward who did. I finally instinctively sat down on the bed and took him in my arms, and the screaming stopped almost at once. He died peacefully in my arms a few hours later. It was not the pleurisy that caused the screaming but loneliness. It was a wonderful education about the care of the dying. I was ashamed of my misdiagnosis and kept the story secret."[54]

Collaboration between oncologists and geriatricians has emerged in the past decade as a clinical model that integrates geriatric principles and assessment into oncology care of elderly persons with cancer. A structured Geriatric Oncology Programme has been promoted to coordinate oncologists, geriatricians, physiotherapists, nurses, and social workers to generate treatment plans for elderly cancer patients.[9,14,55-57] Models for implementing oncogeriatrics in clinical practice include geriatric

	Fit	Vulnerable	Frail
Function impaired	-	+ (IADL)	++ (ADL)
Comorbidities	-	+	++ (3+, severe)
Geriatric syndromes	-	+ / -	++

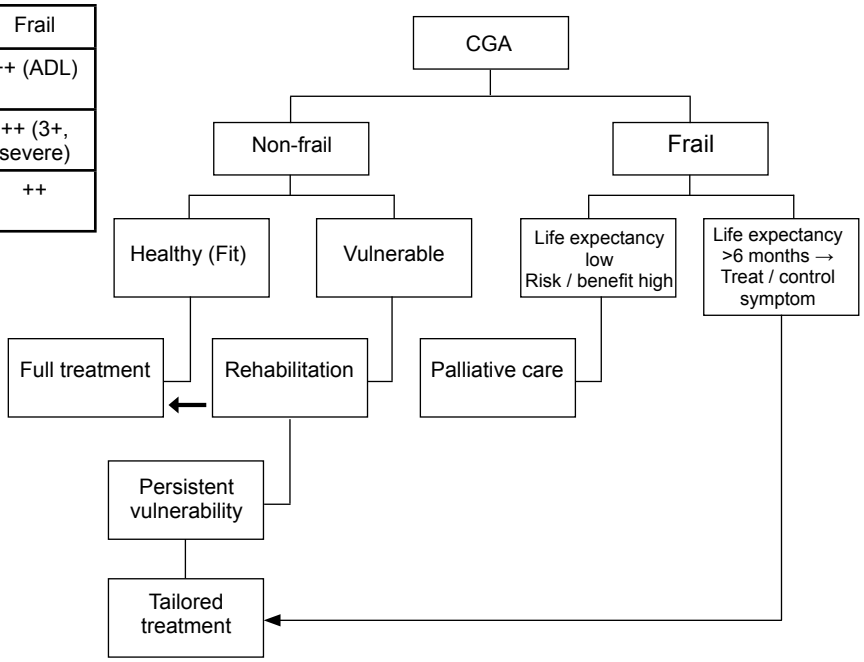

Figure 13.3 Individualised approach to an elderly cancer patient based on comprehensive geriatric assessment[53] (reproduced with permission)
Abbreviations: ADL = activities of daily living; CGA = comprehensive geriatric assessment; IADL = instrumental activities of daily living

oncology units within a general oncology hospital; geriatric consultation teams for patients who remain under the supervision of their oncologist; and referral of high-risk patients to geriatricians outside of the cancer center.[10]

The International Society of Geriatric Oncology recommends that every elderly cancer patient should have a screening assessment to identify frailer elderly patients who require a more detailed CGA by a geriatrician.[9,10] Instead of being excluded from standard oncology treatment, frailer patients may still benefit from cancer treatments after an individualised intervention based on CGA has solved the biological, clinical, and social issues that would otherwise limit the general application of oncology guidelines (Figure 13.4).[9,58] The important focus areas of CGA in an elderly cancer patient are: functional status, comorbidity and its severity, medication use and potential adverse drug reactions, nutritional status, cognition, and social support. Studies of Geriatric Oncology Programmes and Onco-Geriatric approaches have revealed promising outcomes in terms of improved selection of elderly cancer patients for appropriate intervention.[41,59]

In targeting elderly cancer patients, one question often asked is "what age cutoff?" Many oncology studies have used an age of ≥70 years as the age for implementing geriatric assessment, but other age cutoffs have been proposed.[10] Research is in progress to identify whether a shorter geriatric screening tool can identify which older patients with cancer will benefit from a more CGA. As explained above, not all individuals age biologically at the same rate. A very high age cutoff will have a high specificity but low sensitivity for biologically aged patients, while a low age will mean a high sensitivity but low specificity for biologically aged individuals who would benefit from CGA and joint geriatrician-oncologist collaboration. An alternative more sensible approach is a needs-related service that targets those patients whose medical and social problems suggest a benefit from a team approach. A third strategy is to combine the two approaches: to offer an age-related service with a fairly high cutoff age (say 80 years) plus a needs-related service between 65 years and the cutoff age.

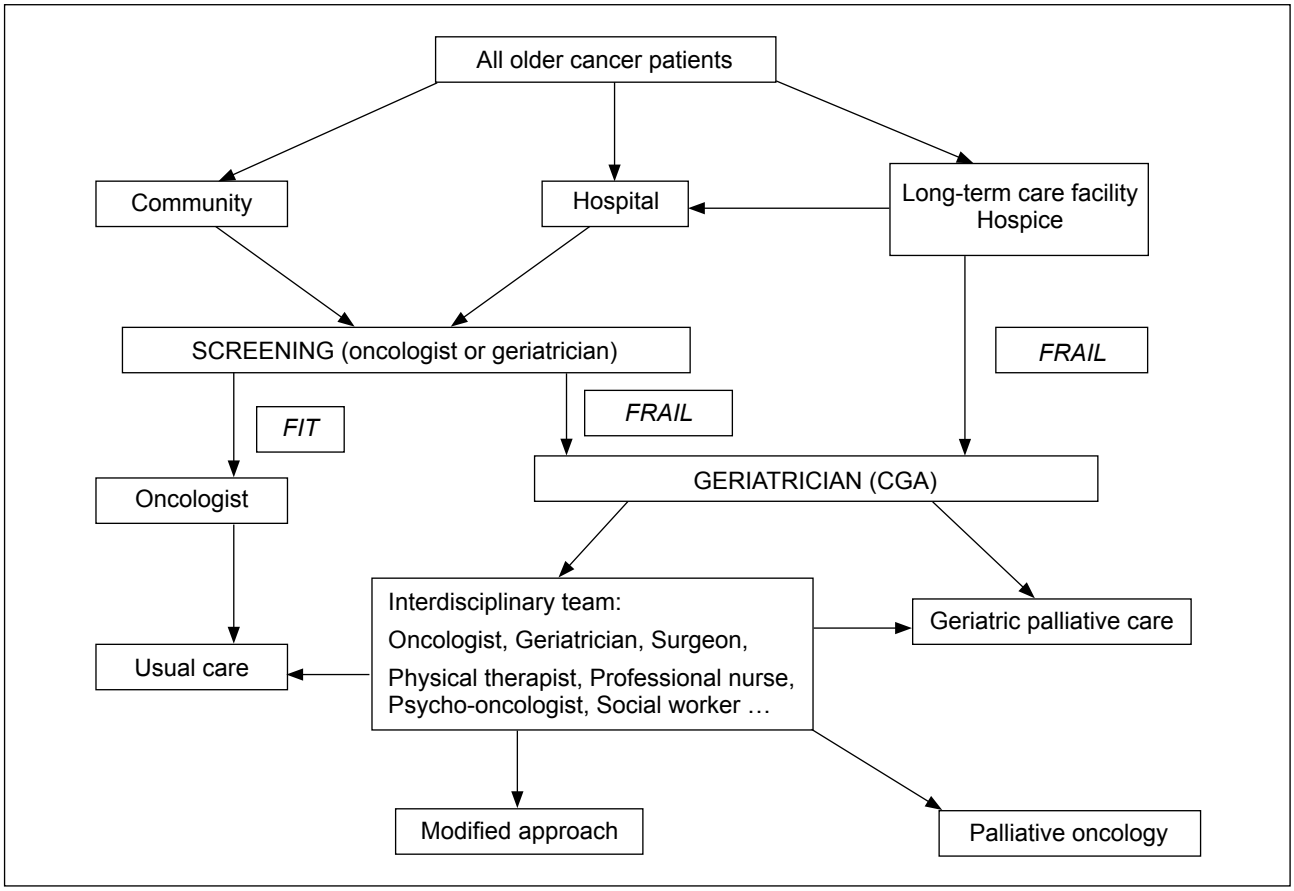

Figure 13.4 Geriatrician-oncologist collaboration in the individualised care of an elderly cancer patient[58] (reproduced with permission)
Abbreviation: CGA = comprehensive geriatric assessment

To conclude, the management of an elderly person with cancer will benefit from the input of a geriatrician, who has expertise in ageing and frailty, diagnosis and CGA, geriatric syndromes, managing multiple comorbidities, polypharmacy and appropriate medication use, rehabilitation to improve function, counselling, and palliative and end-of-life care. Treatment safety in older cancer patients has been enhanced by recent advances in surgical techniques and oncology, e.g. minimally invasive robotic surgery, new radiation techniques, antidotes to reduce chemotherapy toxicity, personalised medicine from genomic profiles, and methods to gauge response to cancer therapy early on in the treatment course.[60] Tallis[61] has argued that the future of old age should be optimistic with enjoyable longevity rather than pessimistic with unenjoyable life. The emerging practice of geriatric oncology will be a promising paradigm to add to the optimism.

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CHAPTER 14

Indications for and Interpretation of Laboratory Investigations in Old Age

Leung-wing CHU, Patrick Ka-chun CHIU

OBJECTIVE

This chapter describes the indications for and clinical interpretation of commonly performed laboratory investigations which are of relevance in geriatric clinical practice in the community.

KEY POINTS

- Ageing is associated with changes in some but not all the laboratory values.
- Frail elders may have decreased leukocyte response to infection.
- A 'normal' serum creatinine level may occur in the presence of impaired renal function.
- A 'normal' serum uric acid level may occur in the presence of acute gouty arthritis and does not exclude gout.
- In elderly patients, autoantibodies may be present in autoimmune diseases as well as non-autoimmune diseases.
- Tumour markers are not specific for tumours and may be elevated in other benign conditions.
- In the interpretation of laboratory results, subtle clinical features including drugs have to be considered in conjunction with the laboratory test results.

14.1 Introduction

Ageing is associated with physiological changes in most organs and tissues of the body. While abnormal laboratory results are commonly seen in the older patients, only a few are due to ageing (Tables 14.1, 14.2).[1] In an elderly person, the co-existence of chronic diseases, medications in use, physical inactivity, and dietary and other lifestyle changes may affect the results of common laboratory investigations. These changes have important implications in the clinical interpretation of 'normal' versus 'abnormal' findings. Misinterpretation of 'abnormal' investigations as changes due to ageing could lead to underdiagnosis and undertreatment. On the other hand, 'normal' laboratory results may not imply normal organ function. For example, elderly patients with mild-to-moderate renal failure may still have serum creatinine levels within the normal reference range because the lean body mass and daily endogenous creatinine production decline in old age.

14.2 Haematological Investigations

14.2.1 Haemoglobin, Total White Cell, Neutrophil, and Lymphocyte Counts

Normal healthy elderly people are not anaemic and the finding of a reduced haemoglobin level indicates anaemia, a common condition among elderly people. The accepted lower limit of normal haemoglobin level is 13 g/dL in men and 12 g/dL in women.[2] Common causes of anaemia include iron deficiency (e.g. gastrointestinal blood loss, dietary deficiency), folate deficiency, vitamin B_{12} deficiency, chronic inflammatory disorders, chronic diseases, malignancy, and protein-energy malnutrition.[2,3] Figure 14.1 highlights the common clinical approach in the diagnosis and management of elderly patients with anaemia (see also Chapter 39).

Table 14.1 Laboratory parameters for which normal values remain unchanged in old age*

• Haemoglobin and haematocrit
• White blood cell count
• Platelet count
• Electrolytes (sodium, potassium, chloride, bicarbonate)
• Blood urea nitrogen
• Liver function tests (transaminases, bilirubin, prothrombin time)
• Thyroid function tests (free T4, free T3)
• Thyroid-stimulating hormone
• Calcium
• Phosphorus

* Ageing does not affect these parameters; abnormal values should prompt further evaluation

Table 14.2 Common abnormal laboratory parameters in old age and their clinical significance*

Parameter	Clinical significance
Erythrocyte sedimentation rate	Mild elevations (10-20 mm/h) may be an age-related change
Glucose	Glucose tolerance decreases
Creatinine	Because lean body mass and daily endogenous creatinine production decline, high-normal and minimally elevated values may indicate substantially reduced renal function
Albumin	The normal range in healthy elderly people is the same as in younger age-groups. In hospitalised elderly patients, mean values of serum albumin decline with age but these generally indicate undernutrition
Alkaline phosphatase	Mild elevations are common in asymptomatic elders; liver or bone disease should be considered if moderately elevated
Serum iron and iron-binding capacity (transferrin)	Decreased values are not changes due to ageing and usually indicate undernutrition and / or gastrointestinal blood loss

* Includes normal ageing and other age-related changes

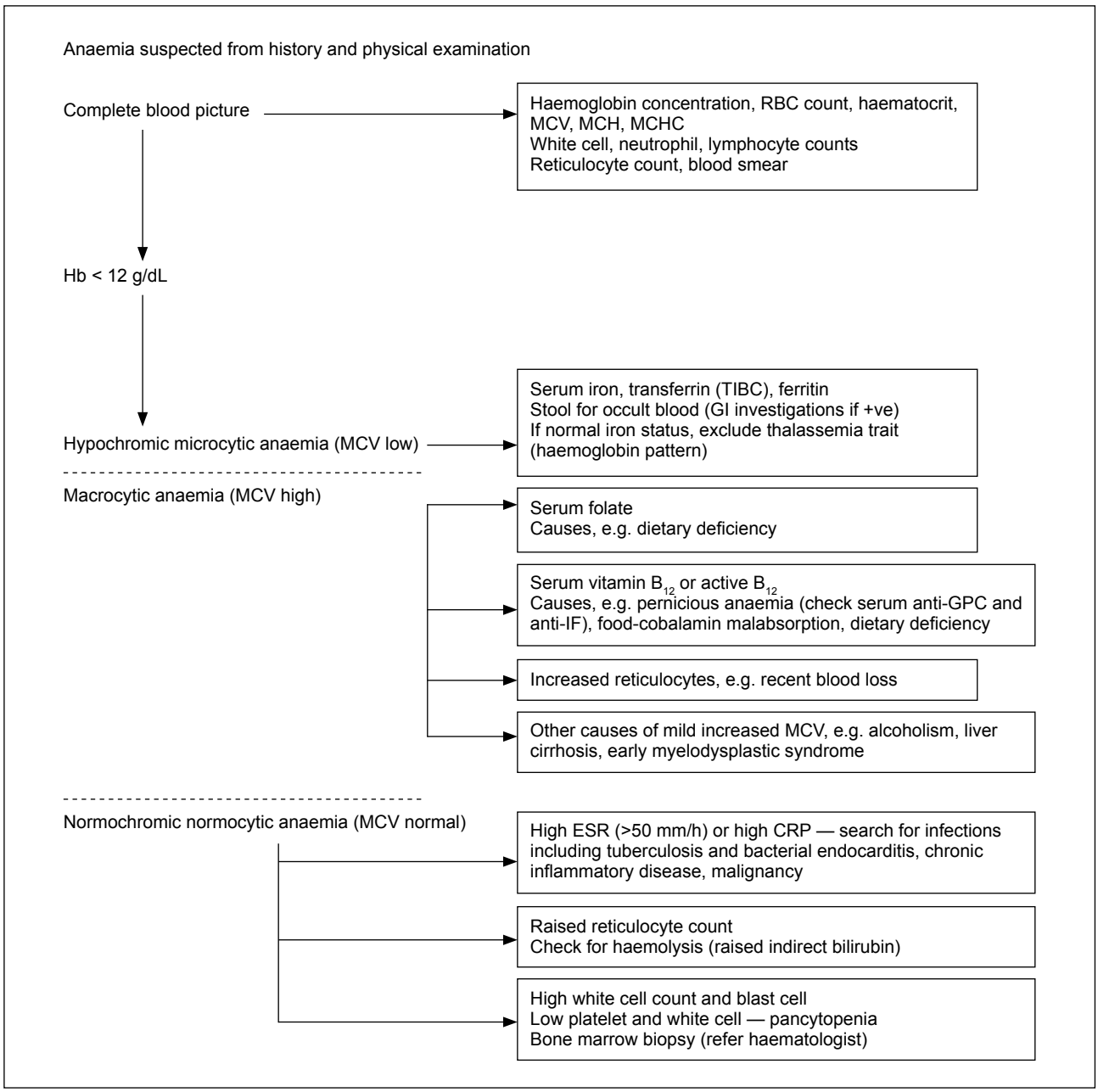

Figure 14.1 Clinical approach to anaemia in elderly people
Abbreviations: CRP = C-reactive protein; ESR = erythrocyte sedimentation rate; GI = gastrointestinal; GPC = gastric parietal cell; Hb = haemoglobin; IF = intrinsic factor; MCH = mean corpuscular haemoglobin; MCHC = mean corpuscular haemoglobin concentration; MCV = mean corpuscular volume; RBC = red blood cells; TIBC = total iron-binding capacity

The normal upper limit of the total white cell count is $11x10^9$ /L. Common causes of an elevated white cell count include infection, inflammation, trauma, surgery, blood loss, and leukaemia. In frail elderly patients with sepsis, it is well recognised that the total white cell count may not be raised or may be raised to a lesser extent than in younger patients. A mildly elevated or a high-normal neutrophil count would support the presence of bacterial infection or inflammation. A peripheral blood film is useful and may show neutrophils with toxic granulation in bacterial infection, abnormal blast cells, or increased number of lymphocytes in leukaemia.[2,3]

14.2.2 Erythrocyte Sedimentation Rate and C-Reactive Protein

The normal upper range of erythrocyte sedimentation rate (ESR) is higher in elderly people and an ESR of 10 to 20 mm/h would be considered as normal. In a study of 27912 subjects aged 20 to 65 years, Miller et al[4] defined a normal range that differed for men and women and increased with age; the upper limit is given by age (in years) divided by 2 in males, and by the sum of age and 10 divided by 2 in females. Griffiths et al[5] extended the study of ESR to 200 elderly people aged 60 to 89 years, and confirmed that the ESR increased with age and that women had higher values than men, and that the Miller's formula for young patients approximately predicted the 98th percentile of ESR in elderly patients. Their results, however, revealed that over half of elderly patients with disease would have rates within the 'normal' range as defined by Miller's formula with mean rates being 19 mm/h and 22 mm/h in their sample of elderly patients with disease. Therefore, they suggested that an ESR of >19 mm/h in elderly men and >22 mm/h in elderly women warrant investigation.[5] An ESR of ≥50 mm/h would deserve detailed investigation of the underlying cause. Other tests for acute-phase reaction like the C-reactive protein (CRP) would be more helpful than the ESR for detecting disease state. CRP is synthesised in the liver and is an acute-phase reactant, the level of which increases markedly within 48 to 72 hours in response to an acute-phase reaction.[3] The level of CRP is elevated in bacterial infection, metastatic malignancy, acute myocardial infarction (AMI), and active rheumatoid arthritis. Serial CRP levels allow a reliable clinical interpretation of an acute clinical event including infection.

14.3 Biochemical Investigations

14.3.1 Electrolytes

Normal ageing is not associated with any change in the normal range of plasma sodium (Na^+), potassium (K^+), or osmolarity.[1-3,6] Fluid and electrolyte (i.e. Na^+ and K^+) imbalance are very common in geriatric patients and tend to occur in sick elderly patients due to infection, heart failure, excessive water or electrolyte administration and drugs (e.g. diuretics).

14.3.1.1 Hyponatraemia

Hyponatraemia is said to be present when the plasma Na^+ concentration is <135 mmol/L. It rarely causes symptoms until the plasma Na^+ concentration falls below 125 mmol/L. Symptoms of hyponatraemia include lethargy, mental dullness, delirium, and seizures. A simultaneous plasma and urine osmolality is helpful when the syndrome of inappropriate antidiuretic hormone (SIADH) is suspected. In SIADH, the plasma osmolality is low and urine osmolality is raised to approximately twice that of the urine.[1-3,6]

If dehydration is present, the hyponatraemia is likely to be due to Na^+ depletion and the causes include use of diuretics,[7] reduced food and salt intake, vomiting or diarrhoea, and diabetic hyperglycaemia. Taking a good history is an important first step in making the appropriate diagnosis. Plasma osmolality may be raised or normal.

If the patient has oedema and fluid retention, dilutional hyponatraemia is likely. Hyponatraemia is commonly mild and will often improve with treatment of the underlying cause of the oedema. Both the plasma and urine osmolality would be low in this group of patients.

Hyponatraemia is common among elder people on tube feeding. This is likely secondary to inadequate sodium chloride content in the liquid diet and can readily be corrected by raising the salt intake.[8]

Pseudo-hyponatraemia is uncommonly encountered in elderly patients who have high serum protein levels (e.g. macroglobulinaemia, multiple myeloma), or hyperlipidaemia.

14.3.1.2 Hypernatraemia

Hypernatraemia is usually diagnosed when plasma Na+ concentration is ≥150 mmol/L. It is uncommon but occurs more often in elderly than in younger patients. Hypernatraemia is always associated with dehydration, which is either due to inadequate water intake or excessive water loss. For excessive water loss, testing the urine Na^+ concentration will help differentiate urinary losses (urine Na^+ >20 mmol/L) versus non-urinary losses (urine Na^+ ≤20 mmol/L). Glucose (e.g. in poorly controlled diabetes mellitus), urea, or mannitol can all induce osmotic diuresis and urinary Na^+ losses. Diuretics, particularly the loop diuretics, would lead to excessive water and salt loss. Excessive renal water loss may also occur

in central diabetes insipidus, which is a rare cause of hypernatraemia. Diarrhoea or excessive sweating can lead to excessive water loss by non-urinary routes.[1-3,6]

Regarding inadequate water intake, the compensatory mechanisms of thirst and oliguria in the presence of fluid deprivation are decreased in elderly patients. Ageing is associated with decreased sensitivity of the central thirst mechanism. However, a more common cause is the restricted access to water or fluid drinks in dependent elderly people. This is a common finding in substandard nursing homes. Hyperosmolar oral or nasogastric feeds without adequate water intake may be another cause of hypernatraemia.

14.3.1.3 Hypokalaemia

Hypokalaemia is present when the plasma K^+ concentration is below the lower limit of the reference range (e.g. 3.5 mmol/L). Hypokalaemia is usually asymptomatic unless it is very low (usually <2.5 mmol/L). Generalised weakness may then occur. Constipation and even paralytic ileus may occur. The electrocardiogram usually shows a U wave and flat T wave.

Over 90% of the K^+ in the human body is intracellular. Shifting of the plasma K^+ into the cell can, therefore, give rise to hypokalaemia.[1-3,6] The causes of hypokalaemia with normal body store (intracellular shift of K^+) are hyperaldosteronism, catecholamines, β_2-agonist, insulin, vitamin B_{12} therapy, and alkalosis.

The causes of hypokalaemia with decreased body stores are related to either insufficient intake or excessive loss: insufficient intake of K^+-containing food, excessive gastrointestinal losses (e.g. diarrhoea, vomiting), and renal losses (e.g. diuretic use, renal tubular acidosis).

14.3.1.4 Hyperkalaemia

Hyperkalaemia is present when the plasma K^+ concentration is greater than the upper limit of normal (usually 5.2 mmol/L). However, falsely raised K^+ level (factitious hyperkalaemia) may occur if the tourniquet is kept in place for too long or when red cells are disrupted during or after phlebotomy. Hyperkalaemia is usually asymptomatic except when the levels are markedly elevated (usually >6.5 mmol/L). Cardiac arrhythmias may occur. The electrocardiogram findings include tall, peaked T waves in the precordial leads followed by a decreased R wave, widened QRS complex, prolonged PR interval, and decreased P waves. In severe hyperkalaemia, the QRS complex will merge with the tall T wave. Ventricular arrhythmias may then lead to cardiac arrest.[1-3,6]

Except in the case of factitious hyperkalaemia, correction of hyperkalaemia should be started immediately while looking for the underlying cause. The clinical setting will usually yield important clues regarding the cause of hyperkalaemia. Acute or chronic renal failure will give rise to hyperkalaemia. Iatrogenic causes include intravenous potassium infusion, oral potassium supplements, potassium-sparing diuretics, non-steroidal anti-inflammatory drugs, angiotensin receptor blockers, and angiotensin-converting enzyme inhibitors. The identified cause should then be removed or treated.

14.3.2 Renal Function Tests: Serum Urea and Serum Creatinine

The kidney size and volume decrease with age after the fourth decade. The glomerular filtration rate decreases by approximately 1% per year after the age of 40 years.

Serum urea and creatinine levels are often used as indicators of renal function. Blood urea concentration is directly related to protein intake and falls with reduced protein intake. The causes of low serum urea include severe liver disease, protein malnutrition, haemodilution, overhydration, and nephrotic syndrome. A common cause of low serum urea levels in elderly people living in residential care homes is protein-energy malnutrition. Primary care physicians must be aware of this pitfall in the clinical interpretation of serum urea level in geriatric patients. Elderly patients who are thin, have a low body mass index (<18.5 kg/m^2) and low serum albumin level are very likely to have protein-energy malnutrition.[1-3,6]

In acute or chronic renal failure, the serum urea concentration is elevated and serum creatinine is usually raised correspondingly. Below are the causes of increased urea and creatinine levels (renal failure):

- pre-renal: dehydration, shock, haemorrhage, hypovolaemia
- renal: nephrotoxic drugs, end-stage renal failure (chronic glomerulonephritis, hypertensive nephrosclerosis, pyelonephritis, or diabetic nephropathy)
- post-renal: outlet obstruction (stones, benign prostatic hypertrophy, carcinoma of prostate)

In the presence of very low protein intake, serum urea may only be raised mildly even in the presence of moderate renal failure. Moreover, the serum creatinine level may also be slightly raised due to non-renal factors. The amount of creatinine produced is proportional to the muscle mass, which decreases with age and is higher in males than in females. Thus, the serum creatinine level remains relatively constant despite a progressive decline in creatinine clearance of approximately 8 ml/min/1.73 m^2 per decade after the age of 30 years. Therefore, the rise in serum

creatinine level associated with renal disease may be masked in elderly patients by a fall in creatinine production. This is another potential pitfall in the clinical interpretation of the renal function test.

Serum creatinine concentration is, thus, not an ideal parameter for assessing renal function in elderly people. Different formulae have been used to estimate creatinine clearance and glomerular filtration rate. These formulae will be discussed in relation to the safe and effective use of drugs in old age in Chapter 15.

14.3.2.1 Proteinuria

Evaluation of protein in urine is an important indicator of kidney function. Bedside Multistix is used to assess the degree of proteinuria semi-quantitatively. Furthermore, spot urine protein-to-creatinine ratio in mg/mg is roughly considered to correspond with 24-hour total urine protein (g/day). This could save elderly patients from the tedious 24-hour urine collection for protein assessment.

14.3.3 Liver Function Tests

The liver function tests include serum bilirubin, alkaline phosphatase (ALP), aspartate aminotransferase (AST), and alanine aminotransferase (ALT). These are relatively crude indicators of liver function. The normal ranges for bilirubin, AST, ALT, and gamma-glutamyl transpeptidase (GGT) are the same in elderly people as those in the younger age-group. The GGT level is a sensitive test of liver function. It plays a role in differentiating the causes of elevated ALP levels. Elevation of both serum GGT and ALP levels suggests an abnormality in the liver. High ALP but normal GGT levels are found in bone diseases. Grossly elevated levels of GGT are often seen in biliary obstruction.[9]

There is no evidence that the normal range of serum ALP changes with advancing age. Surveys of elderly people have detected elevations of serum ALP in asymptomatic elderly people. However, investigations in these elderly persons usually reveal some underlying abnormality in the liver or bone. Serum ALP level serves as a marker of extrahepatic biliary obstruction and intrahepatic cholestasis. It may be elevated in bone-related disorders such as Paget's disease and bony metastasis. Diseases associated with increased osteoblastic activities such as osteomalacia, primary hyperparathyroidism, secondary hyperparathyroidism from chronic kidney disease, and hyperthyroidism may also be associated with elevated ALP levels.[10] Vitamin D deficiency, which is highly prevalent among institutionalised elderly patients, leads to inadequate absorption of calcium, which leads to low serum calcium levels and increased parathyroid hormone levels. This increased parathyroid hormone then causes increased calcium loss from the bone which increases ALP levels. Liver disease or Paget's disease of the bone should be considered if the level is moderately elevated. Very high level (i.e. >1000 U/L) is characteristic of Paget's disease.[1-3,6]

14.3.4 Albumin

Serum albumin is produced in the liver. The normal range in healthy elderly people is the same as that in the younger age-group. In hospitalised elderly patients, the mean value of serum albumin declines with age but this generally indicates undernutrition. In addition to malnutrition, the serum albumin level may be reduced by many other causes including nephrotic syndrome, chronic liver disease, protein-losing enteropathy, and infection. In the presence of infection, an unexplained fall in albumin occurs because the liver switches from albumin to globulin synthesis. Thus, the primary value of serum albumin as a liver function test is limited.[2,3,6]

14.3.5 Creatine Kinase and Troponins

Creatine kinase (CK) is widely distributed in the body tissues, with highest activities found in the skeletal muscle, heart muscle, and brain tissue.[9] CK levels are elevated in cardiac, skeletal, and central nervous system disorders such as myocardial infarction, rhabdomyolysis, polymyositis, malignant hyperthermia, hypothyroidism, stroke, and seizures.[9] Physical activity and intramuscular injection can also lead to elevation of CK levels.

Total serum CK and its MB isoenzyme (CK-MB) are widely employed in the laboratory diagnosis of acute coronary syndrome or AMI. These are elevated within 12-72 hours after the onset of AMI. The cause of raised total CK and CK-MB is due to leakage of the enzyme from the myocardial cells. In the interpretation of CK results, elderly males have higher serum CK activity than the comparable elderly females. In men, there is a progressive decline in CK with age. Therefore, age- and sex-adjusted normal ranges for CK levels should be employed, if available. If the former is not available and the young normal reference range is used, elders with AMI may exhibit CK levels within the young normal range and lead to wrong clinical interpretations.[1-3,6]

In contrast to CK, cardiac troponins are tissue-specific isoforms that can be used to detect damage to the heart. The normal ranges of troponin T (TnT) and troponin I (TnI) are 0.0-0.1 ng/mL and 0.0-0.04 ng/mL, respectively.[10] For practical purposes, there are no major clinical differences between TnT and TnI.[11,12] Cardiac troponin test is a sensitive diagnostic

test for AMI or acute coronary syndrome. Both TnT and TnI are detectable within 3-12 hours after the onset of myocardial injury, peaking at 12-24 hours and remaining elevated for more than 1 week: 8-21 days for TnT and 7-14 days for TnI.[9,12] On the other hand, CK-MB levels return to baseline after 2 to 4 days. Thus, cardiac troponins offer the widest window for detecting myocardial infarction with the highest sensitivity and specificity.[9] However, there are a number of other clinical syndromes which may also give rise to elevated troponins. These include myocarditis, pericarditis, heart failure, pulmonary embolism, renal insufficiency, acute stroke, and sepsis with or without shock.[12] Careful consideration of the clinical circumstances while interpreting the troponin test in the old age will improve the practical utility of this test.

14.3.6 Serum Iron, Iron-binding Capacity (Transferrin), and Ferritin

Serum iron is used in criteria for diagnosing iron deficiency, which is a common cause of anaemia in elderly people. Early morning sampling is preferred because of the diurnal variation in iron concentration. Although low serum iron levels are commonly found in studies involving elderly patients, the decreased values are not due to ageing, and usually indicate undernutrition or gastrointestinal blood loss. Approximately 60-70% of anaemias in elderly people are due to iron deficiency.[2,3,6]

Iron is carried in the plasma bound to a β_1-globulin transferrin. Apart from transferrin, there are other proteins which are also available for binding free iron. Transferrin represents the largest quantity of iron-binding proteins. In men, the serum transferrin level decreases with age — it is approximately 10% less than in the younger age-group. Less consistent decrease has also been reported in elderly women. The level of transferrin is often elevated in iron deficiency. Transferrin is a negative acute-phase reactant protein. Its level may be reduced in acute inflammatory diseases. Transferrin is also diminished in elderly patients with infection, malignancy, malnutrition, and kidney and liver diseases.[11,13] The variability in serum transferrin levels associated with various disease states indicates that this test alone is not specific and sensitive as a diagnostic test of iron deficiency.

Total iron-binding capacity (TIBC) is a measurement of all proteins available for binding free iron. Since transferrin represents the largest quantity of iron-binding proteins, TIBC is an indirect yet accurate measure of transferrin level. It correlates with serum transferrin but the relationship is not linear over wide range of values. Transferrin saturation (or percent saturation) refers to the percent of transferrin and other mobile iron-binding proteins saturated with iron. It is, thus, the ratio of serum iron to TIBC. Transferrin saturation is more useful than either serum iron or transferrin levels as these are frequently affected by factors unrelated to the iron status. In elderly patients, transferrin saturation is found to be the best index of iron status. When it is less than 12%, significant iron deficiency is usually present. The effect of age on transferrin saturation is controversial.

In uncomplicated cases, serum ferritin levels also accurately reflect the bone marrow iron stores in the body. Ferritin is actually the major intracellular iron storage protein in the body. It is present in most cells, particularly in the liver, spleen, and bone marrow. In both young and elderly persons, the serum ferritin level is lower in women (by 30-40%) than similarly aged men. Elders generally have a higher serum ferritin level (by 50-100%) than individuals in the younger age-group. This may be physiological but may also be due to the high prevalence of chronic conditions including chronic renal failure, alcoholism, smoking, myocardial infarction, chronic liver disease, rheumatoid arthritis, and malignancy (e.g. of the liver and ovary).

Low serum iron level, elevated serum transferrin level with low serum transferrin saturation, and low mean red blood corpuscular volume generally mean iron deficiency. Commonly, in anaemia of chronic disease, the red cells may be normochromic or hypochromic with low serum iron and low or normal transferrin. The serum ferritin may be helpful since levels of <40-44 pmol/L (18-20 ng/mL) are associated with an absence of stainable iron in the bone marrow while levels of >111 pmol/L (50 ng/mL) are commonly associated with adequate iron stores. For levels in between these, bone marrow biopsy would be needed to document the body iron status.[2,3,6] It should also be noted that ferritin is an acute-phase protein and is elevated in conditions such as inflammation and infection. This can result in misleadingly elevated ferritin levels in iron-deficient patients with coexisting systemic illness (see also Chapter 39.6). Inflammatory markers such as CRP may have a role in identifying coexisting inflammation. Table 14.3[14] shows the laboratory iron profile of patients with various disease states.

14.3.7 Thyroid Function Tests

Thyroid function tests are often performed to diagnose hypothyroidism and hyperthyroidism. In uncomplicated situations, the serum thyroid-stimulating hormone (TSH) level is suppressed in hyperthyroidism and elevated in hypothyroidism. It is a good and sensitive test of thyroid function.

Frail elders with hyperthyroidism or hypothyroidism

Table 14.3 Laboratory iron status in patients with various disease states[14]

Medical condition	Serum iron	Transferrin	TIBC	Transferrin saturation	Ferritin
Iron deficiency	↓	↑	↑	↓	↓
Iron deficiency with co-existing chronic disease or inflammation	↓	Normal /↑	Normal /↑	↓	↓/ Normal /↑
Iron overload	↑	↓	↓	↑	↑
Haemochromatosis	↑	↓	↓	↑	↑
Malnutrition	↓	↓	↓	Variable	↓
Chronic infection	↓	↓	↓	↓	↑
Anaemia of chronic disease	↓	Normal /↓	Normal /↓	Normal /↓	Normal /↑

Abbreviation: TIBC = total iron-binding capacity

Table 14.4 Interpretation of thyroid function tests[9]

Medical condition	FT4↓	FT4 normal	FT4↑
TSH↓	• Secondary hypothyroidism • Sick Euthyroid	• Subclinical hyperthyroidism • Sick Euthyroid	• Hyperthyroidism
TSH normal	• Secondary hypothyroidism • Sick Euthyroid	• Normal	• Laboratory artefact • Pituitary hyperthyroidism • Past thyroxine dosing within 6 hours
TSH↑	• Primary hypothyroidism	• Subclinical hypothyroidism	• Laboratory artefact • Pituitary hyperthyroidism • Thyroid hormone resistance

Abbreviations: FT4 = Free thyroxine; TSH = thyroid-stimulating hormone

may have no classical thyroid disease symptoms. Atypical symptoms or vague complaints may be the only clues to possible hyperthyroidism (e.g. apathetic hyperthyroidism) or hypothyroidism. The serum TSH level is suppressed in hyperthyroidism and elevated in hypothyroidism. The free T4 (fT4) and the free T3 (fT3) levels are increased in clinical hyperthyroidism and reduced in clinical hypothyroidism.

In patients with subclinical hypothyroidism, the serum TSH level is mildly increased while the fT4 and fT3 levels are within the normal reference range. Approximately, 50-80% of these patients also have positive thyroid antibodies (i.e. anti-thyroglobulin or antimicrosomal antibody) suggesting the presence of primary thyroid diseases.

In elderly patients who are sick with non-thyroid diseases, the serum total T3 and fT3 levels may also be reduced. In severely ill elderly patients, the serum total T4 is reduced while the fT4 and TSH levels may be decreased or normal. These elderly patients often do not have any primary thyroid disorder but have the "sick euthyroid syndrome". Many non-thyroid conditions may give rise to the "sick euthyroid syndrome" and these include infections, trauma, surgery, starvation, and bone marrow transplantation. The abnormal thyroid function tests will return to normal after resolution of the non-thyroid illness. Treatment of these patients with T3 has been suggested but this remains controversial at present.[2,3,6,15] Table 14.4 shows the interpretation of thyroid function tests.[9]

14.3.8 Uric Acid

The serum uric acid level can be employed to diagnose hyperuricaemia. In patients who have one or more clinical attacks of acute monoarthritis or gouty tophi, the presence of hyperuricaemia supports a clinical diagnosis of gouty arthritis. However, a normal serum uric acid level does not exclude the diagnosis of gouty arthritis. Approximately, 20-40% of patients aged between 50 and 70 years with acute polyarticular gouty arthritis exhibit normal serum uric acid levels. In case of doubt, the diagnosis of gout can be confirmed by polarised light microscopy of Na^+ monourate crystals obtained via joint aspiration.[1-3,6]

14.3.9 Glucose

Every elderly person should be screened for diabetes mellitus at regular intervals or when symptoms arise. According to the latest American Diabetes Association criteria, diabetes mellitus is diagnosed when glycated haemoglobin is ≥6.5% or fasting plasma glucose is ≥7.0 mmol/L, 2-hour plasma glucose is ≥11.1 mmol/L during an oral glucose tolerance test or random plasma glucose is ≥11.1 mmol/L. For asymptomatic patients, the test is usually repeated on the second day to confirm the diagnosis.[16]

On the other hand, hypoglycaemia is present

Table 14.5 Common tumour markers used in old age, their interpretation and uses[9,10]

Tumour marker	Marker for (elevated in)	Also elevated in these conditions	Uses*
AFP	Hepatocellular carcinoma	• Testicular and ovarian cancers • Teratocarcinoma • Gastrointestinal tract cancers with or without liver metastases • Acute viral hepatitis, chronic active hepatitis, and cirrhosis	(b), (c), (d), (e)
CA 15-3	Breast carcinoma	• Pancreas, lung, ovarian, colorectal, and liver cancers	(d), (e)
CA 19-9	Pancreatic cancer	• Hepatobiliary, gastric, colon cancers • Non-cancerous conditions: pancreatitis; cholangitis, hepatitis, cirrhosis	(b), (c), (d), (e)
CA-125	Ovarian cancer	• Fallopian tube tumours (100%), cervical (83%) and endometrial (50%) adenocarcinomas, squamous cell carcinoma of vulva or cervix (<15%) • Non-Hodgkin lymphoma and cancers of pancreas, liver, and lung • Pleural effusion or inflammation; peritoneal effusion or inflammation • Diseases of gastrointestinal tract, liver and pancreas, cirrhosis, and renal failure • Healthy persons (1%)	(b), (c), (d)
CEA	Colorectal cancer	• Lung, breast, and gastrointestinal tumours	(b), (c), (d), (e)
PSA	Carcinoma of prostate	• Benign prostatic hypertrophy, prostatitis, acute urinary retention (5-7 times) • Manipulations such as digital rectal examination, indwelling catheter, and cystoscopy	(a) [controversial], (b), (d), (e)

Abbreviations: AFP = alpha-fetoprotein; CA = cancer antigen; CEA = carcinoembryonic antigen; PSA = prostate-specific antigen
* (a) Screening, (b) diagnosis, (c) prognostication, (d) monitoring treatment response, (e) detection of recurrence

if the glucose level is <4 mmol/L. Causes of hypoglycaemia include oral hypoglycaemic drugs, insulin therapy, and insulinoma. The special features of hypoglycaemia in old age are discussed in Chapter 42.

14.3.10 Cholesterol

A serum total cholesterol level of ≥5.2 mmol/L suggests hypercholesterolemia. In men, the serum total cholesterol concentration increases from young age to middle age. It then remains relatively constant until the sixth decade. The level then declines somewhat to below the middle age value. In women, the total cholesterol level is 5-8% below that of men of similar age until menopause. It then increases continuously and becomes higher than that of older men from the seventh decade onwards. This pattern is also observed in Hong Kong and is reported by Janus.[17] The serum low-density lipoprotein (LDL) cholesterol level follows a similar trend as the total cholesterol level. The high-density lipoprotein (HDL) cholesterol level shows no clear pattern with age in men but a gradual decrease with age in women.[2,3,6,17,18]

Elevated total cholesterol levels are correlated with an increased risk of adverse cardiovascular events. Increased levels of LDL cholesterol (>2.6 mmol/L) have similar clinical implications. In the NCEP ATP III (National Cholesterol Education Program Adult Treatment Panel III) guidelines, LDL cholesterol target of <2.6 mmol/L is the primary goal of intervention for preventing coronary heart disease (CHD) in persons who have CHD risk of >20%. For HDL, a low serum HDL cholesterol level of <1.0 mmol/L yields a higher risk of cardiovascular event.[19]

Hypocholesterolaemia is often encountered in financially poor and sick elderly people who have significant malnutrition. However, co-existence of diseases like malignancy, chronic infection (e.g. tuberculosis), liver, and renal or gastrointestinal diseases must be excluded. Careful attention to clinical assessment of elderly people should be given if hypocholesterolaemia is detected.

14.4 Autoantibodies and Immunological Tests

The presence of autoantibodies in elderly people may not indicate autoimmune diseases. A significant proportion of elderly people may show positive autoantibodies. This may be due to the increased exposure to exogenous factors such as intercurrent viral infections or the increased use of medications in elderly people. Medications commonly used in this population such as hydralazine, isoniazid, pyrazinamide, phenytoin, and carbamazepine may cause drug-induced lupus erythematosus and mimic the features of autoimmune diseases.[20]

14.4.1 Antinuclear Antibodies

Antinuclear antibodies (ANAs) are non–organ-specific autoantibodies. Their presence at low titres occurs more frequently in elderly people. The prevalence of ANAs in elderly people from a collection of series including 3462 patients aged ≥50 years was 14%.[20] Measuring ANAs is useful in the initial screening of suspected autoimmune diseases such as

rheumatoid arthritis, systemic lupus erythematosus (SLE), Sjögren's syndrome, mixed connective tissue disease, polymyositis, dermatomyositis, and scleroderma. However, this autoantibody is also found in non-rheumatic diseases such as Grave's disease, Hashimoto's thyroiditis, autoimmune hepatitis, primary autoimmune cholangitis, and in some infections and malignancies.[21,22]

There are different types of ANAs and they are more specific to different nuclear proteins. Their presence is closely associated with systemic autoimmune diseases. The anti–double stranded DNA antibodies are highly specific for the diagnosis of SLE, and are also useful for monitoring the disease progress. The anti–extractable nuclear antigen antibodies include the anti-Smith (Sm) antibody, anti-Ro/SS-A antibody, and anti-La/SS-B antibody. Anti-Sm antibody is also associated with SLE while the anti-Ro/SS-A antibody and anti-La/SS-B antibody are associated with Sjögren's syndrome.

14.4.2 Rheumatoid Factor and Anti-cyclic Citrullinated Peptide Antibody

Rheumatoid factor (RF) is another autoantibody frequently described in elderly people. Similar to ANA, RF is present in healthy elderly people. The prevalence of RF in elderly people from a collection of series including 1794 elderly subjects aged ≥60 years was 22%.[20] RF is commonly used in the diagnosis of rheumatoid arthritis and its presence may indicate a more progressive disease with erosive joints and extra-articular manifestations. Its sensitivity and specificity are both around 70%. Approximately 15% of patients with rheumatoid arthritis are not positive for RF. On the other hand, RF may be present in other autoimmune diseases such as SLE, Sjögren's syndrome, interstitial fibrosis, as well as in various infectious diseases.[22] Anti-cyclic citrullinated peptide antibody (anti-CCP) is a new biomarker and a more specific (around 95% specificity) autoantibody for rheumatoid arthritis. Its sensitivity is similar to that of RF. Patients with positive anti-CCP antibodies tend to run a more aggressive disease course in the early stage.[22]

14.4.3 Antinuclear Cytoplasmic Antibodies

Antinuclear cytoplasmic antibodies (ANCA) are usually used in the evaluation of vasculitis, particularly Wegener's granulomatosis, microscopic polyarteritis, and Churg-Strauss syndrome. The presence of ANCA in elderly people is almost always related to the presence of disease. ANCAs are categorised into cytoplasmic (c-ANCA) and perinuclear (p-ANCA) according to the staining patterns. The presence of c-ANCA is highly specific for Wegener's granulomatosis. The antigen involved is proteinase 3. The p-ANCA, which is targeted against myeloperoxidase, is most commonly associated with microscopic polyarteritis.[21,22]

14.5 Tumour Markers

Tumour markers are biochemical substances which are elevated in the serum of patients with cancers. These are produced by the tumour directly or indirectly from their effect on normal healthy tissue. Tumour markers can be used for screening, diagnosis, prognostication, treatment monitoring, and detection of cancer recurrence.[9] Common tumour markers used in elderly patients include carcinoembryonic antigen, cancer antigen 15-3, cancer antigen 19-9, cancer antigen 125, alpha-fetoprotein, and prostate-specific antigen. These tumour markers can be elevated in different kinds of malignancies or benign conditions. Table 14.5 summarises the clinical uses of these markers and the diseases and conditions in which elevated levels of these tumour markers may be found.[9,10]

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CHAPTER 15

Safe and Effective Use of Drugs in Old Age

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OBJECTIVE

To learn about appropriate and rational use of medications in elderly people, medication-related problems, and recognition and prevention of iatrogenesis, adverse drug reactions (ADRs) and prescribing cascade and their management.

KEY POINTS

- ADRs are common in old age and are a result of multiple interacting factors of ageing; multiple diseases; organ dysfunction; reduced homeostasis; physical, cognitive, and social dysfunction; polypharmacy; prescribing cascade; inappropriate medication; reduced compliance; and age-related changes in pharmacokinetics and pharmacodynamics. Knowledge about age-related changes in pharmacokinetics and pharmacodynamics, physical and mental dysfunction and adverse psychosocial factors in elderly persons are important to guide safe and effective prescription in older adults.
- Adjust dose for renally excreted drugs according to calculated creatinine clearance or estimated glomerular filtration rate with body surface area removed, especially for the frail, old persons. Use the lowest effective dose: 'start low and go slow'.
- Always consider ADRs in the differential diagnosis of geriatric syndromes.
- The risk of ADRs is increased with polypharmacy (more than five drugs), heart failure, liver disease and multiple (four or more) comorbid conditions.
- Beware of intelligent non-compliance when the patient knows 'better' than the doctor. Addressing the reasons for decreased drug concordance and achieving mutual agreement on drug therapy with patient are important.
- Use of traditional Chinese medicine, over-the-counter drugs, or health supplements may increase the risk of drug-drug, herb-drug interactions, or unanticipated ADRs. Use of these medications or supplements should be noted in patient's medication history.

15.1 Introduction

The term 'iatrogenesis', from Greek 'iatros' and 'genesis', means 'brought forth by a healer'. Elderly people are the major medication users and, hence, are susceptible to adverse drug reactions (ADRs). A UK study among adults (aged >16 years) admitted over 6 months in two large general hospitals revealed that 6.5% of all admissions were related to ADRs, and that patients admitted with ADRs (median age, 76 years) were significantly older than patients without ADRs (66 years).[1] Most (72%) of these ADRs were avoidable. To achieve safe and effective medication use, it is important to understand the contributing factors for ADRs in elderly people (Figure 15.1).

15.2 Age-related Changes in Pharmacokinetics

Age-related changes in pharmacokinetics and their implications on medication prescriptions for elderly people are summarised in Table 15.1.

15.2.1 Absorption

Age-related changes do not seem to have significant effect on the extent of drug absorption. The effects of ageing on the absorption of medicines of slow-release formulations have not been systemically evaluated. For medications administered via nasogastric tube, research to date found reduction in drug absorption; thus, therapeutic monitoring may be required.[2-4]

15.2.2 Distribution

Drug distribution refers to where the drug goes after it enters the bloodstream. The distribution phase is represented by a theoretically calculated volume. It is simply the size of a compartment necessary to

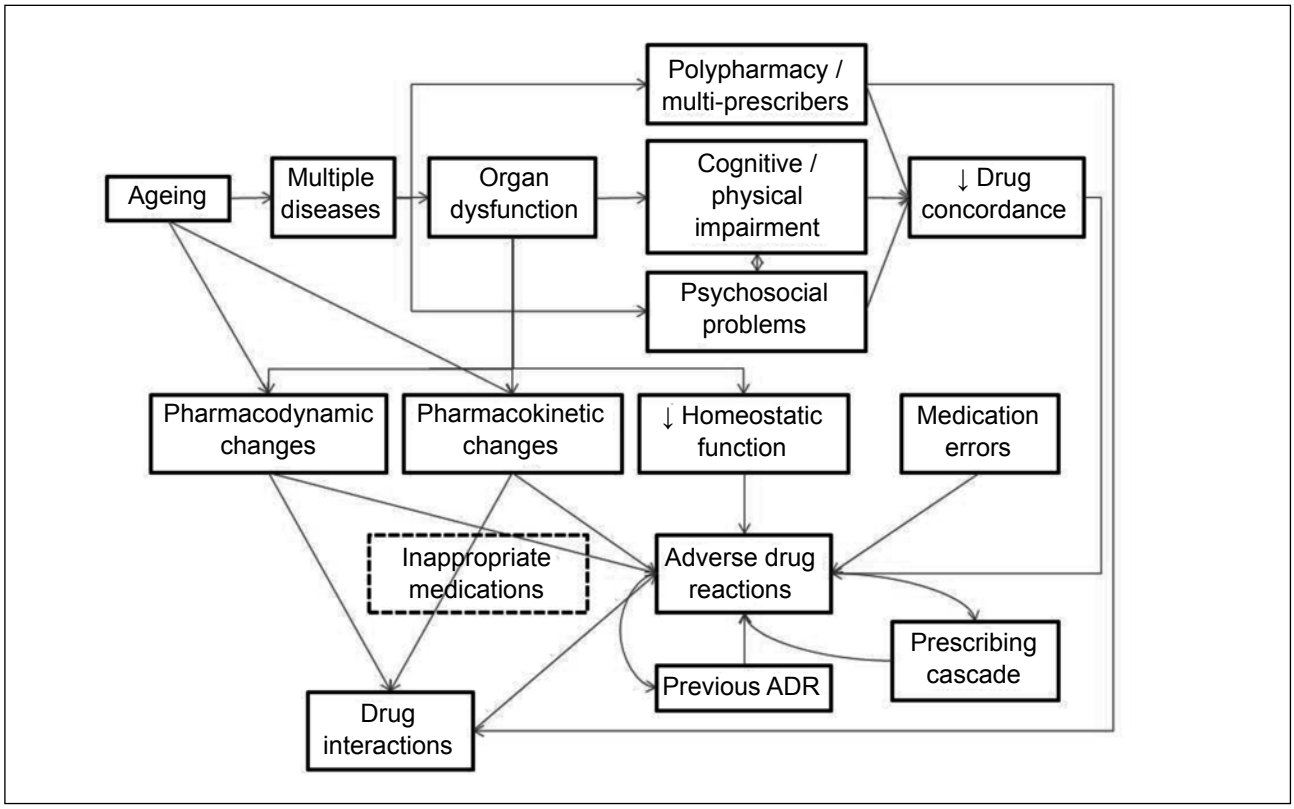

Figure 15.1 Factors contributing to adverse drug reactions in elderly people
Abbreviation: ADR = adverse drug reaction

Table 15.1 Age-related changes in pharmacokinetics and their implications on drug dosing in older people

Pharmacokinetic phase	Age-related change / disease-related change	Implication for dosing	Example
Absorption	• ↑ Gastric pH • Delayed gastric emptying • Altered gastrointestinal motility • ↓ Intestinal blood flow	• Not significant in healthy subjects • Subtle slow rate of absorption in disease states	• Many drugs may have delayed onset of action but the overall therapeutic effect is not affected
Distribution	• ↑ Body fat • ↓ Total body water • ↓ Lean body mass • ↓ Serum albumin level	• ↑ V_D for fat-soluble drugs • ↓ V_D for water-soluble drugs • ↑ Unbound drug levels, but of little clinical relevance	• ↑ $t_{1/2}$ for fat-soluble drugs: diazepam, chlormethiazole, lignocaine • ↓ Loading dose for water-soluble drugs: digoxin, theophylline, aminoglycoside
Metabolism	• Slightly ↓ phase I cytochrome P450 enzymes activity (oxidation, reduction, hydrolysis) • Maintained phase II enzymes activity (glucuronidation, sulfation) • ↓ Liver mass • ↓ Liver blood flow	• No significant change in normal subjects, except in disease states affecting liver functions • ↓ Drug metabolism	• Dose adjustment is not needed in healthy elderly people • No clinical method to estimate dose adjustment in subjects with liver disease • ↓ Liver clearance for drugs with high extraction ratio: chlormethiazole, lignocaine, dextropropoxyphene, glyceryl nitrate, propranolol, verapamil
Elimination	• ↓ Renal blood flow • ↓ GFR, ↓ tubular secretion	• ↓ Elimination of renally cleared drugs	• ↓ Maintenance doses for digoxin, atenolol, diazepam, lithium, morphine, and aminoglycosides

Abbreviations: GFR = glomerular filtration rate; V_D = volume of distribution; $t_{1/2}$ = elimination half-life

account for the total amount of drug in the body if it were present throughout the body at the same concentration as found in the plasma. The equation for the volume of distribution is expressed as:

$$V_D = M/C$$

where V_D is the apparent volume of distribution, M is the total amount of drug in the body, and C is the plasma concentration of drug

As a consequence of decreased total body water, polar drugs that are predominantly confined to the water compartment tend to have smaller volume of distribution. Loading dose of polar drugs should be reduced in order to avoid acute drug toxicity. Aminoglycoside, digoxin, and theophylline fall into this category. The main effect of increased V_D is prolongation of half-life ($t_{1/2}$) following the equation: $t_{1/2} = \ln 2(V_D)/Cl$, where ln is natural logarithm and Cl is clearance of the drug. Hence, increased $t_{1/2}$ has been observed for fat-soluble drugs such as diazepam, chlormethiazole, and lignocaine in elderly people. However, the reduction of V_D for water-soluble drugs tends to be balanced by a reduction in renal clearance with little net effect on $t_{1/2}$. Decreased albumin level is a common finding in patients with malnutrition or uraemia. Reduction in serum protein binding may contribute to increase in free drug level and, hence, the therapeutic and / or toxic effects for drugs that are highly protein-bound. While the clinical relevance of reduced protein binding is probably limited because of increase in free drug clearance,[5] it is worth noting an increased incidence in neurological adverse effects of phenytoin in older adults. This may be associated with decreased plasma protein binding of phenytoin in elderly patients with hypoalbuminaemia, renal disease, or in patients taking concomitant drugs competing for protein binding.[6] Thus, phenytoin toxicity can occur in frail, elderly patients with hypoalbuminaemia even though the total serum drug level is low. Therefore, total drug concentrations must be interpreted with caution in the presence of hypoalbuminaemia that can result in reduced protein binding of drug.[6]

15.2.3 Metabolism

Drug clearance by the liver depends on the liver blood flow, and the capacity of the liver to extract the drug from the blood which is related partly to the metabolising capacity of the liver and partly to the characteristic of the drug. Drugs that are highly extracted by the liver include chlormethiazole, dextropropoxyphene, glyceryl nitrate, lignocaine, verapamil, and propranolol. The liver metabolism of the aforementioned drugs may be limited by the reduction in liver blood flow in elderly people. Drugs with low extraction ratio such as phenytoin, carbamazepine, diazepam, theophylline and warfarin, however, are not affected by the change in liver blood flow. Studies on human liver tissue show that cytochrome P450 activities are largely maintained even in advanced old age.[7] Although there is no precise formula to estimate the liver function with respect to drug metabolism, dosage reduction for hepatically cleared drugs is recommended in patients with significant liver cirrhosis, but not in healthy older adults.[7]

15.2.4 Elimination

Drugs usually become more polar and non-toxic after phases 1 and 2 of metabolism in the liver. Elimination of drugs from the body occurs primarily via renal excretion. The glomerular filtration rate (GFR) declines at an average rate of about 0.8 ml/min/1.73 m^2/year after 30 years of age, though there is marked inter-individual variation. Because of the decline in muscle mass with increasing age, serum creatinine is not a reliable indicator of renal function in old age. Indeed, renal function can still be impaired in an elderly person with a 'normal' serum creatinine level.[8] Several formulae have been developed for estimating a patient's renal function and guiding drug dosing (Table 15.2).[9-14]

Modification of Diet in Renal Disease (MDRD) and Chronic Kidney Disease Epidemiology Collaboration

Table 15.2 Formulae for estimating renal function[9-14]

CG formula[9]
- $eCrCl = (140 - Age) \times Wt \times 1.23 / S_{Cr} \times 0.85$ (if female)

MDRD formulae
- Abbreviated MDRD with IDMS-aligned creatinine assays (for Caucasians)[10]
  $eGFR = 175 \times (S_{Cr} \times 0.0113)^{-1.154} \times (Age)^{-0.203} \times (0.742 \text{ if female})$
- Chinese modification of abbreviated MDRD[11]
  $eGFR = 175 \times (S_{Cr} \times 0.0113)^{-1.234} \times (Age)^{-0.179} \times (0.79 \text{ if female})$

CKD-EPI formulae
- CKD-EPI for Caucasians[12]
  For male:
  $SCr \leq 80$: $eGFR = 141 \times (SCr \times 0.0113/0.9)^{-0.411} \times 0.993^{Age}$
  $SCr > 80$: $eGFR = 141 \times (SCr \times 0.0113/0.9)^{-1.209} \times 0.993^{Age}$
  For female:
  $SCr \leq 62$: $eGFR = 144 \times (SCr \times 0.0113/0.7)^{-0.329} \times 0.993^{Age}$
  $SCr > 62$: $eGFR = 144 \times (SCr \times 0.0113/0.7)^{-1.209} \times 0.993^{Age}$
- Chinese modification of CKD-EPI[13,14]
  $eGFR = \text{above} \times 1.1$ (for Chinese)

Abbreviations (units): Age (years); eCrCl = estimated creatinine clearance (ml/min); CG = Cockcroft-Gault; CKD-EPI = Chronic Kidney Disease Epidemiology Collaboration; eGFR = estimated glomerular filtration rate (ml/min/1.73 m^2); IDMS = isotope dilution mass spectrometry; MDRD = Modification of Diet in Renal Disease; S_{Cr} = serum creatinine (μmol/l); Wt = body weight (kg)

(CKD-EPI) formulae derived from Caucasians have been modified for use in the Chinese to account for possible differences in muscle mass and diet, and these modifications are associated with increased accuracy.[11,13,14] Because the MDRD formula is not precise at higher GFR, estimated GFR (eGFR) computed using MDRD formula is not reported as a numeric value when eGFR is >60 ml/min/1.73 m^2.[15] Most studies show that the CKD-EPI formula is more accurate than the MDRD formula, especially at GFR of >60 ml/min/1.73 m^2.[15,16] There is much debate about which formula should be used for estimating creatinine clearance (CrCl) in elderly patients. A systematic review on methods to evaluate renal function in elderly patients showed that the MDRD formula does not differ appreciably from the Cockroft-Gault (CG) formula in terms of the accuracy with which GFR is estimated.[17] Especially in elderly people, MDRD- and CKD-EPI–derived eGFRs tend to be significantly higher than CG-derived estimated CrCl (eCrCl) and, thus, would often lead to prescription of a higher drug dose.[16,18] Since a renally excreted drug is removed from the body in proportion with the raw GFR (expressed as ml/min as in CG-derived eCrCl) rather than GFR normalised for body surface area (BSA; expressed as ml/min/1.73 m^2 in eGFR from MDRD and CKD-EPI formulae), removal of the BSA normalisation will be necessary when eGFR from MDRD and CKD-EPI formulae are used for drug-dosing decisions, especially in those with low body weight.[16] The US National Kidney Disease Education Program recommends that either a CG-derived eCrCl or an eGFR with BSA normalisation removed are acceptable for drug-dosing decisions.[19] A bedside formula for direct removal of BSA normalisation has been developed[20]:

eGFR (in ml/min) = eGFR (in ml/min/1.73 m^2) × (Wt × 6 + 600) / 1000

where Wt is the body weight

It is important to recognise that all these creatinine-based GFR estimating formulae (including CKD-EPI) have limitations in situations of severe malnutrition or obesity, extremes of body size or age, exceptional dietary intake (vegetarian diet or creatine supplements), skeletal muscle disease, paraplegia, limb amputations, or rapidly changing kidney function[16,19]; all of which are commonly encountered in frail, elderly patients. Also these formulae may not be solely relied upon where precision is required for dosing due to narrow therapeutic indices or high toxicity.[15,19] As accumulation of renally excreted drugs with narrow therapeutic indices such as digoxin, lithium, vancomycin, and aminoglycosides will produce serious drug toxicities, appropriate therapeutic drug monitoring and vigilant clinical monitoring are required for the safe and effective use of such drugs.[21]

According to the US-based NKF KDOQI (National Kidney Foundation Kidney Disease Outcomes Quality Initiative) guideline published in 2002, chronic kidney disease is diagnosed and staged by GFR and evidence of kidney damage. This guideline has been further revised in 2013 by the KDIGO (Kidney Disease: Improving Global Outcomes) CKD Work Group and the updated GFR categories in chronic kidney disease areas are shown in Table 15.3.[15] For drugs excreted unchanged in the urine, dosage adjustment will probably be necessary in patients with moderate-to-severe renal impairment (stages 3-5). For drugs mostly eliminated partially by non-renal mechanisms, dosage adjustment is required, at least in patients with severe renal impairment (stages 4-5).[22] Precautions for prescribing specific drugs in the presence of renal impairment are outlined in Table 15.4.[15,23]

15.3 Age-related Changes in Pharmacodynamics

Different drugs have specific pharmacological mechanisms of action in the body. The ageing process may induce more or less sensitivity to specific medications through changes in the number of receptors, affinity to receptors, and / or post-receptor responses. Elderly people are particularly prone to the unwanted effects on the cardiovascular and / or central nervous systems (Table 15.5).[24]

15.4 ADRs, Iatrogenesis, and Prescribing Cascade

ADRs are defined as any noxious, unintended, and

Table 15.3 GFR categories in CKD[15]

GFR category	Terms	GFR (ml/min/1.73 m^2)
G1*	Normal or high	≥90
G2*	Mildly decreased (relative to young adult level)	60-89
G3a	Mildly to moderately decreased	45-59
G3b	Moderately to severely decreased	30-44
G4	Severely decreased	15-29
G5	Kidney failure	<15

Abbreviations: CKD = chronic kidney disease; GFR = glomerular filtration rate
* In the absence of evidence of kidney damage, neither GFR category G1 nor G2 fulfil the criteria for CKD

Table 15.4 Precautions while prescribing drugs in patients with renal impairment (GFR <60 ml/min/1.73 m^2)[15,23]

Drug class / drug	Precaution (GFR in ml/min/1.73 m^2)
Antihypertensives / cardiac medications	
RAAS antagonists (ACE-I, ARB, aldosterone antagonists, direct renin inhibitors)	Start at lower dose in patients with GFR <45 Assess GFR and measure serum potassium within 1 week of starting or following any dose increase
Beta-blockers	Reduce dose by 50% in patients with GFR <30
Digoxin	Reduce dose based on serum digoxin level
Analgesics	
NSAIDs	Avoid in patients with GFR <30 Prolonged therapy is not recommended in patients with GFR <60 Avoid in patients taking RAAS-blocking agents
Opioids	Reduce dose when GFR <60 Use with caution in patients with GFR <15
Antimicrobials	
Beta-lactam antibiotics (penicillins, cephalosporins)	Neurotoxicity and bleeding disorders when GFR <15 with high doses (dose reduction required)
Aminoglycosides	Reduce dose and / or increase dosage interval when GFR <60 Monitor serum levels (trough and peak)
Macrolides	Reduce dose by 50% when GFR <30
Fluoroquinolones	Reduce dose by 50% when GFR <15
Tetracyclines	Reduce dose when GFR <45
Oral hypoglycaemic drugs	
Sulfonylureas	Avoid agents that are mainly renally excreted (e.g. glibenclamide) Other agents that are mainly metabolised in the liver may need reduced dose when GFR <30 (e.g. gliclazide)
Insulin	Partly renally excreted and may need reduced dose when GFR <30
Metformin	Avoid when GFR <30 Review when GFR <45, consider risk-benefit Probably safe when GFR ≥45 Suspend in patients who become acutely unwell Discontinue at the time of or before an iodinated contrast imaging procedure in patients with GFR between 30 and 60, re-evaluate GFR 48 hours after the imaging procedure, restart metformin if renal function is stable
Chemotherapeutic	
Cisplatin	Reduce dose when GFR <60 Avoid when GFR <30
Melphalan	Reduce dose when GFR <60
Methotrexate	Reduce dose when GFR <60 Avoid if possible when GFR <15
Anticoagulants	
Low-molecular-weight heparins	Halve the dose when GFR <30 Consider switching to conventional heparin or alternatively monitoring plasma anti-factor Xa in those at high risk for bleeding
Warfarin	Increased risk of bleeding when GFR <30 Use lower doses and monitor closely when GFR <30
Non–vitamin K antagonist oral anticoagulants	Avoid when GFR <30 for dabigatran, rivaroxaban, edoxaban; avoid when GFR <25 for apixaban
Lithium	Nephrotoxic with prolonged use even at therapeutic levels Monitor GFR, electrolytes, and lithium levels 6-monthly or more frequently if the dose changes or the patient is acutely unwell Risk-benefit ratio of the drug in specific situations must be weighed

Abbreviations: ACE-I = angiotensin-converting enzyme inhibitors; ARB = angiotensin-receptor blockers; GFR = glomerular filtration rate in ml/min/1.73 m2; NSAIDs = non-steroidal anti-inflammatory drugs; RAAS = renin-angiotensin-aldosterone system

undesired effects of a drug, excluding therapeutic failures, intentional and accidental poisoning, and drug abuse. The reasons why older adults are particularly susceptible to ADRs or iatrogenesis are illustrated in Figure 15.1. Advancing age per se is not associated with increased risk for ADRs. Patient-specific physiological and functional characteristics are probably more important than age in predicting both adverse and beneficial outcomes associated with specific drug therapies.[25] Nursing home patients are, therefore, more vulnerable to ADRs. Of note, a prior history of ADRs was shown to be a strong risk factor for subsequent ADRs, suggesting that some patients might be more susceptible to the negative effects of

Table 15.5 Selected age-related pharmacodynamic changes and their implications on drug dosages[24]

Drug-class / drug	Age-related pharmacodynamic change	Implication
Antihypertensives		
Diltiazem, verapamil	↑ Orthostatic hypotension	Start low and go slow
Diuretics	↑ Sensitivity to drug action	Monitor blood pressure and electrolytes
Anticoagulants		
Warfarin	↑ Inhibition of synthesis of vitamin K-dependent clotting factors	Lower warfarin maintenance dose
Psychotropic drugs		
Benzodiazepines, anticholinergics	↑ Sedation, risk of falls, and paradoxical excitation	Use lowest tolerable doses, and avoid co-ingestion of other psychotropic drugs
Neuroleptics	↑ Extrapyramidal symptoms, postural hypotension and arrhythmias	Use lowest tolerable doses
Analgesics		
NSAIDs, COX-2 inhibitors	↓ Renal blood flow and ↑ renal impairment	Use with caution
Morphine	↑ Analgesia, respiratory depression	Start low and go slow

Abbreviations: COX-2 inhibitors = cyclooxygenase-2 inhibitors; NSAIDs = non-steroidal anti-inflammatory drugs

Table 15.6 Examples of prescribing cascade[28]

Initial treatment	Adverse effect	Subsequent treatment	Subsequent ADR
NSAID	Gastrointestinal bleeding	Proton pump inhibitors	Diarrhoea and *Clostridium difficile* infection
Thiazide diuretics	Hyperuricaemia	Allopurinol	Stevens-Johnson syndrome
Phenothiazine antipsychotic drugs	Extrapyramidal symptoms	Levodopa	Nausea, hypotension, delirium

Abbreviations: ADR = adverse drug reaction; NSAID = non-steroidal anti-inflammatory drugs

drugs because of ethnic, genetic, or cultural factors.[26] Very often, iatrogenesis masquerades as other geriatric syndromes such as incontinence, immobility, instability, falls, intellectual impairment, and failure to thrive. These ADRs are often unrecognised and misdiagnosed as new medical problems.[27] Causality assessment is complicated by issues of polypharmacy and / or inaccurate drug history, and cognitive and communication barriers in the older patients. When new medications are prescribed for the treatment of an ADR, the patient is placed at risk of developing additional ADRs. Examples of 'prescribing cascade' are illustrated in Table 15.6.[28]

The ADRs that result in hospital admissions are usually type A, which is dose-dependent and can be predicted by understanding the drug's pharmacological actions. These are largely due to the use of commonly prescribed drugs. More importantly, they are often preventable.[29] In a prospective study of 513 acutely ill patients of >65 years, in-hospital ADRs were identified in 135 patients. The most commonly observed adverse drug events were acute kidney injury / electrolyte disturbance due to diuretics (25%), falls due to benzodiazepines (18%), acute confusion / falls / sedation / constipation due to opiates (18%), and symptomatic bradycardia / orthostatic hypotension due to beta-blockers (9%).[30] In another large cross-sectional study using a national electronic surveillance system, four medications or medication classes implicated alone or in combination with hospitalisation were: warfarin (33%), insulins (13.9%), oral antiplatelet agents (13.3%), and oral hypoglycaemic agents (10.7%).[31]

Prevention of ADRs can be achieved by avoiding the use of offending drug known to cause ADRs in a patient, and identification of patients who are at risk of an ADR. It is important to stress the need for accurate documentation of an ADR at the time of the event and to provide relevant information to the patient about the ADR. The GerontoNet group, a network of academic departments of geriatric medicine in the European Union, recently developed and validated a practical and simple method of identifying in-hospital elderly patients at increased risk of an ADRs from the GIFA (Gruppo Italiano di Farmacoepidemiologia nell'Anziano; Italian Group of Pharmacoepidemiology in the Elderly) database.[32] The findings of this study confirmed that the number of concomitantly used drugs is the most important risk factor for the occurrence of ADRs. The use of five or more drugs approximately doubled the risk of an ADR, while eight or more drugs quadrupled the risk. Other parameters used in GerontoNet ADR Risk Score are ≥4 comorbid conditions; congestive heart failure; liver disease (alanine transaminase >2 × upper limit of normal); previous ADRs; and renal failure (eGFR ≤60 ml/min/1.73 m^2).[32] However, this tool is yet to be validated locally and in non-

hospital settings. Some experts have criticised the GerontoNet ADR Risk Score for not addressing some important parameters such as presence of potential inappropriate medications or age >85 years.[30]

In addition to type A, the other types of ADRs include:
- type B reactions (non–dose-related, bizarre), which are idiosyncratic, unanticipated from the pharmacological profile, and hence unpredictable, e.g. acute anaphylaxis after penicillin administration
- type C reactions (dose-related and time-related), which are associated with cumulative dose, e.g. hypothalamic-pituitary-adrenal axis suppression by steroids
- type D reactions (time-related, delayed), which occur or become apparent some time after the use of drug, and are usually dose-related, e.g. tardive dyskinesia
- type E reactions (withdrawal, end-of-dose) which occur after drug withdrawal, e.g. myocardial ischaemia after stopping beta-blockers
- type F reactions (unexpected failure of therapy), which are commonly due to drug-drug interactions, e.g. reduced warfarin efficacy due to concomitant administration of phenytoin and rifampicin

15.5 Polypharmacy and Potentially Inappropriate Prescriptions

The term polypharmacy lacks a universally consistent definition. Most define polypharmacy either qualitatively as the use of more than necessary and / or inappropriate medications or quantitatively as the regular use of five or more medications. Elderly people are commonly prescribed multiple medications because of multiple comorbidities. The provision of guideline-adherent medical care increasingly means the addition of more medications to reach disease-specific targets, but this is at the risk of exposing an elderly person to multiple drug-drug and drug-disease interactions that have not been studied in single disease controlled trials. There is evidence that elderly persons do the same or better with careful drug discontinuation. A framework has been proposed to guide the discontinuation of treatments otherwise indicated and this advocates individualised care based on an elderly person's remaining life expectancy, time until benefit, goals of care, and treatment target.[33] Overuse and inappropriate use of medication can be minimised by the use of medications with disease-specific indications, synergistic effect, attention to pharmacological principles, appropriate dosage and duration, alertness to potential adverse effects or interactions, and attention to the patient's need.

Defining an inappropriate medication is challenging — a therapy whose adverse risks exceed its health benefits.[34] Inappropriate medication is a major cause of ADRs in elderly patients.[35] A local study in an extended care hospital showed that 52% of patients were discharged with one or more inappropriate drugs.[36] The most frequent classes of inappropriate medications were drugs for the respiratory and gastrointestinal systems. The commonest reason for inappropriateness was the use of drugs with no apparent indication; followed by inappropriate dose, frequency, or duration; use of duplicate drugs; and use of contraindicated drugs. Appropriateness of prescribing can be assessed by process or outcome measures that are explicit (criterion-based) or implicit (judgement-based). A comparison of different assessment tools has been described elsewhere.[37] These tools are often employed in the research settings on studies of potentially inappropriate prescription. One of the implicit assessment tools — Medication Appropriateness Index[38] — is the most evaluated tool which includes some components of appropriate prescription:
- indicated?
- effective?
- correct dosage?
- correct directions?
- practical directions?
- clinically significant drug-drug interactions?
- clinically significant drug-disease / condition interactions?
- unnecessary duplication with other drug(s)?
- acceptable duration of the therapy?
- the least expensive alternative compared with others of equal utility?

The Beers explicit criteria, introduced in the US in 1991, used a Delphi consensus among geriatric and pharmacology experts who analysed databases of frail, elderly nursing home residents in the US to identify potentially inappropriate medications.[39] Updated versions of Beers criteria were developed for use in outpatient setting, regardless of the patient's residential circumstances in 1997[40] and 2003.[41] The latest Beers criteria were developed by the American Geriatrics Society (AGS) in 2015.[42] It comprised five sub-lists of medications that should be avoided in elderly people; the first lists drugs considered inappropriate independent of any clinical condition; the second lists drugs considered inappropriate for elderly people with certain diseases and syndromes that the drugs listed can exacerbate; and the third lists drugs to be used with caution in older adults. The fourth and fifth lists are new to the 2015 criteria: lists of select drugs that should be avoided or have their dose adjusted based on the individual's kidney function and select drug-drug interactions documented to be associated with harms in older adults. The 2015 AGS Beers criteria are

applicable to all older adults with the exclusion of those in palliative and hospice care, because of the shift in benefit-to-harm ratio in end-of-life decisions and lack of evidence for avoiding drugs in these populations. The limitations of the Beers criteria include several medications which are not available in other countries, and no criteria with reference to underuse of drugs, and duplicate drug classes. One of the strengths of the 2015 AGS Beers criteria is the readiness of incorporation into computerised prescription system which can alert clinicians about a potentially inappropriate prescription at the time of drug prescription.

The Screening Tool to Alert Doctors to Right Treatment (START) and Screening Tool of Older Person's Prescriptions (STOPP) are other newly developed screening tools using Delphi consensus method in Ireland and the UK.[43] The former consists of 22 evidence-based prescribing indicators for commonly encountered diseases in older people and addresses errors such as the omission (for ageist or irrational reasons) of drug therapy likely to be beneficial to patients. The latter is comprised of 65 clinically significant criteria for potentially inappropriate prescriptions in older people and is classified according to physiological systems. It also focuses on the potential problems associated with such prescriptions in the context of the poly-pathology that older people experience. Although START / STOPP takes only 3 minutes to use, further clinical studies on its effectiveness in reducing ADRs are needed.

15.6 Decreased Compliance, Adherence, and Concordance

The conceptualisation and operationalisation of the 'correct' taking prescription medications often reflects the dominant paternalistic approach to healthcare. 'Compliance' is the paternalistic view that the doctor alone makes the treatment decision, while the passive and dependant patient is obliged to comply with the doctor's prescription. 'Adherence', although slightly less paternalistic than 'compliance', is still a paternalistic view that the informed (but still passive) patient will stick to taking their recommended treatment. The use of the term 'concordance' is growing and refers to the process of enlightened communication between patients and their healthcare professional, leading to an agreed treatment and ongoing assessment of this as the optimal course. It is estimated that adherence rates for prescribed medicines are about 50%.[44] Adherence is influenced by patients' beliefs about medication taking, and other factors. For example, these factors include the past experience with medicines, feeling unnatural taking medicines, lack of comprehension on the need to take medicines, long-term risks of medicines, adverse effects of medicines, physician-patient miscommunication, impaired cognitive function, high medication costs, complex medication schedules, and lack of social support. Non-adherence can be classified into intentional and unintentional. Intentional non-adherence is an active decision, occurring when the patient perceives a low health risk of an illness, or has doubts about the drug benefit and / or safety. Sometimes, the patient experiences and knows better about the unwanted effects of a medication than the prescribing doctor, and intelligently foregoes prescribed medications. Unintentional non-adherence, on the other hand, is a passive process whereby patients fail to adhere to prescribing instructions through forgetfulness, carelessness, or circumstances out of their control (e.g. acute illness, inability to open the bottle).

The characteristics of intentional non-adherent medication-taking behaviour are[45]:
- recognition / anticipation of side-effects
- fears of prescribing errors
- lack of faith in prescriber
- failure to accept diagnosis
- fears of addiction
- dislike for taking medicines
- perception of health risk
- testing medicines against symptoms

The characteristics of unintentional non-adherent medication-taking behaviour are[45]:
- forgetfulness
- lifestyle change
- disruption of daily regimen
- period of illness
- drug-related memory loss
- being asymptomatic

Doctors should keep an open mind in approaching non-adherence in elderly patients and be aware of the phenomenon of intelligent non-adherence, which necessitates an open discussion to address their reasons for non-adherence. Tool such as 8-item Morisky Medication Adherence Scale can be applied to evaluate medication adherence in outpatient setting.[46]

A Cochrane systematic review in 2008 showed certain strategies for improving medication adherence.[47] For long-term treatments, more frequent interaction with patients with attention to adherence is more likely to maintain adherence. And like other geriatric syndromes, a multifaceted intervention is often shown to be effective:
- simplifying the dosing regimen
- more thorough patient instruction and counselling
- reminders
- close follow-up

- couple-focused therapy
- psychological therapy
- crisis intervention
- manual telephone follow-up review

15.7 Use of Health Supplements, Over-the-counter Drugs, and Chinese Medicine

Use of traditional Chinese medicine (TCM) is a common practice in Hong Kong. In a recent local population-representative survey of 31762 non-institutional and institutional residents, among those reporting symptoms of medical problems in the 30 days preceding the survey, 1.8% had utilised TCM regularly in the past 6 months prior the survey, 8.8% had consulted a TCM practitioner, and 2.7% had used over-the-counter TCM products.[48] Widespread use of over-the-counter drugs, health foods, and TCM in addition to prescription western medicines were noted in a local study on medication use pattern among 285 community-dwelling older people.[49] Proprietary Chinese medicines (PCM) refer to proprietary products composed solely of Chinese herbal medicines or any materials of herbal, animal or mineral origin, customarily used as active ingredients, that are formulated in a finished dose form. Use of PCM is also very common among local Chinese. It can be obtained from the local community pharmacies without prescription, from the Chinese medicine practitioner as homemade formulae, or from mainland China. Like TCM, PCM is generally safe. In a local retrospective review of patients admitted to the hospital for ADRs secondary to PCM use, the causes of ADRs were adulteration, misuse by the consumer, misuse by prescriber, and drug allergy.[50] PCMs with unknown manufacturer or ingredients were found to be associated with increased risk of ADRs. In Hong Kong, the list of registered PCMs can be found on the website of the Chinese Medicine Council of Hong Kong (http://www.cmchk.org.hk/pcm/eng/#main_dis.htm). PCMs or other health supplements found to contain adulterations/undeclared medicine are regularly under surveillance and updated on the website of the Drug Office of the Hong Kong Department of Health (http://www.drugoffice.gov.hk/eps/do/tc/healthcare_providers/safety_alerts_and_medical_recalls/undeclared_medicines.html).

Despite the fact that older patients may co-administer herbal medicine, health supplements and western medicine, detecting herb-drug, and / or food-drug interactions is challenging.[51] Systematic reviews on interactions between herbs and western drugs revealed that serious consequences are uncommon, except with use of *Hypericum perforatum* (St John's wort) and *Viscum album* (Mistletoe) which may result in transplant rejection, delayed emergence from anaesthesia, cardiovascular collapse, renal and liver toxicity, cardiotoxicity, bradycardia, hypovolaemic shock inflammatory reactions with organ fibrosis, and death. Moderate-to-severe interactions were noted for *Ginkgo biloba*, Panax *ginseng*, *Piper methysticum* (kava), *Serenoa repens* (saw palmetto) and *Camellia sinensis* (green tea).[52] Commonly affected drugs are antiplatelet agents and anticoagulants. For TCM-drug interaction, a useful reference can be found on the Taiwan drug-herb interaction website (http://dhi.cmu.edu.tw/query/).

Toxicity related to the use of TCM, PCM, or health supplements is uncommon in clinical practice. The causality assessment is often complicated by non-specific presentation, unclear temporal relationship, lack of good quality, and the scarcity of primary data. The most dangerous ADR is dysrhythmia following the use of herbs containing aconitum alkaloids such as Radix Aconiti (Chuan Wu), Radix Aconiti Kusnezoffii (Cao Wu), and Radix Aconiti Lateralis Preparata (Fu Zi). These herbs are commonly prescribed for treatment of musculoskeletal pain. Improper decoction of unprocessed herbs has a higher risk of cardiotoxicity. Another commonly encountered problem is herb-induced liver injury (HILI). The belief that herbs and herbal products are harmless is unfounded. Many herbs and herbal products have been reported to cause hepatotoxicity.[53] In a local poison treatment centre which provides consultative service for suspected TCM-related toxicities, two 'highly probable' and seven 'probable' cases were identified among 99 cases admitted to local hospitals for suspected HILI from 2011 to 2013.[54] The causes for HILI in these patients were unclear, with some being idiosyncratic responses with individual susceptibility. A useful reference for the potential toxicity or adverse effects of TCM can be found on the eKG website of the Hong Kong Hospital Authority intranet (http://www.ekg.org.hk/html/gateway/index.htm).

15.8 Effective and Rational Prescription

The Royal College of Physicians of London has published guidelines on effective approaches to prescribing for older people.[55]

- Take a thorough drug history including over-the-counter medication
- Make all reasonable attempts to establish the diagnosis
- Be aware of non-pharmacological treatment options
- Be aware of age-related changes in pharmacokinetics and pharmacodynamics
- Be aware of the limitations of extrapolating the results of clinical trials in younger age groups to older people
- Re-evaluate drug therapy at frequent intervals
- Provide necessary information to help compliance

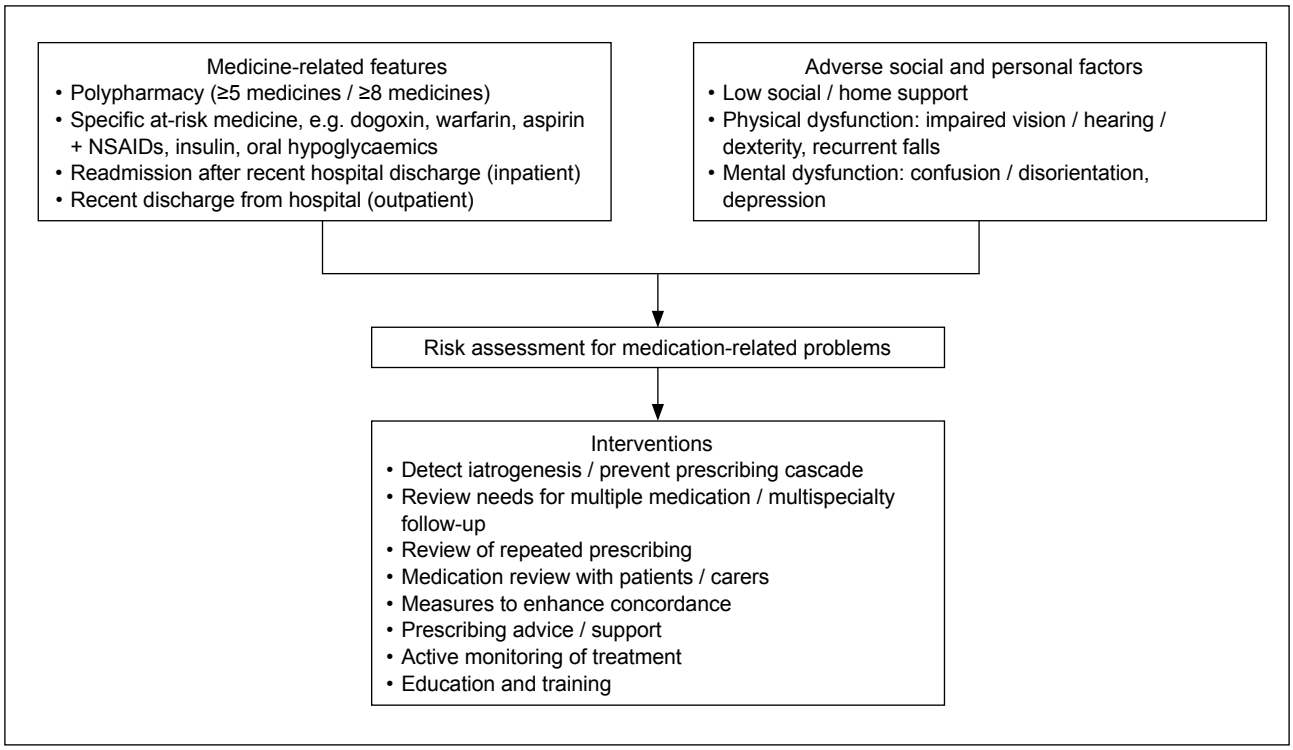

Figure 15.2 Clinical pathway for medication management in at-risk elders
Abbreviation: NSAID = non-steroidal anti-inflammatory drug

Table 15.7 A 10-step conceptual framework aimed at minimising inappropriate medications in older populations[57] (reproduced with permission)

Framework step	Operational strategy
1. Ascertain all drugs	Obtain the list of current drug use by every possible source of information
2. Identify patients at high risk of or experiencing ADRs	Identify all relevant risk factors. Apply risk prediction tools where appropriate
3. Estimate life expectancy	Apply a validated survival prediction rule that best fits patient characteristics
4. Define care goals with reference to life expectancy, level of functional incapacity, quality of life, and patient / caregiver priorities	Goals range from (a) prolonging survival; (b) preventing major morbid events; (c) improving or maintaining function capacity; (d) relieving symptoms
5. Define and confirm existent indications for ongoing treatment with reference to defined care goals	Reconcile current medications with all listed diagnoses in identifying obvious mismatches. Verify diagnostic labels and ascertain current level of disease activity and response to specific therapies
6. Determine time until benefit for preventive disease-specific medications	Review medications aimed at event prevention and compare with estimated life expectancy
7. Determine disease-specific benefit-harm thresholds that may support treatment discontinuation	Estimate the disease-specific absolute risk of events based on patient characteristics (using validated risk prediction rules where appropriate). Estimate the potential treatment-specific reduction in absolute event risk of harm. Determine the appropriate threshold for continuing or discontinuing treatment
8. Review the relative utility of individual drugs	Classify drug utility into: (a) very effective and minimal toxicity for a given indication and few drug-drug and drug-disease interactions. Mortality and morbidity data for older populations consistent and favourable; (b) reasonably effective but some concerns about toxicity in older patients relating to side-effects or drug-drug and drug-drug interactions; (c) questionable efficacy and safety profiles in older patients; (d) minimal effectiveness and considerable potential for toxicity in most circumstances
9. Identify drugs that may be discontinued or have their dosing modified	Discuss with the patient or the caregiver on drug dosage adjustment or complete cessation of the drug(s) which has / have high potential of inducing ADRs
10. Implement and monitor revised therapeutic plan with ongoing reappraisal of drug utility and patient adherence	Set therapeutic goal with the patient and / or caregiver, devise and implement a regimen of drug de-prescribing. Closely monitor the symptom change or disease progression. Implement strategies to maximise patient adherence to the drug regimen. Re-evaluate the therapeutic plan in light of changed patient conditions

Abbreviation: ADR = adverse drug reaction

- Understand the clinical pharmacology of drugs prescribed
- Encourage self-management plans
- Individualise treatment
- Choose treatment endpoints and follow carefully
- Involve the patient or carers in the treatment plan to improve compliance

Like other geriatric syndromes, drug-related problems in elderly people and iatrogenesis are likely benefited from interdisciplinary interventions which involve input from clinicians, geriatricians, pharmacists, nurses, and experts in bioinformatics. A clinical pathway for medication management for at-risk elders is shown in Figure 15.2.

A Cochrane systematic review in 2012 provided limited evidence that interventions such as pharmaceutical care, a service provided by pharmacists, may be successful in ensuring that older people are receiving the right medicines and in reducing medication-related problem. It is not clear, however, if this always results in clinical improvements.[56]

Reducing the number of currently prescribed drugs may be difficult in many patients and may inadvertently increase the risk of morbid events or decrease the quality of life unless performed with care and under close supervision. In order to alleviate this problem, a 10-step conceptual framework is proposed to rationalise drug use and reduce the number of inappropriate drugs prescribed for older people (Table 15.7).[57]

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CHAPTER 16

Psychosocial Aspects of Geriatric Care

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OBJECTIVE

This chapter provides an overview of social gerontology and its implications for elder care. The primary objective of social gerontology is to equip members of the healthcare team as well as other elder care professionals with an understanding of the psychosocial aspects of elder care. This chapter provides not only knowledge and skills needed to provide such care but also a global and philosophical perspective on ageing. It will address the clinical significance of social factors as they relate to the care of individual elderly patients, including societal attitudes, effects of social background and life experience, family relationships, caregiver stress, elder abuse, social isolation, social deprivation, and social networks and support. It will illustrate with case examples clearly demonstrating elders' strengths and the role of helping professionals in the process of care.

KEY POINTS

- Provide assessment and treatment to older patients with empathy.
- Treat older patients as you expect others to treat your parents.
- Understand ageism and its impact on healthcare providers' attitude towards patients and the quality of care they provide.
- Understand that older patients may have internalised ageism and believe disease is a normal part of the ageing process. They may thus delay or refuse treatment.
- Understand that a patient's psychosocial situation may affect his / her motivation to participate in medical treatment and the rehabilitation process.
- Examine the patient's medical needs from a diagnostic to a holistic approach.
- Support of the patient's family caregiver is an important step in the preparation of geriatric patients' post-discharge care.
- Identify elder abuse and assess for changes in an elder's functioning.
- Refer geriatric patients to social work professionals as early as possible for psychosocial care, clinical support, discharge planning, and post-discharge care.

16.1 Social Gerontology and Elder Care: an Overview

Gerontology is the study of ageing that includes all academic disciplines and fields of practice. Gerontologists include researchers, academicians, and practitioners in many fields such as medicine, nursing, dentistry, physical and occupational therapy, psychology, psychiatry, sociology, economics, political science, and social work.[1] The term social gerontology has been used to describe the area of gerontology that is concerned with the psychosocial and cultural aspects of ageing. Gerontology differs to geriatrics that is the study of prevention and management of the diseases of ageing. Gerontology is a multidisciplinary field, and gerontologists see ageing in terms of four different, but interrelated, processes. As indicated in Figure 16.1, an elder's well-being is influenced by his / her past life experience, physical health, mental health, social support, losses and stress management, and psychological and spiritual ways of coping.[1,2] Specifically the four processes are as follows.

- Chronological ageing refers to the number of years that people have lived.
- Biological ageing refers to the physical changes we experience as we age — the reduced capacity and efficiency of some organ systems and the stability of performance of others.
- Psychological ageing refers to the stability and changes to intelligence, brainpower, perceptions, mental functioning, memory, personality, motivation, wisdom, and coping with stress and losses.

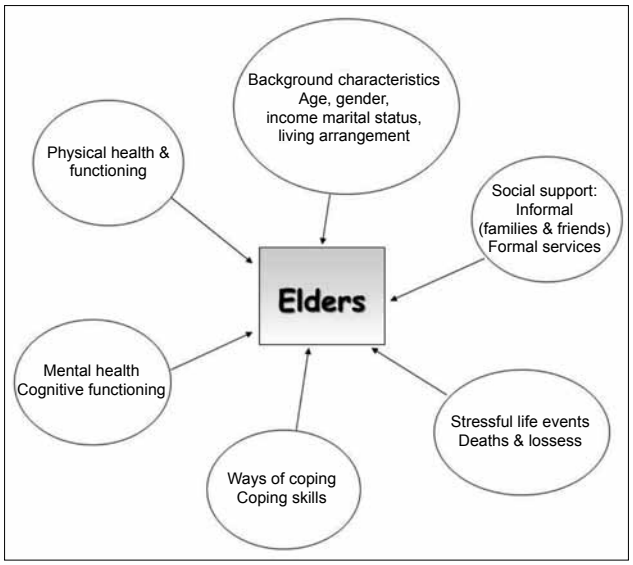

Figure 16.1 The biopsychosocial perspective of ageing[1,2]

- Social ageing refers to the changing roles and relationships experienced over the life course, and the network size and quality of the support system, including both the family support system and the formal support system.

These processes are interrelated and do not occur in isolation. For example, an older person who has a healthy lifestyle — with adequate nutrition and exercise, a positive attitude, adequate financial resources, and strong social support throughout life — may slow his / her biological ageing. In case of the sudden onset of illness, an older person with a positive attitude and strong social support system may be able to recover faster than one with a negative attitude and / or poor social support. In addition, healthier older persons have more options for social roles in old age, such as serving as grandparents or volunteers. Changes in one area cause changes in another. A change in social status can affect psychological status — for example, an older man may become depressed after losing his wife and social support. Psychological changes may impact physical well-being. If a depressed older man loses his appetite and does not eat well, he may experience dizziness and then fall, sustaining a hip fracture. This change in physical health may precipitate another change in social status as he moves in with his son's family, who is supposed to help him recover from the sense of loss he is experiencing due to his wife's death, as well as from malnutrition and the hip fracture. The psychological outcomes of this care arrangement depend on how well professionals and family members understand the man's psychological reaction to and interpretation of all these losses / stresses and the care arrangements. He may be satisfied if he agrees to the arrangements, or he may experience varying degrees of unhappiness and frustration if his family makes all the decisions for him without his involvement. His psychological and emotional reactions depend on how he feels, interprets, and perceives his social reality. If he feels that he is losing control, decision making power, or independence, his depression will be exacerbated.

People are biopsychosocial beings. In caring for elderly people, social work professionals need to pay attention to a patient's physical, psychological, and social needs and provide interdisciplinary support. The team of professionals need to understand the separate processes of the biological, psychological, and social aspects of ageing and be able to work together to provide services and plan programmes both with and for the elders. In addition, they must learn not only how these ageing processes interact with each other but also how an elder adapts to his / her environment. Physically, elders may have different levels of functioning and health conditions. Therefore, social work professionals offer different kinds of programmes and services based on the level of need and functioning of the ageing individual along the wellness and illness continuum (see Figure 16.2).[2,3] Most elders are healthy, and services to these functional elders can be preventive in nature. The role of the social work profession in other care settings such as hospitals, home care situations, nursing homes, and end-of-life programmes is often unique because social work's primary focus is the psychosocial well-being of the elder and their family. Social work professionals understand the interaction between an elderly individual's physical, emotional, and social needs and the adaptations necessary to change the environment to meet those needs. This is also called the person-in-environment perspective.[1] For example, consider a discharge planning arrangement for a male stroke patient who lives alone. Upon discharge, he is able to walk with a walking frame but will not be able to cook for himself. Before this patient is discharged from hospital, the social worker, in consultation with the multidisciplinary team, may need to carry out a home assessment to determine whether he can manage the activities of daily living with a walking frame. The resulting aftercare plan might include home care, home-delivered meals, a community nurse, inpatient or outpatient rehabilitation, accessible transportation to increase community engagement, and a volunteer to check on him regularly to reduce his social isolation. The social worker also provides supportive counselling to the patient to empower and motivate him to maintain adequate self-care and maintain close contact with the social worker to participate in the post-discharge care plan.

Gerontology is a relatively young discipline compared

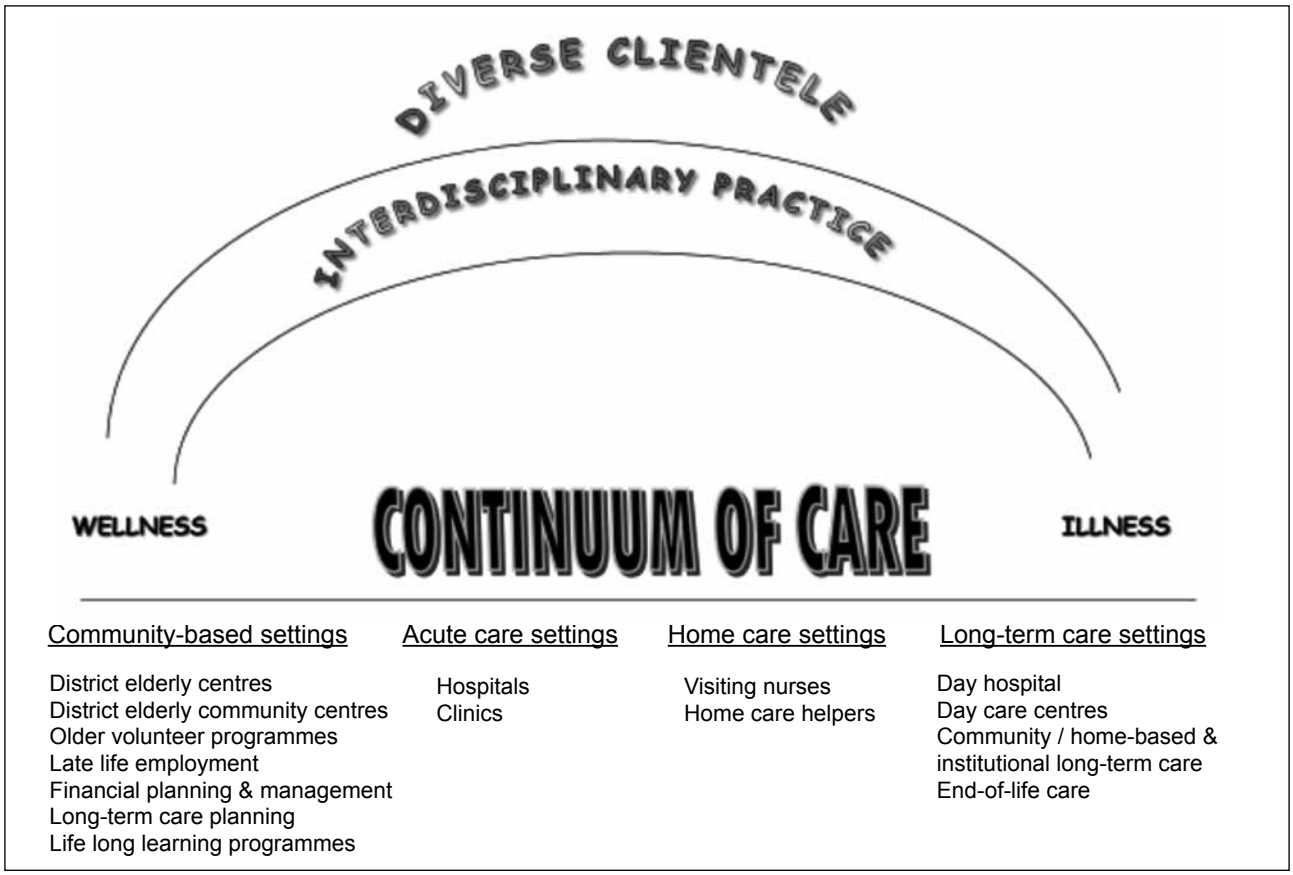

Figure 16.2 Care continuum: from wellness to illness[2]

with other professional fields of study that focus on the human lifespan. Medical and social scientists began ageing research only a few decades ago. In the US throughout the 1970s and early 1980s, interest in gerontology and geriatrics grew by leaps and bounds because of the explosive growth of the ageing population. Despite this synergy, many researchers remained preoccupied with disability and disease, rather than with the positive aspects of ageing. This may be one of the reasons the society has previously stereotyped old age as a life stage characterised only by the 4Ds (disease, disability, dementia, depression). This negative focus of most researchers on ageing in the 1970s and early 1980s meant that study of the effects of lifestyle and other psychosocial factors on the well-being of older persons was neglected. More recently researchers have reported that the majority of older people find the ageing process to be positive, healthy, productive, and satisfying.[4] Gerontology faces a major hurdle, namely, the historically negative view of ageing promoted by both science and society. This ageism has a significant negative impact on our elders.

16.2 Ageism and Its Impact on Elders and Programmes

In society, there are many barriers to working with elders, and the worst of them is ageism.[3,5] Ageism refers to the prejudices against and stereotypes of older persons based solely on their age. Many people perceive ageing negatively, as a time of significant decay, decline, loss, and reduced productivity. As a whole, society fears and misunderstands the normal ageing process and is often biased against older people as a group. These negative stereotypes usually convey the attitude that older adults are less valuable as human beings, thus justifying their inferior or unequal treatment. Most healthcare workers can be guilty of some degree of ageism. There is evidence that medical professionals are less likely to provide vigorous treatment to older patients if they deem them too old and infirm to benefit.[1,5] If healthcare providers can see older patients from a strengths perspective, they may think about them differently. Negative attitudes towards older people may be based on previous experience, what is reported in the media, personal fears of ageing, and cultural stereotypes. Such attitudes are also based on myths. Healthcare professionals need to examine and change their negative attitudes towards the ageing process if they are to be effective in treating not only older people, but a person of any age who faces barriers to healthier functioning in society. The negative stereotypes of ageing that society presents

should also be addressed. For example, many people believe that older adults are frail and stubborn. What many perceive as 'frailty' is often a patient's cautious reaction and adaptation to mobility change. What may be construed as 'stubbornness' often tends to be a time-limited fearful reaction to treatment decisions in which they were insufficiently consulted. In reality, there is evidence that more than 80% of the older populations are healthy and open-minded. Many of them are active, enjoy exercise, and are ready to meet new challenges.[4]

Subconscious latent judgements are often made about older people. Care providers who see frail or depressed older people every day may come to believe that they are seeing people who are ageing normally. This negative image can inhibit a healthcare provider from approaching each new patient with an open mind and can prevent him / her from being able to focus on an older person's strengths. Ageist attitudes develop early in life as children observe parental, media, and social attitudes towards elders. Some parents, who have not addressed their personal emotions and long-standing issues in their relationship with their own parents, may unintentionally send the message to their children that ageing persons in their household are a burden of care, and demanding, needy, stupid, or useless. Even simple remarks such as "I hope I never get like grandma" or "Grandma is too old to change" convey a negative message. Every time parents refer to their own aches and pains as "I must be getting old" or refer to normal forgetfulness as "a senior moment", these subtle messages become clear — that ageing is destined to be painful, and full of illness and disabling conditions. It is important to encourage and educate care providers that underlying such messages is the false belief and personal fear that these issues will inevitably get worse as a person ages.

When the media emphasises the importance of youth, and shows older people in nursing homes and wheelchairs, children and adults will find it difficult to develop a positive attitude towards ageing as a process, and elders as valuable members of families and society. To ensure that older people feel important, and avoid promotion of these negative stereotypes, these negative images about older people must be resisted and professionals and the general population must be educated about the difference between the normal ageing process and the disease process.

During normal ageing, disease is not inevitable. Disease is a different process and people of any age should consult medical doctors for disease treatment. Incorrect and biased attitudes about ageing can affect not only how professionals and society treat elderly people, but also how they view themselves. Elderly people who adopt these negative myths about ageing may become passive and thus limit themselves.

16.2.1 Impact of Ageism on Elders

Ageism makes people feel that there is something wrong with growing old and contributes to a sense of personal and societal hopelessness of the ageing process. Research suggests that ageism has a significant negative impact on older people,[1,2,5] as described below.

Older persons who believe the negative stereotypes associated with ageism are less likely to actively participate in rehabilitation programmes if they do not believe they can recover. They may not welcome choices if they do not believe they are valued or loved by their family or society. Ageism can often affect the choices that are offered. If healthcare professionals believe that some diseases are part of the normal ageing process, timely and active intervention is less likely.

Increasing the level of exercise and mobility, even in those aged >70 years, can have significant effects on functional ability and quality of life.[6-9] Lack of understanding about the ageing process or the belief that decline is inevitable can lead family members and healthcare professionals to withhold vigorous treatment. Psychosocial risk factors such as stress, elder abuse, isolation, and depression can and should be treated as soon as possible, regardless of the patient's age. If an older person or their family believes that depression is part of the normal ageing process, neither the older person nor the family will seek mental health treatment.

When care providers or family members believe that an older person's range of physical and cognitive abilities will deteriorate, their expectations of the individual may be lowered. These restrictions and lowered expectations may reinforce dependent behaviours in older persons (learned helplessness). This behaviour of the care providers and family members may best be conceptualised as "disabling support" versus "enabling support."[1,2]

Ageism can affect the quality and quantity of professional training in all disciplines, for example, medicine, physical therapy, occupational therapy, nursing, and social work. If professional programmes do not recognise the importance of gerontology and geriatrics, their curricula are likely to be deficient in the ageing content. If all licensing examinations for health and social service professionals required a basic knowledge of gerontology and geriatrics, service providers would be better equipped to work with elders. Then, older people — our parents and grandparents — would be treated by competent

healthcare professionals whose knowledge of the ageing process is evidence-based.

Ageism can affect the content and direction of programmes for elders. Older people, just like their younger counterparts, benefit greatly from health prevention, health promotion, and health maintenance programmes. If service providers do not understand normal ageing as well as disease processes, they will be less likely to be optimistic about a patient's prognosis and their likelihood of recovery from any disease. Although changes in physiology are a part of ageing, evidence indicates that many disease processes can be modified and treated.[1,2,5,9]

The latest research on ageism has shown that the more gerontological and geriatric training professionals receive, the less likely they are to have ageist attitudes.[1,2] Research has also established that older people are very resilient. If they are empowered and encouraged to have a positive attitude, they will live longer and be healthier.[1,2] A strong, healthy mind can help a person maintain a healthier body, and in the event of disease, help cope with a failing body. Normal ageing can be a very positive experience, and whether or not it is positive for a given individual depends on how that person deals with stress and losses as he / she ages. In human experience, the nature of stressful life events cannot always be changed, but a positive attitude to live and to cope with adversity and with courage can be adopted. Healthcare providers need to adopt a positive attitude towards ageing and ageing patients. Below is a real-life story of positive ageing:

> An 88-year-old woman, Mrs Wong, had multiple, significant losses in the preceding 10 years. She lost her husband to cancer when she was 78, and her only son to a car accident 2 years later. She lost her vision 3 years ago due to glaucoma, and both of her legs due to a diabetic condition the following year. Then last year, she lost her house due to a neighbourhood fire. Mrs Wong was living in church housing when a social worker first met her and tried to provide her with supportive counselling. To the social worker's surprise, this woman told her that she was thankful that she was still alive as she had her church family, she had a wheelchair, and she had meals-on-wheels service. She also said she had God to be with her every minute of every day. Eager to help other older people who had many similar losses, she served as a volunteer for a telephone assurance support group. In this position, she called other elders and kept them company by phone every day. She remained emotionally and socially connected with her friends and church family and was very content with what she had and what she could do to help.

Regardless of functioning status, life adversities and health conditions, ageing can be a positive, meaningful experience if one chooses to have a positive attitude and live courageously. Those trained in ageing fields can provide critical support, helping their older counterparts to cope and live with meaningful goals as well as have meaningful activities, like Mrs Wong. Ultimately, through volunteering, ageing citizens can provide critical support to society in times of need.

16.3 Normal Ageing Versus Disease Process

In studying gerontology, it is important to distinguish normal ageing changes from disease processes. Disease is not part of the normal ageing process, and it requires medical attention regardless of a patient's age. Normal ageing change is not a disease. When my mother's hair turns gray, I do not have to take her to a doctor because it is a normal ageing process, not a disease. However, many people treat diseases in an older person as part of the normal ageing process, leading them sometimes to delay treatment that would greatly jeopardise that older person's quality of life. Prevention of disease, early intervention, and early treatment are always cheaper and better than negligence and ignorance. In an elderly individual, arthritis is a disease, not a normal ageing process. Disease is disease, regardless of the patient's age, and a diseased patient must consult a doctor because the disease requires medical attention.

Disease processes are strongly influenced by the effects of environment, lifestyle, and disease states that, in turn, are related to ageing but are not due to ageing itself.[1,2,5] In this context, common definitions of ageing rarely distinguish between normal ageing and disease processes. For example, ageing has been perceived as a "progressive, generalised impairment of function resulting in a loss of adaptive response to stress and in a growing risk of age-associated disease." The active ageing, positive ageing, and productive ageing concept, promoted by the World Health Organization (WHO) and gerontologists around the world, encourages the "process of growing older without growing old through the maintenance of physical, mental, social, and spiritual activities throughout a lifetime."[10]

In recent years, there has been a trend to study the concept of healthy ageing. For many years, gerontologists were concerned mainly with the distinction between pathological and non-pathological states. That view of ageing has unacceptably narrow implications: it implies that in the absence of some identifiable clinical disease or disability, all is well. It also implies that in the presence of disease, some kind of personal failure has occurred. Certainly, freedom from disease and disability is an important component of healthy ageing. The WHO also defines health as "a state of complete physical, mental, and social well-being and not merely the absence of

disease, or infirmity."[11] The WHO definition implies that absence of disease is not enough. A person who is not ill may nevertheless be at serious risk of illness or disability. And a person who is not at risk in these respects may nevertheless be living a lonely, inactive old age. A strong, healthy mind can help an older person to age successfully because willpower and inner strength can make it easier to cope with any limitation and disability.[12] Therefore there is a belief that elders should be considered healthy if they can successfully overcome environmental stresses and cope with adversity.

There are also times when it is the environment that is disabling, not the person. If the physical environment is adapted to be age-friendly and user-friendly, disabled people can cope with more autonomy. For example, studies have shown that a blind person can cross the street safely within the time frame of a popular song such as "London Bridge Is Falling Down" played by the traffic light. If the end of the song indicates the end of the time allotted to the street crossing, the blind person can pace himself / herself and manage to cross the street independently. Alternatively, the blind person can decide whether he / she should start the crossing if the remaining time appears to be too short. Gerontologists consider a person to be healthy if they are content with life and have access to internal and external resources to cope with physical disability or psychological losses and live well.[2-4]

16.4 The Strengths Perspective in Elder Care

As noted previously, older people are extremely resilient. Resilience in old age may be best thought of as "the ability to survive and overcome stressors, including disease, disability, and a range of other life transitions and losses."[12] Contributing factors include the ability to reframe the meaning of stress, cope with stress and loss with a positive fighting spirit, maintain a sense of mastery, demonstrate flexibility, and draw on the resources of social support. Research suggests that one's sense of personal control contributes to psychological well-being.[13-15] Resilience is fostered by interdependence with others and grounded in the ability to adapt to changing demands. Finally, resilience is not the cheerful disregard of difficult and traumatic life experiences; neither is it the naive discounting of life's pains. It is, rather, the ability to learn to cope with stressful life events and treat them as life lessons.

An empowerment orientation to social work directs healthcare professionals to search for and enhance resilience and strengths among older persons, especially in times of disease and disability. Even for older adults who seem to have given up, fostering resilience is a catalyst for hope.[16,17] How can individuals prepare for ageing? How can they cope with age-related changes and challenges? One way is to accept life as a progression through stages and see each stage as having not only its own challenges, but also its own rewards. Life courses accumulate unique and meaningful experiences. No matter how sick or frail older people may be, they will always have strengths and meaning.

The extreme resilience of human beings cannot be overemphasised. Most elderly individuals are especially strong because they have had a lifetime of experience dealing with life challenges. Many are able to cope with extreme adversity and difficulties, while continuing to give to others. It is essential for current and future generations to understand that growing old is a natural event of which there is nothing to be afraid. People need to learn to anticipate older age because new roles and new opportunities will arise. Older people can be pillars of the extended family and serve many productive roles such as caretakers of family members in need. In the US, more than 60% of the general population give their time and professional expertise by serving as volunteers and sitting on boards of social service agencies and community organisations.[2,3] In recent years, many retired healthcare professionals in the US have started their own community health clinics with help from foundations so that they can provide free medical care to the uninsured, poorer people within their community.

The majority of older populations are vigorous and stronger than they once were. People in their 60s, 70s, and 80s are doing things they would not have done 30 years ago. The rhythm of the life cycle is also changing. For example, some older people marry, divorce, and remarry. Some change careers in their 60s, or return to school in their 50s and 60s. These trends open up older people's lives to richer social experiences, sometimes making age irrelevant as a predictor of what a person might be doing. Such social changes challenge researchers to review past studies of how people age and how they die. New research should measure the way older people overcome age norms to act differently and courageously against the expectations engendered by outdated cultural norms.[1-3]

16.5 Professional Ethics and Attitudes towards Elderly Patients

The following are the foundations of social work principles that guide the provision of services to elderly people. In the healthcare setting, the entire healthcare team, not just the social worker, should endorse these professional principles because they are important professional ethics and values for any

discipline.[2,5]

- Acceptance: Conveys a positive regard for a patient's strengths and potential for growth
- Individualisation: Affirms each patient's unique and distinctive characteristics
- Non-judgementalism: Maintains a respectful and non-blaming attitude towards patients
- Objectivity: Promotes professional caring, concern, and commitment in working with patients
- Confidentiality: Respects a patient's right to privacy
- Accountability: Ensures competent professional conduct

In addition to these generic social work principles, character traits such as empathy, sensitivity, empowerment, and self-determination are very important to social work ethics and skills, especially when working with elderly patients in a healthcare setting.[2,3,5]

16.5.1 Empathy and Sensitivity

Empathy is the ability to relate to another person's emotions, thoughts, and feelings. It differs to sympathy that implies feeling sorry for a person. Although the difference is subtle, it is important. Along with empathy comes understanding. A social worker who understands what it is like to struggle with physical and mental impairments will have more patience and respect for an elderly patient. It is helpful to imagine experiencing the same problems personally, and this is the notion of 'walking in their shoes'. It is the ability to imagine oneself sick and the care that would be expected from professionals. Alternatively, we can imagine that our own mother becomes ill and consider how we would expect healthcare professionals to treat her. Empathy produces patience, a willingness to listen, and non-judgemental attitudes because one can only imagine what is like to be disabled short-term or even permanently.

Sensitivity is the ability to communicate with elderly patients according to their comfort level. For example, discussing physical health problems with a social worker (stranger) may be particularly uncomfortable for elders. For an elderly woman who has been very modest about personal matters such as bladder and bowel functions, discussing these topics with a stranger may be awkward. The same is true for an elderly man who does not feel his difficulties with maintaining an erection or urinating are a social worker's business. Matters that have been private for elders all their lives tend to become topics for open discussion when social work and other helping professionals get involved. While a solid understanding of an elder's health issue is essential to the development of a care plan, it is important to be sensitive to the feelings and emotions associated with the discussion of these deeply personal health issues.

16.5.2 Empowerment

Empowerment refers to recognising and believing in one's own ability to make decisions and carry out actions to meet self-needs, solve one's own problems, and organise the resources necessary to take control of one's life.[5] An empowering care professional includes older patients in the process of making decisions regarding all options, including the risks and benefits of each. In the educational process, it is imperative that the language used is easily understood yet not condescending, and that information is presented clearly, with all options laid out in a patient manner. Problem-solving recognises the older person's intellectual ability and conveys the worker's belief in the elder's ability to be a full participant in decision making, hence reinforcing the person's belief in their own power and worth. Frequently, older persons are not included in the making of choices, but rather information is presented to family members: "Your mother must go to a nursing home" is all too often the decision offered by well-intentioned healthcare professionals. This approach disempowers patients by excluding them from the decision making process. Research has shown that elders are more likely to feel depressed if they perceive a loss of control or have no choice in the care plan and / or direction of their life.[13,15,17]

16.5.3 Self-determination

The right to self-determination is the most important ethical principle of many helping professions. The right of individuals to determine what is in their own best interest, with certain provisions, is also supported by legal precedent. Self-determination presupposes having options and then having opportunities to make decisions about those options freely rather than by coercion.[2,5] Being able to exercise choice is fundamental to empowerment and plays a significant role in personal sense of well-being. For example, a Swedish study of the daily life of the 'oldest old', living independently with the assistance of community-based support, points to the importance of directly involving patients in decision making.[2,3] The researchers found that respondents who exercised more control over their own lives were better adjusted. In contrast, those who were not as well-adjusted were more likely to be controlled by external forces than by their own choices. The study concluded that plans for caregiving interventions should be driven by the personal preferences of the participants.[3,5]

In western societies, the right to self-determination tends to cease when direct actions will result in death.

Although there is recognition of the right of the individual to refuse treatment, even when the refusal has the likely consequence of death, society continues to grapple with issues of suicide and assisted suicide, even in the face of painful, terminal illness. There are limitations to self-determination in any culture and society, with proponents on both sides of the issues equally committed. The recognised and professionally sanctioned right to self-determination binds helping professionals ethically to help older persons exercise their right to make decisions regarding their own lives.

It is not unusual for families and institutions to bring pressure on an older person through the presentation of 'options' for them to choose from in long-term care decision making. Often, the older person is persuaded and at times coerced to select one option over others, even to the point of presenting information in such a way as to indicate that their own choice is the only option. An elderly person who is at risk of falling but who insists on staying in their own home may be referred to as stubborn, uncooperative, selfish, unfeeling, and worse, with the underlying intention of these statements being to intimidate and undermine their choice. Many times this is done out of a caring notion, but if professionals do not first address their own fears and biases about the elder's right to remain at home, they are engendering ageism. Nonetheless most family members and health and human service professionals believe they are operating in the best interests of the individual. Putting aside the 'options' paradigm for deciding what is in the best interest of another human being may well enable professional to begin viewing the issue from the perspective of the one whose life is being affected. It is then much more likely that the elder, caregivers, family, and healthcare professionals will explore the issues together with less bias, and the ability to find a solution consonant with the desires, wishes, and well-being of the older person will become much more likely.

Regardless of whether healthcare professionals or family members are in agreement with an elder's decision or the basis for that decision, self-determination is an individual right. Making decisions on who is allowed self-determination based on the acceptability of that person's views opens the door for neglect or abuse of any patient based on the beliefs of healthcare professionals. At this juncture, the right to self-determination, provided the rights or welfare of others is not compromised, is a non-negotiable patient / client right, with the exceptions of suicide and assisted suicide.

Although other professional responsibilities affect the quality of care and services provided to older persons, empowerment and self-determination are among the most important. As professionals deal with psychosocial, cultural, health, and mental health issues regarding older people, it becomes apparent that empowerment and self-determination are critical components to all. As older people grapple with the issues of raising grandchildren, maintaining intimate relationships, dealing with long-term care, and finally, the death of loved ones and of themselves, the ability for older persons to make decisions that 'fit' them comfortably is of paramount concern. Finally, for social programmes and policies to be innovative, accessible and effective, they need to be empowering and recognise older people as critical decision makers. If older people are to exercise their right to self-determination, they must first be empowered to do so, and that empowerment must be firmly embedded in the ability of all involved to commit to their basic right to self-determination.

16.6 Social Work Techniques to Help Elderly Patients

As well as allowing older persons to make decisions related to their lives or care, there are practical skills such as encouraging them to tell their life story and using these stories to empower them; recognising strengths / expertise; and providing choices / honouring self-determination that can empower elderly patients. Understanding how to use these empowering skills in working with elders will enable them to be stronger and more positive.[2,5] Besides social work professionals, other members of the healthcare team will benefit from these skills to connect / communicate with older patients. When patients feel emotionally connected with professional, they will be more capable and emotionally stronger to deal with their health problems.

16.6.1 Use Life Stories to Empower Elders

Life stories provide patterns of connection and form the basis of human identity. Cultural and personal stories are often profound sources of strength, guidance, stability, comfort, or transformation, and are often overlooked, minimised, or distorted. The stories that are important to people represent the reservoir of their strengths. Reminiscences reveal the storyteller's abilities, characteristics, past accomplishments, networks of support, and survival skills. To recognise strengths in older adults, particularly those whose health is frail, a vision of their resourcefulness is needed, unobstructed by ageism. To access the power of life stories, the processes of reminiscence and life review must be understood. Finally, to understand the significance of life stories, professional should draw on the narrative perspective with a sense of optimism and hope. Erikson et al suggest that the ultimate foundation for hopefulness is the legacy

of our infancy and trusting relationships with our parents.[1,2] They make clear the significance of life stories as a way to realise a sense of integration in the whole perspective of the cycle of life.

16.6.2 Use of Reminiscence to Strengthen Elders

Reminiscing is the process of recollecting experiences and telling (and retelling) stories that capture the storyteller's perceptions and interpretations of past life. When we reminisce, we remember, and through our memories, we create ourselves, both for ourselves and in relationship to other people. Through that self-construction, consistency is added to life and to sense of self — and through that consistency, a sense of emotional protection. As such, reminiscing provides meaning and continuity to lives. Even if not factually accurate, reminiscing and the narration of memoir provide powerful metaphors for what is believed to be significant of life experiences. Clearly, reminiscence has face value, and several studies suggest additional benefits. Research reports on the results of these studies identify the role and function of reminiscing.[1-5] Among the dimensions identified are problem-solving, or tapping recollections of past problem-solving activities; social bonding, or using stories as ways to connect and reconnect with others; maintaining intimacy, or using recollected images of persons to remember the warmth of emotional bonds in their absence; and teaching and informing, or sharing life stories as ways to communicate morality messages. Interestingly, reminiscence for those who can no longer communicate effectively in words seems to involve powerful images of context and emotion. Memories carry all of our emotions.

16.6.3 Life Stories and Reminiscence are Therapeutic Tools

There is an intricate relationship among life stories, reminiscing, and narrative. The stories we tell about ourselves, of necessity, invoke certain types of memories. When those retrieved memories take the form of personally experienced episodes from the past, this is reminiscing. When reminiscences are logically conjoined and sequentially patterned, there is a form of simple narrative. The telling of a life story, and imbuing it with meaning is a construction of the human mind. The way we represent what we know to ourselves does not correspond to a given external reality that exists independently of our knowledge. We are born into stories: the stories of our parents, our families, and our culture. Their meaning can be constraining, even imprisoning, or they can be freeing and liberating. Everyone has a story to tell, and most stories are a mixture of pain, suffering, frustration, and shame on the one hand and pleasure, joy, satisfaction, and pride on the other. Yet often, people who risk revealing their stories to others find relief and, sometimes, even release. They have often felt trapped in their respective stories, and the simple act of telling them to others brings new meaning and perspective to their lives.

16.6.4 Identifying Strengths and Expertise

There are many ways to characterise strengths and expertise — capacities, competence, and inventiveness, to name a few. Sometimes expertise is obvious; other times it is hidden or undiscovered. Even when people run into difficulties, they still have strengths and expertise, at least the expertise of being the most qualified expert about their own situation. For social professionals, fostering patient expertise can have a profound effect on work with them. As a tenet of empowerment, it creates a new balance of power, elevating patients to the status of collaborative partners. Strategies to promote the expertise of older adults include drawing on strengths-oriented conversations and maintaining their active participation in plans that evolve. Perspectives on successful ageing and resiliency add further dimensions to the understanding of expertise.

Identifying strengths is a conduit for discovering the stories, narratives, and cultural perspectives that form one's identity. Personal and familial parables of falls from grace and redemption, of failure and resurrection, of struggle and resilience may also provide the metaphors from which one may construct a more vibrant vision of the self and world. Strengths-oriented conversations provide resources for coping with current concerns. Working collaboratively with patients is a second avenue for promoting expertise. A key element in maintaining a patient's active participation is ensuring that they have access to information, the power base of decision making. The kinds of information that older adults might need include details of their current situation and the implications and requirements for their health conditions, the differences between ageing and disease, the options for programmes and services and the extent to which they qualify, and the availability of support within their network of family members and friends. As advocates, professionals have a role to ensure patients can exercise their right to choose. Given the effects of Alzheimer's disease or other degenerative disorders, promoting expertise takes a different form, requiring creative imagination and flexibility to see beyond the disabilities and limitations so as to locate possibilities and strengths.

16.7 Elders' Emotional Responses to Life Changes and Illness

Emotionally, elders who go through adverse life

changes (e.g. sensory losses, disability and illnesses, widowhood, retirement, death, and dying) will struggle with many emotions such as anxiety, anger, fear, depression, guilt, uncertainty, insecurity, confusion, frustration and, in extreme situations, hopelessness. Older persons experience the same emotions as younger people but their emotions have a unique configuration. They typically experience more age-related changes and stresses associated with the ageing process than their younger counterparts. The social worker's role is to ensure that elders can process these feelings in a healthy way. Social work professionals need to help elders identify their feelings, accept them, and make necessary psychological and physical adaptations. There are individual variations in terms of how elders respond to or interpret their stress and losses and ways to cope with them.

For many older adults, losing independence or losing control, by being confined to a nursing home, is unthinkable and unbearable. In reality, the probability of any individual ever living in a nursing home is low. In the US, only 4.5% of people over the age of 65 years live in nursing homes, and that percentage has been falling for at least 10 years because of the increased availability of community-based long-term care programmes.[1,7,8] For those who live in the community, fewer than 5% need help with the basic activities of daily living. It is nonetheless true that the capacity for maximum physical effort declines with age, even in the absence of disease and even among lifelong athletes. Fortunately, demands for heroic effort in daily life are seldom encountered. Even after a considerable reduction in reserve capacity, older people retain a level of physical function that is sufficient for active living. Social work professionals need to provide these facts to elders so that they understand the reality of ageing and are relieved of unreasonable fear. Also, social work professionals can educate elders about maintaining a preventive, healthy lifestyle by getting proper exercise and eating a balanced diet. Decline in mental and physical functioning is not inevitable, and an adequate level of functioning can be maintained through healthy lifestyle practices.

16.7.1 Understand the Sense of Loss Experienced by Older Adults

Loss is one of the predominant themes characterising the common emotional problems of older people. Losses in every aspect of later life compel many older persons to expend enormous amounts of physical and emotional energy on grieving and resolving grief, adapting to the changes that result from loss, and recovering from the stresses inherent in these processes. There are many ways that older people can experience the sense of loss, and the meaning associated with loss may vary from person to person. Coping with loss and making feasible adaptations can be challenging for many older people. Older persons, and younger ones as well, may experience different types of loss. Social work professionals who work with elders need to understand the psychological and emotional implications of their loss and be able to support them in sensible ways so that they can make the necessary adaptations. Listed below are examples of some of these losses.

- Relationship loss: divorce, separation, loss of colleagues due to retirement, loss of neighbours due to relocation, and death of loved ones.
- Physical losses: loss of physical health, loss of vision / hearing, loss of mobility, and loss of sexuality.
- Psychological loss: loss of identity or status due to retirement, loss of independence, loss of sense of purpose in life, loss of goals, loss of trust, loss of security, loss of control, and loss of hope.

Older people are confronted by multiple losses that may occur one at a time or simultaneously: death of a significant person, decline in physical health, loss of security caused by widowhood, or burdens caused by other types of financial difficulty. Subjective evaluation of multiple losses may be different because elders' social support, spirituality, and personality can affect their perception in different ways. In the positive ageing example of Mrs Wong, her situation illustrates the ability of elders to cope with multiple losses if they have strong social, emotional, and spiritual support and resources.

16.8 Enhancing Elders' Social Supports

From a life course perspective, ageing of the individual not only means growing old chronologically but also signifies lifelong growth and development in physical, intellectual, social, economic, psychological, and spiritual ways, regardless of the elder's functional status. Active adaptation and coping with losses associated with ageing is a personal growth process.[2,3] One primary mission in elder care is to encourage a nurturing / enabling environment (physical, intellectual, social, economic, psychological, and spiritual) to empower elders' continued growth and development. Research has found elders more likely to report higher life satisfaction scores when emotionally, socially, and physically connected with family and friends.[13-16]

Social work in healthcare settings is usually referred to as medical social work practice. This specialty includes counselling patients and families, helping patients apply for financial assistance, assessing care needs, coordinating multidisciplinary care and arranging for home care and long-term care services, with such discharge planning being a major responsibility for

hospital social work professionals. This responsibility involves planning appropriate continuing care for the patient after discharge. The patient is discharged to their own home, a rehabilitation facility, or a nursing home, and when community services are needed, the social worker makes a referral to a home healthcare agency and other critical community supports such as accessible transportation. Coordinating community services involves mobilising community resources and assessing the patient's needs and eligibility for services. In these cases, a medical social worker may serve as an advocate in securing appropriate benefits and services.

Medical social workers provide counselling to older patients and their family, explore discharge planning and post-discharge care plans. They also act as advocates in what is too often a paternalistic environment. Paternalistic attitudes reflect an attempt to protect the frail older patient, and in this type of environment, social work professionals frequently must act as advocates for the patient's right to self-determination. These paternalistic attitudes often coexist with ageist attitudes that equate physical frailty with mental incompetence. In such environments, social work professionals play a pivotal role as advocates for a patient's right to be informed about treatment options. Sometimes, decisions that have ethical implications create conflict among patients, family members, and healthcare professionals, and social workers may find themselves acting as mediators in these situations. For example, an older male patient who had a bilateral stroke decided to go home. He lived alone and he could manage walking only with a frame. Doctors and physical therapists thought that the patient should go to a nursing home because he required supervision to ensure safe mobility. In the spirit of self-determination, the social worker needed to discuss the pros and cons of various possible discharge destinations with the patient. In a thorough discussion, the social worker learned that the patient worried that his only daughter might not see or visit him if he stayed in an institution. The social worker also worked with the daughter and discussed with her the patient's concern. The daughter was very supportive and promised to visit him 3 times a week and call him every day after his placement in a nursing home. As a result the patient was transferred to a convalescent home and continued his rehabilitation. This example illustrates the need for social work professionals to work with patients and their family to work out reasonable discharge plans that meet the patients' needs, including those related to their physical and psychological functioning. For patients with family, social work professionals may need to work with the family system. For patients without family, social work professionals may need to work with their friends, neighbours, volunteers, and other formal service providers.

16.8.1 How to Work with a Patient's Family and with the Older Patient

Working with family members is an integral part of social work with elderly patients. Holding a 'family meeting' is an effective way to gain understanding of the family dynamics and possible interpersonal conflicts. Such meetings can be very therapeutic, serving as a form of family counselling and as a forum for developing a coordinated care plan and discharge plan. The family meeting allows all parties involved to communicate their concern and support all at once, with the help of the social worker. Through the meeting, the social worker can encourage family members to take a more proactive role in creating the home care plan for an elderly patient and empowering family members to develop their own creative strategies to meet the patient's needs. In addition, the meeting can help family members recognise long-standing family conflicts and resentments and work towards resolving those conflicts in the process of helping an elderly patient. In the context of planning for a patient's discharge, social work professionals need to set up goals for family meetings. The following steps are useful.

- If the goal of the family meeting is discharge planning, the first step is to assess the family and interpersonal dynamics. How much support can family members offer an elderly patient? In other words, what is the capacity or strength of the informal support system in the community?
- The second step is to identify family members who are willing to work with the medical team to support the patient.
- The third step is to help an elderly patient and family members prioritise issues in the midst of conflicting demands.
- And the fourth step is to help families move towards healing past hurts and focus on developing an action plan for their elderly relative.

These goals reflect a basic confidence in the ability of an elderly patient and his / her family to find solutions to the disease challenges, with social work professionals acting to facilitate the family problem-solving process. This approach also recognises the formidable influence of a lifetime of family dynamics as both a help and a hindrance. While families may be able to put their individual differences aside for the sake of determining care options for an elderly member, the same crisis may bring unresolved conflicts to the surface. Both the functional and dysfunctional aspects of a family's system are addressed in this practice model.

16.9 Carers of Frail Elderly Patients

Although community services for frail elders are

available in different societies, including the US and Hong Kong, the majority of impaired elders rely solely on informal caregiving assistance from family and friends.[18-20] In the US, there are more than 22.4 million households with at least one caregiver who provides unpaid help to elders. These caregivers average 18 hours a week of assistance, with one-fifth of all caregivers providing more than 40 hours a week of care, the equivalent of a full-time job. These caregivers' activities range from instrumental support, such as assistance with transportation, grocery shopping, housekeeping, and managing finances, to personal assistance with the most basic activities of daily living. The demand for both formal and informal caregiving is expected to continue to rise as the number of elders in the population grows and longevity among elders with functional disabilities increases. Nearly one-quarter of all elders in the US is functionally disabled or in need of some assistive services. The lower fertility rates of the baby boom generation, the geographical mobility of adult children, and the growing numbers of women in the workforce suggest that in the future, greater caregiving responsibilities will fall to a smaller number of available family caregivers. These demographic changes will have far-reaching implications for the development and delivery of formal services intended to replace the decreasing number of family caregivers.[19,20]

16.9.1 Who are the Family Caregivers?

The majority of caregivers are women, primarily spouses, daughters, or daughters-in-law. Often these caregivers hold full-time jobs and are raising children in addition to their caregiving responsibilities. Women may be socialised to feel more obligated to take care of the emotional and physical needs of ageing relatives. Women are also more likely to develop and maintain deep emotional bonds with ageing parents so that their acceptance of caregiving responsibilities is as much the effect of affectionate ties with ageing parents as it is the response to a subtle expectation by other family members.[19,20]

Adult sons are more likely to become involved in caregiving activities when it is their father who needs assistance as opposed to their mother, presumably because of the intimate nature of personal care. However, men's involvement in caregiving more often involves coordinating formal services from the community rather than providing services directly. Older men are more likely to receive care from their spouses than from adult children, consistent with the greater likelihood that older men will be married. Older wives are often the sole caregivers for their ageing spouses, assuming responsibility not only for all the homemaking tasks but also for the demanding tasks of providing personal care. Research found that care for older men by either a spouse or other female caregivers was often given on the basis of perceived need rather than functional ability. That is, older men were more likely to have meals prepared by someone else because they had no desire to learn to cook, not because they were unable to cook. The same appeared to be the case in other traditionally gender-linked activities, such as housekeeping and shopping.[19,20]

Older women receive caregiving from adult children, particularly daughters, more often than from spouses. And these adult children are more likely than spouses to expand the caregiving network to include more people. This does not mean that older husbands do not provide care for their wives, but that such arrangement is less likely because there are fewer married women than men over age 65.[1-3,20] Social work professionals and other members of the healthcare team need to understand the reality of family caregivers so that a reasonable plan for care of the frail elderly patient can be developed. Care for the caregivers is also crucial because they are equally important in the care plan. The hardest caregiving situation is provision of care to cognitively impaired patients because of the lack of a reciprocal relationship between caregivers and care recipients. In such situations, the elder who needs care may be unable to communicate with the caregiver. In normal family relationships, family members provide mutual and intergenerational support. But for elders afflicted with an illness such as dementia, even responding with affection and gratitude towards their caregivers becomes difficult.

Spousal caregivers for persons with dementia experience dramatic changes in the nature of the marital relationship as the disease progresses. In the middle and later stages of dementia, caregiving elders lose the support and affection once present in the relationship. And, the personal attraction that maintained the relationship ebbs away as the impaired spouse is less capable of responding to his or her partner. A lifetime of mutuality can quickly become a one-sided situation.

16.9.2 Caregiving Experience and Sense of Social Isolation

Spousal caregivers, especially women who are the only caregivers for their husbands, often become socially isolated, and this isolation becomes a high-risk factor for the caregiver's development of both mental and physical health problems.[18,19] It becomes difficult for the caregiver to leave the house without respite care support, and elders with late-stage dementia are often uncommunicative, forcing their caregivers to live in silence as well. Over half of caregivers to elders report they have significantly less time for family and friends than they had previously

or that they have had to give up vacations, hobbies, and other important sources of socialisation. Even caregivers with support systems are often restricted from accessing them because of the demands of caregiving. And caring for an older parent may revive long-standing problems in the relationship between adult children and their parents, exacerbating an already-demanding situation.

The quality of the relationship between caregiver and care recipients is one of the most important determinants of the perception of stress associated with the caregiving arrangement. Adult children (or spouses) who have had an open, mutually affectionate relationship with the care recipient prior to the illness or disability are less likely than those who have not had such a relationship to experience emotional stress or to consider the caregiving responsibilities to be a burden. The ability to detach oneself from parents and establish a healthy level of emotional and instrumental independence is part of the developmental challenge associated with adolescence and young adulthood. Adult children who remain emotionally or financially dependent on parents well into adulthood have great difficulty assuming caregiving responsibilities. Psychologically, they find it difficult to accept the reality that their parents now depend on them rather than being the ultimate source of support and comfort for them. Filial maturity requires that adult children be able to see a parent as an individual with assets and faults that go beyond their role as caregivers of their children. When adult children have not achieved filial maturity or subconsciously are unable to accept the reality that parents may need their assistance, they can perceive caregiving to be a serious burden.

Caregivers of elders who are depressed experience greater stress than their non-depressed counterparts. It is not surprising that watching a loved one withdraw into the lonely world of depression elicits a deep emotional response in the caregiver. A caregiver's psychological reaction to the depressed elder's sadness and loss of interest in most activities that accompanies depression appears to spill over into the family of the caregiver as well, creating tensions in that person's marital and familial relationships. Elders who cannot accept care even in the face of disability, as well as those who relinquish their independence too quickly, increase a caregiver's perception of stress. Caregivers and care recipients are more satisfied with the caregiving relationship when there is a balance between too little and too much care. Elders who consciously work to maintain even limited functional abilities and cultivate a positive attitude are less likely to become depressed and are perceived by their caregivers to be less of a burden than those who do not.

16.9.3 Caregiver Satisfaction

The caregiving experience can be positive and beneficial, and a significant proportion of caregivers report their caregiving experience in very positive terms. Caregiving may provide adult children or spouses a deeply rewarding sense of meaning and purpose. Providing services to a loved one can be a source of great pride, even in previously stormy relationships. The opportunity to resolve lifelong conflicts or unfinished business can be a meaningful experience for both caregiver and care recipient. Caregivers who do not report caregiving as a stressful experience have found successful ways to moderate the effects of stress on both their personal lives and their relationship with the elders. Caregivers who have support systems, including spouses, relatives or friends, report lower levels of stress than do those who lack such support systems. Maintaining a close connection to family members appears to buffer the frustrations and demands of being a caregiver more effectively than does the support afforded by professional counselling. In addition, caregivers who use family / friends and community services such as adult day care or respite care are better able to manage caregiving stress than those who do not use such support. The instrumental support offered by either family members or more formal services creates a sense of control in caregivers that mediates feelings of being overburdened and overwhelmed.[19,20] Social work professionals in hospitals or other healthcare settings need to provide support group and case management services to patients and to family caregivers so as to help the caregiver clearly identify her role, responsibilities and limitations, thereby decreasing the caregivers' burden of care.[21]

16.10 Elder Abuse

Sadly some elders, especially those who are frail, are more likely to be mistreated at home or in an institution.[1,5] Elder abuse is a growing problem in the US. In the US, the term 'elder abuse' refers to any knowing, intentional, or negligent act by a caregiver or any other person that causes harm or a serious risk of harm to a vulnerable older person. Self-neglect, which refers to an elder's inability to provide for his or her own physical care and well-being due to physical, psychiatric, or cognitive limitations, is included in the general rubric of abuse and neglect in the US, where protection of the older person has dominated policy development. Elsewhere, including Hong Kong and the UK, self-neglect is not included in the definition of elder abuse. In Hong Kong, elder abuse refers to the commission or omission of any act that endangers the welfare or safety of an elder. While the abusers may be known or unknown to the elders, the local procedural guidelines on elder abuse (http://www.swd.gov.hk/en/index/site_pubsvc/page_family/

sub_listofserv/id_serabuseelder/) are confined to those involving abused elders and abusers being known to each other, or involving abusers who are responsible for the care of the abused elders. This does not imply that self-neglect or abuse by strangers does not require attention, but rather focuses on the dilemma of harm within a relationship of trust.

In the US, elder abuse includes the following categories:

- Physical abuse: Inflicting, or threatening to inflict, physical pain or injury on a vulnerable elder or depriving him / her of a basic need, like food, water, or medication.
- Emotional abuse: Inflicting mental pain, anguish, or distress on an older person through verbal or nonverbal acts, verbally threatening to kill an elder or throw him / her onto the street.
- Sexual abuse: Making nonconsensual sexual contact of any kind with the elder.
- Exploitation / financial abuse: Illegal taking, misuse, or concealment of funds, property, or assets of a vulnerable elder.
- Neglect: Refusal or failure by those responsible to provide food, shelter, healthcare, or protection for a vulnerable elder.
- Abandonment: The desertion of a vulnerable elder by anyone who has assumed the responsibility for that person's care or custody.

Detailed examples are provided in the following section.

Elder abuse is a serious societal problem because it can lead to harm or death if timely intervention is not provided. It can affect people of all backgrounds and social status, men and women. Although there has always been an awareness that some children and elders are physically, emotionally, or financially exploited by caregivers and family members, it is only since the last half of the 20th century that efforts have succeeded in criminalising such abuse or neglect and holding the perpetrators legally liable for their actions. Statistics show that since the ageing population is growing and people are living longer, the number of frail elders is also growing. Society can no longer ignore the problem of elder abuse. This issue challenges the notion of the family setting as a safe haven for children, women, and elders. Research findings show that a majority of all violence committed against elders, women, and children occur at home. Elders are vulnerable to abuse and neglect due to the greater likelihood of their suffering from physical and cognitive impairments and their need to rely on caregivers and family members for basic physical care. Elder neglect encompasses both active and passive efforts in which elders fail to receive necessary care, resulting in the deterioration of their physical, emotional, or psychological health. It is important to emphasise that neglect of elders is not always intentional but that it always constitutes unacceptable treatment, regardless of intent.

Social work professionals who work with elders on a regular basis must be able to develop the levels of professional sensitivity and a sixth sense to detect something out of the ordinary in an elder's physical, emotional, and social environment that will hint at elder abuse. It is important to remember that elder abuse is not only the traditionally thought of notion of a caregiver lashing out from escalating frustration / anger, but can also include attempts to control an elder's behaviour in ways that violate their health and functioning. The following discussion will provide social work professionals and other health and mental health providers a checklist to use when looking for elder abuse.

16.10.1 Clinical Observation

The social worker must use his or her clinical skills and professional instincts to observe a suspected elder abuse situation as part of the assessment process. Ask the following questions:

- Does the elder have bruises or injuries in various stages of healing?
- Does the elder make any attempt to hide those injuries?
- Is there any evidence of physical injuries that may have been inflicted by others and do not appear to be the direct result of a plausible accident? It is important to remember that elders bruise more easily than younger people due to physical changes in the thickness of the skin and the resiliency of blood vessels that are associated with the biological aspects of ageing. It is not uncommon for frail elders to have mild forearm bruising and skin tears associated with bumping into things or being properly assisted by a caregiver, and this should be questioned carefully.
- Does the elder appear unusually withdrawn or frightened?
- Is he or she unusually vigilant or easily startled?
- Does the elder's emotional state change from calm to agitated or fearful when others are around?
- Does the elder seem to use a lot of self-degrading statements, such as "I am a bother to everyone" or "I am just a worthless old person"? These statements might be indicators of long-term emotional or psychological abuse in which the elder has internalised the abuser's disparaging remarks. They may also be due to negative self-image rooted in depression or anxiety, or even attention-seeking remarks.
- Direct observations are important, but not sufficient in themselves to accurately determine whether behavioural disturbances are the result of abuse or neglect or are due to other emotional

problems. Other signs to look for:
- What does the elder look like?
- Does the elder appear to be malnourished?
- Is his or her personal hygiene appropriate?
- Are clothes reasonably clean and in good repair?
- If incontinence is a problem for the elder, are incontinence products properly used so that the person is dry and comfortable?
- Are appropriate toileting and bathing facilities available?
- Are medications managed according to the prescription?
- Is the elder's immediate living space clean and free of waste and pests?

These questions are very similar to those used in the general process of assessment, but in this case, the social worker is attempting to determine if deficits in any of these areas are due to the failure of caregivers, other social contacts, or the elder himself / herself to provide adequate care.

16.10.2 Assessing Changes in an Elder's Functioning

If the social worker is working with an elder on a regular basis, he or she should be familiar with the elder's functional status and ability to perform both the activities of daily living and instrumental activities of daily living. Consider and ask the following questions:
- Have there been any changes in the elder's ability to perform any of these activities that cannot be explained by declining physical or mental health status?
- How does the elder perform on a mental functioning assessment test? Is he or she oriented to time, space, and person? Can he / she draw a clock face correctly? Is there any short-term memory deficit?
- Have there been acute changes in performance in this area?
- How does the elder score on the Geriatric Depression Scale? Has there been a change in recent weeks or months in depressive mood? It is important to determine what, if any, alcohol, illicit and prescription drug use patterns are in the home if a social worker suspects abuse or neglect by a caregiver, family member, or other social contact.
- Does the elder drink alcohol to excess?
- Does the primary caretaker or do other residents of the home drink or use drugs on a regular basis?
- Are the elder's medications managed properly?

If a social worker is involved with an elder or the elder's family on an ongoing basis and notices significant or acute changes in any of these areas, it is important to pursue a reasonable explanation for them. The social worker may feel that he or she knows an elder or an elder's family very well and that the family would never abuse or neglect the elder. The hidden nature of abuse and neglect nonetheless necessitates the social worker to re-evaluate a familiar situation from a different perspective.

16.10.3 Assessing Elder Abuse through Direct Interviews with the Elder

A social worker who is determining whether there is just cause for suspecting abuse or neglect of an elder needs to interview the elder alone — without the possible abuser present. The presence of the possible abuser may prevent the elder from answering questions honestly out of fear of retaliation. In addition, implicating a possible abuser in front of another person may result in escalation of the abuse. For many elders who are highly dependent on others (even those who are abusing or neglecting them), the prospect of losing their sole source of care and support is very frightening. A social worker should be very direct in asking the elder questions about circumstances that constitute abuse or neglect even if these are very sensitive questions. Ask the elder directly:
- Is he or she is being hit, beaten, slapped, or shoved? Has he or she has ever been left alone for long periods of time, tied to a bed, or locked in a room? Has money ever been taken from him or her without permission?
- Has he or she ever been forced to sign over property or money against his or her will?
- Has medication, food, or medical care ever been withheld by a caregiver, family member, or other social contact?
- Has the elder been threatened by anyone?
- If the elder has bruises or other injuries, what explanation does he or she give for the cause of the injuries?
- Does the severity of an injury correlate with the explanation?
- Is there evidence that injuries were treated promptly?
- Is there evidence of over- or under-medication, poor nutrition, or dehydration?
- Does the elder defend the alleged abuser for providing substandard care?
- What does your intuition tell you about the relationship between the alleged abuser and the elder?
- Determining whether an elder has been abused or neglected depends on a careful blend of empirical observations that serve as direct evidence of such mistreatment and the honing of a social worker's professional intuition to detect and evaluate more subtle clues about the way an elder is being treated. An elder has the right to be protected from abuse and neglect by others. An elder also has the right to deny that such abuse or neglect exists,

even if the social worker's professional intuition indicates otherwise.

If the social worker has strong evidence of abuse or neglect, either through direct observation of injuries or by the admission of the elder, it is imperative that a formal report be filed promptly with the supervisor. If there is no direct evidence of abuse or neglect, but the elder is at high risk for mistreatment or the social worker suspects past or fears future abuse, it is important to consider what interventions may prevent future harm.

16.11 Social Services to Elders in Hong Kong

If an elderly patient exhibits symptoms of psychosocial problems, he / she should be referred to a medical social worker so that timely intervention and a post-discharge care plan can be developed. Conceptually, along the ageing wellness and illness continuum (see Figure 16.2), different types of service are provided. These programmes include a wide range of community-based and institutional care services that include financial assistance, senior centre services, accessible transportation, exercise, disease management groups, counselling, hospital discharge planning, day care services, carer support services, home care services, rehabilitation, and long-term care services. For a detailed description of services to elderly people in Hong Kong, readers should refer to Chapters 3 to 11 or go to the Services for the Elderly section of the Hong Kong Social Welfare Department's website <http://www.swd.gov.hk/en/index/site_pubsvc/page_elderly/>.

Social work intervention for elders may involve the clinical intervention for individual elders and / or family, post-hospital care plan, home care plan, long-term care plan, and institutionalisation plan. In the process, social work professionals assess an elder's psychosocial, physical, psychological, financial, spiritual, and social needs and environments. Social work services may aim to develop appropriate service plans to support the elders and family. At the same time, social workers will work with elders and families to empower them so that they can use their inner strength and external assistance to cope with health-related and other challenges.

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CHAPTER 17

Ethical and Legal Aspects of Care for Elders

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OBJECTIVE

To learn the principles of medical ethics, special considerations when applied to the care of elders, the process of clinical decision making (assessment, consent, setting objectives for care, appropriate involvement of patient and family), and the current status of advance directives and guardianship in Hong Kong.

KEY POINTS

- Older people's autonomy in medical decision making is hampered by ageism, low socioeconomic status, and variable decision-making capacity.
- It is the doctor's responsibility to assess an older person's decision-making capacity in different clinical situations on an individual basis, and promote patient participation in decisions as far as any limited capacity allows.
- Good ethical decisions strike a balance between the ethical principles of autonomy, beneficence, non-maleficence and equity; as well as taking into consideration the social context. When older patients are mentally incapable, the doctor should consult colleagues and closest family members or caregivers; gather information and observations from all involved; and try his best to reach a consensus in the best interest of the older patients.
- It is not the responsibility of family members to make ethical decisions. They should nonetheless be encouraged to provide insight into what the patient would want. The doctor should respect the expressed wishes of older patients before they become mentally incapable.
- Relief of a patient's suffering must be the prime concern in what is inevitably a terminal state.

17.1 Introduction

Ethical concerns are common in the management of older patients. Technical advances in medicine have opened up new therapeutic possibilities for previously incurable or fatal medical conditions. In many countries, socioeconomic development has also significantly improved the access of older people to specialised medical care. At the same time, older individuals and their family are increasingly aware of their individual right to medical interventions.

Despite this, medical advances have not prevented the accumulation of physical and mental disabilities in older people. These chronic disabilities lead to a decline in their quality of life, and place a great burden on family caregivers and society in general. Some medical interventions, e.g. tube feeding, may also be very uncomfortable and burdensome. Therefore, in managing frail older people, physicians often have to balance preservation of life with maintenance of quality of remaining life. These medical decisions for individual patients should be guided by ethical principles.

17.2 Principles of Medical Ethics

Modern codes of medical ethics have been greatly inspired by the moral rules of the centuries-old Hippocratic Oath.[1] It is of interest to note that the first premise Hippocratic Oath is that the doctor owes loyalty to his teachers and his brethren. Obligations to exercise skill for the benefit of patient's health come second. Abortion, direct euthanasia, and abetting suicide are prohibited. Improper sexual relationships with patients are banned. Confidentiality in all dealings with patients is imposed. Traditionally the physician as gentleman paradigm demanded that they should show professional solidarity, and practise benevolent paternalism towards their patients. As long as he behaved in a gentlemanly and benevolent fashion his own conscience and integrity would guide him towards ethical solutions in dilemmas surrounding how to treat a patient. However entering the modern era the general public, political parties and governments, especially in the developed first world countries, began to uphold the notion of human rights and the individual's right to make choices.[2] We should see later how this is going

Table 17.1 The four general principles of medical ethics[3]

Ethical principle	Meaning	Example
Respect for autonomy	Respect values and preferences of a person	Genuine attempt to understand why a patient rejects medical advice
Justice	All persons have an equal claim to healthcare and prioritisation must be fair	Non-discriminatory care irrespective of age
Beneficence	Doing good to others	Relieving pain in a cancer patient
Non-maleficence	Avoiding harm	Avoid use of restraints

to affect the evolution of medical ethics.

The four general principles of medical ethics widely accepted in the West are autonomy, justice, beneficence, and non-maleficence (Table 17.1).[3] Although these four principles can serve as a useful reference when tackling ethical issues in the care of patients and have been claimed to be independent of religion and culture,[3] others have emphasised that moral practice cannot be abstracted from the contextual dimension. Moral life happens in a given society, with its cultural background, and economic and political circumstances. The patient-doctor relationship is indeed part of a broader patient-doctor-society relationship. An example is the variation in truth-telling practices throughout the world with regard to cancer patients.[4] While the principle of autonomy would imply informing a patient of their diagnosed cancer illness (the right to know),[5] it is common in cultures with strong family ties, such as Chinese, Japanese and Italians, to have the psychological shock of a cancer diagnosis absorbed, shared, and dealt with by the whole family. In the extreme, some family members would rather bear the bad news among themselves than share it with the patient.[6-8]

When applying the four ethical principles in resolving real clinical situation one should take into account the fact that the principles themselves are often conflicting. For example, a particular effective treatment may bear significant side-effects (beneficence vs. non-maleficence). Effective treatment may be refused by the patient based on his / her belief system (beneficence in clash with autonomy).

In the modern world, the autonomy of the patient assumes paramount importance but may conflict with other principles. The common law presumes that an adult has the mental capacity to make decisions unless proved otherwise. The right to autonomous choices challenged the traditional paradigm of medical professional paternalism.

In the case of Montgomery v Lanarkshire Health Board, the Supreme Court of the UK noted that not only are 'patients now widely regarded as persons holding rights' but are also 'widely treated as consumers exercising choice.' Non-consensual treatment of a patient, even for his own good (beneficence), violates his autonomy. The principle of informed consent can be seen as emanating from the doctrine of autonomy.[2]

The well-accepted principles for clinical use in modern medicine include the following:

- a mentally capable patient has the ultimate right to choose or refuse any form of medical intervention;
- in an emergency situation when the wish of the patient is unknown, one tends to lean towards the preservation of life; and
- it is not necessary to offer futile treatment

17.3 Ageism

Advanced age is often quoted as a reason for not offering effective medical treatment. The arguments include the following:

- older people are not productive to society;
- the elderly are less likely to respond to medical treatment;
- the elderly are more likely to have complications from treatment; and
- an elder in her 80s has already lived to her life expectancy

The first argument is a political one and most doctors would not agree that medicine is merely for the productivity of the population whom it serves. It is true that some frail older people are less likely to benefit from some medical interventions, but age is not a reliable clinical criterion. Moreover, scientific evidence has increasingly revealed that older people benefit from many medical interventions to which they would previously have been denied access, e.g. the treatment of hypertension, thrombolysis for myocardial infarction, and renal replacement therapy. The second and third arguments also ignore the heterogeneity of older people by generalising them as a group with uniform characteristics: 'the elderly', a commonly used term that carries misleading connotations in clinical medicine, as pointed out by Isaacs.[9] With increasing age, there is much greater biological diversity. The potential benefit and harm from treatment can only be judged clinically on an

individual basis. Furthermore, treatment-related harm (iatrogenesis) among elderly people can be minimised or avoided by enhancing a clinician's knowledge of geriatric medicine (see Chapter 15.4). The last argument confuses life expectancy at birth with life expectancy at an older age (see Chapters 1.2.2 and 13.1, respectively).

17.4 Threat to the Dignity and Autonomy of Older Persons

Older people have low social status in many countries. A comparative study of the views of older persons in six European countries reported that participants found it much easier to identify situations in which dignity was lacking rather than present, with areas of concern focused on the themes of respect and recognition, participation, and dignity in care.[10] The issue of elder abuse (see Chapter 16.10) has been relatively ignored compared with child abuse.

In Hong Kong, older people are more likely to be poor, poorly educated, and disabled. These often result in low self-esteem. This problem is particularly prevalent in older people in institutional care. The autonomy of older patients can therefore be easily overlooked.

A common scenario is an older person with diseases or disabilities who is being coerced by family members into a residential care home placement. Although it is true that the wishes and needs of family caregivers should also be respected, in such situations, healthcare professionals should not take sides. Their role instead is to promote mutual understanding and to assist in the establishment of a consensus. Community care may be a viable option after rehabilitation with flexible use of social services.

Physical restraint is another difficult area in the care of older people. In a study of 14 residential care institutions in Hong Kong, bedside rails were used in 62.5% of residents, while other physical restraints were used in approximately 25% of residents, with trunk restraint being most commonly used, followed by limb restraint and chair restraint.[11] Although physical restraint is sometimes used in confused patients as a precautionary measure against falls or as a means to retain life-sustaining devices (e.g. nasogastric tube), less invasive alternatives (e.g. attention to personal needs and bodily discomfort, use of bed chair pressure sensor) should be explored. It is not justifiable to restrain mentally alert older people even when they have occasional episodes of acute confusion. The physical and psychological harm of physical restraints has been widely reported. Research evidence has revealed that restrained residents in nursing homes may show more agitated behaviour and have constipation, incontinence, pressure sores, sensory deprivation, reduced functional capacity and dependency, and are at higher risk of injurious falls.[12] Physical restraints should not be accepted as convenient 'solutions' to confusion and falls, geriatric giants that require proper assessment, diagnosis, and treatment of underlying causes. The use of physical restraints and their justification should therefore be clearly documented and closely monitored. In Hong Kong, a multidisciplinary task group under the Hospital Authority consisting of geriatricians, nurses, and allied health professionals has formulated clinical guidelines for the appropriate and proper use of physical restraints.[13] A successful restraint reduction programme in a geriatric rehabilitation department in Hong Kong resulted in a reduced length of hospital stay by an average of 5 days among confused patients without significant increase in the incidence of falls.[14]

17.5 Assessment of Decision-making Capacity

The assessment of an older patient's decision-making capacity should be task-specific. For example, the intellectual demand for a decision about cardiorespiratory resuscitation would be greater than that about whether to have a bath. In order to have sufficient decision-making capacity, the patient needs to be able to communicate a preference consistently. The patient should be able to understand the pros and cons of the preference. The patient's reasoning for having the preference should be understandable and reasonable.

The UK Mental Capacity Act (2005)[15] stipulated that a person lacks capacity in relation to a matter if at the material time he is unable to make a decision for himself in relation to the matter because of an impairment of, or a disturbance in the functioning of, the mind or brain.
A person is unable to make a decision for himself if he is unable:

(a) to understand the information relevant to the decision;
(b) to retain that information;
(c) to use or weigh that information as part of the process of making the decision; or
(d) to communicate his decision (whether by talking, using sign languages or any other means)

One pitfall that should be avoided is the tendency to dismiss the possibility of meaningful communication with dementia patients because of a 'global' assessment of competency. What is more important is clinical assessment of specific patient capacities, e.g. the capacity to care for oneself, to make informed decisions, or to communicate one's decisions. Because assessing capacity for medical decision making varies with each clinical situation, it is important to remember that although patients may not have a

functional ability to handle legal or financial matters, they may still have an adequate capacity for making decisions about their own medical care.[16]

In order to have a fair assessment, the medical condition of the patient should be optimised, e.g. stop sedatives, relieve discomfort. The interview should be held in private and with as few distractions as possible. There should be plenty of room for the patient to express himself / herself. Cognitive testing may be helpful but is not the deciding factor. Apart from confusion or dementia, depression should be identified as this can also impair decision-making capacity. If there is any doubt, the assessment should be repeated later on.

17.6 Decision Making for Mentally Incapacitated Persons

When an older patient is judged to be mentally incapable of making a decision, the attending doctor is obliged to make the decision according to the patient's best interests. The first step is to decide if the intervention or investigation is essential to the well-being of the patient with the chance of a successful outcome. The burden of the intervention, e.g. tube feeding, on the quality of life of the patient has to be considered. This part of the assessment is more controversial and subject to individual variation.

It is important to have a full discussion with concerned family members. Family members do not have any legal rights in medical decision making. Nonetheless based on past experience, they can form an opinion of the patient's probable choice of action, although it is important to distinguish this from their own opinion. The latter is liable to be influenced by their personality, personal attitudes, relationship with the patient, their emotional state, and even their own interests. Therefore while it is important to understand how family members feel about the problem, the burden of decision making should not be placed on them. Different family members may have different opinions. It is helpful to identify a representative among them for liaison. Sometimes, a patient may have designated a specific individual to make proxy decisions when he / she becomes mentally incapable. Such empowerment has been legally provided by the Enduring Power of Attorney (EPA) in the US and the Lasting Power of Attorney. In Hong Kong, the EPA legislation does not provide the donee with the power to make medical decision or consent to treatment (though he / she may manage monetary matters and property in the interest of the mentally incapacitated person). However, reform is underway. It is submitted that the current state of the EPA law should be replaced by the proposed Continuous Power Attorney whereby a person with capacity can appoint a designated person to handle his / her finance, personal care matter, and medical decision making.

In 1999, the Guardianship Board, an independent statutory corporation, was established in Hong Kong to appoint guardians for the mentally incapacitated person.[17] The Board has a multidisciplinary composition of psychiatrists, geriatricians, lawyers, and other professional lay people. The appointed guardians are allowed to make surrogate decisions about the medical as well as social management of a mentally incapacitated person. A public guardian is required when a mentally incapacitated person does not have any family member who is willing to act on his / her behalf or when the attending caring professional team has reason to believe that the family members are not serving the best interests of the disabled person.

17.7 Life-sustaining Treatments

In hospital and nursing home care, ethical dilemmas about end-of-life issues are very common. This has a direct consequence for the quality of care of frail older people. A commonly accepted concept, which originated from Catholic theologians, is that medical interventions are optional while basic care, e.g. food by mouth, shelter, clothing, is morally obligatory. Intravenous / subcutaneous fluids, tube feeding, artificial ventilation, and cardiorespiratory resuscitation are all considered medical interventions. Although they are potentially life sustaining or saving, they should be scrutinised in terms of their clinical effectiveness and their impact on quality of life.

In the long-term care setting, the justification for tube feeding is particularly pertinent. In their advanced stages, cerebrovascular disease, dementia, and other neurodegenerative diseases commonly cause dysphagia. Depression and dementia are also associated with loss of appetite. Long-term tube feeding is useful for delivering nutrition but can cause great discomfort to older people. This is commonly evidenced by attempts to remove a feeding tube and often results in the application of hand restraints and thus further discomfort and distress.

The burden of tube feeding on older people is therefore very great. There is no evidence that tube feeding can prolong life in the long run because of the underlying frailty of subjects and the associated risks of tube feeding and restraints. Careful hand feeding should be considered if tube feeding is not well tolerated. Palliative care without tube feeding may also be justified in the terminally ill patient. If older individuals are mentally capable, their preference should be respected. If such decision-making capacity is lacking, the choice will have to be made

by the attending doctor in consultation with family members as previously mentioned. It is important to review the decision from time to time as the clinical condition may change.

17.8 Assisted Suicide and Euthanasia

Assisted suicide occurs when a person is assisted in any way to commit suicide. For example, a doctor can assist in suicide by intentionally leaving strong sedatives within easy reach of a patient who wants to commit suicide. If the doctor personally administers a lethal procedure with the intention to end the patient's life, this is euthanasia. Some regard this as active euthanasia. Both assisted suicide and euthanasia are illegal in Hong Kong. In the Netherlands, euthanasia is allowed under strict guidelines in consenting adults with incurable diseases.

If a person is deliberately deprived of treatment or assistance that is essential to life, e.g. food for a newborn baby, with the intention of ending that life, this is regarded as passive euthanasia. There is no ethical or legal difference between active and passive euthanasia. If a patient with terminal cancer fails to eat adequately, it is ethical not to initiate tube feeding if the patient does not want it but the patient should continue to be encouraged to eat and drink.

It is not uncommon for older patients to ask doctors to terminate their lives because of persistent suffering. Instead of contemplating euthanasia, doctors can do much to relieve suffering by offering treatment for depression or pain and by providing psychosocial support. If a treatment is intended to relieve suffering, e.g. morphine for terminally ill cancer patients for pain relief, it is ethically acceptable even though it may potentially shorten the patient's life.

17.9 Advance Care Planning and Advance Directives

Older adults frequently have an acute medical illness that requires serious treatment decisions to be made. At the time of the medical crisis, the patient is often unable to communicate their preferences for treatment. As a result, the decision-making responsibility falls on the patient's family and providers who may not have knowledge of the patient's wishes. Advance care planning allows a patient's autonomy to be supported by inviting patients, while they retain their capacity, to express their health-related goals that can guide future medical decisions. It is a process of communication between the patient, family, and medical providers to prepare for future medical decision making. It clarifies the patient's goals, values, and treatment preferences in the event of serious illness and at the end of life.

Advance directives allow patients to provide written instructions about future medical decisions that are applied when the patient lacks decision-making capacity. There are two types. A proxy directive allows patients to assign one or two individuals the durable power of attorney for healthcare or healthcare proxy to make medical decisions on their belief in the event of decisional incapacity. An instructional directive or a living will allows patients to specify the types of treatment they would and would not wish to receive under particular circumstances such as when they are incapacitated by a terminal condition or in a persistent vegetative state. The legal status of durable power of attorney for healthcare and living will varies in different western countries, and has been a subject of constant debate.

In Hong Kong, there is a legally binding durable power of attorney for financial management. To date, there is no legally binding advance directive for healthcare. In 2006, the Law Reform Commission of Hong Kong released its report on "Substitute Decision-Making and Advance Directives in relation to Medical Treatment,"[18] that argues against enacting legislation on advance directives because the concept has not yet become familiar to the local community. The Commission has made available a model form of advance directive for public use. Recent studies have shown that although the Hong Kong lay public lack knowledge about an advance directive, when informed, the acceptability among local Chinese was high.[19,20]

The Hong Kong Geriatrics Society has responded with its position statement on advance directives.[21] The Society welcomes the use of advance directives as a means to promote patient autonomy and improve communication between patients and healthcare professionals at a time when the patient lacks the capacity to make a decision. Nonetheless, the Society cautions against solely relying on advance directives because of their limitations shown below.

- Goal setting is of utmost importance in geriatric care. The listing of potential procedures in advance directives may divert attention away from the overall treatment goals and may give rise to inappropriate care.[22]
- Patients' wishes for care often change over time and an instruction for future care cannot be assumed to reflect a permanent wish in the future.[23] Advance directives should be periodically reviewed to determine whether wishes have changed in relation to changing health status and goals of care.
- The complexity of illness in old age makes it difficult for a patient to be adequately informed of unforeseen circumstances to which the advance directive may be applied. Treatment preferences

may be difficult to extrapolate to specific clinical situations and this has led to misapplication of advance directives.[24]

Advance directives alone frequently do not affect the quality of end-of-life care nor improve family's knowledge of patient preferences.[25-29] Without good communication between patients, families and providers, documented directives can lead to conflict between family members and providers who disagree about their interpretation when patients lose their decisional capacity.[30] Medical providers can facilitate advance care planning discussions with patients and their families and enable patients to have control over their future care, ease the burden of the decision-making responsibility on families, and eliminate potential conflicts that may arise. To do so effectively, the approach should redirect focus away from asking patients to make premature decisions about interventions such as mechanical ventilation and tube feeding, given the difficulty of predicting the particular intervention and the circumstances under which the decision should be applied. Instead, these discussions should focus on informing patients and families about the patient's health status and prognosis and allowing patients to reflect on their goals and values in the context of their evolving health over time.[31,32]

17.10 Reaching a Consensus

Medical ethical decisions are often not straightforward. For a variety of reasons, the opinion of the patient, their family members, and the attending doctor may differ. The doctor should make every effort to reach a consensus with the other parties by careful listening, explaining, and counselling.

In the ethical approach, the process is as important as the outcome. This involves examining relevant factors related to the patient's medical condition, including diagnosis, treatments, and prognosis; understanding quality-of-life issues before, during and after the present illness; exploring the preferences of the patient, family (or surrogate), and healthcare team; listening and addressing the concerns / goals / values of patients, families, and staff. The clinician should be sensitive to the personal and psychological struggle faced by people with severe illnesses who may also be facing frightening or threatening outcomes and / or death. One should also consider the context, including the available support systems, resource availability, religious or ethnic preferences before setting objectives of care.

As mentioned before, the patient has the final say about the decision. However, when the patient is mentally incapable, the clinical team should first ascertain whether there is a legal proxy (under EPA or guardianship provision) who is granted the power to make medical decision. The team should also make sure that any relevant and applicable advance directives be given due effect. In the absence of legally binding decision-making mechanism, the clinical team should consult the family members and any concerned parties to reach a consensus based on the best interest principle. In determining what is in the best interests of patient, one should take into account:

(a) past and present wishes and feelings;
(b) beliefs and values that may have influenced the decision being made, had the person had capacity; and
(c) other factors that the patient would be likely to consider if they had capacity

But when the patient is mentally incapable and the doctor and family members cannot reach a consensus, a second opinion should be sought. A doctor who is not involved in the care of the patient and preferably experienced in medical ethics can give an independent opinion and helpful advice. Hospitals can set up a more authoritative medical ethics committee to arbitrate. A law court is rarely helpful except for landmark cases, e.g. the Tony Bland case in the UK. Mr Tony Bland was a young man who had a severe head injury during a stampede in a football match. He remained in a vegetative state for 3 years. His family requested termination of enteral feeding. The attending doctor was uncertain about the legality of such practice and referred the case to the Law Court. The case was finally considered in the House of Lords who determined that it was legal to withdraw enteral feeding in a patient in a persistent vegetative state.

John Morley,[33] Christine Cassel, and Harry Moody have argued against the dominance of the three C's (competency, consent, and confidentiality) in the current legal model of ethics, and advocated a genuine social ethics that stresses care, commitment, compassion, and courage to approach the ethical dilemmas in caring for elderly people (Table 17.2).

17.11 Summary

The autonomy of older patients and their right to effective medical interventions should be respected in all civilised societies. The decision-making capacity of frail older people for a specific task should be carefully assessed, and can be enhanced. Every

Table 17.2 Legal and social models of ethics

Legal model	Social model
Competency	Sliding scale of capability
Consent	Consensus building
Confidentiality	Communication Clarification

effort should be made to reach a consensus with the patient and their family. In the mentally incapacitated patient, the doctor should be ultimately responsible for medical decision making, although surrogate opinion should be sought from family members or guardians. Although a living will can be informative about a patient's previously held attitudes towards life-sustaining treatments during the final phase of life, the limitations and problems of advance directives have to be borne in mind in clinical practice. Doctors can do much to relieve suffering in patients with incurable diseases without resorting to euthanasia that is not only illegal but also ethically unacceptable.

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CHAPTER 18

Palliative Care and Pain Management

Raymond See-kit LO

OBJECTIVE

To learn how to provide palliative care and pain relief to elderly patients with cancer and non-cancer illnesses, including a holistic approach, and pharmacological and non-pharmacological interventions.

KEY POINTS

- Palliative care is a significant component of comprehensive care for our elders, and should not be neglected.
- Palliative care in elders adopts a holistic approach with a shift in emphasis from the biomedical to the biopsychospiritual paradigm. Palliative care is active, total and holistic, and should be offered at any stage of an incurable illness.
- Spiritual / existential care, dignity, and quality of life are relevant in elders and should not be ignored.
- Older patients bear the brunt of cancer burden, and geriatricians should be ready to shoulder the responsibility of elderly people with cancer.
- Palliative care is also required in elders with advanced non-cancer conditions.
- Symptoms that require palliative care are subjective and multidimensional. Like other diseases in elders, they can have an atypical and non-specific presentation.
- Symptoms in elders often have remediable causes, and require an individualised approach. Therapeutic interventions should carry maximum benefit and cause minimal harm.
- Management of pain involves the total pain concept with its underlying physical, psychological, social, and spiritual / existential components.
- Evaluation of pain should be comprehensive in both cognitively intact and impaired elders, and should consider its functional and social impact.
- Adequate knowledge about the properties of opioids and adjuvant therapy is needed to enable safe and adequate provision of pain relief in elders.
- The World Health Organization analgesic ladder is appropriate for application in elders.
- Judicious titration of opioids is safe for older patients with pain who do not respond to step one or two of the analgesic ladder.
- Side-effects of opioids can be minimised by prompt management.
- Non-pharmacological methods for pain and symptom relief should not be forgotten.

18.1 Introduction

18.1.1 Introduction to the Concept of Palliative Care for Elders

A vital component of comprehensive care for our elders is palliative care.[1] The ageing elderly face increasing medical and psychosocial problems with deteriorating heath. Palliative care aims to relieve symptoms and enhance quality of life. It should be provided whenever an advanced incurable illness is diagnosed and cater to both patients and their relatives.[2,3] Geriatric care and palliative care complement each other and are not mutually exclusive.[4] Despite the wide promotion of the concept of palliative care, older patients still receive suboptimal palliative care.[5] For example, ageism in cancer care still exists.

18.1.2 The Essence of Palliative Care

Palliative care adopts a holistic approach, with a shift in emphasis from the biomedical to the biopsychospiritual paradigm.[6] It offers care to patients with advanced incurable disease, and the ultimate aim is to optimise quality of life.[7] Palliative care neither hastens death nor prolongs suffering. It offers care and support not just to patients but also to their family, helping relatives through the

bereavement process.[8] Palliative care involves active, total, and holistic care. Active care refers to active treatment of symptoms, prevention of complications, and support of patients and families. The care is holistic: it is patient- and family-centred, covering all the physical, psychological, social, and spiritual / existential needs.

Several commonly held misconceptions about palliative care should be clarified. First, palliative care is not passive care. It does not comprise oversedation of patients with morphine or neglect of symptoms that can be easily treated. Instead, there is prompt and effective palliation of symptoms that necessitates an intensive approach, although this does not imply numerous tests, multiple pills, or aggressive interventions. A second misconception is that doctors have little role in psychospiritual care that can be left to the nurses, chaplains, and clinical psychologists. It should be remembered that doctors lead or coordinate an interdisciplinary team to deliver comprehensive and holistic care. Doctors are not just organ specialists or symptomatologists, but should also be understanding and empathic towards patient needs. A third misconception is that care for the dying is the sole responsibility of palliative care physicians. This is evidently inappropriate as doctors from different specialties will all have to face end-of-life issues in clinical practice, but especially geriatricians who deal with older patients and the frequently accompanying issues of death and dying. While a basic approach to comfort care at the end of life needs to be adopted by all, specialist palliative care should be available whenever necessary. A fourth misconception is that palliative care is only applicable at the end of life. Palliative care and acute care are not mutually exclusive: there is evidence that early palliative care in conjunction with acute care yields better outcomes.[9,10]

18.2 Special Considerations in Geriatric Palliative Care

18.2.1 The Need for Palliative Care in Older Patients

The older patient with cancer particularly requires a palliative care approach. Over the last 20 years, it has been well recognised that older persons bear the brunt of the cancer burden.[11] Data from the US Cancer Statistics Review show that the risk of developing cancer for those aged >65 years is 11 times that for those aged <65 years.[12] Almost 70% of all cancer deaths in the US occur in individuals aged >65 years.[13] They are less likely to receive opioid treatment for cancer pain[14] and they are under-represented in prospective clinical trials in cancer and cancer management.[15] Despite an increasing prevalence, older patients with cancer have been badly treated,[16] and in 2014, ageist practices were still evident.[17] A fundamental change in policy, with a 'geriacentric' focus on clinical trials, tolerable treatments, decision making, and access needs to be advocated.[17] Chemotherapy near the end of life is a difficult decision, and should not be just an overzealous attempt at not 'giving up'.[18] Oncologists, palliative care physicians and geriatricians should be ready to shoulder the responsibility of caring for older patients with cancer.

It should be emphasised though that palliative care in old age is not just for those with cancer. There are many other conditions for which it is appropriate. End-stage organ failure, e.g. heart failure, chronic obstructive airway disease, cirrhosis, and renal failure, are clearly conditions with physical and psychosocial symptoms that need palliation. An early study of care for the dying in the UK showed that in addition to dyspnoea, people who died from heart disease also had pain, nausea, constipation and low mood, none of which were well managed for at least 6 months.[19] The quality of life of these patients was poor, and the discomfort and stress was worse than that experienced by patients with cancer.[20]

Elders also have various neurodegenerative diseases such as dementia, Parkinson's disease, motor neuron diseases, and cerebellar degeneration. All are incurable with a progressive decline. As with cancer and other advanced conditions, palliative care should be offered early when there is a need. An elderly patient newly diagnosed with Parkinson's disease may require intensive counselling and support. Palliation of pain may be neglected at any stage in motor neuron disease. Feeding problems, pain, and behavioural problems in advanced dementia are all issues of concern that require good palliation.[21] There are other significant palliative care issues such as advance care planning and forgoing futile treatment that are especially important for older individuals and should not be ignored.

18.2.2 Principles of Symptom Palliation in Older Patients with Incurable Illness

Elderly patients in need of palliative care face the same wide-ranging physical, psychological, social, and spiritual symptoms as their younger counterparts. Among this spectrum of symptoms, physical symptoms are of paramount importance and should be promptly alleviated so the patient can focus on in-depth psychospiritual support. For those elderly patients with cancer, the prevalence of the different types of physical symptoms is remarkably consistent. In two separate prospective studies of palliative care patients, totalling up to 600 palliative care patients, the three most common symptoms that required intervention by the palliative care unit were fatigue, pain, and dyspnoea.[22,23] Other frequent

symptoms include cough, anorexia, constipation, and nausea. Pain is not just prevalent in cancer, but also common in non-cancer conditions. There is compelling evidence that a significant majority of community-dwelling patients and institutionalised elderly experience pain that interferes with normal functioning. Musculoskeletal pain is especially common, with an age-related increase in prevalence.

It is prudent to remember that palliative care symptoms, just like other symptoms in elders, are often atypical and non-specific. For example, it may be difficult to distinguish problems and complications of cancer from those of normal ageing and chronic diseases. Symptoms from brain metastases may be difficult to identify if there is pre-existing mild cognitive impairment. Early signs of spinal cord compression as a cause of immobility may be missed. Symptoms of hypercalcaemia are non-specific in older people. Vomiting may not be all related to cancer and its treatment, but can be due to constipation or urinary tract infection. Cancer itself is a complicated problem to manage, and the additional conditions associated with ageing add much to the complexity of diagnosis and decision making.

In managing symptoms in palliative care patients, it is important to remember that symptoms are inherently a subjective multidimensional experience. They are physical and psychological phenomena that arise from pathological states or disorders. The same amount of pathological impairment can have different impact and meaning for different individuals. One man's agony is just another man's ache. Comprehensive assessment must therefore take account of the multidimensional nature, and a patient's account of their symptoms should always be taken seriously. For example, pain should be considered from all aspects, physical, psychological, social, and spiritual components, that overlap and contribute to the end result of total pain. Likewise, dyspnoea can generate a vicious cycle of anxiety, fear, and panic that further aggravates the breathlessness. Nausea or vomiting may have a psychological element, for example being associated with the sight of chemotherapy or smell of a particular kind of food, that persists even when the organic cause is removed.

18.2.3 Spiritual / Existential Issues, Dignity and Quality of Life for Our Elders

It is a common misconception that elderly Chinese do not understand or perceive the spiritual or existential issues related to ill health. Likewise, there have been doubts about whether the concepts of dignity and quality of life apply to the local elderly population. Accumulating local clinical and research experience reveal that they do.

The spiritual / existential dimension of palliative care refers to unfinished business, hope, dignity, the meaning of suffering, meaning of life, value of life, and the sanctity of life, not just religious issues and life after death. In a longitudinal study of Hong Kong Chinese patients with advanced cancer with a mean age of 60 years, the spiritual / existential domain was the domain that was most related to patients' overall quality of life.[23] A total of 462 consecutive patients were recruited in the cross-cultural validation of an international palliative care–specific quality-of-life scale, and all patients were able to understand and respond to the existential items of the questionnaire. The items assessed included the meaning of personal existence, achieving life goals, whether life is worth living, and feeling good about oneself. This provided evidence that the existential domain is indeed relevant and not to be neglected for our elders. In some cases, elders realised for the first time that it was the existential issues that had been bothering them, and that the interview with the questionnaire served as a useful opportunity to raise these issues and was itself therapeutic. Palliative care physicians and geriatricians now need to promote the importance of spiritual care for elderly patients to colleagues, students, and trainees. Spiritual care can indeed be taught and shared.[24]

A notion closely connected to and overlapping spiritual and existential issues is dignity. It is less abstract than previously thought, with recent work attempting to better conceptualise the theme. In end-of-life situations, it can be categorised into illness-related concerns such as freedom from symptom distress and level of independence; social dignity inventories such as privacy boundaries, social support, and care tenor; and dignity-conserving repertoire such as role preservation, maintenance of pride and hope, autonomy, and acceptance.[25] The concept of dignity for older people is being actively advocated internationally.[26]

The ultimate aim of palliative care is to promote quality of life.[7,8] Quality of life for elders at the end of life need not be poor. Local research evidence has already shown that palliative care services are able to uphold the quality of life at the end. In a cohort of 58 patients with a mean age of 60 years, as close to 5.6 days pre-death, overall quality of life was satisfactorily maintained at 7 out of 10 (10 is the best score).[27] There should be no ageism in maintaining quality of life in palliative care patients. Elders should have the right to 'live until they die'.

18.3 Provision of Palliative Care in the Community

A significant component of palliative care for elders will be delivered, indeed better delivered, in the

community. Elderly patients with an advanced incurable disease need not be hospitalised throughout the disease and until death. Local research evidence has already demonstrated that the quality of life of palliative care patients can be further improved in the community, provided they are fit for discharge.[28] Yet it remains a challenge for both geriatricians and palliative care physicians together to continue the best palliative and supportive care for our patients in the community. Further liaison and collaboration between the community geriatric services and palliative day and home care teams is essential if this goal is to be achieved.

The existing palliative day care units provide medical follow-up and minor treatment procedures, as well as coordinating patient services according to patient needs. The day centre can arrange urgent home visits or outpatient appointments, and direct admissions. The modern palliative day centre typically combines the medical and social model and provides peer support, social / recreational activities, and respite care, as well as individualised rehabilitation. Various modes of therapy such as art therapy, music therapy, aromatherapy, and relaxation therapy are particularly useful. Such therapies can provide a positive focus and a welcome distraction from suffering and distress hence improve quality of life. Local studies have demonstrated the benefit of Tai Chi and aromatherapy in a palliative day care setting.[29,30] Psychospiritually, earlier intervention using anticipatory grief therapy can help elderly people better cope with their declining condition / health,[31] and day centres provide a good environment for such support. Meanwhile palliative home care teams aim to offer continuity of care of physical symptoms at home, together with psychospiritual, social, and practical support for patients and their family. Local evidence is also available of the effectiveness of home care services in improving quality of life.[32] Full support of our elderly palliative care patients at home is vital: they need to know that they are not abandoned in the final part of their life journey, and also that their symptoms can be promptly attended to, with unnecessary emergency attendances or hospitalisations minimised. A palliative hospital support team provides continuity of care when community patients are readmitted to hospital, and can follow up with support and advice, or referral to a hospice. In the author's service setting, the hospital support team takes on new referrals and provides joint care with the parent team in designated comfort beds on acute wards.

The family physician or general practitioner in the community also has a key role to play in the provision of seamless palliative care. They may have been involved in care from the time of diagnosis of an incurable illness or of breaking the news to the patient. Shared care or more active follow-up care by general practitioners is naturally appropriate, depending on their interest and expertise and the local set up of services. They can help with titration of analgesia and morphine, and be alert to ongoing or new physical symptoms. They are also in a good position to provide emotional support to the patient and their family. When circumstances are favourable, patients who so wish, are able to die at home. It has been shown that with adequate support, patients can also die comfortably in local nursing homes.[33] The needs of elders with advanced cancer are unmet, and palliative care in residential care homes needs to be actively supported.[34,35] Finally, the role of the family physician and general practitioner does not end with the patient's death, as they can continue to offer advice and lend support to the bereaved relatives.

18.3.1 Palliative Care in Residential Care Homes

While ageing and dying in a place of choice have been much advocated, many of our older adults in society have to face the reality of residing and being cared for in a residential care home. It may not be the ideal placement for most of our older adults, and it is imperative that palliative care is offered to those with advanced incurable illness in these institutional settings. Transition to life in a care home imposes major stresses that affect quality of life, and palliative care conditions will aggravate the suffering and burden. It may be too optimistic to expect every older adult and family to accept dying in residential care homes. Nonetheless palliative care in any case must not be withheld.

Promoting palliative care in residential care homes is much needed, although it should be provided with care and caution. In a local study that explored factors that could enhance end-of-life care in old age homes, knowledge, culture and attitude were found to be important.[36] The quality of care in residential care homes in Hong Kong is heterogeneous, and the readiness of accepting care for the dying differs widely. Support for both residents and the staff who care for dying patients in residential care homes should not be neglected. Rapid staff turnover is another issue. Community geriatric assessment teams and palliative home care teams should join forces to provide appropriate outreach care for elderly individuals with advanced illness who reside in care homes. Both teams should provide continuous care for patients when they enter such a residential care home and also provide access to in-hospital medical and palliative care beds. Palliative care teams can transfer their skills in care at the end of life to geriatric teams, whereas the geriatric teams should provide support with emergencies and admissions. There

may be gaps in providing appropriate care to elderly residents at care homes, but with concerted efforts hopefully these gaps can be filled.

18.4 Advance Care Planning in Palliative Medicine

Advance care planning is an integral part of palliative care, and should be considered earlier rather than later. Advance care planning can certainly help professionals, patients, and families to better communicate early about important health decisions, while a formal advance directive can safeguard patient preference and autonomy. Various issues require advance care planning when patients are faced with an incurable illness.

There are several principles in advance care planning. It should be emphasised that formulating an advance care plan is not equivalent to giving up on the patient, and the patient should not feel despair. Making an advance care plan does not necessarily mean the patient is at a terminal stage of illness or the end of their life. In the author's experience, it is easier to initiate discussion about advance care planning when palliative care is first discussed with the patient and his / her family. It should also be remembered that advance care planning is a dynamic process, and adequate time is required to make informed, not hasty, decisions. As a patient's condition deteriorates, the reality of the situation becomes evident and the patient and their family may accept some care options previously rejected. Advance care planning is only an option, not a prerequisite to palliative care. If patients and families are not ready to make a decision, this should be respected. All advance care planning should be a process shared by patients and professionals. Decisions made should not be paternalistic or uninformed.

A range of advance care planning decisions can be considered in the light of inevitable decline in terminal disease. Because of the futility and indignity of the procedure when death is expected, an advance decision is often made to not attempt cardiopulmonary resuscitation. Other decisions include do not operate, do not ventilate, do not transfuse, or do not transfer to an acute hospital. In countries where dying at home is possible, do not hospitalise is also a valid decision to be contemplated. In all circumstances, advance care planning decisions are optional, not mandatory, and patient choices and preferences should always be respected. If patients are willing to commit, then proceeding to a formal advance directive should be pursued. Efforts are continually made to promote advance care planning in the clinical setting,[32] but government support is essential to inform the public about the concept.

18.5 Palliative Care for Elders with Non-cancer Conditions

Elders with non-cancer conditions often have worse symptoms and have more distress than those with cancer. End-stage organ failure and neurodegenerative conditions especially require attention. The disease trajectory and prognosis of non-cancer conditions is less predictable than of cancer conditions, although this does not preclude palliative care. Likewise with cancer conditions, acute care and palliative care are not mutually exclusive. If there is a need for palliative care, this should be provided at any stage of the illness. For example, advanced cardiac and respiratory conditions are frequently associated with total dyspnoea syndrome, and with psychospiritual factors that compound the physical distress and necessitate early palliative care. Neurodegenerative conditions such as Parkinson's disease and motor neuron disease are frequently painful. Dementia and other conditions with cognitive impairment require advance care planning early. Heart failure patients have multiple symptoms just like cancer patients. The Gold Standards Framework indeed suggests helpful triggers for general practitioners in initiating palliative care: if a patient is likely to pass away in the next 12 months; if a patient self-requests palliative care; and if there are any poor prognostic indicators.[37] Palliative care should not be delayed when indicated. As with cancer conditions, palliative care should aim to improve care and quality of life for both patients and families in a non-cancer setting, and evidence is beginning to emerge that this can be achieved with a structured programme.[38,39]

End-stage renal failure is a good example of an advanced non-cancer condition that can benefit much from a structured palliative care approach. Patients with low creatinine clearance need advance care planning early, to decide whether to consider renal replacement therapy with dialysis. Dialysis is associated with implications and burden, and might not benefit all, especially the frail old population. Patients with renal failure frequently have troublesome symptoms such as fatigue, itch and cold intolerance, in addition to the common symptoms that need palliative support. Palliative care physicians and renal physicians have demonstrated good synergistic efforts in collaboration, and set a good prototype for other non-cancer palliative care models. Likewise, patients with terminal breathlessness require a good palliative approach, and the use of either invasive or non-invasive ventilation should be very carefully considered. Such procedures are poorly tolerated in many frail elderly, and withholding or withdrawing requires a palliative approach, with consideration of physical and psychospiritual needs.

18.6 Pain Management in Elderly Patients

Older adults are more likely to have pain than younger individuals, giving rise to much distress to both patients and carers. Inadequate pain control for older people is common and there are numerous adverse sequelae such as impaired ambulation, deconditioning, anxiety, fear, and depression. Common reasons for failure to control pain are lack of training on the part of the doctor, not recognising pain, and a reluctance to prescribe opioids.

In the management of pain, it is important to be alert to the total pain concept that acknowledges pain as a subjective sensation, that inevitably will have not just a physical component, but also psychological, social and spiritual components or consequences as listed in Table 18.1.

18.6.1 Evaluating Physical Pain in Elders

Much effort has been spent heightening awareness of the importance of pain control, and guidelines / updated recommendations have been promulgated on the management of persistent pain in older persons.[40,41] General key points in history taking, physical examination, and treatment for older patients with pain are shown below.

18.6.1.1 History taking

- Any presence of pain should be actively looked for in assessment and review for every older patient.
- Physical pain characteristics such as intensity, character, frequency, location, duration, any precipitating and relieving factors must be elicited.
- The patient's subjective account of severity and intensity of pain should be systematically evaluated through a pain scale and recorded.
- Detailed analgesic history including over-the-counter medicines, Chinese medicine, and complementary therapy, together with their effectiveness should be sought.
- In interviewing older patients with pain, the impact of pain on their independence, physical, and psychosocial function, must be assessed, e.g. activities of daily living, sleep, appetite, energy, exercise, mood, social and leisure activities, and quality of life.
- The patient's beliefs and attitudes towards pain should be explored, as they can affect their compliance with analgesics and other pain treatment.

18.6.1.2 Physical Examination

- Careful examination of the reported pain site, and other common sites of pain and pain referral.
- Attention to musculoskeletal systems, e.g. joint inflammation / deformity, postural changes, myofascial pain, fibromyalgia, muscle spasms.
- Attention to neurological system, e.g. signs of neuropathic pain with numbness, paraesthesia, allodynia, hyperalgesia.
- Attention to skin and pressure areas, e.g. abscesses, sores, cellulitis.
- Attention to oral and dental hygiene, e.g. mouth ulcers, mucositis, ill-fitting dentures.
- Observation of any limitation by pain of limb strength, physical performance, or activities of daily living.

18.6.1.3 Assessing Pain in Cognitively Impaired Individuals

For the cognitively impaired, history from caregivers is vital and pain behaviour in particular should be recognised and identified. For example, unusual behaviour or agitation in a demented individual may signify underlying pain. Common pain behaviours are listed below[40]:

- facial expressions of pain, grimacing, frowning, and distortion
- vocalisations of moaning, groaning, grunting, or being verbally abusive
- body movements, assuming a rigid tense posture, guarding, fidgeting, increased pacing or rocking
- changes in personal interactions, e.g. resisting care, aggression, disruptive behaviour, or becoming withdrawn
- changes in activity patterns, e.g. refusing food,

Table 18.1 Potential components of total pain in elders (different components may overlap)

Physical pain	Psychological pain	Social pain	Spiritual / existential pain
Pain from cancer	Worry	Poor living conditions	Indignity
Pain from metastases	Anxiety	Financial hardship	Meaning of suffering
Pain from treatment	Fear	Inadequate communication	Meaning of life
Pain from joints	Negativity	Inadequate information	Purpose of life
Pain from trauma	Low self-esteem	Loneliness	Value of life
Pain from wound	Despair	Isolation	Sanctity of life
Pain from sores	Depression	Neglect	
Pain from immobility	Demoralisation	Abuse	
Pain from poor oral / dental hygiene	Derealisation	Burden on family / carers	
		Family disharmony	
		Discharge placement	

increase in rest periods, increased wandering
- changes in mental state, e.g. increased confusion, crying, irritability

18.6.2 Differentiating Mechanisms of Physical Pain

It is essential to be able to distinguish the various underlying mechanisms that give rise to physical pain,[42] as their treatment strategies are different. Common mechanisms are described below.

18.6.2.1 Nociceptive Pain

This refers to the visceral or somatic pain experienced in response to a noxious stimulus acting on pain nociceptors. Nociceptive pain may arise from a tumour, injury, mechanical destruction, intense heat or cold, and usually responds well to traditional analgesics and opioids.

18.6.2.2 Neuropathic Pain

This refers to pain from a pathophysiological process that is associated with damage or a lesion of the peripheral or central nervous system, e.g. diabetic neuropathy, herpetic neuralgia, post-stroke thalamic pain, phantom limb pain, or a tumour-compressing nerve roots or peripheral nerves. Neuropathic pain responds less well than nociceptive pain to traditional analgesics and often responds better to antidepressants and anticonvulsants.

18.6.2.3 Inflammatory Pain

As the name implies, this refers to spontaneous pain and hypersensitivity to pain in response to tissue damage and inflammation, e.g. following trauma or surgery or in inflammatory conditions such as rheumatoid arthritis and vasculitides.

18.6.2.4 Functional Pain

This refers to hypersensitivity to pain as a result of abnormal responsiveness or function of the nervous system, in which heightened gain or sensitivity of the sensory apparatus amplifies symptoms. Examples are fibromyalgia, irritable bowel syndrome, tension headaches, and some forms of non-cardiac chest pain.

18.6.3 Goals of Palliating Pain in Elders

The goals of palliating pain are pain free at rest, at night, on movement, and without excessive sedation.

18.6.4 General Principles

- All pharmacological interventions should balance benefits and risks.
- Older people are more susceptible to adverse drug side-effects and interventions, and should be frequently and carefully monitored.
- Individually tailored drug regimens should be used rather than blanket treatment.
- Start with the lowest anticipated effective dose. The best route of administration is oral, and drugs should be administered regularly over 24 hours and not on an as-needed basis.
- With care, analgesics can be used safely and effectively in elders.
- Pharmacological and non-pharmacological methods of pain relief should be combined.

18.6.5 World Health Organization Analgesic Ladder

A stepwise approach with an analgesic ladder is recommended by the World Health Organization for control of subacute or chronic pain, especially cancer pain (Figure 18.1).[43] Step 1 is the use of non-opioids; step 2 is the use of weak opioids plus or minus non-opioids; and step 3 is strong opioids plus or minus non-opioids. At each step, adjuvant therapy should also be considered. Analgesics can be stepped up as well as stepped down, using this analgesic ladder approach.

18.6.5.1 Non-opioids

Non-opioid drugs refer to paracetamol and non-steroidal anti-inflammatory drugs (NSAIDs). The maximum recommended daily dose of paracetamol should not exceed 4 g per day, and dose reduction may be needed in hepatic insufficiency. With NSAIDs, there is a ceiling dose for each NSAID, and there is no value in using two NSAIDs together. Paracetamol and NSAIDs can be used in combination.

18.6.5.2 Weak Opioids

Codeine and tramadol are commonly available locally. Dologesic, doloxene and doloxene co. have already been withdrawn from the market. The usual dosage of codeine is 30-60 mg 3-4 times per day. Its opioid strength is equivalent to about one-tenth that of oral morphine, although this varies with individuals as there is genetic variation in the enzyme CYP2D6 that metabolises codeine to morphine. Codeine itself has little direct analgesic effect.[44] Codeine is also useful if the patient has a coexisting cough and requires symptomatic relief. Side-effects include drowsiness and constipation.

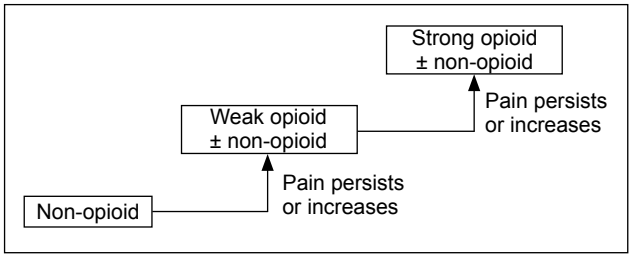

Figure 18.1 Analgesic ladder for control of pain[43]

With regard to tramadol, low dosages will have a weak-to-moderate opioid effect, and higher dosages can have side-effects similar to those of morphine. It is not just an opioid with moderate strength, but also has a central neurotransmitter mechanism of action, inhibiting uptake of noradrenaline and serotonin, therefore with additional analgesic effect in neuropathic pain. At a maximum dose of 100 mg every 6 hours, the equianalgesic effect would not exceed that of 60 mg of morphine over 24 hours. It should not be a substitute for morphine when strong opioids are indicated.[40] Side-effects of serotonin syndrome have been reported when tramadol is co-prescribed with selective serotonin reuptake inhibitors (SSRIs). The starting dose is 50 mg every 4-6 hours, but this can be lowered in frail elderly to 25 mg using drops.

18.6.5.3 Strong Opioids

Morphine is commonly used and remains the first choice of strong opioid analgesic. Other alternative opioids available locally include fentanyl and methadone. The latter two may be used as an alternative in elders, but should be used only in specialist hands. A strong opioid can be used together with a non-opioid, but there is no value in combining an already strong opioid with a weak opioid.

18.6.6 Prescribing Morphine and Managing Its Side-effects

Compared with the young, elderly people have a decreased morphine clearance and smaller volume of distribution.[45] The traditional advice is therefore to 'start low and go slow' to prevent undue adverse reactions in older people. Elderly patients generally require a lower morphine dose than younger patients,[46] but each patient's morphine dose should be titrated individually and care must be taken that elders do not suffer unnecessarily with too low a dose of morphine for too long when in severe pain.

The best route of morphine administration is oral, in either syrup or slow release tablet form: slow-release morphine sulphate (MST). Syrup morphine lasts for about 4 hours whereas MST lasts for about 12 hours. If end-of-dose failure with MST every 12 hours is encountered, the dose interval can be shortened to every 8 hours.

When starting morphine, it is best to prescribe morphine syrup every 4 hours round the clock. A double dose can be given at nighttime so the patient does not wake up due to pain. A 4-hourly as-needed dose of morphine, same as the 4-hourly regular dose must also be prescribed and administered if necessary for breakthrough pain. Sometimes a lower as-needed dose can be given more frequently, even hourly, but this must be carefully monitored. Anticipated incident pain can be pretreated with a stat dose of morphine syrup about 45 minutes before. The morphine dose is increased by 20-50% or by the amount of breakthrough morphine used in the previous 24 hours. A commonly used titrating regimen for increasing morphine dose is as follows, though this serves as a guide only: 5 mg → 7.5 mg → 10 mg → 15 mg → 20 mg → 30 mg → 40 mg and so on. The lowest dose to start can be 1-2 mg, for very frail elders who are opioid-naïve.

Only when the pain is adequately titrated should the morphine syrup be changed to MST. MST allows a better compliance with only two doses per day and achieves better sustained analgesia at night. The equivalent MST dose can be calculated by multiplying the 4-hourly syrup dose by 6 to give the 24-hour total dosage requirement, and dividing it by 2 to get the MST dose every 12 hours. For patients who are unable to swallow, morphine can be given parenterally through a syringe driver, continuously delivering the drug subcutaneously over 24 hours, or through an intravenous pump. The equivalent 24-hour subcutaneous dose is about half the total daily oral dose, and the intravenous dose is about one-third.

18.6.6.1 Side-effects of Morphine

- Constipation: The only side-effect that will persist as long as the patient is on morphine. The best approach is to prevent and treat any constipation before prescribing morphine. Always prescribe laxatives when prescribing morphine. Patients on high doses of morphine often need both stimulant and osmotic laxatives, e.g. Senokot and lactulose.
- Nausea and vomiting: Not every patient on morphine will develop nausea and vomiting. It has been estimated that only about one-third of patients will be affected. Treatment is best with drugs that are effective on the chemoreceptor-trigger zone where morphine induces the vomiting, e.g. haloperidol 0.25-0.5 mg twice daily, prochlorperazine 5 mg 3 times a day, or metoclopramide 10 mg 3 times a day. The side-effect of vomiting is transient and usually subsides after a few days. It should be borne in mind that not all vomiting is due to opioids.
- Drowsiness and sedation: Again not every patient on morphine will develop drowsiness and sedation, and the side-effect will subside after a few days. If drowsiness is troublesome or persistent, it can be counteracted by giving psychostimulants of methylphenidate 10 mg every morning and 5 mg noon in the short term, or by trial of substituting the morphine with another opioid.
- Respiratory depression: This side-effect, although much feared, is in fact rare if there is judicious use

of morphine, used appropriately in physical pain that is responsive to morphine. Caution is needed for patients with lung disease and carbon dioxide retention, where lower doses should be used and titrated very slowly. Carbon dioxide retention should not in itself preclude the judicious use of opioids in terminal stage disease, especially where symptom control is of paramount importance.
- Dry mouth: This can be alleviated by regular mouth care and also use of 65% glycerine for local lubrication as a hygroscopic agent.
- Postural hypotension: This needs to be carefully monitored to prevent falls. All elderly patients on morphine should be advised about fall prevention, and precautions when changing posture. Other drugs that may aggravate postural hypotension should be avoided or minimised.
- Urine retention: This can occur due to the effect of morphine on the urinary sphincter, especially in men with benign prostatic hypertrophy. Hytrin can be tried to counteract this side-effect. Nonetheless a more common reason for opioids to cause urine retention is faecal impaction that may occur if regular laxatives are not prescribed.

18.6.6.2 Myths of Opioids

Several myths are common amongst patients and professionals, and should be clarified / dispelled.
- Physical tolerance: Many patients are reluctant to start morphine for fear that the effectiveness will reduce as disease progresses. This is not usually a problem because there is no ceiling dose of morphine, and dosage can be increased whenever necessary. Also for elders with advanced incurable illness, life expectancy is not usually long enough for significant tolerance to develop.
- Psychological addiction: Many patients worry that they will become addicted to the opioid. Experience tells us that as long as the opioid is prescribed for physical pain, psychological addiction will not develop. The morphine can be successfully tailed down or stopped when the cause of pain is removed.
- Side-effects of morphine are worse than the pain: this is clearly untrue. Side-effects can be prevented, alleviated or minimised, and elders do not have to suffer unnecessarily with pain due to fear of morphine and side-effects.

18.6.7 Alternative Opioids

18.6.7.1 Fentanyl

Fentanyl is an alternative opioid that can be administered via a transdermal, transmucosal, or parenteral route. In Hong Kong, only the transdermal patch and the parenteral injection are available. The transdermal patch lasts for approximately 72 hours. Peak effect of the first dose takes 18-24 hours. Currently available lowest dose of 25 μg per hour is recommended for patients who require about 60 mg of oral morphine over 24 hours. Although it may be used in opioid-naïve patients, it should not be used when pain is fluctuating or uncontrolled, and preferably started only when the effective equivalent dosage of morphine has been determined. Side-effects are generally the same as morphine though may be less, especially with constipation. Its use in elderly people is limited, due to its unpredictable absorption with differences in body temperature, subcutaneous fat, and water compared with the young.[40] One advantage of fentanyl is its safety in renal failure, as the drug is not renally excreted. In terminal illness, subcutaneous use of fentanyl in a syringe driver is preferred when a patient is in renal failure or has intractable intestinal obstruction. Advice of a palliative care specialist should be sought.

18.6.7.2 Methadone

An alternative opioid that differs to morphine by acting more on the kappa receptor, and itself also a N-methyl D-aspartate (NMDA) receptor antagonist, so useful in addition for neuropathic pain. Methadone alone or in combination with morphine has been used in the specialist palliative care setting. Nonetheless it has a long half-life that exceeds the duration of analgesia, and is difficult to titrate in elders. Its pharmacodynamics and pharmacokinetic properties also vary much between individuals. Excessive sedation and respiratory depression may result. Conversion from morphine dose is best through an ad libitum approach. Its use in elders is limited.[40]

18.6.8 Adjuvant Therapy

Adjuvant therapy can be added at each step of the analgesic ladder, and can be pharmacological or non-pharmacological.

18.6.8.1 Non-pharmacological

This includes various modalities such as cognitive behavioural therapy, mindfulness therapy, aromatherapy, other relaxation / diversional therapy, and physical modalities like transcutaneous nerve stimulation, braces or splints, acupuncture, massage, and exercise. It is important to remember the total pain concept, where pain is multidimensional and non-pharmacological pain relief should not be neglected.

18.6.8.2 Pharmacological

Not all kinds of pain are equally responsive to morphine, and adjuvant therapy when used early can help greatly to control pain. Two notable examples are bone pain that responds more to NSAIDs, and neuropathic pain that responds best to antidepressants and anticonvulsants. A combination

of both antidepressants and anticonvulsants may be necessary in neuropathic pain.

18.6.8.3 Antidepressants

A commonly used tricyclic antidepressant is nortriptyline, which is less sedating, at a starting dose of 10-25 mg at night. Caution must be exercised in elders due to its well-known side-effects of arrhythmias, sedation, postural hypotension, urine retention, and other anticholinergic effects. Nonetheless its effective dose is less than the usual antidepressant dose. Its action is probably through central and peripheral action, by inhibiting serotonin and noradrenaline reuptake in presynaptic terminals and modulating the neurotransmission of pain.[47] The onset of action is faster than the antidepressant action. Classic teaching is that the persisting type of neuropathic pain, such as diabetic peripheral neuropathy, responds best to antidepressants. SSRIs, however, have not been found to be very effective.

18.6.8.4 Anticonvulsants

Commonly used drugs include valproate, carbamazepine, or clonazepam. Their action is probably through a membrane-stabilising action, reducing neuronal hyperexcitability by inhibiting sodium channels.[44] Side-effects of drowsiness and ataxia are well known and patients should be closely observed. Allergic reaction to carbamazepine requires genetic testing first. Starting doses are valproate at 300 mg twice daily, carbamazepine 100-200 mg twice daily, and clonazepam at 0.25-0.5 mg at night. A sustained release preparation for carbamazepine and valproate is available. Classic teaching is that the anticonvulsants are especially effective for shooting and paroxysmal pain such as trigeminal or postherpetic neuralgia.

Newer second-line anticonvulsants include gabapentin. Its action is mainly through voltage-gated calcium channels.[48] Its efficacy has been demonstrated in large randomised controlled trials in elders with postherpetic neuralgia and diabetic neuropathy. Side-effects are somnolence, dizziness, cognitive disturbance, and fatigue.[49] Starting dose is 100 to 300 mg daily, increasing gradually to 300 mg 3 times daily if there is no excessive somnolence or other intolerable side-effects, and adjusted according to renal function.[50] Pregabalin is also available, and has an additional therapeutic effect for generalised anxiety disorder.

18.6.8.5 Steroids

Dexamethasone is useful in nerve compression, e.g. nerve root compression by spinal metastases; and hepatic pain due to distension of the liver capsule by liver tumour or metastases. It is also useful for headaches associated with raised intracranial pressure from brain metastases. Dexamethasone at 4-8 mg per day initially can be tried.

18.6.8.6 Bisphosphonates

Intravenous Zometa or pamidronate can be useful for pain from bone metastases, especially in breast cancer or myeloma.

18.6.8.7 Baclofen

An antispasmodic useful for pain from muscle spasms and also has some activity against neuropathic pain through its NMDA receptor antagonist property. Starting dose can be at 2.5-5 mg 3 times daily.

18.6.8.8 Ketamine

Another adjuvant therapy available for treating neuropathic pain. It acts partly through NMDA receptor antagonist, blocking the 'wind up' phenomenon seen in chronic neuropathic pain. Ketamine is an anaesthetic agent that also contributes to its analgesic effect, although side-effects on cognitive function and the cardiovascular system preclude its general use. Ketamine can be given orally, but it is usually given subcutaneously by infusion in the palliative care setting, and should be prescribed by specialists only.

18.6.8.9 Antiarrhythmics

Mexiletine is an alternative drug for neuropathic pain and its action is also through membrane-stabilising action by blocking sodium channels. It should be avoided in patients with conduction block and bradyarrhythmia. It should be reserved for use by specialists, only when pain is refractory to all other medications.

18.6.8.10 Interventional Procedures

Interventional procedures, e.g. intercostal nerve blocks, coeliac plexus blocks, sympathetic blocks, epidurals and intradurals, can also be considered, but with careful weighing of the risk and invasiveness of an individual procedure against the analgesic benefit in the context of an elderly person.

18.6.8.11 Palliative Chemotherapy or Radiotherapy

Palliative chemotherapy or radiotherapy can be useful especially in pain scenarios associated with cancer.

18.7 Summary

With concerted effort by all professionals devoted to geriatrics and palliative care, adequate palliation for our older patients can be achieved. Our elders need not suffer unnecessarily, even at the end of

life. Quality of life at the end of life can equally be enhanced in old age.

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CHAPTER 19

End-of-life Care

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OBJECTIVES

To understand the concept of end-of-life (EOL) care for both community-dwelling elders and those in institutions. To learn how to advocate EOL care with a multidisciplinary approach.

KEY POINTS

- EOL care encompasses all elements of support to persons nearing the end of their lifespan, including the management of symptoms such as pain; and provision of psychological, social, spiritual, community, and practical support as far as possible.
- A good death is one that is free from avoidable distress and suffering for the patient, their family and caregivers; it accords with the patient's or family's wishes; and is reasonably consistent with clinical, cultural, and ethical standards.
- Written orders in the form of an advance directive (AD) provide instructions for care in the event the patient becomes incapacitated and unable to make decisions. An AD can be drawn up when the person is mentally competent.
- An advance care plan (ACP) is a set of written instructions that informs healthcare providers of the type of care a person wants when he / she is seriously ill or dying. Continuous discussion among and adaptation by the healthcare team and the family are necessary throughout the disease trajectory.
- Common treatment decisions in ACP include the application of cardiopulmonary resuscitation, mechanical ventilation, tube feeding, artificial hydration, use of antibiotics, and the place of care.
- When a death occurs in a residential care home for the elderly, a bereavement service should be provided not only to family members, but also to staff and other residents.

19.1 Demographics Data in Hong Kong

The population in Hong Kong is ageing and life expectancy is increasing. The life expectation for males increased from 72.3 years in 1981 to 80.3 years in 2011 and for females from 78.5 years to 86.7 years.[1] The major killers in 2011 were malignant neoplasm (33%), pneumonia (14.2%), heart disease (14.2%), cerebrovascular disease (7.2%), chronic lower respiratory disease (6.2%), and kidney disease (3%). The prevalence of cognitive impairment is also rising (mortality rate 1.2% in the population).[2] There is an increasing demand for residential care homes for the elderly (RCHEs): up to September 2013, 8% of Hong Kong elders were institutionalised.[3,4] With advances in health and medical services, the disability-adjusted life years of elders is prolonged, thus better foresight in advance care planning and end-of-life (EOL) care are crucial, especially for those with known life-limiting chronic disease.

19.2 Definition of EOL Care

Terminology is not consistent and EOL care is referred to in many ways that often overlap, including palliative care, hospice care, comfort care, supportive care, and terminal care. Each term originates from a different cultural or historical background. In some local settings, EOL care has been used synonymously with palliative care for they are very similar in their approach. Generally speaking, palliative care is given to a patient at any time and at any stage of an irreversible illness, whether or not the disease is terminal. In contrast, EOL care describes the physical, psychosocial, and spiritual care and support given during the time preceding death and can start days, weeks, and sometimes even months earlier. It can be considered to be the time period when family and healthcare professionals are aware of the patient's limited lifespan and would not be surprised if death occurred within about 6 months. EOL care encompasses all elements of support to people nearing the end of their life, and includes management of symptoms such as pain, and provision of psychological, social, spiritual, community, and practical support as far as possible. These patients normally fall into one of the following categories:

- advanced, progressive, and incurable condition(s)
- general frailty and decline, punctuated by episodes of acute deterioration and some recovery, such as respiratory and heart failure
- a prolonged gradual decline such as the frail elderly with dementia

It is worth noting that most, if not all of these older patients, have multiple comorbidities, rather than a single chronic disease or cancer. Progressive chronic illness can follow one of the three distinct illness trajectories[5,6]:

- steady progression and usually a clear short terminal phase, e.g. cancer
- gradual decline punctuated by intermittent serious acute episodes, and sudden death, e.g. respiratory and heart failure
- prolonged gradual decline, e.g. frailty, dementia

19.2.1 Appropriate EOL Care: a Good Death

A good death is a highly individualised concept, strongly dependent on the individual's beliefs and cultural background. In general, a good death is one that is: free from avoidable distress and suffering for the patient, their family and caregivers; it also accords with patient and family wishes; and is reasonably consistent with clinical, cultural, and ethical standards.[7] The concept of a 'good death' has been described as including to be able to know when death is coming, retain control of what happens, having dignity and privacy, pain and symptom control, choice and control where death occurs (at home or elsewhere), access to information and expertise, access to spiritual or emotional support, access to hospice care in any location, control over who is present, able to issue advance directives, have time to say goodbye and leave when it is time to go instead of prolonging life pointlessly.[8,9]

19.3 Making an Advance Directive and Advance Care Plan

After identifying the patient who may benefit from EOL care, a care plan should be formulated. The right to self-determination and autonomy is a fundamental human right that guarantees human dignity and the right to physical integrity. To achieve this, written orders in the form of an advance directive (AD) can be drawn up when the patient is mentally competent. They provide instructions for care in the event the patient becomes incapacitated and unable to make decisions. In Hong Kong, standard forms are available in the Hospital Authority. Life-sustaining treatment, including tube feeding, can be withheld if there is a valid AD when the patient is in an irreversible coma, persistent vegetative state, terminal illnesses, or other end-stage irreversible life-limiting condition. Nonetheless until recently ACP and AD have seldom been discussed in Hong Kong.[10] The formulation of AD can be accompanied with a standard do-not-attempt cardiopulmonary resuscitation (DNACPR) [non-hospitalised] order which needs to be renewed every 6 months (see below).

Prior to an AD, the patient, their family and healthcare team can discuss the planning and delivery of EOL care, with consideration given to the patient's beliefs, preferences, and resources alongside best medical practices. An advance care plan (ACP) is a set of written instructions that informs healthcare providers of the type of care a person wants when he or she is seriously ill or dying. DNACPR (non-hospitalised) can also be formulated alongside with ACP. In some foreign countries, ACP must be established by a mentally sound person. Hong Kong has adopted a broader approach and allow establishment of ACP for a mentally incapacitated person via discussion with the family members. Changes in the patient's condition or performance status may lead to changes in the care plan, and continuous discussion and adaptation will forge an individualised care plan throughout the disease trajectory. Recently, information of ACP and DNACPR has been provided by Hospital Authority in both English and Chinese for the public at the Smart Patient website.[11]

The following are common treatment decisions that should be included when drawing up an ACP with patients and their family.

19.3.1 Cardiopulmonary Resuscitation

In the event of cardiac arrest, cardiopulmonary resuscitation (CPR) is required to sustain circulation. In an audit of the survival rate following in-hospital cardiac arrests in Hong Kong, the rate of return of spontaneous circulation was 37.5% and survival to discharge was only 5% (age range, 38-97 years).[12] In addition, the force required for CPR may cause fractured ribs or a collapsed lung. Thus discussion with patients and their family about the decision for DNACPR is vital for those patients in whom CPR would likely be futile. The establishment of the DNACPR plan will help ensure that the patient dies in a peaceful and dignified manner. If the patient and family do not understand the procedure of CPR, medical or nursing staff can provide explanation with the aid of pictures or even a video so that they can better understand the risks and benefits.

It is also important to reassure the patient and their family that a DNACPR decision applies only to CPR. It does not imply that other treatments for reversible causes will be abandoned. In other words, DNACPR forms one part of ACP.

In October 2014, the Hospital Authority implemented

new guidelines for DNACPR that now include DNACPR decisions for seriously ill non-hospitalised patients. In the new guidelines, the DNACPR form has been standardised, and for both hospitalised and non-hospitalised patients there is a mandatory requirement for at least two doctors, one being a specialist, to be involved in the DNACPR decision. In addition, the DNACPR directive for non-hospitalised patients is restricted to special categories of seriously ill patients with an AD and seriously ill incompetent patients with an explicit ACP.

The DNACPR (non-hospitalised) order has to be renewed every 6 months by the attending doctor. If the patient is mentally incapacitated (e.g. suffering from dementia), it can be made with the family members who need to sign on the DNACPR (non-hospitalised) form. Nonetheless, at the time of writing this chapter, ambulance crews of the Fire Services Department are excluded from the guidelines, and the implementation of those that apply to non-hospitalised patients are applicable only to accident and emergency departments (AED) of the Hospital Authority. If the patient is admitted, the healthcare teams in wards can base on the DNACPR (non-hospitalised) to activate a new DNACPR for inpatient after discussion with the patients or the family members.

19.3.2 Mechanical Ventilation

Intubation with ventilatory support in severe pneumonia may sustain a patient while they recover from an acute infection. However, it is not always possible to wean those patients with poor baseline lung function and recurrent exacerbation of chronic obstructive pulmonary disease off ventilatory support. This results in the need for a tracheostomy and the possibility of remaining on a ventilator for the rest of their life.

Non-invasive positive pressure ventilation also may not be a comfortable procedure. Patients need to wear a tight mask with air pumped into the lungs via the nostrils and mouth. It may cause pressure ulcers over the face, a dry mouth, gastric bloating, and pneumothorax.[13] The decision to discontinue ventilatory support is difficult. The family may feel they have made a decision to terminate life, although it is in fact accepting the natural process of dying. In order to make such a decision more comfortable, it is better to plan well in advance in an ACP, preferably before ventilatory support is commenced.

19.3.3 Tube Feeding

As death approaches, reduced appetite is common as body systems start to shut down despite the ongoing need for fluid and food.[14] When a patient cannot or will not eat or drink, medical staff may consider use of a feeding tube. While recovering from an acute illness, a feeding tube may be helpful, but at the EOL it may cause more discomfort and induce more complications than not eating. A recent Cochrane Review found inconclusive evidence that enteral tube feeding provides any benefit in dementia patients in terms of survival time, mortality risk, nutritional parameters, and improvement or reduced incidence of pressure ulcers.[15] Numerous studies have already shown that tube feeding cannot prevent aspiration pneumonia since the gastric contents and accumulation of secretions in the oral pharynx are more likely to be potential sources of recurrent pneumonia.[16]

Sometimes patients may try to remove a feeding tube, resulting in use of restraints or sedation that may cause further distress. Refusing food might be a conscious decision. After excluding mood problems, a trial of short-term tube feeding may be offered to see if it makes a difference, then stopped if there is no improvement.

19.3.4 Artificial Rehydration

In artificial hydration, fluid is administered intravenously or subcutaneously.[17] In the past, artificial hydration was used to prevent death from dehydration that was considered a painful way to die. Gradual dehydration is, however, usually not painful and results in a lessening of awareness about discomfort so that the patient is allowed to succumb naturally.

If the body is shutting down, it cannot eliminate the excess fluids provided by intravenous or subcutaneous hydration, and limb oedema and even pulmonary oedema will develop.[18] Drip site cellulitis or phlebitis may develop, especially in immunocompromised patients. Ultimately, artificial hydration will only prolong dying. It will not prolong living. Legally, artificial hydration is considered a medical treatment that may be refused at the EOL.

19.3.5 Antibiotics

The acceptance of antibiotic treatment may be attributable to the belief that antibiotics are not a burden and should be prescribed for any infection. The use of antibiotics is not fully contradictory to the practice of good EOL care when they are prescribed for symptom control rather than cure.[19] For example, when infections cause pain and discomfort, such as cellulitis and bacterial peritonitis, antibiotics play an adjuvant role.

Most patients who receive EOL care are immobile or bed bound and at increased risk of aspiration and other infections. In these circumstances, the use of antibiotics to treat fever and recurrent infection becomes controversial. If first-line antibiotics are

unable to relieve a patient's suffering, a detailed discussion should be held with the patient or their relatives about whether or not other antibiotics should be tried during this terminal stage of life, especially for those with poor venous access and poor general condition with multiple organ failure. Antibiotic therapy in such situations is unlikely to improve the clinical condition but instead may prolong dying and suffering. Therefore a decision about antibiotic use should be included in the ACP in order to avoid futile treatment. Good antibiotic stewardship is also needed to avoid the emergence of multidrug-resistant organisms.

Pneumonia and urinary tract infection are the two most common infections in patients with advanced dementia. A study showed that over 18 months, 53% of demented residents in nursing homes had a febrile episode, and 41% had pneumonia.[20] Another study reported pneumonia or urinary tract infection as the immediate cause of death in up to 71% of cases.[21] The use of antibiotics in these patients is a controversial issue: their use has been shown to have no demonstrable effect on mortality.[22]

19.3.6 Place of Care

In Hong Kong, dying in a RCHE or at home is not yet common due to cultural taboo and legal issues. Recent studies among Hong Kong RCHEs, however, revealed that up to 30% of residents would prefer receiving EOL care in a RCHE.[23]

Even when elderly patients and their proxies have set up an AD requesting comfort care or dying in their current place of care,[24,25] they are often transferred to a nearby acute hospital if their medical condition deteriorates. Aggressive treatment is then inevitable, leading to unnecessary suffering and futile treatment.[26,27] There is evidence that elderly patients are frequently overtreated, particularly those with a critical illness requiring emergency operation, resuscitation, or intensive care.[28]

Despite this, in the absence of an AD or ACP, it can be overwhelming for the family to be asked to make healthcare decisions for their loved one. In this situation, two approaches can be adopted in decision making. One approach is for the family to put themselves in the place of the patient and try to choose as he or she would. This is called a substituted judgement approach. A second approach is to decide what would be best for the patient, known as the best interests approach. Patients and their family should also be made aware that AD orders and ACP are not permanent and can be changed at any time.[29] A general checklist of an ACP is outlined in Table 19.1.

On some occasions, there may be disagreement among medical staff, the patient and his / her family about a medical care plan. If there is an AD that explains the patient's preferences, it should determine care. Without the guidance of an AD, it may be necessary to obtain a second opinion from a senior doctor with relevant experience, or even seek the opinion of the Ethics Committee in the hospital setting.

Table 19.1 Checklist of an advance care plan

- Cardiopulmonary resuscitation
- Mechanical or non-mechanical ventilation
- Insertion of feeding tube
- Artificial rehydration or nutrition
- Intravenous antibiotics
- Blood transfusion
- Blood taking
- Transfer back to acute unit
- Other treatments, e.g. abdominal paracentesis

19.4 Mood and Spiritual Needs

Care that extends beyond attention to physical needs and includes alleviation of a patient's emotional, social, and spiritual problems should be an essential component of EOL care.[30] Regrettably though, medical staff are often focused only on the patient's disease and physical deterioration, and neglect or do not notice mood problems and psychosocial needs.

Spiritual needs involve finding meaning in one's life and ending disagreements with others if possible. This allows the patient to find peace by resolving unsettled issues with friends or family. Sometimes, asking open questions such as "Where do you find strength?" or "How do you make sense of what is happening?" and "How do you feel about the future?" can be a useful and non-confrontational way of exploring a patient's spiritual needs.[30]

19.4.1 Depression

Patients approaching the EOL may understandably feel depressed or anxious. Medical staff might misinterpret mood problems as symptoms of physical problems. For example, a low mood may be regarded as malaise due to end-stage disease and advanced age. Physicians also tend to avoid increasing the drug load of patients with multiple comorbidities and worry about the potential side-effects of antidepressants.

Some reliable symptoms of major depression in a patient with end-stage disease are persistent dysphoria, anhedonia, feelings of helplessness, hopelessness and worthlessness, and loss of self-esteem.[31] The somatic symptoms of depression might be unreliable as they partly reflect the underlying organ failure. Setting a screening question such as "Do you feel depressed most of the time?" may help

identify a depressed patient. This has been proven to be a highly sensitive and specific question in this population.[32]

19.5 EOL Care in the Dying Phase

The last weeks of life for most people are characterised by a progressive physical decline, frailty, lethargy, worsening mobility, reduced oral intake, and little or no response to medical intervention. This dying phase, however, may not be so evident in those with advanced dementia. When the patient is close to death, clinical signs such as reduced consciousness level, cardiovascular changes (e.g. peripheral vasoconstriction), and change in respiratory pattern (e.g. Cheyne-Stokes breathing) are common. When dying is inevitable, the focus of care should move away from cure of the disease to providing comfort. Review of the ACP with the family and confirmation of earlier EOL decisions is essential. The physician should explain to the family that the patient is now dying and when and how death might be expected to occur. If the family does not accept that the patient is dying, the physician will need to explain clearly the basis of this judgement and provide enough time and opportunity for the family to ask questions (Table 19.2).[33]

19.5.1 Prescription during EOL

During the dying phase, some medications may be useful to soothe the distress. Generally, there are three principles about prescription during EOL.[34]

19.5.1.1 Stop Non-essential Drugs

In general, this means stopping all drugs that are not providing a symptomatic benefit, such as lipid-lowering agents and antihypertensives. The definition of essential medication is controversial, however, and benefits should be carefully weighed against adverse side-effects, e.g. over-sedation hindering meaningful social interactions. Continuation of some medications such as proton pump inhibitors for the prophylaxis of gastrointestinal bleeding, or laxative or stool softeners for patients on narcotic analgesics, may be considered useful for symptom control and should be continued until the very last moment. Analgesics, sedatives, anti-emetics, antipyretics, diuretics, steroids, mucolytics, and heart rate–controlling agents are considered essential medications in view of their importance in symptom control in the dying phase. Antibiotics are usually stopped but can be continued if they are providing symptomatic benefit.

19.5.1.2 Convert Essential Drugs to the Subcutaneous Route

Essential drugs may need to be administered subcutaneously, especially in patients who are having trouble swallowing tablets and in those who may have nausea and vomiting. Regular drugs can be delivered via a syringe driver or subcutaneous infusion pump that runs over 24 hours. The subcutaneous route is preferred over the intravenous one as it is relatively non-invasive, provides easy dose titration, and allows drug combinations.

19.5.1.3 Anticipatory Prescribing

Depending on the availability of 24-hour professional medical or nursing support in the care setting, regardless of current symptoms, patients in the end stage of life should have prompt and timely access to an analgesic, an anxiolytic, an antiemetic, and drugs to reduce bronchial secretions on an 'as required' basis, so that there is no delay in responding to a symptom when it occurs.

19.5.2 Useful Tips in Symptom Control in EOL

Besides pharmacological treatment, which is covered in Chapter 18 on Palliative Care and Pain Management, there are numerous non-pharmacological measures to make patients more comfortable in their dying phase,[30] such as physiotherapy, occupational therapy, aromatherapy, music therapy, meditation, and body massage.

19.5.2.1 Pain

Not everyone who is dying experiences pain, but over a third do. After determining the nature and origin of pain, an appropriate analgesic should be prescribed. Medical staff should explain to the patient and family that EOL care should focus on pain relief without

Table 19.2 Questions that the family may raise before making a decision on any investigation and treatment[33]

Before making a decision on any investigation and treatment, the medical team should answer the following questions that the family may raise:
1. What is the purpose of the test or treatment?
2. Will the treatment speed up or slow down the dying phase?
3. Will the treatment help the patient get better and change his / her quality of life?
4. How long will the treatment take to make a difference?
5. What are the side-effects of the treatment?
6. What will happen if the treatment does not work or if they do not try this treatment?
7. Since there is no cure, what can be expected to happen in the coming days or weeks?

worrying about possible long-term problems of drug dependence or abuse.

19.5.2.2 Breathing Problems and Respiratory Secretions

Shortness of breath is a common experience at the EOL. One can try to raise the bed, open a window, or have a fan to circulate air in the room. Administering oxygen via a mask or nasal cannula may also help reduce breathlessness.

When patients are very near death, their noisy breathing makes a gurgling or rattling sound, caused by build-up of fluid in the throat (death rattle). Turning the patient to rest on one side, or adding some medication such as hyoscine may help to reduce this.

19.5.2.3 Agitation

Some patients show fluctuating changes in consciousness level towards the EOL. If the patient becomes agitated or restless, soft music, staying with family in a peaceful environment, simple physical contact such as holding hands, a touch, or a gentle massage may help. These can make the patient feel connected to their loved ones and soothe their anxiety. Sedation may be needed if these measures do not help. The aim is for the patient to be in a sleep-like state, not to hasten death. Comfort measures such as analgesics should be continued but sedatives can be stopped at any time when the patient's level of consciousness declines.

19.5.2.4 Skin Irritation

Skin problems can be very uncomfortable, especially for those whose skin is drier and more fragile. Dryness on parts of the lips and eyes can be a common cause of discomfort near death. A lip balm or some olive oil may help. A damp cloth placed over closed eyes or regular eye drops might relieve dryness. Ice chips can be used if the inside of the mouth appears dry or wiping the mouth with a damp cloth or cotton ball can be attempted.

It is not uncommon to have pressure ulcers in the EOL stage. Close monitoring of any discolouration especially on the heels, hips and sacral region, regular turning, and using a foam pad or special mattress may help to prevent pressure ulcers (refer to Chapter 44 on Pressure Ulcers).

19.5.2.5 Digestive Problems

Nausea, vomiting, and constipation are common in the end stage of life. Causes can be physical (immobility, weakness, and bowel obstruction), biochemical (poor intake, dehydration, and hypercalcaemia), central (raised intracranial pressure), or drug-related (particularly opioids). The appropriate antiemetic should be chosen on the basis of the underlying cause. Occasionally, more than one antiemetic is needed for better symptom control. Constipation is best treated with a combination of a stimulant and a stool softener. If the patient cannot tolerate an oral laxative, rectal suppositories may be needed although a balance must be struck between the benefit and the discomfort of the intervention.

19.5.2.6 Temperature Sensitivity

Close observation of temperature changes may be needed when patients cannot speak for themselves. For example, repeated removal of a blanket may indicate that they feel hot. If the patient is hunching the shoulders, pulling the covers up or even shivering, another blanket and warm pad may help.

19.6 Multidisciplinary Approach in EOL Care

A multiprofessional team including staff from different healthcare and non–healthcare professions is proven to improve satisfaction, and can identify and deal more with patient and family needs,[35] shorten the stay in acute hospital units, and improve pain and symptom control[36] when compared with conventional care. Doctors and nurses with special training play an irreplaceable and invaluable role in EOL care. If the physician takes up the role of gatekeeper of information and the one to break bad news to patients, the nurse will be the one who can assume the role of communicator and translator of information and feelings, helping the patient and their family to understand and obtain the best possible care. Shared decision making between physician and nurse specialist can result in more appropriate care and increased physician / nurse, patient, and family satisfaction. This can be achieved by providing further training to nurses about pain control and comfort care,[37] using specialised daily round forms, having joint grand rounds, patient care seminars and inter-professional dialogues, offering communication training, and building up a collaborative practice model.[38] Geriatricians possess the specialist skills for EOL care across the different settings of acute, subacute, and community situations, especially in the management of polypharmacy, delirium, pressure sores, incontinence, and malnutrition etc., as well as providing communication, psychological, and spiritual support as necessary. In addition, geriatricians are often consulted for specialist advice on ethical and legal issues, and treatment decisions.[39] In addition to the physician and nurse in charge, the following professionals play a role in EOL care.

19.6.1 Role of the Family Physician

Family physicians have an important role in coordinating EOL care in the community. They can

identify those in their practice who are nearing death, make appropriate specialist referrals, and initiate EOL planning with a patient's family, including setting up of an AD or ACP. Good EOL care is all about planning, coordination, communication, and ensuring continuity of care. Enhanced training for nurses to boost their confidence in managing death and dying is needed.

19.6.2 Role of the Physiotherapist

The physiotherapist is concerned with identifying and maximising movement potential, within the spheres of promotion, prevention, treatment, and rehabilitation.[40] Their services can be directed to three areas: delivering direct patient care, educating the patient-family care unit and fellow health professionals, and functioning as a team member.[41] They offer treatment to maintain optimum respiratory and circulatory function; prevent muscle atrophy and shortening; prevent joint contractures; optimise pain control; enhance independence and function; and educate and encourage participation of the carer.[42]

19.6.3 Role of the Occupational Therapist

Occupational therapists are trained to deliver care that is attentive to the unique and individual needs of their clients in need of intervention. They can address activities of daily living, psychological and emotional issues, exercise programmes, splinting and positioning, energy conservation, relaxation techniques, seating and mobility, comfort, adaptive and assistive equipment, support and education for family caregivers, connecting the patient with community services, and conducting home assessments.[43]

Some literature indicates that in the end stages of life, people with terminal illness may develop a sense of living through occupational engagement that involves: living while dying; reworking everyday life; being guided by the will of the body, focusing on relationships, attending to the small things in life, and engaging existential orientations.[44] These types of intervention play a major role in assisting individuals to live life fully, safely and comfortably at home, preventing injury, and controlling pain while facing death.

19.6.4 Role of the Social Worker

Social workers in EOL care should demonstrate an attitude of compassion and sensitivity to their patient, respecting their right to self-determination and dignity.[45] Their role in EOL care can be defined by five main functions: assessment, counselling, liaison with local resources and agencies, training and development activities, and staff support.[46] The social worker can facilitate the setting up of patient support groups, bereavement groups, and a volunteer team to supplement the work of EOL care, particularly in the community. They can offer training to the voluntary worker and act as the link between them, the carers, and the hospital.[6]

19.6.5 Other Healthcare Professionals

The following professionals should also be available to offer support:

- dietitian
- speech therapist
- clinical psychologist
- pharmacist
- podiatrist
- prosthetics and orthotics
- chaplain

19.7 Bereavement

19.7.1 Bereavement of Staff

Staff in a care team can experience loss, both physical and psychological, following the death of a resident with whom they have established a bond and relationship. Unresolved grief may result in withdrawal from close or meaningful involvement with other residents, clients or colleagues. An opportunity to formally acknowledge their loss and access to adequate bereavement support should be set up (Table 19.3[47]).

Staff empowerment in grief and bereavement support is important when developing EOL care in RCHEs. This can be achieved by workshops, individual interviews, reviewing staff feedback about a previous death, conducting a written survey and close observation.

19.7.2 Bereavement of RCHE Residents

The impact of loss and grief on other residents in the same RCHE and families should not be overlooked. Residents can develop very close relationships as they are often in contact with each other on a daily basis. It is therefore important for the loss experienced by other residents to be acknowledged. They may experience a range of feelings following a person's death including fatigue, shock, sadness, anger,

Table 19.3 Strategies to support the aged care team dealing with grief[47]

• Publicly acknowledging the death
• Avoid throwing oneself into work with no time allowed for grieving
• Permission to cry and show anger
• Seek help and accept support when offered
• Memorial services, grief box, ceremonies
• Staff debriefing
• Getting involved in training activities and workplace activities
• Follow up the bereaved family

helplessness, loneliness, and anxiety. They may also experience physical symptoms including hollowness in the stomach, tightness in the chest or throat, breathlessness, over-sensitivity to noise, weakness in muscles, and lack of energy as the result of the loss. Some behavioural changes may be observed in bereavement including appetite disturbances, avoidance of reminders of the lost person, social withdrawal, absent-mindedness, sleep disturbances, crying, calling-out, or treasuring objects. In some extreme cases, the loss can lead to a disordering of thought including hallucination, disbelief, confusion, pre-occupation, and sense of presence.[47]

A memorial service is a useful means to provide bereavement support in RCHEs. An introduction to life-and-death education to residents can also facilitate them to prepare for their own EOL journey in the future.

19.8 Staff Empowerment and Engagement

The health profession, for all its wisdom and expertise, is generally not good at prospectively identifying those who are nearing death. The concept of dying is a very difficult concept in the medical system because healthcare staff are not trained to let people die.

Reflection and continuous learning about EOL care should be integrated into daily practice. This can be achieved through structured multidisciplinary meetings, mortality and morbidity meetings, and joint ward rounds with colleagues who have a special interest and expertise in EOL care. Regular meetings with visiting medical officers in RCHEs should be encouraged to further the understanding of each other's role and services, and to improve coordination of care across traditional boundaries.

Junior doctors are often required to care for dying patients. However, they are often making difficult decisions about titrating opioid analgesics, anxiolytic sedatives, or antisecretory drugs. Thus EOL care should be an important part of undergraduate and postgraduate medical training and an attachment to a geriatric or palliative care team (PCT) can broaden their experience of EOL care in the hospital and in the community.

19.9 EOL Care for People with Cognitive Impairment

Advanced dementia is regarded as a 'terminal illness' with a similar symptom burden and prognosis to advanced cancer. Compared with patients with oncological disease or end-organ failure, demented patients generally have poorer access to good-quality EOL care. Interventions such as enteral tube feeding, artificial hydration, antibiotic therapy, and fever management remain in use despite little evidence that they improve quality of life or other outcomes. Some studies have suggested that patients with advanced dementia have symptoms similar to those of patients with terminal cancer, including pain, dyspnoea, eating problems, pressure ulcers, and agitation.[20,48-50] Residents of RCHEs with advanced cognitive impairment have been shown to be frail, exhibit multiple comorbidities and a high mortality, and be frequent users of outpatient, emergency, and inpatient services.[51]

Due to their impaired cognitive function, their family and the healthcare team may find difficulty in identifying their needs. In such circumstances, direct observation or a validated observational tool such as Abbey Tool,[52] Disability Distress Assessment Tool[53] for pain assessment, and Non-Communicative Patient's Pain Assessment Instrument[54] can be used.[55] EOL care for people with dementia should involve the intervention in the hospital, nursing home, and the community. Studies have proven that establishing a clear treatment plan with the goal of enhancing patient comfort can minimise the prescription of unnecessary drugs in the last week of life,[56] increase the likelihood of prescribing analgesia, and decrease transfers to the acute hospital.[57]

19.10 Pilot EOL Programmes in the Community

Different pilot community EOL programmes have been running in Hong Kong since 2008/2009. These programmes usually involves collaboration with the AED, the Community Geriatric Assessment Team (CGAT), and staff of RCHEs to facilitate EOL care for residents and direct admission from RCHEs to inpatient care. Knowledge and service gaps among staff and residents have been addressed by the geriatric team with training provision.[58] The use of AD, ACP and DNACPR (non-hospitalised) forms, and family conferences have been introduced during which options for resuscitation, place of care, and use of a ventilator, artificial hydration, tube feeding, and antibiotics will be proactively discussed with the patient and their family. Below are two examples of pilot community EOL programmes in Hong Kong.

1. In New Territories East, an EOL programme which aims at promoting quality EOL care for RCHE residents, through the establishment of ACP and introduction of a new care pathway has been in place.[59] This pathway bypassed the emergency room and acute medical wards by facilitating direct clinical admission to an extended care facility. A study on this programme shows that nearly 40% of EOL patients could be managed entirely in an extended care setting without compromising the

quality of care and survival.

2. In Hong Kong West, CGAT piloted the EOL programme for RCHE in cooperation with two care and attention homes in September 2009.[60] The objectives of the programme were to provide palliative care for older residents in RCHEs, ensured elderly persons could enjoy a dignified and comfortable period in a familiar environment before they died, reduced acute hospital admission and to serve as a model of care in Hong Kong. In the programme, there were weekly EOL clinics and family conferences. ACP and DNACPR (non-hospitalised) would be established. If patients were mentally sound, AD would also be created if the older residents wish to. The patients / family members could select one of the two pathways, namely the Hospital Pathway and AED Pathway. In the Hospital Pathway, elderly patients would be clinically admitted to a geriatric step-down hospital (instead of acute hospital) which was suitable for EOL care via an expedite pathway. In the AED Pathway, elderly would stay in RCHE as long as possible with support from the RCHE staff and EOL team of Hong Kong West CGAT. At the last moment (patients become unconscious, breathing becomes shallow and / or blood pressure starts to drop), they would be transferred to AED by ambulance. Resuscitation would still be carried out by the ambulance men as The Fire Services Ordinance in Hong Kong stipulates that resuscitation has to be carried out to their clients. When the patients reach AED, resuscitation would not be offered. Instead, a quiet environment would be offered to elderly patients, allowing them to pass away peacefully in the AED. The pilot showed that AED Pathway was logistically feasible and could serve as a new model of care in some local better staffed and equipped RCHEs to foster dignified death.

19.11 Pilot EOL Programme in Hospital

As many older patients may pass away in wards, there is a need to enhance EOL services at the inpatient setting. In some geriatric hospitals, pilot EOL programmes have been in place to enhance EOL care. For example, in Hong Kong West, TWGHs Fung Yiu King Hospital has established the End of Life Clinical Plan (EOL CPi) since 2012. A study on the EOL CPi shows that a tailor-made EOL clinical plan could be useful to guide clinical team in fostering dignified death among dying older patients in a geriatric step-down hospital.[61]

19.12 Advance Care Planning and New Development of EOL Care in Residential Care Homes in Hong Kong

In October 2015, a programme to "Enhance CGAT Support to EOL Patients in Residential Care Homes for the Elderly" was implemented by Hospital Authority in Hong Kong. It was first commenced in four clusters, namely Hong Kong West, Hong Kong East, New Territories West and East in 2015. In the programme, the usual CGAT service is enhanced, with link nurse as case managers for elderly people in RCHE, including those with advanced dementia, approaching EOL stage. CGAT also collaborated with palliative specialists to develop and carry out this programme. PCT assists in providing training to RCHE and CGAT staff, as well as in complex case management. In the programme, ACP would be discussed and DNACPR (non-hospitalised) order would be made (if agreed by patient or family members). AD may also be signed if the patient is mentally sound. According to the plan of Hospital Authority, the "Enhance CGAT Support to EOL Patients in Residential Care Homes for the Elderly" service will expand to all clusters in Hong Kong by 2018/2019.

19.13 Careful Hand Feeding for Advanced Dementia

Feeding problems are common in older people with advanced dementia. When eating difficulties arise, unless there is a valid AD refusing enteral feeding, tube feeding is often started. Tube feeding itself has many pitfalls and complications. To date, no benefits in terms of survival, nutrition and prevention of aspiration have been demonstrated. The use of feeding tube leads to patient discomfort, increased use of restraints and greater likelihood of pressure sore development.[62,63] Studies show that RCHE residents with feeding tubes are frequently transferred to emergency to take care of tube complications such as blockage and dislodgement.[64] So far, studies have not shown survival benefits in older people with tube feeding.[65] In the 2014 positional statement of feeding tubes in advanced dementia published by the American Geriatrics Society, feeding tubes are not recommended.[66] It emphasises that careful (or comfort) hand feeding (CHF) should be offered as it is at least as good as tube feeding for the outcomes of death, AP, functional status, and comfort.[67] Similar recommendations are made by the Australian and New Zealand Society for Geriatric Medicine.[68] In September 2015, the Hospital Authority Guidelines on Life-Sustaining Treatment in the Terminally Ill was updated, providing a clear picture of CHF from the ethical perspective.[69] In some geriatric step-down hospitals, CHF is already incorporated as one of the management strategies for EOL patients. Notwithstanding, there are many practical issues if tube feeding is not used in older patients with advanced dementia locally. Training of doctors, nurses and other members of the healthcare teams are vital to the promulgation of CHF. There is an urgent

need to enhance the present wards in public hospitals with a more elderly friendly environment. RCHEs staff training and ratio will be important factors of whether CHF can be smoothly practiced in the community of Hong Kong. Without a well-prepared receiving end, patients on CHF will be soon put on enteral tubes. The Social Welfare Department can play a role in to encourage RCHE in practising EOL care and CHF. More palliative care training should be given to primary doctors who look after older people with advanced dementia.[70]

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CHAPTER 20

FALLS

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OBJECTIVE

To learn the prevalence and impact of falls in old age, the causes and risk factors of falls, as well as medical assessment, multidisciplinary multifactorial risk factor assessment and intervention approach, and evidence-based strategies in prevention of falls and fractures.

KEY POINTS

- Falls occur commonly in the older population. Fall (instability) is considered to be one of the giants of geriatrics.
- Fall risk screening is important. Older people should be screened for risk of falls at every medical encounter at least yearly with the following screening tools: (a) asking the older people, "Did you have any fall in the past one year?"; and (b) Timed Up and Go test.
- One can follow the algorithms for the management of older people who have fallen. Both intrinsic and extrinsic (especially medication and environmental) factors should be delineated.
- Fall prevention strategies with proven efficacy are available for older people in the community, nursing homes, and hospital settings.
- Older people with high risk of future falls should be referred to multidisciplinary, multifactorial assessment and intervention usually at fall clinics or geriatric day hospitals.

20.1 Introduction

External causes (including intentional self-harm, various accidents and falls) rank the sixth leading cause of death in Hong Kong. Transport accidents, intentional self-harm, and falls occur commonly in the older population.[1] The risk of falls and accidents increases with age. These occur both inside and outside the homes of the older people and are due to both external and internal factors. Older people have more adverse consequences of falls compared with the younger population (Table 20.1).

20.2 Epidemiology of Falls in Old Age

Fall is commonly defined as an event that results in the patient or a body part of the patient coming to rest inadvertently on the ground or other surface lower than the body.[2] This definition is widely adopted in Hong Kong.

Data from international literature show that approximately 30% of community-dwelling people over the age of 65 years fall each year. The fall rate in institutions is higher. About 20% of fall incidents require medical attention and less than 10% of falls result in a fracture.[3]

In Hong Kong, the annual fall rates in older Chinese community-dwelling people ranged from 18-29%.[4-10] Among these falls, 47% occurred indoors and 53% occurred outdoors.[4] A higher incidence of falls in winter was also observed.[11] Fallers are associated with significantly greater number of hospitalisations and visits at specialist doctor clinics and emergency department than non-fallers.[12] Approximately 70-75% of local fallers reported injuries,[4,9] while fractures occurred in 4.6-8% of the subjects.[4,5] Besides, 4.4% of those who fell died within 9 months[9] and the 1-year fall-related mortality rate was around 1.3% (Table 20.2).[4]

Fall rates have been shown to be generally lowest in acute settings in hospitals and highest in long-stay institutions with those in rehabilitation setting being in between. The fall rate usually ranged from 2.3-7 per 1000 patient bed-days.[13] Approximately 30% of inpatient falls resulted in injury, with 4-6% resulting in serious injury. The reported overall fall rate in public hospitals in Hong Kong was, however, only 0.43 per 1000 patient bed-days and the rates also increased with patients' age.[14] Further studies are needed to determine whether this low fall rate is due to underreporting of minor falls or some unknown factors.

20.3 Causes and Risk Factors of Falls

Falls in older people are typically due to an interaction of various predisposing risk factors as well as precipitating causes. Risk factors are generally classified as intrinsic and extrinsic (including environmental).[15] Older people have many age-related changes (degeneration or illness) that predispose them to falls (Table 20.3).[15] Precipitating causes include acute illnesses like cardiac arrhythmia, urge incontinence, dizziness, and risk-taking behaviour.

Table 20.1 Consequences of falls and accidents in older people

↑ Hospitalisation
↑ Inpatient stay
↑ Disability / dependence
↑ Rehabilitation period
↓ Confidence / self-esteem
↓ Social activities
↑ Institutionalisation
↑ Risk of dying

Table 20.2 Complications of fall in older people

Injuries
Painful soft tissue injuries
Fractures
Hip / femur
Humerus
Wrist
Ribs
Subdural haematoma
Hospitalisation
Complication of immobilisation
Iatrogenesis
Disability
Due to physical injury
Due to fear, loss of confidence
Institutionalisation
Death

Eleven most important risk factors as identified from international literatures include: muscle weakness, history of falls, gait deficit, balance deficit, use of assistive devices, visual deficit, arthritis, impaired activities of daily living (ADL), depression, cognitive impairment, and age over 80 years. Other possible risk factors are the use of psychotropic medication, antiarrhythmic medications, digoxin, and diuretics.[16] Local studies also showed that increasing age, fall history, decline in ADL, living alone, multiple medication usage, gait abnormalities, use of walking aids, self-perceived poor health, and comorbidities were independent risk factors for falls in the community.[4-10] Generally speaking, poor health and musculoskeletal conditions are predictive of falls. Environmental hazards are also important in causing falls in older people (Table 20.4).

There is also interaction and synergism between multiple risk factors. Tinetti et al[17] showed that the fall rate rose from 27% with no or one risk factor to 78% with four or more risk factors. Local studies also showed the additive effects of independent risk factors. Proportion of fallers with one or more falls increased from 10.7% in people with no independent risk factors to 41.7% in people with three or more independent risk factors.[9]

20.4 Assessment and Management

Initial medical assessment of older people with falls involves a fall-focused history, detailing circumstances of fall, precipitating factors, and consequences. Injuries should be attended to in

Table 20.3 Age-related factors contributing to instability and falls

Factor	Description
Changes in postural control and blood pressure	Decreased proprioception Slower righting reflexes Decreased muscle tone Increased postural sway Orthostatic hypotension Postprandial hypotension
Changes in gait	Feet not picked up high enough Men develop flexed posture and wide-based, short-stepped gait Women develop narrow-based, waddling gait
Increased prevalence of pathological conditions predisposing to instability	Degenerative joint disease Fractures of hip and femur Stroke with residual deficits Muscle weakness from disuse and deconditioning Peripheral neuropathy Disease of deformities of the feet Impaired vision Impaired hearing Impaired cognition and judgement Other specific disease processes (e.g. cardiovascular disease, Parkinsonism)
Increased prevalence of conditions causing nocturia	e.g. Congestive heart failure, venous insufficiency
Increased prevalence of dementia	-

acute fall. A witness should be sought to look for unrecognised syncope due to amnesia or cognitive impairment. Other relevant history includes past medical illnesses, medications, and functional status (Table 20.5). Examination should include testing of gait, balance, lower limb joint function, and relevant cardiovascular and neurological examination. Postural blood pressure, vision, feet, and footwear should also be examined (Table 20.6). Further investigations to be undertaken should be guided by the history and examination findings.

In the Get-up and Go Test, the abnormalities are graded on a 5-point scale by observing the deviation from a confident and normal performance.[19] In the Timed-up and Go Test,[18] the patient is observed and timed while he / she rises from an arm chair, walks 3 metres, turns, walks back, and sits down again. Regular footwear should be worn and customary walking aid used but without any physical assistance. Those who complete the test in less than 20 seconds are usually independent in mobility, toileting, and going out. Those requiring more than 30 seconds usually need assistance for many mobility tasks. Further studies of the Timed Up and Go test point to a lower cut-off at 12 seconds for normal mobility and fitness (non-frailty).[20,21] Other detailed assessments of balance or gait (e.g. Berg Balance Scale,[22,23] Functional Reach Test,[24] and Performance Oriented Mobility Assessment[25] are usually undertaken by the multidisciplinary team (Appendix 1[22-25]).

Older persons reporting a single fall with a clear cause and with no demonstrable balance or gait abnormalities may not require further evaluation. Pre-syncope or syncope should be identified, investigated, and managed accordingly. Other modifiable risk factors for falls should be recognised and corrected. Examples are postural hypotension, unnecessary psychotropic medication, hypoglycaemia, and visual impairment. Patients should also be counselled on the importance of regular exercise and a healthy balanced diet rich in vitamin D. Patients with recurrent falls, especially unexplained falls, falls requiring medical attention, or gait and balance problems should be considered for referrals to specialised centres, like falls clinic for multifactorial assessment and intervention.[16]

Comprehensive assessment by a multidisciplinary team (Table 20.7) is usually needed to delineate all the contributing risk factors and formulate a management plan. This multidisciplinary, multifactorial assessment and intervention (MMAI) approach has clear evidence in clinical studies that it will reduce fall rates in older people. MMAI is best delivered by a multidisciplinary team that includes a physician and nurse with experience in falls and care of older

Table 20.4 Environmental hazards

Furniture that is unstable and / or of inappropriate height
Beds / toilets of inappropriate height
Uneven stairs and inadequate or no railing
Steps and kerbs at entrance
Cracked and uneven sidewalks
Obstacles on the floor, e.g. cords, wires, frayed carpets
No grab bars
Slippery floors and bathtubs
Poor lighting or glaring

Table 20.5 Assessment of older people with falls: history

General medical history
Fall history
Any loss of consciousness?
• Could you remember in detail what happened after the fall?
• Could you get up after the fall?
• Any witness?
Circumstances of the fall
• Time and place
• Witness' report
• What were you doing?
• Relationship to changes in posture, head movement, cough, toileting
Premonitory or associated symptoms
• Dizziness, vertigo
• Palpitations, chest pain, shortness of breath
• Sudden neurologic symptoms (weakness, sensory disturbance, dysarthria, ataxia, confusion, dysphasia)
• Aura
Incontinence (urine / bowel)
Medications (especially antihypertensive, psychotropics)
Patient's own thought about the cause
• Aware of impending fall? Or unexpected?
• Accidental? Tripped or slipped / pushed by someone?

Table 20.6 Assessment of older people with falls: physical examination

Vital signs
• Temperature
• Respiratory rate
• Blood pressure / pulse and postural change (lying, standing)
Skin
• Turgor, pallor, trauma
Eyes
• Visual acuity
Cardiovascular
• Arrhythmias
• Signs of aortic stenosis
• Carotid bruits
• Carotid sinus sensitivity
Extremities
• Arthritic joints and deformities
• Range of motion
• Fractures
• Podiatric problems (calluses, bunions, ulcers, poor shoes)
Neurologic
• Mental status
• Focal signs
• Muscles (weakness, rigidity, spasticity)
• Peripheral nerves (especially position sense)
• Cerebellar (especially heel-shin test)
• Extrapyramidal signs (resting tremor, bradykinesia, other involuntary movements)
Gait and balance
• Timed Up and Go test[18]

people, physiotherapist, and occupational therapist. Other supportive members include prosthetists and orthotists, podiatrists, and social workers.[3] The assessment is geared to the delineation of intrinsic and extrinsic (including environmental) risk factors of falls for the individual, especially looking for the remedial ones. The intervention, an interdisciplinary one, must follow the assessment findings and usually includes the following[3,16]:

- group or home-based exercise like balance, gait and strength training, such as tai chi;
- modification and withdrawal of medications especially psychotropic medications;
- treatment of postural hypotension and cardiovascular disorders, e.g. arrhythmia;
- environmental hazard modifications;
- vitamin D supplementation for patients with or at risk of vitamin D deficiency; and
- assistive device use

Fall clinics and geriatric day hospitals are the usual venues for MMAI and local experience has shown the feasibility of successful implementation. The benefit of MMAI in fall prevention is considered to be modest — about one-quarter reduction in future falls.[3] The number needed to treat is estimated to be about 11.[26] A sufficient duration, intensity, and frequency of exercises to facilitate physiological changes have to be considered. Appropriateness of and adherence to exercise programmes for each individual should be addressed. There is also the need to translate clinical practice into home-based programme so that the intervention gains are maintained. Attention should also be paid to improve confidence and decrease fear of falling.

20.5 Fall and Fracture Prevention in Older People

There is strong evidence from randomised controlled trials showing the efficacy of fall prevention strategies in older people.[3] Many good evidence-based guidelines are present to help the development of fall prevention services.[16] However, direct evidence

Table 20.7 Comprehensive multidisciplinary fall assessment of older people

History of fall circumstances — time, place, activity at the time of falls, triggering causes, and consequences of falls; proxy interviews needed if the faller is demented or too ill to report
Review of medications
History of acute or chronic medical problems
Examination of sensory systems especially visual acuity, vestibular function, and proprioception
Testing of gait and balance and lower extremity joint function
Examination of mental status, cognitive functions, and mood
Other neurological assessment including muscle strength, lower extremity peripheral nerves, reflexes; tests of cortical, extrapyramidal, and cerebellar functions
Cardiovascular assessment including heart rate, rhythm, electrocardiogram, postural blood pressure, heart sounds and murmurs and, if appropriate as in the case of syncopal falls, tilt table test, and carotid sinus massage
Incontinence
Assessment of foot condition and footwear
Walking aids assessment
Environmental hazard evaluation
Psychological and behavioural problems and risk behaviours

Table 20.8 Overview of fall and fracture prevention strategies in older people
Interventions shown in *italics* were shown to be effective according to latest Cochrane review or national guidelines.[3,16] The others are logical approaches without solid evidence of effectiveness

Community	Nursing home	Hospital
• *Exercise: Multicomponent, group or home-based* • *Balance exercise like tai chi* • *Home safety assessment and modification** • *MMAI** • *Vitamin D supplement in vitamin D deficiency* • *Expedited first eye cataract surgery* • *Withdrawal of antipsychotic medications* • *Bone health assessment and treatment*	• *Vitamin D supplement* • *MMAI* • Safe environment • Staff education • Medication review • Avoidance of restraint • *Hip protectors*† • *Treatment of osteoporosis*	• *MMAI* • Fall risk screening • Patient and staff empowerment • Safe environment • Attention to immobility, urinary and cognitive problems • Avoidance of restraints • Timely reporting and monitoring of falls events • Coordinated multidisciplinary teamwork and rehabilitation‡ • Secondary prevention of fractures and fall assessment‡

Abbreviation: MMAI = multidisciplinary, multifactorial assessment and intervention
* Mainly for older adults with high risk of falls
† Indicated for secondary prevention in older adults with high risk of recurrent falls
‡ Tertiary prevention in patients with hip fracture

that fall prevention leads to a decrease in significant injuries and fractures, though logical, is lacking. As majority of osteoporotic fractures result from falls, it is crucial that all at-risk patients should have both falls risk assessment and bone health assessment.[27] Table 20.8[3,16] summarises those evidence-based strategies to prevent falls and fractures in different settings and different target populations.

In the community, screening older people with minor falls or high fall risks in the general public followed by MMAI has demonstrated clear evidence of effectiveness. For primary screening, the following are recommended.[16,27]

- An older person should be asked at least once a year about falls in the past 1 year.
- Those who report a single fall should undergo the Timed Up and Go test.[11] Suboptimal performance of the test should lead to MMAI.
- The site of screening should be any contact point of the older people with the health service or social service centres. These may include general outpatient clinics, general practitioners, accident and emergency departments, social day centres or any general health check service. Screening should be a part of routine assessment, just like checking blood pressure, for all older people in attendance (Figures 20.1 and 20.2).

20.5.1 Community Home-dwelling Setting

A Cochrane systematic review of 159 randomised controlled trials with a total of 79 193 participants revealed that among community-dwelling older people, the only interventions that reduced both the rate of falls* (rate ratio, RaR†) and risk of falling (risk ratio, RR‡) were exercise and home safety

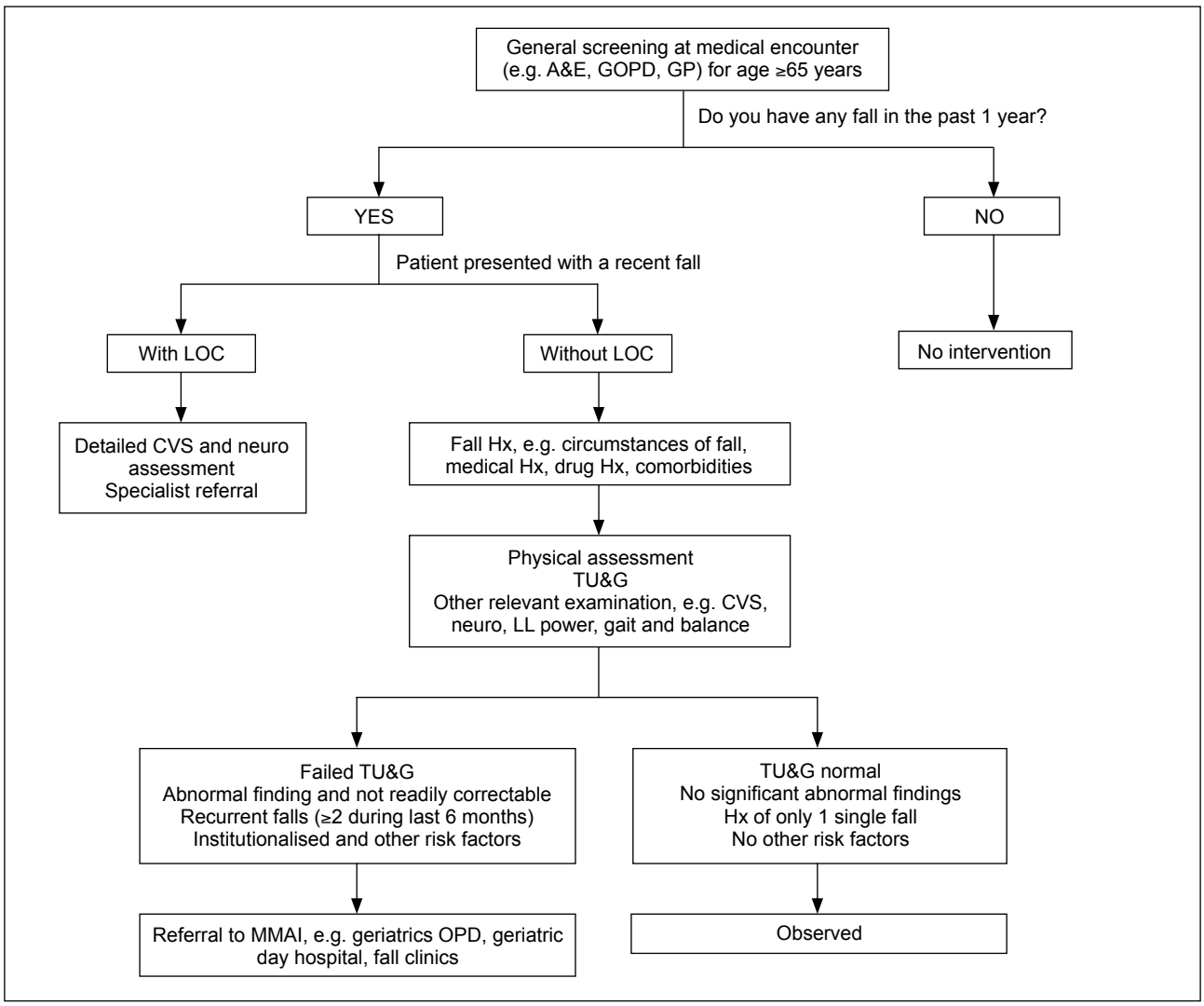

Figure 20.1 Algorithm for fall screening and subsequent assessment and management for older people
Abbreviations: A&E = accident and emergency; CVS = cardiovascular diseases; GOPD = general outpatient department; GP = general practitioner; Hx = history; LL = lower limb; LOC = loss of consciousness; MMAI = multidisciplinary, multifactorial assessment and intervention; neuro = neurological diseases; OPD = outpatient department; TU&G test = Timed Up and Go test

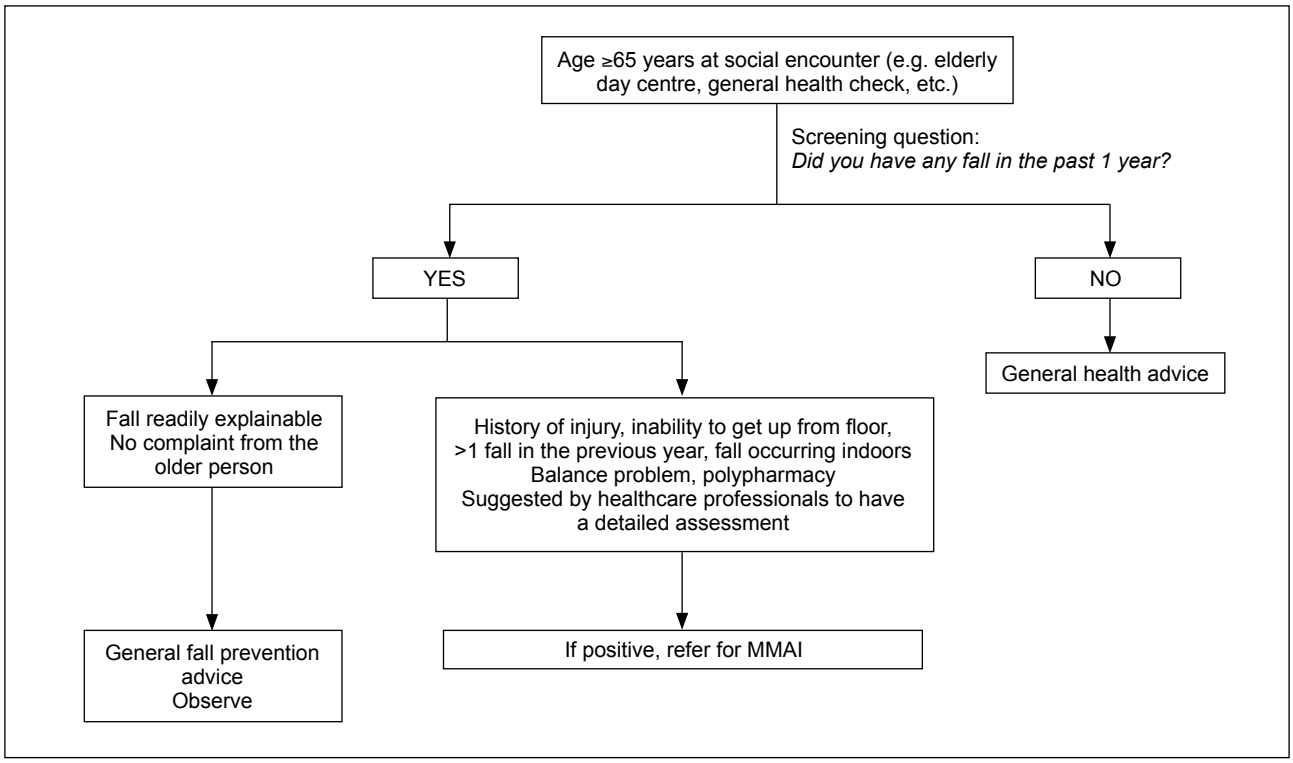

Figure 20.2 Algorithm for fall screening at social setting for older people
Abbreviation: MMAI = multidisciplinary, multifactorial assessment and intervention

modification.[3] Multifactorial interventions, which included individual risk assessment, also reduced rate of falls (RaR=0.76; 95% confidence interval [CI], 0.67-0.86) but not risk of fall.

* Fall rate=total number of falls per unit person time (e.g. falls per person-year)

† RaR=comparison of fall rates between intervention and control groups

‡ RR=comparison of number of people who fell once or more (fallers) between intervention and control groups

Both multi-component group (RaR=0.71, 95% CI=0.63-0.82; RR=0.85, 95% CI=0.76-0.96) and individually prescribed home-based multi-component exercise (RaR=0.68, 95% CI=0.58-0.80; RR=0.78, 95% CI=0.64-0.94) significantly reduce rates and risk of fall.[3] Greater benefits are seen in classes that include exercises that challenge balance and are of higher intensity, like ≥2 hours per week.[28] Examples are tai chi or Otago Exercise Programme. Otago Exercise Programme is a home-based exercise programme designed to improve strength, balance, and endurance. It is individually tailored and the difficulty increases with time. A stable and sustainable programme is established after a series of visits from an instructor and a walking component are often included.[29] It is effective in reducing risks of death and falls in community-dwelling older people.[30] The tai chi group exercise was a simplified one in the western studies with an emphasis on 'single leg standing' and 'trunk flexibility'. In Hong Kong, most people practise full forms and should be at least as effective, if not better. This can be promoted as a territory-wide health recommendation. Brisk walking alone is not recommended for high-risk individuals.[31]

Home safety assessment and modification interventions were best delivered within the setting of a multifactorial fall prevention programme, preferably by an occupational therapist (RaR=0.81, 95% CI=0.68-0.97; RR=0.88, 95% CI=0.80-0.96). These were more effective in people at increased risk of falling, like those with impaired vision or recent hospitalisation.[3]

A healthy balanced diet rich in calcium and vitamin D is recommended. Vitamin D supplementation reduces falls in patients with low vitamin D level and should also be considered for older adults who are susceptible to deficiency. A dose at least 800 IU of vitamin D per day is recommended.[3,16] However, intermittent administration of large doses of vitamin D (e.g. 500 000 IU annually) is not advised, as it was associated with more falls and fractures in some studies.[32,33]

Visual impairment is an independent risk factor

for falls. Cataract surgery on the first eye should be expedited (within 4 weeks) in older persons in whom surgery is indicated.[34] Use of multifocal lens may increase the risk of fall by reducing contrast sensitivity and depth perception in the lower visual field during outdoor activities and walking the stairs. Older adults participating in regular outdoor activities should wear single lens glasses. Dual chamber cardiac pacing should be considered for older patients with cardio-inhibitory carotid sinus hypersensitivity and unexplained recurrent falls.[3]

Lastly, the assessment of bone health (e.g. bone mineral density) should be undertaken in patients at risk of fragility fracture and advice on lifestyle measures should be given. Specific pharmacological treatment should also be considered if indicated.[35] The age of patient, comorbid conditions, therapeutic efficacy, side-effect profile, and cost and ease of administration are all important considerations affecting selection of treatment.

20.5.2 Community Institutionalised Setting

For older people living in institutions, vitamin D supplementation reduces rate of falls (RaR=0.63, 95% CI=0.46-0.86) but not risk of falling (RR=0.99, 95% CI=0.90-1.08) in patients with low vitamin D level.[36] A Cochrane review in 2012 found that MMAI was associated with a possible but not conclusive benefit in decreasing the rate of falls (RaR=0.78, 95% CI=0.59-1.04) and risks of falling (RR=0.89, 95% CI=0.77-1.02).[36] Staff education programmes including the recognition of at-risk inmates, environmental safety, and exercise programmes are important components of MMAI implemented in institutions. Community Geriatric Assessment Service teams play an instrumental role in the serviced residential care homes in Hong Kong.

The use of chair or bed alarms to alert caregivers to the movement of high-risk fallers can be tried but there is insufficient evidence for or against their use. For those recurrent fallers who fail to show improvement with education and training, interventions should be targeted at prevention of injury rather than fall. The use of hip protectors has been found to marginally reduce the rate of hip fractures (RR=0.81, 95% CI=0.66-0.99) in nursing or residential care settings.[37] Adverse effects were minimal but adherence, particularly in the long term, was poor. Hip protectors should be considered in high-risk elderly patients who can tolerate wearing them. Compliance should be monitored and their use could be improved by engaging patients and staff in the selection and implementation process.[38]

The use of restrainers to prevent falls should be discouraged because of the major adverse effects like injuries in the older people. No evidence was found to support the use of restrainers to prevent falls in major reviews while withdrawal of restrainers resulted in less injury. Stringent criteria should be applied to the use of restrainers, taking note that the restriction of the elders' mobility in order to prevent falls is a major sacrifice of their independence. It directly affects their ability to ambulate safely by reducing muscle mass, strength, joint flexibility, and vasomotor stability. Thorough consideration of non-restrictive care alternatives like increase in surveillance should always be sought and a systematic documentation of restrainer use should be enforced and audited.[38]

Staff education in long-stay setting plays an important role in fall prevention. Staff should develop high awareness about the problem, understand the basic principles of fall prevention and management, learn the skills to depict reversible risk factors and identify potential fallers, and cultivate the proper attitude of restrainer use. Structured staff education programme is a crucial component of fall prevention in these settings.[39] Although exercise as a single intervention itself failed to reduce falls in long-term care facilities, it was included in most successful multifactorial programmes. Regular exercise programmes, supervised by staff, should be offered to residents who are willing and capable of participating.[39]

Treatment of osteoporosis is proven to be effective for preventing fractures in institutionalised older people.[40] Adequate calcium intake (elemental calcium, 1200 mg daily) should be ensured and vitamin D supplement should be provided to patients with proven or suspected vitamin D deficiency.[40]

20.5.3 Hospital Setting

A Cochrane review in 2010 examined four multifactorial intervention trials in the hospital setting (three acute and one subacute) including 6478 patients to show a significant reduction in the rate of falls (RaR=0.69, 95% CI=0.49-0.96) and risk of fall (in three trials: RR=0.73, 95% CI=0.56-0.96; n=4824).[36] These interventions generally included a combination of furnishing adaptation, mobility aids, communication aids, protection aids, and staff and patient empowerment. The use of screening tools such as Morse Fall Scale,[41] STRATIFY,[42] and nursing precautions to detect high-fall-risk patients are logical approaches and are practised widely (Appendices 2[41] and 3[42]). However, there was no solid proof of successfully using these tools to prevent falls in hospitals. The 2013 NICE (National Institute for Health and Care Excellence) guideline for fall prevention does not recommend the routine use of these tools to predict inpatients' risk of falling in hospital. Instead, for patients at risk of falling, MMAI

was recommended.[43] Special attention should be paid to patients with mobility problems, elimination needs, and cognitive impairment. Hospital staff should avoid applying restraints to patients with delirium, and alternative means should be tried. Post-fall case evaluation may help to document service gaps or practice defects. Acute illness often overshadows the clinical picture of the patient. It may affect the comprehensive fall assessment during the acute ward stay. After the acute illness, patients with persistent high fall risks should be referred to MMAI.

In conclusion, fall prevention requires different strategies in different settings and target populations. This is achievable through assessment and implementation of interventions with proven efficacy.

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Appendix 1 Assessment tools of balance or gait[22-25]

Berg Balance Scale[22,23]
This is a validated 14-item scale which assesses the body balance by functional tasks like sit to stand, transfers, reaching forward, and picking up objects from the floor. Each item is scored on a 5-point scale (0-4); a higher score indicates better performance. A score of <40-45 predicts an increase in fall risk. This test is, however, more useful for monitoring the effectiveness of an intervention and quantification of function in clinical practice and research. A change of 8 points is indicative of a genuine change in function.

Functional Reach Test[24]
In this test, an individual who fails to reach out 6-10 inches is at an increased risk of recurrent falls.

Performance Oriented Mobility Assessment[25]
This tests the balance and gait through a number of items or everyday activities, including the ability to sit and stand from an armless chair, ability to maintain standing balance when pulled by an examiner, and ability to walk normally and manoeuvre obstacles. Patients' adaptive or abnormal responses to these manoeuvres are evaluated and the gait is observed while the patient is walking down a hallway in a usual and at a rapid pace. A reliable cut-off score for predicting falls with this test is yet to be established.

Appendix 2 Morse Fall Scale (adapted from Reference 41*)

Assessment item	Result	Score
Fall history in past 3 months	Yes	25 (0 if 'No')
More than 1 active diagnosis	Yes	15 (0 if 'No')
Aids for ambulation	Furniture	30
	Crutches, stick, frame	15
	None / bed rest / assistance needed	0
Intravenous line or saline lock	Yes	20 (0 if 'No')
Gait and transfer	Impaired (unsteady, sit-to-stand difficulty)	20
	Weak (need touch for balance)	10
	Normal / bed rest / immobile	0
Mental state	Overestimates / forgets one's limitation	15
	Good insight to one's ability	0

* Cut-off threshold ≥45: sensitivity 78%, specificity 83%; cut-off threshold ≥55: sensitivity 83%, specificity 68% with respect to the patient groups studied as mentioned in reference 41

Appendix 3 STRATIFY risk assessment tool[42*]

Assessment	Response / score
1. Did the patient present to the hospital with a fall or has he / she fallen in the ward since admission?	Yes / 1 (0 if 'No')
Do you think the patient is:	
2. agitated?	Yes / 1 (0 if 'No')
3. visually impaired to the extent that daily function is affected?	Yes / 1 (0 if 'No')
4. in need of especially frequent toileting?	Yes / 1 (0 if 'No')
5. having transfer and mobility score of 3 or 4?†	Yes / 1 (0 if 'No')

* Cut-off threshold ≥2: sensitivity 93%, specificity 88% with respect to the study population mentioned in reference 42
† Transfer score: 0 = unable, 1 = major help needed (one or two people, physical aids), 2 = minor help (verbal or physical), 3 = independent
Mobility score: 0 = immobile, 1 = independent with aid of wheelchair, 2 = walks with help of one person, 3 = independent

CHAPTER 21

Dizziness and Syncope

Chi-fai KO, Pan YEUNG

OBJECTIVE

To learn the epidemiology, causes, diagnosis, evaluation and management of dizziness, and syncope in old age.

KEY POINTS

- The causes of dizziness and syncope in an elderly person may be multifactorial.
- Categorisation of dizziness into vertigo, dysequilibrium, pre-syncope, and non-specific dizziness may narrow down the differential diagnoses.
- Unselected and prolonged use of antivertiginous drugs should be discouraged.
- It is important to review drug history when evaluating an elderly person with dizziness or syncope.
- Initial evaluation in syncopal patients yields clinical clues in over 60% of cases.
- A witness account is very important in the evaluation of syncope in an elderly person.
- Following a standard syncope diagnostic protocol will improve the diagnostic yield in syncopal patients.
- Cardiac syncope carries a poorer prognosis in terms of increased mortality and recurrence rate.

21.1 Definition and Epidemiology of Dizziness

Dizziness is a common symptom in elderly people. It can be difficult to manage because it is subjective and difficult to define and quantify. Objective diagnostic criteria are lacking. The problem tends to be recurrent and response to treatment is usually incomplete.

The term dizziness is used to describe various abnormal sensations relating to perception of the body's relationship to space. Based on symptoms, it can be categorised into four subtypes: vertigo, presyncope, dysequilibrium, and non-specific dizziness. A study of 1300 patients with dizziness showed that 68.9% were vertigo, 12.4% dysequilibrium, 1.8% presyncope, and 16.9% non-specific.[1] Subtype categorisation helps to narrow down the differential diagnosis as each subtype may point to a different set of aetiologies.

Dizziness occurs in 9.6-24% of community-dwelling elderly.[2,3] Together with syncope, it constitutes 7% of attendances at emergency departments among elders aged ≥75 years.[4] It is more common in women[1,2,5] and in advanced age.[2,6] Every eight out of 1000 individuals of all age-groups will present to their general practitioner with dizziness over a 12-month period. Of these, 60.5% will be aged >60 years, 75% will have improvement in symptoms, and 13% will require specialist referral.[6] Elders who have dizziness are more prone to falls[3] and functional impairment.[2,7] Among older adults who present to a primary care service with dizziness, up to one-third will have dizziness-related impairment that has persisted for over 6 months.[8]

In a Hong Kong study,[9] dizziness in adults contributed 4% of emergency department attendance. Among this group of patients, 38.5% were elderly with age >65 years. The majority (68.3%) presented with non-vertiginous dizziness and 31.7% presented with vertigo. Among those who presented with vertigo, 94% were diagnosed with a peripheral cause and in 3% the cause was central vertigo.

21.2 Subtypes of Dizziness

21.2.1 Vertigo

Vertigo is the sensation that either the environment or the individual is spinning or undergoing angular motions. It can be further classified into peripheral vertigo and central vertigo (Table 21.1). The former is more common than the latter.[6] Peripheral vertigo is caused by disorders of the inner ear or vestibular nerve, e.g. benign positional vertigo, Ménière's disease, and acute labyrinthitis (Table 21.2[10,11]). Central vertigo is usually caused by ischaemia of

Table 21.1 Features of peripheral and central vertigo

	Central vertigo	Peripheral vertigo
Site of pathology	Posterior circulation territory of central nervous system	Inner ear or vestibular nerve
Balance during acute phase	Unable to stand or walk without falling	Impaired balance but still able to walk
Direction of nystagmus	Nystagmus changes direction with gaze. Any direction	Nystagmus does not change direction with gaze. Horizontal, or mixed with torsional
Improves with visual fixation	No	Yes
Auditory symptoms	Rarely	Often
Focal neurologic symptoms / signs	Often	Rarely
Duration of nystagmus	Persist for weeks to months	Last for 1-2 days

Table 21.2 Differential diagnosis of peripheral vertigo[10,11]

	Pathology	Clinical feature	Treatment
Benign positional vertigo	A loosened otolith migrated to posterior semicircular canal	Short-lived vertigo, lasting less than a minute, precipitated by change of head position; confirmed by positive Hallpike manoeuvre	Canalith repositioning manoeuvre[10,11]
Ménière's disease	Endolymphatic hydrops of the inner ear structures	Episodic vertigo lasting minutes to hours, accompanied by fluctuating hearing loss and tinnitus	Low-salt diet, diuretic; surgical decompression in severe cases; antivertiginous medication in the acute phase
Acute labyrinthitis / vestibular neuronitis (if hearing not affected)	Mostly viral in origin	Abrupt onset of severe vertigo with hearing affected; usually lasts for 1-5 days, followed by 2-3 weeks of gradual improvement; positive head-thrust test	Antivertiginous medication in the acute phase, prolonged use not recommended; vestibular rehabilitation

the posterior circulation, such as vertebrobasilar insufficiency and vascular cervical dizziness, or less commonly neoplasm.

Transient ischaemic attack of the brainstem results in vertebrobasilar insufficiency, and presents with abrupt onset of vertigo lasting from 20 minutes to several hours and associated neurological features such as diplopia, dysarthria, and limb weakness / numbness. Similar features may also occur in severe degeneration of the cervical spine when the vertebral arteries are compressed by the cervical vertebrae during head turning or extension, a condition termed vascular cervical dizziness. Magnetic resonance angiography may be used to confirm the clinical impression in both conditions. Vertebrobasilar insufficiency should be treated with an antiplatelet agent and correction of vascular risk factors.

21.2.2 Dysequilibrium

Dysequilibrium is a sensation of imbalance or unsteadiness. It is always more prominent when the patient is standing or ambulating and should be considered in patients with postural dizziness as a differential diagnosis of orthostatic hypotension (OH). It is caused by any disturbance of the neurosensory structures related to postural control, such as visual, vestibulospinal, proprioceptive, somatosensory, cerebellar, or motor function. Older adults depend more on vision to maintain balance compared with young individuals, as indicated by excessive body sway in older adults when their eyes are closed (low Romberg's quotient — body sway when eyes open / body sway when eyes closed).[12] Usually more than one causative factor is involved in dysequilibrium.

Proprioceptive cervical dizziness is one example. The facet joints of the cervical spine contain proprioceptive receptors. Disturbance of these receptors during flare-up of osteoarthritis or acute muscle spasm may lead to proprioceptive dysfunction. The patient may complain of dizziness and imbalance if this dysfunction cannot be compensated for by vision. Apart from correction of visual impairment and treatment of underlying diseases, use of walking aids to enhance sensory input and postural control as well as balance training are useful treatment modalities.

21.2.3 Pre-syncope

Pre-syncope is a sensation of impending faint. It indicates a transient hypoperfusion of the cerebral cortex. The causes are similar to those of syncope and will be discussed in the later part of this chapter.

21.2.4 Non-specific Dizziness

Non-specific dizziness refers to dizziness that is vague and cannot be distinctly identified as vertigo, dysequilibrium, or pre-syncope. It is most often associated with anxiety, depression, or other

psychological disorders. Other possibilities such as electrolyte disturbances or metabolic disorders should also be considered.

Ruckenstein and Staab[13,14] use the term 'chronic subjective dizziness' to describe unexplained persistent non-vertiginous dizziness associated with:

- chronic hypersensitivity to one's own motion or the movement of objects in the environment, and / or
- exacerbation of symptoms in settings with complex visual stimuli or when performing precision visual tasks

The dizziness, which is a form of an undifferentiated somatoform disorder, may present as lightheadedness, heavy headedness, subjective imbalance not apparent to others, spinning inside the head, feeling that the floor is moving from underneath, or feeling of dissociation from one's environment.

21.3 Evaluation and Management of Dizziness

The temporal characteristic of dizziness, whether it is episodic or continuous, as well as the duration of each attack, should be ascertained. The presence of any associated symptoms or signs should also be noted. Hearing loss and tinnitus point to cochlear problems. Diplopia, dysarthria, and limb weakness suggest cerebral / brainstem problems. Palpitations indicate a cardiac origin. Identification of triggering factors will also help determine the causes of dizziness. Head and neck movement hint at cervical or carotid sinus problems. Change of posture may suggest OH. Ambulation indicates multiple neurosensory impairment. Finally, a comprehensive medication history is important when evaluating an elderly patient who complains of dizziness.

Examination should be directed by clues from the history, e.g. perform Hallpike manoeuvre to look for benign positional vertigo; otoscopic examination together with head-thrust test to examine the vestibulo-ocular reflex. If peripheral vertigo is suspected, a positive head-thrust test confirms the vestibular origin of vertigo.[15] In general, checking for visual impairment, OH, and irregular rhythm should be sought. Attention should be paid to the cardiovascular, neurologic, and musculoskeletal system. Video clips on how to perform the Hallpike manoeuvre are available online (https://www.youtube.com/watch?v=LxD-lgqix-s).

When the cause is unclear, investigations for dizziness should include complete blood count, electrolytes, lipid levels, and thyroid function test. Autoimmune markers and syphilis serology should be considered in a patient with unexplained vertigo and fluctuating bilateral hearing loss. Magnetic resonance imaging / angiography should be performed in a patient suspected to have a central disorder, whereas computed tomographic scanning provides a better imaging for bony labyrinth.

Benign positional vertigo should be treated with the canalith repositioning manoeuvre. Video clips on how to perform this manoeuvre are available online (https://www.youtube.com/watch?v=LxD-lgqix-s). Chronic subjective dizziness and psychogenic dizziness should be treated with a selective serotonin reuptake inhibitor.[16] Vestibular rehabilitation is useful in the patient with persistent peripheral vertigo, as it may enhance the central compensation for lesions in the vestibular system.[17] Table 21.3 shows commonly used antivertiginous medications.[18] They are sometimes given liberally to patients who complain of dizziness without first trying to diagnose the underlying cause. This practice should be discouraged. Nonetheless a patient with persistent dizziness and no identifiable cause should be referred to a specialist for further work-up.

21.4 Definition and Epidemiology of Syncope

Transient loss of consciousness (TLOC) is a term used to describe disorders characterised by transient, self-limited, and non-traumatic loss of consciousness. It comprises two syndrome complexes: syncope and non-syncopal TLOC.[19] Syncope refers to TLOC caused by global cerebral hypoperfusion. TLOC as a result of all other mechanisms is called non-syncopal TLOC. Syncope is characterised by its rapid onset, brief duration, loss of postural tone, and complete recovery with no neurologic sequelae. Patients

Table 21.3 Antivertiginous medications (please refer to the Beers Criteria[18] for their use in elders)

Drug class	Action	Medication
Antihistamines	Histamine (H_1) antagonist H_3 antagonist	Dimenhydrinate, promethazine, cyclizine, cinnarizine Betahistine
Phenothiazine	Dopamine (D_2) receptor antagonist — blocking chemoreceptor trigger zone	Prochlorperazine, chlorpromazine
Benzodiazepines	$GABA_A$ agonist	Diazepam, clonazepam
Anticholinergics	Muscarinic antagonist	Hyoscine (scopolamine)

usually recover within a few minutes, unless they are maintained in an erect or sitting position. Any presentation that deviates from these clinical features should alert the clinician to the possibility of non-syncopal TLOC. Global cerebral hypoperfusion for >5 seconds is sufficient to cause syncope and more prolonged hypoperfusion of >15 seconds may result in generalised tonic spasm, myoclonic jerks, and urinary incontinence. Pre-syncope refers to a condition in which patients feel as though syncope is imminent. Symptoms associated with pre-syncope may be relatively non-specific (e.g. dizziness), and tend to overlap with those associated with the premonitory phase of true syncope. The symptom of syncope often coexists with dizziness and falls. A study of patients attending a syncope clinic revealed that more than half of them had overlap of these symptoms.[20]

The prevalence of syncope varies with the setting of clinical studies. In a French study,[21] TLOC constituted 1.21% of attendance at emergency departments, of which 63% were syncope and 12% non-syncopal TLOC. The rest were unexplained. Among this group of emergency attendees, 63% required admission. In the Framingham Heart Study,[22] which followed 7814 community dwelling individuals for a mean of 17 years, the incidence rate of first TLOC was 6.2 per 1000 person-years, with a recurrence rate of 21.6%. The incidence increases in individuals of older age (>70 years) and in those with a history of cardiovascular disease. It has been reported that only 56% of individuals with a TLOC will seek medical advice.

TLOC is associated with an increased risk of death by 31% over a period of 17 years. This risk is particularly significant in patients with cardiac syncope. Reflex syncope usually carries a benign prognosis, reflecting the better prognosis of the underlying aetiologies. Cardiac syncope also carries a significantly higher risk of recurrence.[22]

21.5 Classification and Differential Diagnosis of Syncope

Based on the pathophysiology, the causes of syncope can be classified as follows: reflex, orthostatic, and cardiac (Table 21.4). Sympathetic responses to environmental challenges play an important role in the pathophysiology of syncope. Reflex syncope is caused by an excessive sympathetic response to physiological stress, e.g. venous pooling. Signals sent via brainstem nuclei trigger the Bezold-Jarisch reflex[23,24] (Figure 21.1) resulting in sudden sympathetic withdrawal (vasodilation) and vagal stimulation (bradycardia). Patients with reflex syncope will have two groups of prodromal symptoms — autonomic activation and hypoperfusion.[25] Prodromal symptoms of autonomic activation include nausea, abdominal discomfort, sweating, or feeling cold. Orthostatic syncope results from an inadequate sympathetic response to fluid shift with insufficient venous return, such as venous pooling in an upright posture, post-exercise, or post-meal. Prodromal symptoms of hypotensive syncope result from retinal and cerebral hypoperfusion, and include lightheadedness, blurred or darkened vision, and shoulder pain.[25] Causes of non-syncopal TLOC are shown in Table 21.5.

The distribution of causes reported in the Framingham Heart Study were as follows: cardiac 9.5%, neurologic 9%, neurally mediated 28.7%, OH 9.4%, medication induced 6.8%, and unknown in 36.6%.[22] In elderly people, the cause for syncope is frequently multifactorial. For example, OH often coexists with vasovagal syncope[20] and the role of medication is much more significant when compared with younger adults.

Table 21.4 Causes of syncope according to pathophysiology

Pathophysiology	Example
Reflex (neurally mediated) syncope	Vasovagal syncope Situational syncope (cough / swallow / micturition) Carotid sinus syndrome
Orthostatic syncope	Hypovolaemia Increased venous pooling in the lower body • pregnancy, varicose veins • standing still after rigorous exercise (post-exercise) Failure to activate the sympathetic activity • autonomic failure • postprandial hypotension Inadequate vasoconstrictor response to sympathetic stimulation • on vasodilators, such as alpha-blockers, calcium channel blockers
Cardiac arrhythmia	Sinus node dysfunction (including bradycardia / tachycardia syndrome) A-V conduction defect Paroxysmal supraventricular or ventricular tachycardia
Structural cardiac or cardiopulmonary disease	Outflow obstruction (aortic stenosis, hypertrophic cardiomyopathy, left atrial myxoma) Acute myocardial infarction / ischaemia

Ageing has an effect on the prevalence of different causes of syncope. In young adults (age <40 years), more than 90% of syncope is caused by reflex syncope whereas in adults aged >60 years, reflex syncope causes barely >50% of cases. The prevalence of orthostatic syncope and cardiac syncope increases significantly compared with the young.[26]

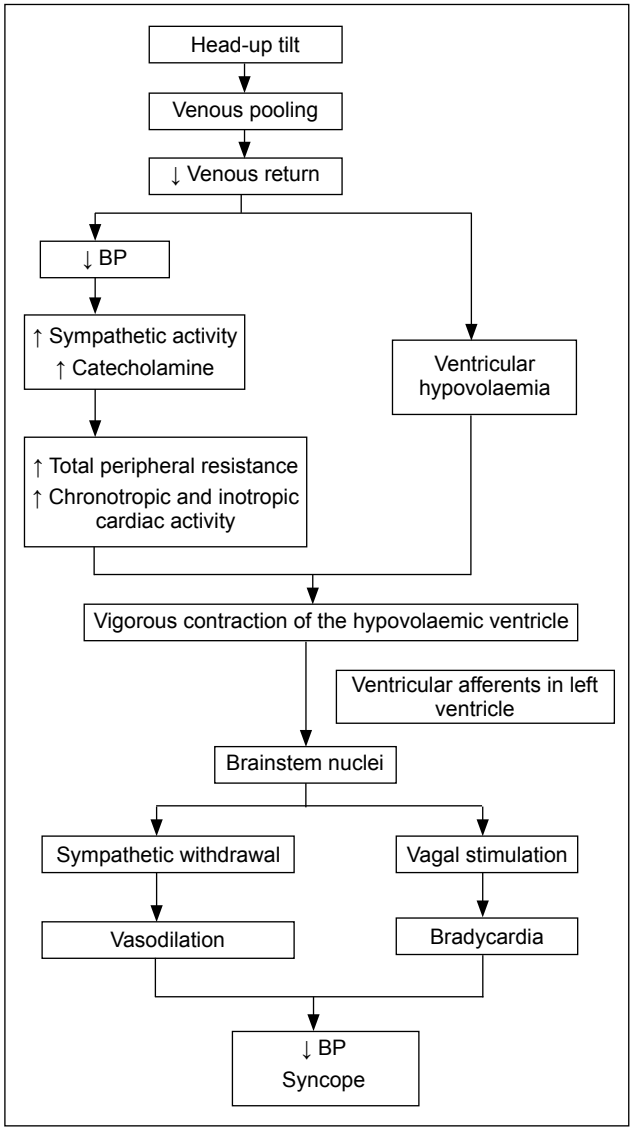

Figure 21.1 Bezold-Jarisch reflex
Abbreviation: BP = blood pressure

21.6 Evaluation of Syncope

Assessment of syncope aims to identify the underlying cause and estimate the short-term prognosis. Since cardiac syncope carries a significantly higher mortality and recurrence rate, it is important to differentiate cardiac from non-cardiac syncope.

An initial assessment should include history taking, physical examination, measurement of supine and erect blood pressure, as well as a standard electrocardiogram (ECG). This initial assessment will achieve a certain or highly likely diagnosis in up to 63% of patients with 88% accuracy.[27]

History taking is of utmost importance in diagnosing syncope. A witness account should be sought to clarify the actual event as some elders may have retrograde amnesia about the syncopal episode.[28] The initial evaluation should clarify any precipitating or predisposing events; any prodrome; details of the syncopal episode; any post-event abnormalities; medication history; medical history (especially of recurrent syncope, cardiac, and neurologic problems); and family history of sudden death or congenital arrhythmia (Table 21.6).

Seizure may be suggested by presence of aura, a prolonged loss of consciousness longer than 5 minutes, presence of twitching at the outset, incontinence, tongue biting, as well as a postictal state. Brainstem transient ischaemic attacks should be suspected if vertigo, dysarthria, diplopia and limb weakness / numbness are present. Syncope preceded by neck turning should point to carotid sinus syndrome (CSS) or vascular cervical dizziness whereas arm movement may hint at subclavian steal syndrome. Vasovagal syncope usually occurs in crowded and warm places after prolonged standing, with a prodrome of autonomic activation. Syncope preceded by a specific event such as cough, micturition or swallowing suggests situational syncope. Syncope occurring soon after standing up suggests OH whereas that which occurs after meals indicates postprandial hypotension (PPH).

Physical examination should be guided by the

Table 21.5 Causes of non-syncopal transient loss of consciousness

Pathophysiology	Example
Disorders with impairment or loss of consciousness	Metabolic disorders (e.g. hypoglycaemia, hypoxia, hyperventilation) Epilepsy Intoxication Vertebrobasilar transient ischaemic attack Cerebrovascular steal syndrome
Disorders without impairment of consciousness	Drop attacks Psychogenic attack (somatisation disorders) Transient ischaemic attack of carotid origin

history and in general should include pulse rhythm, supine and erect blood pressure, cardiovascular system, and neurologic system. Differences in blood pressure or pulse between the two arms should be sought if subclavian steal is suspected. ECG should be performed in all instances. Clues to diagnosis (refer to Table 21.6) should direct further work-up and treatment should be started accordingly.

For patients who do not have a certain diagnosis, further investigations should follow a standard diagnostic protocol (Figure 21.2[29]). Patients should be worked up along the line of reflex or orthostatic syncope if they have no clinical features suggestive of cardiac syncope as mentioned in Table 21.6. Presence of any clinical features suggestive of cardiac syncope should prompt the clinician to work along the line of cardiac syncope.[30,31] With adherence to this syncope diagnostic protocol, the diagnostic yield of syncope work-up can be improved to 78%.[32] The range of

Table 21.6 Clinical features of different syncopal subtypes

Syncopal subtype	Clinical feature
Reflex syncope	Prodromal symptoms of autonomic activation After prolonged standing in hot, crowded environment Associated with emotional stress, pain, fear Happens shortly after cough / micturition / defecation Associated with head-turning or pressure over carotid sinus Absence of underlying cardiac disease
Orthostatic syncope	Happens during or after a meal Happens after exertion Shortly after assumption of upright posture After prolonged standing Recent change of medication regimen
Cardiac syncope	Preceded by palpitations Happens during exertion or when supine Presence of underlying cardiac disease Family history of sudden death

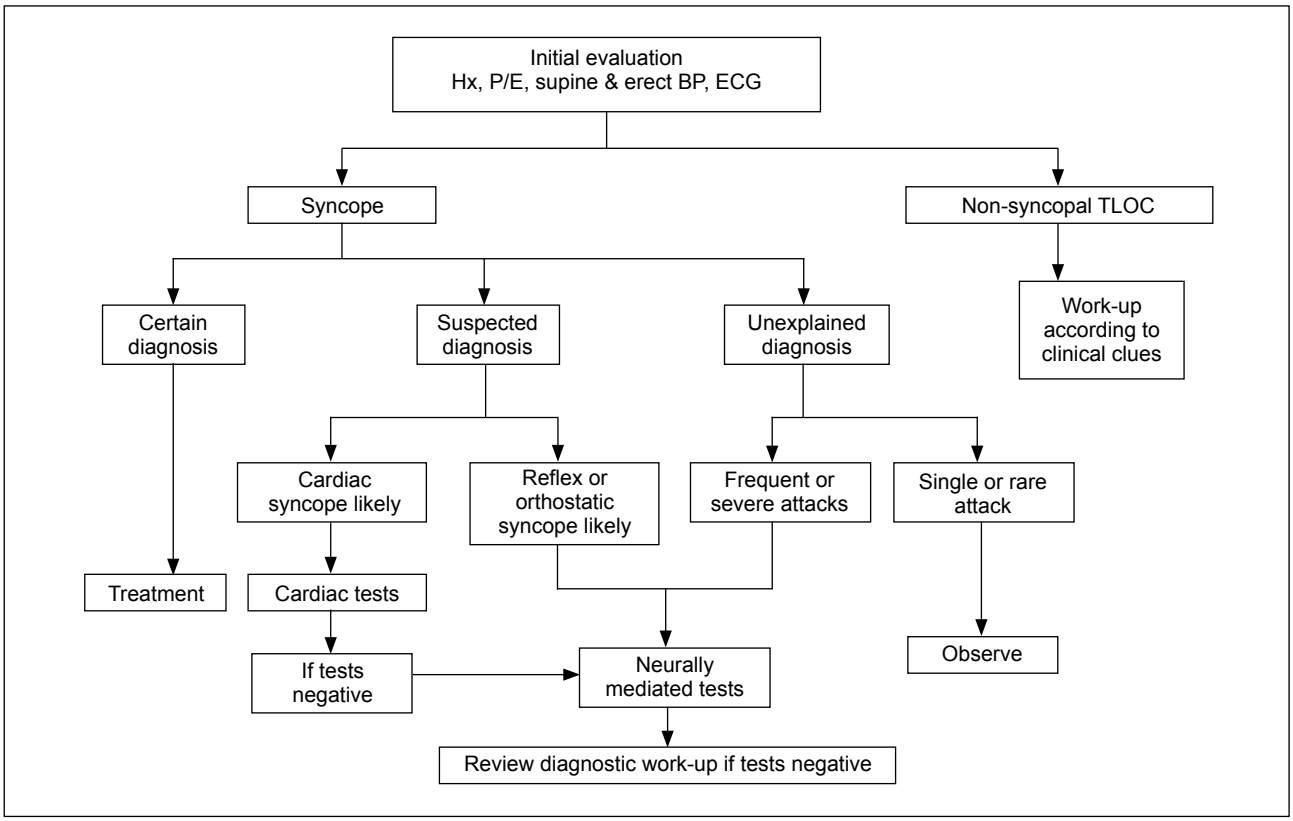

Figure 21.2 Work-up of TLOC (adapted from O'Dwyer and Kenny[29])
Abbreviations: BP = blood pressure; ECG = electrocardiogram; Hx = history; P/E = physical examination; TLOC = transient loss of consciousness

diagnostic tests available is illustrated in Table 21.7. They should be applied according to clinical clues.

An atypical presentation is a characteristic feature among elderly patients. There is no exception in evaluating syncope. Among older adults with tilt-positive vasovagal syncope, they are less likely to report total LOC or near LOC and more likely to present with unexplained falls. The provocative factors of prolonged standing and a hot environment are also less common in older adults.[33] The diagnosis of the cause of syncope is possible on the basis of the history alone in around a quarter of younger adults, but only possible in 5% of older adults.[31] As a result, elderly patients rely more on the diagnostic tests to confirm or exclude a diagnosis.

Several risk prediction tools have been developed to predict the prognosis of syncopal patients. They have all identified similar risk factors for worse prognosis over a 1-2 year period. The risk factors in common are abnormal ECG, older age, and presence of underlying heart disease.[19] In a meta-analysis, however, these risk prediction tools were no better than clinical judgement in predicting short-term serious outcome.[34]

Hospitalisation after an episode of syncope is indicated for the following situations[35]:

- suspected or known underlying structural heart disease
- ECG abnormalities suggesting arrhythmic syncope (high grade A-V block, long pause, pre-excitation syndrome, increased QT interval, Brugada syndrome)
- syncope occurring during exercise
- syncope causing a motor vehicle accident or severe injury
- family history of premature sudden death
- associated with acute myocardial ischaemia, acute aortic dissection, or signs of congestive heart failure

21.7 Use of Common Syncope Diagnostic Tools

21.7.1 Head-up Tilt Test

The head-up tilt test (HUTT) was first reported in 1986 to be useful in investigating unexplained syncope.[36] It is now the gold standard in diagnosing vasovagal syncope. HUTT monitors the haemodynamic response to venous pooling during a head-up tilt. It incorporates the use of an electric tilt table and a non-invasive beat-to-beat continuous blood pressure monitoring device. The initial standard protocol proposed a 60° passive tilt for 45 minutes,[37] often referred to as the 'Westminster protocol'. The mean time to syncope during passive tilt has been found to be 24 minutes with a standard deviation of 10 minutes.[37] The time of 45 minutes was chosen because it is the mean time to syncope plus 2 standard deviations. In year 2000, a shortened HUTT referred to as 'Italian protocol' was proposed.[38] This requires a 60° tilt for 35 minutes. After the initial 20 minutes, a sublingual spray of 400 μg of glyceryl trinitrate is given as provocation. The authors have adopted the use of this Italian protocol in HUTT and a proforma currently in use in the authors' centre is illustrated in Figure 21.3.[39] The Italian protocol has high specificity, up to 97% in older adults and 94% in younger subjects.[39]

The HUTT is considered positive if there is symptom reproduction associated with hypotension and / or bradycardia characteristic of a vasovagal response. The test is false positive if there is haemodynamic change without syncope. Psychogenic pseudosyncope should be considered if there is apparent loss of consciousness without haemodynamic change. The test should be interrupted if completed after 35 minutes, when syncope / presyncope occurs or when there is significant hypotension (decreased systolic blood pressure [SBP] >50% from baseline or SBP <90) persisting for ≥3 minutes in patients known to have ischaemic heart disease or cerebrovascular disease. HUTT is contraindicated in patients with severe left ventricular outflow obstruction, severe mitral stenosis, critical proximal coronary artery disease, or critical cerebrovascular disease.[40]

Apart from the indications mentioned in the diagnostic protocol (Figure 21.2[29]), HUTT is also indicated in elderly people with unexplained persistent dizziness and unexplained recurrent falls. HUTT is also useful in those suspected to have OH that has not been documented at the bedside. HUTT

Table 21.7 Common tests for diagnosis of syncope

Cardiac test	Neurally mediated test
24-Hour ambulatory electrocardiogram monitor (Holter)	Head-up tilt test
Echocardiography	Carotid sinus massage
Exercise stress test (Treadmill)	24-Hour ambulatory blood pressure monitor
Loop recorder	Autonomic function tests
Electrophysiologic study	

GTN-HUT proforma Syncope clinic Geriatric day hospital Princess Margaret Hospital	(Patient's gum label)

- Fasting is not required and usual medications should be continued
- Supine for 10 minutes; tonometry monitor and ECG recorder should be connected
- Tilt upright to 60° for 20 minutes; continuous BP and P observed
- If syncope / presyncope does not occur, give sublingual GTN spray (400 µg)
- Continue BP and P observation at the same angle for another 15 minutes
- If syncope / presyncope occur, the patient should be returned to supine position and test interrupted

Time (min)	BP	P/min	Time (min)	BP	P/min
Supine			19		
1			20		
2			Give sublingual GTN spray, keep patient at upright 60°		
3					
4			21		
5			22		
6			23		
7			24		
8			25		
9			26		
10			27		
11			28		
12			29		
13			30		
14			31		
15			32		
16			33		
17			34		
18			35		

Reproduction of syncope / presyncope: YES / NO
If YES, what are the BP and P prior to / during the event? BP _____ P_____
Is the test positive? YES / NO

Name of physician: _____ Signature: _____ Date: _____

Figure 21.3 GTN-HUT proforma[39]
Abbreviations: BP = blood pressure; ECG = electrocardiogram; GTN = glyceryl trinitrate; P = pulse

is more sensitive in identification of OH[41] and is more able to identify delayed OH. Patients who present with features typical of vasovagal syncope do not need a HUTT to confirm the diagnosis.

21.7.2 Continuous Ambulatory ECG Monitoring

In patients suspected to have cardiac syncope, 24-hour ambulatory ECG monitoring (Holter study) is a useful diagnostic tool, but the authors have observed it is often unselectively used in patients who present with TLOC. In well-selected cases, such as those patients who present with features suggestive of cardiac syncope (Table 21.6), Holter study may confirm the arrhythmia (correlation between symptoms and arrhythmia) or exclude a cardiac cause (reported syncope / presyncope without significant arrhythmia) in approximately 20% of patients.[42,43]

In patients suspected of cardiac syncope but with a non-diagnostic Holter result, repeating the Holter study within a 1-year period will not increase the diagnostic yield.[42] However, increasing the

monitoring period for up to a month by the use of an external loop recorder will increase the diagnostic yield, either confirming or excluding arrhythmia as the cause of syncope, to around 50%.[43]

21.8 Common Subtypes of Syncope

21.8.1 OH

OH, or postural hypotension, is defined as a reduction of SBP by at least 20 mm Hg or of diastolic blood pressure by at least 10 mm Hg within 3 minutes of standing or head-up tilt to at least 60° on a tilt table. If the drop occurs after 3 minutes, it is a variant called delayed OH.[44] Usually, it is more prominent in the early morning and after meals. It may be symptomatic or asymptomatic and is a significant causative factor for dizziness, falls, and syncope. Orthostatic blood pressure measurement should be taken after lying supine for 5 minutes and then 1 and 3 minutes after standing. It is good practice to mention whether the patient is symptomatic when OH is documented. Repeated morning measurements are recommended since OH is not always reproducible in older adults. OH is associated with increased all-cause mortality and coronary events.[45-47] It is common among elderly people, being found in 30% of community-dwelling elderly people aged ≥70 years.[48] Its prevalence increases with age, institutionalisation, and hypertension.[44,49]

Causes of OH include medication (e.g. antihypertensives, vasodilators, diuretics, anti-parkinsonian drugs), hypovolaemia (e.g. acute blood loss, severe dehydration, adrenal insufficiency), and prolonged immobility with deconditioning and autonomic insufficiency (e.g. diabetes, multiple system atrophy, pure autonomic failure). An association between OH and vitamin D deficiency has also been reported.[50]

Interventions for OH include:

- correcting any reversible factors such as medication or hypovolaemia
- teaching the patient to rise slowly from a bed or chair
- exercise involving dorsiflexion of the ankles before standing
- exercise programme to improve conditioning and teach physical counterpressure manoeuvres (PCM) [Table 21.8[51]]
- increasing salt and caffeine (two cups of coffee each morning) consumption (caffeine is an adenosine antagonist that inhibits adenosine-induced vasodilatation)
- wearing elastic stockings over the lower limbs from calf to thigh or abdominal binders
- elevating the head of the patient's bed to 5°-20° (although a recent study did not confirm its effectiveness[52])
- pharmacological treatment if the above measures fail:
  - Fludrocortisone 0.1-1 mg daily (start with 0.1 mg, titrate upward at 0.1 mg increments weekly or biweekly until mild pedal oedema develops or maximum dose of 1 mg reached)
  - Midodrine[53-55] 2.5 mg twice daily to 10 mg 3 times daily (peripheral alpha 1–adrenergic receptor agonist causing venous and arterial constriction; to be avoided in patients with coronary or peripheral arterial disease, heart failure, urinary retention, thyrotoxicosis, and acute renal failure)
  - Droxidopa[56] 100-600 mg 3 times daily (noradrenaline precursor)

21.8.2 PPH

Ingestion of food causes splanchnic blood pooling that results in a compensatory increase in sympathetic nervous system activity and plasma renin activity. Loss or blunting of this compensatory response may lead to PPH. Gut vasoactive hormones may also play a role.

PPH is defined as a decrease in SBP by at least 20 mm Hg within 2 hours of the start of a meal or a decrease in the absolute level of SBP after a meal to <90 mm Hg when the SBP before a meal is >100 mm Hg.[57] It occurs in 24-43% of nursing home residents and is more commonly seen in patients with autonomic failure.[57-59] Apart from syncope, it may present as dizziness, weakness, or angina after meals. PPH is often unrecognised. It has been reported that up to 50% of unexplained syncope in elderly people is due to PPH.[60] PPH is associated with increased incidence of falls, syncope, new coronary events, new stroke, and all-cause mortality.[61,62] Nutrient composition and temperature of meals may affect the magnitude of the hypotension. Carbohydrates, more specifically glucose, especially when served warm have been found to play a significant role.[63] Ambulatory 24-hour blood pressure monitoring may be helpful

Table 21.8 Physical counterpressure manoeuvres (PCM)[51]

Type of PCM	Detail of manoeuvre
Leg crossing	Crossing of legs combined with tensing of legs, abdominal, and buttock muscles
Handgrip	Maximal voluntary contraction of dominant hand on any available object
Arm tensing	Contraction of the 2 arms by gripping one hand with the other and concomitant abduction of both arms

Table 21.9 Interventions for postprandial hypotension

Intervention	Description
Maintaining a patient's intravascular volume	• Increase salt intake • Increase fluid intake • Caffeine 250 mg (2 cups of coffee) each morning or before meals
Adjusting the size and composition of a patient's meals	• Frequent small meals • Limit carbohydrate content
Assumption of a recumbent position after meals if symptoms are severe	-
Discontinuing potentially offending medications such as diuretics, antihypertensives, or vasodilators	-

Table 21.10 Drug treatment in postprandial hypotension[64]

Drug	Dosage	Mechanism of action
Caffeine	250 mg every morning or before meals	Adenosine antagonist that inhibits adenosine-induced vasodilatation
Alpha-glucosidase inhibitor — acarbose	100 mg before meals	Reduces complex carbohydrate breakdown, delays gut glucose absorption
Droxidopa	1 g 3 hours before meals	Prodrug that will be converted to noradrenaline in the nervous system
Octreotide	50 µg subcutaneous before meals	Inhibits vasodilation of the splanchnic vasculature by inhibiting vasoactive peptides
Guar gum	4-9 g before meals	Bulking agent, delays gastric emptying and glucose absorption in the small intestine

in detecting PPH in suspected cases. Interventions for PPH are shown in Table 21.9. Pharmacological treatments are listed in Table 21.10.[64]

21.8.3 Vasovagal Syncope

Vasovagal syncope is diagnosed when precipitating events such as fear, severe pain, emotional distress, instrumentation, or prolonged standing are associated with typical prodromal symptoms.[19] No further investigation is necessary if the presentation is typical. For doubtful cases, the HUTT may be employed to confirm the diagnosis. The haemodynamic response to the test is effected through the Bezold-Jarisch reflex (Figure 21.1). The test is considered positive if syncopal symptoms are reproduced together with hypotension and / or bradycardia.[40]

Patients with recurrent vasovagal syncope should be advised to avoid predisposing situations and to lie down if the prodrome occurs. Chronic vasodilator therapy should be tailed down. Therapies that have been shown by randomised controlled trials to be effective in preventing vasovagal syncope include PCM and midodrine.[51,54,65-67] Details of PCM are shown in Table 21.8.[51]

Various other treatment modalities have been suggested to prevent recurrence of vasovagal syncope but their effectiveness is unconfirmed. They include:

- increasing fluid and salt intake;
- tilt-training;
- prescription of medications such as beta-blockers (negative inotropic), disopyramide (negative inotropic and anticholinergic), hyoscine (anticholinergic), paroxetine / fluoxetine (selective serotonin reuptake inhibitor), etc.; and
- insertion of a permanent pacemaker to prevent bradycardia

21.8.4 CSS

Carotid sinus hypersensitivity (CSH) is present if carotid sinus massage (CSM) produces asystole exceeding 3 seconds (cardioinhibitory response), if there is a decrease in SBP that exceeds 50 mm Hg (vasodepressor response), or a combination of the two (mixed response). If a spontaneous syncopal response is induced by CSH, it is termed CSS.

CSH was reported to be common (25-45%) in patients who present with dizziness, fall, or syncope.[20,68] Thus CSM is recommended in all syncopal patients aged >40 years.[19] Later findings have shown CSH to be present in 39% of asymptomatic people aged >65 years,[69] and pacing of cardioinhibitory CSH in patients with recurrent unexplained falls or single syncope had no significant effect on the number of subsequent events.[70,71] Thus it is thought that CSH may be an age-related physical finding and coincidental with syncope.

CSS should be suspected if situations such as shaving, a tight collar, or sudden turning of the head precipitate an attack. It should be confirmed by a positive CSM. For CSS with a cardioinhibitory response, pacemaker implantation may prevent recurrence. Midodrine has been reported to be useful in CCS with a vasodepressor response,[72] but its effectiveness needs to be confirmed.

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CHAPTER 22

Urinary Incontinence

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OBJECTIVE

To learn the prevalence and impact of urinary incontinence (UI) in elderly people, the different types of UI and their presentation, the reversible and permanent causes, as well as the diagnosis, assessment, and management of UI in old age.

KEY POINTS

- A thorough understanding of the causes and management of UI in older people will help to improve their quality of life.
- UI is a symptom of disease in old age and should be appropriately investigated.
- UI is more common in older people, especially those in residential care.
- Normal physiological changes of ageing will affect function of the urinary bladder but itself will not result in incontinence in old age.
- Reversible causes of UI are common in older people and are amenable to investigation and treatment.
- Most patients who develop UI can be managed in a primary care setting with a thorough history taking, physical examination, and simple investigations.
- Patient education is an important aspect of the management of UI in older people.
- General measures to manage diet, fluid intake, and bowel habits as well as use of toilet aids will be helpful to most patients who are urinary incontinent.
- Pelvic floor exercises are highly effective in stress incontinence and may also be beneficial in urge incontinence.
- Patients who have urge incontinence may benefit from behavioural training such as bladder retraining.
- Because of the high prevalence of multi-morbidities and polypharmacy in frail older persons, the occurrence of UI in this population will usually be the result of an interplay of different factors. Intervention should then be multifaceted after a comprehensive geriatric assessment.

21.1 Introduction

Urinary incontinence (UI) is one of the 'I's in geriatrics (Immobility, Instability, Intellectual impairment, Incontinence, Iatrogenesis). UI is a common symptom among older people and is one of the main causes of institutionalisation. UI also affects their quality of life and sometimes hinders social interaction. The underlying causes are not well understood and attributed by many to the phenomenon of ageing. Nonetheless, UI in older people often arises from conditions that can be tackled by proper assessment, investigation, and management.

22.2 Definition and Terminology

The International Continence Society (ICS) has standardised the definition of UI and related conditions (Table 22.1).[1] In each specific circumstance, UI should be further described by specifying relevant factors such as type, frequency, severity, precipitating factors, social impact, effect on hygiene and quality of life, the measures used to contain the leakage and whether or not the individual seeks or desires help.

The ICS no longer recommends the term "overflow incontinence" as this term is considered confusing and lacks a convincing definition. If used, a precise definition and any associated pathophysiology should be stated. The term "chronic retention" implies a significant post-void residual (PVR) urine. A literature review does not show an evidence-based maximum PVR that is considered normal. The amount of residual urine that precludes treatment by various therapies has also not been determined. The US Agency for Health Care Policy and Research (now reauthorised as Agency for Healthcare Research) guidelines (based on panel expert opinion) state

Table 22.1 International Continence Society definitions of selected lower urinary tract disorders relating to urinary incontinence[1]

Terminology	Description
Urinary incontinence	The complaint of any involuntary leakage of urine
Stress urinary incontinence	The complaint of involuntary leakage on effort or exertion, or on sneezing or coughing
Urge urinary incontinence	The complaint of involuntary leakage accompanied by or immediately preceded by urgency
Mixed urinary incontinence	The complaint of involuntary leakage associated with urgency and also with exertion, effort, sneezing, or coughing
Chronic retention of urine	A non-painful bladder that remains palpable or percussible after voiding. Such patients may be incontinent
Nocturnal enuresis	The complaint of loss of urine occurring during sleep
Continuous urinary incontinence	The complaint of continuous leakage

that, for both sexes, a PVR of <50 ml is considered adequate bladder emptying and >200 ml is considered inadequate.[2] Both the American and the European Urology Association guidelines suggest a threshold of 300 ml to identify male patients at risk of an unfavourable outcome following treatment for benign prostatic obstruction.[3,4]

22.3 Epidemiology

The prevalence of UI in older people has been the subject of research in many western countries.[5] Studies have shown that the prevalence increases with age. The varying prevalence in different populations in different studies reflects the different defining inclusion criteria for investigations, different methodologies for data collection, as well as a high tendency for underreporting. In general, the estimated prevalence of UI in middle-age and older women in the general population appears to be in the range of 30-60% when most inclusion definition of UI ('ever', 'any', or 'at least once in the past 12 months') are used; while the prevalence of daily UI ranges from 5% to 15%, rising to over 15% in women aged >70 years who are institutionalised. Independent risk factors for UI in females include pregnancy, labour and vaginal delivery, impaired physical function, cognitive impairment, and diabetes.

The epidemiology of UI in men has not been investigated to the same extent as in females, but it appears the prevalence is less, about half that in women. There seems to be a more steady increase in prevalence with age for men compared with women. Established risk factors that predispose men to UI include increasing age, presence of lower urinary tract symptoms, urinary tract infection, functional and cognitive impairment, neurological disorders, and prostatectomy.

The UK Department of Health[6] has suggested the following figures on UI prevalence for easy reference:
For people living at home:

- between 1 in 20 and 1 in 14 women aged 15-44 years
- between 1 in 13 and 1 in 7 women aged 45-64 years
- between 1 in 10 and 1 in 5 women aged ≥65 years
- over 1 in 33 men aged 15-64 years
- between 1 in 14 and 1 in 10 men aged ≥65 years

For people (both sexes) living in institutions:

- 1 in 3 in residential homes
- almost 2 in every 3 in nursing homes
- 1 in 2 to 2 in 3 in wards for elderly patients and elderly mental infirmary

In Hong Kong, prevalence studies have been conducted in various environments and are summarised in Table 22.2.[7-13]

22.4 Physiological Changes Associated with Ageing

Normal micturition involves the ability to store and to void urine in a suitable place and at a convenient time, and constitutes a cycle of events. Continence is maintained so long as the pressure within the bladder is lower than urethral resistance. Urine will escape when this balance is reversed.

The bladder normally accommodates about 600 ml of urine and is capable of emptying itself completely, but it is capable of containing 2 litres and more. As the bladder fills up, its muscular wall accommodates the increase in volume without any significant rise in bladder pressure. As the normal bladder fills up, an awareness of distension reaches consciousness at some point beyond 250 ml.

The detrusor muscle forms the smooth muscle of the fundus of the bladder and comprises a meshwork of interlacing bundles. The trigone provides a firm base of the bladder. The base plate has a sphincteric effect when it is flat and unbroken, but contributes to bladder emptying when it has been broken.

The delicate balance between the detrusor and the sphincter mechanism is maintained by the coordinated reflex activity of the autonomic and somatic nerves mediated peripherally by afferent and efferent pathways between the lower urinary tract and

the micturition centre in the second, third, and fourth sacral segments of the spinal cord and centrally by the modulating inhibitory and facilitatory influence of the higher centres in the nervous system.

With ageing, there are changes to the bladder that include reduced bladder capacity, reduced bladder and urethral compliance, reduced maximal urethral closure pressure, increased PVR volume, and altered diurnal rhythm of urine production. However, while these changes may predispose an individual to incontinence, they are not in themselves a cause.

22.5 Causes

There are many causes of incontinence in older people.[14] It is necessary to consider a classification of the causes of UI. A useful clinical classification of UI is to separate transient from established incontinence.

22.5.1 Transient Causes

Transient incontinence is that which accompanies other underlying acute disease, and in which the incontinence is generally reversible and resolves as the disease is successfully treated.

Resnick suggested the mnemonic of "DIAPPERS" for easy reference and this is widely used (Table 22.3).[15] The Fourth International Consultation on Incontinence[16] made two alternations to the mnemonic: (1) atrophic vaginitis does not itself cause UI and should not be treated for this purpose; and (2) current consensus criteria for urinary tract infection are poorly sensitive and specific in nursing home residents and overtreatment of asymptomatic

Table 22.2 Prevalence figures for UI in Hong Kong[7-13]

Setting	Methodology	Prevalence
Health survey of community-dwelling elders[7,8]	Self-reported symptoms in questionnaire / interview	3.2-4.9%
Elders visiting general practitioners[9]	Self-completed questionnaires	• 18-39 Years: male 2.5%; female 13%; overall 8.6% • 40-59 Years: male 5.2%; female 34.5%; overall 19.9% • 60-74 Years: male 18.1%; female 36.6%; overall 26.4% • ≥75 Years: male 29.5%; female 39.5%; overall 35%
Elders admitted to acute medical wards[10]	UI identified by nursing staff	10.9%
Elders admitted to geriatric wards[11]	UI identified by doctors / nursing staff	36%
Elders staying in convalescence hospital[10]	UI identified by nursing staff	37.6%
Elders with stroke admitted to rehabilitation facilities[12]	UI identified by nursing staff	64.3% with micturition disorder: • 44.9% with UI (14.5% of this group with significant PVR >199 ml) • 17.8% on indwelling catheter • 1.6% continent with PVR >199 ml
Elders staying in care and attention homes[10]	UI identified by nursing staff	23.2%
Elders staying in a residential care institution[13]	Self-reported symptoms during interview	54.1%

Abbreviations: PVR = post-void residual urine volume; UI = urinary incontinence

Table 22.3 Transient causes of urinary incontinence: the "DIAPPERS" mnemonic[15]

Condition	Description
D: Delirium	Confusional states in older people are usually caused by acute medical illnesses such as respiratory tract infection, myocardial infarction, septicaemia, etc.
I: Infection of the urinary tract	Acute inflammation of the bladder can result in increased sensory input to the bladder mucosa and detrusor overactivity. Symptoms will include urgency, frequency, and urge incontinence.
A: Atrophic vaginitis or urethritis	Occurs in postmenopausal women and can result in urgency and frequency. However, it is not currently considered a cause of urinary incontinence and was withdrawn from the mnemonic in 2009.
P: Pharmaceutical	There are various mechanisms whereby medication can affect continence (Table 22.6).
P: Psychological disorders	Frequency and urgency can be related to anxiety. Depression in an older person can affect behaviour and result in inappropriate urination.
E: Excessive urine output	The ageing bladder may not be able to handle the increased urine output resulting from pathological or iatrogenic conditions. Diabetes mellitus, hypercalcaemia, and diuretics are some causes of polyuria.
R: Restricted mobility	Immobility can be related to physical illnesses or conditions such as stroke, parkinsonism, and arthritis. Lack of assistance or presence of environmental obstacles to toileting will exacerbate the problem.
S: Stool impaction	Faecal impaction is discussed in Chapter 23.4.6. The condition will result in reduced functional bladder capacity, spurious diarrhoea and faecal incontinence, as well as a predisposition to urinary tract infection.

bacteriuria should be avoided.

22.5.2 Established Causes

Established causes of UI are classified according to the mechanism affecting the lower urinary tract organ, namely the bladder (either detrusor overactivity or underactivity) or the sphincter (either increase or reduction in outlet resistance), resulting in different types of UI. These are summarised in Table 22.4.

22.5.2.1 Urge Incontinence

Urge incontinence is the involuntary loss of urine associated with an abrupt and strong desire to void (urge). It is usually, but not always, associated with the urodynamic findings of involuntary detrusor contractions referred to as detrusor overactivity or detrusor motor instability. Although involuntary detrusor contractions can be associated with neurological disorders (neurogenic detrusor overactivities), they can also occur in individuals who appear to be neurologically normal. Patients with urge incontinence typically present with irritative symptoms of an overactive bladder, including frequency (voiding more than every 2 hours), urgency, and nocturia (the need to wake at night once or more to void).

22.5.2.2 Stress Incontinence

Stress incontinence is the involuntary loss of urine with increase in intra-abdominal pressure (e.g. due to cough, laugh, exercise). It is due to weakness of the pelvic floor musculature and urethral hypermobility. It may also be caused by bladder outlet or urethral sphincter weakness. Stress incontinence is common in older women. It may be infrequent and involve a very small amount of urine and need no specific treatment in women who are not bothered by it. It is most often associated with weakened supporting tissues and consequent hypermobility of the bladder outlet and urethra caused by lack of oestrogen and / or previous vaginal deliveries or surgery. Obesity and chronic cough can exacerbate this condition.

22.5.2.3 Overflow Incontinence

Although the term "overflow incontinence" is no longer recommended by the ICS, it is still commonly used in clinical settings. Urinary retention with overflow incontinence can result from anatomic or neurogenic outflow obstruction, a hypotonic or acontractile bladder or both. The most common causes include prostatic enlargement, diabetic neuropathic bladder, and urethral stricture. Low spinal cord injury and anatomic obstruction in females (uterine prolapse) are less common causes. Some patients with suprasacral spinal cord lesions develop detrusor-sphincter dyssynergia and consequent urinary retention. A patient may have a history of voiding symptoms during urination with gradual loss of bladder sensation. Nonetheless, symptoms of overflow incontinence are non-specific and urinary retention can easily be missed on physical examination. Thus a PVR urine volume must be ascertained to exclude this condition.

Table 22.4 Established causes of urinary incontinence

Cause	Detrusor overactivity	Reduced outlet resistance / outlet laxity	Increased outlet resistance / outlet obstruction	Detrusor underactivity	Causes that are unrelated to lower urinary tract disorders
Mechanism	Uninhibited bladder contraction causing leakage of moderate-to-large amounts of urine	Increase in intra-abdominal pressure, e.g. coughing, sneezing, laughing or other physical activity, causing leakage of small-to-moderate amount of urine	Distension of bladder resulting in increased intravesical pressure with leakage of variable amount of urine		Inability or unwillingness of a normal continent elderly individual to go to the toilet
Aetiology	• Local bladder pathology: ○ Cystitis ○ Bladder stone / neoplasm • Neurological disorder: ○ Stroke ○ Dementia ○ Parkinson's disease ○ Normal pressure hydrocephalus • Idiopathic	• Oestrogen deficiency • Childbirth • History of pelvic floor surgery • Obesity • Post-prostatectomy	• Prostatic enlargement • Urethral stricture • Spinal cord disease causing detrusor-sphincter dyssynergia • Uterine prolapse • Faecal impaction	• Diabetic neuropathy • Sacral nerve damage • Medications	• Musculoskeletal or neurological disorders • Impaired mental status • Unfamiliar environment • Depression • Sedating medication • Use of physical restraints
Type	Urge incontinence Mixed incontinence	Stress incontinence	Overflow incontinence / chronic retention of urine with incontinence		Functional incontinence

22.5.2.4 Functional Incontinence

Functional incontinence refers to incontinence associated with the inability or lack of motivation to reach a toilet in time. Factors that contribute to functional incontinence can also exacerbate other types of persistent incontinence.

Table 22.5 Some of the conditions that can contribute to urinary incontinence in older people

Type	Condition
Urological and gynaecological	• Urinary tract infection • Bladder stones • Bladder cancer • Urinary tract fistula • Urinary strictures • Prostate gland enlargement • Pelvic floor weakness • Surgery, e.g. lower bowel resection • Pelvic irradiatio
Medical	• Poor mobility • Immobility • Medication • Constipation • Faecal impaction • Acute medical illness • Alcohol excess • Diabetes mellitus • Obesity
Neurological	• Muscle weakness • Stroke • Parkinson's disease • Brain tumour • Hydrocephalus • Multiple sclerosis • Peripheral nerve disorders, e.g. diabetes mellitus • Detrusor-sphincter dyssynergia
Psychological	• Confusion • Dementia • Depression • Stress, anxiety • Disturbed or attention-seeking behaviour
Environmental	• Inappropriate furniture height • Cot side or bed rails • Excessive distance to toilet • Poor lighting • Toilet signs inadequate or absent • Clothing difficult to manipulate • Cold air

22.5.2.5 Multifactorial Causes of UI in Frail Older Persons

Because of the high prevalence of multi-morbidities and polypharmacy in frail older persons, occurrence of UI in this population will usually be the result of an interplay of different factors including urological, neurological, mental, comorbid, iatrogenic as well as functional, environmental, and psychosocial domains. Causes of UI in frail older persons are multifactorial and a comprehensive geriatric assessment is required to delineate the contribution of different pathologies to the problem of UI in a particular patient.

22.6 Evaluation and Investigation

Thorough assessment of older people with incontinence depends on a detailed history to evaluate the above causes and examination of the relevant systems.[2,14] An awareness of the various urological, gynaecological, medical, neurological, psychological, and environmental causative factors of UI in older people is essential. Table 22.5 shows some of the conditions that can contribute to UI in older people, and Table 22.6 shows the medications that can affect continence state.

In patients with a sudden onset of incontinence, the reversible causes of acute incontinence can be excluded by a brief history, physical examination, measurement of PVR urine and basic laboratory studies (urinalysis, culture). Elderly patients with persistent incontinence should be evaluated with a focused history, targeted physical examination, urinalysis, and measurement of a PVR urine. The history should focus on the characteristics of the incontinence, current medical problems and medications, the most bothersome symptoms, and

Table 22.6 Medications that can affect continence state

Effect	Drug class
Delirium, sedation	Anticholinergics, antipsychotics, TCA, anti-Parkinsonian drugs, sedative / hypnotics, narcotic analgesics, alcohol
Cough, exacerbating stress incontinence	ACEI
Polyuria	Diuretics, alcohol
Bladder stimulation causing detrusor overactivity	Caffeinated beverages, cholinergic agents
Bladder relaxation causing urinary retention	Anticholinergics, calcium channel blocker, antihistamine, antispasmodics, TCA
Sphincter contraction causing urinary retention	Alpha-adrenergic agonist, SSRI, SNRI
Sphincter relaxation causing stress incontinence	Alpha-adrenergic blockers
Constipation and faecal impaction	Anticholinergics, narcotic analgesics, antispasmodics, calcium channel blockers, etc.
Oedema associated with nocturnal diuresis, nocturia and nighttime incontinence	Calcium channel blockers, vasodilators, glitazones, NSAID, gabapentin

Abbreviations: ACEI = angiotensin-converting enzyme inhibitor; NSAID = non-steroidal anti-inflammatory drug; SNRI = serotonin and norepinephrine reuptake inhibitor; SSRI = selective serotonin reuptake inhibitor; TCA = tricyclic antidepressant

the impact of the incontinence on the patient and caregivers. Key aspects of the history should include active medical problems, medications, fluid intake, genitourinary history, symptoms of incontinence, neurological symptoms, psychological symptoms, bowel habits, and environmental factors. Completing a bladder chart can help to assess symptoms and follow-up of response to treatment.

Physical examination should focus on the abdomen, rectum, and genitourinary tract along with an evaluation of lumbosacral innervation. During the history and physical examination, special attention should be paid to factors such as mobility, mental state, medications, and accessibility to a toilet. The following aspects of examination should be included: mobility and dexterity, gait disorders, mental state, neurological examination, abdominal examination for bladder distension, rectal examination, prostatic enlargement, and pelvic examination.

Further investigations of patients with more severe UI include urodynamic studies that can be simple or complex. Simple urodynamic studies include the examination of urine flow, simple cystometry and measurement of PVR urine. Patients with difficult symptoms that warrant drug treatment or surgery will require complex urodynamic studies including videourodynamic studies that can help detect the presence of obstruction, detrusor instability, impaired contractility, or bladder neck problems. For patients with chronic urinary retention, an ultrasound of the kidneys, ureters, and bladder should be carried out to evaluate any renal involvement such as stones or hydronephrosis. For patients with limited bladder capacity, cystoscopy may be required to evaluate any structural abnormality within the bladder.

22.7 Management

Management of UI in older people is best planned following thorough assessment to delineate various specific causes of the UI with treatment directed at any specific underlying condition.[2,14,17-19] Attention should be paid to factors that contribute to UI. In frail older persons, because of the multifactorial nature of the aetiology of UI, intervention should be multifaceted following a comprehensive geriatric assessment.

General measures that can help to alleviate the symptoms of UI in most patients include education about diet and fluid intake, regulation of bowel habits, avoidance of bladder irritants (caffeinated beverages), environmental modifications, and provision of toileting aids. A course of rehabilitation will usually enhance the physical and functional ability of the patient and help reduce the occurrence of incontinence. A consensus statement on healthy bladder[20] has been published to promote the achievement of optimal bladder health by increasing healthy bladder habits and behaviours, raising awareness of risk factors, healthcare seeking, clinician engagement, and reducing stigma and other barriers to treatment.

For patients with stress incontinence, pelvic floor exercises with or without biofeedback are highly effective in relieving symptoms. Some patients with urge incontinence can also benefit from pelvic floor

Table 22.7 Drugs used to treat different types of UI

Type	Pathogenesis	Drugs (proprietary name in brackets)	Side-effect / problems
Urge UI	Detrusor overactivity	Anticholinergic agent: oxybutynin (Ditropan), propantheline, tolterodine (Detrusitol), trospium (Spasmolyt), solifenacin (Vesicare), darifenacin (Enablex)	Gastrointestinal: dry mouth, constipation Urological: urine retention Neurological: delirium, fatigue Ocular: blurred vision Cardiac: arrhythmia
		Selective beta3-adrenoceptor agonist: mirabegron	Hypertension, tachycardia Caution in patients with cardiovascular diseases
		Botulinum toxin intradetrusor injections	High rate of urinary retention
Stress UI	Reduced outlet resistance / outlet laxity	Alpha-adrenergic agonist: phenylpropanolamine, ephedrine, pseudoephedrine	Hypertension Increase incident of stroke
		SNRI: duloxetine	Nausea
Overflow UI	Increased outlet resistance / outlet obstruction	Alpha-adrenergic blocker: prazosin (Minipress), terazosin (Hytrin), doxazosin (Cardura), alfuzosin (Xatral SR / XL), tamsulosin (Harnal)	Postural hypotension Intraoperative floppy iris syndrome
		5alpha-reductase inhibitor: finasteride (Proscar), dutasteride (Avodart)	Slow onset of action Only act on big prostate
	Detrusor underactivity	Cholinergic agonist: distigmine (Ubretid), bethanechol (Urecholine)	Gastrointestinal: diarrhoea, abdominal pain Urological: increase intravesical pressure

Abbreviations: SNRI = serotonin and norepinephrine reuptake inhibitor; UI = urinary incontinence

exercises since pelvic floor contraction is a strong inhibitor of detrusor overactivity.

Behavioural modifications are important in the treatment of UI. For the cognitively intact individual, bladder training aims to reduce the occurrence of inappropriate urge and to increase the functional capacity of the bladder. In cognitively impaired individuals, a regular or tailor-made toileting regimen, e.g. prompt voiding or timed voiding, aims to increase the chance of dryness.

Table 22.8 Complications of an indwelling urinary catheter

Urinary infection
Indwelling urinary catheter blockage
Local ulceration at urethra (traumatic hypospadias) and bladder
Stricture
Encrustation
Urine leak
Discomfort / pain
Hinders walking

Various drugs have been used to treat different types of UI (Table 22.7).

For patients with detrusor overactivity, anticholinergic agents with M3-specific antimuscarinic activity are commonly prescribed. Their clinical efficacy is modest[21] and the long-term adherence to the therapy appears low.[22] Oxybutynin is the prototype drug but the prevalence of side-effects (most commonly dry mouth) is high. Newer antimuscarinic agents have reduced the incidence of dry mouth. A selective beta-3 adrenoceptor agonist[23] has recently been introduced as a new class of medication used for detrusor overactivity. Its role over antimuscarinic agents is yet to be defined. Its potential effect on the cardiovascular system may limit its use in patients with co-existing cardiovascular problems. Intradetrusor injection of botulinum toxin[24] is reserved for patients with neurogenic detrusor overactivity refractory to conventional medical treatment although there is a considerable incidence of urinary retention after the procedure. There is also an emerging role of Botox injection in idiopathic detrusor overactivity.[25]

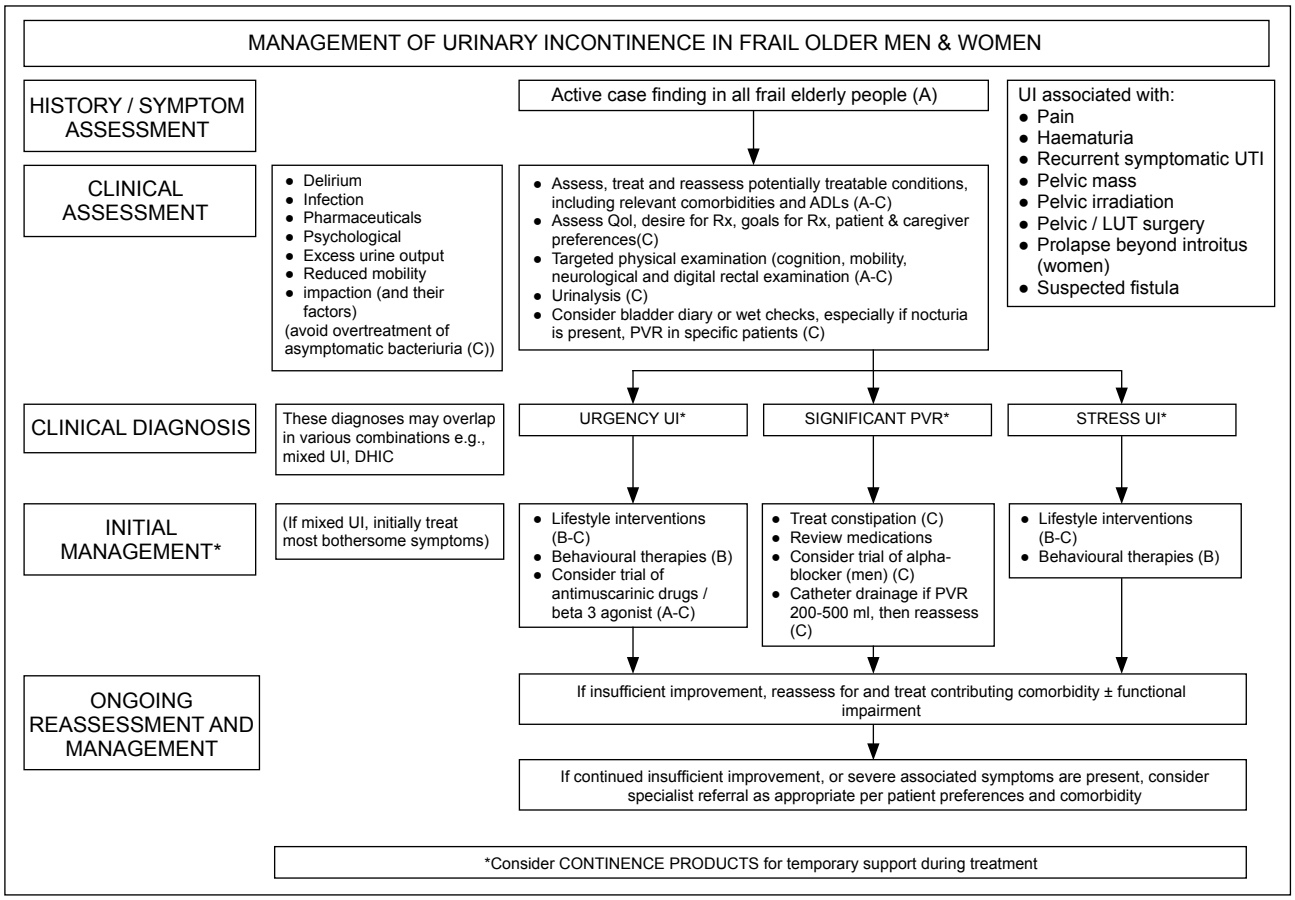

Figure 22.1 Algorithm for initial management of urinary incontinence in frail elderly people*[28]
(Reproduced with permission from International Continence Society)
Abbreviations: ADL = activities of daily living; DHIC = detrusor hyperactivity with impaired contractility; LUT = lower urinary tract; PVR = post-void residual; QoI = quality of life; Rx = treatment; UI = urinary incontinence; UTI = urinary tract infection
* (A), (B), (C) refers to grades of recommendation based on evidence from randomised controlled trial, non-randomised / experimental study, and non-experimental descriptive study, respectively

Alpha agonists can increase internal sphincter tone while duloxetine acts on the rhabdosphincter. Both drugs can theoretically improve stress incontinence but the occurrence of side-effects limits their use.[21] Pelvic floor exercises remain the mainstay of treatment in patients with stress incontinence.

Systemic administration of oestrogen in postmenopausal women does not seem to help UI.[26] There is evidence that oral oestrogens result in worsening incontinence compared with placebo, although local application (e.g. vaginal creams or tablets) may help.

Male patients with bladder outlet obstruction secondary to benign prostatic hyperplasia can be treated with alpha-adrenergic antagonists and / or 5alpha-reductase inhibitors. Alpha-blockers have a faster onset of action but commonly result in postural hypotension and falls. 5Alpha-reductase inhibitors can shrink prostate size but they usually take at least half a year before their clinical effect can be observed. Many patients will eventually require prostatectomy.

Cholinergic agents, which stimulate detrusor contraction, have been used in patients with detrusor underactivity and chronic urinary retention. Such stimulant drugs are generally ineffective, however.[27] There is also a risk of further elevation of the already high intravesical pressure with a genuine risk of damaging the upper urinary tract and exacerbating obstructive uropathy.

Most patients with chronic urinary retention will eventually require catheterization in order to protect the upper urinary tract. Long-term indwelling catheterisation has many potential complications (Table 22.8). Selected patients can benefit from clean intermittent catheterisation that results in a lower urinary infection rate if performed correctly and better patient acceptance.

Patients with resistant incontinence not amenable to conservative measures and drugs may require surgical intervention. Female patients with stress incontinence who fail to respond to pelvic floor exercises will need to consider surgery. With the introduction of minimally invasive surgery and tension-free vaginal tapes, many older women can benefit from less invasive procedures to treat their stress incontinence.

22.8 Algorithm for Initial Management of UI in Frail Disabled Elderly People

Recommendation from the Sixth International Consultation on Incontinence for initial management of UI in frail elderly people is shown in Figure 22.1.[28]

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CHAPTER 23

Constipation and Faecal Incontinence

Bing-chung TONG

OBJECTIVE

To learn the normal mechanism of faecal evacuation and maintenance of continence as well as the epidemiological data, types, causes, assessment, and treatment of constipation and faecal incontinence.

KEY POINTS

- Maintenance of faecal continence requires normal rectal and sphincteric function and is also dependent on stool volume and consistency.
- A precise definition of constipation should take account of stool frequency, stool consistency, requirement for straining or manual manoeuvre, and sensation of incomplete evacuation or anorectal block.
- Constipation is common in elderly people, particularly in the context of physician visits and laxative use.
- Causes of constipation in elderly people are an interplay of physiological, pathological, and iatrogenic factors.
- Promotion of a healthy bowel habit helps to prevent and treat constipation.
- There are different classes of laxative. A rational prescribing approach is a key to relieve symptoms.
- Diagnosis of faecal impaction in the context of non-specific complaints requires a high index of suspicion.
- An abdominal X-ray is sometimes needed to diagnose faecal impaction.
- Faecal incontinence has a significant impact on the individual, caregiver, and society.
- Faecal impaction with overflow incontinence is the single most important reversible cause of faecal incontinence in elderly people, especially in those who are institutionalised.

23.1 Introduction

Defecatory disorders are important topics in geriatric medicine. Constipation is commonly encountered in our daily clinical practice, and many elderly patients are chronic users of laxatives. Faecal incontinence has a significant physical, psychosocial, and economic impact on the individual, caregivers, and society. Assessment of constipation and faecal incontinence requires knowledge of the physiology and pathophysiology of the defecatory mechanism as well as an appreciation of the interplay of different individual disease processes, iatrogenesis, and environmental factors. Many treatment modalities are available.

23.2 Normal Physiology of Defaecation

The colon receives around 1-1.5 litres of fluid, salt, fibre, and other residues each day. Approximately 90% of water and salt is reabsorbed and 100 g of stool, which consists of 70 ml of water and 30 g of solid material, is produced. The degree of desiccation of bowel content is related to the transit time along the colon.[1-4]

Colonic motor activities include non-propulsive contractions that are responsible for mixing the bowel content and propulsive contractions that move the content forwards and backwards. There is a circadian rhythm of colonic activities such that the transverse / descending colon exhibits more activity during waking hours while nocturnal activity is dominated by periodic rectal motor activity. The high-amplitude propagated contractions induce mass migration of colonic content. These contractions often occur after a meal (the gastrocolic reflex), and may be accompanied by the urge to defecate.

Movement of faeces into the rectum stimulates tactile receptors in the anal canal and stretch receptors in the pelvic floor and rectum, triggering the urge to defecate. Smooth muscle of the internal anal sphincter

(IAS) relaxes transiently (rectoanal inhibitory reflex) and the external anal sphincter (EAS) simultaneously contracts (rectoanal contractile reflex). A rectoanal sensorimotor response has recently been described in which there is a transient contractile response of the anal sphincter associated with rectal sensory perception.[5] The clinical and physiological significance of the sensorimotor response has not been determined. The decrease in anal pressure allows rectal content to be in contact with the sensitive receptors at the anal canal momentarily, aiding in recognition of whether the material is solid, liquid, or gas (sampling response). Solids can be retained, and gas can be passed relieving the intrarectal pressure. Voluntary contraction of the EAS can extend the period of continence and allow time for a compliance mechanism within the colon to compensate for the increase in rectal volume (rectal accommodation response). As the colon accommodates the new volume, stretch receptors are no longer activated and the urge disappears. Further rectal distension leads to inhibition of the EAS, making the body less able to postpone defaecation. When time and place are appropriate, relaxation of the sphincters and the puborectalis muscle together with rectal contraction allow passage of faeces away from the body.

Faecal continence depends on the following mechanisms.[1,6]

- Reservoir function of the rectum: This relates to the adaptive compliance of the rectum along with rectal capacity and distensibility.
- Rectal and anal sensation: An intact sensation provides a warning of imminent defaecation and is essential for discriminating different stool consistency and flatus. Impaired sensation can also lead to faecal impaction and its complications.
- Sphinteric function: The IAS contributes to 70-85% of the resting sphincter pressure and is chiefly responsible for maintaining anal continence at rest. The EAS provides voluntary control of continence in response to cough, rectal distension, and anal dilatation. The anal endovascular cushion is represented by vasculature at the submucosal and subcutaneous tissue of the anal canal and is able to expand and contract and to take up slack, thus contributing to the finest degree of anal continence.
- The anorectal angle: The angulation of 80-90 degrees between the axis of the rectum and the anal canal is maintained by contraction of the puborectalis muscle. As a result of the angulation, the flap of anterior rectal mucosa lies over the upper end of the anal canal and functions as an occlusion (flap-valve theory). Increase in intra-abdominal pressure accentuates the angulation and forces the anterior rectal mucosa more firmly over the upper anal canal, thus preventing faecal leakage. The significance of the anorectal angle and the flap valve theory in maintaining faecal continence are nonetheless controversial.
- Stool volume and consistency: Excessive volume and fluid stool are more difficult to retain inside the rectum.

23.3 Age-related Changes in Defecatory Function

Colonic transit does not appear to change with ageing but the reservoir volume of the rectum is reduced and the perception of rectal distension is often impaired. Anatomically, the thickness of the IAS is increased, and this is thought to be related to fibrosis. On the contrary, there is a thinning of the EAS that may indicate progressive denervation. Functionally, both the resting pressure (IAS contraction) and the squeeze pressure (by contraction of EAS) of the anal canal decrease with ageing, this reduction being more pronounced in women. It is difficult to tell whether the dysfunctions are related to ageing or disease processes.[3]

23.4 Constipation

23.4.1 Definition of Constipation

Different people define constipation differently. It is reported that 99% of the population have between three bowel movements a week and three bowel movements a day.[7] Many older people insist on a daily bowel opening in order for them to be healthy. According to the American Gastroenterological Association (AGA) Medical Position statement,[8] the symptom of constipation from the patient's perspective (in decreasing order of importance) includes straining, stool that is excessively hard, unproductive urge, infrequency, and feeling of incomplete evacuation. The Rome III criteria for functional constipation,[9] which describes a group of functional disorders that present as persistently difficult, infrequent or seemingly incomplete defaecation, helps to give a better idea of what constipation is, at least for epidemiological purposes (Table 23.1).

23.4.2 Epidemiology of Constipation

The prevalence of constipation in the general population worldwide ranges from 0.7-79%.[10] Interpretation of prevalence studies of constipation should take account of the definition used in any particular study. For example, a Canadian telephone survey reported a prevalence of 27.2% for self-reported constipation ("Have you felt constipated?") in the past 3 months and 38.6% if the period was extended to 12 months.[11] If Rome I and Rome II criteria were used for the same group of participants, the prevalences would have been 16.7% and 14.9%, respectively. Studies on whether the prevalence of constipation increases

Table 23.1 Rome III criteria for functional constipation* [9] (reproduced with permission)

- Must include two or more of the following:
  - Straining during at least 25% of defecations
  - Lumpy or hard stools in at least 25% of defecations
  - Sensation of incomplete evacuation for at least 25% of defecations
  - Sensation of anorectal obstruction / blockage for at least 25% of defecations
  - Manual manoeuvres to facilitate at least 25% of defecations (e.g. digital evacuation, support of the pelvic floor)
  - Fewer than three defecations per week
- Loose stools are not present without the use of laxatives
- Insufficient criteria for irritable bowel syndrome

* Criteria fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis

Table 23.2 Diagnostic criteria of constipation predominant irritable bowel syndrome (IBS)[9] (reproduced with permission)

Diagnostic criteria for irritable bowel syndrome
- Recurrent abdominal pain or discomfort at least 3 days per month in the last 3 months associated with two or more of the following:
  - Improvement with defecation
  - Onset associated with a change in frequency of stool
  - Onset associated with a change in form (appearance) of stool
- Criteria fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis

Subtyping IBS by predominant stool pattern
- IBS with constipation (IBS-C) — hard or lumpy stools 25% and loose (mushy) or watery stools 25% of bowel movements
- IBS with diarrhoea (IBS-D) — loose (mushy) or watery stools 25% and hard or lumpy stool 25% of bowel movements
- Mixed IBS (IBS-M) — hard or lumpy stools 25% and loose (mushy) or watery stools 25% of bowel movements
- Unsubtyped IBS — insufficient abnormality of stool consistency to meet criteria for IBS-C, -D, or -M

with age have yielded markedly inconsistent results. Nonetheless it is generally believed that physician visits for constipation are significantly more common among elderly people[12] and laxatives are used more commonly by elderly women.[13]

In Hong Kong, the prevalence of constipation as defined by the Rome II criteria[14] has been reported to be 14.3%, with a female-to-male ratio of 1.3:1. The prevalence did not differ between younger and older age-groups. A Hong Kong population survey of various heath conditions in 2003/2004, however, found an increased prevalence of acute constipation within the preceding month in older age-groups (14.1% in the group of 65-74 years and 22.8% in those older than 75 years compared with 8.8-11.5% in the younger age-group).[15] Other local data concerning constipation include a prevalence of 1.7% for the constipation predominant subtype of irritable bowel syndrome[16] and a prevalence of 27.5% for constipation in diabetic patients.[17] It was found that 70.5% of diabetic patients had gastrointestinal symptoms. Apart from constipation, 34% had diarrhoea and 16.8% had a sensation of epigastric fullness.

23.4.3 Pathophysiology of Constipation

23.4.3.1 Functional Constipation

Below shows three main types of functional constipation.[18,19]

- Slow-transit constipation is a disorder of colonic motor function attributable to ineffective colonic propulsion. The cause can be a primary dysfunction of colonic smooth muscle (myopathy) or its innervation (neuropathy) or can be secondary to dyssynergic defecation.
- Dyssynergic defecatory disorder (synonyms: anismus, pelvic floor dyssynergia, paradoxical pelvic floor contraction, and obstructive constipation) is caused by the paradoxical anal contraction or involuntary anal spasm during defaecation.
- Normal-transit constipation is an ill-defined category in which patients perceive difficulty with evacuation or presence of hard stool. The patients may experience bloating and abdominal pain and may exhibit increased psychological distress. Many have symptoms that constitute a diagnosis of constipation-predominant irritable bowel syndrome (Table 23.2).[9]

23.4.3.2 Disease-related Constipation

Many medical conditions and medications are associated with constipation (Table 23.3)[20] and are often more prevalent in elderly people. Nonetheless the cause of constipation in elderly people is usually multifactorial, with an interplay of various disease processes and iatrogenic factors.

23.4.4 Clinical Assessment of Constipation

The first question to be answered during a clinical assessment will relate to whether the patient is truly constipated. A stool diary, paying attention to frequency of bowel opening and stool form, is

Table 23.3 Common medical conditions associated with constipation[20]

Mechanical obstruction	Metabolic condition	Drug
• Colorectal cancer	• Diabetes mellitus	• Opiates
• External compression from malignant lesions	• Hypothyroidism	• Anticholinergic agents
• Strictures	• Hypercalcaemia	• Calcium channel blockers
• Large rectocele	• Hypocalcaemia	• Tricyclic antidepressants
• Megacolon	• Hypokalaemia	• Antipsychotics
• Postsurgical abnormality	• Hypomagnesaemia	• Calcium supplements
Painful anal conditions	• Uraemia	• Iron supplements
• Anal fissure	• Heavy-metal poisoning	• Calcium-containing antacids
• Prolapsed piles	Neurological conditions	• Antidiarrhoeal agents
• Perianal abscess	• Parkinson's disease	Miscellaneous
Connective tissue disorder	• Spinal cord injury or tumour	• Depression
• Scleroderma	• Cerebrovascular disease	• Cognitive impairment
• Amyloidosis	• Multiple sclerosis	• Immobility
	• Autonomic neuropathy	

Table 23.4 Bristol Stool Form scale[21,22]

Type	Appearance
1	Separate hard lumps, like nuts
2	Sausage-shaped but lumpy
3	Like a sausage or snake but with cracks on its surface
4	Like a sausage or snake, smooth and soft
5	Soft blobs with clear-cut edges
6	Fluffy pieces with ragged edges, a mushy stool
7	Watery, no solid pieces

Table 23.5 Indications for gastroenterology referral in constipated individuals[24]

- Recent onset or chronic constipation associated with:
  - unintentional weight loss
  - anaemia
  - abdominal pain
  - blood per rectum or presence of faecal occult blood
- Unexplained change in bowel habit, involving stool frequency or consistency
- Failure to alleviate constipation despite compliance with adequate lifestyle modification
- Chronic constipation necessitating use of high doses of laxatives
- Recent-onset faecal incontinence

preferable. It may sometimes be helpful to refer to the Rome criteria for clarification.[9] For functionally constipated individuals, stool consistency, which can be assessed and graded by the Bristol Stool Form scale (Table 23.4[21,22]), correlates with colonic transit.[23] A sensation of incomplete evacuation and anorectal blockage together with the need to use manual manoeuvre to facilitate defaecation may suggest dyssynergic defaecation. Abdominal pain relieved by a bowel motion is the key to diagnosis of irritable bowel syndrome. Dietary and lifestyle factors should be managed. Since medical diseases as well as iatrogenesis are more prevalent in elderly patients, the differential diagnoses listed in Table 23.3[20] should be considered with symptoms and signs of the individual disease entity actively elicited. Clinicians should be able to pick up 'red flag' symptoms and signs so that any underlying sinister cause is not missed. Recommendations for gastroenterologist referral (Table 23.5)[24] for patients with alarming clinical features are available for reference. Medication history, in particular laxative use, should be reviewed. Rectal examination should be focused on detection of perineal and perianal pathology, assessment of sphincter tone, and presence of faecal impaction. Proctoscopy examination revealing blood coming down from a level above the anal canal is an important sign for locating pathology proximal to the anal canal, instead of attributing any rectal bleeding to haemorrhoid. Laboratory investigations should be directed by clinical findings. Complete blood count, renal function and electrolytes, calcium, glucose, thyroid function, faecal occult blood, and colonoscopy may be indicated in specific clinical settings. The American College of Gastroenterology advises against routine use of these investigations in every constipated individual.[25] Sophisticated investigations such as balloon expulsion test, colonic transit study, anorectal manometry and defaecography[26] will be available in tertiary gastroenterological referral centres. Last but not the least, the psychosocial well-being of the constipated individual should also be assessed and counselling may be needed.

23.4.5 Management of Constipation

Individuals who do not have symptoms leading to a diagnosis of constipation may only need reassurance and explanation of what constitutes a normal healthy bowel habit. The need to avoid excessive laxative use should also be stressed. A healthy bowel habit includes a prompt response to the urge to defecate. The establishment of a consistent time for defecation is desirable, with the aid of the gastrocolonic response during the postprandial period, particularly in the morning. During defaecation, maintaining the body upright with the hips flexed will make the anorectal angle less acute. This will theoretically help stool

passage. Availability of appropriate toilet facilities is important for an individual with mobility problems. Use of a bedpan should be avoided, wheelchair accessibility should be considered and toilet facilities may require modification (e.g. hand rail or raised toilet seat).[18,27,28]

Dietary fibre can increase stool weight and accelerate colonic transit. A fibre intake of 20-30 g per day is usually recommended. Fibre supplementation in the form of wheat bran, fruits (prune, blackberries, grapes), and vegetables (beans, peas, broccoli) in a palatable form for institutionalised older people is of particular importance. A high-fibre diet may result in gastrointestinal upset such as bloating and flatulence, but can be minimised by starting low and going slow. Despite a lack of evidence that extra fluid and exercise help to relieve constipation, maintaining adequate hydration, particularly for those who are taking a bulk-forming or osmotic laxative, and encouraging mobility are generally beneficial. Observations from refeeding of patients with anorexia nervosa suggest that an adequate calorie intake is also important for normal colonic transit.

Many patients do resort to laxatives for their bowel problem despite trying lifestyle modification. Bulk-forming laxatives, including natural (psyllium) or semi-synthetic (methylcellulose) fibre are the most commonly used. These agents increase colonic residue and stimulate peristalsis. As with other fibre supplements, the gastrointestinal side-effects may be intolerable to some patients. Anaphylaxis and asthma are significant but rare side-effects of psyllium.

Osmotic laxatives draw water into the intestine along an osmotic gradient and soften stool. Saline laxatives containing magnesium should be used with caution in patients with renal insufficiency. Lactulose is commonly used but sorbitol appears to be a cheaper and equally effective alternative. These poorly absorbed sugars cause significant gas production and hence a side-effect of bloating and flatulence. There has been an increase in use of polyethylene glycol in small packs in recent years since the polymers, not being metabolised by gut bacteria, cause less bloating and cramping than lactulose. This is available in Hong Kong over-the-counter as sachets (macrogol 4000). Osmotic laxatives can cause fluid and electrolyte disturbance particularly when taken in excess and with inadequate fluid.

Stimulant laxatives have gained a bad reputation since they have been linked to serious side-effects of pseudomelanosis coli, cathartic colon, enteric neuronal degeneration together with malignant and abuse potential. Nonetheless, with the exception of liquid paraffin (which can cause malabsorption of fat-soluble vitamins and anal seepage with long-term use and lipoid pneumonia when aspirated) and phenolphthalein (which was observed to have carcinogenic potential in an animal study), most stimulants (senna, bisacodyl, docusate, and sodium picosulphate) are generally thought to be safe and effective when used no more than 2-3 times per week, even for a prolonged period. The malignant potential for melanosis coli has been questioned and also the presence of the entity of cathartic colon. The microscopic neuronal degeneration is not specific to laxative use and human study has provided inconclusive evidence of the link of colonic cancer to stimulant laxative use.

Newer agents have been introduced to the market in the past decade.[29,30] 5-Hydroxytryptamine receptor 4 agonists are prokinetic agents that induce peristalsis. Prucalopride has been proven to be effective in improving symptoms of constipation and is available in Hong Kong. Secretagogues promote intestinal secretion and thus produce softer stools. They also accelerate intestinal transit. Lubiprostone (acts on type 2 chloride channels) and Linaclotide (acts on guanylate cyclase C) have been proven effective in clinical trials. A peripherally acting mu opioid receptor antagonist methylnaltrexone administered subcutaneously is effective in inducing laxation in palliative care patients with opioid-induced constipation when conventional laxatives have failed.

Other agents that have been reported to be of use in treating constipation include colchicine and misoprostol although side-effects have limited their use.

The AGA medical position statement on constipation 2013[31] has suggested a practical approach to medical management of constipation. An initial gradual increase in fibre intake is suggested, with or without addition of inexpensive osmotic agents, such as milk of magnesia or polyethylene glycol. Depending on stool consistency, the next step may be to supplement the osmotic agent with a stimulant laxative (e.g. bisacodyl or glycerin suppositories), preferably administered 30 minutes after a meal to synergise the pharmacologic effect with the gastrocolic response. Newer agents may be considered when symptoms do not respond to traditional laxatives.

Biofeedback[32,33] is an instrument-based educational programme used for dyssynergic defaecation directed at coordinating pelvic floor muscle relaxation with a Valsalva manoeuvre. The instruments used can be electromyography (EMG) [intra-anal or perianal] or anal-canal pressure feedback (such as manometry or balloon expulsion) devices. Patients are also taught diaphragmatic breathing techniques. Sensory training

can also improve the threshold for rectal sensory perception and promote better awareness for stooling. On the whole, the symptomatic improvement rate has been reported to be 44-100%.[32,33] Locally, biofeedback in the context of a multidisciplinary treatment with diet modification and bowel habit adjustment can achieve a more than 70% subjective and objective improvement.[34]

Surgery is reserved for a highly select group of patients. Rectal surgery might be needed in patients with pelvic floor obstruction due to clinically significant rectocele or rectoanal intussusception. Subtotal colectomy with ileorectal anastomosis might be needed in patients with slow-transit constipation refractory to medical treatment.

There has been an increase in the use of complementary and alternative medicine[35,36] in the western world in recent years. Manual colonic massage appears to be the simplest treatment. This has been widely practised in palliative and long-term care settings. Other complementary and alternative medicine includes probiotics, acupuncture, homeopathy, shiatsu, herbalism, reflexology, and aromatherapy.

23.4.6 Faecal Impaction

Faecal impaction is the inability to pass a collection of stool, usually hard in consistency. Constipation results in faecal retention; the colon's normal absorption of salt and water contributes to the hardening of stool, and peristalsis causes packing. Typical symptoms include anorexia, nausea, vomiting, and abdominal pain. The hard stool irritates the colonic or rectal mucosa resulting in mucus production that dissolves and liquefies the surface bowel content. As a result, there is leakage of liquid faeces around hard-impacted faeces causing paradoxical diarrhoea (spurious diarrhoea) and, as a result of impaired rectal sensation and anal sphincter relaxation, faecal incontinence. The urinary bladder is in close proximity to the rectum so that faecal impaction can result in urinary frequency, retention, and overflow incontinence. The patient will be prone to urinary tract infection. Other complications include stercoral ulceration with bleeding or perforation, intestinal obstruction, idiopathic megacolon, volvulus, and fecaloma. Mental confusion has also been a reported complication, presenting as delirium in an individual with pre-existing cognitive impairment.[37-39]

Diagnosis of faecal impaction, especially when a patient presents with non-specific complaints like anorexia and nausea, requires a high index of suspicion. Rectal examination is crucial in all cases but judicial use of plain abdominal X-ray can help identify proximal or 'high' impaction. It should be noted that impacted stool can be of any consistency (soft to rock-hard) and can take many forms (a single mass or multiple pellets).

Manual fragmentation and extraction, with the aid of lignocaine jelly, of the faecal mass is often needed as the first treatment of faecal impaction. Repeated enema or suppository, to the extent of daily use for a week, may also be required. Whole-gut irrigation using 2 litres of polyethylene glycol electrolyte balance colonic lavage solution is a useful alternative in non-emergency cases without complete obstruction. Occasionally, lavage directed by sigmoidoscopy or surgery may be needed in severe cases. Prevention of further impaction is essential. This may be achieved with lifestyle methods but the use of a regular bulk-forming or osmotic laxative with adequate hydration may be necessary in some cases.

23.5 Faecal Incontinence

23.5.1 Definition of Faecal Incontinence

Faecal incontinence is defined as uncontrolled passage of faecal material recurring for 3 months or longer,[40] in an individual with a development age of at least 4 years.

23.5.2 Epidemiology of Faecal Incontinence

Epidemiological data for faecal incontinence should be interpreted with caution because of a high chance of underreporting. Table 23.6 shows the prevalence of faecal incontinence in the western population in general.[41,42] The prevalence of faecal incontinence increases with age. In younger persons, the condition is more common in women (8 times more common at age 45 years) but this difference narrows with increasing age (prevalence similar at age 70 years). It should also be noted that double incontinence is 12 times more common than isolated faecal incontinence in elderly patients.[41,42]

In Hong Kong, the prevalence of faecal incontinence was 2.6%[43] and 2.9%[44] in two community-based studies. In a study of the prevalence of urinary incontinence in institutionalised elderly in Hong Kong, 63.8% has concomitant faecal incontinence.[45] The impact of faecal incontinence is listed in Table 23.7.

23.5.3 Causes of Faecal Incontinence

Mechanisms for maintaining continence have already been discussed in Chapter 23.2. Clinically there are three types of faecal incontinence[46]:

- passive incontinence: involuntary discharge of faecal matter or flatus without any awareness, caused by loss of perception and / or impaired rectoanal reflexes either with or without sphincter dysfunction;

- urge incontinence: discharge of faecal matter or flatus despite active attempts to retain these contents, caused by predominant disruption of sphincter function or the rectal capacity to retain stool; and
- faecal seepage: undesired leakage of stool, often after a normal bowel motion, caused by incomplete evacuation or stool and / or impaired rectal sensation.

Table 23.6 Prevalence of faecal incontinence in western population[41,42]

Population group	Prevalence
General population	2-13%
Community-dwelling older people	3-27%
Long-term care facilities	20-58%
Hospitalised setting	
Geriatric wards	20-30%
Psychogeriatric wards	58%
Patients hospitalised with dementia	80%
Patients having neurological disease	
Stroke	30-40% At admission; 9% at 6 months
Multiple sclerosis	51%
Spinal cord injury	11% Weekly; 50% occasionally

Table 23.7 Impact of faecal incontinence

Marker of poor overall health and increased mortality
Causes anxiety, embarrassment, and social isolation
Experiences more urinary tract infections and pressure ulcers
Second most common cause for institutionalisation
Increased health expenditure

Table 23.8 lists the aetiologies of faecal incontinence.[47] Faecal impaction with overflow incontinence is the single most important reversible cause in elderly people, especially in those who are institutionalised. Drug-induced diarrhoea is another common cause to be looked for. Vaginal delivery-related complications underlie most cases of postpartum faecal incontinence and may be related to direct sphincter damage or damage to pudendal or other pelvic nerves. The presentation may be delayed in the latter case.

23.5.4 Clinical Assessment of Faecal Incontinence

Since faecal incontinence can arise from diverse pathologies, evaluation of patients should aim to identify treatable conditions. Symptoms and signs of colorectal disease listed above should be elicited. History of obstetric complications, anorectal surgery, pelvic malignancy and radiotherapy, as well as neurological disorders are important in contributing to incontinence and the possibility of iatrogenesis should be determined. A detailed neurological examination is mandatory with particular attention paid to perianal sensation. The patient's mental state, mobility, and dexterity should be assessed and environmental issues including toilet accessibility are other important domains to be looked into. Rectal examination should be performed to look for 'low impaction'. Any perineal or perianal pathology should be noted. The sphincter tone should be assessed. An abdominal X-ray can show 'high impaction.' Other laboratory or endoscopic investigations should be directed by relevant clinical findings. More advanced investigations such as proctography, manometry, EMG, pudendal nerve latency study, defecography and endoanal ultrasonography or magnetic resonance

Table 23.8 Aetiologies of faecal incontinence[47]

Overflow incontinence
• Faecal impaction
Inadequate reservoir capacity / compliance problem
• Inflammatory bowel disease, irradiation proctitis, post-surgery, chronic rectal ischaemia, collagen vascular disease
Inadequate rectal sensation
• Various neurological diseases: dementia, stroke, spinal cord disease, diabetic and other neuropathy, multiple sclerosis
Defective sphincter mechanism or pelvic floor mechanism
• Anatomical sphincter defect
o Obstetric injury: third-degree tear, episiotomy wound complication, forceps injury
o Anorectal surgery: haemorrhoidectomy, anal fistula surgery, sphincterotomy
o Neoplasm
o Inflammation
• Denervation of puborectalis and external sphincter
o Postpartum pudendal neuropathy
o Idiopathic neurogenic incontinence: relating to multiple vaginal deliveries, chronic straining at stool, and descending perineum syndrome
o Secondary to spinal cord, cauda equina and pelvic floor nerve injuries and diabetic neuropathy
• Others
o Rectal prolapse
Diarrhoea state
• Neoplasm, infective, inflammatory bowel disease, malabsorption, irradiation enteritis, diabetes mellitus, medication (laxative, metformin, colchicine, misoprostol, ticlopidine, proton pump inhibitor)

imaging may be indicated in selected cases.[47]

23.5.5 Management of Faecal Incontinence

The primary pathology of faecal incontinence should be treated as far as possible. In institutionalised older people, detection and management of faecal impaction is crucial (see Chapter 23.4.6), since the consequent spurious diarrhoea may lead to a mistaken prescription of antidiarrhoea drugs. This may increase faecal impaction further and thus worsen the condition. A high-fibre diet or bulk-forming agents are usually added if faecal impaction is excluded or treated. This can solidify stool and may enhance sphincter function. Antidiarrhoeals such as loperamide are helpful in cases of chronic diarrhoea. While pelvic floor exercises alone are not thought to be effective in improving sphincter function, biofeedback using balloon manometry and EMG monitoring can help to improve sphincter strength and rectal sensation. The success rate in improving faecal continence can be up to 50-75%. Behavioural therapy includes advising patients to go to the toilet after every meal, when the colorectal response is likely to produce a filled rectum, even without such sensation. For cognitively impaired individuals, prompt voiding might be effective, and this is best combined with a fluid prompt protocol and mobility exercises. Planned evacuation, use of regular antidiarrhoeals alternating with enema, may be required in order to make the incontinence more manageable. Surgery (sphincter repair, neosphincter, or diversion) will be reserved for selected patients with intractable incontinence. Sacral nerve stimulation and radiofrequency energy to the sphincter are other modalities to be considered. Environment modification and incontinence aids such as pads and napkins are often necessary when a primary disease process is not totally reversible. Psychological support is essential.[24,41,48,49]

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CHAPTER 24

Dementia and Delirium

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OBJECTIVES

To learn the clinical diagnosis and management of dementing illnesses in elderly people. To recognise delirium superimposed upon dementia in an elderly person and appreciate its clinical significance. To understand the non-pharmacological and pharmacological management of the cognitive and non-cognitive symptoms of dementia.

KEY POINTS

- Dementia is prevalent among elders in Hong Kong, and the prevalence and impact of the disease is increasing with ageing of the population.
- The aetiology of dementia is multiple, and can be differentiated by careful clinical evaluation, neuropsychological testing, neuroimaging, and observation of the progression of illness.
- It is important to exclude causes of cognitive impairment other than dementia that may be reversible.
- Changes to clinical diagnostic criteria for neurocognitive disorder in the 5th revision of the Diagnostic and Statistical Manual of Mental Disorders are highlighted. Advanced neuroimaging studies and biomarkers are not required in usual clinical settings, but may play a role in research and for ascertainment of the aetiology of dementing illness.
- Cholinesterase inhibitors are mainly used for symptomatic treatment of Alzheimer's disease. The clinical benefits in cognition and global functioning are marginal. Management of the psychological symptoms of dementia should be largely non-pharmacological. Psychoactive drugs are mainly reserved for psychological symptoms that cause extreme distress or that pose an imminent threat of harm to self or others. Attention to the side-effects of the latter drugs should be given, especially the extrapyramidal effects and increased risk of stroke. Recruitment of supportive services for patients and family members is an essential part of management.
- Delirium can present in a hyperactive or hypoactive form; the latter being often under-recognised.
- Delirium should be carefully excluded or treated in elders who present with a change in mental, cognitive, or behavioural status. Delirium may indicate underlying cognitive decline; hence patients who have recovered from the episode should be monitored for development of dementia in subsequent years.
- Delirium can be managed by optimising the psychosocial and physical environment of the patient, and treating or reversing precipitating factors. If a psychoactive medication is used, always start low and go slow.

24.1 Dementia

24.1.1 Introduction

The first case of Alzheimer's disease (AD), reported in a 51-year-old woman, was published in 1907 by Alzheimer and titled "On an unusual illness of the cerebral cortex".[1] The hallmark of the pathology of AD is the presence of senile plaque and neurofibrillary tangles in the brain. Clinical diagnosis is mainly based on the presentation and progression of the illness. Investigation aims to exclude other causes of cognitive dysfunction that may be reversible.

Dementia is a common condition with a significant impact on both the patient and the patient's family. It is associated with decline in self-care ability leading to incontinence, tendency to fall, mood problems, and other behavioural problems and institutionalisation. It also causes significant stress for caregivers. Specific pharmacological management of AD and other dementias produces only modest effect in the early-to-moderate stages. Non-pharmacological treatment is equally important, particularly in the management of non-cognitive symptoms of dementia and in reducing caregiver stress.

Memory impairment is a common complaint of elders. It can be age-related or pathological with different outcomes. Therefore it is important to recognise and provide proper assessment of cognitive decline in elderly people.

24.1.2 Epidemiology and Impact of Dementia

The estimated prevalence of dementia varies considerably globally, partly because of differences in methodology and diagnostic criteria. There is a 4-fold variation in the overall estimated standardised prevalence for those aged ≥60 years, from 2.07% (West Sub-Saharan Africa) to 8.5% (Latin America) from systematic review and meta-analysis (studies based on the Diagnostic and Statistical Manual of Mental Disorders, 4th edition [DSM-IV] or 10th revision of the International Statistical Classification of Diseases and Related Health Problems [ICD-10] criteria up to March 2009).[2]

Based on 76 dementia prevalence studies in highly developed areas drawn from mainland China, Hong Kong, and Taiwan between 1980 and 2012 that used the DSM-IV diagnostic criteria, the estimated prevalence of dementia in people aged ≥60 years is 4.6%. The prevalence varies from 5.1% in north to 4.4% in central to 3.9% in south Asia.[3] The prevalence of dementia has been shown to increase with age, approximately doubling every 5 years until around the age of 90 years. Women have a higher prevalence than men across all age-groups of >65 years (Figure 24.1).[3]

The prevalence of dementia increases with population ageing. In Hong Kong, the percentage of community-dwelling people aged ≥60 years with dementia increased from 0.6% in 2000 to 1.1% in 2004 and remained stable until 2008.[4] Females have a higher prevalence of dementia than males because of their longer lifespan. Those who live in institutions also have a higher prevalence of dementia than community-dwelling people. Longer life expectancy and ageing baby boomers will increase the number and percentage of Hong Kong people, especially the oldest old. By combining the community and institutional populations, the estimated number of people aged ≥60 years with dementia will increase from 103 433 in 2009 to 332 688 in 2039, an increase of 222%.[4]

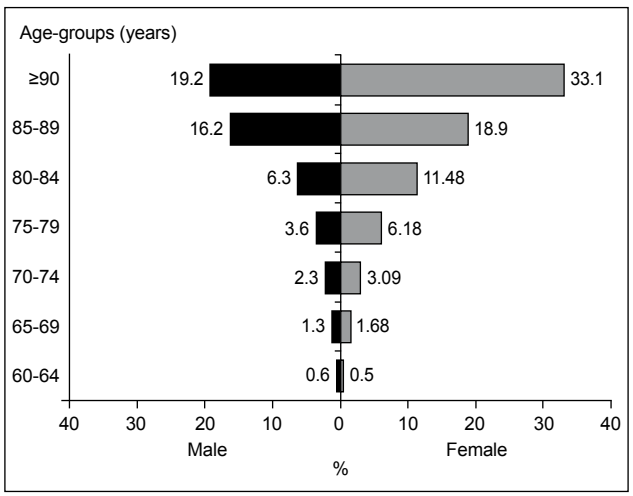

Figure 24.1 Age- and sex-specific prevalence rate of dementia among elderly people in China, Hong Kong, and Taiwan between 1980 and 2012[3]

As the population of Hong Kong ages, dementia is becoming a more common cause of death. Between 2001 and 2016, deaths attributed to dementia (ICD-10: F01-03) increased 392% (from 3.8 to 18.7 deaths per 100 000 population), whereas those attributed to the principal cause of death, malignant neoplasms, increased by only 14% (from 169.9 to 193.3 deaths per 100 000 population).[5] The impact of dementia can be estimated by disability-adjusted life years (DALYs), which was 286 313 DALYs (females 203 262 DALYs, males 83 051 DALYs) for elderly people aged ≥60 years in Hong Kong in 2006. The burden of disease from dementia was disproportionately carried by women. The majority of this burden was due to disability, with years lost due to disability comprising 99% of DALYs. The remaining 1% of the burden was due to years of life lost from dementia.[4]

24.1.3 Aetiology of Dementia

Dementia has a variety of aetiologies. AD is by far the most common, accounting for an estimated two-thirds of all cases of dementia (Table 24.1). In a series of 454 consecutive patients referred to the memory clinic of a regional hospital in Hong Kong from 1999 to 2004, 385 were diagnosed with dementia. Of these, 55.8% had AD dementia, 26.2% had vascular dementia (VaD), 6.0% had other irreversible dementia (e.g. Parkinson's disease, post-concussion), 5.2% had dementia with Lewy bodies, 4.9% had undetermined dementia, and 1.8% had frontotemporal dementia (FTD).[6] Reversible causes were evident in 5.2% and included depression, delirium, anxiety disorder, psychosis, hyponatraemia, and vitamin B_{12} deficiency.

24.1.4 Characteristics of Cognitive Impairment and Diagnostic Criteria of Normal Ageing, Mild Cognitive Impairment and Disease

Age-associated neuropathological changes in the brain occur in normal elderly persons. These include lipofuscin accumulation and neurofibrillary tangles

Table 24.1 Aetiologies of dementia

Degenerative disorders	Cortical dysfunction: Alzheimer's disease, Pick's disease, frontotemporal dementia
Subcortical dysfunction	Progressive supranuclear palsy, Parkinson's disease, Huntington's disease, Lewy body dementia
Vascular dementia	Multi-infarct dementia, strategic infarcts, Binswanger's disease
Toxic and metabolic disorders	Hypothyroidism, chronic renal failure, Wilson's disease, vitamin B_{12} deficiency, carbon monoxide, alcohol, chronic hypoxia, chronic liver failure
Psychological disorders	Depression
Intracranial hypertension or space-occupying lesions	Normal-pressure hydrocephalus, brain tumours, subdural haemorrhage
Cerebral infection	Neurosyphilis, progressive multifocal leukoencephalopathy, human immunodeficiency virus
Others	Head trauma

within neurons, senile plaques, granulovacuolar degeneration, cerebral arteriolar amyloid deposition, and evidence of lacunar infarcts. These, however, do not correlate with the intellectual performance in later life, nor with cerebral atrophy detected on computed tomographic (CT) scan of brain.

Forgetfulness is a common clinical symptom. A systematic approach to the problem and a basic knowledge of cognitive functional changes with ageing is essential. Some aspects of cognitive function remain markedly stable, or even improve, as individuals age. The ability to retrieve and use information that has been acquired throughout a lifetime remains stable across the lifespan. For example, older adults are very good at defining words, answering questions that rely on general knowledge, detecting spelling errors, or carrying out skills related to jobs that they have held for many years. Another important area of preservation is emotion regulation. Older adults seem to improve their mood regulation after the age of 60 years. They are able to rebound more quickly from negative mood states, and appear to focus more on positive information in their environment and to choose activities based on their potential for emotional fulfilment. Other memory domains such as procedural memory (acquisition and use of behavioural skills), perceptual representation (identifying objects in the surrounding world), semantic memory (general knowledge and concept) and working memory (short-term memory) remain intact. Nonetheless the ability to store and manipulate new information may decline with age. Older adults often complain of word-finding difficulties and poor contextual memory. For example, older adults may not recognise someone who they have seen before, or complain of forgetting the place (the context) where they met. Other cognitive functions — including executive functions and reasoning, processing speed, and attention — may also decline from middle age onwards. Psychological testing in elderly people produces diverse results. Standard intelligence tests such as the Wechsler Adult Intelligence Scale applied to cross-sectional studies that compare young with old consistently showed significantly poorer performance by old people, with about 20% loss of score between the ages of 20 and 70 years. This, however, should be interpreted with caution because elderly people in their 70s may have received less education and nutrition during their development compared with the younger adults in their 20s.

Mild cognitive impairment, or MCI (as defined by the National Institute on Aging–Alzheimer's Association [NIA-AA]) or mild neurocognitive disorder (NCD) due to AD (as defined by the 5th revision of the Diagnostic and Statistical Manual of Mental Disorders [DSM-5]) has a 10% per year conversion rate to dementia.[7] The progression, however, is not always linear with time. In a group of 285 cognitively normal subjects with a mean age of 78.9 years, the cognitive state of up to 20% of subjects was found to fluctuate between normal, MCI, and dementia.[8] Subjects who developed MCI and later returned to normal can subsequently progress to dementia. A longitudinal review of cognitive function for patients with MCI is important to avoid missing the diagnosis of dementia conversion. The most notable cognitive deficit for patients with AD is an inability to remember information encountered in the recent past. This deficit extends across different types of encoding tasks (e.g. incidental or intentional; deep or shallow processing) and exists regardless of the stimulus materials (e.g. pictures, words, faces, autobiographical events, emotional stimuli) or the task's retrieval demands (e.g. recall, forced-choice recognition, yes-no recognition). Deficits in episodic memory tend to be the best way of distinguishing people with AD from healthy older adults. The working memory and executive function (on tasks requiring dual-task performance, the ability to flexibly shift attention and to attend to goal-relevant information) are also impaired in patients with AD. In contrast, the semantic memory (general knowledge) is relatively spared in AD until the disease progresses to late stage.

Some cognitive impairment is a natural part of the normal ageing process. Distinguishing normal cognition from MCI, and between MCI and dementia is difficult. New diagnostic criteria for dementia are being developed to address new findings in the pathophysiology, biomarkers, genetics, and neuroimaging techniques of AD. The NIA-AA published new diagnostic guidelines for AD to replace the NINCDS-ADRDA (National Institute of Neurological and Communicative Disorders and Stroke and the Alzheimer's Disease and Related Disorders Association) criteria that have been commonly used in research since 1984.[9-12] In 2013, the American Psychiatric Association released the DSM-5 to replace the DSM-IV that was published in 1994. The new NIA-AA guidelines and the DSM-5 have many similarities. The NIA-AA guidelines, however, have a primary focus on further research directions while the DSM-5 focuses exclusively on clinical diagnosis. Further, the NIA-AA guidelines are for diagnosis of AD, while the DSM-5 includes diagnostic criteria for various forms of dementia. The new NIA-AA guidelines for AD are designed to reflect research results that indicate that AD starts to affect the brain many years before the patient becomes symptomatic. Hence, it supports the notion that dementia can be detected and treated during the preclinical stage. The NIA-AA expert workgroups proposed three phases of AD progression over time — preclinical AD (where the disease is present in the brain but there are no symptoms), MCI due to AD (also called prodromal AD), and dementia caused by AD. Another important change in the criteria is the incorporation of biomarkers into the diagnostic criteria. Two biomarker categories are highlighted — biomarkers that indicate the level of beta-amyloid accumulation in the brain, and biomarkers that indicate injured or degenerating nerve cells in the brain. A summary table of NIAA-AA criteria and guidelines for AD for clinical use is shown in Table 24.2.[12,13]

Dementia is categorised as NCD in the DSM-5. NCDs are defined as being acquired and degenerative, rather than inborn and apparent in childhood. The NCD category is then further divided into mild and major NCD. The DSM-5 details six cognitive domains that may be affected by NCDs — complex attention, executive ability, learning and memory, language, visuoconstructional perceptual ability, and social cognition. Mild NCDs are defined by 'modest cognitive decline' in one or more domains and do not interfere with independence. Major NCDs are defined by 'significant cognitive decline' in one or more domains and do interfere with independence. Table 24.3 shows the comparison of DSM-IV and DSM-5 criteria for dementia (or major NCD). Following the determination of a mild or major NCD, the condition is further specified as due to AD, frontotemporal NCD, Lewy bodies, vascular, traumatic brain injury, Parkinson's disease, human immunodeficiency virus infection, Huntington's disease, prion disease, another medical condition, or multiple aetiologies (Table 24.4). Therefore the DSM-5 expands the scope to a spectrum of neurocognitive degenerative disorders that may affect younger adults. Further research is needed to show the effect of change in DSM-5 on the management of dementia or major NCD.

Table 24.2 NIA-AA new diagnostic criteria and guidelines for AD for clinical use[12,13]

All-cause dementia

1. Interferes with ability to function at work or activities
2. Represents a decline from previous levels of function
3. Not due to delirium or psychiatric disorder
4. Impairment diagnosed through (a) history and (b) objective cognitive assessment (bedside or neuropsychological testing)
5. Cognitive impairment in at least two domains:
   - Ability to acquire — remember new information
   - Impaired reasoning — handling complex tasks (poor judgement)
   - Impaired language
   - Personality change
   - Visuospatial (impaired ability to recognise faces, objects)

Dementia due to AD

1. Probable AD dementia (core clinical criteria) — includes meeting the clinical criteria for all-cause dementia along with insidious onset; clear history of worsening of cognition by report or observation; and initial and most prominent cognitive deficits include amnestic presentation and / or deficits in language presentation, visuospatial presentation, and executive function
2. Possible AD dementia — diagnosis for patients who meet core clinical criteria but exhibit an atypical course of cognitive decline or mixed aetiological presentation

MCI due to AD

Core clinical criteria — clinical and cognitive assessments that establish concern of change in cognition over time; impairment in ≥1 cognitive domain (scores on cognitive tests for individuals with MCI may be 1-1.5 standard deviations below the mean for their age and education-matched peers on culturally appropriate normative data); preservation of independence in functional abilities; not demented, and aetiology of MCI consistent with AD, including where relevant, AD genetic factors

Abbreviations: AD = Alzheimer's disease; MCI = mild cognitive impairment; NIA-AA = National Institute on Aging–Alzheimer's Association

Table 24.3 A comparison of DSM-IV and DSM-5 criteria for dementia

DSM-IV criteria for dementia	DSM-5 criteria for major NCD (formerly dementia)
A1. Memory impairment A2. At least one of the following: - Aphasia - Apraxia - Agnosia - Disturbance in executive functioning	A. Evidence of significant cognitive decline from a previous level of performance in one or more cognitive domains: - Complex attention - Executive ability - Learning and memory - Language - Visuoconstructional perceptual ability - Social cognition
B. The cognitive deficits in criteria A1 and A2 each cause significant impairment in social or occupational functioning and represent a significant decline from a previous level of functioning	B. The cognitive deficits interfere with independence in everyday activities. At a minimum, assistance should be required with complex instrumental activities of daily living, such as paying bills or managing medications
C. The deficits do not occur exclusively during the course of a delirium	C. The cognitive deficits do not occur exclusively in the context of a delirium
	D. The cognitive deficits are not better explained by another mental disorder (e.g. major depressive disorder, schizophrenia)

Abbreviations: DSM = Diagnostic and Statistical Manual of Mental Disorders; NCD = neurocognitive disorder

Table 24.4 DSM-5 criteria for subtypes of NCDs

AD NCD	The following 3 conditions are met: • Clear evidence of decline of memory and learning AND at least one other cognitive domain • Progressive, gradual decline • Not a mixed aetiology (e.g. cerebrovascular disease or other disease affecting cognition) Or, alternatively, a causative AD gene is found
Frontotemporal NCD	Relative sparing of learning memory, and criteria for behavioural variant or language variant met • Behavioural variant o Decline in social cognition and / or executive abilities o Three or more of the following: apathy, loss of empathy, perseveration, disinhibition, hyperorality, or dietary changes • Language (primary progressive aphasia): language decline o Non-fluent type: word pronunciation disorder o Semantic subtype: loss of single-word meaning, anomia
Lewy bodies NCD	Two or more of the following: • Fluctuating variations in attention alertness • Well-formed visual hallucinations • Parkinsonism develops AFTER cognitive change begins • REM behavioural sleep disorder (acts out dreams) • Severe neuroleptic sensitivity: catatonia, muscle rigidity
Vascular NCD	There is evidence of cerebrovascular disease from neuroimaging of history and examination One of the following: • Onset temporally related to a stroke or strokes • Prominent loss of executive function and loss of speed of processing (bradyphrenia)
NCD due to multiple aetiologies	Evidence by history, examination, tests that the NCD is due to >1 of the other established aetiologies One of the causes is NOT substances

Abbreviations: AD = Alzheimer's disease; DSM-5 = Diagnostic and Statistical Manual of Mental Disorders, 5th revision; NCD = neurocognitive disorder; REM = rapid eye movement

24.1.5 Evaluation of Cognitive Impairment and Dementia

The aim of the clinical assessment is to establish the diagnosis, detect the possible aetiology, and formulate a management plan. A detailed history from the patient and more often from the caregiver is the most important determinant of an accurate diagnosis. The patient and the caregivers are often uncertain about the onset of symptoms because the appearance of dementia is insidious. The diagnosis rests on a history of cognitive impairment notable over several months or years and the progressive nature of the deficit. Memory impairment is a common symptom. Patients with dementia may have difficulties in handling complex tasks (e.g. balancing a cheque book), reasoning (e.g. unable to cope with unexpected events), spatial ability and orientation (e.g. getting lost in familiar places), language (e.g. word finding), and / or disturbed behaviour or perception. Enquiry about reversible causes of dementia is useful. These include risk factors for cardiovascular disease such as high blood pressure and diabetes, alcohol or drugs intake (e.g. analgesics, anticholinergics, psychotropic medication, and

sedatives) and presence of obstructive sleep apnoea or any concurrent neurological disease. The diagnosis of dementia must be distinguished from delirium and depression, also known as pseudodementia. Patients with depression are more likely to complain about memory loss than those with dementia. The latter are frequently brought to physicians by their caregivers, while depressed patients often present by themselves. The patient with depression may have signs of psychomotor slowing and make a poor effort on testing, while those with dementia often try hard but respond with incorrect answers. Enquiry about social support is essential when planning further management. Input from other disciplines such as an occupational therapist, physiotherapist, or social worker will help to address the multidimensional needs of the patient and the caregiver.

Objective testing tools such as the Mini-Mental State Examination (MMSE) [see Chapter 2] can only indicate the level of cognitive functioning at the time of testing. It may be used as a screening tool, but by itself cannot be used to diagnose dementia. For diagnosis, it is important to follow standard diagnostic criteria such as DSM-5 (Table 24.4) but not all symptoms are present in the early stage of the disease. Global cognitive impairment is the eventual fate but cognitive impairment is usually patchy in the early stage. The Chinese version of the MMSE is a brief and validated test based on a 30-item test and covers orientation, registration, attention and calculation, recall, language, and praxis. It serves as a screening instrument to detect cognitive impairment in AD (see Chapter 2). Its limitations include insensitivity in detection of frontal lobe dysfunction and monitoring of cognitive change with treatment in patients with AD. Its test-retest and inter-rater reliability are inadequate for detecting small changes in cognition, making this test inappropriate for assessing treatment response in AD.[14] The MMSE has not been used as a primary outcome measure in any blinded trial of cholinesterase inhibitors (ChEIs) but sometimes has been used as a secondary outcome measure. The results of these clinical trials showed that the drug-placebo differences on the MMSE range from 0.68 to 1.36 points over 12-26 weeks. This difference is smaller than the average measurement error of the MMSE score of 2.8 points, suggesting that the test is insensitive for monitoring the change induced by ChEIs.[15] The MMSE should therefore be used as a screening tool for initial evaluation of cognitive impairment, not for serial monitoring of the response to ChEIs. Another popular simple screening test is the clock face drawing and copying test (see Chapter 2) that is sensitive in detecting impairment in orientation, conceptualisation of time, visual spatial organisation, hemi-neglect, and agraphia. Since depression is an important reversible cause of pseudodementia, the Chinese version of the Geriatric Depression Scale (see Table 25.7 of Chapter 25) should be used for screening. A cut-off point of ≥8 may suggest depression in an elderly person.

During history taking, the severity of the dementia can often be assessed at the same time. A useful global staging scale such as Clinical Dementia Rating scale (Table 24.5) can be used. This will score only for decline from previous usual level due to cognitive loss, not for impairment arising from other factors.

Physical examination may be completely normal, especially in early AD. Some aspects deserve attention. It is important to assess the patient's vision and hearing. Diagnosis of dementia may be mistakenly made because of optical illusions or communication problems. The patient's mood should also be noted. Atrial fibrillation, high blood pressure, cardiac disease, and carotid bruit are important physical signs that may suggest a vascular cause for the dementia. Examination of the central nervous system can reveal much useful information. The presence of focal neurological deficits or exaggerated reflexes may indicate VaD or mixed dementia. Gait disturbance such as apraxic gait occurs early in normal-pressure hydrocephalus. The presence of Parkinsonism features may narrow the aetiology to Parkinson's disease, Parkinsonism plus syndrome such as progressive supranuclear palsy, or cerebral Lewy body disease. Primitive reflexes such as grasp reflex, palmomental reflex and rooting reflex may occur in frontal lobe dementia, VaD and in very late AD. Other higher cognitive dysfunctions such as early involvement of speech and prominent behavioural symptoms may direct the diagnosis to frontal lobar degeneration.

Investigations are performed mainly to detect the reversible causes in dementia (Table 24.6[16]). The American Academy of Neurology recommends screening for vitamin B_{12} deficiency and hypothyroidism in patients with dementia.[16]

24.1.6 Roles of Neuroimaging and Biomarkers in Diagnosis of Dementia

Advanced neuroimaging or measurement of biomarkers is not required in all persons with cognitive impairment. The NIA-AA advocates incorporating biomarkers / advanced neuroimaging into the diagnostic criteria for preclinical AD. This is a new conceptual phase to encompass individuals with pathophysiological changes in the brain who are cognitively normal. The tests include measurement of amyloid-β (Aβ) accumulation (cerebrospinal fluid [CSF] Aβ42 and positron emission tomography [PET] imaging with amyloid tracer) and measurement of neuronal injury (CSF tau / p-tau, fluorodeoxyglucose

Table 24.5 Clinical Dementia Rating Scale (CDR)

Score	Healthy CDR 0	Very mild CDR 0.5	Mild CDR 1	Moderate CDR 2	Severe CDR 3
Memory	No memory loss or slight inconsistent forgetfulness	Consistent slight forgetfulness; partial recollection of events; 'benign' forgetfulness	Moderate memory loss; more marked for recent events; defect interferes with everyday activities	Severe memory loss; only highly learned material retained; new material rapidly lost	Severe memory loss, only fragments remain
Orientation	Fully orientated	Fully orientated except for slight difficulty with time relationships	Moderate difficulty with time relationships; orientated for place at examination; may have geographic disorientation elsewhere	Severe difficulty with time relationships; usually disorientated in time, often to place	Orientated to person only
Judgement and problem solving	Solves everyday problems and business affairs well; good judgement in relation to past performance	Slight impairment in solving problems, similarities, differences	Moderate difficulty in handling problems, similarities, differences; social judgement usually maintained	Severely impaired in handling problems, similarities, differences; social judgement usually impaired	Unable to make judgements or solve problems
Community affairs	Independent function at usual level in job, shopping, volunteer, and social groups	Slight impairment in these activities	Unable to function independently at these activities although may still be engaged in some; appears normal on casual inspection	*No pretence of independent function outside home* Appears well enough to be taken to functions outside a family home	*No pretense of independent function outside home* Appears too ill to be taken to functions outside a family home
Home and hobbies	Life at home, hobbies, intellectual interests well maintained	Life at home, hobbies, intellectual interests slightly impaired	Mild but definite impairment of function at home; more difficult chores abandoned; more complicated hobbies and interests abandoned	Only simple chores preserved; very restricted interests, poorly maintained	No significant function in home
Personal care	Fully capable of self-care	Fully capable of self-care	Needs prompting	Requires assistance in dressing, hygiene, keeping of personal effects	Requires much help with personal care; frequent incontinence

Table 24.6 Investigations for dementia patients* [16]

Routine	• Complete blood cell count • Glucose • Urea and electrolytes, calcium • Liver function tests • Thyroid function • Vitamin B_{12} and folate • Chest X-ray • Electrocardiography • Non-contrast CT scan of brain
Not routinely done	• HIV serology • Electroencephalography • Sleep study • Neuropsychological testing • Linear or volumetric MRI, CT measurement • Functional brain imaging e.g. functional MRI, FDG-PET, SPECT • Amyloid PET scan • Genetic testing or other biomarkers • Lumbar puncture (unless presence of metastatic cancer, suspicion of CNS infection, reactive serum syphilis serology, hydrocephalus, age <55 years, rapidly progressive or unusual dementia, immunosuppression, suspicion of CNS vasculitis)

Abbreviations: CNS = central nervous system; CT = computed tomography; FDG-PET = fluorodeoxyglucose positron emission tomography; HIV = human immunodeficiency virus; MRI = magnetic resonance imaging; SPECT = single-photon emission computed tomography

* Genetic testing is usually not required in the clinical management of dementia, except in familial early-onset dementia; biomarkers may have a potential role in the early diagnosis of dementia in its preclinical stages

PET [FDG-PET] / functional magnetic resonance imaging [MRI], and structural MRI). Positive findings on these tests in patients with dementia suggest higher certainty of an AD pathological process but otherwise do not establish an AD diagnosis. In MCI patients, the presence of biomarkers also increases the probability

of MCI converting to AD. Reduction in hippocampal volume has been found in AD patients.[17] However, an age-related reduction in hippocampal volume is also noted in AD patients. It is unclear whether measurement of hippocampal volume adds to the accuracy of the diagnosis over the clinical assessment alone. Functional brain imaging with FDG-PET, functional MRI, perfusion MRI, or perfusion single-photon emission computed tomography reveals distinct regions of low metabolism and hypoperfusion in AD. These areas include the hippocampus, the precuneus (mesial parietal lobes) and the lateral parietotemporal cortex. Amyloid PET tracers (e.g. with florbetapir) measure the amyloid lesion burden in the brain. It can be positive in AD, but also Lewy body disease and in about 20% of normal elderly people. It increases the certainty that clinically assessed dementia is due to AD or FTD. In addition, measurement of biomarkers such as CSF Aβ42 and CSF t-tau / p-tau helps differentiate AD from other dementias including FTD and Lewy body dementia. For younger-onset dementia and non-AD, functional neuroimaging and clinico-radiological correlation helps in reaching a definitive diagnosis.[18-20]

24.1.7 Management of Cognitive and Non-cognitive Symptoms of Dementia

The management of a patient with dementia is highly individualised, focused on the needs of the patient and the caregiver. Patients with mild-to-moderate dementia often present with cognitive symptoms. Non-cognitive or behavioural and psychological symptoms usually occur in moderate-to-late dementia. In general, therapy can be divided into non-pharmacological and pharmacological strategies that target the cognitive and non-cognitive symptoms. The primary goals of treatment are to improve quality of life and maximise functional ability by improving cognition, mood, and behaviour. The principles of treatment are to:

- identify and treat comorbid physical illnesses such as hypertension, diabetes, and pain syndrome;
- exclude and treat coexisting delirium;
- set realistic goals according to the patient's functional ability and social support;
- maximise and maintain cognitive and functional performance;
- identify and treat any abnormal behaviours; and
- limit the use of psychotropic medication

24.1.7.1 Non-pharmacological Interventions to Improve Cognitive and Functional Performance

Since cognitive impairment is frequently exacerbated by medications, use of psychotropic drugs or hypnotics should be minimised whenever possible. Safety issues such as those related to driving and getting lost, and end-of-life issues such as advance directives may be addressed after the diagnosis is made. To improve the patient's functional performance, behavioural modification such as scheduled toileting for urinary incontinence, graded assistance, positive reinforcement, and environmental modifications can be implemented, e.g. dim lighting and sounds of nature may enhance eating behaviour. Multi-strategy group therapies aimed at cognitive stimulation training including reality orientation, reminiscence therapy, multisensory stimulation, exercise, and music therapy have been shown to benefit cognition in patients with mild-to-moderate dementia over and above any medication effects.[21] Training of the caregiver in strategies to manage daily activities, home support services, support groups, day care centre, respite care unit, and voluntary community agencies such as the Hong Kong Alzheimer's Disease Association[22] may help reduce caregiver stress and maintain the patient in the community.

24.1.7.2 Pharmacological Treatment in Alzheimer's Disease

There is no convincing evidence that pharmacological treatment can prevent dementia. Most data on preventing dementia come from observational studies. Prospective studies and randomised controlled trials have not shown an overall benefit from ChEIs, non-steroidal anti-inflammatory drugs, statins, oestrogen replacement, *Ginkgo biloba*, or omega-3 fatty acids in preventing dementia. High-dose vitamin E supplementation (2000 IU per day) has been inconsistently associated with less functional decline (3.15 units in the Alzheimer's Disease Cooperative Study–Activities of Daily Living [ADCS-ADL] inventory) in a group of patients with mild-to-moderate AD after a mean follow-up of 2 years.[23]

Two types of pharmacological treatment are available for the treatment of AD — ChEIs (donepezil, rivastigmine, and galantamine) and a N-methyl-D-aspartate (NMDA) receptor antagonist (memantine) [Table 24.7]. Patients with AD have reduced cerebral cholinergic function. ChEI increases cholinergic transmission by inhibiting cholinesterase at the synaptic cleft. Excitation of NMDA receptors by glutamate is involved in the processes of learning and memory. Excessive NMDA stimulation can induce excitotoxicity. Hence, memantine, by blocking NMDA receptors, can protect neuron damage and result in cognitive improvement.

There is convincing evidence that ChEIs have efficacy in cognition and global functioning at all stages of AD. ChEIs are also useful in mixed dementia (AD and VaD) and Parkinson dementia. These drugs are not indicated for pure VaD or for treating

Table 24.7 Pharmacological treatment of cognitive dysfunction

Drugs	Initial dosage	Recommended final dose	Common side-effect	Comment
Donepezil (Aricept)	5 mg daily for 4-6 weeks	10 mg daily	Nausea, vomiting, diarrhoea, insomnia, bradycardia	
Rivastigmine (Exelon)	1.5 mg bd, increase by 1.5 mg per dose every 2 weeks	6 mg bd	Same, weight loss	Nausea is reduced when given with meals
Rivastigmine patch	4.6 mg/24 hours for 4 weeks	9.5 mg/24 hours	Same, weight loss	Appears to have less nausea
Galantamine (Reminyl)	4 mg bd, increase by 4 mg per dose every 4 weeks	8-12 mg bd	Same, weight loss	Nausea is reduced when given with meals
Memantine	5 mg daily, increase by 5 mg per dose/week	10 mg bd	Dizziness, hypotension, constipation	

Abbreviation: bd = twice daily

neuropsychiatric symptoms. The choice between the three ChEIs is largely based upon cost, individual patient tolerability, and physician experience as there is no clear evidence that one ChEI is more efficacious than the other. Donepezil has relatively little peripheral anticholinesterase activity and appears to have less gastrointestinal side-effects compared with the other two ChEIs. This, combined with its once-daily dosing, has made it a popular drug of choice. The average benefit of ChEIs in AD patients is a small improvement in cognition and activities of daily living. An average improvement in the Alzheimer's Disease Assessment Scale–Cognitive Subscale (ADAS-Cog) of about 2-3 out of 70 points over 6 months is achieved with ChEIs compared with an average decline of about 5-6 points per year in a control group. It is suggested that aspects of both language and memory contribute to the improvement in cognition recorded in clinical trials. A Cochrane systemic review of 10 randomised, double-blind, placebo controlled trials demonstrated that treatment of people with AD of all severities with ChEIs produced improvements in cognitive function, on average -2.7 points, in the midrange of the 70-point ADAS-Cog.[24] A meta-analysis consisting of 96 publications from 1986 to 2006 concluded that improvement in cognitive measures (weighted mean difference [WMD] for ADAS-Cog: -2.83 points in donepezil; -3.91 points in rivastigmine; -2.46 points in galantamine) and global assessment of dementia (WMD for Clinician's Interview-Based Impression of Change Plus Caregiver Input [CIBIC-Plus]; -0.45 points in donepezil; -0.36 points in rivastigmine; WMD for Clinical Dementia Rating Scale Sum of Boxes score: -0.44 points in donepezil) was associated with the use of ChEIs. A change of >4 points in ADAS-Cog and any change in the CIBIC-Plus are regarded as clinically significant. Hence, the beneficial outcomes in cognition associated with use of ChEIs are clinically marginal, although statistically significant in patients with AD of all severity levels.[25] Despite this, the response to ChEIs may be quite variable: as many as 30-50% of patients show no observable benefit although up to 20% show a greater than average response (at least 7-point improvement in ADAS-Cog, an equivalent to reversing the disease process by approximately 1 year).[26] In the patient with advanced AD, the Severe Impairment Battery (SIB), a neuropsychological test used to assess cognitive functioning in patients with advanced dementia, was also shown to have less decline with donepezil.[27] There is no clinical predictor to identify who will respond favourably to ChEIs. It is recommended that they are administered at a low dose for 4-8 weeks to monitor the patients' tolerance, then increased gradually to the maximum tolerable dose. An interval review should be carried out 3-6 months after commencement of treatment to reassess the cognitive and global functioning response. Most blinded trials of ChEIs have been for 6 months, few have lasted longer than a year. Additional open-label observational studies have suggested that patients treated with donepezil for 4.9 years declined significantly more slowly than anticipated based on rates of change observed in those who received placebo during the blinded portions of the studies.[28] ChEIs are therefore a symptomatic treatment but not disease-modifying. They can be continued indefinitely until the patient progresses to AD of advanced stage. If ChEIs are to be discontinued, they should be tapered over 2-3 weeks to avoid abrupt worsening of cognitive function.

A variety of secondary outcome measures have been investigated in clinical trials of ChEIs, which reported small improvement in ADL, behaviour, and delay to nursing home placement with the use of ChEIs, albeit different trials used different assessment instruments with different ChEIs. In the absence of direct comparative trials, it is difficult to draw a conclusion about the effects of ChEIs on these secondary outcomes. In clinical practice, ADL rarely improve in the course of dementia with ChEIs and the drug effects on ADL or functioning are likely

revealed through reduction in the rate of decline in cognition.

Memantine was approved by the US Food and Drug Administration in 2003 for the symptomatic treatment of moderate-to-severe AD. It is generally well tolerated. A Cochrane systematic review of double-blind, placebo-controlled, randomised trials of memantine showed a small beneficial effect on cognition (SIB: 4.12 points on the 100-point scale) in patients with moderate-to-severe AD in two 6-month studies.[29] In another 6-month trial, memantine was associated with an improvement in cognition (ADAS-Cog: 1.9 points) and global assessment of dementia (CIBIC-Plus: 0.3).[29] Nonetheless memantine appears to be ineffective in mild AD. A systematic review of three clinical trials did not find statistically significant beneficial effect on cognition (ADAS-Cog: -0.17; 95% confidence interval [CI], -1.60 to 1.26) or global assessment (CIBIC-Plus: -0.09; 95% CI, -0.30 to 0.12) in patients with mild AD.[30] Use of memantine is associated with an improvement in secondary outcome measures of ADL (ADCS-ADL: 1.39; 95% CI, 0.39-2.39) and behaviour (Neuropsychiatric Inventory [NPI] -3.19; 95% CI, -5.09 to -1.29).[29] Of note, patients who took memantine appeared to be less likely to become agitated.

The benefits of memantine as an add-on therapy for patients with moderate-to-severe AD who have been on stable-dose donepezil or rivastigmine has been evaluated.[31-34] The results for cognition, global assessment, ADL functioning, and behaviour were mixed. In a clinical trial that compared different strategies (continue donepezil, discontinue donepezil, discontinue donepezil and start memantine, or continue donepezil and start memantine) in a group of 295 community-dwelling patients with moderate-to-severe AD (MMSE score, 5-13), the outcome measures for cognition (MMSE) and ADL (Bristol Activities of Daily Living Scale) were of no difference in the donepezil group, memantine group, or in the presence or absence of the other,[34] suggesting that add-on memantine therapy offers no additional benefit in patients who fail to respond to donepezil.

24.1.7.3 Pharmacological Treatment in VaD

Patients with cognitive impairment due to cerebrovascular pathology should be screened and treated for vascular risk factors with antihypertensive drugs, hypoglycaemic drugs, antiplatelet drugs, and statins.

There is some degree of overlap in the neuropathology of AD and VaD. Deficient cholinergic neurotransmission has been postulated to contribute to the cognitive impairment of VaD. While ChEIs are not endorsed for use in patients with pure VaD, they are often used in part because of the known association of AD with VaD. In a Cochrane systematic review of two large-scale, randomised, double-blind, parallel-group controlled trials consisting of 1219 patients with mild-to-moderate cognitive decline due to probable or possible VaD, donepezil (5 mg or 10 mg/day) was associated with an improvement in cognitive function, global function, ADL, and instrumental ADL.[35] The benefits were confirmed in a meta-analysis. Nonetheless donepezil efficacy for cognitive function measures was only marginally clinically significant.[25] The secondary outcome measurement such as ADL was statistically insignificant. There were insufficient data for analyses of cognitive and non-cognitive outcomes for rivastigmine or galantamine in the meta-analysis.

The effects of memantine in VaD have been evaluated in the Cochrane systematic review.[29] In mild-to-moderate VaD, two 6-month studies showed that memantine improved cognitive function (1.85 ADAS-Cog points; 95% CI, 0.88-2.83 points, p=0.0002), and behaviour (0.84 NPI points; 95% CI, 0.06-0.91 points, p=0.03) but this was not supported by clinical global measures (CIBIC-Plus). In patients with VaD of all severities taking memantine, agitation appeared to be less likely to develop. Another meta-analysis that included clinical trials evaluating behaviour outcomes, however, failed to show a statistically significant result. While the demonstrated efficacy is limited, many clinicians may use memantine for patients with VaD because of a lack of other proven therapy.

24.1.7.4 General Approach to the Non-cognitive Symptoms of Dementia

Behavioural and psychological symptoms of dementia (BPSD) affect up to 90% of patients with dementia at some stage in their illness. These symptoms include agitation, aggression, delusions, hallucinations, wandering, depression, and sleep disturbances. BPSD helps in the initial diagnostic process, e.g. depressive symptoms occur in VaD and cerebral Lewy body disease, apathy in FTD, and paranoid delusions in AD. BPSD are more likely to occur in the later stage of dementia. They can be distressing, increase the likelihood of moving to a nursing home, and contribute to the cost of caring for demented patients. To help the caregiver or informant to characterise the BPSD and tackle the problem, the following 'ABC' approach can be adopted.

- Antecedent: to identify some triggering causes for the BPSD, e.g. pain or urinary urgency may present as agitation. Questions about environmental triggers should also be explored. Look for what happens before the event: who, how, what, where, when, and why the BPSD occurs.
- Behaviour: can be psychological such as delusions,

hallucinations, depressed mood, sleeplessness and anxiety; or behavioural such as physical aggression, wandering, and restlessness.
- Consequences: to explore the relationship between the patient and the caregivers, to help understand the problems the caregivers are exposed to during the caring process. Moreover, the outcome and the effect of both non-pharmacological and pharmacological interventions should be regularly evaluated.

The appropriate communication skills and attitude to dementia patients can be summarised in the mnemonic "RESPECT":
- **R**ecognise
- **E**mpathise, avoid "no"
- **S**upport
- **P**revent
- **E**nhance
- **C**are and comfort
- **T**ake time

Non-pharmacological Intervention for BPSD

Non-pharmacological intervention must be tried first before pharmacological treatment because it is more acceptable to the patient and there are no pharmacological adverse effects. Music therapy has been shown to reduce agitation, aggression, and mood disturbance under various conditions including eating and bathing. Video tapes of family members reduces verbally disruptive behaviour more than music. Bright light therapy appears to reduce aggression, agitation, and diverse behavioural disturbances. Walking and light exercise appear to reduce wandering, aggression, and agitation. Pet therapy has been reported to improve socialisation. Aromatherapy, exercise training, massage, and touch therapy have also demonstrated some benefits for BPSD.

Pharmacological Intervention for BPSD

If non-pharmacological treatment of potential causes of BPSD is unsuccessful, pharmacological therapy can be considered for BPSD that cause extreme distress or pose an imminent threat of harm to self or others. It should be prescribed at the lowest effective dose for the shortest possible duration, taking into account the significant risks compared with potential benefits.[36,37] Few randomised controlled trials have addressed the optimal psychopharmacological agents for the treatment of behavioural problems in dementia. Recommendations are based on small trials. In a systematic review of double-blind, placebo-controlled, randomised controlled trials or meta-analyses of drug therapy studies published from 1966 to 2004, pharmacological therapies were found to be not very effective for the management of BPSD.[38] No difference among typical antipsychotics (haloperidol, thioridazine, thiothixene, chlorpromazine, trifluoperazine, and acetophenazine) was found. The efficacy was small at best and adverse effects were common. There may be a slight benefit from haloperidol with aggression but it is unclear if this benefit outweighs the adverse effects, such as extrapyramidal symptoms and sedation. To avoid side-effects, typical neuropsychotics should be commenced in a 'start low and go slow' manner and stopped when they are no longer indicated. There have been no published randomised controlled trials to compare the efficacy of typical and atypical antipsychotics. Among atypical antipsychotics studied (olanzapine, risperidone), somnolence, injury, and falls were the common adverse drug events. Olanzapine and risperidone might be associated with increased risks of stroke and mortality. The pooled incidence of cerebrovascular events in patients taking olanzapine or risperidone has been reported to be 1.3-3.3%. Thus they should be used with caution in patients with cardiovascular risk factors. Because of the increased risk of stroke and greater rate of cognitive decline and mortality in dementia persons with the use of neuroleptics (typical or atypical), the 2015 Beers Criteria of the American Geriatrics Society recommend avoiding antipsychotics for behavioural problems of dementia or delirium unless non-pharmacological options (e.g., behavioural interventions) have failed or are not possible and the older adult is threatening substantial harm to self or others.[39] Online non-pharmacological strategies for reducing antipsychotic use are available for the settings of nursing home (http://www.nursinghometoolkit.com) and hospital (http://www.hospitalelderlifeprogram.org). Drugs with strong anticholinergic properties, such as tricyclic antidepressants, should be avoided in patients with dementia. Selective serotonin reuptake inhibitors (SSRIs) are preferred. SSRIs except citalopram have been found to have no efficacy for treatment of BPSD other than depression. Data on mood stabilisers (carbamazepine, divalproex sodium, sodium valproate) in the treatment of agitation are conflicting.

24.2 Delirium

24.2.1 Introduction

With the ageing population and the increasingly earlier detection of cognitive decline, there is a great need for better service planning to create a dementia-friendly inpatient environment and to prevent the occurrence and complications of delirium — a common, costly, and debilitating condition faced by acute elderly inpatients. Some of the large hurdles are the clinicians' lack of awareness of the presence and implications of delirium, the lack of interest in managing these patients, and the poor treatment of the condition resulting in further prolongation and worsening in severity of delirium through

inappropriate prescription of physical and chemical restraints.

24.2.2 Epidemiology, Costs, and Mortality

To date there is a lack of local epidemiological data in this area. Delirium is estimated to occur in 30-60% of geriatric admissions to acute medical wards.[40] In the US, the inpatient costs attributable to delirium and its complications are estimated to be US$40-150 billion per year.[41,42] Increased mortality is significantly associated with delirium. A study has shown that delirium on admission is associated with a 5-fold increased risk of mortality at 6 months post discharge from acute care.[43-45]

24.2.3 Relationship with Geriatric Giants

The high hospital costs and mortality induced from the presence of delirium are caused by increased length of hospital stay and the complications arising from delirium. The prolonged hospital stay further translates into various geriatric giants that are closely related to delirium.[46]

The use of physical restraints will result in 'incontinence', 'instability', and 'immobility' because patients are not allowed to move freely and excrete in their usual way. Meanwhile, physical restraints also do not prevent 'falls' effectively as patients get even more agitated and become prone to risky behaviour. Pharmacological treatment for delirium can be regarded as a form of 'iatrogenesis', exposing patients to the adverse effects of extra medications, including psychoactive drugs and sedatives. These agents may further perpetuate delirium. Persistent delirium often causes 'deconditioning', resulting in the development of sarcopenia and decubitus ulcers.

Importantly, delirium and its relationship with cognitive impairment and dementia have been widely studied.[47,48] Dementia is the most common and strongest risk factor for the development of delirium. The first incidence of delirium in an elderly individual implies an underlying decline in brain reserve as well as the development of progressive cognitive impairment in the near future.[47,49,50] Delirium also has the tendency to accelerate cognitive impairment each time it occurs.

With all the above inter-relationships of delirium with other geriatric giants, it is not difficult to understand the degree of functional decline and the higher chance of a delirious elderly individual being discharged from hospital to an institution.[51-53]

24.2.3.1 Risk Factors

Delirium is usually multifactorial, although one single factor can lead to delirium in a vulnerable elderly individual. Inouye et al[54] have proposed the multifactorial model of delirium, where the development of delirium is dependent on complex inter-relationships between vulnerable patients with several predisposing factors and exposure to noxious insults or precipitating factors. The clinical implication is that addressing a single risk factor may not be able to resolve the delirium and that a multicomponent approach will be more effective for both prevention and treatment.

The leading 'predisposing' factors in acute medical wards are dementia or cognitive impairment, functional impairment, visual impairment, history of alcohol misuse, and advanced age (>70 years). Comorbidity burden or presence of specific comorbidities, including stroke and depression, are associated with an increased risk in all populations. While 'precipitating' factors vary across populations, drugs play an important role, especially polypharmacy and the use of psychoactive drugs. Use of physical restraints was also one of the leading factors in medical inpatients. Various abnormal laboratory results, including electrolyte disturbances, impaired glycaemic control, and acute kidney injury are risk factors in all populations.[40]

Elderly people are more susceptible to drug-induced delirium due to changes to pharmacodynamics and pharmacokinetics with age. The increase in total body fat and decrease in lean body mass and water can result in a prolonged half-life of lipid-soluble agents. The reduced albumin levels and creatinine clearance contribute to the larger volume of distribution and raised free drug levels. Comorbidities such as dementia and stroke will result in impaired integrity of the blood-brain barrier, hence allowing more potential toxic agents to enter the brain.

24.2.4 Pathophysiology

Much knowledge has been gained over the years about the underlying pathophysiology of delirium.[55] Each delirium episode is probably represented by a unique cascade of contributing components.[40] As a result, a single cause or mechanism for delirium onset is difficult to identify. More evidence, however, suggests that several different sets of interacting biological factors result in disruption of the neuronal networks in the brain, leading to acute cognitive dysfunction.

Some of the leading mechanisms being proposed to contribute to delirium are neurotransmitters, inflammation, physiological stressors, metabolic derangements, and electrolyte disorders. Many 'extrinsic' factors interfere directly with neurotransmission or cellular metabolism, including drugs, other 'biological' factors such as

hyperadrenalism, electrolyte disturbances, hypoxia and hypercapnia, and impaired glucose oxidation.

The key neurotransmitters implicated are acetylcholine deficiency and dopamine excess, and correlate with the adverse effects of anticholinergic or dopaminergic drugs.

24.2.5 Evaluation of Delirium

24.2.5.1 Subtypes of Delirium

In general, there are three psychomotor subtypes of delirium. The hyperactive type is characterised by agitation and hypervigilance. The hypoactive type demonstrates lethargy, with a markedly decreased level of motor activity, and is most often underdiagnosed. The mixed type has features from both ends of the spectrum during different times of the same episode.

24.2.5.2 Detection for Delirium

Delirium is fundamentally a clinical diagnosis.[56] History from family members, carers, and nursing staff is extremely important in making the diagnosis. Information from them helps distinguish delirium from the usual BPSD. If no history can be obtained, the patient should be presumed to be delirious until proven otherwise, especially in high-risk elderly groups with advanced age and multiple comorbidities.

The most widely validated instrument for screening and identification of delirium in the acute geriatric care setting is the Confusion Assessment Method (CAM).[57,58] Key CAM components are (1) an acute onset and fluctuating course of symptoms plus (2) inattention, together with either (3) disorganised thinking or (4) altered level of consciousness (Table 24.8). The updated reference standard diagnostic criteria are stated in the DSM-5.

For clinical and research purposes, the bedside friendly CAM is quick and suffices for daily practice, offering up to 95% sensitivity (95% CI, 55-100%) and 95% specificity (95% CI, 72-99%) if performed by geriatricians.[56,59,60]

Table 24.8 The Confusion Assessment Method (CAM)

The CAM instrument

1. **[Acute onset]** Is there evidence of an acute change in mental status from the patient's baseline?

2A. **[Inattention]** Did the patient have difficulty focusing attention, e.g. being easily distractible, or having difficulty keeping track of what was being said?

2B. **(If present or abnormal)** Did this behaviour fluctuate during the interview, that is, tend to come and go or increase and decrease in severity?

3. **[Disorganised thinking]** Was the patient's thinking disorganised or incoherent, such as rambling or irrelevant conversation, unclear or illogical flow of ideas, or unpredictable switching from subject to subject?

4. **[Altered level of consciousness]** Overall, how would you rate this patient's level of consciousness? (Alert [normal]; vigilant [hyperalert, overly sensitive to environmental stimuli, startled very easily]; lethargic [drowsy, easily aroused]; stupor [difficult to arouse]; coma [unarousable]; Uncertain)

5. **[Disorientation]** Was the patient disoriented at any time during the interview, such as thinking that he or she was somewhere other than the hospital, using the wrong bed, or misjudging the time of day?

6. **[Memory impairment]** Did the patient demonstrate any memory problems during the interview, such as inability to remember events in the hospital or difficulty remembering instructions?

7. **[Perceptual disturbances]** Did the patient have any evidence of perceptual disturbances, e.g. hallucinations, illusions, or misinterpretations (such as thinking something was moving when it was not)?

8A. **[Psychomotor agitation]** At any time during the interview did the patient have an unusually increased level of motor activity such as restlessness, picking at bedclothes, tapping fingers, or making frequent sudden changes of position?

8B. **[Psychomotor retardation]** At any time during the interview did the patient have an unusually decreased level of motor activity such as sluggishness, staring into space, staying in one position for a long time, or moving very slowly?

9. **[Altered sleep-wake cycle]** Did the patient have evidence of disturbed sleep-wake cycle, such as excessive daytime sleepiness with insomnia at night?

The CAM diagnostic algorithm

Feature 1: Acute onset or fluctuating course

This feature is usually obtained from a family member or nurse and is shown by positive responses to the following questions: Is there evidence of an acute change in mental status from the patient's baseline? Did the (abnormal) behaviour fluctuate during the day, that is, tend to come and go, or increase and decrease in severity?

Feature 2: Inattention

This feature is shown by a positive response to the following question: Did the patient have difficulty focusing attention, for example, being easily distractible, or having difficulty keeping track of what was being said?

Feature 3: Disorganised thinking

This feature is shown by a positive response to the following question: Was the patient's thinking disorganised or incoherent, such as rambling or irrelevant conversation, unclear or illogical flow of ideas, or unpredictable switching from subject to subject?

Feature 4: Altered level of consciousness

This feature is shown by any answer other than 'alert' to the following question: Overall, how would you rate this patient's level of consciousness? (alert [normal]), vigilant [hyperalert], lethargic [drowsy, easily aroused], stupor [difficult to arouse], or coma [unarousable])

The diagnosis of delirium by CAM requires the presence of features 1 and 2 and either 3 or 4.

24.2.5.3 Role of Specific Investigations for Delirium

Electroencephalography may show moderate-to-severe generalised slowing of brain activity in delirium, distinguishing the condition from dementia and other psychiatric diagnoses. It may also help exclude non-convulsive status epilepticus.

Neuroimaging, mostly CT in our local setting, has been routinely used to exclude primary neurological causes of delirium such as stroke, bleeding, and space-occupying lesions. Lumbar puncture should be reserved for when there is clinical suspicion of a central nervous system infection, or in the future, when CSF biomarkers will be clinically useful for making the diagnosis.

Among the above investigations, all will produce extra stress and distress to the already-delirious elderly patient in hospital. Hence sensible judgements in choosing the appropriate investigation should be exercised on an individual basis. Sometimes a trial of therapy, including removal of noxious agents and potential precipitants, is a useful form of diagnostic tool that causes minimal harm.

24.2.6 Prevention and Interventions for Delirium

24.2.6.1 Non-pharmacological Therapy and Models of Care

There are various models of care for the prevention and interventions of delirium.[61-65] The most traditional approach is the Hospital Elder Life Program, where a multicomponent intervention strategy with proven effectiveness and cost-effectiveness in the prevention of delirium and functional decline is implemented through targeting risk factors for delirium.[66-68] In 2016, a Cochrane review analysed interventions for preventing delirium in hospitalised patients.[69] Non-pharmacological, multi-component interventions (which included reorientation, early patient mobilisation, and education) reduced the risk of developing delirium by 31% compared to usual care. This finding was consistent in both medical (general medical, geriatric) and surgical (majority orthopaedic) settings.

Progressing from the above models, the acute care for the elderly (ACE) unit model has been the focus of attention and research interests in managing elderly medical inpatients including those with delirium.[70] The delirium room (DR) concept in the ACE unit is the current research trend. Flaherty et al[71] described an improvement in ADL function and equalisation of length of hospital stay in delirious patients managed in the DR compared with patients without delirium. More DRs blossomed in different parts of the world, with different setups and interventions put under trial.[72]

Most DRs and ACE units, apart from intensive nursing supervision, adopt the underlying concept of the Dementia Care Bundle (Table 24.9). Locally in Hong Kong, various ACE units have been set up in various public hospitals. In 2009, the Prince of Wales Hospital designated a 24-bed unit as the ACE unit, and aimed to deliver the 10 ACE-focus areas announced by the Hospital Authority in a structured manner. One of these areas was delirium (acute confusion). The relevant nursing interventions carried out in the unit are listed in Table 24.10.

24.2.6.2 Pharmacological Therapy and Approach of Management

To date, there is a lack of compelling evidence for which agent or even class of agents is the most effective and safe for the prevention and treatment of a delirious episode. Much experience has been gained from the drugs used to treat the non-cognitive

Table 24.9 Key elements of the Dementia Care Bundle

1. Staff know key information about the patient within 24 hours of admission.
2. Personalised communication takes place with the patient frequently, skilfully, and compassionately.
3. Nutrition and hydration are adequate for the individual patient, are in line with patient preference, and are provided in a way that is geared to the patient's capability.
4. A safe and orientating environment is maintained.

Table 24.10 Examples of nursing interventions in an acute care for the elderly unit

1. Fulfil patient's needs. Explain treatment.
2. Orientate patient to surroundings.
3. Ensure glasses / dentures / hearing aids / amplifiers fit and are working.
4. Invite relatives to visit more frequently.
5. Actively look for urinary retention / constipation / pressure sores / pain.
6. Remove physical restraints for 10-15 minutes every shift while observing for persistence of delirium. Consider complete removal of restraints if delirium is resolved.

symptoms of dementia (BPSD).

The general approach, of utmost importance and priority, is the removal wherever possible of all potential deliriogenic agents. A temporal relationship from the onset of delirium to any change in drug dosages or new additions should be established, including recent ad-hoc doctor consultations, over-the-counter and herbal drugs, and any iatrogenic events (including recent hospital admissions, endoscopic procedures, physical restraints, and even bedside procedures such as urinary and feeding catheter changes). Next, as in all acute geriatric admissions, acute medical events should be sought and identified as any of them can precipitate a delirious episode in an elderly individual with limited brain reserve.

Melatonin given before bedtime has been shown in isolated small-scale randomised controlled trails to prevent delirium in high-risk geriatric inpatients (medical and surgical), based on the principle that sleep deprivation and melatonin deficiency are implicated in the pathogenesis of delirium.[73,74] Large-scale studies and meta-analyses are needed to determine whether this method of preventing delirium is useful.

Antipsychotics should be avoided as far as possible due to their anticholinergic and extrapyramidal side-effects. Although atypical antipsychotic agents have often been stated as having similar efficacy to haloperidol but with reduced propensity for extrapyramidal side-effects,[75] atypical antipsychotics do have dose-dependent extrapyramidal and anticholinergic side-effects similar to traditional antipsychotics.[76] High-quality reviews have concluded that there is lack of evidence to justify the use of antipsychotics, whether atypical or traditional, for prevention or treatment of delirium; and the risks of treatment with antipsychotics clearly outweigh the benefits.[76]

Trazodone should be the only agent in the class of antidepressants to be used, as SSRIs and tricyclic antidepressants have the potential to cause delirium. Benzodiazepines are reserved for use in patients undergoing sedative or alcohol withdrawal, those with Parkinson's disease, and those with neuroleptic malignant syndrome,[77] since clinical trials have shown them to be associated with prolongation and worsening of delirium symptoms.[78]

'Start low, go slow' with the above agents remains the pearl of wisdom as in all geriatric prescriptions. Another key principle of pharmacotherapy for delirium is to discontinue it as soon as possible, once the delirious episode has resolved together with treatment of the underlying cause. Communication with nursing staff and family members should not be neglected. They should be kept informed of the patient's mental status and be involved in discussions of when best to stop drug treatment.

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CHAPTER 25

DEPRESSION

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OBJECTIVES

To appreciate the extent and impact of the problems of depression and suicide in elderly people; to understand the epidemiology, symptomatology and diagnosis of depression in elderly people; to appreciate the importance of appropriate investigations and rational pharmacotherapy in the management of depression in elderly people; and to understand the importance of early detection of depression and timely intervention for the prevention of suicide in elderly people.

KEY POINTS

- Depression is one of the most important causes of disease burden in the world, but is frequently underdetected and undertreated in the primary healthcare setting.
- The prevalence of depression in elderly people varies with respect to the clinical setting and is substantial.
- The accurate diagnosis of depression depends on careful history taking and mental state examination.
- The presentation of depression in elderly people is often modified by the ageing process and may be 'atypical'.
- Standardised screening instruments, such as the Geriatric Depression Scale, are available to aid detection and diagnosis of depression in elderly people.
- Pharmacological treatment should be rational and based on putative molecular mechanisms, and prescribers should be aware of the pharmacokinetic and pharmacodynamic changes associated with ageing.
- Generic psychosocial interventions are as important as pharmacotherapy in the management of depression in elderly people.
- Suicide in elderly people is associated with a number of modifiable risk factors, the most important of which is depression.
- It is possible to reduce suicide in elderly people by early detection of depression and timely intervention.

25.1 Introduction

Depression is one of the commonest psychiatric disorders in old age, and yet, it is frequently underdetected and undertreated in the primary care setting. The fact that loss is common in this particular stage of the human life often leads to the notion that the presence of depressive symptoms is somehow understandable and 'normal' in old age. In addition, the presentation of depressive disorders in elderly people may be modified by old age, making early detection difficult. In fact, depression in elderly people is a highly treatable illness, and its prognosis is at least as good as depression presenting in younger age-groups.[1] If left untreated, depression is associated with significant disability, functional decline, diminished quality of life, increased burden for caregivers, increased service utilisation, and increased mortality due to comorbid medical conditions and suicide.[2] Recently, the World Health Organization projected that unipolar depressive disorders would become the leading cause of global burden of diseases by the year 2030,[3] as a result of expected demographic change in the world. One of the most undesirable and tragic complications of depression in elderly people is suicide. In Hong Kong, the rate of suicide in elderly people is 2 to 3 times higher than in the general population, and about 30% of all deaths due to suicide occur among those aged ≥65 years.[4] With the rapidly ageing population in Hong Kong, the recognition and management of depression and prevention of suicide in elderly people will become increasingly important public

health issues.

25.2 Epidemiology

Depression is common in elderly people, but its reported prevalence rate varies widely, mainly as a result of differences in populations studied, diagnostic definitions used and methodologies employed. In general, the prevalence rate increases from the community to the primary care setting, with the highest prevalence being found in institutional settings such as hospitals and residential care homes. There is consistent evidence for a female preponderance in the prevalence of both subsyndromal depressive symptoms (also termed minor depression) and major depressive disorder.[5]

In the community setting, the prevalence of subsyndromal depressive symptoms is approximately 15% while the prevalence of major depressive disorder is between 1% and 3%.[6] The local prevalence of subsyndromal depressive symptoms among the community-dwelling elderly was reported to be between 12.5% and 37.8%, with higher rates found in those with disabilities.[7-9] For major depressive disorder, a study by Chiu[10] reported a community prevalence of 1.54% in people aged >70 years. Table 25.1 summarises the prevalence of depression in elderly people in different care settings including primary care,[11] acute hospital,[12] and residential care homes.[13]

A number of risk factors have been found to be associated with depression in elderly people in epidemiological studies and are listed in Table 25.2.[2] It is noteworthy that the causal and temporal relationships between these risk factors and depression have not been established.

Table 25.1 Prevalence of depression in elderly people in different care settings[6-13]

Care setting	Prevalence of subsyndromal depressive symptoms (%)	Prevalence of major depressive disorder (%)
Community	15	1-3
Primary care	20	10-12
Acute hospital	20-25	10-15
Residential care	30-40	16

Table 25.2 Risk factors for depression in elderly people[2]

Female sex
Being widowed or divorced
Medical illness, e.g. stroke, neurological disorders
Functional disability
Family and personal history of depression
Social isolation
Life events
Medications, e.g. antihypertensives, steroids, antiparkinsonian drugs
Caregiving

25.3 Diagnosis

Depression is a syndromal diagnosis based on eliciting a specific cluster of symptoms through careful history taking and mental state examination, supplemented by relevant physical examination and laboratory investigations. No confirmatory laboratory test is available for diagnosing depression, and the role of laboratory investigations mainly lies in excluding possible organic causes for the observed depressive symptoms.

A diagnosis of major depression is made if the elicited symptoms fulfil the diagnostic criteria of either one of the two major classification systems currently in use, namely, the International Statistical Classification of Diseases and Related Health Problems, tenth revision (ICD-10),[14] and the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5).[15] To make a diagnosis of depression, the required number of symptoms (four for ICD-10 and five for DSM-5) must be present for most of the time, on most days, for a minimum of 2 consecutive weeks. The core depressive symptoms included in the two diagnostic criteria are similar, and specifiers are available for documentation of additional features like severity, psychosis, and remission. Of the two classification systems, DSM-5 is generally preferred because its criteria tend to be more explicit and operationalised. The DSM-5 criteria for major depressive disorder are listed in Table 25.3.

25.4 Diagnostic Difficulties

Many studies have found that primary care physicians could identify no more than 50% of patients with diagnosable depressive disorders[6] despite the seemingly explicit nature of standard diagnostic criteria. One of the main reasons is that the presentation of depression in elderly people may be atypical, making recognition and diagnosis difficult.

25.4.1 Minimisation of Feelings of Sadness

There is evidence that elderly people with depression have a tendency to minimise feelings of sadness to their doctors.[16] This may be especially relevant in Chinese elders, who tend not to bother their doctors with emotional difficulties. Some elders might even feel ashamed of disclosing their symptoms and worries. It may be more fruitful if proactive enquiries are made on areas like recent loss of interest (anhedonia), reduced self-esteem, guilt, and suicidal ideation.

Table 25.3 DSM-5 criteria for major depressive disorder

A. Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure
   Note: Do not include symptoms that are clearly attributable to another medical condition
   1. Depressed mood most of the day, nearly everyday, as indicated by either subjective report (e.g. feels sad, empty, hopeless) or observations made by others (e.g. appears tearful)
   2. Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation)
   3. Significant weight loss when not dieting or weight gain (e.g. a change of >5% of body weight in a month), or decrease or increase in appetite nearly every day
   4. Insomnia or hypersomnia nearly every day
   5. Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down)
   6. Fatigue or loss of energy nearly every day
   7. Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick)
   8. Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others)
   9. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide
B. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning
C. The episode is not attributable to the physiological effects of a substance or to another medical condition
D. The occurrence of the major depressive episode is not better explained by schizoaffective disorder, schizophrenia, schizophreniform disorder, delusional disorder, or other specified and unspecified schizophrenia spectrum and other psychotic disorders
E. There has never been a manic episode or a hypomanic episode

Abbreviation: DSM-5 = Diagnostic and Statistical Manual of Mental Disorders, 5th edition

25.4.2 Somatisation or Disproportionate Complaints Associated with Physical Disorder

Depression presenting as a physical complaint is common among elderly patients.[17] In a local study, 70% of patients presenting to an academic general practice clinic with diagnosable depressive disorders had a chief complaint of a somatic nature, such as dizziness, chest pain, dyspepsia, and headache.[18] Alternatively, some patients may present with a physical complaint disproportionate to known pathology. Systematic history taking is needed to tackle this problem, which should include enquiry about any recent change in the concerned somatic symptoms and a thorough physical review, including laboratory investigations, if indicated.

25.4.3 'Neurotic' Symptoms of Recent Onset

Anxiety is commonly associated with depressive disorders in elderly people.[19] Apart from typical anxiety symptoms (e.g. nervousness, tension, unfounded worries, and autonomic hyperarousal), comorbid anxiety spectrum disorders like generalised anxiety disorder, panic disorder, phobic disorder, obsessive-compulsive disorder and hypochondriacal disorder, might also be present. Depressive elders with prominent concurrent anxiety symptoms or comorbid anxiety disorders are associated with increased suicidal risk and poorer treatment outcome versus the normal population, and, hence, more intensive and aggressive approach in management is warranted.

25.4.4 'Trivial' Acts of Deliberate Self-harm

Seemingly 'trivial' acts of deliberate self-harm like wrist slashing or overdoses in elderly people should be taken seriously and should be promptly referred to the specialist psychiatric service for assessment and management, as there is no reliable correlation between the medical severity of the act and seriousness of the psychiatric problem. There is consistent evidence that most elderly people who eventually commit suicide due to moderate depression and manipulative attempts of deliberate self-harm are rare in elderly people.[20]

25.4.5 Pseudodementia

It is not uncommon for elderly patients with depression to present with cognitive impairment (memory deficits, executive dysfunction, and slowed processing speed).[21] The characteristic features of 'pseudodementia' include a clear onset of cognitive impairment of short duration with the patient complaining at length about their memory, "don't know" answers on cognitive function test accompanied by considerable irritation, a personal or family history of depression, and the presence of depressive symptoms such as diurnal mood variation and negative cognitions.[1] There is growing evidence that a significant proportion of depressed elderly, despite treatment, remains cognitively impaired.[21,22] Depression in elderly people is also believed to be associated with increased risk of developing dementia later in life.[23,24]

25.4.6 Depression Superimposed on Dementia

Depressive symptoms are common, especially, in the early stages of dementia,[25] and the presence of dementia may prevent recognition of a comorbid depressive disorder because certain symptoms such as apathy, psychomotor retardation, and decreased concentration overlap with symptoms of dementia. Interview with an informant is, therefore, essential in differentiating the diagnosis. Particular attention should be paid to features such as crying spells, feeling of loneliness, anxiety symptoms, early morning wakening, negative cognitions, preoccupation with death and dying, and suicidal ideation.

25.4.7 Accentuation of Premorbid Personality Traits and Recent Change in Behaviour

Clinicians who are experienced in working with elderly patients will readily recognise this phenomenon of recent change or exaggeration of usually undesirable personality traits in patients with depression. This may take the form of dramatic theatricality or ceaseless importunate behaviour. In others, shoplifting and late-onset alcohol or substance-related problems might be the markers of depression. Again, the key to assessment is careful history taking to delineate a recent onset of these behaviours, and to elicit the depressive symptoms that may not be immediately apparent on cursory examination.

25.5 Assessment

The most important element of assessment in an elderly patient with suspected depression is history taking and mental state examination, preferably, in the presence of an informant. Diagnostic checklists are useful as a guide but are not meant to be inviolable. This is especially true in elderly people when clinical presentations are modified by old age.

Assessment should aim at eliciting the exact onset, nature and duration of symptoms, bearing in mind the possibility of atypical presentations as detailed in the previous section. Any hints of suicidal ideation should be taken seriously, and evaluation of suicide risk factors (see chapter 25.7) and intent (planning, preparation, method) should be done for all patients. Care should be taken in the assessment of any coexisting physical illnesses and associated functional impairment. A detailed drug history should be obtained, as some medications may be a cause of depression on their own. Common examples of depression-inducing drugs include antihypertensives (e.g. methyldopa, beta-blockers, calcium channel blockers), corticosteroids, antiparkinsonian drugs, and cyclooxygenase-2 inhibitors. In addition, the availability of social support (from family, friends and social agencies) should be assessed as these may have bearings on the ability of the individual to comply with treatment (as in those with cognitive impairment), the risk of self-harm (as in those who may have suicidal ideation), and the need for further specialist input (as in those with complicated psychosocial problems). Assessment of cognitive function using validated assessment instruments (e.g. Cantonese version of the Mini-Mental State Examination,[26] or Montreal Cognitive Assessment Hong Kong Version[27]) should also be performed.

Physical examination should be carried out to assess the extent of coexisting physical illnesses and their role, if any, in the aetiology of the patient's depression. Common physical conditions that may cause depression are listed in Table 25.4.

Laboratory investigations, if indicated, should be selective and carried out with the purpose of ruling out any organic causes. These are listed in Table 25.5.

Locally validated instruments, such as the Geriatric Depression Scale (GDS),[28] are widely used for screening and assessing the severity of depressive symptoms in elderly people. GDS is a self-rating scale that is easy to administer and is particularly suitable for elderly patients as it focuses on cognitive aspects of depression and excludes many of the somatic symptoms. It has been endorsed for use in primary care by the Royal College of General Practitioners of the UK.[29] In Hong Kong, the GDS has been translated into Cantonese in a short form which consists of 15 items.[30] A standardised verbal instruction manual has

Table 25.4 Common physical conditions that may cause depression

- Endocrine / metabolic, e.g. hypo- / hyper-thyroidism, Cushing's disease, hypercalcaemia, pernicious anaemia, folate deficiency
- Organic brain disease, e.g. stroke, brain tumours, Parkinson's disease, Alzheimer's disease, multiple sclerosis
- Occult carcinoma, e.g. carcinoma of pancreas or lung
- Chronic infection, e.g. neurosyphilis

Table 25.5 Investigations for depression in elderly people

Investigation	Remark
Complete blood picture	Yes
Liver and renal function	Yes
Thyroid function and calcium	Yes
Vitamin B12 and folate	If indicated by nutritional status
VDRL	If neurologically indicated
CT brain	If clinically indicated

Abbreviations: CT = computed tomography; VDRL = venereal disease research laboratory

also been validated.[31] A cut-off score of 8 was found to be sensitive and specific in detecting depression in the local elderly population. The English and Cantonese versions of the GDS Short Form are shown in Tables 25.6 and 25.7, respectively.

25.6 Management

As recommended by the World Psychiatric Association, management of depression in elderly people should involve a wide range of possible interventions that may be focused on the patient, on his or her living conditions, or on the family or other caregivers.[32] The overall objectives are:

- resolution of depressive symptoms and signs;
- reintegration into the family and social environment, when possible;
- prevention of relapse or recurrence; and
- restoration of functioning and social roles

25.6.1 Pharmacotherapy

The mainstay of pharmacological treatment for depressive disorders in elderly people is antidepressants, although short-term use of hypnotics or benzodiazepines may be necessary for symptomatic relief in some patients.

The monoamine hypothesis is the dominant biochemical theory that accounts for the biological aetiology of depression. In brief, depression is believed to result from a deficiency of monoamine neurotransmitters, namely, noradrenaline (norepinephrine), serotonin, and dopamine. The majority of antidepressants act by increasing the activity of these neurotransmitters in the brain through various mechanisms.

25.6.1.1 Principles of Prescription

Before prescribing any medication, it is important to establish a therapeutic alliance with the patient, without which the patient is unlikely to comply with either pharmacological treatment or the physician's advice. Listening to the patient, providing adequate information, allowing ample time for questions, and regular and frequent review, are all important components in building such an alliance (see 25.6.2 Psychosocial Management).

Due to changes in both pharmacokinetics and pharmacodynamics with advancing age, the prescription principle of 'start low, go slow' is especially applicable in elderly people. In addition, elderly patients often take longer to respond to an antidepressant. Instead of the usual time lag of 2 to 4 weeks in antidepressant response in younger patients, elderly patients may need as long as 8 weeks to respond to a trial of antidepressant.[1] It is particularly important to explain to the patient about this delay in onset of action of antidepressants as the seemingly lack of effect in the first few weeks of treatment is an important cause of non-compliance and disengagement in the therapeutic alliance. Furthermore, as elderly people are more susceptible to adverse drug reactions, the patient must be closely monitored in the initial stage of acute treatment so that side-effects are promptly addressed and managed. Since many elderly patients may have comorbid medical conditions and may be on long-term maintenance medications for these conditions, the choice of antidepressant would also need to take into account the possibility of drug interactions.

25.6.1.2 Choice of Antidepressant

There is convincing evidence that antidepressants are effective in treating depression in elderly people, including those with comorbid medical conditions.[33,34] However, there is no good evidence to show that any one class of antidepressant is superior

Table 25.6 Geriatric Depression Scale Short Form: English version*

Choose the best answer for how you have felt over the past week	Answer
1. Are you basically satisfied with your life?	No
2. Have you dropped many of your activities and interests?	Yes
3. Do you feel your life is empty?	Yes
4. Do you often get bored?	Yes
5. Are you in good spirits most of the time?	No
6. Are you afraid something bad is going to happen to you?	Yes
7. Do you feel happy most of the time?	No
8. Do you often feel helpless?	Yes
9. Do you prefer to stay at home, rather than going out and doing new things?	Yes
10. Do you feel you have more problems with your memory than most?	Yes
11. Do you think it is wonderful to be alive now?	No
12. Do you feel pretty worthless the way you are?	Yes
13. Do you feel full of energy?	No
14. Do you feel that your situation is hopeless?	Yes
15. Do you think most people are better off (in their lives) than you are?	Yes

* Score 1 point for each question if giving the answer as indicated. A cut-off score of ≥8 is suggestive of possible depression

Table 25.7 Geriatric Depression Scale (GDS) Short Form: Cantonese version
This instrument (GDS-SF Cantonese version) was translated and validated by Wong et al[31] based on the GDS Chinese version[30]

老人抑鬱短量表

姓名:_____ **年齡:**_____ **性別:**_____ **備註:**_____

以下的問題是人們對一些事物的感受，答案是沒有對與不對。請想一想，在過去一星期內，你是否曾有以下的感受。如有的話，請圈「是」，若無的話，請圈「否」。

	是	否
1. 你基本上對自己的生活感到滿意嗎？ 問 <1> 喺上個禮拜裡面，你滿唔滿意自己嘅生活呢？ 問 <2> 咁你係滿意多啲，抑或唔滿意多啲呢？	---	1
2. 你是否已放棄了很多以往的活動和嗜好？ 問 <1> 喺上個禮拜裡面，你有冇放棄好多以前嘅活動或者嗜好呢？ 問 <2> 喺上個禮拜裡面，好多以前你鍾意做嘅嘢，係咪已經冇做啦？ 問 <3> 咁喺上個禮拜裡面，你喺朝早或日頭有冇啲嘢做吓？ 例如：晨運、落街行吓，同人傾吓偈，或者響屋企做吓家務呢？	1	---
3. 你是否覺得生活空虛？ 問 <1> 喺過去呢個禮拜裡面，你係咪覺得生活空虛呢？ 問 <2> 喺上個禮拜裡面，你係咪覺得做人都幾百無聊賴呢？	1	---
4. 你是否常常感到煩悶？ 問 <1> 喺上個禮拜裡面，你係咪成日覺得好煩悶呀？	1	---
5. 你是否很多時感到心情愉快呢？ 問 <1> 你上個禮拜心情係咪成日都咁好呢？ 問 <2> 咁你上個禮拜，係開心嘅時候多，定係唔開心嘅時候多啲呢？	---	1
6. 你是否害怕將會有不好的事情發生在你身上呢？ 問 <1> 喺上個禮拜裡面，你有無擔心有啲唔好 嘅嘢 會發生喺你身上呢?	1	---
7. 你是否大部份時間感到快樂呢？ 問 <1> 喺上個禮拜裡面，你係咪成日都覺得開心呢？ 問 <2> 咁你喺上個禮拜裡面，係開心多啲，抑或唔開心多啲呢？	---	1
8. 你是否常常感到無助？ 問 <1> 喺上個禮拜，你有冇覺得無論做乜嘢，都係冇用呢？ 問 <2> 喺上個禮拜，你有冇覺得無論做乜嘢，都係無補於事呢？	1	---
9. 你是否寧願留在院舍/屋企裡，而不出外做些有新意的事情？ 問 <1> 喺上個禮拜裡面，你係咪寧願留低響老人院/屋企，都唔想落街做啲有新意嘅事呢？ 問 <2> 喺上個禮拜裡面，你係咪寧願留低響老人院/屋企，都唔想落街做啲特別嘅事情呢？ 問 <3> 咁你上個禮拜其實想唔想落街行吓，做啲你平時少做嘅嘢呢？	1	---
10. 你是否覺得你比大多數人有多些記憶的問題呢？ 問 <1> 喺上個禮拜裡面，你有冇覺得你嘅記性比其他老人家差呢？ 問 <2> 咁比起兩三個禮拜前，你上個禮拜記性有冇差到呢?	1	---
11. 你認為現在活著是一件好事嗎？ 問 <1> 喺上個禮拜裡面，你有無覺得做人係一件好事呢？ 問 <2> 喺上個禮拜裡面，你覺得做人係有意思嘅，係唔係呢？	---	1
12. 你是否覺得自己現在一無是處呢？ 問 <1> 喺上個禮拜裡面，你係咪覺得自己好無用呢？ 問 <2> 喺上個禮拜裡面，你有冇覺得自己完全冇用呢？	1	---
13. 你是否感到精力充足？ 問 <1> 喺上個禮拜，你係咪覺得精力充沛呀？ 問 <2> 喺上個禮拜，你係咪好夠精力呀？ 或 問 <3> 喺上個禮拜，你係咪好夠精神呀？	---	1
14. 你是否覺得自己的處境無望？ 問 <1> 喺上個禮拜裡面，你有無覺得你嘅處境係無哂希望呢？	1	---
15. 你覺得大部份的人的境況比自己好嗎？ 問 <1> 喺上個禮拜裡面，你係咪覺得大部份人嘅情況都好過你？	1	---

總分: _____

備註:
聽覺： 良好 滿意 普通 差
理解能力： 良好 滿意 普通 差
合作程度： 良好 滿意 普通 差
清醒程度： 良好 滿意 普通 差

in terms of efficacy or speed of onset of action. The choice of antidepressant is, therefore, determined by patient preference, symptom profile, tolerability, previous response to treatment, potential drug interactions, safety in overdose, and physician's clinical experience.

In Hong Kong, the selective serotonin reuptake inhibitors (SSRIs) have largely superseded the traditional tricyclic antidepressants (TCAs) and become the standard first-line antidepressant for treating depression in elderly people. The newer antidepressants such as serotonin–norepinephrine reuptake inhibitors (SNRIs), noradrenergic and specific serotonergic antidepressants (NaSSAs), serotonin antagonist and reuptake inhibitors (SARIs), and norepinephrine–dopamine reuptake inhibitors (NDRIs) are becoming more popular second-line alternatives. Table 25.8 summarises the properties and dosages of the various commonly used antidepressants in Hong Kong.

TCAs

TCAs are one of the oldest groups of psychiatric drugs and, therefore, have been most extensively studied. Depending on the subgroup, most TCAs act by inhibiting presynaptic reuptake of noradrenaline and serotonin. One of the most extensively studied TCA in elderly people is nortriptyline, with a usual starting dose of 10 to 30 mg at night, gradually increasing up to 75 to 100 mg at night. Another commonly prescribed TCA in Hong Kong is dothiepin, which has a starting dose of 25 to 50 mg at night and a therapeutic dose of 75 to 150 mg at night for elderly people. Common side-effects of TCA, as a group, include anticholinergic side-effects (dry mouth, blurred vision, constipation, urinary retention, and delirium), antiadrenergic side-effects (dizziness and postural hypotension), antihistaminic side-effects (sedation and weight gain), and cardiotoxic effects (arrhythmia and heart block). Because of these side-effects, TCAs can be very dangerous in overdose, and are, generally, considered unsuitable for patients with significant risk of suicide. On the other hand, since most TCAs are sedative, they may be helpful in alleviating insomnia and lessening the need to prescribe an additional hypnotic.

SSRIs

The SSRIs act by selectively inhibiting the neuronal presynaptic reuptake of serotonin, causing a net increase in the quantity of serotonin in the synaptic cleft. The SSRIs have become first-line antidepressants in elderly people because of their more favourable side-effect profile. In particular, they are, generally, not sedative and have very little anticholinergic side-effects. They are generally not cardiotoxic, making them safer in patients with risk of deliberate self-harm. The main side-effects include nausea, vomiting, diarrhoea, headache, agitation, and sexual dysfunction. Inappropriate antidiuretic hormone secretion and gastrointestinal bleeding have also been reported to be more common in elderly people versus the general population.[35,36] The SSRIs are inhibitors of the cytochrome P450 enzyme 2D6, and may cause drug interactions with warfarin, digoxin, and cimetidine. The SSRIs available in Hong Kong include fluoxetine, paroxetine, fluvoxamine, citalopram, escitalopram, and sertraline. Citalopram, escitalopram, and sertraline may have some advantages in elderly people in that they are more specific inhibitors of serotonin reuptake and less potent inhibitors of cytochrome P450 enzyme 2D6, making drug interactions less likely.[37] The usual starting dose in elderly patients for both citalopram and escitalopram is 10 mg in the morning, increasing

Table 25.8 Commonly used antidepressants in Hong Kong

Drug group	Commonly used drugs in elders	Starting dose	Usual therapeutic dose	Common side-effects
TCA	Nortriptyline	10-30 mg Nocte	75-100 mg Nocte	Dry mouth, constipation, hypotension, sedation
	Dothiepin	25-50 mg Nocte	75-150 mg Nocte	
SSRI	Citalopram	10 mg OM	20 mg OM	Nausea, vomiting, diarrhoea, headache, agitation, sexual dysfunction
	Escitalopram	10 mg OM	20 mg OM	
	Sertraline	25-50 mg OM	150-200 mg OM	
SNRI	Venlafaxine XR	75 mg Daily	150-225 mg Daily	Nausea, sweating, high blood pressure at high doses
	Desvenlafaxine	25 mg Daily	50 mg Daily	
	Duloxetine	30 mg Daily	60 mg BD	
	Milnacipran	25 mg Daily	50 mg BD	
NaSSA	Mirtazapine	15 mg Nocte	30-45 mg Nocte	Weight gain, sedation
SARI	Trazodone	25-50 mg Nocte	150-300 mg Nocte	Sedation, dry mouth, nausea
NDRI	Bupropion SR	150 mg Daily	150 mg BD	Dry mouth, headache, insomnia

Abbreviations: BD = twice a day; NaSSA = noradrenergic and specific serotonergic antidepressant; NDRI = norepinephrine–dopamine reuptake inhibitor; Nocte = at night; OM = every morning; SARI = serotonin antagonist and reuptake inhibitor; SNRI = serotonin–norepinephrine reuptake inhibitor; SSRI = selective serotonin reuptake inhibitor; TCA = tricyclic antidepressant

to 20 mg, if tolerated. For sertraline, the usual starting dose is 25 to 50 mg in the morning, gradually increasing to 150 to 200 mg in the morning or in divided doses, according to tolerability and clinical response. Since the SSRIs are generally nonsedative, it is often worthwhile to offer a short course (not more than 2 weeks on an as-required basis) of hypnotics to address the common symptom of insomnia before the onset of the antidepressant effect.

SNRIs

SNRIs share the dual-action reuptake inhibitory properties of TCAs, but do not have the side-effects of TCAs.[37] At lower doses, the properties of SNRIs resemble those of SSRIs while at higher doses, their dual-action properties become prominent. They may be particularly beneficial to depressed patients with comorbid chronic pain conditions.[38-40] Common side-effects include nausea, sweating, insomnia, nervousness, tremor, and raised blood pressure at high doses. Hence, caution should be exercised in patients with hypertension. Four SNRIs, namely, venlafaxine XR, desvenlafaxine, duloxetine and milnacipran, are currently available in Hong Kong. Both venlafaxine XR and desvenlafaxine can be administered once daily. For venlafaxine XR, the starting dose is 75 mg once daily, which can be increased up to a maximum of 225 mg once daily, in a relatively short period of time. For desvenlafaxine (which is the active metabolite of venlafaxine), it is advisable to start with 25 mg daily, increasing to 50 mg once daily, if tolerated. The starting dose of duloxetine is usually 30 mg, gradually increasing to a maximum of 60 mg twice daily. For milnacipran, the usual starting dose is 25 mg daily, and the recommended therapeutic dose is 50 mg twice daily.

NaSSAs

Mirtazapine belongs to the group of NaSSAs. It exerts its antidepressant effect by inhibiting the presynaptic alpha-2 adrenergic receptor, which disinhibits the release of both noradrenaline and serotonin.[37] It also antagonises serotonin 2A, 2C and 3, and histamine H_1 receptors, giving rise to its anxiolytic and sedative effects without causing sexual dysfunction and gastrointestinal problems. Common side-effects with mirtazapine include sedation, dizziness, and weight gain. The usual starting dose in elderly people is the same as in adults: 15 mg at night, which can be increased up to a maximum of 45 mg at night. Mirtazapine is particularly useful in patients presenting with severe insomnia and comorbid anxiety symptoms.

SARIs

Trazodone belongs to the group of SARIs. It exerts its antidepressant effect through blockade of serotonin 2A and 2C receptors, and serotonin transporter. It also antagonises alpha-1 adrenergic and histamine H1 receptors. At low doses, trazodone works like a hypnotic, mainly through its serotonin 2A, alpha-1 adrenergic and histamine H_1 antagonism. Trazodone exhibits antidepressant properties only at high doses. Common side-effects include nausea, vomiting, sedation, dizziness, and hypotension. The usual starting dose is 25 to 50 mg at night. Slow titration to the range of 150 to 300 mg at night is recommended to minimise daytime sedation.

NDRIs

Bupropion SR belongs to the group of NDRIs. It acts by blocking norepinephrine and dopamine reuptake. It is generally activating, and the common side-effects include dry mouth, headache, insomnia, and sweating. To minimise the risk of seizure, bupropion SR should be started at 150 mg daily and slowly titrated to the recommended therapeutic dose of 150 mg, twice daily.

25.6.1.3 What to Do If There is No Response?

Up to 30% of depressed elderly patients do not improve with first trial of antidepressant treatment.[41] When this happens, a number of steps should be taken (Table 25.9). First, the diagnosis of the patient should be reviewed. For example, an unforthcoming, severely depressed patient may be harbouring unrecognised psychotic symptoms, making him or her unresponsive to antidepressant monotherapy. Second, a review of physical conditions should be carried out to ensure that any previously undetected organic causes of depression are corrected (see Table 25.4 for a list of common physical conditions that may cause depression). Third, the compliance of the patient should be checked. Non-compliance may be due to unacknowledged side-effects, misunderstanding, or myth about all psychiatric medications being addictive, lack of supervision in the cognitively impaired and physically frail patients, and lack of insight. The physicians should, therefore, adopt a proactive approach in providing adequate information about the nature, expected

Table 25.9 Strategies in non-response to initial antidepressant treatment

1. Review diagnosis
2. Look for and correct previously unrecognised organic cause of depression, if any
3. Assess and enhance compliance
4. Look for and address ongoing psychosocial stresses
5. Review antidepressant treatment:
   (a) If partial response after 4-6 weeks: increase dose to maximally tolerated dose cautiously; allow adequate duration of treatment
   (b) If little or no response after 4 weeks: switch to another class of antidepressant

duration and possible delay in response associated with antidepressant treatment. Soliciting assistance from the patient's relatives and / or community agencies in providing supervision to the patient is also helpful in addressing compliance problems. Fourth, a depressed patient may fail to recover because of ongoing psychosocial stress. Common examples in elderly people include unresolved grief, longstanding marital discord, or perceived disability associated with chronic physical illnesses. Simple counselling and provision of tangible support may be able to address these problems and bring about improvement in symptoms. Finally, a review of antidepressant treatment should be carried out. If there is partial response to the initial treatment, a further increase in dose up to the maximally tolerated dose is indicated. If there is little or no response with 4 weeks' initial treatment, a switch to another class of antidepressant is indicated.

For patients with doubtful diagnosis, severe depressive symptoms (including psychotic symptoms), high risk of self-neglect or self-harm, inadequate response to antidepressant treatment, complicated psychosocial situations and lack of social support, referral to specialist psychiatric service for further assessment, intensive treatment (e.g. augmentation and combination pharmacotherapies, formal psychological interventions, electroconvulsive therapy) and multidisciplinary management should be considered.[32]

25.6.1.4 What is the Duration of Antidepressant Therapy?

Depression in old age is known to be associated with chronicity and high risk of relapse,[42,43] and antidepressant treatment has been shown to reduce the risk of relapse or recurrence in elderly depressed patients by 28%.[44] However, the duration of antidepressant therapy in patients who have recovered remains controversial. An expert consensus guideline on pharmacotherapy for depressive disorders in elderly patients recommends continuation of treatment with antidepressant medication at full therapeutic dose for at least 1 year if a patient has had a single episode of severe unipolar major depression; for a patient who has had two episodes, treatment should be continued for 1 to 3 years. If there is a history of three or more episodes, treatment should extend beyond 3 years.[45] For patients with multiple poor prognostic factors[32,43] (Table 25.10), longer duration of maintenance antidepressant treatment should be considered. In a primary care setting, the pragmatic approach for an elderly patient with first episode of depression who has achieved remission with antidepressant treatment would be to continue the full therapeutic dose for 12 months, and reassess the risk of recurrence at that point.

25.6.2 Psychosocial Management

Although the expertise for specific forms of psychotherapeutic intervention is mostly restricted to specialist settings, generic psychosocial management in the primary care setting is just as important and enhances the effect of pharmacotherapy.

All forms of psychological interventions, ranging from simple counselling to psychoanalytical psychotherapy, share a number of basic processes which, if used properly, can enhance insight into one's illness, improve compliance and may even be able to bring about change in one's life. These basic processes include: (a) listening and talking; (b) release of emotion; (c) giving information; (d) providing a rationale; (e) restoration of morale; (f) suggestion; (g) guidance and advice; and (h) establishing a therapeutic relationship.[46]

The primary care physician should aim to establish a therapeutic alliance with the patient, starting at the first assessment. Listening to a patient's complaint and providing support would help in establishing a therapeutic relationship. After a diagnosis is made, psychoeducation should be provided to both the patient and the family. They should be educated about the nature and pathogenesis of depressive illness, the proposed treatment, the expected side-effects of medication, the delay in response to antidepressant, and the expected duration of treatment after remission is achieved. It is often useful to employ a 'stress-diathesis model' when discussing the diagnosis with a patient. In this model, depression is reframed as the consequence of 'stress' (which may be biological such as a stroke or psychosocial such as bereavement) on the brain, resulting in an imbalance in brain chemistry, thereby, giving rise to a range of symptoms the patient experiences; whether an individual develops depression or not would depend on the patient's vulnerability (e.g. personality, coping skills, availability of support and genetic loading). The advantage of this model is that

Table 25.10 Factors associated with poor outcome in elderly depressed patients

Severe depression at baseline
Presence of psychotic symptoms
Executive function impairment
Slow initial recovery
Residual depressive symptoms
Comorbid psychiatric disorders
≥3 Past depressive episodes
Chronic stress
New or chronic physical illness
Poor physical functioning
Poor perceived social support
Family history of depression

it implies that depression is modifiable. The patient is encouraged to take an active role in modifying his or her vulnerability and combat 'stress' by complying with treatment, developing coping skills and seeking social support. By empowering the patient, he or she is more likely to comply with treatment in the long term.

Patients requiring formal psychological intervention should be referred to the specialist psychiatric service. Formal psychological interventions that have been found to be effective in treating depression in elderly people include interpersonal therapy,[47,48] problem-solving therapy,[49,50] cognitive behavioural therapy,[51] and mindfulness-based cognitive therapy.[52] For moderate-to-severe depression, the combination of pharmacotherapy and psychological treatment has been shown to be superior to either treatment given alone.[53,54]

25.7 Suicide in Elders

Suicide is one of the major causes of mortality in elderly patients with depression. The suicide rate in elderly people in Hong Kong has remained high and consistently accounted for 30% of all suicide deaths.[4] While the suicide rate across all age-groups in Hong Kong in 2012 was 12.7 per 100 000 population, the suicide rate among people aged ≥65 years was 25.4 per 100 000 population in the same year.[4] Similar trend has been reported elsewhere in the world.[20,55]

Although elderly suicide, as a phenomenon, is a result of complex interplay of biological, psychosocial and cultural factors, its prevention is intricately related to the early detection and optimal management of depression. Psychological autopsy studies reveal that suicides in elderly people are characterised by lethality of the act, fewer warnings to others as well as greater planning and resolve.[56] More importantly, it has been consistently shown that 70-95% of elderly suicide completers have a major psychiatric disorder, predominantly depression, at the time of death.[57-59] Other risk factors associated with completed suicide in elderly people include male sex, history of suicide, presence of physical illnesses, a personality characterised by methodical, rigid and emotionally restricted traits, being single, widowed or divorced, unemployment, indebtedness and poor social support.[58,60,61] However, controlled studies reveal that most of these factors appear to act through the overriding effect of depression.[20] In view of the central role of depression in the causation of suicide in elderly people and the fact that depression is highly treatable, targeted intervention for high-risk patients may be able to reduce rate of suicide in elderly people. Since over half of elderly suicide completers contact their general practitioners 1 month before their death (not necessarily presenting with mood symptoms or suicidal idea),[58,61,62] early detection in the primary care setting would be crucial.

Locally, the Elderly Suicide Prevention Programme (ESPP), a targeted intervention strategy, has been developed in the public old age psychiatric service aiming at early detection and treatment of elderly persons at risk of suicide.[63] The ESPP adopts a two-tiered multifaceted care management model. It facilitates early detection by targeted education programme aiming at the general public, frontline workers of social agencies with access to elderly in the community, social workers, and primary care physicians. In addition, it improves access to specialist psychiatric service by providing a short waiting time in a fast-track clinic, with coordinated multidisciplinary input and intensive follow-up of elderly patients at risk of suicide. The programme has been evaluated and shown to be associated with a reduction in 2-year completed suicide rate in old-age suicide attempters (suicide rate, 1.99% in the ESPP group compared to 7.58% in the pre-intervention cohort).[64]

25.8 Summary

Depressive disorders are common in elderly people. Despite standardised diagnostic criteria, the diagnosis is frequently missed in the primary care setting because ageing modifies the presentation of depressive disorder in elderly people. The impact of suboptimal detection and treatment is considerable as depression is associated with significant mortality and morbidity in elderly people. The increased awareness of depression in elderly people as well as the use of standardised screening instruments such as the GDS may help to address this problem. Effective and well-tolerated treatments such as the newer antidepressants are available and most patients respond favourably to these. Generic psychosocial interventions, feasible in the primary care setting, may be able to further enhance the effect of pharmacotherapy. The ability of primary care physicians to detect and treat depression early in elderly people with appropriate referral for difficult, complicated or suicidal cases is crucial in complementing the efforts in the public old age psychiatric service in the implementation of targeted intervention strategies to better manage elderly depression and prevent suicide in elderly people in Hong Kong.

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CHAPTER 26

Anxiety Syndrome

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OBJECTIVES

To understand the comorbidities of anxiety syndrome in elderly people, the common types of anxiety disorder and their corresponding pharmacological treatment, and the practical use and potential side effects of commonly prescribed medications. To present an overview of practical cognitive behavioural therapy for anxiety syndrome.

KEY POINTS

- Anxiety disorders are common in late life. Comorbidity with depression, somatisation, and medical illnesses are special considerations in elderly people.
- Comorbidity of depression and anxiety is frequent. Suicidal risk and disability are both increased in this type of comorbidity.
- There is a strong association between somatisation and anxiety disorder in elderly people. Somatisation may improve when anxiety and depression are treated.
- Real physical illness in a somatising anxious elderly person should not be overlooked.
- Although the term "neurasthenia" is widely used by Chinese elderly people, it is in the strict sense an outdated psychiatric diagnosis in the West.
- Common types of anxiety disorder include generalised anxiety disorder, panic disorder, adjustment disorder, post-traumatic disorder, and social phobia.
- Treatment of anxiety disorders includes recommendations on change of lifestyle, psychosocial treatment, and pharmacological treatment.
- Cognitive behavioural therapy is an effective form of psychotherapy for anxiety syndrome.

26.1 Epidemiology of Anxiety Disorders

26.1.1 The Worldwide Trend

Anxiety disorder is the most prevalent class of mental illness. In a large study by the World Health Organization (WHO), the lifetime prevalence of any anxiety disorder was 14.3% and 12-month prevalence was 8.3%.[1]

Although anxiety disorders in general are less prevalent among elderly people than among younger adults, they remain relatively common in late life. The US National Comorbidity Survey Replication found that the 12-month prevalences of the DSM-IV (Diagnostic and Statistical Manual of Mental Disorders, 4th edition) anxiety disorder, generalised anxiety disorder (GAD), and any phobia in a representative sample of those aged ≥65 years were 7.0%, 1.2% and 4.7%, respectively.[2] People with anxiety disorders make heavy use of medical services; despite this, most cases of anxiety disorder in late life are not diagnosed or treated.[3]

Anxiety disorders have a mean age of onset in the 20s. Left untreated, they tend to be chronic, with symptoms that are either continuous or episodic. Most primary anxiety disorders in late life have persisted from a younger age. New-onset anxiety in elderly people is often associated with depressive illness or medical disorders. At any age, anxiety disorders are more common in women than men.[3]

26.1.2 The Hong Kong Trend

In the Shatin Community Mental Health Survey in 1993, GAD had a high lifetime prevalence of 7.77% for males and 11.11% for females.[4] This was the most common psychiatric disorder in Shatin, next only to tobacco dependence.[4] A later community survey found that the 6-month prevalence of GAD was 4.1%.[5] A more recent territory-wide epidemiological study showed that the prevalence rate of GAD increased with age and the 1-week prevalence rate of GAD in elderly people aged 66-75 years was 5.52%.[6]

26.2 Comorbidities of Anxiety Syndrome

26.2.1 Comorbidity of Anxiety and Depression

The comorbidity of depression and anxiety was a consistent finding across all sites in the Cross-National Study (6-17%).[7] Persons with major depression were at increased risk of having panic disorder at all the sites. Suicide is an important consequence of depression and anxiety comorbidity. When anxiety disorders are comorbid with depression, the suicide attempt rate is increased, adding further burden on the family of sufferers.[7]

The impact of comorbidity in terms of increase in disability was shown in the WHO–Mainz Center study of a primary care population.[7] Degree of disability caused by depression and GAD was comparable with that induced by chronic somatic disorders. More importantly, comorbidity between different psychiatric disorders or psychiatric disorders and chronic somatic disorders, induced maximal rates of disability that were greatly superior to those of the 'pure' condition alone.

26.2.2 Somatisation and Anxiety Disorders in Elders

Epidemiological studies have consistently shown a strong positive correlation between number of somatic symptoms and anxiety symptom counts,[8] and also between functional somatic symptoms and diagnosis of panic disorder.[9] Somatisation disorder has been shown to be more common in older than younger panic disorder female patients, suggesting that somatisation may have a specific link with anxiety disorder in elderly people.[10]

Little data are available on the course and treatment of somatisation in elderly patients with anxiety disorder. Preliminary evidence indicates that somatisation may improve when anxiety and depression are treated. In a longitudinal study of 41 elderly persons referred to an old-age psychiatry service in south London, decreased anxiety and depression scores were accompanied by an increase in normalising attributional styles and a decrease in somatic attributional styles.[11]

26.2.3 Comorbidity of Anxiety Disorders and Medical Illnesses in Elders

A special consideration in elderly people is the increased likelihood of real physical illness associated with the somatisation symptoms of anxiety disorders. There is a complex interplay between anxiety, medical illness, and the medications used to treat the following conditions.[3]

- Realistic worry and pathologic anxiety could be consequences of physical illness.
- Anxiety itself might contribute to medical morbidity or mortality, for example, people with a high level of anxiety are at increased risk of hypertension, arrhythmias following myocardial infarction, and death from cardiovascular disease.
- Anxiety might be misdiagnosed as a medical condition or vice versa. Some medical conditions, such as hyperthyroidism, hypercalcaemia, pheochromocytoma, or paroxysmal atrial tachycardia, might mimic an anxiety disorder.
- Anxiety-like symptoms might be caused by toxicity from stimulants (e.g. theophylline, β-agonists, sympathomimetics, thyroxine, amphetamines, caffeine) or withdrawal from depressants (e.g. benzodiazepines, alcohol).
- Increased disability could result from excessive benzodiazepine use by people with physical illness and anxiety.

26.2.4 Neurasthenia in Chinese Elders

The term 'neurasthenia' is widely used by general practitioners and laymen in Hong Kong to refer to a non-specific type of mild psychiatric condition. Although it may serve the purpose of destigmatisation of a psychiatric disorder, such a term is in the strict sense an outdated psychiatric diagnosis. To understand the term 'neurasthenia', we must look at it from the historical perspective.

The concept of neurasthenia was introduced in 1869 by the American neurologist George Beard to describe a 'new' disease associated with profound physical and mental fatigability. Beard's clinical description was very broad and included more than 50 symptoms and signs. Charcot reclassified the symptoms in 1889, trying to isolate the most important ones: neuromuscular asthenia, brachialgia, dyspepsia with drowsiness, abdominal distension, constipation, and morbid mental state characterised by intellectual asthenia with some loss of memory, sad and irritable mood.[12] Many writers commented on over-inclusiveness of the Beardian concept of neurasthenia, especially its lack of precision and consistency. Currently, International Classification of Diseases (10th revision) retains the diagnosis of neurasthenia. While this diagnostic category is not used in some countries, it is widely used in others as an approved diagnosis, particularly in non-western countries such as China.[13]

The term 'neurasthenia' has fallen into disuse in the US since the early 20th century. At around the same time, the term 'neurasthenia' was transmitted to China, and translated into "*shenjing shuairuo*"神經衰弱in Chinese. According to traditional Chinese medicine, *shen* is emblematic of vitality, the capacity of the mind to form ideas, and the desire of the personality to live life. *Jing* refers to the meridians or channels

that carry *qi* (vital energy) and *xue* (blood) through the body. *Shenjing* means 'nerve' or 'nervous system'. *Shuai* (degenerate) and *ruo* (weak) may follow undue nervous excitement, producing a variety of psychic and somatic symptoms. The neurasthenic notion of weakness closely resembles the popular and non-stigmatising illness category of *xu*; therefore *shenjing shuairuo* readily found acceptance among both Chinese professionals (as a disease) and lay people (as an illness).[14]

26.2.5 Anxiety Disorders and Parkinson's Disease in Elders

Clinically significant anxiety symptoms occur in 20% to >50% of patients with Parkinson's disease. Notably, anxiety and depressive syndrome frequently co-occur. The degree of comorbidity is in excess of that found in patients without the disease. Besides GAD, patients can also experience social phobias and panic disorders.[15]

Several theories have been proposed to explain the phenomenon, though little is known. The anxiety could be a psychological reaction to the stress of the illness. Degeneration of subcortical nuclei and ascending dopamine, and norepinephrine and serotonin (5-hydroxytryptamine) pathways within the basal ganglia–frontal circuits may also be responsible for symptoms of anxiety. Episodic anxiety could also be associated with motor fluctuation. To date, evidence from treatment is sparse and of small sample size, so pharmacological agents should be used with careful evaluation of known risks against putative benefits.

26.2.6 Comorbidity of Anxiety Disorders with Dementia and Cognitive Decline

Anxiety disorders are frequently comorbid with dementia and cognitive decline. This comorbidity is partially due to the fact that cognitive impairment is common among elderly people. Symptoms of anxiety may also arise in response to concern about cognitive changes, and may further interfere with cognitive function via poor concentration, sleep impairment, and autonomic arousal.[16] In addition, longitudinal studies have demonstrated a causal relationship, suggesting that anxiety is predictive of the development of dementia or cognitive decline.[17,18] Nonetheless the reported prevalence of anxiety in patients with dementia varies widely, reflecting differences in methodology and difficulties in diagnosing anxiety in the presence of dementia.[16]

26.3 Common Types of Anxiety Disorders

The following is an abridged version of the criteria in the DSM-5 (Diagnostic and Statistical Manual of Mental Disorders, 5th edition),[19] and can serve as a quick revision of the clinical features of common types of anxiety disorder.

26.3.1 GAD

Characteristics of GAD include:

- excessive anxiety and worry, occurring more days than not for at least 6 months
- the person finds it difficult to control the worry
- may be associated with the following symptoms:
  - restlessness
  - easily fatigued
  - difficulty concentrating
  - irritability
  - muscle tension
  - sleep disturbance

GAD can be distinguished from non-pathological anxiety by:

- worries of GAD are difficult to control and interfere significantly with functioning. Worries of everyday life are perceived as more controllable and can be put off until later
- worries of GAD are more pervasive, pronounced, distressing, of longer duration, and frequently occur without precipitants
- everyday worries are much less likely to be accompanied by physical symptoms, e.g. excessive fatigue, restlessness, and irritability

26.3.2 Panic Disorder

Characteristics of panic disorder include:

- Recurrent unexpected panic attacks. A panic attack is an abrupt surge of intense fear or intense discomfort that reaches a peak within minutes, and during which four (or more) of the following symptoms occur:
  - palpitations
  - sweating
  - trembling
  - shortness of breath
  - feeling of choking
  - chest pain or discomfort
  - nausea or abdominal distress
  - dizziness
  - chills or heat sensations
  - paresthesias
  - derealisation or depersonalisation
  - fear of losing control or going crazy
  - fear of dying
- May be followed by one of the following:
  - persistent concern about having further attacks
  - worry about the implications of the attack (e.g. losing control, having a heart attack, going mad)
  - a significant change in behaviour related to the attacks
- Absence of agoraphobia

26.3.3 Agoraphobia

Characteristics of agoraphobia include:

- Marked fear or anxiety about two (or more) of the following five situations:
  - using public transportation
  - being in open spaces
  - being in enclosed places
  - standing in line or being in a crowd
  - being outside of the home alone
- The individual fears or avoids these situations because of thoughts that escape might be difficult or help might not be available in the event of developing panic-like symptoms or other incapacitating or embarrassing symptoms
- The agoraphobic situation almost always provokes fear or anxiety
- Agoraphobic situations are actively avoided, require the presence of a companion, or are endured with intense fear or anxiety
- The fear or anxiety is out of proportion to the actual danger posed by the agoraphobic situation and to the sociocultural context.
- The fear, anxiety, or avoidance is persistent, typically lasting for ≥6 months

26.3.4 Adjustment Disorders with Anxious Mood

Characteristics of adjustment disorders with anxious mood include:

- Emotional or behavioural symptoms in response to an identifiable stressor, occurring within 3 months of the onset of the stressor
  - marked distress that is in excess of what would be expected, or
  - significant impairment in functioning
- Lasts no longer than 6 months after the stressor has ceased

26.3.5 Post-traumatic Stress Disorder

Characteristics of post-traumatic stress disorder include:

- Exposure to actual or threatened death, serious injury, or sexual violence in one (or more) of the following ways:
  - directly experiencing the traumatic event(s)
  - witnessing, in person, the event(s) as it occurred to others
  - learning that the traumatic event(s) occurred to a close family member or close friend.
  - experiencing repeated or extreme exposure to aversive details of the traumatic event(s)
- Presence of one (or more) of the following intrusion symptoms associated with the traumatic event(s), beginning after the traumatic event(s) occurred:
  - recurrent, involuntary and intrusive distressing memories of the traumatic event(s)
  - recurrent distressing dreams
  - dissociative reactions (e.g. flashbacks) in which the individual feels or acts as if the traumatic event(s) were recurring
  - intense psychological distress on exposure to cues
  - marked physiological reactions to cues
- Persistent avoidance of stimuli associated with the trauma
  - avoidance of or efforts to avoid distressing memories, thoughts, or feelings about or closely associated with the traumatic event(s)
  - avoidance of or efforts to avoid external reminders that trigger the distressing memory, thoughts, or feelings about or are closely associated with the traumatic event(s)
- Negative alterations in cognitions and mood associated with the traumatic event(s), beginning or worsening after the traumatic event(s) occurred, as evidenced by two (or more) of the following:
  - inability to remember an important aspect of the traumatic event(s)
  - persistent and exaggerated negative beliefs or expectations about oneself, others, or the world
  - persistent, distorted cognitions about the cause or consequences of the traumatic event(s) that lead the individual to blame himself / herself or others
  - persistent negative emotional state
  - markedly diminished interest or participation in significant activities
  - feelings of detachment or estrangement from others
  - persistent inability to experience positive emotions
- Marked alternations in arousal and reactivity associated with the traumatic event(s), beginning or worsening after the traumatic event(s) occurred, as evidenced by two (or more) of the following:
  - irritable behaviour and angry outbursts typically expressed as verbal or physical aggression toward people or objects
  - reckless or self-destructive behaviour
  - hypervigilance
  - exaggerated startle response
  - problems with concentration
  - sleep disturbance

26.3.6 Social Anxiety Disorder (Social Phobia)

Characteristics of social anxiety disorder include:

- Marked fear or anxiety about one or more social situations in which the individual is exposed to possible scrutiny by others
- The individual fears that he or she will act in a way or show anxiety symptoms that will be negatively evaluated
- The social situations almost always provoke fear

or anxiety
- The social situations are avoided or endured with intense fear or anxiety
- The fear or anxiety is out of proportion to the actual threat posed by the social situation and to the sociocultural context

26.4 Assessment

26.4.1 Assessment of Anxiety Disorders

The Hospital Anxiety and Depression Scale (HADS) developed by Zigmond and Snaith[20] in 1983 comprises 14 questions, 7 each on anxiety and depression. Each question is scored on a scale of 0 to 3. It has been validated among Chinese elders in general practice in Hong Kong.[21] The optimal cut-off depression score of 6 and anxiety score of 3 were lower than those found by the original authors. Nearly half of elderly people found to have a positive HADS score had never been diagnosed with any psychological illness, showing that HADS has great potential to serve as a screening instrument for Chinese elders in general practice.

26.4.2 Assessment of Panic Disorder[22]

The following should be assessed for panic disorder:
- date and time of the attacks
- circumstances of the attacks — including places, whether alone or accompanied, and what sort of activity at that period, expected or unexpected
- duration of the attack
- the physical sensations, the psychological feelings and thoughts, and the behaviour in response to the attack
- the outcome of the attack, especially any avoidant behaviour, both short term and long term

26.5 Treatment of Anxiety Disorders in Late Life

26.5.1 General Recommendations[22]

- Avoid caffeine
- Avoid excess alcohol consumption and nicotine
- Exercise moderately to relieve tension and improve sleep
- Monitor over-the-counter medication use
- Adhere to prescribed treatment regimen

26.5.2 Psychosocial Treatment

- Cognitive behavioural therapy (CBT)
- Graded exposure to feared stimulus
- Relaxation training and breathing exercises
- Explore and solve any underlying family or social problems

26.5.3 Pharmacological Treatment

26.5.3.1 Selective Serotonin Reuptake Inhibitors

Antidepressants are effective for GAD in late life, both in the presence and absence of co-morbid depression. A selective serotonin reuptake inhibitor (SSRI) is the first treatment of choice. There is no systematic evidence to support the use of antidepressants in other anxiety disorders in this age-group, but extrapolation from studies in younger adults suggests that SSRI would also be the treatment of choice for panic disorder.[23] Caution should be exercised in those who are at high risk of falls and fractures as a higher risk has been reported among elderly people prescribed SSRI antidepressants.[24,25]

The side-effects of SSRIs include: gastrointestinal effects; insomnia; anxiety and jitteriness early in treatment; sexual dysfunction; and rarer side-effects such as dystonic reactions, low sodium, and seizures. Sedation and anticholinergic effects are uncommon, thus use is more appropriate in elderly people at risk of cognitive impairment. SSRIs should not be stopped abruptly as patients with anxiety spectrum disorders are particularly sensitive to discontinuation symptoms. The dose should be reduced as slowly as tolerated over several weeks to months.[26]

26.5.3.2 Serotonin and Norepinephrine Reuptake Inhibitors

Venlafaxine is useful for GAD in older patients with comparable efficacy and tolerability compared with its use in younger patients.[27] Duloxetine is another approved serotonin and norepinephrine reuptake inhibitors (SNRIs) that has demonstrated efficacy in older patients with GAD.[28]

Side-effects of SNRIs include nausea, headache, constipation, dizziness, dry mouth, and somnolence. Systematic monitoring of blood pressure during treatment is required with venlafaxine as blood pressure may be elevated at higher doses.[29] It is best avoided if the patient is at risk of arrhythmia.

26.5.3.3 Benzodiazepines

Benzodiazepines provide rapid symptomatic relief from acute anxiety states. However, they should only be used to treat anxiety that is severe or subjecting the individual to extreme distress because of the potential to cause dependence and withdrawal symptoms. Benzodiazepines should be used at the lowest effective dose for the shortest period of time (maximum 4 weeks), while medium and long-term treatment is formulated.[30] These recommendations should be applied to the majority of patients. A very small number of patients with severely disabling anxiety may benefit from long-term treatment with a benzodiazepine and these patients should not be denied treatment.

The National Institute for Health and Care Excellence (NICE) recommends that benzodiazepines should

not be used to treat panic disorder. They should be used with care in post-traumatic stress disorder.[30]

The side-effects of benzodiazepines include: sedation, cognitive impairment, ataxia, dependence, withdrawal reactions and rebound anxiety, and paradoxical disinhibition. They are also associated with fracture[31] and disability in mobility and activities of daily living.[32]

For the classification of benzodiazepines, please refer to Chapter 27.

26.5.3.4 Buspirone

Buspirone is an agonist at type 1A serotonin (5-hydroxytryptamine[1A]) receptors. Buspirone is effective in the treatment of GAD but does not produce useful effects in panic disorder. It may have some modest effect in social phobia. In contrast to benzodiazepines, buspirone needs to be administered for several days or weeks before a useful anxiolytic effect is obtained. For reasons that are not fully understood, patients who have previously taken benzodiazepines tend to do badly on buspirone.

The side-effects of buspirone include: nausea, dizziness, and headache. The advantages are: no sedation, less cognitive impairment, and less dependence or tolerance.

26.5.3.5 Pregabalin

Pregabalin binds to the α2δ subunit of the voltage-dependent calcium channel in the central nervous system.[33] It is effective in the treatment of GAD in elderly people, with improvement in both psychic and somatic symptomatology. The onset of efficacy of pregabalin is about 2 weeks after initiating treatment.[34]

The side-effects of pregabalin include: dizziness, sedation, dry mouth, amblyopia, and impaired coordination.

26.5.3.6 Antipsychotics

The use of typical antipsychotic drugs in the management of anxiety is not recommended, because of the risk of adverse side-effects. The role of an atypical antipsychotic is less clear. There is evidence that Quetiapine XR monotherapy is effective in the short term in improvement of anxiety symptoms in older patients with GAD.[35] Nonetheless adverse effects including increased risk of death, stroke, and extrapyramidal symptoms cannot be ignored.

26.5.3.7 Tricyclic Antidepressants

Tricyclic antidepressants (TCAs) are effective against symptoms of anxiety, but the use of these drugs is limited by the risks they pose to physically ill patients, and their toxicity in overdose. They are not supported by the NICE guideline, except as part of a specialist Step 4 intervention.[30]

The side-effects of TCAs include: cardiotoxicity (especially in drug overdose); agitation and jitteriness early in treatment; anticholinergic side-effects, e.g. dry mouth, blurred vision, constipation, urinary retention; drowsiness; sexual dysfunction; and postural hypotension.

26.5.3.8 Beta-blocking Drugs

These have been in use for anxiety for the past 30 years and have an established place in the treatment of anxiety in clinical psychiatric practice. Their major effects are peripheral, and symptoms that are most responsive include awareness of fast heartbeat, flushing, palpitations, and tremor. They are useful in the treatment of performance anxiety in acute stress situations, such as speaking in public and playing a musical instrument. They have no sedative effects or sensorimotor impairment and have no risk of dependence.

26.5.4 Practical Pharmacotherapy for Anxiety[26]

26.5.4.1 GAD[30]

SSRIs should be used as first-line treatment. SNRI and pregabalin are alternative choices. Benzodiazepines should not be used beyond 2-4 weeks. SSRIs should initially be prescribed at half the normal starting dose for treatment of depression as initial worsening of anxiety may be seen when treatment is started. The dose can be titrated upwards to the normal antidepressant range as tolerated. The same advice applies to venlafaxine and duloxetine. Response is usually seen within 6 weeks and continues to increase over time. The optimal duration of treatment has not been determined but should be at least 1 year.

26.5.4.2 Panic Disorder

SSRIs should be used as first-line treatment. The same starting dose and dose titration to GAD should be used. Onset of action may be as long as 6 weeks. The optimal duration of treatment is unknown, but should be at least 8 months. Benzodiazepines are not recommended as a first-line monotherapy. Nonetheless time-limited adjunctive use of a benzodiazepine with an antidepressant has proven clinically useful. The addition of clonazepam to SSRIs at the start of treatment in mixed-age patients can lead to a more rapid response.[36] Imipramine and clomipramine, the tertiary amine TCAs that have been found to be effective in treating panic disorder in younger persons, are not generally recommended for use in elderly persons owing to their potential

to cause orthostatic hypotension, cardiotoxicity, and anticholinergic side-effects.[36]

26.5.4.3 Adjustment Disorder with Anxious Mood

Short-term benzodiazepines are the agents of choice for treating patients with overwhelming anxiety that needs to be brought under rapid control. They are particularly useful for the anxiety and sleep disturbance that commonly occur and which, if left untreated, severely affect the individual's coping ability after a stressor. There is the risk, however, that some patients will find it hard to stop; therefore benzodiazepines should be reserved for patients where extreme distress is disrupting their normal coping strategies.

26.5.4.4 Post-traumatic Stress Disorder

A short course of a benzodiazepine may be required immediately following the incident to promote sleep and to help minimise mental rehearsal of the trauma. SSRIs should be considered as first-line treatment. Lower starting doses are also required, with high doses often being required for full effect. Response is usually seen within 8 weeks, but can take up to 12 weeks. Treatment should be continued for at least 6 months and probably longer.

26.5.4.5 Social Phobia

SSRIs should be used first. A benzodiazepine may be useful when necessary. Response is usually seen within 8 weeks and treatment should be continued for a year and probably longer.

26.6 Practical CBT for Anxiety

26.6.1 The Cognitive Behavioural Model

CBT is one of the most effective treatments for anxiety disorders across all ages among the adult population.[37,38] It not only enjoys strong empirical support, but is also one of the treatment options suggested by the NICE clinical guidelines for various anxiety disorders.[30,39]

CBT, as its name indicates, represents an integration of two distinct schools of psychotherapy: cognitive therapy and behaviour therapy. Cognitively, it gears patients to developing a cognition-oriented coping plan, realising and changing their irrational thoughts. Behaviourally, it prepares and guides patients to face stress-producing real-life situations through graded in-vivo exposure, role play, relaxation training, and contingent reinforcement. It is generally believed that the synthesis of these two approaches provides a comprehensive treatment with greater efficacy and applicability than cognitive therapy alone.

In general, the CBT approach not only aims to modify maladaptive thinking, but to assist the development of adaptive cognitive constructs. The goal of treatment is also to assist the implementation of more functioning and adaptive cognitive constructs for processing information relating to the self, the future, and the world.[40,41] Nonetheless as the manifestation and underlying mechanism varies among different types of anxiety disorder, this implies that specific components and method of delivery of the CBT intervention and treatment focus will be different.

26.6.2 The Cognitive Model

Patients with anxiety disorders consistently and continuously perceive daily events as out of control.[42] As such, some cognitive theorists suggest that the perceived lack of control and the sense of helplessness is one of the central themes in the study of anxiety.[43]

Other theorists suggest that systematic biases in processing information, such as over-generalising or interpreting ambiguous stimuli in a catastrophic- or threat-related manner trigger maladaptive anxiety in the form of intense worry and fear.[44,45] It is believed that these distorted ways of information processing are either self-created or acquired through learning. On this point, Borkovec et al proposed that pathological worrying, along with intense fear and distorted beliefs, help create and maintain anxiety in a maladaptive manner, pushing an individual towards the disordered end of the anxiety continuum.[46] It has also been theorised that worrying is a cognitive maladaptive tactic aimed at avoidance of aversive images and intense, unpleasant emotions generated by a distorted way of thinking and misinterpretation of information received. This form of cognitive thinking has been coined by Roemer and Orsillo as experiential avoidance, a maladaptive cognitive tactic for dealing with anxiety that once formed can become self-maintaining and self-reinforcing.[47]

26.6.3 Cognitive Training in Action

Following the cognitive perspective, this aspect of treatment usually starts off with problem identification. This involves educational elements including recognition, labelling, and self-monitoring of physiological and emotional cues. The aim is to assist patients to eventually control behavioural reactions that often accompany these cues.

As treatment progresses, it slowly moves on to tackle 'faulty' cognitive beliefs through cognitive restructuring, conceptualising the process as a problem-solving endeavour. This process usually involves a series of repeated practice that encompasses identification of immediate thoughts, replacement of maladaptive thoughts with more adaptive ones, attribution retraining (learning how to distinguish between internal-external, global-local, and stable-

unstable attributions), and then finally implementing alternative attributions and thoughts in given circumstances, and evaluating the implementations.

26.6.4 The Behavioural Model

From the behavioural perspective, behaviour is learnt from one's experiences with significant others, peers, and the environment. Daily events are thought to be influential elements in influencing one's learning opportunities, and in turn impose a potentially pathogenic experience, making an impact on learning and conditioning.[37] It is theorised that in this interactive process, one learns from one's significant others as well as from one's own experience that certain situations, objects, or events that are seemingly benevolent to others might be extremely anxiety-provoking for oneself. At the same time, it is believed that the employment of certain maladaptive but immediately stress-relieving actions have the effect of promoting and encouraging the continuing existence of such behaviour through the mechanism of reinforcement.[48]

26.6.5 Behavioural Exposure Training in Action

The purpose of exposure training is to assist patients to unlearn existing dysfunctional constructs and behaviour and to relearn a more adaptive one.[49-51] Through these procedures, not only relevant cognitions, emotions, and physiological responses are identified, this also provides an opportunity to attenuate anxiety, as well as to break the pattern of avoidance and escape.[49,51,52]

To operationalise the behavioural model in the treatment of anxiety disorders, two major types of exposure procedures are developed. (1) In-vivo exposure employs a direct real life approach. For example, if the patient has social phobia, the patient may be asked to give a speech in front of a small group of people. (2) Imaginal exposure involves asking the patient to imagine an anxiety provoking situation. These two types of exposure procedure are usually employed either in conjunction or separately with a graded approach, where mildly feared situations are targeted first, followed by more strongly feared ones. This usually involves the construction of exposure hierarchies, in which feared situations are ranked according to the anticipated fear reaction. Generally, higher-level exposures are not encouraged until the patient has mastered the ability to adaptively cope with lower levels of anxiety provoking situations.

26.7 Summary

The outcome for anxiety disorder is one of guarded optimism. Many people become chronic (23%) or have subclinical symptoms (47%) at 6-year follow-up. Those high in neuroticism are at greater risk of persistent anxiety.[53] Pathological anxiety should not be ignored because it not only causes suffering in itself, it also creates future distress when allowed to proceed unchecked.

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CHAPTER 27

Sleep Disorders in Old Age

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OBJECTIVES

- To be able to identify the causes of insomnia in older adults.
- Be able to carry out a comprehensive assessment of sleep disorders.
- Understand the importance of sleep hygiene, in addition to pharmacotherapy and cognitive behavioural therapy.
- Know the symptoms of obstructive sleep apnoea (OSA), and treatment options.
- Identify other problems that cause poor sleep in older adults and their management.
- Learn about rapid eye movement sleep behaviour disorder, its diagnosis, and management.

KEY POINTS

- Sleep disturbance is common in older adults. Although partly due to age-associated physiological changes, a significant proportion of these older people who complain of insomnia have impaired physical and mental health and thus merit more medical attention.
- Persistent insomnia that lasts longer than 4 weeks is regarded as significant insomnia.
- Multiple factors can disrupt sleep, including poor sleep habits, medical illnesses, medications, and neurodegenerative and psychiatric disorders.
- Management of sleep disorders in older adults includes improving sleep hygiene, pharmacotherapy, cognitive behavioural therapy, and environmental modifications.
- Short-acting benzodiazepines and non-benzodiazepine hypnotics are preferred in older people.
- If a hypnotic is to be used, low dose, short-term use, and intermittent dosing are recommended.
- The risk factors for OSA are obesity, male gender, older age, postmenopausal women, smoking, and alcohol.
- Treatment of OSA includes conservative, medical, and surgical interventions.
- Continuous positive airway pressure is the first-choice therapy for OSA.

27.1 Epidemiology

Few local studies have addressed the important issue of sleep disorders in elderly people. In a survey that examined the sleep habits and 1-year prevalence of sleep disturbances (difficulty in falling asleep, broken sleep and early morning wakening) as well as insomnia (subjectively inadequate or poor sleep) in the Hong Kong elderly population,[1] occasional or persistent sleep disturbance was reported by 75% and insomnia by 38.2%. Advanced age was associated with a higher rate of sleep disturbance, and females reported a higher rate of insomnia. Sleep disturbance is very common among Hong Kong elderly people. Although this may be partly due to age-associated physiological changes, those with a concomitant complaint of insomnia often have impaired physical and mental health and thus merit more medical attention. A local study from a general practice clinic, however, reported a disproportionately low prevalence of subjects who took sleeping pills within a 1-year period (2.8%), and revealed that the number of night-time awakenings increases steadily with increasing age.[2] In another local survey of 1034 older adults, 0.8% reported a history of sleep-related injury.[3] The estimated prevalence of rapid eye movement (REM) sleep behaviour disorder (RBD) was 0.38%.[3]

In large surveys in international literature, reports of the prevalence of sleep disturbances among older adults aged ≥65 years are similar, with 25-45% had insomnia or poor sleep quality.[4-6] The symptoms of insomnia appear to increase with age and are more prevalent in women.[7] Daytime somnolence is another common complaint among the older population. Napping occurs in approximately one-third of older community-living adults.[6,8]

Other primary sleep disorders common in older

people are the movement disorders: restless leg syndrome (RLS) and periodic limb movements of sleep (PLMS), sleep-disordered breathing (SDB) or obstructive sleep apnoea (OSA), RBD, and advanced sleep phase syndrome.

27.2 Definition

According to the International Classification of Sleep Disorders (ICSD) version 2,[9] sleep disorders are classified into eight groups on the basis of their pathophysiological features (Table 27.1).[10]

Insomnia lasting only a few days is often a result of acute and transient stress and is usually regarded as a normal phenomenon. Insomnia that lasts longer than a few weeks is considered significant.

27.3 Sleep Problems in Older People

27.3.1 Ageing and Sleep

Sleep complaints in older people may be viewed as a multifactorial geriatric syndrome.[11] Among these complaints or problems, the most common ones are insomnia and daytime sleepiness. There are often multiple underlying factors, some or all of which may be modifiable. It is helpful to first understand sleep in normal ageing.

27.3.1.1 Decreased Total Night-time Sleep

Compared with younger persons, elderly people tend to achieve less total night-time sleep. They have more night-time arousals and awakenings. The result is a reduced sleep efficiency — percentage of time in bed-spent asleep. The deepest stages of non-REM sleep are frequently reduced or non-existent in elderly people; whether or not there is reduced REM sleep is controversial.[11,12] It has been observed that a marked reduction of REM sleep in healthy older adults is associated with higher mortality.[13] In a local cohort study, mortality increased sharply with time spent ≥10 hours in bed at night among community-living older adults. Night-time sleep ≥10 hours was associated with 1.75 times (men) and 2.88 times (women) higher mortality risk over 5 years, independent of frailty.[14]

Table 27.1 The ICSD-2 lists 81 sleep disorders in eight major categories[10]

1. Insomnias
2. Sleep-related breathing disorders
3. Hypersomnias of central origin
4. Circadian rhythm sleep disorders
5. Parasomnias
6. Sleep-related movement disorders
7. Isolated symptoms, apparently normal variants, and unresolved issues
8. Other sleep disorders

Abbreviation: ICSD-2 = International Classification of Sleep Disorders, version 2

27.3.1.2 Phase Shift

Older people tend to go to sleep earlier in the evening and to wake earlier in the morning. Early morning awakening is a common complaint in elderly people. If the onset of sleep is not correspondingly earlier, sleep deprivation and excessive daytime sleepiness may result.

27.3.1.3 Daytime Napping

Daytime napping reduces the drive for sleep at the usual bedtime hour with consequent delayed sleep onset and a further decrease in the duration of night-time sleep.

27.3.1.4 Day-night Reversal

The development of a 'night-owl' pattern is less common, with bedtime delayed until the early morning hours and then continuing until mid-afternoon.

27.3.2 Factors That May Disrupt Sleep in Older People

It is important to bear the following multiple factors in mind when evaluating sleep complaints in elderly patients.

27.3.2.1 Poor Sleep Habits

An irregular sleep-wake pattern can undermine the ability of the circadian system to effectively offer sleepiness and wakefulness at appropriate times. Caffeine intake can impair night-time sleep. Alcohol consumption in the evening, while initially sedating, prevents deeper sleep and increases arousals during the later part of the night. Excessive wakeful time in bed may increase arousal that is reinforced nightly.

27.3.2.2 Medical Conditions

Acute and chronic medical illnesses, such as arthritis (pain), prostatic hypertrophy, detrusor instability and cardiovascular (congestive heart failure, angina), gastrointestinal (gastroesophageal reflux disease, constipation, ulcer syndrome), and pulmonary diseases (chronic obstructive airway disease, rhinitis with nasal obstruction), may precipitate sleep disruption. Sleep disorders are common among patients with Parkinson's disease (PD; see Chapter 34.3.3) due to periodic limb movement disorder or depression. Pain and discomfort may delay sleep onset and shorten the duration of sleep. Deterioration of the sleep-wake cycle can accompany neurodegenerative disorders, particularly Alzheimer's disease.

27.3.2.3 Medications

Some antidepressants (particularly selective serotonin reuptake inhibitors), decongestants, bronchodilators, antihypertensives, corticosteroids, levodopa, and

many other medications can have a stimulating effect and thereby disrupt sleep. Diuretics can promote repeated awakening to micturate. Medications with sedating side-effects can produce excessive daytime sleepiness that in turn impairs the drive to sleep at night time. Alcohol induces sedation initially, but increases intermittent wakefulness towards the end of the night.

27.3.2.4 Psychiatric Disorders

Numerous psychiatric disorders are associated with insomnia, such as major depressive disorders, dementia, schizophrenia, and anxiety disorders. Significant life events such as physical limitations, loss of loved ones, and change of living environment also contribute to insomnia.

27.3.2.5 Nocturnal Polyuria Syndrome

The nocturnal polyuria syndrome (commonly known as nocturia) is a common condition among older people that causes interrupted sleep.[15] The syndrome can be due to medications such as diuretics taken too late in the evening, a high water intake at dinner, caffeine or alcohol in the evening, or age-related physiological changes such as detrusor instability of the bladder, benign prostatic hypertrophy in older men, vaginal atrophy and thus reduced sphincter control in older women, or reduced bladder volume due to ageing. The decline or even reversal in the circadian pattern of urine production, the reduced urine concentrating ability of the kidneys, and the higher plasma natriuretic hormone in old age especially at night time also contribute to a larger urine volume production at night among older individuals. These combined factors lead to nocturnal polyuria syndrome that can be managed pharmacologically and behaviourally.

Medications

Various drugs have been studied, including desmopressin (DDAVP), tricyclic antidepressants (imipramine), detrusor antispasmodics (oxybutynin, tolterodine), and even frusemide. In one study, frusemide given 6 hours before bedtime significantly reduced nocturnal polyuria. The proposed mechanism was that frusemide reduced intravascular volume and renal blood flow after the initial 6 hours of diuresis, thereby reducing nocturnal urine production. In the case of DDAVP, although highly effective, concern about the associated hyponatraemia and fluid retention has limited its popularity.

Behavioural Management

Behavioural management should be suggested for every patient with this syndrome as it is free from side-effects and effective in some cases. It includes reducing evening fluid intake as well as caffeine and alcohol consumption, and prompted voiding before bedtime for those with cognitive impairment and night-time incontinence.

27.3.3 Adverse Health Outcomes Associated with Sleep Disorders

Poor sleep has been reported to be associated with several important health outcomes in older people.

27.3.3.1 Poorer Health and Poorer Physical Function

Studies in community-living older people have demonstrated the association between poorer health-related quality of life and poor sleep quality.[6,16,17] Older persons with poorer sleep and lower sleep efficiency have poorer physical performance, evidenced by lower grip strength, slower walking speed, and inability to stand up from a chair without assistance.[18]

27.3.3.2 Falls

Poorer sleep is associated with increased falls and accidents among the older population. Both untreated insomnia and insomnia un-responsive to hypnotics have been shown to be associated with higher risk of fall in a large nursing home population.[19]

Similarly, among community-living older adults, short sleep duration of <7 hours per night and fragmented sleep with low sleep efficiency has also been linked to an approximately 30-40% higher risk of fall in the following year.[20]

27.3.3.3 Cognitive Impairment

Sleep disturbance has been associated with cognitive impairment in various cohort studies among older adults. Short sleep duration, daytime napping, chronic insomnia, and duration of waking after sleep onset have all been found to be related to impaired cognition.[17,21,22] In a local study of 3000 community-living older adults, phase shift (earlier bedtime and earlier wake up time) was associated with lower cognitive function.[23]

27.3.3.4 Mortality

Long sleep-onset latency (time to sleep onset after going to bed) of >30 minutes, and a low sleep efficiency of <80% have been associated with higher risk of mortality among older community-living individuals.[13] Both short and long night-time sleep duration have been reported to be related to excessive mortality risk: results were not always consistent. In a local study of several thousand community-living older people, only long night-time sleep duration (≥10 hours) was associated with higher mortality, independent of physical frailty.[14]

27.4 Assessment

27.4.1 History

The two most common symptoms are insomnia and excessive daytime sleepiness. Insomnia is the subjective complaint of restless, disturbed, shortened, or non-restorative sleep. It is important to distinguish between genuine poor sleep and unrealistic expectation or inaccurate perception of the quantity and continuity of sleep. Excessive daytime sleepiness is the inability to maintain wakefulness during the daytime, and the tendency to fall asleep unintentionally and at inappropriate times.

The bed partner should be interviewed as a collateral informant, particularly about arousals during sleep, breathing pattern and movements. The patient may be instructed to keep a sleep log or diary to document the hours of bedtime and awakening, the time in bed and day-time naps. Caffeine and alcohol intake, use of sleeping pills, time of meals and exercises, use of the toilet during sleep time, and noises that disturb sleep can also be documented.

Ten questions to ask[24]:

- What is the time of retiring and rising and the variation between weekdays and weekends or holidays?
- Does your sleep restore alertness or vigour?
- How much sleep do you need to feel ideally alert and energetic?
- How often is that amount of sleep obtained in relation to the nightly average?
- How were your sleep habits and requirements previously?
- Is sleep difficult to get into, disturbed during its course or foreshortened?
- Is sleepiness during waking hours a problem? Do you have episodes of muscle weakness associated with laughter (cataplexy)? Do you or your bed partner notice intermittent snoring, gasping or gagging? Do you have leg twitches in sleep? Ask about sleepwalking and hallucinations during sleep.
- Do you take any medication, alcohol or caffeine-containing drinks and how much?
- Do you have any personal emotional or physical problems (e.g. pain)?
- What is your sleep environment like? What do you do before going to bed?

27.4.2 Physical Examination

- Obesity
- Enlarged tonsils, big tongue, small jaw, short neck
- Pallor
- Neurological deficits
- Thyroid and adrenal diseases
- Hypertension
- Heart failure
- Renal failure
- Chronic obstructive airway diseases

27.4.3 Investigations

- Blood tests: haematological tests, renal and liver function, thyroid function
- Toxicology screening may be needed
- Polysomnography (PSG): details of the duration, distribution and transition pattern of arousals and different sleep stages, sleep efficiency. Indications for PSG include SDB, sleep-onset REMs in narcolepsy, continuous positive airway pressure titration for treatment of SDB, treatment evaluation of SDB, and the diagnosis of RBD.[25] Results from PSG are subject to night-to-night variability in sleep quality, environmental factors, and patient positioning.[26,27]
- Actigraphy: a cost-effective method to assess sleep-related problems that can be carried out in the patient's home. Measurement is based on the detection of body movements recorded by wristwatch-like devices. The results largely correlate with PSG-based results in the investigation of SDB and PLMS.[28] It can also be used to evaluate interventions for sleep-related disorders, although it may overestimate sleep time in those lying in motionless wakefulness.[28] In addition, discrepancies between sleep diary measures and actigraphy can be affected by subjective quality of sleep, gender, cognitive function, and disability.[29] Errors can be minimised by repeating measurements over a number of days, or by using multiple methods.
- Multiple Sleep Latency Test: narcolepsy is diagnosed in the presence of REM sleep latency of <5 minutes.

27.5 Treatment of Insomnia in Older People

27.5.1 General Measures

Observe the following rules of sleep hygiene[24]:

- Follow a regular sleep schedule: Go to bed at the same time each day. Avoid naps during the day. Get up at the same time each morning on weekdays and weekends.
- Establish a bedtime routine: A regular pattern of activities (brushing teeth, washing, setting the alarm clock) can set the mood for sleep.
- Maintain a proper sleep environment (dark and quiet, right temperature).
- Use the bedroom primarily as a place for sleep, not for other activities associated with wakefulness.
- Avoid substances that interfere with sleep, e.g. alcohol and caffeine.
- Exercise regularly, but not in the late evening.

Daily exercise and exposure to daylight can help reinforce the circadian cycle.
- Relax, e.g. read for a while, warm bath, warm milk, light music, aromatherapy, etc.

27.5.2 Pharmacotherapy

27.5.2.1 Hypnotic Agents

Selected patients may benefit from temporary use of sleep-promoting medication. If hypnotics are considered, the first choice would be a short-acting benzodiazepine. Non-benzodiazepine hypnotics (Table 27.2[9]) such as zopiclone and zolpidem are newer choices. Generally, a low dosage and short-term use are recommended. Intermittent dosing has advantages of minimising dependence and withdrawal effects. Patients may be given guidelines on use, such as the recommendation that a hypnotic agent be used no more than 2 to 4 nights per week. The availability of a sleep medication may be reassuring to the patient on particularly difficult nights. That reassurance will likely decrease the patient's distress on nights when no medication is used.[30]

Hypnotic agents should not be regarded as the ultimate solution to a sleep problem. They should be used under limited circumstances, following evaluation of the patient's symptoms and in the context of good sleep habits.

27.5.2.2 Hypnotic Use in Frail Older Adults and Falls

While hypnotics may be used to promote sleep or prolong sleep, their use is not without risk in older adults, in particular those at high risk of fall. Older patients with cognitive impairment, a history of falls, or PD are at higher risk of fall with the associated significant morbidity and even mortality. Alternatives should be sought and, perhaps to a certain degree, insomnia may have to be tolerated.

27.5.3 Cognitive Behavioural Therapy for Chronic Insomnia

27.5.3.1 Stimulus Control Therapy

The theoretical basis for stimulus control therapy is rooted in classical conditioning theory. Stimulus control instructions limit the amount of time patients spend awake in the bed and bedroom. The fundamental goals are to de-condition pre-sleep arousal and re-associate the bed and bedroom environment with rapid-onset and well-consolidated sleep. Below are typical instructions.
- Keep a fixed wake time 7 days a week, irrespective of how much sleep achieved during the night
- Avoid any behaviour other than sleep or sexual activity in the bed or bedroom
- Sleep only in the bedroom

Table 27.2 Non-benzodiazepine drugs used as hypnotics[9]

Drug	Action	Remark
Chloral hydrate	• Rapid onset (30 minutes) • Duration of 6-8 hours • Dose of 0.5-2 g	• Gastrointestinal irritation • Can lead to transient increased levels of warfarin and risk of haemorrhage in patients on anticoagulant
Zolpidem (Stilnox)	• Dose of 5-10 mg • Rapid onset • Half-life of 1.5-4.5 hours	• Imidazopyridine. Not benzodiazepine but binds selectively to a subset of benzodiazepine receptors • Impairs short- and long-term memory, psychomotor performance, postural sway • May cause tolerance, confusion, memory and behavioural disturbance, psychotic reaction • Side-effects: drowsiness, dizziness, headache, gastrointestinal upset • Abuse potential less than benzodiazepines, less enjoyable • Safer in overdose, reversed by benzodiazepine antagonist flumazenil
Zopiclone (Imovane)	• Optimal dose of 7.5 mg • Rapid onset • Half-life of 4 hours • No hangover effect	• Chemically unrelated to Zolpidem and benzodiazepines, but similar pharmacological actions — sedative, hypnotic, anticonvulsant, muscle relaxant, and anti-aggressive • Little evidence that it offers any advantage in terms of its potential to induce tolerance, withdrawal, or dependence • Fatalities following overdose reported, which is managed with flumazenil
Melatonin	• Dose of 1-5 mg • Optimal dose not determined	• Pineal gland manufactures and releases serotonin that is catalysed in vivo to melatonin — a natural endogenous regulator of sleep and light-dark accommodation • Safe and minimal side-effects (over-sedation). No potential for physical dependency or addiction • Available as a health product
Chlormethiazole	• Rapid onset and action, short half-life • Dose of 250-500 mg	• Anticonvulsant properties, can be used in alcohol withdrawal • Indicated for use as a hypnotic only in elderly people although they may be more sensitive • Dependence and abuse reported • Caution in cardiac, pulmonary disease, and hepatic impairment

- Leave the bedroom around 15 to 20 minutes after waking
- Return to bed only when sleepy

27.5.3.2 Sleep Restriction Therapy

This technique is a systematic, controlled form of partial sleep deprivation designed to consolidate sleep rapidly and then gradually increase the scheduled time allotted for sleep when adequate sleep consolidation (efficiency) has been achieved.

Sleep restriction therapy consists of curtailing the amount of time spent in bed so that this time frame matches closely the amount of time the patient actually spends sleeping. Time in bed is then extended in 15-minute increments each week, pending adequate sleep consolidation. Scheduled morning wake time is held constant throughout treatment. Sleep opportunity is never restricted to less than 4 to 5 hours to avoid excessive daytime sleepiness. The rule governing the prescription of an earlier bedtime is that the patient must achieve an average sleep efficiency (sleep divided by time in bed) of at least 85% for a week before the time in bed is extended. The subject's sleep diary data should be analysed to calculate weekly sleep efficiency averages to guide titration of sleep opportunity.

27.5.3.3 Relaxation Training

Relaxation techniques are used to decrease the psychophysiological hyper-arousal that is thought to interfere with sleep initiation and maintenance in chronic insomnia. The following are three most common relaxation techniques.

- Breathing exercises: Diaphragmatic breathing is used to make breathing slower, deeper, and mechanically driven from the abdomen as opposed to the thorax. Such breathing resembles that which occurs naturally at sleep onset.
- Progressive muscle relaxation: This technique is used to diminish muscle tension, and involves systematically tensing and releasing the major muscle groups while focusing attention on the physical sensations of tension contrasted with relaxation.
- Use of imagery: Imagery is used to reduce sleep-related worry and to decrease intrusive pre-sleep cognitive arousal.

27.5.3.4 Cognitive Therapy

Cognitive therapy is based on the observation that patients with chronic insomnia have negative thoughts and beliefs about their condition and its consequences, and these further contribute to sleep disturbance. For example, a patient may worry about presumed catastrophic consequences of a poor night's sleep. Helping patients to challenge the reality of these beliefs and reframe their expectations about sleep may decrease the anxiety they experience as bedtime approaches.

27.5.3.5 Cognitive Behavioural Therapy: a Multicomponent Approach[31]

The multicomponent approach that combines stimulus control, sleep restriction, sleep hygiene education, cognitive therapy, and relaxation therapy has its benefits. It permits the clinician to target several different pathways thought to contribute to insomnia. Several studies have demonstrated robust effects of cognitive behaviour therapy in elderly patients with insomnia.[32,33]

27.5.3.6 Bright Light Therapy

Bright light therapy aims to modulate melatonin release and has been used with variable success in healthy and demented older people. The intensity of the artificial light ranged from 3000 to 10 000 lux in studies. The individual was exposed to the light for 1 to 2 hours either in the early morning (for those finding it difficult to fall asleep) or early evening (for those finding it difficult to maintain sleep).[34-36] This therapy may be particularly useful for institutionalised older persons or homebound individuals with little regular sunlight exposure.

27.6 Other Sleep Disorders

27.6.1 Sleep-disordered Breathing

Apnoea is defined as a cessation of airflow for at least 10 seconds, and is classified as obstructive or central based on the presence or absence of respiratory effort. Hypopnoea is defined as any one of three features: a substantial reduction in airflow (>50%); a moderate reduction in airflow (<50%) with desaturation (>3%); or a moderate reduction in airflow (<50%) with electroencephalographic evidence of arousal. The apnoea-hypopnoea index (AHI) is the number of apnoea and hypopnoea episodes per hour of sleep. An AHI of ≥5 is required for a diagnosis of SDB. Although the severity of SDB can be classified by the AHI from mild (AHI 5-14) to severe (AHI ≥30), clinical symptoms should be present for the treatment of mild cases. It should be noted that although laboratory evidence of SDB increases with age, clinical symptoms may be milder in older adults when compared with the middle-aged.[37] Treatment should aim to ameliorate symptoms and should not be based on laboratory severity alone.

Symptoms of SDB, particularly of obstructive SDB (also known as OSA syndrome) include daytime sleepiness, poorer quality of life, low mood, higher risk for accidents especially falls, and cardiovascular presentations including atrial fibrillation and cardiomyopathy.[11]

Central SDB, especially if severe, can be associated with increased mortality. It may reflect a disordered breathing response due to sleep-wake transitions, hypoxemia, use of respiratory suppressants, hypothyroidism, and autonomic or neuromuscular dysfunction. Work-up would thus depend on the suspected underlying cause(s). In general though, the adverse health outcomes of SDB are diminished in old age.

Apart from treatment of reversible underlying causes such as hypothyroidism, or withdrawal of culprit medications, continuous positive airway pressure remains the mainstay of treatment for SDB. Patient tolerance and compliance, however, pose major hurdles to treatment success.

27.6.2 Movement Disorders

27.6.2.1 RLS

In older adults, RLS is largely secondary rather than idiopathic or familial.[38] Physical examination is usually normal but associated medical conditions such as iron deficiency and kidney failure should be sought. Four essential diagnostic criteria are helpful to confirm the diagnosis (the URGE acronym[39]):

- U=Urge to move legs, usually but not always accompanied by unpleasant sensations in the legs
- R=Rest induces symptoms
- G=Getting active brings relief (e.g. walking, stretching)
- E=Evening and night makes symptoms worse

Supportive features include:

- family history from first-degree relative;
- positive therapeutic response to levodopa or dopamine receptor agonist; and
- periodic limb movements occur in sleep

The levodopa test may be useful to establish the diagnosis. It employs a single levodopa dose of 100 mg that should reduce the symptoms by approximately half.[40] The immobilisation test in the evening can also be used: the patient obtains temporary relief by moving around, but symptoms reappear after sitting or lying down for 10-20 minutes.

Secondary causes such as iron deficiency, if present, should be treated. Current evidence, however, suggests that iron supplementation in those with adequate iron stores is not recommended. Exacerbating drugs such as antidepressants should be stopped if they are no longer required. Caffeine, nicotine, and alcohol are best avoided.[11] The mainstay of pharmacotherapy is levodopa or dopaminergic agents. Daily doses of levodopa range from 100 mg to 300 mg, usually taken at night. A dopamine agonist (ropinirole, pramipexole, and rotigotine) may have the benefit of causing less blood pressure drop and nausea than levodopa. Long-term levodopa treatment may lose its efficacy and induce worsening of symptoms (augmentation).[38]

27.6.2.2 PLMS

PLMS has a higher prevalence than RLS, and is the most common movement disorder in older adults.[11] It is characterised by cyclical leg kicking in sleep at intervals of 20-40 seconds, and may or may not lead to arousals from sleep. It commonly occurs without concomitant RLS. Management and work-up are similar to that of RLS.

27.7 RBD in Older Adults

Parasomnia is an involuntary behaviour that occurs during sleep or during the sleep-wake transition. The American Sleep Disorders Association classification describes a total of 24 parasomnias including REM sleep parasomnias and others. RBD was first described in 1986 and is characterised by a history of excessive motor activities during nocturnal sleep that may lead to physical injuries and is associated with pathological absence of muscle atonia during REM sleep.[41] It usually occurs during the middle and last third of the night, and may be associated with injuries due to jumping or falling out of bed, harm to bed partner, and is recognised as a treatable cause of recurrent falls at night.[42] Patients often report vivid frightening dreams.[43] RBD is more common in older adults, and especially among older males. The mean (± standard deviation) age of presentation was 59 ± 15.4 years in a series of 70 patients with RBD.[44] The estimated prevalence of RBD ranges from 0.38% (among people aged ≥70 years in Hong Kong) to 0.5% (among non-institutionalised adults in the UK).[3,45] RBD is frequently underrecognised or misdiagnosed as another psychiatric illness.

27.7.1 RBD and Neurodegenerative Diseases

RBD is classified as idiopathic or secondary, the latter when it occurs in the presence of another neurodegenerative disease. In patients who present with idiopathic RBD, especially males older than 50 years, there is a substantial risk for neurodegenerative disease. The risks of developing neurodegenerative diseases: PD, dementia with Lewy bodies (DLB), or Alzheimer's disease have been reported to be 17.7% at 5 years, 40.6% at 10 years, and 52.4% at 12 years, respectively.[46]

RBD-associated PD is a common secondary form. The frequency of RBD in PD has been estimated to range from 15-59%.[47] Compared with PD patients without RBD, those with RBD are more likely to be older, male, and have orthostatic hypotension, cognitive impairment, or non-motor complications of

Parkinsonism such as hallucinations. DLB is another neurodegenerative disease commonly associated with RBD. Indeed, REM sleep parasomnias are regarded as an early sign of DLB, usually presenting a few years before onset of other typical features of DLB such as fluctuations and hallucinations.[48] Other conditions associated with RBD include multiple system atrophy, and a neoplastic or vascular lesion involving the REM-control areas or causing suppression of locomotor generators of the pons or medulla.

27.7.2 RBD and Medications

Acute REM sleep parasomnias may be triggered or revealed by medications.[47] These medications include serotonin-norepinephrine reuptake inhibitor antidepressant, selective serotonin reuptake inhibitor antidepressants, tricyclic antidepressant, monoamine oxidase inhibitor, beta-blocker, and opioid (e.g. tramadol).

27.7.2.1 Diagnosing RBD

The ICSD requires the following criteria for the clinical diagnosis of RBD.

- Presence of REM sleep without atonia on PSG
- The presence of at least one of the following conditions: (1) sleep-related behaviours, by history, that have been injurious, potentially injurious, or disruptive (e.g. dream enactment behaviour); (2) abnormal REM sleep behaviour documented during PSG monitoring
- Absence of epileptiform activity on electroencephalogram during REM sleep (unless RBD can be clearly distinguished from any concurrent REM sleep-related seizure disorder)
- Sleep disorder not better explained by another sleep disorder, a medical or neurological disorder, a mental disorder, medication use, or substance use disorder

PSG, including electromyography of the chin, arms, and legs, is mandatory for the diagnosis of RBD. PSG is helpful in excluding severe OSA that can cause symptoms that mimic RBD, known as "pseudo-RBD", and that resolve with treatment for OSA.[49] PSG is also helpful in excluding nocturnal seizures and parasomnias arising from non-REM sleep (e.g. sleepwalking, also known as somnambulism), as the distinction between RBD and these entities may not be made reliably on clinical grounds. PSG with video recording can detect some tonic or phasic abnormalities of muscle tone during REM sleep. Neuroimaging studies are not routinely indicated in idiopathic cases but are useful if secondary RBD is suggested by history and neurological examination.

27.7.2.2 Treatment of RBD

Non-pharmacologic treatment of RBD includes preventive measures to improve safety of the sleep environment. These include the use of a hospital-style bed with padded and raised side rails, removal of sharp, heavy, or dangerous items from the sleeping area, or the bed partner choosing to sleep in another bed or bedroom.

Clonazepam is the first-line pharmacologic management for RBD. Clonazepam suppresses abnormal behaviours without restoring REM atonia, and is thought to act by inhibiting the locomotor generator during REM sleep. There are no double-blind, randomised controlled trials of therapy for RBD. Based on large case series using clonazepam in patients with RBD, the response rates of 87-90% were similar in both idiopathic and secondary forms.[47] In 55% of patients, behaviours completely resolved on clonazepam.[50] Doses ranged from 0.25 to 4.0 mg per day at bedtime, with dosages between 0.5 mg and 2.0 mg being the most common. Side-effects such as excessive daytime sleepiness, confusion, and cognitive impairment may be seen in up to 58% of patients, resulting in medication discontinuation in up to one third.[51] In conditions where clonazepam may be relatively contra-indicated, such as the presence of sleep apnoea, dementia, or a high risk of falling, melatonin can be used as the second-line therapy. In contrast to clonazepam, melatonin restores REM atonia. It has been proposed that melatonin may normalise the circadian timing of REM sleep and thus decrease RBD behaviours. The response rates range from 71-83%.[47] Side-effects are mild and include morning sleepiness and headaches. Doses range from 3 mg to 12 mg taken 30 minutes prior to bedtime. Other agents have been used with varying success and include cholinesterase inhibitors (donepezil, rivastigmine), non-benzodiazepine hypnotic (zopiclone), and dopaminergics (levodopa, pramipexole).

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CHAPTER 28

Impaired Vision and Hearing

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OBJECTIVE

To learn the epidemiology, assessment, diagnosis, and management of impaired vision and hearing in old age.

KEY POINTS

Visual Impairment

- Visual acuity should be checked periodically.
- Urgent referral to an ophthalmologist is advised for painful red eye or sudden loss of vision.
- Annual ophthalmological examination is recommended for all diabetics.
- Always obtain the names of eye drops when taking a history.
- Register the patient as partially sighted if there is sufficient impairment.
- Seek low visual aids advice.

Hearing Impairment

- Deafness becomes more common with increasing age.
- Hearing loss leads to impaired functioning and health.
- Always check for cerumen impaction in hearing impaired older adults. It represents a significant factor in 30% of elderly people.
- Avoid ototoxic medication in hearing impaired individuals.
- Counsel about use of a hearing aid and its maintenance.
- Individuals with communication difficulty due to permanent hearing loss may benefit from an aural rehabilitation programme.

28.1 Introduction

Visual and hearing problems are the two most common sensory impairments in older adults. Both impairments become more common with increasing age, and are included as one of the common geriatric syndromes of "I"s. Visual and hearing impairment will also predispose to other syndromes of "I"s, that include inanition (resulting from difficulty in acquiring and preparing food), instability (fall), isolation (both physical and social), incontinence (functional incontinence), immobility, and iatrogenesis (e.g. being misdiagnosed as dementia). These sensory disabilities are predictive of subsequent reduction in mobility and activities of daily living, and are associated with an increasing need for social services and medical care.[1] In addition, declining vision and hearing are associated with increased mortality, imbalance, hip fracture, and depression; all of which pose important challenges for caregivers and families.[2,3] It is important that primary healthcare providers identify older adults with these sensory deficits, elucidate the cause, and initiate appropriate therapies so as to reduce disability, minimise handicap, and improve quality of life.

28.2 Visual Impairment

28.2.1 Epidemiology

Functional blindness (low vision or blindness) is defined as vision worse than 6/60 in one or both eyes tested with a Snellen chart (see Chapter 2.6 for visual acuity assessment). Approximately 10% of the older population have low vision.[4] The prevalence appears even greater among institutional elders. A survey of 100 older adults living in residential homes in London showed that 51% had cataracts and 34% had evident refractive errors.[5]

Epidemiological data for vision impairment in Hong Kong are scarce. A local study revealed that 29.5% of the community-dwelling older adults reported

poor vision or blindness.[6] On the other hand, 41.7% of the participants of elderly health centres of the Department of Health were noted to have cataract.[7] According to the report by Hospital Authority in March 2001, adults aged ≥65 years constituted 57.7% of those registered with low vision or blindness in Hong Kong.[8]

The most common eye diseases that cause vision impairment in older age are macular degeneration (36%), cataracts (25%), glaucoma (8%), and diabetic retinopathy (2%).[9]

28.2.2 Structural Changes in Ageing Eyes

Eyelids will undergo loss of fat, decreasing muscle tone, and diminished elasticity resulting in ptosis, ectropion, and entropion. Tear secretion will also decrease, thus resulting in dry eye states that in turn predispose to inflammation and infection. Sunken eyeballs due to loss of orbital fat are common in old age and hence not a reliable sign of dehydration. Arcus senilis is also common but not significant. The corneal endothelial cells may degenerate — a condition called cornea guttata that in turn may occasionally predispose epithelial breakdown causing severe discomfort and impaired vision.

Endothelial proliferation and sclerosis of the trabecular meshwork together with scleral rigidity may give rise to elevated intraocular pressure (IOP) in ageing eyes, thus predisposing to glaucoma. Sclerotic changes in the iris vasculature together with increased hyalinisation in the connecting tissue result in a thickened and rigid iris: ageing pupils tend to be small and slow to react to light and accommodation. This hinders adaptation to dark. The lens becomes less elastic and transparent, thus predisposing to presbyopia and cataracts. Vitreous humour commonly detaches from its normal attachment to the retina in the ageing eyes, thus predisposing to retinal tears, detachment, and vitreous haemorrhage.

28.2.3 Functional Changes in Ageing Eyes

Static visual acuity as tested by a visual acuity chart declines with age. Dynamic visual acuity also decreases as target velocity increases. The size of the visual field as well as the contrast sensitivity will decrease with age. Colour vision also declines with age and is relevant to certain tasks such as seeing a traffic light while driving or crossing the street. Older adults are more sensitive to dazzling light and take longer time to recover when exposed, thus increasing the risk of falls and accidents. Similarly, dark adaptation ability also decreases in ageing eyes, predisposing to difficulty in walking when moving from a lighter to a darker area, for example, when entering a movie theatre or dimly lit restaurant.

28.3 Presbyopia

Presbyopia begins around the age of 45 years and is slowly progressive. By the age of 70 years, accommodation is universally lost.[10] Patients will complain of difficulty in focusing on near objects and reading material. They may experience ocular discomfort or aching pain, tearing, and fatigue when doing near work. Optimal refractive correction with additional lens power (usually bifocal glasses) will bring near objects into focus and reduce symptoms.

28.4 Cataract

28.4.1 Introduction

Cataract is a principal cause of vision loss among elderly people. Opacification of the lens usually begins in the fifth to seventh decades of life. The prevalence increases with advancing years and approaches 100% by the age of 90 years.[11]

The opacity may occur in the cortex, the nucleus, or the posterior subcapsular region of the lens. Cortical cataracts involve peripheral lens fibres and are well tolerated until the opacification extends into the centre of the lens. Nuclear cataracts, the most prevalent, are characterised by a diffuse yellowing of the lens. They worsen over time, and surgery is usually needed. Posterior subcapsular cataracts are poorly tolerated because they reduce contrast sensitivity and produce glare sensitivity. The risk factors for developing cataracts are listed in Table 28.1.[11-13]

28.4.2 Assessment and Diagnosis

The initial symptoms of cataracts are decreased glare tolerance and contrast sensitivity followed by blurred vision. Distance visual acuity is most significantly impaired by nuclear cataracts. Vision deteriorates to blindness as the cataract advances. The sclerotic lens results in myopia that temporarily corrects presbyopia, causing a misconception that eyesight is improving. This condition is called 'second sight'. Fundoscopy, contrast sensitivity, and visual acuity testing are helpful in diagnosis.

28.4.3 Management

Treatment is largely limited to cataract surgery. Other

Table 28.1 Risk factors for development of cataracts[11-13]

• Diabetes mellitus
• Ultraviolet B light (cortical cataract)
• Corticosteroids (posterior subcapsular cataract)
• Ocular trauma or intraocular surgery
• Prolonged glaucoma therapy with topical medications
• Smoking (cortical cataract)
• Alcohol
• Normal ageing

Table 28.2 Indications for cataract extraction[12,14]

- Corrected visual acuity of <6/18 with symptoms of poor vision in the absence of other ocular disease
- Visual acuity is 6/12 or better with symptoms of disabling glare, monocular diplopia or occupational impairment
- Cataract with complications like glaucoma
- Cataract with retinal disease requiring monitoring

Table 28.3 Types of primary glaucoma

	Acute: narrow angle	Chronic: open angle
Symptom	Sudden eye pain, seeing halos, blurred vision, nausea, and vomiting	Asymptomatic in early stage; insidious loss of vision leading to tunnel vision
Sign	Red eye, tense eye, cloudy cornea, fixed irregular pupil	Raised IOP, scotoma, cupped disc
Pathology	Sudden impairment of anterior chamber drainage	Gradual increase in IOP

Abbreviation: IOP = intraocular pressure

Table 28.4 Risk factors for developing glaucoma[11,16]

- Diabetes mellitus
- Hypertension
- Family history of glaucoma
- Migraine headache
- Severe myopia
- Increasing age
- Medications (anticholinergic, antidepressant, antipsychotic)

choices for early cataract include improved lighting, pupil dilatation, and glare reduction with tinted glass. Indications for surgery are focused on subjective symptoms as well as objective vision assessments (Table 28.2).[12,14] In general, cataract surgery is indicated when the patient's activities of daily living are affected or if the cataract interferes with the diagnosis or treatment of other ocular diseases.[15,16] Small-incision phacoemulsification is a popular technique that involves fragmenting the lens nucleus with ultrasonic vibrations and subsequent aspiration of the lens material. This facilitates implantation of a foldable intraocular lens into the eye. No suturing is required because of the small incision (usually a few millimetres), thus facilitating prompt recovery of vision and rehabilitation.

28.5 Glaucoma

28.5.1 Introduction

Glaucoma is the second most common cause of irreversible vision impairment in older adults. It is a group of eye disorders characterised by increased IOP, progressive cupping and atrophy of the optic nerve head with initial loss of peripheral vision followed by central vision. The risk of progressing to glaucoma is believed to vary directly with the level of IOP and duration of raised IOP. Systemic reviews showed that a raised IOP as measured by Goldmann applanation tonometry (IOP ≥20.5-22 mm Hg) has a pooled sensitivity of 46% and specificity of 95% in detecting open angle glaucoma, while that measured by non-contact tonometry (IOP ≥21 mm Hg) has a pooled sensitivity of 92% and specificity of 92%.[17] Primary glaucoma is classified as narrow-angle (10%) or open-angle (90%) [Table 28.3]. Secondary glaucoma is due to processes that anatomically or functionally block the outflow channels (e.g. trauma, central retinal vein occlusion, uveitis, ocular tumours, cataract extraction). The risk factors for developing glaucoma are shown in Table 28.4.[11,16]

28.5.2 Assessment and Diagnosis

It is important to obtain a patient's medical history — paying particular attention to family history, risk factors, and possible secondary causes. Physical examination includes local eye examination, tonometry, fundoscopy, and visual field testing.

28.5.3 Management

Treatment for primary glaucoma is medical and surgical. Medical therapy is usually the initial mainstay of treatment. There are currently five types of medication commonly employed, and familiarity with their side-effects will help subsequent management of patients (Table 28.5). Systemic carbonic anhydrase inhibitors are generally reserved for cases refractory to topical treatment. Surgical options include laser or surgical trabeculoplasty and laser iridotomy and iridectomy. In chronic open-angle glaucoma, surgical options are considered when medical treatment has failed. In acute narrow-angle glaucoma, surgical treatment is usually the definitive treatment following urgent stabilisation with medical therapy.

28.6 Age-related Macular Degeneration

28.6.1 Introduction

Macular degeneration affects about 30% of the geriatric population.[11,12,14] It is also the most common

Table 28.5 Drugs for glaucoma

Type	Side-effect
Beta blockers (topical)	Hypotension, bradycardia, falls, syncope, depression, nightmares, bronchospasm
Adrenergics (topical)	Palpitation, headache, insomnia, increase in blood pressure
Cholinergics (topical)	Sweating, visual disturbance, gastrointestinal upset, bronchospasm
Prostaglandin analogues (topical)	Muscle pain, arthralgia, chest pain, rash, upper respiratory tract infection
Carbonic anhydrase inhibitors (topical or systemic)	Gastrointestinal upset, delirium, depression, malaise, renal calculi

Table 28.6 Risk factors for developing ARMD[13,14,18,19]

- Increasing age
- Excessive sunlight exposure
- Smoking
- Hypertension
- High serum cholesterol level
- Family history of ARMD

Abbreviation: ARMD = age-related macular degeneration

cause of blindness in older adults.[11,14,16] It is defined as atrophy or degeneration of cells in the central macular region of the retinal pigment epithelium resulting in loss of central vision. The exact aetiology is unknown although there are multiple risk factors (Table 28.6).[13,14,18,19] On ophthalmologic examination, early age-related macular degeneration (ARMD) appears as whitish-yellow pinhead patches or drusen (hyaline deposits). ARMD has two forms: non-exudative (dry) and exudative (wet) forms. The non-exudative form is characterised by varying degrees of atrophy of the outer retina, retinal pigment epithelium, and Bruch's membrane. Discrete lesions will coalesce as time goes on. Fundoscopic examination may reveal drusen and areas of depigmentation alternating with zones of hyperpigmentation. It is the more common form but with generally less severe vision loss. Of note, 10% of patients with the dry form will develop the wet form of degeneration. The exudative (wet) form is due to subretinal neovascularisation. Serous fluid from the choroids filters through defects in Bruch's membrane, causing partial detachment of the retinal epithelium and irreversible loss of central vision. Fundoscopic examination shows haemorrhage and exudates in the macula secondary to bleeding from subretinal neovascular nets. Sometimes, detachment of the sensory retina in the macula will also be revealed.

28.6.2 Assessment and Diagnosis

Symptoms include diminished central vision with difficulty in reading and recognising faces. Straight lines may look distorted, for instance, telephone poles may appear crooked. Colours may seem faded, and colours or objects may look different through each eye. There may be problems with depth perception. Objects may disappear from the field of vision, thus causing difficulty in mobility. Evaluation includes visual acuity assessment, vision testing with an Amsler grid, fundoscopic examination of the macula, and visual field testing.[20] All patients with ARMD should be advised to screen their central vision on a daily basis using an Amsler grid to detect progressive disease and enable early ophthalmological intervention.

28.6.3 Management

There is no specific treatment for the non-exudative form although zinc supplementation and antioxidant vitamins may slow progression. Treatment of exudative ARMD includes laser photocoagulation, verteporfin, and intravitreal injections of vascular endothelial growth factor inhibitors.[20] Regular evaluation is important because recurrent neovascularisation may occur within 2 years of successful treatment in half of the patients.[12] The use of anti-angiogenic therapy for exudative ARMD under some conditions (e.g. no permanent damage to the central fovea) is suggested. Anti-angiogenic therapy is the use of a recombinant, humanised, monoclonal antibody that neutralises all active forms of vascular endothelial growth factor A.[21]

28.7 Other Important Eye Disorders

28.7.1 Diabetic Retinopathy

Diabetic retinopathy is a chronic disorder of the retinal microvasculature and accounts for 10% of blindness in older adults. It is categorised into three phases: background, pre-proliferative, and proliferative. The prevalence of retinopathy increases with the duration of diabetes. Treatment of proliferative retinopathy with laser therapy helps preserve visual function. Annual retinal examination by an ophthalmologist is essential because treatable disease may progress to irreversible damage in an asymptomatic patient.

28.7.2 Amaurosis Fugax

Amaurosis fugax refers to transient monocular blindness. It is a transient ischaemic attack of the retina by emboli. The presence of this symptom should prompt investigation of an embolic source such as atrial fibrillation, carotid artery stenosis, infective endocarditis, or haematologic disorder.

28.7.3 Herpes Zoster Ophthalmicus

Herpes zoster is a common ocular infection. Pain

may be severe and precedes vesicle eruption by 1 to 3 days. Involvement of the ophthalmic division of the trigeminal nerve can result in conjunctivitis, keratitis, uveitis, and secondary glaucoma. Ocular herpes zoster requires urgent ophthalmological referral. Treatment includes antiviral, steroid, and / or antiglaucoma agents.

28.7.4 Giant Cell Arteritis

Giant cell or temporal arteritis is an important ocular emergency in older adults. It is a large-vessel vasculitis that often affects the vessels of the aortic arch and their branches. The most dreaded complication is irreversible loss of vision that occurs in 15% of patients, most commonly due to ischaemic optic neuritis with involvement of the ciliary branches of the internal carotid artery. Giant cell arteritis is suspected when there is a history of occipital or temporal headaches, scalp tenderness, and high erythrocyte sedimentation rate. Diagnosis is confirmed by temporal artery biopsy. Treatment requires a course of high-dose corticosteroids. Such therapy may protect the second eye from future vision loss.

Table 28.7 Low-vision aids

• Magnifying glass or high-plus spectacles
• Large-print books, newspapers, and magazines
• Black and white TV sets
• Talking books, watch or clock
• Large-button telephones
• Simple aids like liquid-level indicator and electronic gas lighter
• Tactile markers for cookers
• Written communication system (e.g. Braille)
• Long cane or guide dogs

Table 28.8 Environmental modification to help low vision[22,23]

• Soft light
• Flat paint
• Tinted glasses
• Floor lamps
• Night light
• Using colour contrast between placemats and dishes
• High technology options, such as video magnification with closed circuit television

28.8 Vision Rehabilitation

Reduced vision in older adults is a significant factor in rehabilitation as it minimises mobility, increases risk of fall and injury, and degrades the quality of life. Vision-impaired older adults, however, can be maintained in functional living with the appropriate use of low-vision aids (Table 28.7), skills training, environmental modification (Table 28.8),[22,23] and psychosocial support. More resources on vision impairment can be found in Table 28.9.

28.9 General Patient and Family Education about Impaired Vision

Older adults should be advised of the following.[11,24]

- Take a well-balanced diet and avoid smoking
- Ensure adequate rest and sleep
- Be careful when using sprays, preferably at a distance
- Watch television at a comfortable distance with good lighting
- Make sure lighting is sufficient when reading and try to take a short break every half an hour to relax the eyes by looking at some distant objects
- Do not use hands or a public towel to rub the eyes
- Do not use other people's eye medications or glasses

Table 28.9 Local resources for the visually impaired

Organisation	Correspondence
Hong Kong Blind Union	Unit 13-20, G/F, Tsui Ying House, Tsui Ping Estate, Kwun Tong Tel: 2339 0606 Fax: 2338 7850 Email: info@hkbu.org.hk Webpage: www.hkbu.org.hk
Asian Foundation for the Prevention of Blindness	Unit 713, 7/F, Tower 2, Cheung Sha Wan Plaza, 833 Cheung Sha Wan Road Tel: 2789 3383 Fax: 3005 7110 Email: info@afpb.org.hk Webpage: www.afpb.org.hk
Rehabilitation Alliance Hong Kong	G/F, Unit 12-13, Wang Hau House, Wang Tau Hom Estate Tel: 2337 0826 Fax: 2337 1549 Email: info@rahk.org.hk Webpage: www.rahk.org.hk
Hong Kong Society for the Blind	East Wing, 248 Nam Cheong Street, Sham Shui Po Tel: 2778 8332 Fax: 2788 0040 Email: enquiry@hksb.org.hk Webpage: www.hksb.org.hk

- Keep spectacles clean and always put them in the same safe place when not in use
- Seek support and advice from associations for the blind

28.10 Hearing Impairment

28.10.1 Epidemiology

Hearing loss is the most common sensory impairment in elderly people. It affects about 30% of community-dwelling elders and its prevalence increases with age, reaching 80% in the eighth decade and in nursing home residents.[25-27] A study carried out in Canton, China found that in those aged 60-69 years, 23.3% of male and 37.8% of female participants were hearing impaired and the proportion rose to 53.6% for the male and 59.6% for the female counterparts aged ≥70 years.[28] A local study revealed that 76.6% of the community-dwelling older adults reported hearing impairment or deafness.[6] Another local study found that 19.1% of the patients attending elderly health centres of the Department of Health had hearing loss.[7] According to the report by Hospital Authority in March 2001, adults aged ≥65 years constituted 25.5% of those registered as hearing impaired or deaf in Hong Kong.[8] Consequently, deafness is ranked as one of the top five disabilities associated with ageing in Hong Kong.[29]

28.10.2 Ageing Changes in the Ears

Cerumen glands, which are modified apocrine sweat glands, decrease in number and activity and cause an overall reduction in moisture. This may explain, in part, why ear wax impaction tends to be more common in the ear canals of older patients. Total ear canal occlusion by wax can lead to a conductive hearing loss of 15-30 db.[30] Atrophy of the epithelial sebaceous glands results in a decrease in the oiliness and wetness of the skin, thus predisposing to pruritus in the external auditory canal.

Degenerative changes are noted in the ossicular chain in the middle ear (Table 28.10). The significance of these changes as they relate to sound transmission, however, seems to be negligible as revealed by audiometric studies.

Ageing changes to the inner ear and central auditory system (Table 28.11) predispose older adults to sensorineural deafness, namely, presbycusis. Presbycusis refers to a slowly progressive bilaterally symmetrical hearing loss that primarily affects high frequencies and is associated with the ageing process. The clinical manifestations of presbycusis have been categorised into four different types. Sensory presbycusis is the degeneration of hair cells and supporting cells at the base of the cochlea and cochlear fibre degeneration. These changes produce an abruptly sloping, slowly progressive high-frequency sensorineural hearing loss. Neural presbycusis is a loss of cochlear neurons resulting in problems of transmission information coding. It causes high-frequency hearing loss, but speech understanding is poorer than one would expect from audiometry. Strial or metabolic presbycusis is characterised by atrophic changes in the stria vascularis. Audiometrically it is evidenced by a relatively flat pure tone response with excellent speech discrimination. Finally, cochlear conductive or mechanical presbycusis results from alterations in cochlear mechanics produced by mass or stiffness changes or spiral ligament atrophy. It has a characteristic audiometric pattern of a descending straight line. Speech discrimination and the degree of pure tone hearing loss are directly related. Table 28.12 lists the multiple risk factors for developing presbycusis.

28.10.3 Functional Implications of Hearing Loss in Elderly People

Older adults with hearing loss report greater difficulties with functional activities than those without hearing impairment (Table 28.13).[2] They also rate their self-health poorer than those without hearing loss including those with hypertension, heart disease, stroke, and depression.[2] Hearing loss affects communication, thus reducing socialisation.

Table 28.10 Ageing changes to the middle ear ossicular chain

• Fibrillation, fraying, vacuolation, and calcification in the articular cartilages
• Hyalinisation and calcification of the joint capsule
• Narrowing, fusion, or obliteration of the joint space

Table 28.11 Ageing changes to the inner ear and central auditory system

Type	Side-effect
Inner ear	• Angiosclerosis • Calcification of membranes • Atrophy and cell loss • Bioelectric and biomechanical imbalances
Central auditory system	• Degeneration and loss of ganglions cells and their fibres in the cranial nerve VIII • Cranial VIIIth nerve canal closure with destruction of nerve fibres • Atrophy and cell loss in the auditory regions in the brainstem and the cortex

Table 28.12 Risk factors for developing presbycusis

- Genetic programmed senescence
- Lifelong acoustic trauma
- Chronic alcohol abuse
- Diabetes mellitus
- Hypertension
- Hyperlipidaemia
- Vascular disease

Table 28.13 Comparison of older adults with and without hearing loss[2]

	Hearing loss	No hearing loss
Difficulty walking	30.7%	21.3%
Difficulty getting outside	17.3%	12.0%
Getting into or out of bed or a chair	15.1%	9.8%
Managing medication	7.7%	4.8%

Sometimes there is misinterpretation of hearing loss as a cognitive, affective, or personality disorder, especially in an acute hospital care setting. Increasing evidence has linked age-related hearing loss to more rapid progression of cognitive decline and incidental dementia.[31]

28.10.4 Assessment and Diagnosis

The history should establish previous noise exposure, family history of hearing loss, use of ototoxic medications, and cardiovascular problems. Chronic allergy or respiratory conditions may be associated with fluid in the middle ear. Ear infections earlier in life may have resulted in a perforation or cholesteatoma. It is important to ask the patient about the timing of hearing loss (e.g. insidious, progressive, or sudden) and the ears affected (unilateral or bilateral).

An examination of the ears should be performed. The ear canal should be examined with an otoscope to identify a mass or ear wax. The eardrum should be visualised and any perforations or other abnormalities (cholesteatoma or middle ear fluid) noted.

There are three common screening methods that can be used by a general practitioner (see Chapter 2.7 for details of hearing tests). One method is to use simple tests such as a whispered voice, watch tick, or finger rub test. They are reported to have a sensitivity of 90-100% and a specificity of 80-85% but are criticised for a lack of data on interobserver variability. The second method is utilising a standardised screening questionnaire like the HHIE-S (Screening Version of Hearing Handicap Inventory for the Elderly) [see Chapter 2.7]. Finally a hand-held audiometer can be used for screening purposes. It has a reported sensitivity of 94% and specificity of 72%.[25,32,33]

Last but not the least, performing the Rinne and Weber tests using a 512-Hz tuning fork will help differentiate a conductive hearing loss from a sensorineural hearing loss. Further evaluation of the hearing loss including pure tone audiometry, tympanometry, and brainstem auditory evoked potential require referral to an otorhinolaryngologist.

28.11 Other Common Hearing Disorders

28.11.1 Ototoxic Medications

Medications can cause hearing impairment. Ototoxicity is often potentiated by pre-existing renal disease. Common implicated drugs include high-dose loop diuretics, high-dose salicylates, aminoglycosides, and some cytotoxic agents (e.g. cisplatin). They all damage the cochlea directly. Usually, except for salicylates, removal of the offending drug does not reverse the sensorineural hearing loss.

28.11.2 Otosclerosis

Otosclerosis is a common cause of conductive hearing loss in older adults. It has an autosomal dominant inheritance. The pathological mechanism is fixation of the ossicular chain causing immobility of the middle ear ossicles.

28.11.3 Chronic Suppurative Otitis Media

Chronic suppurative otitis media is a relatively common condition in elderly people. It is divided into safe and unsafe types. The safe type is identified by its association with a central perforation of the eardrum on otoscopy. The disease is characterised by profuse mucopurulent discharge, conductive hearing loss, and tinnitus. Treatment requires repeated aural toilet and topical antibiotic. Myringoplasty with tympanoplasty need to be performed. The unsafe type is associated with marginal perforation instead, roof erosion, and cholesteatoma formation. There is usually minimal ear discharge but patients complain of hearing difficulty. Diagnosis is made by the typical otoscopic appearance of bony erosion or deep retraction pockets. The disease can progress to affect temporal bone and the intracranial cavity. Limited disease is managed with tympanoplasty and extensive disease requires modified or radical mastoidectomy.

28.11.4 Meniere's Disease

Meniere's disease is characterised by episodic vertigo, aural fullness, tinnitus, and fluctuating hearing loss. The pathophysiology is thought to be secondary to endolymphatic hydrops exposing the hair cells in the inner ear to progressive damage. The exact aetiology is unknown. The onset of the disease usually starts

in middle age and therefore should rarely be first diagnosed in elderly people although it contributes to hearing loss in older adults.[29,34] Elders usually tolerate surgical treatment poorly. Management is conservative with antivertiginous medication, prophylactic salt restriction, and aural rehabilitation.[35]

28.12 Aural Rehabilitation

Older adults who have communication difficulties caused by permanent hearing loss should receive aural rehabilitation. As each patient has specific characteristics with regard to the degree of pure tone hearing loss, the ability to understand speech, noise tolerance, manual dexterity for manipulation of small components, cognition, and overall lifestyle, the rehabilitation programme must be tailor-made to suit each individual's specific needs. An aural rehabilitation team includes an otologist, audiologist, occupational therapist, speech therapist, social worker, patient, and caretaker. A comprehensive hearing rehabilitation programme includes many components (Table 28.14).[29,36,37] More resources on hearing impairment are shown in Table 28.15.

Table 28.14 Components of aural rehabilitation[29,36,37]

- Counselling and education
- Hearing aid prescription, maintenance, and follow-up care
- Assisted communication techniques including speech reading, auditory training, and hearing aid orientation
- Assisted listening devices (e.g. FM, loop and infrared systems)
- Special sensory devices (e.g. flashing telephone or doorbells, vibrating alarm clock, captioned TV)

28.13 General Patient and Family Education about Hearing Impairment

Patients should be advised to dry their ears after washing hair and swimming, avoid prolonged use of earphones and turning the volume too high, use ear protection when working in a very noisy environment, avoid blowing the nose too hard, and seek advice from a doctor if the following conditions exist[11,26,29,37,38]:

- foreign body getting into the ear
- ear pain or discharge or tinnitus
- difficulty in hearing other person's speech over the phone
- frequently asking others to repeat themselves
- difficulty in hearing with background noise
- difficulty in understanding speech when meeting new people
- always turning up the volume of radio and television in order to hear well

The family should also be given advice about how to communicate with the deaf patient (Table 28.16).

Table 28.15 Local resources for the hearing impaired

Organisation	Correspondence
Hong Kong Association of the Deaf	Choi Hung Centre Nos. 109 & 111-118, G/F, Chi Mei House, Choi Hung Estate Tel: 2327 2497 Fax: 2327 7445 Email: info@hongkongdeaf.org.hk Webpage: www.hongkongdeaf.org.hk
Hong Kong Society for the Deaf	Head Office, Room 903, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai Tel: 2527 8969 Fax: 2529 3316 Email: headoffice@deaf.org.hk Webpage: www.deaf.org.hk
Rehabilitation Alliance Hong Kong	G/F, Unit 12-13, Wang Hau House, Wang Tau Hom Estate Tel: 2337 0826 Fax: 2337 1549 Email: info@rahk.org.hk Webpage: www.rahk.org.hk

Table 28.16 How to communicate with the deaf

- Get listener's attention before speaking
- Alert listener to changes in topic before proceeding
- Do not shout
- Speak clearly and slowly but not in an exaggerated way
- Do not sit with your back to the light
- Face listener directly in clear light to afford visual cues
- Do not cover mouth while talking
- Ask the patient if he or she can hear you, adjust tone of voice if necessary
- Try to reduce background noise, e.g. turn down radio
- Rephrase message instead of repeating if listener does not understand, avoiding high-frequency sounds
- Check that if patient has an aid which is properly worn and functioning
- Do not turn and walk away while talking

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CHAPTER 29

Impaired Immunity and Infections

Tak-kwan KONG

OBJECTIVES

To learn the causes of secondary immunodeficiency in old age, the special presentations and impact of infections in old age, the relationship between infections and the other geriatric giants, and the preventive strategies as well as approach towards treating fever of unknown origin in elderly patients.

KEY POINTS

- The geriatric giants of iatrogenesis, isolation, depression, and malnutrition all add on and interact to increase the risk of infection in old age.
- The risk of infection is much higher among residents of care homes and long-term care facilities.
- The fever response to infection in old age may be absent, sluggish, or of lower intensity, and the pattern may be one of a rise from low baseline temperature.
- Instead of focal organ-specific symptoms, an elderly person with infections may present with geriatric syndromes of falls, confusion, incontinence, functional decline, or a change in appetite (e.g. poor feeding and refusal to feed).
- Since the classical presentations of infections in a frail elderly person may be replaced by functional decline, careful observation and assessment of change in the functional status of an elderly individual would provide additional sensitive evidence (though non-specific) of an emerging infectious illness.
- Secondary causes of immunodeficiency are common in old age, and can be reversed or minimized by treating comorbid diseases, correcting malnutrition, avoiding iatrogenesis, and appropriately using drugs that might suppress body defence.
- Tuberculosis in common in old age, especially in the male sex and in residents of care homes. It is an important and common treatable cause of fever of unknown origin in old age.

29.1 Infections in Old Age as a Geriatric Giant

It has been proposed that the decline in immunity associated with ageing (immunosenescence) explains the increased prevalence of autoimmune disorders, cancers, and infections in old age.[1,2] However, there are a number of potentially reversible secondary causes of immunodeficiency in old age including malnutrition, comorbid diseases, polypharmacy and iatrogenesis, HIV infection, psychosocial isolation, depression, and stress.[2,3] Early recognition and management of these secondary causes of immunodeficiency are important in reducing the problem of infections in old age.

Although the news about infections in elders have hit the headlines every now and then, the issue is relatively neglected. Infections in elders, typically, tend to affect the under-privileged and malnourished, remain hidden and underdiagnosed, carry a high morbidity burden with hidden mortality, and yet, accorded low priority. Like other geriatric giants, infections in old age are common and complex, with gigantic impact on elderly persons (see Chapter 29.3), their families, and the society; yet, these are often ignored.

Infections account for a significant proportion of hospitalisations for elderly people. Epidemiological data show rising age-specific incidences of both established infectious diseases like tuberculosis[4] (Figure 29.1) and herpes zoster[5] (Figure 29.2), and emergent infectious disease like severe acute respiratory syndrome (SARS)[6] [Figure 29.3]. Infections showing increased frequency with older age include urinary tract infections, pneumonia, tuberculosis, skin and soft tissue infections (including infected pressure ulcers, herpes zoster), gastroenteritis (including *Clostridium difficile*), osteomyelitis, bacteraemia, infective endocarditis, and intra-abdominal infections (cholecystitis, diverticulitis, and intra-abdominal abscesses).[7]

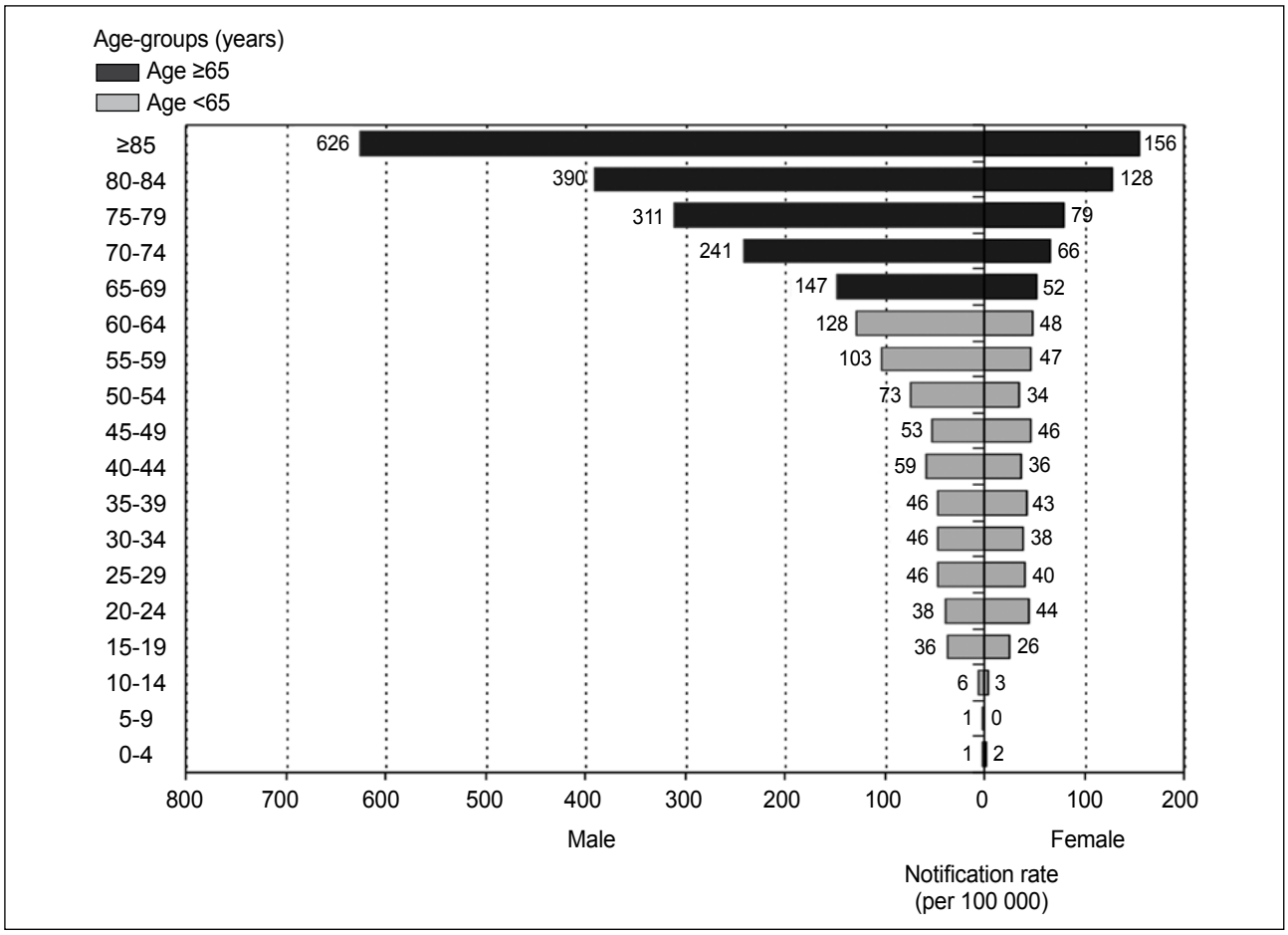

Figure 29.1 Age- and sex-specific notification rates of tuberculosis in Hong Kong in 2014[4]

The increased prevalence of infections in old age is due to the increasing mean age of the general population as well as the increasing risk of infections in old age (Figure 29.4).[6,8-27] While ageing and comorbid diseases render elders vulnerable to infections,[8,9] the geriatric giants of iatrogenesis,[10-14] isolation, depression,[15,16] and malnutrition[17-21] all add on and interact to increase the risk of infections through mechanisms outlined in Figure 29.4. While studies have thrown light on the association of malnutrition with depressed immunity leading to the concept of malnutrition-associated immune deficiency syndrome,[17] specific nutritional deficiencies have also been reported to make an elderly person vulnerable to common infections. For example, vitamin D deficiency predisposes to tuberculosis[28] and folate deficiency to aspiration pneumonia.[29]

Increased use of acute hospital care and long-term care facilities among elders also exposes them to medical and nursing procedures as well as the microbiological flora of the hospital / nursing home with the consequent risk of nosocomial and nursing home-acquired infections.[21,22] Thus, the risk of pneumonia in those aged >75 years was estimated as twice that of those aged 60-74 years[23] and, in residents of long-term care facilities, was reported as 4 to 10 times that of community-dwelling older adults.[24,25] In Hong Kong, the prevalence of active tuberculosis in residential care homes was estimated at 1.2-2.6% in a survey of 16 care homes reported in 1996 (91% and 61% of the study population aged >70 years and >80 years, respectively) in the North East New Territories region,[26] and 0.67% (1.1% for men, 0.53% for women; mean age of study population, 82 years) in a later study in 15 care homes in two districts (Hong Kong West and Tai Po) reported in 2006.[27] These prevalence rates are 2 to 3 times of those in community-dwelling elderly population. Similarly, the SARS rate among residents of care homes (0.13%) was higher than the general population in 2003 in Hong Kong, being 5 times that in the general population and 3.2 times in the elderly population aged ≥65 years.[6]

29.2 Clinical Presentation

The impaired immune and inflammatory response

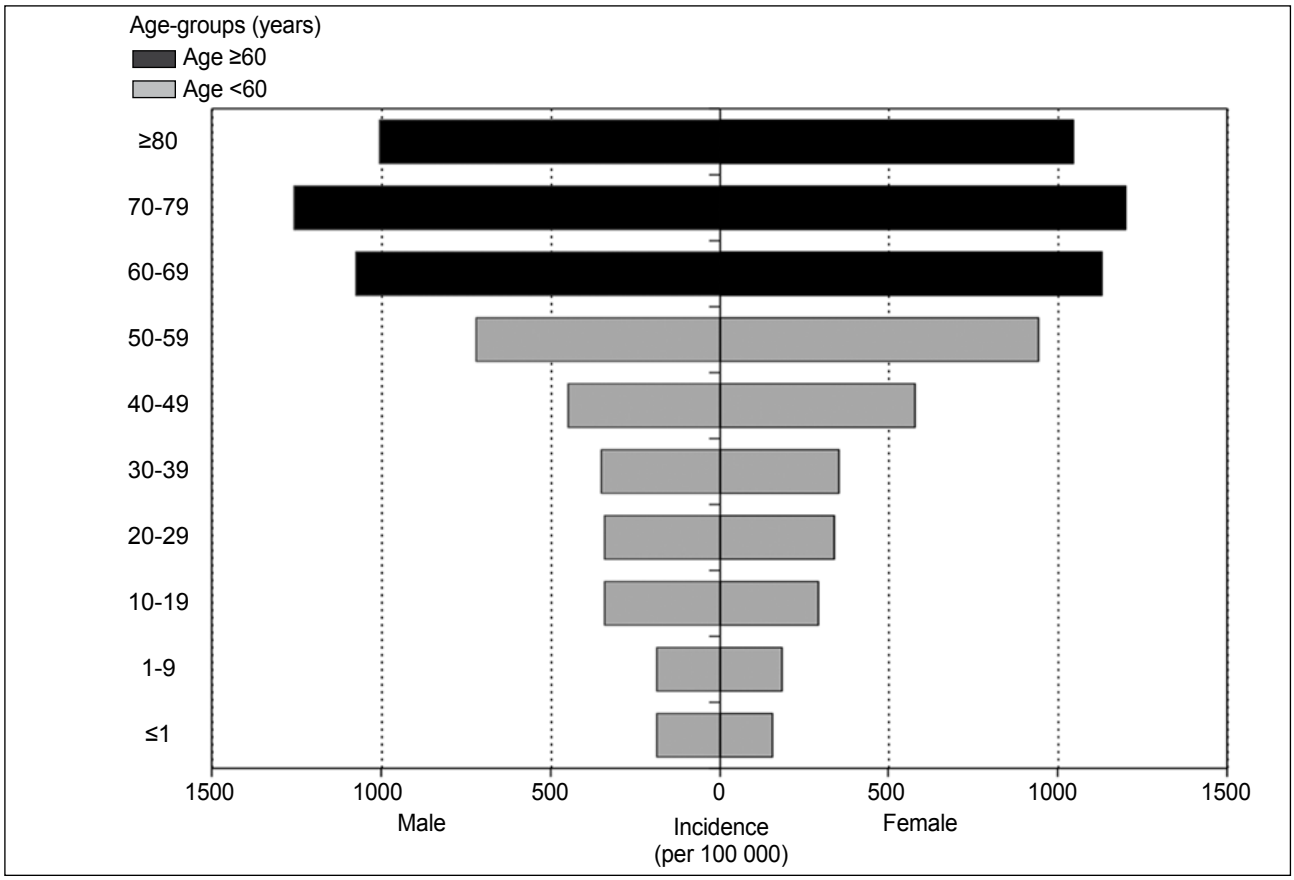

Figure 29.2 Age- and sex-specific annual incidence of herpes zoster in Taiwan from 2000 to 2005[5]

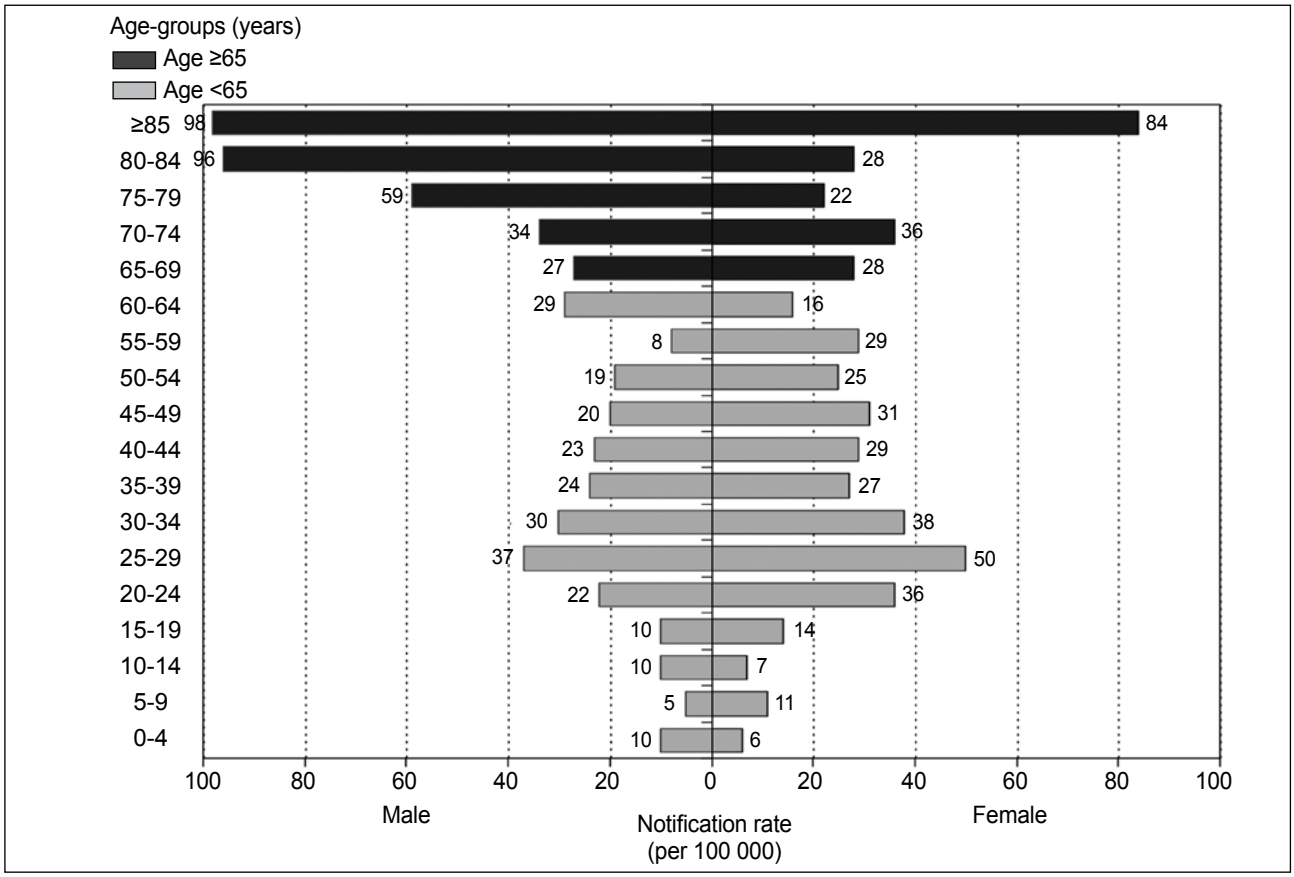

Figure 29.3 Age- and sex-specific notification rates of SARS in Hong Kong in 2003[6]
Abbreviation: SARS = severe acute respiratory syndrome

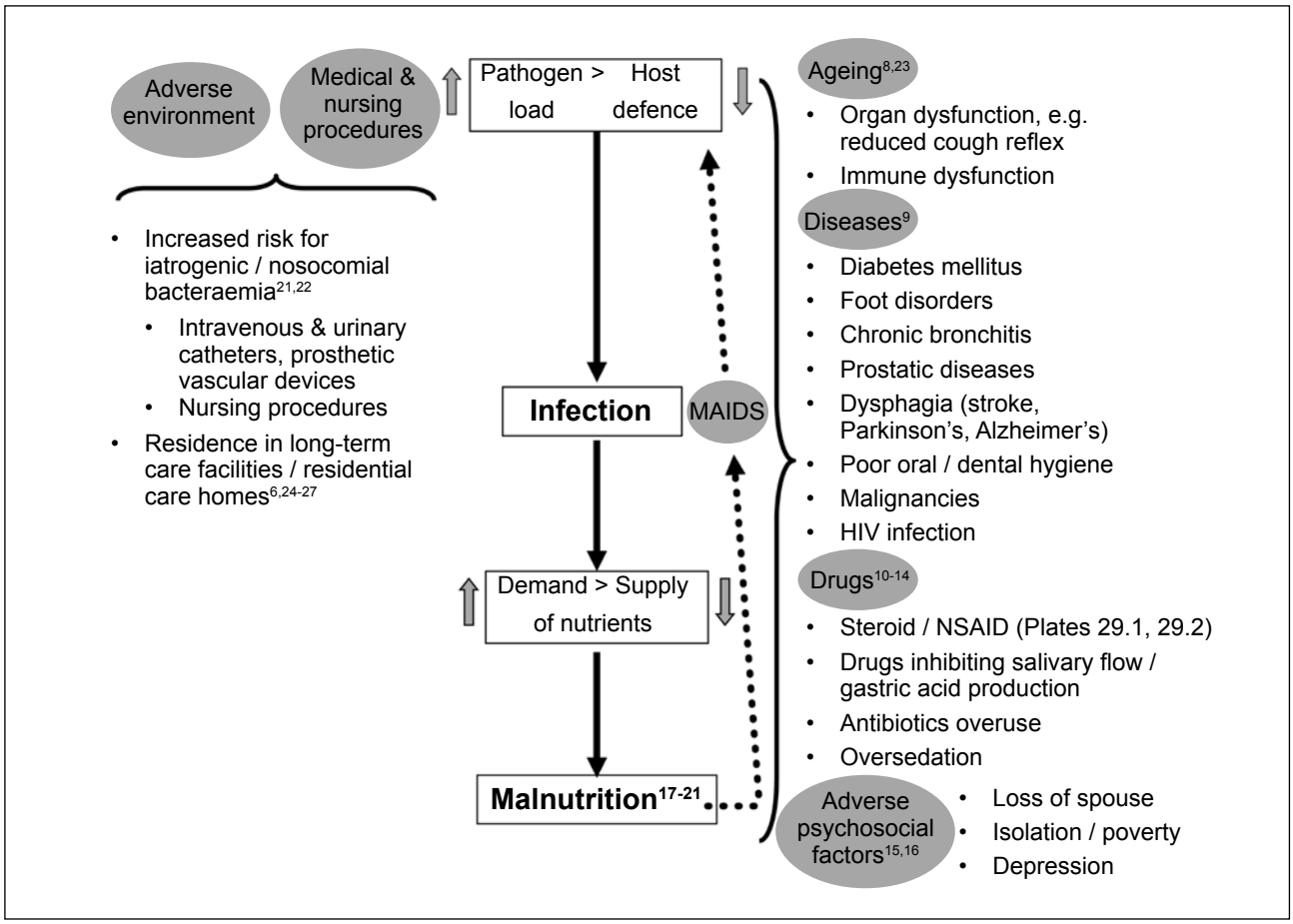

Figure 29.4 Factors for the increasing risk of infections in old age[6,8-27]
Abbreviations: HIV = human immunodeficiency virus; MAIDS = malnutrition-associated immune deficiency syndrome; NSAID = non-steroidal anti-inflammatory drug

in old age lead to altered clinical presentations of infections among elders.[2,3,30] Thus, the fever response to infections may be absent or sluggish among elders, and tend to be of lower intensity, especially among frail elders.[31-34] This is illustrated by the differences in distribution of the first temperature among SARS victims for younger adults, elders, and elders residing in residential care homes. While elders have a lower febrile response compared with younger adults, frail elders residing in residential care homes have an even lower febrile response than the fitter community-dwelling elders (Figure 29.5).[6] However, underdetection of fever during sepsis in elderly patients may occur because of lack of vigilance or inappropriate body temperature recording.[34-36] Tympanic and rectal temperature measurements have been found to be better than oral and axillary recordings for detecting pyrexia in elderly patients.[37-39] Careful body temperature recording and continual monitoring have enabled the detection of a fever response, though often delayed, to infections among elders.[31,34] Since elderly patients, especially the frail ones, may have a lower baseline temperature, it may be necessary to rely on a change in temperature rather than on the absolute temperature, in detecting a febrile response in old age.[31,40,41] Studies on body temperature in geriatric patients have led to a proposal of redefinition of fever in old age as a 2 to 4 standard deviation (SD) rise in temperature from an elderly individual's own baseline normal temperature, computed from the average of three (or more) recent non-illness temperatures.[42] Thus, if the mean non-illness body temperature of a residential care home population is 36.5°C, and the SD is 0.3°C, a care home resident with a baseline temperature of 36°C would be regarded as febrile for a temperature rise of 0.6°C (2 SDs) and the likelihood of fever increases with a higher rise of 1.2°C (4 SDs). While the temperature rise may be blunted during sepsis, phasic rise in pulse rate (Figure 29.6) may still be present and this can be a clue to an underlying infection yet to be diagnosed. Some elderly patients with infectious illness may even present with hypothermia[43] because of defective homeostatic system. On the other hand, the presence of fever does not necessarily imply infection, and other differential diagnoses should be borne in mind as guided by the clinical picture, e.g. arthritis, gout (see Plate 33.5), arteritis, and lymphoma.[44]

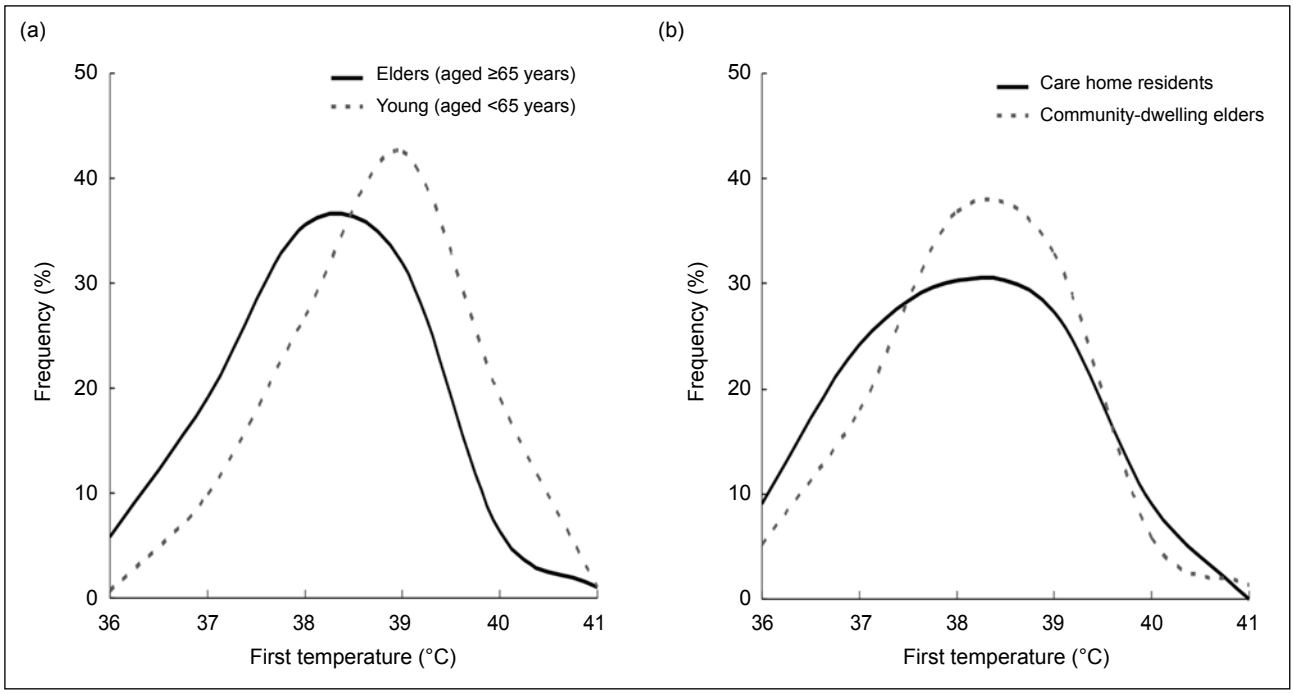

Figure 29.5 Frequency distribution of first temperature among 1467 laboratory-confirmed SARS victims in different population groups (a) elders versus younger adults, and (b) care home residents versus community-dwelling elders[6]
Abbreviation: SARS = severe acute respiratory syndrome

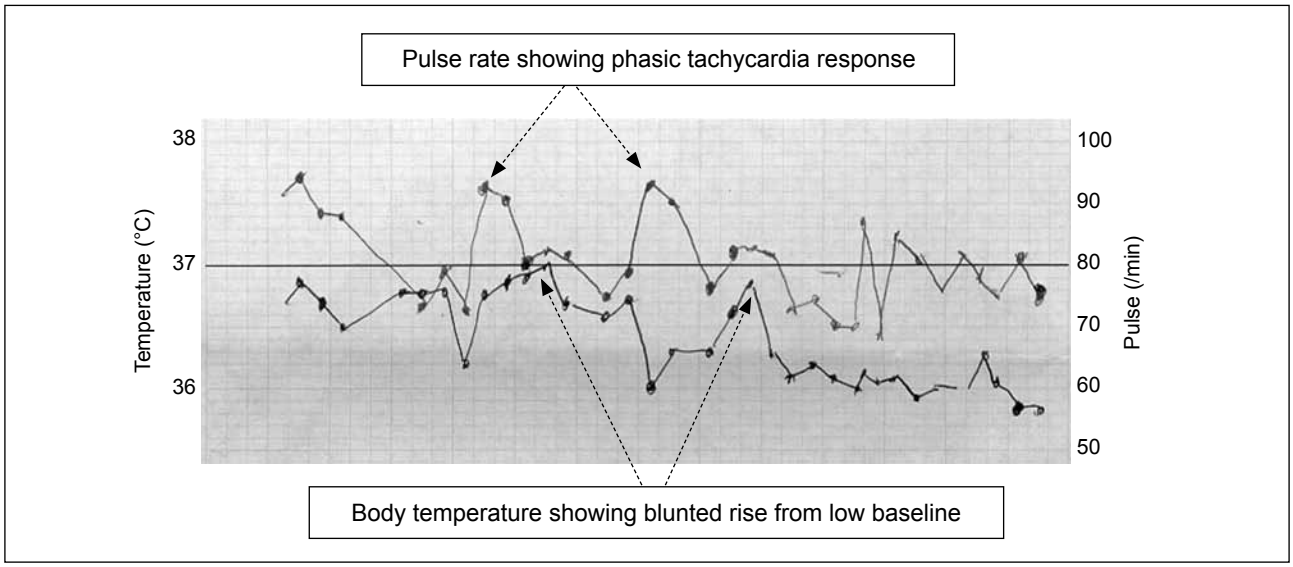

Figure 29.6 Temperature chart of an elderly patient with sepsis, showing blunted temperature rise, but phasic rise in pulse rate

An elderly patient with sepsis may report fewer symptoms because of reduced pain appreciation or communication issues. Instead of focal organ-specific symptoms, their complaints tend to be non-specific and global: a change in appetite (poor feeding, refusal to feed), anorexia, fatigue, weakness, or weight loss. Presentations with geriatric syndromes with functional / cognitive impairments are common: falls, delirium, incontinence, and functional decline.[42,45] Because of the common occurrence of multiple pathologies and the interdependence and limited reserve of the different organ systems, an elder may start with an infection in one organ system and present with problems in another organ system. Thus, pneumonia may present as worsening of a chronic illness, such as congestive heart failure,[46]

or as a fall with fracture in an elderly person with osteoporosis or arthritis (Plate 29.2). Pneumonic infiltrate may be absent radiologically because of initial dehydration or, when present, be masked by underlying or coexisting illnesses such as chronic obstructive pulmonary disease or congestive heart failure.[47] The altered presentations of tuberculosis[48-53] and infective endocarditis[54-56] in old age also pose diagnostic challenge. Tuberculosis, a common infection among elderly people in Hong Kong (Figure 29.1), may have many faces of presentation,[57,58] may simulate malignancy or coexist with malignancy.5[9,60]

29.3 Impact

Studies have revealed the high mortality and morbidity associated with pneumonia and respiratory tract infections in old age, especially among frail elders or in those presenting non-classically (e.g. when patients are afebrile). These may be related to the poor immune response to infection and delayed recognition of infection.[47,61-63] Infection may exhaust the functional reserve of an elderly individual, resulting in functional decline and increase in dependency, disability or handicap, with consequent stress on caregivers. Fear of contracting infection, best exemplified by the emergence of SARS epidemics in 2003, may result in isolation, reduced activities, and delayed presentation of other illnesses; all of which are potentially hazardous to elderly people.[6] Delayed or missed diagnosis of infectious disease in elders has been reported to result in serious outbreaks of tuberculosis,[64] parainfluenza,[65] influenza,[66] multidrug-resistant pneumococcal pneumonia,[67] SARS,[68,69] Legionnaires' disease,[70,71] and *C. difficile*–associated diarrhoea.[72]

29.4 Prevention

Prevention of infectious diseases is particularly important in elderly people because this age-group of population tends to consume a large proportion of medical and hospital services. They are at risk for a higher age-specific rate of infection and a more severe course of illness with functional impact or fatality than the normal population. There is also the potential for diagnostic uncertainty and the impact of spread from undiagnosed infectious illnesses or emergence of resistant organisms (e.g. vancomycin-resistant enterococci, and methicillin-resistant *Staphylococcus aureus*) from overuse of empirical antibiotics.[73] Early detection of infections in elderly patients by trained medical professionals with diagnostic acumen and familiarity with the complex presentations of illnesses in old age is, thus, of paramount importance. Since the classical presentations of infections in a frail elderly person may be replaced by functional decline, careful observation and assessment of change in the functional status of an elderly individual would provide additional sensitive evidence (though non-specific) of an emerging infectious illness. This has to be matched by a healthcare system and a multidisciplinary care setting that can meet the diagnostic challenge, special needs and complexity of disease conditions in frail elders.

As secondary causes of immunodeficiency are common in old age, these should be reversed by treating comorbid diseases, correcting malnutrition, avoiding iatrogenesis, and appropriate use of drugs that might suppress body defence. The emergence of the new *C. difficil*e, which was reported to cause multi-institutional outbreaks of *C. difficile*–associated diarrhoea with high morbidity and mortality in elders, is a timely reminder of the importance of the appropriate use of antibiotics to reduce the emergence of new resistant strains of microorganisms from antibiotic pressure.[74] Probiotics (e.g. *Lactobacillus* species) may expedite the recovery of the microflora following damage by antibiotics, thus, limiting overgrowth of *C. difficile*.[75] The appropriate use of invasive procedures, good nursing care (of skin, foot, oral cavity, and bladder), as well as good infection control behaviour of healthcare workers will help to minimise the introduction of nosocomial pathogens into elderly patients. Reduction in the use of restraints and a rehabilitative approach to encourage activities and appropriate positioning will help to promote immunity and reduce aspiration. All these have to go in hand-in-hand with public health (e.g. influenza vaccination, see Chapter 32) and environmental measures, as well as social interventions (e.g. adequate meal service to preserve nutrition).

29.5 Fever of Unknown Origin

The traditional definition of fever of unknown origin (FUO)[76] as illness of more than 3 weeks' duration, fever higher than 38.3°C on several occasions, and diagnosis uncertain after 1 week of study in hospital, needs to be modified for elderly patients.[77] The febrile threshold of 38.3°C may have to be lowered[77] because of lower baseline body temperature and blunted febrile response to infections in old age, as discussed above. A week of inpatient evaluation is not necessary because of the development of community geriatric services and the ability to perform diagnostic investigations in outpatient settings. FUO should be considered when a febrile illness does not resolve itself in the period expected for self-limited infection and when cause of fever cannot be determined despite considerable diagnostic effort.

A study in Belgium that compared FUO in elderly patients (aged >65 years) with that in younger patients revealed that a precise diagnosis was more readily reached in elderly patients (88% vs. 70%) and FUO in elderly patients was often the result of atypical presentation of a common disease.[78] Infection

was the cause in 25% of elderly patients with FUO, with tuberculosis occurring much more commonly in elderly than younger patients with FUO (12% vs. 2%).[78] Connective tissue diseases such as giant cell arteritis, polymyalgia rheumatica and rheumatoid arthritis accounted for 31% of causes in elderly patients, and giant cell arteritis was the most frequent specific diagnosis in elderly FUO (17%).[78] Malignancy accounted for 12% of FUOs in all elderly patients, much higher compared with that in younger patients (5%).[78] Since these diseases are treatable, the causes of FUO in elderly patients should be investigated and diagnosed promptly.

However, the spectrum of diseases causing FUO may vary across different races and geographic locations. A study in Beijing among elderly Chinese patients with FUO (mean age, 71 years) showed that a diagnosis could be made in 90% of the elders with FUO, and infection was the leading cause in 49.4% of them, with tuberculosis (both pulmonary and extrapulmonary) being the most frequent specific diagnosis (26%).[79] Connective tissue diseases, including vasculitis, accounted for 17.9% of cases, but no confirmed case of giant cell arteritis was diagnosed in this study because no temporal artery biopsy was performed. Malignancy accounted for 15.6% of the cases, and lymphoma was the most frequent malignancy causing FUO (7.3%). In both the Beijing and Belgium studies of FUO in elderly patients, tuberculosis emerged as the most important specific infection and accounted for half of the infectious causes of FUO in old age. The importance of considering disseminated or intra-abdominal tuberculosis as a common cause of FUO in Hong Kong has long been recognized in clinical practice.[80] The relevance of giant cell arteritis for the Chinese will be discussed in Chapter 33.

The evaluation of elderly patients with FUO requires a geriatric perspective that differs from that needed in younger patients. Compared with younger adults, the differential diagnosis of FUO in elderly people is different, disease presentation frequently nonspecific, symptoms difficult to interpret, improvement often slower and less dramatic, and maintaining functional independence is a more important goal. The limited reserve that characterises elderly persons makes them vulnerable to irreversible changes and functional deterioration if the underlying illness is not diagnosed and treated promptly. The investigation of elderly patients, who may not tolerate prolonged repeated FUO investigation, should be guided by the spectrum of underlying diseases rather than by reported results of selected diagnostic tests, and considering the more common causes before the rare ones. The extent of investigation, adoption of an observational approach, or initiation of a therapeutic trial should be individualised according to the frailty and clinical stability of an elderly person. A stepwise approach to FUO in elderly patients adapted from Tal et al[77] for the local setting is as follows.

- Confirm fever by serial measurements of body temperature.
- Take careful history and perform a physical examination focusing on intra-abdominal diseases, cardiac disorders, tuberculosis, musculoskeletal disorders, and cancers; and travel history.
- Discontinue all nonessential drugs and see if fever subsides.
- Search and perform a workup for tuberculosis.
- Perform relevant investigations: chest radiographs, total blood count with differential count, erythrocyte sedimentation rate (ESR), a chemistry panel, urinalysis, multiple cultures of urine and blood, thyroid-stimulating hormone. Consider testing for HIV, Epstein-Barr virus, and cytomegalovirus infections, and presence of antinuclear antibody in specific cases.
- Perform an abdominal ultrasonography, echocardiography, transoesophageal echocardiography, and computed tomography of the abdomen and chest.
- Search for pulmonary embolism, particularly in bedridden patients.
- Scanning with gallium-67–, indium-111–labelled autologous leukocytes or 18-fluorodeoxyglucose positron emission tomography scan
- Consider therapeutic anti-tuberculosis trial / broad-spectrum antibiotic trial.
- Further invasive investigations may be considered depending on clinical suspicion, patient well-being and fitness:
  - consider temporal artery biopsy, if compatible clinical features and ESR of >40 mm/h
  - consider liver and bone marrow biopsy
  - laparoscopy

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CHAPTER 30

Nutrition Disorders in Older Adults

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OBJECTIVE

To understand the definition, prevalence, consequences, and management of nutrition disorder in older adults and the application of common nutritional screening tools.

KEY POINTS

- Undernutrition is common, especially in high-risk older adults.
- Nutritional screening should be part of a routine clinical assessment.
- Risk factors of undernutrition should be reviewed.
- An individual care plan should be devised and progress monitored.
- An understanding of age-related physiological changes in body composition is important.
- The differences between, as well as overlap of, anorexia, cachexia, and sarcopenia should be understood.
- Malnutrition has severe consequences.
- Early diagnosis and treatment is the key to success in improving outcome.
- Body mass index in old age is of limited value.
- Overweight in old age is protective for survival.

30.1 Introduction

Malnutrition has been used interchangeably with the term 'undernutrition' or 'undernourished'. Nonetheless malnutrition encompasses both overnutrition and undernutrition and describes a deficiency, excess, or imbalance of a wide range of nutrients, including micronutrients, resulting in adverse health outcomes. To avoid ambiguity, we deliberately employ the term 'nutrition disorders' to indicate excess, deficiency, or imbalance in nutritional status. Since being underweight or undernourished is definitely associated with adverse outcomes, particularly in hospitalised patients and in long-term care facilities, it will be discussed in more detail than being overweight. The advantageous and disadvantageous effect of being overweight in old age will be discussed where relevant.

30.2 Age-associated Changes in Body Composition

An understanding of age-associated changes in body composition is important in distinguishing undernutrition changes from those due to the ageing process. Body weight peaks between age 55 and 65 years in women and between age 34 and 54 years in men. Fat-free mass (FFM), or muscle and bone mass, starts to decrease and fat mass increases from mid-life. After 70 years of age, fat mass also tends to decrease, resulting in a decline in total body weight. Men are estimated to lose up to 6.6 kg between age 70 and 81 years compared with 5.7 kg in women over the same period. It is worthwhile to note that the concurrent reduction in FFM may render body fat percentage relatively constant or cause it to rise slightly despite a decrease in absolute fat mass. Therefore a higher percentage of fat in older adults may imply a more rapid relative loss of FFM (bone and muscle), not of being overweight or obese.

At the same time, the distribution of adiposity shifts from the periphery (limb and subcutaneous) to the trunk (visceral). It is still questionable whether this central adiposity shift carries as much cardiometabolic risk as that in mid-life. Studies in Hong Kong community-living older adults have observed that older men may benefit from being overweight and centrally obese and older women were resistant to the hazard of these two conventional cardiometabolic risk factors.[1] In this Hong Kong cohort study, the best surviving men were those with a body mass index (BMI) of 24-26 kg/m^2 and waist-hip ratio of 0.90-0.94. It is possible that the World Health Organization (WHO) or Asia-Pacific classification of BMI is not applicable in old age. Overall, a BMI of <20 kg/m^2 should always be cautioned as being underweight

and a high BMI of up to 26 kg/m^2 may be best for survival and even if higher, may not confer any excess mortality.

Although being underweight is always disadvantageous in old age, being overweight can be a disadvantage in terms of morbidity despite being advantageous in mortality. Woo et al[2] have shown that the relationship between BMI and walking speed is U-shaped. Walking speed was slower at both low and high BMI values. Being obese but lacking muscle (sarcopenic obesity) is an apparent cause of disability. Perhaps the optimal balance between muscle and adiposity is the major determinant of good physical function in older adults.[3]

Three patterns of weight loss have been recognised in old age but the pathophysiological mechanisms overlap as detailed below.

30.2.1 Wasting / Anorexia

This is an involuntary loss of weight, primarily attributed to insufficient food intake. It may be related to both medical and psychosocial factors, and may overlap with cachexia or sarcopenia or both. Loss of appetite is central to the development of wasting and is a major cause of undernutrition in old age.

30.2.2 Cachexia

Cachexia is an involuntary loss of muscle mass due to catabolism, and results in changes in body composition. Nonetheless weight loss may not be apparent initially. It is characterised by a raised metabolic rate, an acute immune response, and increased protein degradation. This involves the activation of interleukin 1 (IL1), tumour necrosis factor α (TNFα), and IL6. These are cytokines responsible for the metabolic response to injury or stress and exert their effects on hormone production and hormonal control of metabolism, resulting in an increased resting energy expenditure, and a shift from the synthesis of albumin to the production of acute phase proteins. Overall, nitrogen balance becomes negative and muscle mass is lost. Cachexia is seen in many other diseases such as rheumatoid arthritis, congestive heart failure, human immunodeficiency virus infection, and cancer, and also in situations of metabolic stress such as trauma, infections, and pressure ulcers.

30.2.3 Sarcopenia

Sarcopenia is an involuntary loss of muscle mass associated with ageing rather than the effect of age-associated disease. The exact aetiology is unknown. A lack of physical activity is definitely crucial to the development of sarcopenia but inactivity is not the only cause. Growth hormone, testosterone, and oestrogen have all been investigated as possible mediators of sarcopenia. The pro-inflammatory cytokines such as TNFα, IL1, IL6, serotonin and interferon gamma, which may also play a major role in cachexia, are known to stimulate protein breakdown in muscle. Ageing is characterised by decreasing production of growth and sex hormones, and elevation of pro-inflammatory cytokines. There is substantial evidence that activation of TNFα leads to systemic inflammation, muscle loss and anorexia, which similarly is occurring in the ageing process.

It has also been proposed that the role of central nervous system (CNS) degeneration is central to the development of sarcopenia and in particular differentiates this type of weight loss from wasting and cachexia.[4] We have observed that cognitively impaired older adults have lower muscle mass and weaker muscle strength than the cognitively normal.[5] Furthermore, those who had lower muscle mass and strength had faster cognitive decline.[6] All these support the interlinkage between CNS degeneration and muscle loss.

30.3 Epidemiology of Nutrition Disorders

A local survey in Hong Kong of 4000 community-dwelling adults ≥65 years revealed a prevalence of being underweight (BMI <18.5 kg/m^2) of 4.6%, normal weight (BMI 18.5-22.9 kg/m^2) of 37%, overweight (BMI, 23-24.9 kg/m^2) of 27%, and being obese (BMI ≥25 kg/m^2) of 31%.[2] The cohort was nonetheless underrepresented by the physically frail so the genuine prevalence of being underweight is likely to be higher.

Undernutrition is common but underrecognised in hospitalised older patients. A local study conducted in an acute geriatric ward revealed that being underweight was undiagnosed in 50% of the elderly patients. Height and body weight were not recorded in 56% and 23% of cases, respectively. Of those with weight recorded, 23% lost over 6 kg and 37% had a serum albumin level of <30 g/L.[7]

A survey in UK hospitals revealed that the in-hospital prevalence of malnutrition was 13-40% and 28% of inpatients were at risk of undernutrition.[8] The prevalence was higher in specific subpopulations, e.g. 34% of all emergency admissions and 52% of admissions from care homes.[9] In a Hong Kong local survey conducted in an old age home, Lee et al[10] reported a 26.1% prevalence of being underweight (BMI <18.5 kg/m^2). In the UK, specific micronutrient deficiencies have also been investigated, e.g. folate deficiency has been described in 29% of the independent elderly population and 35% of those in institutional care.[11]

30.4 Causes of Undernutrition

30.4.1 Appetite and Anorexia

Anorexia is likely the major cause of undernutrition. It is apparent that dietary energy intake decreases with age although the underlying mechanisms are not clearly understood. Ageing seems to be associated with an impaired ability to regulate food intake. Older people fail to reduce their energy expenditure during periods of negative energy balance and the weight lost will take longer to be re-gained.

Satiety depends on a process involving relaxation of the stomach wall and the hormone cholecystokinin that slows gastric emptying, increases with ageing, and results in earlier satiety. Reduced stomach capacity in old age can also lead to earlier satiety.

Other gut peptide hormones, including ghrelin, cholecystokinin, peptide-YY, glucagon-like peptide 1, oxyntomodulin, and pancreatic polypeptide regulate appetite. Ghrelin has a stimulatory effect, the others are all inhibitory. It is possible that ghrelin secretion may reduce during ageing, illness, or trauma. It remains uncertain whether dysregulation of these gut hormones is the underlying mechanism of the anorexia of ageing.

Cytokines are also involved in the regulation of appetite. Ageing is associated with elevation of TNFα, IL1, IL6, and serotonin; all of which are known to cause anorexia and may also be involved in increased muscle breakdown and nitrogen loss. These cytokines are also increased during infection, injury, and chronic inflammation.

Many neurotransmitters are involved in the regulation of food intake, of which CNS nitric oxide in the brain may play a central part in appetite regulation. Opioids are thought to increase food intake. Ageing is associated with a loss of opioid receptors and reduced brain concentrations of endogenous opioids. Therefore, older persons may be less sensitive to endogenous opioid action.

30.4.2 Taste and Smell

Loss of taste and smell is common in older adults and can be exacerbated by disease and medications. Possible accountable mechanisms include a reduction in the number of taste buds, or a decrease in the sensitivity of taste sensation receptors. Many drugs can change taste and smell, including drugs in the following groups: lipid-lowering drugs, antihistamines, antibiotics, anti-inflammatories, bronchodilators and other asthma drugs, antihypertensives, Parkinson's disease treatments, and antidepressants. Drug reconciliation can help improve taste and smell. Dietary flavour enhancer is another alternative.

30.4.3 Oral Health and Dental Status

Oral health and dentition generally deteriorate with ageing. Edentate people report greater difficulty with eating, more chewing problems, and more mouth dryness. Chewing problems are associated with poor general health and lower quality of life. Energy intake has been shown to be lower in edentate people, as were many micronutrients, fibre, and protein. Nutrient intake is better and BMI is higher in dentate people.

30.4.4 Dysphagia

Oropharyngeal dysphagia can apparently reduce dietary intake. Dementia, stroke, and Parkinson's disease are commonly associated with dysphagia. Dyspnoea in chronic obstructive pulmonary disease and heart failure is also a major cause of dysphagia. Old age home residents with dysphagia are frequently fed by care workers. Insufficient resources and feeding can also account for undernutrition in this group of people.

30.4.5 Disease and Disability

Common diseases or conditions associated with undernutrition are listed here:

- end-organ failure: chronic obstructive pulmonary disease, congestive heart failure, chronic kidney disease;
- malabsorption;
- diabetes mellitus, thyrotoxicosis;
- stroke, Parkinson's disease, dementia;
- acute and chronic infection including tuberculosis;
- physical disability: arthritis, poor mobility;
- drugs: distaste, nausea and vomiting, delayed gastric emptying, anorexia, diarrhoea, and malabsorption;
- malignancy; and
- frequent hospitalisation

30.4.6 Lifestyle and Psychosocial Factors

Several lifestyle and psychosocial factors have been studied as determinants of undernutrition and found to be significant independent determinants of intake in older adults. They include cooking knowledge, loneliness and isolation, income, food beliefs and attitudes, stress and bereavement, services and assistance available, dentition, food availability, food expenditure, functional disability, and vision.

Reduced food intake is a symptom of depression. Bereavement, anxiety, and stress are also thought to be associated with changes in food intake, leading to unintentional weight loss.

30.4.7 Dementia and Confusion

In hospitalised older adults, an inverse relationship between energy intake and cognition suggests

that cognition may impair the ability or desire to eat. Weight loss and altered eating behaviour is a recognised feature in dementia. Nearly half of all patients with Alzheimer's disease lose the ability to feed themselves 8 years after diagnosis. Nonetheless eating problems, such as slower oral manipulation of food and slower swallow response, may appear even in the early stages of Alzheimer's disease. In addition, anorexia and dementia share some underlying pathophysiological mechanisms including activation of the cytokine systems. It is known that olfactory changes occur during Alzheimer's disease, and smell is an important component of taste. This may also affect eating behaviour and food intake.

30.5 Impact and Consequences

Undernutrition can lead to serious consequences in terms of morbidity and mortality. It is seen as a marker for illness but usually ignored in daily clinical practice, both in the hospital and the community. The associated adverse health outcomes include the following.

30.5.1 Higher Mortality

Although being underweight undoubtedly confers excess mortality, whether being overweight is deleterious to survival in old age remains questionable. In a local cohort study of 2000 older men and 2000 older women, the best BMI for survival in men was 24-26 kg/m^2 and a high BMI in women did not increase mortality.[1] Flicker et al[12] observed a similar pattern in the Australian older population. The best BMI for survival was within the overweight range of BMI 25-30 kg/m^2 in Australian older men and women.[12] It appears that the WHO classification of BMI should be interpreted differently when applied in old age. Weight reduction management in overweight older adults should only be based on specific indications and not generally based on the WHO BMI classification.

30.5.2 Physical Limitations and Disability

Although being underweight is associated with physical limitations, Woo et al[2] have demonstrated that overnutrition or high BMI was also a risk of disability, likely related to sarcopenic obesity (co-existence of obesity and muscle loss). A local study observed a similar relationship with muscle mass.[13] The paradoxical excess risk occurring with high muscle mass can be explained by the associated high BMI. Perhaps a well-balanced adiposity to muscle ratio is the key to independence in old age.[3] In addition, underweight and muscle loss can predict faster cognitive decline, giving rise not only to physical but also cognitive disability at the same time.[6]

30.5.3 Impaired Immunity and Delayed Wound Healing

Immune function is also affected, increasing the risk of infection due to impaired cell-mediated immunity and cytokine, complement, and phagocyte function. Delayed wound healing is also well described in malnourished surgical patients. Being underweight is a risk factor for pressure ulcer occurrence and also delays healing.

30.5.4 Psychosocial Effects

In addition to these physical consequences, undernutrition results in psychosocial effects such as apathy, depression, anxiety, and self-neglect.

30.5.5 Increased Hospital Length of Stay and Treatment Cost

Underweight older patients tend to stay in hospital longer, have more frequent admissions, and therefore demand higher health expenditure.

30.6 Assessment, Screening, and Management

30.6.1 BMI

BMI has been conventionally employed to represent the nutritional status of older persons. It, however, tends to overestimate being overweight and underestimate being underweight because of progressive height loss related to ageing.[14] Taking into account an underestimation in being underweight, in general, a BMI of <20 kg/m^2, rather than 18.5 kg/m^2, should be regarded as a warning sign of undernutrition. In addition, many frail and undernourished older patients cannot stand, making height measurement impractical. Alternative methods have been developed to estimate stature, including knee height, arm span, and long bone length. All are compromised by reliability and validity limitations. A locally developed estimation formula[15] should be adopted as far as possible because the estimation can vary widely across various ethnicities. The classification of BMI is shown in Table 30.1.

30.6.2 Serial Body Weight

If available, significant weight change is more informative than a single body weight or BMI measurement. A 5% loss in 1 month or a 10% loss in 6 months is regarded as significant unintentional weight loss. It is strongly recommended that the body weight of older patients should be charted at every clinic or hospital encounter to enable serial monitoring, similar to routine blood pressure charting. Chair weighing scales should be provided for disabled patients who are unable to stand.

Table 30.1 Classification of body mass index (BMI) in adult Asians and Europeans

Classification	BMI (kg/m²) in Asians	BMI (kg/m²) in Europeans	Risk of comorbidities	
			Asians	Europeans
Underweight	<18.5	<18.5	Low (but increased risk of other clinical problems)	
Normal range	18.5-22.9	18.5-24.9	Average	
Overweight	≥23	≥25		
Pre-obese	-	25-29.9	-	Increased
At risk	23-24.9	-	Increased	-
Obese I	25-29.9	30-34.9	Moderate	Moderate
Obese II	≥30	35-39.9	Severe	Severe
Obese III	-	≥40	-	Very severe

30.6.3 Other Anthropometric Measures

Other anthropometric measures include:
- triceps or subscapular skin-fold thickness;
- mid-arm muscle circumference and mid-arm muscle area; and
- waist-hip ratio

30.6.4 Biochemical Indices

There is no reliable biochemical marker for malnutrition. The commonly quoted at-risk biochemical levels are shown in Table 30.2, but it is important to note that biochemical data are altered during acute insult, oedema, or chronic disease state (e.g. renal or liver failure). Caution is thus required in their interpretation, especially among older hospitalised patients and those with concurrent inflammatory changes.

Table 30.2 Laboratory levels that indicate risk of malnutrition

Laboratory test	At-risk level
Serum albumin (g/L)	<35
Total lymphocyte count (cells x 10^9 /L)	<1.5
Serum transferrin (g/L)	<1.4
Serum pre-albumin (mg/L)	<170
Total iron binding capacity (μmol/L)	<44.8
Serum cholesterol (mmol/L)	<3.89

30.7 Nutritional Screening

Although assessment reflects nutritional status, screening is a simple way to identify older persons at risk in a wider population. The screening process identifies patients who require referral to dietitians for a more sophisticated assessment and management plan. Rescreening of inpatients at 7-day intervals throughout a hospital stay is recommended to detect at-risk patients with significant weight loss at a later stage. The screening tools are nonetheless not substitutes for detailed nutritional assessment. The common screening tools include: brief self-reported screening instruments (Mini Nutritional Assessment or MNA); nutrition screening initiatives (clinician-determined brief screening instruments, Subjective Global Assessment or SGA); and multi-method techniques.

The MNA[16] is simple, quick, and can be used in hospital, outpatient clinics, and nursing homes. It is a gold standard against which other tools can be judged. The results compare well with a standard extensive nutritional assessment. The SGA[17] is a clinical tool that involves two steps: clinical history and physical examination. Patients are classified as well nourished, moderately malnourished, or severely malnourished.

The Nutrition Screening Initiative is a tiered tool to screen older people who are at increased nutritional risk. The first level is a self-check list, followed by a two-level approach by professionals.[18]

30.7.1 Malnutrition Universal Screening Tool

The MUST (Malnutrition Universal Screening Tool)[19] is a simple, rapid, and easy method to screen patients that aims to identify those at risk by incorporating:
- current weight (BMI);
- history of recent unintentional weight loss; and
- likelihood of future weight loss

30.8 Management

The strategy for implementation of nutritional intervention is multifaceted and varies among different settings, e.g. hospital, community, and institution. Reversible psychosocial risk factors should be sought and rectified. At times there may be a need to draw up a management plan tailored for individual needs with particular attention paid to personal choice, and cultural and socioeconomic background. A multidisciplinary approach is needed in high-risk older adults, especially in those who are hospitalised.

Nutrition therapy includes review and analysis of the medical and dietary history, physical examination, biochemical data, and anthropometric measurements. The choices of nutritional modalities are:
- diet modification, counselling, and individualised dietary plan; and
- special diet and therapies such as nutrient

supplementation, enteral, or very rarely parenteral nutrition

Simply providing regular meals or food with better nutritional content may be enough to address nutritional risk. In hospitalised patients, additional measures may include broader food choices, flavour enhancers, liberating dietary restrictions, medication reconciliation, adequate staff for assistive feeding, protected meal times, freely allow private food and many common sense methods. Addition of oral nutritional supplements or temporary enteral tube feeding may be considered in some special cases. Patients rarely require parenteral nutrition. Need for parenteral nutrition usually occurs in the context of an inaccessible or non-functioning gastrointestinal tract. Serial body weight charting and accurate intake charting are important monitoring tools to gauge progress.

Oral nutrition supplementation is rather commonly prescribed in inpatients and has been advocated as a nutrition therapy. Mortality may be reduced in older people who are undernourished. There may also be a beneficial effect on complications but there is no evidence of improvement in functional benefit or reduction in length of hospital stay with supplements. Additional data from large-scale multicentre trials are still required.

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CHAPTER 31

URINARY TRACT INFECTIONS

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OBJECTIVE

To learn the epidemiology, clinical presentation, investigation, and management of urinary tract infection occurring in various conditions that are commonly encountered in older persons.

KEY POINTS

- Asymptomatic bacteriuria is common in older persons but antimicrobial treatment is indicated only in those undergoing urogenital surgery.
- Urosepsis in older persons is likely to be due to a complicated urinary tract infection. Urine should be sent for culture prior to prescribing antibiotics and therapy adjusted according to the results of sensitivity testing.
- Duration of antibiotics for complicated urinary tract infections should be 7 days in women and 10-14 days in men.
- A Foley catheter in place for longer than 2 weeks usually results in bacteriuria. Short-term antibiotic therapy for 5-7 days is indicated if the patient is symptomatic.

31.1 Introduction

Urinary tract infections (UTIs) are among the most common infections encountered in medical practice. Among older persons, UTI is the most common bacterial infection encountered in the outpatient setting, nursing home, and hospital. Co-existing diseases as well as the ageing process predispose to the development of symptomatic UTI in elderly persons. Diagnosis and treatment of UTI in older persons differ significantly to that for younger patients. Clinically relevant information is reviewed in this chapter to update the management of UTI in older persons. Much has been published about previously uncertain issues related to UTI since the first edition of this chapter. This edition provides updated information about the evolving evidence for diagnosis, treatment, and prevention of UTI.

31.2 Terminology

Bacteriuria is the presence of bacteria in bladder urine.[1] In clinical practice, bacteriuria is diagnosed by urine culture. The urine culture can be affected by the method used to collect the sample. In a voided urine sample, bacteria normally resident in the terminal urethra can be dislodged into urine as it exits the bladder through the urethra. This results in urine contamination. To avoid such errors, quantitative bacterial counts have been used since the 1950s to distinguish between contamination and true bacteriuria.[2]

Bacteriuria can be either symptomatic or asymptomatic. Asymptomatic bacteriuria is defined as isolation of a specified quantitative count of bacteria in an appropriately collected urine specimen obtained from a person without symptoms or signs referable to urinary infection.[3] Increased urinary leukocyte[2] and cytokine level in bacteriuria indicates a host response.[4] Hence asymptomatic bacteriuria reflects infection rather than colonisation of the urinary tract. But treatment is generally not indicated.

Symptomatic bacteriuria is the occurrence of bacteriuria in the presence of symptoms attributable to infections of the urinary tract and antimicrobial treatment is indicated. Symptomatic bacteriuria is used interchangeably with UTI in this chapter.

Uncomplicated UTI is an acute infection that occurs in the absence of any anatomical, functional, or metabolic abnormalities.[5] The vast majority of UTIs in community-dwelling women seen in family practice belongs to this category and requires minimal evaluation. Uncomplicated UTI occurs rarely in men and is usually managed as complicated.

Complicated UTI is associated with anatomical

abnormalities (e.g. increased residual urine volume, prostatic obstruction, nephrolithiasis, or indwelling Foley catheter), medical conditions (e.g. diabetes mellitus, renal insufficiency, or other immunodeficiency associated diseases) or unusual organisms (suspected in nursing home residents or those with a recent hospital stay). UTI in older persons should be considered complicated. Complicated UTI is more likely to be recurrent, defined as three episodes in the previous 12 months or two episodes in the previous 6 months, and may fail to respond to standard antibiotic treatment.

31.3 Epidemiology

Asymptomatic bacteriuria is a highly prevalent condition. It is more common in females and the incidence increases rapidly with age. It increases from 1-5% in healthy premenopausal women, to 2.8-8.6% in postmenopausal women, and >15% in elderly women aged ≥70 years living in the community.[6] It is more common in older people living in nursing homes than in the community. In a Swedish study, the prevalence of asymptomatic bacteriuria in nursing home residents was 26% for women and 16% for men in municipal institutional care.[7] Asymptomatic bacteriuria is rare in healthy young men but becomes increasingly common after the age of 60 years. This is reflected in a change to the male-to-female ratio for asymptomatic bacteriuria from 1:30 in young adults to 1:2 in an elderly population.[8]

The prevalence of symptomatic UTI is influenced by gender, age, presence of comorbid diseases, and functional disability. In Hong Kong, uncomplicated UTI was clinically diagnosed in 0.7% of a cohort of 1160 patients who consulted a general practitioner for infection in the community[9] while the point prevalence of hospital acquired UTI was 0.8% among 1021 patients in a local university hospital.[10] Clinically diagnosed UTI increases from 0.17 per 1000 days for elderly men and 0.9 per 1000 days for elderly women living in the community, to 0.1-2.4 per 1000 days for nursing home residents.[11]

31.4 Microbiology

Molecular biology techniques to sequence bacterial 16S ribosomal RNA (16S rRNA) have replaced standard urine culture as the gold standard for detecting urinary bacteria. Research using these techniques has changed the long-held dictum that urine is normally sterile and the healthy bladder is pristine.

Healthy female urine analysed by these techniques shows a diverse microbiota that is not detected by standard urine culture. The microbiota includes bacteria with known pathogenic potential such as *Escherichia coli*.[12] Many of the organisms identified in urine by 16S rRNA gene sequencing can also be cultivated using an expanded quantitative urine culture protocol when the standard laboratory urine culture reports no growth at 10^3 colony-forming unit (CFU) per mL.[13] These new techniques show that standard urine cultures reported as culture-negative or insignificant growth actually contain varied bacterial communities. Conversely, in a urine culture that is positive for a single pathogen, that pathogen may be only a tiny fraction of the actual bacterial population. For example, a urine culture that was reported as positive for *E. coli* and analysed by polymerase chain reaction techniques for 16S rRNA found that only 1-2% of urinary bacteria were *E. coli*, while 40-50% were *Aerococcus* and 17-25% were *Actinobaculum*.[14]

Studies based on standard urine culture show that UTIs are caused by a small number of bacterial species. Uropathogenic *E. coli* (UPEC) account for 70-90% of community-acquired UTI. A study of culture specimens collected by general practitioners in Hong Kong revealed that *E. coli* isolates comprised 65.3% of culture-positive community-acquired UTI.[15]

E. coli is normally a commensal in the gastrointestinal tract. Commensal bacteria play a vital role in host health. They promote polysaccharide digestion and nutrient uptake by host intestinal cells. A number of bacteria-regulated genes in host intestinal cells have been identified and they promote normal intestinal homeostasis.[16] A debilitated host or a breach of the gastrointestinal barriers can result in disease when some strains of E. coli acquire virulence factors that allow the bacteria to adapt to new niches.

UPEC possess virulence factors that enable them to overcome host defences in the urinary tract. These defences prevent microbes from ascending the urethra and adhering to uroepithelium in the bladder. UPEC invasion of bladder uroepithelium leads to the formation of intracellular bacterial communities (IBC).[17] IBC have biofilm-like properties that permit adherence to the bladder wall for extended periods. Biofilm has been shown to be up to 1000-fold more resistant to antibiotics than planktonic bacteria in the urine and can re-emerge to cause recurrent infection. Hence, apart from the vagina and perineum, the bladder wall that harbours IBC is also a reservoir for UTI. UPEC forms biofilm on foreign bodies in the urinary tract, such as indwelling catheters and stones, enabling similar properties of antibiotic resistance and recurrent infection. IBC also complicate interpretation of urine culture results in the clinical setting because bacterial density in urine may not correlate with its density in the bladder wall.

UTIs that affect non–community-dwelling patients,

such as nursing home residents or hospital patients, have a higher proportion of microorganisms other than *E. coli*. A study of nursing home residents found that *E. coli* accounted for 53.6% of positive urine cultures, while other Enterobacteriaceae (*Proteus, Klebsiella, and Providencia*) accounted for 34.8% and Gram-positive bacteria (*Enterococcus and Staphylococcus*) accounted for 8.6%.[18] *Proteus mirabilis* and *E. coli* occur with similar frequency in UTI affecting male residents in nursing homes.[7,19] *Streptococcus agalactiae* (group B streptococcus) is an uncommon cause of UTI that can be associated with urinary tract abnormalities or chronic renal failure. *Staphylococcus saprophyticus* is an important cause of acute uncomplicated UTI in young women but is rarely isolated in complicated UTI.[20]

Commensal organisms that normally reside in the distal urethra and perineum include anaerobic bacteria, *Lactobacillus*, *Corynebacterium*, and *Staphylococcus epidermidis*. These organisms do not usually cause UTIs. They are most often reported as contaminants in standard urine culture results.

31.5 Pathogenesis

Microorganisms invade the urinary tract either by ascent from the perineum up the urethra or, less commonly, by the haematogenous route. The importance of the ascending route explains the frequency of bacteriuria in women compared with men because the shorter female urethra affords less protection against bacteria resident in the vagina and perineum. Declining oestrogen level in women after menopause affect the vaginal epithelium, urethra, and bladder trigone with consequent urogenital atrophy. Lactobacilli that contribute to an acidic environment in the vagina are also reduced. This predisposes to vaginal colonisation by faecal bacteria. These factors collectively increase the risk for ascending UTI in older women.

Unobstructed flow of urine in the urinary tract is an essential defence against infection. Voiding removes bacteria in the bladder and urethra by its flushing action. Any disruption of normal voiding such as urinary retention, nidus formation, or reflux of urine back to the upper tract increases the risk and severity of infection. Incomplete bladder emptying occurs more frequently in older persons and predisposes to development of UTI. This can be caused by obstructive uropathy (benign prostatic hypertrophy in men, uterine prolapse or cystocele in women) or neuromuscular disease.

An indwelling urinary catheter is associated with acquisition of bacteriuria and is discussed later on in this chapter.

31.6 Clinical Presentation

The clinical features of UTI can be similar to the classic features seen in younger age-groups. Clinical syndromes are divided into upper tract (pyelonephritis) or lower tract involvement (acute urethral syndrome, cystitis, and prostatitis) and are diagnosed from the clinical presentation (Table 31.1).

31.6.1 Special Geriatric Considerations

Apart from classic features, UTI in elderly people can also present with fever and retention of urine. Fever in the presence of an indwelling Foley catheter can be empirically treated as UTI if other localising signs of infection are absent.

The symptoms listed above are not specific for UTI. Apart from cystitis, other conditions that affect the lower urogenital tract, e.g. urethritis and vaginitis, can cause dysuria. Chronic symptoms of frequency, nocturia, urgency, and incontinence do not indicate infection unless there is an increase in severity or new onset. They should prompt a pelvic examination in women or evaluation of the prostate in older men. An accurate history is important because chronic symptoms are common in older persons regardless of whether infection is present.[21] Treatment of bacteriuria in patients with chronic incontinence does not improve the severity of incontinence.[22]

Diagnosis of UTI can be difficult in older persons who cannot provide a reliable history, as in a patient with dementia. The typical fever response to infection may be blunted. They may present with an altered mental state, decreased appetite, abdominal pain, or new onset of incontinence. These non-specific changes should prompt a search for symptoms more suggestive of UTI, e.g. crying out or grimacing when urinating, urinating more often than usual for that patient, increased incontinence, and increased confusion or agitation. There should be close monitoring for fever and other signs of UTI.

Despite its high prevalence, UTI is over-diagnosed

Table 31.1 Typical symptoms of urinary tract infection

Cystitis
Severe dysuria
Increased urinary frequency
Nocturia
Urgency
Increased incontinence
Foul-smelling urine
Pyelonephritis
Fever, rigors
Flank pain (usually unilateral)
Tenderness
(and / or) Lower urinary tract symptoms

and over-treated in old age.[23] A study of women aged ≥70 years with emergency department–diagnosed UTI found that only 57% had confirmatory findings on urine culture.[24] Non-specific symptoms such as anorexia, fatigue, malaise, insomnia, and weakness do not predict bacteriuria.[21] Similarly, falls in nursing home residents are not associated with bacteriuria plus pyuria.[25] In the absence of other localising symptoms and without an indwelling Foley catheter, UTI accounts for only 10% of fever episodes in the institutionalised elderly.[26]

Inappropriate use of antibiotics fosters antibiotic resistance and healthcare-associated infection such as *Clostridium difficile*. This is a concern particularly for nursing homes due to the remarkably high prevalence of both asymptomatic bacteriuria and symptomatic UTI. Consensus criteria for the diagnosis of UTI can improve infection control practices and avoid unnecessary use of antibiotics in nursing homes (Table 31.2).[27]

31.7 Investigation

31.7.1 Inspection

A study of children in the emergency department found that urine that appears crystal clear indicates absence of bacteriuria with a negative predictive value of >97%.[28] The study on urine clarity was repeated with female adult patients and its negative predictive value for bacteriuria was found to be 86.3%, suggesting that clear urine is insufficiently accurate to exclude UTI.[29] Nonetheless cloudy urine is not a reliable indicator of bacteriuria and pyuria, as phosphate crystals that form in normal urine can cause a similar appearance. *P. mirabilis* infection produces a distinct fishy smell but this is encountered infrequently. Gross haematuria is seen in certain severe forms of cystitis but this is usually absent. Instead, stones and tumours are more common causes of haematuria.

31.7.2 Microscopy

Microscopy of urine specimens detects bacteriuria, white blood cells or pyuria, and squamous cells.

31.7.2.1 Pyuria

Bacteriuria induces an inflammatory response in the urinary tract, resulting in a sharp increase in the urinary excretion of white cells. The upper limit of normal urinary excretion is 400 000 white cells per hour. This corresponds to a count of ≥10 white blood cells per μL of freshly voided urine.[30] Most clinical laboratories report this as >5 white blood cells per high-power field under microscopic examination of urine that has been centrifuged and resuspended.[31]

The most common cause of pyuria is bacteriuria. When pyuria is detected in symptomatic patients with negative urine cultures, a false-negative result is likely and empirical antibiotics should be considered. Other possible causes of sterile pyuria are listed in Table 31.3.[32] For older persons, particularly if they reside in nursing homes, the presence of pyuria and positive urine culture should not be routinely treated

Table 31.2 Society for Healthcare Epidemiology of America criteria for urinary tract infection in long-term care facilities[27]

For residents without an indwelling catheter (both criteria 1 and 2 must be present):
1. At least one of the following signs or symptoms subcriteria:
   (a) Acute dysuria or acute pain, swelling, or tenderness of the testes, epididymis, or prostate
   (b) Fever or leukocytosis and at least one of the following localising urinary tract subcriteria:
      (i) Acute costovertebral angle pain or tenderness
      (ii) Suprapubic pain
      (iii) Gross haematuria
      (iv) New or marked increase in incontinence
      (v) New or marked increase in urgency
      (vi) New or marked increase in frequency
   (c) In the absence of fever or leukocytosis, then at least two of the following localising urinary tract subcriteria:
      (i) Suprapubic pain
      (ii) Gross haematuria
      (iii) New or marked increase in incontinence
      (iv) New or marked increase in urgency
      (v) New or marked increase in frequency
2. One of the following microbiologic subcriteria:
   (a) At least 10^5 CFU/mL of no more than 2 species of microorganisms in a voided urine sample
   (b) At least 10^2 CFU/mL of any number of organisms in a specimen collected by in-and-out catheter

For residents with an indwelling catheter (both criteria 1 and 2 must be present)
1. At least one of the following signs or symptoms subcriteria:
   (a) Fever, rigors, or new-onset hypotension, with no alternative site of infection
   (b) Either acute change in mental status or acute functional decline, with no alternative diagnosis and leukocytosis
   (c) New-onset suprapubic pain or costovertebral angle pain or tenderness
   (d) Purulent discharge from around the catheter or acute pain, swelling, or tenderness of the testes, epididymis, or prostate
2. Urinary catheter specimen culture with at least 10^5 CFU/mL of any organism(s)

Abbreviation: CFU = colony-forming unit

Table 31.3 Causes of sterile pyuria[32]

Infection-related	Recent antibiotic usage, e.g. UTI treatment in past 2 weeks Urethritis by chlamydia, Neisseria gonorrhoeae, mycoplasma, ureaplasma Infection of nearby organs, e.g. gynaecological, prostate, penis, appendix Other infections, e.g. viral, tuberculosis, fungal, parasitic (e.g. trichomoniasis / schistosomiasis)
Non–infection-related	Foley catheter usage (present or recent) Recent invasive urological processes Stones / neoplasms / fistula / foreign bodies (e.g. surgical urethral mesh or stent) Other causes: irradiation, polycystic kidney, renal vein thrombosis, renal transplant rejection, interstitial nephritis or cystitis, papillary necrosis, inflammatory disease, e.g. SLE

Abbreviations: SLE = systemic lupus erythematosus; UTI = urinary tract infection

as active infection. The strong association between pyuria and UTI was originally based on studies of non-catheterised patients living in the community.[33] In the absence of symptoms, pyuria in nursing home residents does not necessarily indicate UTI. A study of incontinent nursing home residents showed that asymptomatic bacteriuria was present in 56% of urine specimens with pyuria. Asymptomatic bacteriuria was also present in 31% of urine specimens without pyuria, indicating pyuria is not a good predictor of bacteriuria in this population.[34] Pyuria is also a poor predictor of bacteriuria in patients who require intermittent catheterisation.[35] Presence of pyuria in catheterised patients in hospital did not differentiate asymptomatic bacteriuria from symptomatic UTI.[36]

31.7.2.2 Squamous Cells

Squamous epithelial cells line the female urethra and the distal portion of the male urethra. Hence a few are commonly seen in normal urine specimens. The vagina is also lined with these cells and when the urine specimen shows massive amounts, it generally indicates vaginal contamination and a repeat specimen should be collected.

31.7.2.3 Bacteriuria

Urine Sample Collection for Culture

The best specimens are collected from the first morning urine because bacterial counts are highest after overnight incubation in the bladder. Bacteria have a relatively slow doubling time in urine (every 30-45 mins). This can affect bacterial count in urine that is voided frequently from the bladder, as occurs in acutely dysuric patients. Urine samples collected after forced drinking of fluids or at random times can also yield falsely low counts.

Collection of urine by mid-stream clean catch is easily contaminated by bacteria in the terminal urethra and perineum if done improperly. The first voided 10 ml of urine can contain 1000 to 10 000 organisms per mL as a result of the bacteria dislodged from the urethra.[37] In women, collection can be improved by instructions to spread the labia and to ensure only the mid-stream is collected without the initial portion or the 'afterdrip'. Accuracy is not improved by perineal washing or antiseptic use.[38] Men should be instructed to retract the foreskin. Mid-stream clean-catch urine may be difficult to obtain in elderly persons who are unable to cooperate due to impaired cognition or uncontrolled incontinence. Alternative methods include collecting from a freshly applied condom catheter in men.[39]

Single catheterisation for a catheter specimen of urine is used when a voided specimen cannot be collected but a urine culture is essential for clinical management. The procedure itself carries a 1-2% risk of introducing UTI.[40] More than 90% of patients with long-term Foley catheters will have bacteriuria after 30 days of catheterisation,[41] with the majority remaining asymptomatic.[42] Urine cultures should only be taken in the presence of symptoms, like fever or loin pain, that suggest infection. Urine samples are aspirated directly from the catheter and should never be taken from the urine bag because bacteria multiply in the bag.

Urine specimens should be cultured by the laboratory within 2 hours of collection as most uropathogenic bacteria grow well in urine kept at room temperature. Refrigeration at 4°C stabilises bacterial counts for at least 24 hours. Use of culture bottles that contain preservatives (e.g. boric acid) can keep the organism count stable without refrigeration. Care should be taken to collect at least 3 mL of urine to avoid spuriously low counts due to a possible antibacterial or dilution effect of the preservative.

Interpretation of Urine Culture Results

Interpretation of the urine culture is based on four key criteria: identification of the organism; its count in urine; the mode of urine specimen collection; and clinical information about the patient.

A single bacterial isolate with a count $>10^5$ CFU/mL usually indicates infection and $<10^4$ CFU/mL indicates contamination of the urine specimen. Most cases of pyelonephritis and cystitis are correctly

diagnosed based on these criteria. Isolation of multiple pathogens or bacteria species that are not the usual cause of UTI should raise the possibility of specimen contamination.

For symptomatic patients, however, there is difficulty in applying the '$>10^5$ CFU/mL' rule in all cases. Various factors can account for a spuriously low-count bacteriuria despite symptomatic UTI. Kass[43] demonstrated significant bacteriuria at a level of $>10^5$ CFU/mL in specimens collected from the first voided morning urine. Subsequent workers found that bacteriuria count was influenced by the time of collection. Women who had bacteriuria counts of $>10^5$ CFU/mL in their first voided morning urine specimens had reduced counts of 10^2-10^4 CFU/mL when studied later in the clinic.[44] If left untreated, symptomatic women with counts of 10^2-10^4 CFU/mL had persistent symptoms and developed counts of $>10^5$ CFU/mL 2 days later. Catheterised patients with counts of $>10^3$ CFU/mL who were left untreated also developed counts of $>10^5$ CFU/mL within 2 days.[45] There is less contamination of the urine specimen in men and a predominant isolate with count of 10^3 CFU/mL is likely to reflect infected bladder urine (sensitivity 0.97, specificity 0.97).[46]

31.7.3 Dipstick Testing

Dipstick urinalysis is convenient and is routinely performed in clinical practice. It began in 1956 with the advent of Clinistix for glucose, followed by tests for other analytes including nitrite (1972) and white blood cells (1984).[47]

31.7.3.1 Leukocyte Esterase Test for Pyuria

Neutrophils contain esterases that are not found in the serum, urine, or urothelium. These esterases are released from lysed neutrophils in urine. A stain reacts with the leukocyte esterase and the result can be read within 2 minutes. Sensitivity is acceptable (72-97%) although false-negative results can be due to high levels of glucose, albumin, ascorbic acid, boric acid, gentamicin, cephalexin, doxycycline, nitrofurantoin, or urobilinogen that inhibit the reaction. False positives occur with clavulanic acid, imipenem, or vaginal contamination.

31.7.3.2 Nitrate Reductase (Griess) Test

In Gram-negative bacilli that cause UTI, this enzyme reduces nitrate to nitrite. Detection of nitrite indicates the presence of Gram-negative bacilli. Other important causes of UTI such as *Enterococcus*, *S. saprophyticus*, or *Candida* do not reduce nitrate and are not detected. The first morning voided urine is preferred because optimal results are obtained from urine that has been collected in the bladder for at least 4 hours. Results from incubating a culture of *E. coli* in urine showed an increasing proportion of positive nitrite dipstick test, from 1/20 cultures showing a positive result after 4 hours of incubation to 19/20 cultures showing a positive result after 6 hours.[48] Ascorbic acid and abnormal amounts of urobilinogen can cause false-negative results.

Testing for UTI in nursing home residents should include urine dipstick analysis for leukocyte esterase and nitrite. A urine culture should be obtained if either is positive. If both are negative, the combined result has 100% negative predictive value for UTI in nursing home residents.[49]

31.8 Management

31.8.1 Asymptomatic Bacteriuria

Antibiotic treatment of asymptomatic bacteriuria in pregnant women significantly reduces the incidence of pyelonephritis.[43] This led to the false claim in earlier medical literature that asymptomatic bacteriuria was associated with increased morbidity or mortality in other patient populations. Subsequent studies in schoolgirls, adult women, diabetic women, nursing home residents, spinal cord–injured patients, and catheterised patients consistently show that treatment of asymptomatic bacteriuria does not confer any benefit. Instead, these studies showed that treatment increases the risk of adverse drug effects and emergence of resistant organisms in urine. Asymptomatic bacteriuria in older persons also tends to develop and resolve spontaneously.[50] A prospective randomised clinical study of asymptomatic bacteriuria in a large cohort of 673 young women with recurrent UTI showed that antibiotic treatment was harmful. Asymptomatic bacteriuria helped protect against symptomatic recurrent UTI; 76% of the untreated group remained asymptomatic up to 1 year, compared with only 17% in the treated group.[51]

Apart from clinical trials, asymptomatic bacteriuria investigated with molecular methods shows a phenomenon called bacterial interference. Patients with an asymptomatic bacteriuria strain seem to be prevented from superinfection with other strains. This is due to microbial interaction in competing for nutrients and producing toxic molecules. Thus a microbial balance is established in the human microbiome that is considered to be a potent defence against superinfecting pathogenic bacteria.[52]

Based on these findings, routine urine cultures should not be taken when urinary tract symptoms are absent. An exception would be when invasive genitourinary procedures are planned in which case asymptomatic bacteriuria should be eradicated prior to surgery.

Pyuria is usually present in bacteriuria and does

not discriminate between symptomatic UTI or asymptomatic bacteriuria.[33-35] In the absence of symptoms, pyuria is not an indication for antibiotic treatment of bacteriuria.

31.8.2 Symptomatic UTI

Symptomatic UTI should be treated with antibiotics. General recommendations about duration of therapy and preferred regimens are listed in Tables 31.4[53] and 31.5.[54]

Microbiological cure rates of 70-80% are expected in ambulatory elderly women. Ambulant men with prostatitis have lower cure rates. Nursing home residents have recurrent infection in at least 50% of cases by 4-6 weeks post-treatment. Because of such a high bacteriuria recurrence rate, the goal of treatment is to treat symptoms, but not to sterilise the urine. Recurrence may be symptomatic or asymptomatic and post-treatment cultures are not necessary if the patient is asymptomatic.

31.8.3 Prophylaxis

A Cochrane review in 2004 recommended that healthy non-pregnant women who experience frequent recurrence of acute cystitis can be given low-dose prophylactic antibiotics at bedtime for at least 6 months.[55] The antibiotic can also be given as post-coital prophylaxis for UTI associated with sexual intercourse. The review included study populations of both pre- and post-menopausal women. It should be noted that this recommendation is not appropriate for women in nursing homes. Antibiotics in the review included nitrofurantoin 50-100 mg/day (monitor for pulmonary and hepatic toxicity), co-trimoxazole 80/400 mg per day, ciprofloxacin 250 mg/day, and norfloxacin 200 mg/day.

Cranberries are a traditional remedy for UTI. There have been numerous trials of cranberry products to treat UTI but no randomised controlled trials and no good quality evidence to suggest that it is effective for treatment of UTIs.[56] In a change from previous years' reviews, a Cochrane review in 2012 also found that cranberry juice cannot be recommended for the prevention of UTI.[57]

31.9 UTI Associated with Foley Catheter

31.9.1 Epidemiology

Dr EB Foley devised a self-retaining catheter to control post-prostatectomy haemorrhage in 1929. This has evolved into an essential instrument widely used in the management of urinary retention and incontinence. It is also frequently overused and is the most common cause of nosocomial infection in hospitals and nursing homes. Bacteriuria develops at the rate of approximately 5% per day of catheterisation. By 1 month (>28 days), the duration that defines long-term catheterisation, almost all catheterised patients will be bacteriuric.

Table 31.4 Duration of antibiotic therapy for urinary tract infection (UTI)[53]

	Women	Men
Short-course regimen for uncomplicated UTI	3-6 Days	Should be managed as complicated UTI
Cystitis	7 Days	7-14 Days
Pyelonephritis	14 Days	14 Days
Relapsing UTI due to prostatitis		Culture-directed antimicrobial therapy 6-12 weeks

Table 31.5 Antibiotic regimens for empirical treatment of acute urinary tract infection[54]

	Usual organism	Preferred regimen	Alternative	Special consideration / usual duration of treatment
Cystitis	*Escherichia coli*; *Staphylococcus saprophyticus*, Group B streptococcus	PO nitrofurantoin or amoxicillin-clavulanate	PO levofloxacin	Encourage high fluid intake. Increasing numbers of fluoroquinolone-resistant *Escherichia coli* in the community have already been found, thus restricting the empirical use of fluoroquinolones as suggested in IDSA and EAU guidelines
Acute pyelonephritis	*Enterobacteriaceae Enterococcus* (*Pseudomonas* in catheter-related, obstruction, transplantation)	IV amoxicillin-clavulanate	(Piperacillin-tazobactam if suspect *Pseudomonas aeruginosa*) or Imipenem or Meropenem	Blood culture and MSU cultures, need to exclude obstructive uropathy. IV until afebrile for 24-48 hours, then complete 14 days course with oral drugs Carbapenem is recommended for severe or rapidly deteriorating clinical cases

Abbreviations: EAU = European Association of Urology; IDSA = Infectious Diseases Society of America; IV = intravenous; MSU = midstream urine; PO = per oral

31.9.2 Microbiology

Long-term indwelling Foley catheters are usually associated with polymicrobial bacteriuria of two to five organisms, unlike short-term catheterisation that is usually associated with a single organism.

The commonest uropathogen is *E. coli*. Other commonly isolated uropathogens include *Pseudomonas aeruginosa*, *P. mirabilis*, *Klebsiella*, *Enterobacter*, *Providencia*, *Serratia marcescens*, *S. epidermidis*, and *Staphylococcus aureus*.[58]

There are two populations of bacteria in catheter-associated UTI (CA-UTI). One has a planktonic phenotype, grows in suspension in the urine, and is susceptible to antibiotics identified by in-vitro sensitivity tests. The other has a biofilm phenotype and grows within a protective biofilm on the catheter.[59] These bacteria survive urinary concentrations of antibiotics even if in-vitro tests show that they are antibiotic-sensitive. Urine specimens obtained from the infected catheter tend to have polymicrobial cultures and show higher counts because of these biofilm bacteria. To treat symptomatic UTI, any Foley catheter that has been placed longer than 7 days should be changed and a urine specimen obtained from the fresh catheter before antimicrobial therapy is initiated. This results in a shorter time to afebrile status and a lower rate of clinical relapse after treatment.

31.9.3 Clinical Considerations

Catheter-associated bacteriuria (CA-bacteriuria) is usually asymptomatic. Antibiotic treatment of asymptomatic CA-bacteriuria has no effect on the subsequent development of fever or blockages. Usually, the urine is rapidly re-infected after completing a course of antibiotic therapy. Hence CA-bacteriuria should not be treated. The presence of pyuria in CA-bacteriuria is not an indication for antimicrobial treatment.[35] The absence of pyuria in a symptomatic patient suggests a source of sepsis other than the catheterised urinary tract.[55]

Symptomatic CA-UTI is diagnosed in a patient with a catheter urine culture growth of at least 10^3 CFU/mL of at least one bacteria species in a patient with an indwelling urinary catheter or intermittent catheterisation.[58] CA-UTI is also diagnosed with the same criteria applied to voided urine if the patient had a urinary indwelling or condom catheter removed within 48 hours. There should be symptoms or signs compatible with UTI and no other identifiable source of infection. Patients usually present with fever. Other symptoms like dysuria and urgency may be due to discomfort from the catheter. Localised infections can occur in male patients and include urethritis, urethral abscess, epididymitis, and prostatitis. Many episodes of fever are short-lived and resolve without treatment. Nonetheless there may be possible progression to bacteraemia, sepsis and death, especially in high-risk patients.

Selection of antibiotics for symptomatic infections is based on previous urine culture results or presumed infecting organism. Antibiotic choice is similar to that for patients without a Foley catheter. Empirical antibiotics should be adjusted according to culture results. Short courses should be prescribed to prevent the emergence of antibiotic resistance. Generally, if the clinical response is satisfactory, only 5-7 days is recommended.

Encrustations are another complication of infection associated with a long-term indwelling Foley catheter. They are formed by crystals of magnesium and calcium phosphate in the biofilm matrix. *P. mirabilis* has a major role in the encrusting process. Encrustations can result in catheter blockage and severe UTI. Efforts to prevent encrustation, such as prophylactic antibiotics and urinary acidification, are not recommended at present. Drinking cranberry juice does not reduce encrustation.[60] Apart from encouraging a high fluid intake daily, patients need to be closely observed and the catheter changed if there are signs of encrustation or cystitis.

31.9.4 Prevention

The introduction of a closed drainage system, to block ascending infection up the Foley catheter, was a milestone in catheter care. The early practice of using open drainage systems resulted in bacteriuria affecting all catheterisations within a few days. The closed drainage system can preserve sterility for a longer period and reduces the risk of antibiotic resistant microorganisms. Nonetheless with the closed drainage system, measures like bladder irrigation, instillation of antibiotics into the catheter or collecting bags, and local application of antiseptics do not further reduce infection rates. The use of silver alloy–coated catheters does not decrease the frequency of CA-UTI. Nitrofurazone-coated catheters are associated with a small decrease in CA-UTI but are accompanied by increased rates of catheter removal and discomfort. Thus the current evidence does not support the routine use of antimicrobial coated catheters.[61]

Alternatives to catheterisation should be considered. These include the use of intermittent catheterisation or suprapubic catheterisation to relieve obstruction that is not amenable to surgery. Patients with uncontrolled incontinence can be treated with diapers or condom-type catheters.

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CHAPTER 32

Respiratory Tract Infections

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OBJECTIVES

- To understand the pathogenesis of pneumonia and the spectrum of respiratory tract infection in older people.
- To understand the clinical characteristics and aetiology of community-acquired pneumonia (CAP) and nursing home–acquired pneumonia (NHAP) in older people.
- To understand the risk factors for aspiration pneumonia in older people.
- To understand that pulmonary tuberculosis is still prevalent among older people in Hong Kong.
- To understand the evolving recommendations for empirical antibiotic therapy for NHAP.
- To appreciate the current influenza and pneumococcal vaccination programme for older people in Hong Kong.

KEY POINTS

- Pneumonia results from age-related changes in the respiratory, immune, and neurological systems.
- Older people are more likely to present with atypical features of pneumonia, such as a paucity of respiratory symptoms and absence of fever. Confusion and geriatric syndromes are also the common atypical presentations.
- *Streptococcus pneumoniae* is the most frequent bacterial pathogen in CAP and NHAP, although Gram-negative bacteria and *Staphylococcus aureus* are more common in the latter because of aspiration.
- Risk factors for aspiration in older people include neurological dysphagia and gastric dysmotility. Stroke is the most important cause of neurological dysphagia.
- Reactivation of latent infection is the major cause of pulmonary tuberculosis in older people in Hong Kong.
- It is controversial whether NHAP should be treated as community-acquired or nosocomial in origin.
- Aspiration pneumonitis is not caused by bacterial infection. Physicians may adopt a 'de-escalation' therapy or prescribe antibiotics of shorter duration.
- Physicians should be more cautious about the side-effects of anti-tuberculosis drugs in older people.
- Pneumonia can be prevented by influenza vaccination, good oral hygiene, and minimising the use of sedative medications.

32.1 Introduction

Respiratory tract infection is the most common infection among adults and lower respiratory tract infection is a leading cause of hospitalisation in older patients. Compared with younger adults, older people are more susceptible to pneumonia, and more likely to present with atypical features, such as absence of fever, refusal to feed, acute confusion, and functional decline. Upper respiratory traction infection (URTI), such as influenza virus and respiratory syncytial virus (RSV), also poses a threat to the health of older people, especially those resident in nursing homes. It may lead to secondary bacterial pneumonia, acute exacerbation of chronic pulmonary and cardiac disease, and death. In Hong Kong, pulmonary tuberculosis (TB) remains highly prevalent, in particular among the immunocompromised and older people. The atypical presentations in older people may result in delayed diagnosis. Thus, pulmonary TB should always be excluded in older patients with pneumonia. Due to comorbidity and disability, the risk of respiratory tract infection is much higher among older people resident in nursing homes than among those living in the community.

32.2 Pathogenesis of Pneumonia

Ageing is associated with physiological changes in the respiratory, immune, and neurological systems that increase the vulnerability of older people to

pneumonia. In the respiratory system, the lungs have lower elastic recoil, higher residual volume, and functional residual capacity. There is also a decrease in chest wall compliance, strength of respiratory muscles, and mucociliary clearance of respiratory cilia.[1,2] In the immune system, the innate and adaptive immunity of the respiratory tract decline with age.[3,4] In the neurological system, cough and gag reflexes diminish with age.[5] Oropharyngeal dysphagia is more common among older people, including increased delay in and decreased response of pharyngeal swallowing, decreased duration of cricopharyngeal opening, and decreased pharyngeal peristaltic amplitude and velocity.[6]

Aspiration of pharyngeal secretions occurs even among healthy adults during sleep.[7] Neurological dysphagia, disruption of the gastroesophageal junction, and anatomical abnormality of the upper digestive tract are risk factors for aspiration. The risk of aspiration is relatively high among older people because of the increased incidence of dysphagia or gastroesophageal reflux disease. Aspiration does not result in bacterial pneumonia unless normal pulmonary defence mechanisms are impaired or overwhelmed by a large volume of infected aspirated material.[8] Risk factors for aspiration pneumonia include functional dependency, tube feeding, poor oral health, comorbidity, medications, and smoking. Dysphagia is also considered a risk factor for aspiration pneumonia although it is generally not sufficient to cause pneumonia unless other risk factors are also present.[9]

The need for assistance in feeding, oral colonisation with *Staphylococcus aureus*, periodontal disease, and dental caries are the most important dental and oral risk factors for aspiration pneumonia among older people.[10] Several categories of drugs, including proton pump inhibitors, neuroleptics, and atypical antipsychotics increase the risk of aspiration pneumonia.[11-13]

32.3 Spectrum of Respiratory Tract Infections

32.3.1 Viral URTI

The clinical presentation and severity of viral URTIs vary considerably. The typical presentation is acute onset of systemic symptoms such as fever, chills, malaise, myalgia, and headache in the first 3 to 4 days. Rhinitis or pharyngitis may also occur. Respiratory symptoms, such as sore throat, dry cough, rhinorrhoea, and wheezing tend to occur later. The symptoms are usually self-limiting, and are expected to resolve in 5 to 7 days. Occasionally, viral or even secondary bacterial pneumonia may develop. It should be noted that an atypical presentation is very common in older patients, especially those living in nursing homes. Delirium, changes in functional status, and acute exacerbation of underlying pulmonary or cardiac disease are the most frequent atypical features.[14] Epidemics are typically reported in the late autumn and throughout the winter. In Hong Kong, influenza activity usually commences in January and peaks in February and March every year; and there may be a second peak in the summer. Transmission typically occurs after contact with the aerosolised respiratory secretions of an acutely infected person. An overcrowded living environment increases the risk of spreading infection among nursing home residents. If outbreaks of influenza-like illness are reported in nursing homes, appropriate infection control procedures should be implemented, such as isolation and antiviral therapy for the infected individuals, and prophylaxis for other residents with a contact history.[15]

The causative viral agents include influenza virus types A, B and C, and other viruses such as RSV, parainfluenza and adenovirus. Influenza virus type C is uncommon while type B causes less severe illness than type A. RSV is also recognised as a common cause of outbreaks of influenza-like illness in nursing homes during influenza seasons.[16] No clinical features can reliably differentiate infections caused by influenza A virus and RSV.[17]

Common respiratory viruses, such as influenza virus, RSV, and parainfluenza virus can be detected by viral culture or immunofluorescence assay of nasopharyngeal aspirate or swab. Positive results depend on viral quantity, greatest at the onset of illness.[15]

32.3.2 Community-acquired Pneumonia and Nursing Home–acquired Pneumonia

32.3.2.1 Epidemiology and Pathogenesis

Pneumonia is approximately 10-fold more frequent in older adults compared with younger adults. Its incidence is higher among older people resident in a nursing home than in those living in the community. Community-acquired pneumonia (CAP) and nursing home–acquired pneumonia (NHAP) represent the leading cause of death in older patients aged ≥80 years.[18] The mortality rate has been reported to be 13-40%, depending on the severity of pneumonia.[19]

Aspiration plays an important role in CAP and NHAP among older people. An early study showed that silent aspiration was more common in older patients hospitalised with pneumonia compared with healthy control subjects (71% vs. 10%, P<0.02).[20] Use of feeding tubes, such as a nasogastric tube, gastrostomy, or jejunostomy does not reduce the risk

of aspiration.[21] On the contrary, feeding tubes are linked to a higher risk of pneumonia and pneumonia-related mortality.

Another important pathogenic mechanism of pneumonia in older people is upper airway colonisation by pathogenic bacteria. Potential respiratory pathogens can be identified from the dental plaque of over 60% of dependent older people.[22] Gram-negative bacteria (GNB) have been found to colonise 60-73% of critically ill older patients in an acute medical ward and 22-37% of institutionalised older people.[10,23] Table 32.1 summarises the risk factors for bacterial colonisation of the upper respiratory tract.

32.3.2.2 Clinical Presentation

Atypical presentations of pneumonia are more common in older patients compared with their younger counterparts. The classic triad of cough, dyspnoea, and fever has been shown to be present in only 56% of elderly people and absent in 10% of older patients hospitalised with CAP.[24] Confusion is a common 'atypical presentation'. A local study showed that confusion was more common in NHAP than CAP (42% vs. 13%, P<0.001), in particular in those with a history of cerebrovascular accident and dementia.[15] Other atypical features included geriatric syndromes (falls, incontinence, and anorexia) and acute exacerbation of chronic diseases (chronic pulmonary disease, congestive heart failure, and diabetes).

32.3.2.3 Aetiology

Streptococcus pneumoniae is the most frequent bacterial pathogen of CAP and NHAP, followed by *Haemophilus influenzae*, *Enterobaceriaceae*, and *S. aureus*. Both colonisation by and infection with GNB is a function of the number and severity of concomitant illnesses, and functional status. The risk of GNB-related pneumonia increases among nursing home residents and in patients with functional impairment. *Pseudomonas aeruginosa* is more common in patients with bronchiectasis.[25-28] *S. aureus* is more commonly isolated in NHAP than CAP. In the US, methicillin-resistant *S. aureus* (MRSA) is an increasingly recognised pathogen in the nursing home population. Concomitant infection with two or more pathogens is common in older patients with pneumonia.[29]

Table 32.1 Risk factors for bacterial colonisation of the upper respiratory tract

- Antibiotic therapy
- Endotracheal intubation
- Smoking
- Impaired salivation, such as anti-parkinsonism, antidepressants, and diuretics
- Periodontal diseases and dental plaques
- Malnutrition
- Severe medical illnesses and surgery

Atypical pathogens and *Legionella pneumophila* display strong geographic variations. In Hong Kong, atypical pathogens have been isolated in 10-13% of older patients hospitalised with CAP or NHAP. The most common isolates were *Chlamydophila pneumoniae* and *Mycoplasma pneumoniae*.[30,31] In clinical practice, the incidence of *Legionella* infection may be underestimated because of the non-specific presentations (constitutional symptoms, myalgia, diarrhoea, and paucity of pulmonary signs) and the relatively low sensitivity of diagnostic tests. Advancing age, male gender, cigarette smoking, chronic lung disease, and immunosuppression are risk factors for *Legionella* infection.[32] Contrary to the traditional belief that *L. pneumophila* was the most common cause of 'atypical pneumonia' in older people,[33] local research suggests that it was rare (<1%).[30,31]

Respiratory viruses have been detected in about 10% of older patients hospitalised with CAP or NHAP.[15] Influenza A virus and RSV are the most frequent isolates. Human metapneumovirus (MPV) was initially found to be the second most common cause of viral URTI among children. Using reverse-transcriptase polymerase chain reaction (RT-PCR) on nasopharyngeal specimens, MPV has been shown to be an important cause of outbreaks of respiratory tract infections in nursing homes. The attack rate of respiratory diseases was 27-36%. Pneumonia was present in 17-25% of symptomatic cases. The mortality rate was very high, reaching 33-50% of laboratory-confirmed cases.[34,35] Our local data suggest that RSV and MPV are the most frequent viral pathogens of NHAP.[31]

32.3.3 Aspiration Pneumonia and Aspiration Pneumonitis

Aspiration is the inhalation of oropharyngeal or gastric contents into the larynx or lower respiratory tract, resulting in aspiration pneumonitis or pneumonia. Aspiration pneumonitis is the chemical injury to the lung parenchyma that is induced by the sterile gastric contents while aspiration pneumonia is an infectious process caused by oropharyngeal secretions that are colonised by pathogenic bacteria. Aspiration pneumonia usually occurs among older people with neurological dysphagia or gastric dysmotility.[36] An early study suggested that aspiration pneumonia was more common in NHAP than CAP (18% vs. 5%).[37] There are no reliable clinical features to distinguish between aspiration pneumonia and aspiration pneumonitis. A local study further

indicated that no bacterial or viral pathogen was identified in 32% of NHAP cases despite extensive microbiological investigations, including the use of RT-PCR on nasopharyngeal specimens. Aspiration pneumonitis presumably accounted for some of these cases. The in-hospital mortality rate was much lower in patients with indeterminate aetiology than in those with established aetiology, although statistical significance was not reached (6% vs. 19%, P=0.077).[31]

32.3.4 Pulmonary Tuberculosis

In Hong Kong, TB remains prevalent although the notification rate (per 100 000 person-years) decreased from 101-114 in 1991-2000 to 66-109 in 2001-2012.[38] Failure to eradicate TB is attributed to the phenomenon 'ageing of the epidemic'. A fall in the proportion of active cases from progressive primary infection and exogenous re-infection is superseded by endogenous reactivation in older people that constitutes a significant proportion of the notified cases. This can be illustrated by the age-related changes in the notification rate (per 100 000 person-years) that increased from 45 among people aged 20-24 years to 322 among those aged ≥85 years in 2012.[38]

TB is more common among older people than younger adults because of socioeconomic deprivation, immunosenescence, and comorbidity (diabetes mellitus and malignancy).[39] Elders, who acquired TB with or without treatment many years ago, represent a huge potential source of latent TB. Thus treatment of latent TB is an emerging area for exploration.

Table 32.2 illustrates the comparison of clinical features of TB in older and younger adults in Hong Kong.[40] Older patients are more likely to present late with non-specific features, resulting in a delay in diagnosis and commencement of anti-TB therapy.

32.4 Treatment

32.4.1 Viral URTI

Treatment of viral infections aims to prevent complications such as dehydration, decline in functional status, and secondary bacterial pneumonia. Supportive measures with antipyretics and analgesics should be given. Patients should be advised to stop using sedatives, antihistamines, or common ingredients of cough mixtures. Antibiotics are not indicated unless bacterial infection is suspected, e.g. purulent sputum or abnormal chest radiographs.

With the exception of four anti-influenza agents, including adamantanes (amantadine and rimantadine) and neuraminidase inhibitors (oseltamivir and zanamivir), there are no other licensed antiviral agents. Amantadine and rimantadine have activity against influenza A only while oseltamivir and zanamivir have activity against both influenza A and B. They are effective for prophylaxis, and also reduce disease severity, duration of symptoms, and viral shedding when given within 48 hours of symptom onset in uncomplicated influenza. Adverse effects on the central nervous system limit the use of amantadine in older people. Drug resistance develops rapidly in 30-80% of cases after use of amantadine and rimantadine for a few days. Fewer side-effects are reported with oseltamivir and zanamivir. The former may cause gastrointestinal upset, such as nausea and vomiting, but this usually resolves with discontinuation of drugs. The latter should be avoided in patients with chronic pulmonary disease because of bronchospasm. Influenza viruses resistant to neuraminidase inhibitors are very rare, i.e. 0.4-1%. Adamantanes are therefore not recommended as first-line antiviral therapy.[41,42] Table 32.3 shows the antiviral prophylaxis and treatment of influenza in

Table 32.2 Comparison of clinical features of tuberculosis in older and younger patients[40]*

	Older patients (age ≥65 years)	Younger patients (age <65 years)
Pulmonary involvement alone	81.8%	76.0%
Non-specific symptoms		
Dyspnoea	24.3%	9.0%
Weight loss	23.4%	17.9%
Malaise	16.6%	5.1%
Haemoptysis	17.5%	21.3%
Lymphadenopathy	3.9%	13.6%
Late presentation (>4 weeks)	37.4%	29.3%
Comorbidity	65.2%	28.3%
Chest radiograph		
Moderate or extensive disease	63.9%	37.1%
Lower zone involvement	15.6%	8.1%
Investigations		
Positive tuberculin test	61.9%	80.3%
Positive sputum smear	38.9%	30.5%
Positive sputum culture	66.1%	56.2%

* The differences in all variables, except haemoptysis, reached statistical significance with P<0.05

Table 32.3 Antiviral prophylaxis and treatment of influenza

Drug	Prophylaxis / treatment
Oseltamivir (Tamiflu)	Treatment: 75 mg bd for 5 days
	Prophylaxis: 75 mg daily for 1 week in patients living in the community; or at least 2 weeks in case of ILI outbreaks in nursing homes
	Reduction of dosage in patients with CrCl <30 ml/min
Zanamivir (Relenza)	Treatment: 10 mg bd for 5 days

Abbreviations: DBP = diastolic blood pressure; SBP = systolic blood pressure

Table 32.4 CURB score[43] and CURB-65 score[44] *

Confusion
Urea level >7 mmol/L
Respiratory rate ≥30/min
Blood pressure (SBP <90 mm Hg or DBP ≤60 mm Hg)
Age ≥65 years

Abbreviations: DBP = diastolic blood pressure; SBP = systolic blood pressure

* Scoring: 1 point given for each feature present (range 0-4 points for CURB; 0-5 points for CURB-65)

older patients living in the community and nursing homes. Oseltamivir is the most frequently used antiviral agent for prophylaxis and treatment of influenza.

32.4.2 Community-acquired Pneumonia and Nursing Home–acquired Pneumonia

CAP and NHAP can be managed in both inpatient and outpatient settings. There are several factors that guide physicians when deciding the site of care: pneumonia severity, availability of medical support in the community or nursing home, and social factors.

Pneumonia severity can be predicted by the CURB score[43] or CURB-65 score[44] (Table 32.4). A CRUB cut-off score of ≥2 identifies patients with severe pneumonia. The CURB-65 score[44] stratifies patients into three levels of severity: low risk for score 0-1, intermediate risk for score 2, and high risk for score ≥3 (corresponding to CURB score of ≥2).

Antibiotics are almost always administered empirically in patients with CAP or NHAP, because aetiology can rarely be defined at presentation. The guidelines, which are published by the British Thoracic Society (BTS) and co-published by the American Thoracic Society (ATS) / Infectious Diseases Society of America (IDSA), are most commonly adopted. *S. pneumoniae* is the most common causative bacteria. The BTS (2009) and ATS / IDSA (2007) guidelines recommend a beta-lactam / beta-lactamase inhibitor, a cephalosporin or an anti-pneumococcal fluoroquinolone as a first-line antibiotic in adult patients with CAP.[45,46] Local IMPACT guidelines, however, do not support first-line use of fluoroquinolones because resistance is emerging rapidly in *S. pneumoniae* (levofloxacin MIC ≥8 μg/ml). The excessive use of fluoroquinolones may also delay the diagnosis of pulmonary TB and induce antibiotic resistance in *Mycobacterium tuberculosis*.[47,48] The ATS / IDSA guidelines recommend use of a macrolide together with a beta-lactam in all CAP patients for atypical coverage.[46] On the contrary, BTS guidelines[45] recommend that such a combination of antibiotic therapy be reserved for patients with moderate-to-high severe CAP as indicated by their CURB score of ≥2. Doxycycline may replace macrolide in some regions, including Hong Kong, where the prevalence of macrolide-resistant *M. pneumoniae* is high.[49] Anti-pseudomonal penicillin or cephalosporin should be considered in patients with structural lung disease, such as bronchiectasis.[46]

The BTS (2009) guidelines recommend that CAP and NHAP share similar empirical antibiotic therapy, although the incidence of GNB and *S. aureus* is higher in NHAP because of aspiration pneumonia.[45] In contrast, the ATS / IDSA (2005) guidelines re-categorise NHAP as healthcare-associated pneumonia (HCAP) that should be treated empirically with antibiotics that cover nosocomial pathogens, such as multidrug-resistant *P. aeruginosa* and MRSA.[50] It should be noted that the concept of HCAP is based on the findings of several studies in the US, and may not be totally applicable in our locality.[30] Debate continues about whether NHAP should be treated as CAP or HCAP.[51,52] In our IMPACT guidelines, NHAP is not a separate entity to CAP.[48] Our local data support that NHAP should be treated empirically as CAP. Thus, nursing home residence should not be the only indication for empirical broad-spectrum potent antibiotics. Anti-pseudomonal antibiotics with or without vancomycin may be considered in patients who have been recently hospitalised or have severe pneumonia (CURB score of ≥2).[30]

32.4.3 Aspiration Pneumonia and Aspiration Pneumonitis

Empirical antibiotic therapy for aspiration pneumonia is no different for CAP or NHAP. Aspiration pneumonitis usually resolves with supportive measures and antibiotic therapy is not indicated. At presentation, however, it can be hard to differentiate between aspiration pneumonia and pneumonitis. It is reasonable to prescribe empirical antibiotics in older patients with aspiration pneumonia or pneumonitis.

It is advisable to adopt a 'de-escalation' approach (change from a broad-spectrum, potent antibiotic to a narrow-spectrum, less potent one) or refrain from prescribing prolonged antibiotics in older patients with neurological dysphagia or gastroesophageal reflux, and history of an aspiration event.[31]

Prevention is always better than cure. Aspiration can be prevented by assisted hand feeding, dietary modification, good oral and dental hygiene of older patients, and elevation of the head of the bed during feeding. Physicians should minimise the use of sedatives, especially psychotropic medications, to reduce the risk of aspiration. It is important to be aware that tube feeding does not reduce the risk of aspiration.[36]

32.4.4 Pulmonary Tuberculosis

A higher rate of adverse reactions to treatment (49% vs. 34%, P<0.05) and higher mortality (16% vs. 1%, P<0.05) have been observed in older patients with TB infection, compared with younger adults.[39] Chemoprophylaxis for latent TB based on the tuberculin skin test is recommended by the ATS to prevent reactivation of latent TB,[53] although this approach may not be appropriate in Hong Kong, especially among older people, because of the lack of a diagnostic tool, a huge infected pool, difficult interpretation of tuberculin test, longer duration of treatment, and potential serious treatment complications such as hepatotoxicity.

Clinicians must pay extra attention to tolerability of the standard four-drug regimen (rifampicin, isoniazid, pyrazinamide and ethambutol) when treating older patients, particularly in the initial stages. Failure to thrive is a common non-specific adverse event observed in older patients and secondary to anti-TB drugs. Our personal experience suggests that one may consider starting with two drugs, then step up the regimen to three or four drugs when patient tolerance has developed. Dosage adjustment may be warranted according to age-related pharmacokinetics and comorbidity. Pyridoxine supplementation should be considered for those with poor nutritional intake or at increased risk of neuropathy. When the susceptibility pattern of cultured bacilli is shown to be favourable, the combined use of rifampicin and isoniazid may suffice because of a lower bacterial load.

In Hong Kong, the Department of Health adopts the direct observation therapy for TB to enhance drug compliance. Patients have to attend the chest clinic every day. This arrangement may not be practical for those who are functionally dependent so a common practice is to prescribe anti-TB drugs for a longer period of time, e.g. 1-2 months, with drug compliance monitored by community nurses.

32.5 Prevention

32.5.1 Vaccination

Influenza and pneumococcal vaccines effectively reduce respiratory tract infections and their related complications. Although vaccine efficacy is generally lower in older people because of immunosenescence, it is this population who is most likely to benefit from vaccination. However, immune response to vaccination may be blunted by malnutrition[54,55] and psychosocial stress[56,57] commonly found in frail elderly patients and their carers. Thus, attention should also be paid to these factors that modulate immune response.

The World Health Organization recommends annual influenza vaccination. The current vaccines are trivalent and contain two type A and one type B viruses.[58] It is made from highly purified, egg-grown viruses that have been inactivated and is contraindicated in people with a history of anaphylactic hypersensitivity to eggs. In Hong Kong, it should be administered around November each year to ensure adequate immune protection for the coming epidemics. The Centre for Health Protection recommends that vaccination be given to high-risk people, including those aged ≥65 years, nursing home residents, institutionalised psychiatric and mentally handicapped patients, and adult patients with chronic illnesses. With the effect of herd immunity, all healthcare workers involved in direct elder care are also encouraged to receive the vaccine every year in order to prevent influenza outbreaks in nursing homes or healthcare facilities.[59] The effectiveness of influenza vaccination in community-dwelling older people is illustrated in Table 32.5.[60] A systematic review concluded that influenza vaccination could reduce pneumonia and death related to pneumonia or influenza in institutionalised older people.[61] A local study further indicated that trivalent seasonal influenza vaccine reduced all-cause and pneumonia-

Table 32.5 Effectiveness of influenza vaccination in older people[60]

Patient group (age ≥65 years)	Relative risk reduction
Hospitalisation for acute and chronic respiratory illnesses	32%
Hospitalisation for pneumonia and influenza	39%
Hospitalisation for congestive heart failure	27%
All-cause mortality	50%

related mortality by 28% and 20% respectively, in institutionalised older people.[62]

Polyvalent pneumococcal polysaccharide vaccines (PPV) have been in clinical use for more than 30 years. The 23-valent pneumococcal vaccine (PPV-23) is the most commonly used PPV among adults in industrial and developing countries. Like other polysaccharide vaccines, PPV-23 is not believed to confer long-lasting protection. One single revaccination is suggested 5 years after the first dose. In Hong Kong, pneumococcal vaccine PPV-23 has been included in the Government Vaccination Programme for older people aged ≥65 years since 2009. One-time revaccination is recommended 5 years after the first vaccination among elders aged ≥65 years for those with additional high-risk conditions (history of invasive pneumococcal disease, immunocompromised states, chronic disease, with cochlear implants). Revaccination with three or more doses is not recommended because of an uncertain safety profile.[63] From October 2017, the Hong Kong Government provides free or subsidised 13-valent pneumococcal vaccine (PCV13) to eligible high-risk elderly persons to strengthen their immunity against pneumococcal infection.[64] In this newly launched pneumococcal vaccination programme, one dose of PCV13 will be provided to eligible elderly persons with high-risk conditions, followed by one dose of PPV-23 a year later. If an eligible elderly person has already received PPV-23, one mop-up dose of PCV13 will be given a year later. For those without high-risk conditions, PPV-23 will continue to be provided.

Compared with influenza vaccines, the efficacy of pneumococcal vaccines is less certain. In older people, vaccination has been shown to effectively reduce the risk of invasive pneumococcal disease but not non-bacteraemic pneumococcal pneumonia and other pneumonia.[65]

32.5.2 Other Measures

Apart from vaccination, there are several simple measures that can prevent aspiration pneumonia in older people, as mentioned in Chapter 32.4.3.

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CHAPTER 33

Geriatric Musculoskeletal Disorders

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OBJECTIVE

To learn the special presentations and impact of musculoskeletal disorders in old age, the need for holistic assessment in guiding therapeutic options, and the appropriate use of drugs (non-steroidal anti-inflammatory drugs [NSAIDs], steroids, colchicine, allopurinol) to reduce iatrogenesis.

KEY POINTS

- Elders with musculoskeletal problems may present with pain; fever; falls; fractures; neurological complications; complications of immobility or drug treatment; functional decline; or psychosocial problems.
- Iatrogenic events can masquerade as rheumatic disorders, e.g. steroid-induced osteoporosis, diuretic-induced gout, statin-associated myopathy.
- Elders are most prone to the adverse effects of NSAIDs that include an increased risk of acute kidney injury when used in combination with renin-angiotensin system inhibitors or diuretics.
- Osteoporosis and fractures are common in old age and have a major impact on elderly people, with physical and psychosocial consequences. Anti-osteoporosis treatment should be individualised, balancing the risks and benefits of bisphosphonates with consideration of a drug holiday for patients at lower risk of fracture.
- Up to 55% of men and 30% of women with symptomatic vertebral crush fractures have underlying secondary osteoporosis, treatment of which may lead to a significant increase in bone density and symptomatic improvement.
- Osteoarthritis is not a single degenerative joint disease related to ageing and 'wear and tear' of the joint. It is a synovial joint syndrome of overlapping osteoarthritic disorders with multiple pathogenic contributors resulting in an imbalance between degradation caused by mechanical stress and the process of repair.
- Subacute polyarticular gout with early tophi formation over the fingers, often associated with diuretic use, is the pattern commonly seen in elderly onset gout.
- Because of the increased toxicity of NSAIDs and colchicine in old age, corticosteroids are preferred when treating an acute attack of gout in elderly patients with multiple comorbidities.
- To minimise the risk of allopurinol hypersensitivity syndrome, and to reduce the chance of a mobilisation attack of gout, a 'start low, go slow' approach to dosing should be adopted.
- Elderly onset rheumatoid arthritis patients are characterised clinically by disabling morning stiffness and marked pain that affects the large proximal joints.
- Paget's disease is a potentially treatable disease but can cause significant morbidity in elderly patients with complications such as fracture, bone pain, secondary osteoarthritis, deafness, dental problems, and osteosarcoma.
- Although giant cell arteritis (GCA) / polymyalgia rheumatica (PMR) is rare among Chinese, it is a treatable cause of distressing pain and functional decline in elderly people. If an elderly patient does not improve following treatment with the appropriate steroid regimen for GCA / PMR, alternative diagnoses (e.g. tuberculosis, lymphoma) should be seriously considered even when a positive temporal artery biopsy has been obtained.

33.1 Special Considerations in Geriatric Musculoskeletal Disorders

Musculoskeletal problems in old age are common and their presentation can include pain (backache, loin pain, chest pain, arthritis, myalgia), fever (inflammation, infections), falls and fractures, neurological complications (cervical myelopathy, paraplegia, cauda equina syndrome), complications

of immobility (constipation, incontinence, pressure sore), functional decline, and consequent psychosocial problems (depression, anxiety). Despite the common occurrence of pain in musculoskeletal disorders, there are barriers to the detection of pain in an elderly person. These include individual social, emotional, cognitive, and subjective factors.[1] A non-report of pain does not mean an absence of pain. The presence of pain in an elder may be undetected or misunderstood because of non-specific signs and symptoms (Table 33.1),[1] and should lead to a search for evidence of pain of musculoskeletal origin: pain upon motion, stiffness, tender joints, or limited range of motion.

Diagnosis of musculoskeletal disorders can be challenging in old age (Table 33.2).[2-11] The combination of ageing, multiple pathologies, and polypharmacy mean elderly patients are particularly susceptible to drug-related adverse events (see Chapter 15). In an ageing population, it is not unusual for such iatrogenic events to masquerade as rheumatic disorders. Although steroid-induced osteoporosis is well known, drugs that may induce gout, lupus, vasculitis, arthralgia, or myopathy are often overlooked.[2] Recognition of iatrogenic rheumatic syndromes in old age should prompt a thorough re-evaluation of medications, avoidance of over-investigations, appropriate treatment via drug reduction or cessation, and prevention of a prescribing cascade (see Chapter 15).

Table 33.1 Non-specific signs and symptoms of the presence of pain in elderly people[1]

- Frowning, moaning, grimacing, fearful facial expressions, grinding of teeth
- Sighing, groaning, fearfulness, breathing heavily, withdrawn
- Bracing, guarding, rubbing
- Fidgeting, increasing or recurring restlessness
- Striking out, increasing or recurring agitation
- Eating or sleeping poorly
- Mental status change
- Decreasing activity levels
- Depressed affect of sudden onset
- Resist certain movements during care, rigidity
- Change in gait or behaviour
- Paucity of interaction, speech
- Loss of function — lack of adaptation skills

Musculoskeletal pain is one of the most common reasons elderly patients seek medical care. Unrelieved pain inhibits respiration, decreases mobility, and decreases functional status, and may precipitate iatrogenic events such as pneumonia, constipation, and deep vein thrombosis. Prolonged hospital and institutional stays may decrease an elderly patient's quality of life and initiate social isolation. The pain and distress of rheumatological / musculoskeletal disorders that are inaccurately diagnosed and improperly treated may result in doctor shopping and self-medication with over-the-counter remedies, some of which have turned out to be potent steroids (that the patient may be taking unknowingly) with disastrous consequences of hypertension, hyperglycaemia, osteoporosis, and sepsis (Plates 33.1, 33.2).

An initial approach to musculoskeletal pain

Table 33.2 Diagnostic challenges in geriatric musculoskeletal disorders[2-11]

Diagnostic challenge	Example
Iatrogenic rheumatic syndromes[2]	Steroid-induced osteoporosis, diuretic-induced gout, statin-associated myopathy, drug-induced arthralgia, drug-induced lupus, vasculitis, scleroderma
Age-related skeletal changes common	Underdiagnosis of secondary causes of osteoporosis[3-5] (attributed to senile osteoporosis) Attributing problem to degeneration / osteoarthritis[5,6]
Symptomatic treatment instead of exact diagnosis	NSAID-masked gout[7]
Multiple pathologies / aetiologies	Coexistence of other non-musculoskeletal pathologies that can cause pain, e.g. ischaemic heart disease
Over-reliance on diagnostic imaging	Inferring malignancy from radiological studies (see Plate 13.1)
Under-use of biopsy / histological confirmation	Inferring infection (instead of inflammatory musculoskeletal conditions) from fever and systemic symptoms
Presenting problem remote from diseased organ / system	Geriatric presentations (falls and fractures) from focal disease[8]
Ready availability of 'screening' biochemical test battery	Interpretation of significance of high alkaline phosphatase, hypercalcaemia
Altered pattern in old age	Polyarticular gout,[9] elderly-onset rheumatoid arthritis,[10] elderly-onset systemic lupus erythematosus[11]
Symptoms (e.g. deafness) attributed to old age	Paget's disease of bone
Symptoms / signs attributed to non-rheumatic disorders	Acute gouty arthritis presenting as fever misdiagnosed and treated as sepsis Acute gouty arthritis presenting as limb weakness misdiagnosed as stroke

Abbreviation: NSAID = non-steroidal anti-inflammatory drug

management in an elderly patient begins with acceptance of pain complaints as genuine, addressing both physiological and psychosocial components, and focusing upon the underlying aetiologies that may be numerous. Aims of treatment are to treat the underlying disorder(s), relieve pain, enhance functional status, and augment activities of daily living.[1] Management of musculoskeletal disorders in old age is challenging because drug treatment may be associated with adverse reactions, operative interventions may be associated with complications, and functional decline and disabilities need to be addressed.

Non-steroidal anti-inflammatory drugs (NSAIDs) are the most frequent medication used in old age, whether prescribed or self-administered over-the-counter. Elderly patients are nonetheless most prone to their adverse effects (Table 33.3) if they are not used appropriately and cautiously.[12] In particular, older patients are more likely to have cardiovascular disease and an age-related decline in renal function, increasing the risks of cardiovascular, haematological, and renal adverse events. Multiple drug interactions involving NSAIDs are particularly relevant for elderly patients who are the victims of polypharmacy. NSAIDs are associated with an increased risk of acute kidney injury when used in both triple and dual combinations with either renin-angiotensin system inhibitors or diuretics.[13]

Given these risks, recent guidelines for the management of chronic pain in elderly patients recommend using NSAIDs rarely and only in carefully selected patients.[12] The lowest effective dose should be used for the shortest period of time necessary. The currently available NSAIDs fall into three categories: non-selective NSAIDs that act via inhibition of cyclooxygenase (COX)-1 and COX-2; selective inhibitors of COX-2 (e.g. celecoxib); and topical NSAIDs that inhibit both COX-1 and COX-2 with less systemic NSAID exposure but similar efficacy to oral NSAIDs. Topical agents are an option only in patients with localised pain in superficial joints. Safe pain management in older patients therefore requires cautious choice of selective and non-selective oral NSAIDs, topical NSAIDs or non-NSAID analgesics.[12]

Table 33.3 Adverse effects of NSAIDs

- Gastrointestinal irritation / bleeding
- Fluid retention
  - o precipitate heart failure
  - o uncontrolled hypertension / interfere with antihypertensive therapy
- Renal impairment (acute / chronic)
- Liver impairment
- Central nervous system side-effects (headache, drowsiness, confusion [particularly indomethacin])
- Drug interactions (e.g. increased risk of gastrointestinal bleeding with anticoagulants)
- Toxic epidermal necrolysis
- Activation of tuberculosis
- Increased risk of cardiovascular events (myocardial infarction, stroke) in cyclooxygenase-2 selective NSAIDs

Abbreviation: NSAIDs = non-steroidal anti-inflammatory drugs

The toxicity profile of different NSAIDs varies considerably, with indomethacin showing the greatest toxicity (and thus should be avoided in elders), and ibuprofen the least. As the geriatric patient population has often been excluded from pre-marketing studies of drugs, post-marketing surveillance is important to provide information on the risk-benefit ratio of newly released drugs in elderly patients. Such surveillance has led to the withdrawal of, for example, benoxaprofen[14] in the 1980s because of hepatic toxicity; and the COX-2 selective NSAID rofecoxib[15] in 2004 because of increased cardiovascular events (myocardial infarction, stroke). Caution is therefore needed in the prescription of new drugs that might give rise to unknown adverse effects in elderly patients. Jüni and Dieppe[16] have argued that the heavily marketed view that the COX-2 selective NSAIDs are safer than conventional NSAIDs is flawed. In a local study that compared patients prescribed celecoxib with those prescribed diclofenac plus omeprazole[17] and mean age of 66.5 ± 14.2 and 68.8 ± 13.2 respectively, neither regimen provided a good nor even acceptable level of protection from recurrent ulcer bleeding. There was also additional concern about renal toxicity: among patients with renal impairment at baseline, 51.4% of those prescribed celecoxib and 40.7% of those prescribed diclofenac plus omeprazole experienced renal adverse events. Thus, drug treatment for musculoskeletal conditions in old age should be individually designed and based on renal and liver function profile, as well as the presence of cardiovascular risk factors and gastrointestinal disorders. Constant alertness for drug-drug and drug-disease interactions is important.

Elderly patients with musculoskeletal disorders are often under the care of orthopaedic surgeons, and close orthogeriatric collaboration is necessary to improve the quality and efficiency of care of frail elderly patients with multiple, complex medical problems. Elders who present with fractures after a fall may have undiagnosed underlying medical disorders that impair their stability, e.g. hypo- or hyper-thyroidism, pneumonia, of which the latter may become apparent only after surgery as 'postoperative' pneumonia (Plate 29.2).[8] Too much focus on neuromusculoskeletal problems may result in underlying systemic diseases being missed, e.g.

infective endocarditis,[6,18] miliary tuberculosis,[19] and giant cell arteritis (GCA). Careful preoperative diagnosis and management of medical problems are thus required. Elders are also prone to perioperative complications such as postural hypotension after spinal anaesthesia,[20,21] postoperative cognitive deficit[22,23] (Case 33.1), dehydration, fluid overload, hyponatraemia,[24] sepsis, and confusion. Early mobilisation and rehabilitation are essential to restore the preoperative level of function. All these will require close collaboration of the geriatrician and orthopaedic surgeon, as well as a multidisciplinary team. The clinical effectiveness of orthogeriatric care, pioneered in Hastings in the 1960s and 1970s, was proven in the 1990s, with reduced mortality and improved functional status. Evidence-based guidelines on hip fracture care have been developed and published (see Chapter 12).

Case 33.1 Postoperative cognitive deficit in a 68-year-old woman

A 68-year-old woman, with a 7-year history of hypertension and right nephrectomy for renal stone at age 62, underwent a left total knee replacement because of distressing knee pain (secondary to pseudogout) that was refractory to medications. She was assessed for confusion 2 weeks postoperatively. Prior to surgery, she was mentally clear and alert, and was independent in instrumental activities of daily living and household activities. Record review showed intra-operative drop in blood pressure during spinal anaesthesia; transfusion of two units of blood because of a reduced haemoglobin level of 8.1 g/dL on day 3; and administration of a stat dose of Adalat (nifedipine) 5 mg sublingually for an episode of high blood pressure on day 4. An urgent computed tomographic brain revealed periventricular infarcts (Plate 33.1). This case illustrates the effects of the combined insults of hypotension from spinal anaesthesia,[17,18] blood loss, and antihypertensives, as well as anaemia leading to ischaemic injury to the watershed area within her brain, itself made vulnerable by pre-existing hypertension. Her cognitive impairment persisted and she developed vascular dementia (Binswanger's disease).[25]

33.2 Osteoporosis

33.2.1 Definition and Classification of Osteoporosis, and Fracture Risk Assessment

Osteoporosis has been defined as a disease of increased skeletal fragility accompanied by low bone mineral density (BMD) and microarchitectural deterioration, with a consequent susceptibility to fracture.[26] By using population standards, BMD measurements are expressed as standard deviation units above or below the mean value for normal young adults (T-score) or relative to the mean value for control subjects of the same age (Z-score). In 1994, the World Health Organization (WHO) established BMD measurement criteria based on T-score allowing the diagnosis of osteoporosis before fracture occurrence (Table 33.4).[27] The Z-score is not used to define osteoporosis, but a low Z-score helps to identify individuals with BMD lower than expected for their age and, in the context of suspicious clinical data, should prompt a search for secondary causes of osteoporosis.[28]

Although the WHO study group has recommended a reference database of femoral neck BMD measurements in white women aged 20-29 years for application worldwide, studies demonstrate that the application of this recommended Caucasian reference might lead to a distorted T-score and misclassification of osteoporosis among other ethnicities. The Osteoporosis Society of Hong Kong recommends an Asian normative database for diagnosing osteoporosis in Asian subjects.[29] A nationwide BMD reference database for the lumbar spine and femoral neck in Chinese adults has been established by pooling the published BMD data from 91 studies including 51 906 males and 88 006 females (≥20 years) in 38 cities in China.[30]

Although the WHO has defined osteoporosis as a BMD T-score of < –2.5, this does not necessarily represent a threshold for treatment. Study has revealed that 82% of postmenopausal women with fractures had a T-score better than –2.5.[31] Osteoporosis defined by BMD (T-score ≤ –2.5) has low sensitivity despite high specificity. Treatment threshold differs from diagnostic thresholds (as defined by BMD T-score) because for the same T-score, fracture risk varies with age and the presence of clinical risk factors. Treatment threshold is also determined by the cost, risk, and benefits of treatment. The Sheffield WHO Collaborating Center team (http://www.shef.ac.uk/FRAX/) has developed a web-based Fracture Risk Assessment Tool (FRAX) that integrates clinical risk factors, femoral neck BMD, country-specific mortality and fracture data and

Table 33.4 The WHO classification of osteoporosis based on BMD measurements, with reference to the number of SDs from the BMD in an average young adult (T-score)[27]

Status	T-score
Normal	≥ –1 SD
Osteopenia	> –2.5 SD or < –1 SD
Osteoporosis	≤ –2.5 SD
Established osteoporosis	≤ –2.5 SD, with one or more associated fragility fractures

Abbreviations: BMD = bone mineral density; SD = standard deviation; WHO = World Health Organization

calculates the 10-year fracture probability in order to help healthcare professionals identify patients who need treatment.[32] The clinical risk factors for fractures identified in the FRAX prediction model include advanced age, low body weight, previous fragility fracture, parental history of hip fracture, glucocorticoid treatment, current smoking habit, alcohol intake, rheumatoid arthritis (RA), and other secondary causes of osteoporosis. FRAX, however, does not include other important risk factors for fracture, in particular those related to frailty and fall risk.[33] A prospective study in Hong Kong of 1435 community-dwelling, postmenopausal, treatment-naive women (mean age, 63.4 ± 8.3 years) followed up for 5.0 ± 2.3 years revealed that the most important independent risk factors for low-trauma fracture were the use of walking aids and history of falls.[34] Other predictive factors in this local study included being homebound, calcium intake of <400 mg/d, age of >65 years, history of fracture, and BMI of <19 kg/m^2. Subjects with three to eight clinical risk factors had a predicted 10-year risk of osteoporotic fracture of 25%, increased to 30% if they also had total hip BMD T-score of ≤ −2.5.[34]

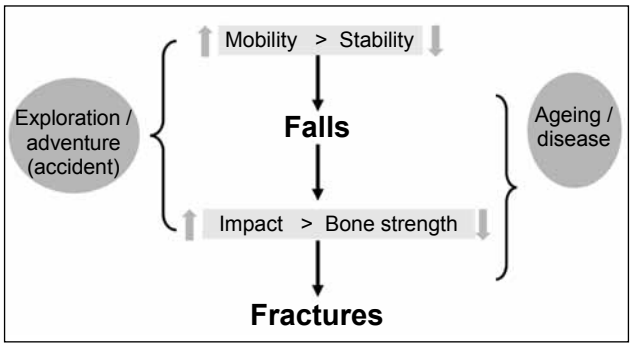

Figure 33.1 Relationship between falls, osteoporosis, and fractures

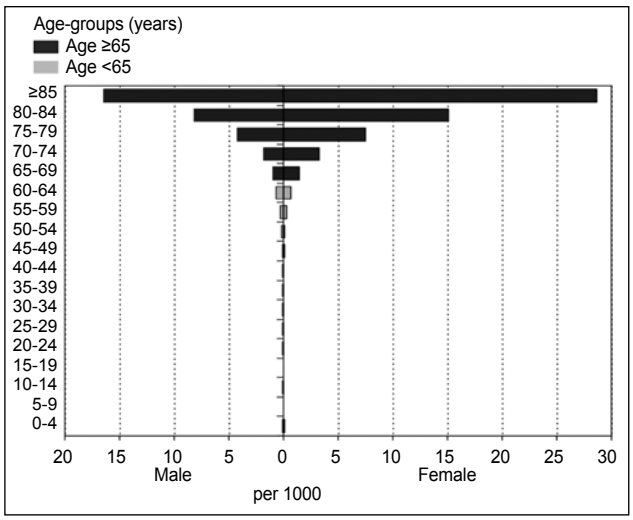

Figure 33.2 Incidence of hip fractures in Hong Kong population in 2004 by age-groups and sex (data from Hospital Authority, Hong Kong)

33.2.2 Epidemiology and Impact

The relationship between falls, osteoporosis, and fractures is shown in Figure 33.1; fall as an important geriatric giant is detailed in Chapter 20. The major fractures in elderly people involve the forearm, vertebral body, humerus, pelvis, and hip. The incidence of these fractures increases with advancing age and is higher in women than men (Figure 33.2), because of their lower peak bone mass, more rapid bone loss, and greater risk of falls. Osteoporosis has a major impact on elderly people, with physical and psychosocial consequences (Table 33.5), best described in the patient's own words (Case 33.2).[35]

Case 33.2 How osteoporosis affects the lifestyle of an 82-year-old woman[35]

"One of the hardest things for me is getting in and out of the car. I cannot shop very long like I used to be able to. To put my arms up while I am looking through a clothes rack really hurts me. When I walk, I am looking around all the time with my face looking down at the sidewalk. I cannot go up and down curbs because of jolting and hurting my back. When you cannot look at the flowers for fear of falling, walking is not nearly as much fun. One of the things I love to do and cannot do anymore is sit at my sewing machine, because I am stooped over and I have too much pain when I do that."

A cross-sectional study of 769 community-based female subjects in Hong Kong found that the mean bone mass of women over the age of 60 years was 30% lower than that of young healthy women, and more than 50% of women over the age of 70 years had osteoporosis.[36] In Hong Kong, it was estimated that there were about 300 000 women and 100 000 men with osteoporosis and low bone mass in 2001; and that by 2030, the numbers will be more than double.[37] Locally, osteoporosis resulted in 5490 hip fractures in elders (aged ≥65 years) or 550/100 000 elders in 2013, causing severe pain and disability. The predominance of hip fractures in people over 65 years of age and the increase in age-specific incidence of hip fractures

Table 33.5 Consequences of osteoporosis

Physical	Psychosocial
Pain	Social isolation
Reduction in physical activity	Loss of independence
Body deformity (kyphosis, reduced height)	Fear and anxiety
Muscle weakness	Depression
Fatigue	Anger

with age that has been reported in the West[38] have also been observed in Hong Kong (Figure 33.2). The main risk factors for osteoporotic hip fracture in Hong Kong Chinese have been identified as a low calcium intake, a lack of load-bearing activity, smoking, and frequent falls.[39] A local cross-sectional analysis of the problem of osteoporosis in 1225 women enrolled for health screening revealed that the cumulative lifetime risk of having an osteoporotic fracture for an ambulatory free–dwelling woman at the age of 80 years was 21%; the adverse risk factors for fracture were lower body weight, lower body mass index, previous gastrointestinal surgery, and osteoarthritis (OA).[40]

Studies in Hong Kong and the UK have shown that a low serum 25-hydroxyvitamin D level is common among hip fracture patients who are less ambulant and home-bound. Vitamin D deficiency could not be predicted from routine blood biochemistry measures of calcium metabolism (levels of serum calcium, phosphate, or alkaline phosphatase).[41-43] Low vitamin D levels are also prevalent in institutionalised elders.[44]

33.2.3 Vertebral Fractures and Secondary Osteoporosis

Much attention has focused on osteoporotic fractures of the femur and wrist, while vertebral crush fractures have been relatively neglected in diagnosis and management.[45] A study in Hong Kong in 1995 revealed that 30% of women and 17% of men who were aged 70-79 years had experienced one or more vertebral fractures.[46] A follow-up (mean, 4.0 ± 2.8 years; range, 1-14 years) study of a cohort of 4386 community-dwelling Southern Chinese recruited into the Hong Kong Osteoporosis Study since 1995 revealed that the clinical vertebral fracture incidence rate for elders (aged >65 years) was 299/100 000 person-years in men, and 594/100 000 person-years in women.[47] This study also demonstrated that although the hip fracture incidence in Asians is lower than that in Caucasians, the incidence of clinical vertebral fractures was similar. Thus the FRAX fracture prediction model that assumes a universal spine-to-hip fracture ratio across ethnic groups may be biased.[47]

In an elderly patient with multiple pathologies, the diagnosis of vertebral crush fracture as the source of presenting distress may be masked (Case 33.3). This underdiagnosis is liable to occur when an elderly person presents with the complications of vertebral crush fractures or analgesic treatment, e.g. pneumonia (from reduced coughing effort), exacerbation of chronic bronchitis, constipation (from immobility or side-effects of narcotic analgesics), heart failure (from fluid retention after NSAIDs).

Case 33.3 An 80-year-old man with chest pain, cough, and dyspnoea

An 80-year-old man with a history of chronic smoking, beta-thalassaemia trait, hypertension, hypertensive heart disease with heart failure, and previous thoracic herpes zoster was distressed by acute severe knife-stabbing pain over bilateral subcostal regions that was aggravated by turning and lying down. Further questioning revealed a history of recent strenuous pulling exercise and lifting of his great grandchild. He also complained of recent constipation. Examination revealed spinal tenderness and X-rays revealed collapse of T7 and L2 vertebral bodies as well as faecal loading (Plate 33.2). He was given analgesics (diclofenac, dologesic, nasal calcitonin) for pain relief, but was hospitalised 1 week later for chest pain, cough, and dyspnoea. His blood pressure had increased dramatically (pain, side-effect of non-steroidal anti-inflammatory drug [NSAID]), and his renal function worsened (NSAID side-effect). X-ray revealed pneumonia and faecal impaction; and his sputum grew *Streptococcus pneumoniae*. He improved after his pneumonia was treated, faecal impaction cleared, NSAID withdrawn, blood pressure stabilised, and mobility restored with a course of rehabilitation.

Up to 55% of men and 30% of women with symptomatic vertebral crush fractures have underlying secondary osteoporosis (Table 33.6),[3,45] treatment of which may lead to a significant increase in bone density and symptomatic improvement. These conditions should therefore be sought by taking a thorough history (drug review, past operations including gastrectomy and oophorectomy, past radiotherapy for nasopharyngeal carcinoma, alcohol or smoking history), physical examination, and appropriate investigations (e.g. workup for possibility of myeloma when diffuse osteoporosis occurs in the presence of raised erythrocyte sedimentation rate [ESR] and serum globulin level).

Table 33.6 Causes of secondary osteoporosis[3,45]

Steroids
Hypogonadism
Hyperthyroidism
Neoplasia (especially myeloma)
Alcohol excess
Chronic smoking
Post-gastrectomy
Post-oophorectomy
Primary hyperparathyroidism
Anticonvulsant drugs
Other / multiple causes

33.2.4 Clinical Presentations and Diagnosis

An elderly person with osteoporosis may present with the consequences of osteoporosis (Table 33.5) or its complications as described above, together with a history of traumatic events that may be overt (e.g. fall, sudden movement, lifting, jumping) or covert (e.g. cough, exercise). Spontaneous fractures have nonetheless been reported in the long bones of bedridden elderly people, without any apparent external force or abuse, during daily care procedures.[48-51] Risk factors implicated in these spontaneous 'care' fractures include age >80 years, female gender, joint contractures, paretic limbs, dementia, previous fractures of the same bone, and a marked decrease in bone mass and bone quality secondary to immobility, disease, and malnutrition.[50-54] The incidence of spontaneous fractures in long-term care facilities has been estimated in two prospective cohort studies in the US and France at approximately 1 per 100 residents per year.[48,53] Spontaneous fractures raise concern not only because of the increased morbidity (pain, functional decline), mortality, and cost, but the repercussions of the possibility of inadequate care and elder abuse with potential medicolegal complaints.[48,54,55] Staff need to explain to families that fractures can occur in at-risk frail elderly persons without direct trauma.[53,54] Proposed measures to reduce spontaneous fractures in long-term care facilities include identification of at-risk residents, educating staff to recognise pain and distress in non-verbal residents, and the use of techniques designed to reduce bone stress during routine patient care (e.g. avoid torsion movement during transfer).[53,54]

Osteoporotic kyphosis may cause the ribs to press against the pelvis with consequent abdominal pain (ribs on pelvis syndrome)[56,57] or groin pain (costo-iliac impingement syndrome).[58] Failure to recognise such rib impingement syndromes may result in unnecessary extensive investigations or inappropriate treatments due to misdiagnosis.[57] Thoracic kyphosis is associated with dyspnoea and ventilatory dysfunction of a restrictive and obstructive type,[59] and may even result in dysphagia.[60] Physical examination typically reveals tenderness over an area of fracture, associated bruising, externally rotated leg in case of hip fracture, spinal deformity (kyphosis), loss of height, or the subsequent development of a protuberant abdomen due to laxity of the abdominal muscles. Dual-energy X-ray absorptiometry (DEXA) is currently the most validated method of measuring BMD. The appropriate use of BMD measurement in elderly people as suggested by Francis is shown in Table 33.7.[61]

Table 33.7 The use of bone density measurement in elderly people[61]

Indications for BMD measurement
• Fractures after minimal trauma in previously fit individuals
• History of early menopause (<45 years) in women up to the age of 70 years
• Underlying secondary causes of osteoporosis
• Apparent osteopenia on X-ray
Treatment of osteoporosis justified without BMD measurement
• Fractures after minimal trauma in frail individuals
• Prolonged oral steroid therapy

Abbreviation: BMD = bone mineral density

33.2.5 Management

Although the Royal College of Physicians[62] and the National Osteoporosis Guideline Group[63] in the UK recommends that prevention of osteoporosis should be directed towards selected case finding rather than population-wide screening, the National Osteoporosis Foundation[64] and the US Preventive Services Task Force[65] in the US have recommended screening for osteoporosis in women aged >65 years. The National Osteoporosis Foundation also recommends screening for osteoporosis in men aged >70 years.[64] Evidence for the effectiveness of osteoporosis screening has been demonstrated by an observational study showing that the receipt of hip DEXA scans in older adults (aged ≥65 years) was associated with a 36% lower hip fracture incidence (4.8 per 1000 person-years vs. 8.2 per 1000 person-years) during 6 years of follow-up compared with usual medical care.[66] The Osteoporosis Society of Hong Kong 2013 Guideline did not recommend population screening for osteoporosis but instead advocated a case-finding approach by increasing physician awareness of the clinical risk factors for osteoporosis.[29] The reasons for and against implementing an osteoporosis screening programme in Hong Kong have been examined by Hui[67] using WHO criteria for a disease screening programme. He argued that large-scale screening was not a valid option in Hong Kong based on currently available evidence.

Treatment of osteoporosis is generally recommended in individuals at high risk of fracture: those who have a BMD T-score of ≤ −2.5, a history of fragility fracture, or a FRAX score indicating increased fracture risk.[63,64,68] An algorithm for the management of osteoporosis is summarised in Figure 33.3.[61,62,68] For both primary and secondary prevention, lifestyle advice is important and includes adequate nutrition (especially with calcium and vitamin D), regular weight-bearing exercise, and avoidance of smoking and excessive alcohol. Once a patient has fractured, pain should be relieved by physical measures, appropriate bed rest, and analgesics. History taking, physical examination, and investigations should address risk factors and possible secondary causes of

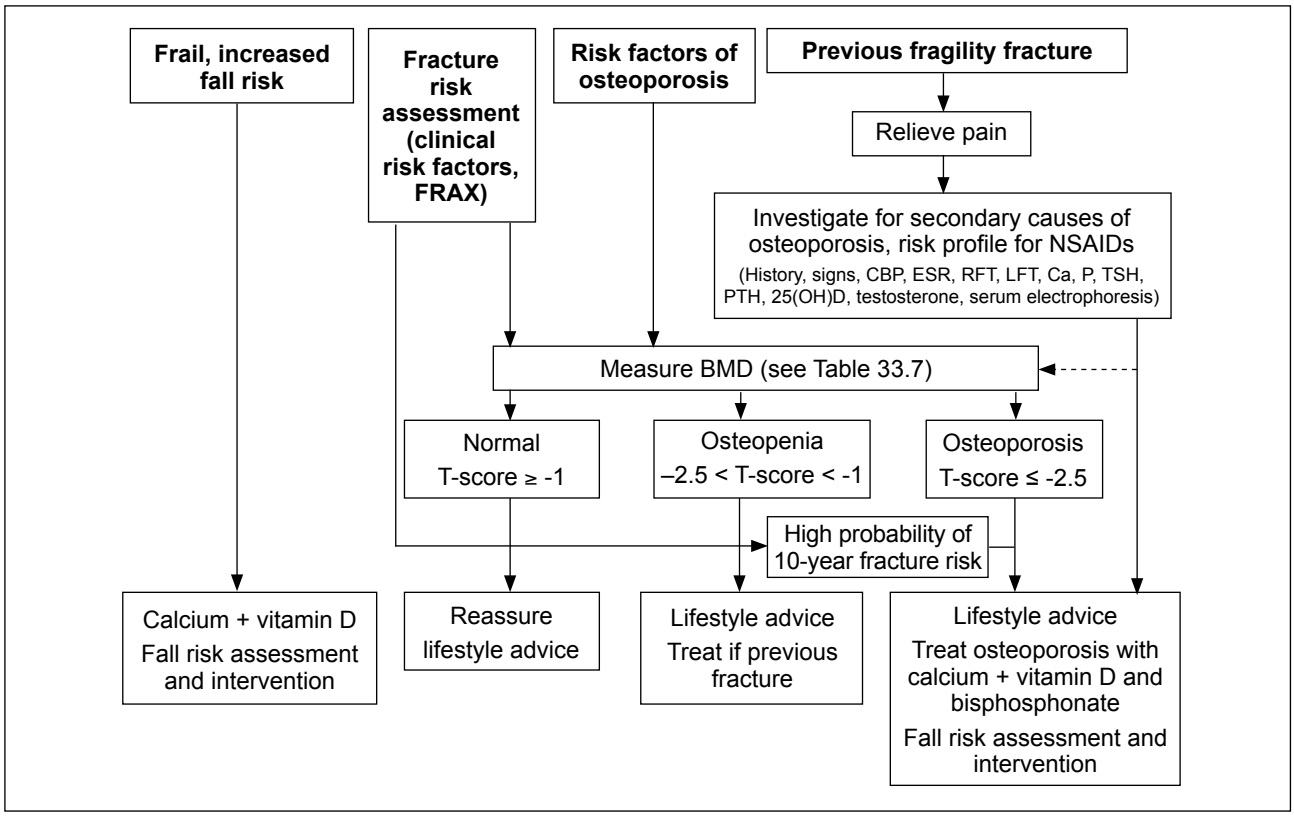

Figure 33.3 Algorithm for the management of osteoporosis[61,62,68]
Abbreviations: 25(OH)D = 25-hydroxyvitamin D; BMD = bone mineral density; Ca = calcium; CBP = complete blood picture; ESR = erythrocyte sedimentation rate; LFT = liver function test; NSAIDs = non-steroidal anti-inflammatory drugs; P = phosphate; PTH = parathyroid hormone; RFT = renal function test; TSH = thyroid-stimulating hormone

osteoporosis, fall risk, and a profile of the individual's vulnerability to the toxicity of NSAIDs. Decision making should also take into account biological age, frailty, premorbid physical and cognitive functioning, likely compliance, and patient preference. Frail elders who were immobile or housebound before fracture will benefit more from calcium and vitamin D supplementation, as they are more likely to be deficient in vitamin D. Elders who were mobile and independent before fracture will benefit from more active investigation and therapeutic intervention, as quality of life may be improved by reducing the risk of further fractures.

The Women's Health Initiative study, while confirming the well-known benefit of hormone therapy in the prevention and treatment of osteoporosis, nonetheless found that long-term hormone therapy users had an increased risk of cardiovascular disease, stroke, and breast cancer that outweighed the benefit of fracture reduction, even in women at high risk of fracture.[69,70] This led to new clinical guidelines that advise against the use of hormone therapy as a first-line measure for prevention and treatment of osteoporosis in postmenopausal women and emphasise more alternatives such as exercise, increasing calcium and vitamin D intake, and the use of other effective antiresorptive agents such as bisphosphonates.[68,71,72]

Plant-derived oestrogens, especially soy phytoestrogens (isoflavones), may serve as a natural substitute for hormone replacement therapy. The Shanghai Women's Health Study, a large cohort study in China that examined the relationship between usual soy food consumption and fracture incidence in 24 403 postmenopausal women with no history of fracture or cancer and who completed a food frequency questionnaire at baseline and during a mean of follow-up of approximately 4.5 years, concluded that soy food consumption might reduce the risk of fracture in postmenopausal women, particularly among those in the early years following menopause. For this subgroup, the risk of fracture was reduced by 48% among those with the highest intake of soy protein compared with those with the lowest. In women who had been menopausal for >10 years, the risk reduction was 29%.[73]

Exercise may reduce fracture risk by enhancing bone strength and reducing fall risk by improving muscle strength and balance. Both impact and non-impact exercise had a positive effect on lumbar spine bone

density in postmenopausal women, whereas only impact exercise had a positive effect at the femoral neck.[74] Patients with vertebral fractures benefit from resistance exercises that strengthen back extensor muscles, but back flexion exercises are harmful and increase the risk of new vertebral fractures.[75] Tai Chi is popular among local Chinese, and has been found to retard bone loss; improve muscle strength, body flexibility, coordination, and balance; as well as reducing the risk of fragility fractures.[76-78]

Meta-analysis and pooled analysis have demonstrated that vitamin D 800 IU/day combined with calcium was effective in reducing osteoporotic fractures in both community-dwelling and institutionalised middle-aged to older adults.[79-81] Vitamin D reduces fracture risk probably by improving bone health and reducing the risk of falls through enhancement of muscle strength and neuroprotective reflexes.[82]

Continuous treatment in postmenopausal women with osteoporosis with the second-generation bisphosphonates has been shown to increase BMD and reduce fracture rates at vertebral and non-vertebral sites, such as the hip and forearm, without an adverse effect on mineralisation.[83] The major side-effect of oral bisphosphonates is oesophageal irritation. To reduce drug-induced oesophagitis and to ensure adequate absorption, a bisphosphonate should be taken in the morning, on an empty stomach, with a full glass of water, and the patient should remain upright for 30 to 45 minutes before eating. A concern of long-term bisphosphonate use is the risk of osteonecrosis of the jaw and atypical femoral shaft fractures with minimal or no preceding trauma, reported to occur at respective rates of <1 and 1 to 6 cases per 10 000 patient years.[84,85] Because benefits are retained after discontinuation of alendronate or zoledronic acid, drug holidays after 5 years of alendronate therapy or 3 years of zoledronic acid therapy may be considered for patients at lower risk for fracture.[68,86] Further discussions on the different anti-osteoporosis agents (alendronate, risedronate, ibandronate, zoledronic acid, calcitonin, raloxifene, denosumab, teriparatide, strontium ranelate) are to be found in Chapter 12. A detailed review of these anti-osteoporosis agents can be found in the Osteoporosis Society of Hong Kong 2013 Guideline.[29]

33.3 OA

A local survey in 2000 revealed that 40.6% of non-institutionalised elders aged ≥60 years reported having arthritis, second only to hypertension as the most common cause of chronic conditions in old age.[87] OA is estimated to affect one in three people aged ≥70 years in Hong Kong with over 80% having OA of the knee. Women are nearly 4 times as likely to have OA of the knee as men.[88]

The view of OA as a single degenerative joint disease related to ageing and 'wear and tear' of the joint has been replaced by the current concept of a synovial joint syndrome of overlapping osteoarthritic disorders with multiple pathogenic contributors resulting in an imbalance between degradation caused by mechanical stress and the process of repair.[89] Thus OA is now considered a heterogeneous condition with numerous factors leading to its pathological hallmark of cartilage loss and the clinical manifestation of joint pain with movement, stiffness, bony swelling and crepitus, instability, and loss of movement and function. The major pathogenic factors include chondrocyte regulation of the extracellular matrix, genetic influences, loading (overweight, injury), local mechanical factors, and inflammation (crystals, cytokines).[90,91] Although these multiple factors interact with one another, individual patients may have a predominant pathogenic contributor (e.g. inflammation due to crystals) that can be the target of treatment.

Management of OA of the knee and hip should start with patient education, weight reduction, physical therapy to strengthen related muscles, and an exercise programme. Drug treatment for OA is targeted towards symptomatic relief in a stepwise approach, beginning with simple analgesics (e.g. paracetamol), with NSAIDs added if pain is not relieved or when the OA is of the predominant inflammatory type.[90,91] Intra-articular steroids are effective in OA, particularly for the knee.[92] Glucosamine or chondroitin provide symptomatic relief; their mechanism is thought to shift cartilage metabolism to a positive balance.[93,94] Colchicine can be used in inflammatory OA that is unresponsive to NSAIDs or intra-articular corticosteroids since such patients often have evidence of calcium pyrophosphate dihydrate (CPPD) crystals,[95,96] but this has to be used with caution among elders (see Chapter 33.4 below). For elderly patients with OA of the knee or hand, topical NSAIDs may be preferable to systemic NSAIDs for pain relief because of the lower risk of gastrointestinal and cardiovascular adverse events. Because of the proven efficacy and tolerability of topical NSAIDs, they are now included in international guidelines for the treatment of patients with OA of the hand or knee, and some guidelines have further recommended topical NSAIDs be considered before oral analgesics.[97] Acupuncture has been shown in a randomised controlled trial to be an effective adjunctive therapy for reducing pain and improving function in patients with OA knee.[98] Surgery (including total joint replacement) should be considered in patients with moderate-to-severe symptomatic OA that does not respond to medical management and with impaired activities of daily living.

33.4 Gout

Gout is the most common cause of inflammatory arthritis. The overall prevalence of gout in the UK has risen from 1.4% in 1997 to 2.5% in 2012.[99] The prevalence increases with age: 1.5% in men and 0.13% in women aged 35-39 years (male-to-female ratio, 11.2); 13.8% in men and 4.5% in women aged 75-79 years (male-to-female ratio, 3.0); 14% in men and 5.6% in women aged >90 years (male-to-female ratio, 2.5).[1] In a meta-analysis that included six studies of the prevalence of gout in China, an increasing prevalence of gout was also found; rising from 0.9% in 2000-2005 to 1.1% in 2006-2010 to 1.4% in 2010-2014.[100] These epidemiological trends of a rising prevalence over the past decade and with increasing age have been attributed to increased longevity, large use of diuretics, comorbidities associated with hyperuricaemia, and more time for deposition of monosodium urate crystals to occur in older persons.

Elderly-onset gout, defined as gout with onset at age of ≥65 years, differs from typical (younger-onset) gout in epidemiology, clinical presentation, and management.[9,101-106] Elderly-onset gout is the most common inflammatory arthropathy in older people. Among patients with an onset of gout after the age of 65 years, the gender distribution between men and women is equal, and in those with onset after 80 years of age, women predominate.[101-103] The clinical presentation of elderly-onset gout often differs from gout in younger age[101-103,107-117] (Table 33.8, Plates 33.3, 33.4), and is characterised by more tophi, polyarticular onset with hand involvement, and frequent association with diuretics and renal impairment. It seems that elderly patients, especially women, are at risk of developing early tophaceous deposits and polyarticular gout without a history of acute monoarthritis.[103] Failure to recognise this altered presentation in old age may result in underdiagnosis (NSAID-masked gout for indolent forms)[7,9] or misdiagnosis as other arthritic disorders (e.g. RA, OA).[104,105,107,118] or even as stroke with acute hemiparesis.[108] Failure to recognise that gout can involve the spine leads to misdiagnosis of axial or spinal gout as compression fracture, metastatic cancer, or infection (Plate 33.5).[119] In gout patients who present with back pain unresponsive to conservative measures, axial gout should be considered as a probable differential diagnosis.[119]

The major pathogenic factors of gout include development of hyperuricaemia leading to urate saturation; formation of monosodium urate crystals from body fluids saturated with urate; and interactions between monosodium urate crystals and leukocytes to mount inflammatory responses. Factors that exacerbate gout and their possible mechanisms are shown in Table 33.9.[9,104,105,120]

Although typical or classic gout can be clinically

Table 33.8 Clinical features of gout: typical versus elderly-onset[101-103,107-117]

	Typical gout	Elderly-onset gout
Age of onset (years)	Mid-40s	Over 65
Sex distribution	Male predominant	Equal sex[101-103]
Arthritis	Acute monoarthritis (Plate 33.3)	Subacute polyarticular gout[101,107,108] (Plate 33.4)
	Lower limb (big toe)	Upper limb (fingers), may spare the feet[101,102,107]
Tophi	Late (after years of attacks)	Early (or without a history of gouty arthritis) tophi formation over fingers[102,109]
Systemic reaction	-	Fever (Plate 33.5), leukocytosis, malaise[107,108]
Associated features	Obesity, hyperlipidaemia, insulin resistance syndrome, hypertension, alcohol use	Diuretic use[101,102,110-112] Low-dose aspirin[113] Blood dyscrasias (multiple myeloma, polycythaemia)
Multiple pathology / aetiology	-	Coexistent gout and osteoarthritis / Heberden's nodes[111,112] (Plate 33.4) Coexistent gout and seronegative rheumatoid arthritis[114] Coexistent gout and septic arthritis[115-117]

Table 33.9 Exacerbating factors in gout[9,104,105,120]

Triggering factor	Mechanism
Infection	Lactic acidosis
Minor trauma	Intra-articular flaking of urate crystals
Recent surgery	Increased tissue breakdown
Dieting	Ketoacidosis
Diuretics, low-dose aspirin, alcohol	Rapid rise in urate levels
Hypouricaemic therapy (allopurinol), high-dose salicylates, sudden alcohol withdrawal	Rapid fall in urate levels, allowing release of more urate crystals from deposit (mobilisation attacks)

diagnosed by the New York criteria[121] (Table 33.10) or the new 2015 Gout Classification Criteria,[122] the occurrence of an altered presentation commonly found in old age should alert one to consider gout in elderly patients when these non-classic features (Table 33.8) are present in the setting of known exacerbating factors (Table 33.9). In particular, the possibility of gout should not be ignored in a patient with OA.[104,105,114] The diagnosis of gout can be confirmed by aspiration of synovial fluid from affected joints or aspiration of a suspected tophaceous nodule, and the detection of needle-shaped, negatively birefringent urate crystals (Plate 33.5c) under polarised light microscope.[9,26,120] Serum urate concentration is of limited diagnostic value as a normal level is found in up to half of patients during an acute attack of gout and many patients with hyperuricaemia never develop acute gout.[9,26,120] In patients with long-standing disease or in elderly patients with early tophaceous disease, X-rays may reveal well-defined gouty erosions (cortical break with sclerotic margin and overhanging edge) in or around joints in the hands and feet. X-rays also help to exclude other diseases (chondrocalcinosis, calcific periarthritis, RA, septic arthritis).[9,26] Advances in gout imaging facilitate the identification of urate deposits on ultrasound images as the double contour sign (hyperechoic enhancement over the surface of the hyaline cartilage) and on dual-energy computed tomography as the presence of colour-coded urate at articular or periarticular sites.[123] These characteristic imaging features have been combined with biochemical findings, clinical features, signs, and symptoms into a scoring system for the new 2015 classification criteria for gout that facilitate research on gout and elucidate the key features of the disease (http://goutclassificationcalculator.auckland.ac.nz/).[122]

The differential diagnoses of acute gouty arthritis in elderly patients include pseudogout (CPPD deposition disease), acute septic arthritis, joint trauma, RA, cellulitis, and deep venous thrombosis.[9,104,105,120] CPPD deposition disease is mainly a disease of elderly people with three most common clinical forms of presentation: acute attacks of mono- or less frequently poly-arthritis (pseudogout); chronic arthropathy associated with OA; or an incidental asymptomatic radiological finding (chondrocalcinosis). Acute pseudogout (Plate 33.6), like gout, can be precipitated by acute medical illness, trauma, or surgery[124]; associated with systemic features of fever, confusion, and leukocytosis[125]; and relieved by colchicine. Knee involvement is characteristic of CPPD deposition disease, but wrist, shoulder, and hip joints are also frequently involved. The diagnosis of pseudogout is confirmed by synovial fluid aspiration that yields rhomboid-shaped, weakly positively birefringent CPPD crystals (Plate 33.6c) under polarised microscopy. Acute gouty arthritis and septic arthritis can be difficult to differentiate clinically (Plate 33.5), and may even coexist in the same joints[117,118] Thus, any suspicion of septic arthritis should be confirmed by synovial fluid and blood cultures.[104,120]

An acute attack of gout can be treated with NSAIDs, colchicine, or corticosteroids; the choice should be based on the patient's biological age, overall health and frailty, and clinical profile of cardiac function, renal function, and any underlying gastrointestinal disease (Figure 33.4).[29,104,105,120,126-128] Because of the increased toxicities of NSAIDs and colchicine[129,130] in old age, corticosteroids have been promoted as the preferred treatment for acute gout in elderly patients with multiple comorbidities.[29,104,105,120,126,131] There is frequent reluctance to use corticosteroids to treat acute gout in elderly patients because of concern about their hyperglycaemic, osteoporotic, and immunomodulatory effects.[132] Immunological and osteoporotic effects, however, are not expected to occur with short-term use, and a transient increase in blood glucose concentrations can be managed while acute gout is treated.[106,132,133] Thus, for patients with gout and concomitant diabetes mellitus, steroids remain an appropriate treatment of acute gout, because diabetic patients often have renal impairment that renders them prone to the toxicities of NSAIDs or colchicine.[133] In a multicentre randomised controlled study of patients who presented with acute gout to emergency departments in Hong Kong, oral prednisolone 30 mg/day and indomethacin resulted in similar clinically significant reductions in pain.[134] The challenge of managing acute gout in an elderly person and its control by use of appropriate prednisolone dosage and regime is illustrated in Case 33.4.

Table 33.10 Clinical diagnostic criteria for typical gout[121]
(Clinical gout is diagnosed in the presence of two of the following four criteria)

- A clear history of at least two attacks of painful joint swelling with complete resolution within 2 weeks
- A clear history or observation of podagra (the attack of excruciating pain, erythema, and subsequent overlying desquamation of the first metatarsophalangeal joints)
- The presence of a tophus
- Rapid therapeutic response after NSAIDs / colchicines

Abbreviation: NSAIDs = non-steroidal anti-inflammatory drugs

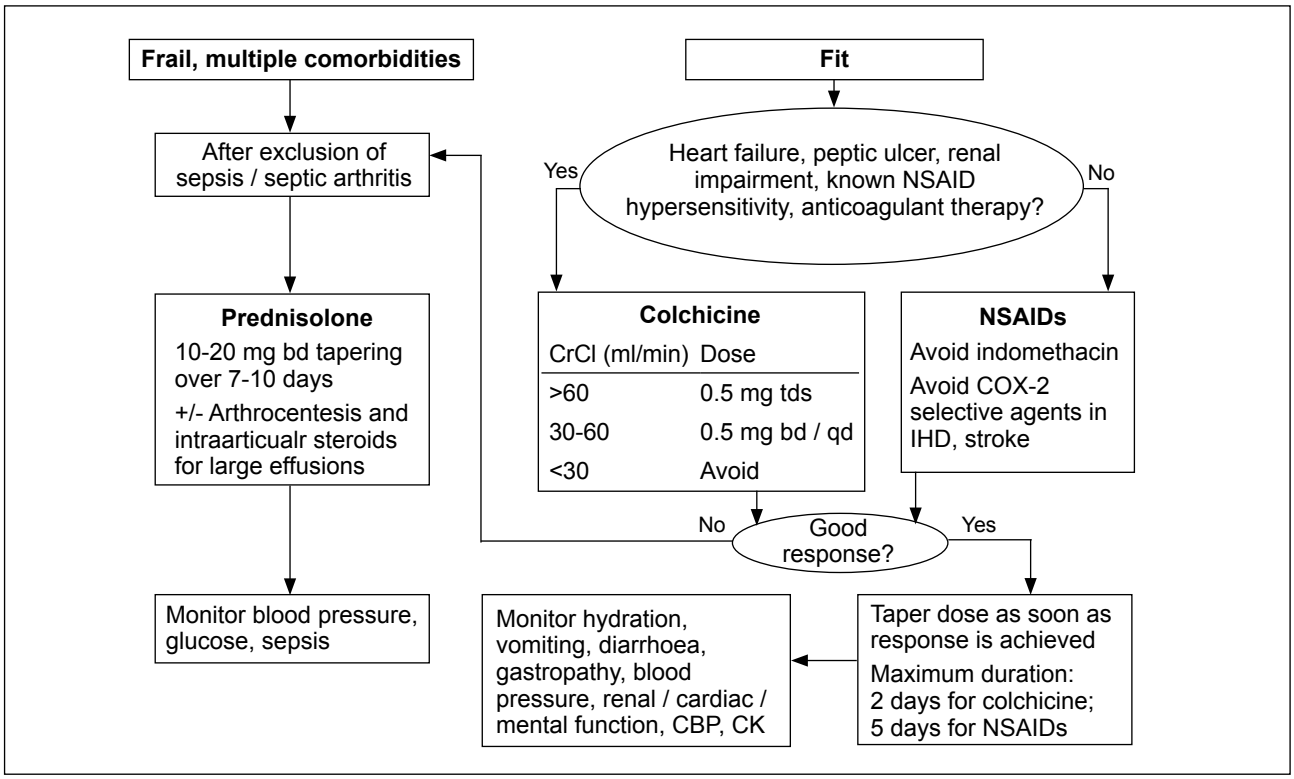

Figure 33.4 Algorithm for the treatment of acute gout[29,104,105,120,126-128]
Abbreviations: bd = 2 times a day; CBP = complete blood picture; CK = creatine kinase; COX-2 = cyclooxygenase-2; CrCl = creatinine clearance; IHD = ischaemic heart disease; NSAID = non-steroidal anti-inflammatory drug; qd = daily; tds = 3 times a day

Case 33.4 An 80-year-old woman immobilised and distressed by polyarthritis and abdominal pain

An 80-year-old woman with independent premorbid mobility and known history of hypertension and polyarticular gout (big toes, ankles, knees) presented with severe bilateral ankle pain and swelling limiting her mobility to wheelchair-dependency. Two months before, she was treated by an orthopaedic surgeon with diclofenac SR 100 mg daily and colchicine 0.5 mg tds for acute gouty arthritis of her ankles. She had some improvement in her ankle pain, but she could not tolerate the medication because of epigastric pain with reduced appetite. She thus switched to see a rheumatologist, and was given 6 days of prednisolone 35 mg daily and colchicine 0.5 mg bd. Her ankle pain improved while on prednisolone. However, she had increasing ankle pain and swelling after completion of her prednisolone course, so severe that she could not walk and required a wheelchair for mobility. She was also seen by a surgeon for 2-month on-and-off abdominal pain, constipation alternating with diarrhoea, mild anaemia, high erythrocyte sedimentation rate (ESR; 100 mm/h), positive occult blood in stool, for which she was investigated with immunological screen (rheumatoid and antinuclear factors), sigmoidoscopy, computed tomography of the abdomen and pelvis, but the investigatory results were non-revealing. Distressed by her ankle pain, she had just resumed her diclofenac SR, but her oral intake was much reduced both because of diclofenac-induced epigastric discomfort and deliberate dieting to reduce gout.

At presentation, she had acute arthritis of both ankles, hyperuricaemia (serum urate 0.5 mmol/L), hypotension and bradycardia (blood pressure 100/56 mm Hg, pulse 55/min), anaemia (haemoglobin 9.1 g/dL), and impaired renal function (urea 13.9 mmol/L, creatinine 129 μmol/L, creatinine clearance [by Cockcroft-Gault equation] 31 ml/min, estimated glomerular filtration rate [by Modification of Diet in Renal Disease equation] 34 ml/min/ 1.73m2). She had rebound of acute gouty arthritis upon abrupt stoppage of prednisolone course, treatment complications (diclofenac-related gastritis with epigastric pain, reduced oral intake, and anaemia; diclofenac-related acute kidney injury; colchicine-induced diarrhoea; carvedilol-induced bradycardia and hypotension), and disease complications (acute gout resulting in

immobility, which in turn caused constipation). The combination of reduced oral intake, diarrhoea, and hypotension add on the diclofenac-related acute kidney injury to cause further impairment of her renal function, which resulted in urate retention and worsened her gout.

Her acute gouty arthritis of ankles responded to a prednisolone course tailing down over 2 weeks. Her anaemia was corrected (haemoglobin rising from 9.1 to 12.7 g/dL) after 2 months of oral iron. Her epigastric pain subsided upon stopping diclofenac and a course of lansoprazole. Her blood pressure stabilised with reduction of carvedilol. Here ESR normalised after treatment of acute gout and anaemia. Her renal function improved (serum creatinine reduced from 129 to 99 μmol/L). Her mobility was restored to premorbid level of independent walking. With the use of allopurinol her urate was normalised and her gouty attacks reduced.

This case illustrates that elderly patients with gout may be under care of multiple specialties and at risk of prescribing cascade, polypharmacy, iatrogenesis, and over-investigations. A holistic approach and appropriate prescription would help to relieve her pain, restore her mobility, and optimise her medical condition.

Measures to prevent recurrent attacks of gout are shown in Table 33.11.[104,127,135,136] Chronic urate-lowering therapy with allopurinol should be considered in established gout with frequent gouty attacks (more than once per year), tophi, stage 2 (or worse) chronic kidney disease, or past urolithiasis.[137] Normalisation of serum urate levels in symptomatic patients will eventually lead to prevention of further attacks, resorption of tophi, and improvement in renal function, possibly due to reduced renal urate deposition or reduced use of NSAIDs.[104] Anti-inflammatory cover with low-dose colchicine for the first 6 months of allopurinol initiation reduces the chance of a mobilisation attack of gout from the sudden decrease in urate level.[138] In renal impairment, the clearance of colchicine is decreased and thus its dose should be reduced: 0.5 mg once daily if the creatinine clearance (CrCl) is 35 to 49 ml/min and 0.5 mg every 2 to 3 days if it is 10 to 34 ml/min.[135] The target serum urate level recommended is <0.36 mmol/L,[127,139,140] or even lower at 0.3 mmol/L for those with visible / palpable tophi (reflecting a higher total urate burden) to allow for their more rapid resolution.[106] An unnecessarily low serum urate level should be avoided because patients with a sustained low serum urate level may be at increased risk of Parkinson's disease or dementia.[141-143] This concern for sustained hypouricaemia has nevertheless to be balanced against data to suggest that hyperuricaemia contributes to the progression of chronic kidney disease,[144] and cardiovascular mortality.[145,146] Despite the American and European rheumatology societies[127,139] advocate in their guidelines a treat-to-target strategy for patients with gout and hyperuricaemia, such a strategy is not recommended by the American College of Physicians,[147] who concluded from a review of the evidence base that this strategy of "treating to a target serum urate level" is based on physiology and observational evidence only without support from experimental testing.

Concern over the rare, but extremely severe, hypersensitivity reaction to allopurinol has contributed to its underuse, especially in patients with renal impairment. While about 2% of patients on allopurinol, particularly elderly patients with renal impairment, develop a pruritic maculopapular rash,[148] a more severe, but less frequent (0.3-0.9% of patients) reaction is an allopurinol hypersensitivity syndrome (AHS, Plate 33.7) of severe cutaneous adverse drug reactions or severe cutaneous adverse reactions (SCAR) [Stevens-Johnson syndrome, toxic epidermal necrolysis, drug reaction with eosinophilia and systemic symptoms], hepatitis, and renal failure, with mortality up to 25%.[104-106,149-151] Severe

Table 33.11 Measures to prevent recurrent attacks of gout[104,127,135,136]

Prophylactic measure	Remark and precaution
Dietary restriction	Impractical; a strict purine-free diet has only a small effect on serum urate levels
Avoid excess alcohol	-
Review need / dose for diuretics	Consider alternatives (losartan or calcium channel blockers) for managing hypertension / cardiac disease
Short-term colchicine prophylaxis for first 6 months of urate-lowering therapy to reduce mobilisation attacks	With caution, low dose (0.5 mg alt day to 0.5 mg twice a day), short-term, or avoid with renal insufficiency (marrow suppression and myoneuropathy reported for chronic low-dose colchicines with CrCl <50 ml/min,[135] fatal interaction with clarithromycin reported[136])
Chronic urate-lowering therapy (allopurinol, febuxostat)	Observe indications, 'start low, go slow', monitor renal function, and reduce dose of allopurinol according to CrCl, monitor liver function for febuxostat

Abbreviation: CrCl = creatinine clearance

AHS is more likely to occur in elderly patients with reduced renal function.[51-53,105,152] The accumulation of renally excreted oxipurinol, the toxic metabolite of allopurinol, has been implicated in triggering hypersensitivity vasculitis syndrome.[51-53,105,153] Hande et al[149] have proposed a guideline to prevent this life-threatening reaction by reducing the maximum maintenance allopurinol dose relative to CrCl (Table 33.12).

Concern has been raised that adherence to Hande's dosing guidelines may have led to underdosing and suboptimal management of gout because of failure to achieve target serum urate concentration.[154,155] Stamp et al[154] showed that a higher starting dose of allopurinol rather than the maximum maintenance dose is a risk factor for AHS, and that gradual escalation above CrCl-based doses is safe and effective when kidney and liver function are appropriately monitored. However, the sample size of 45 patients in Stamp's safety analysis was insufficiently powered to draw any firm conclusion given the low incidence of 0.7/1000 patient-years for AHS,[156] and controversy remains over allopurinol dosing in patients with renal impairment.[155,156] International rheumatology societies guidelines differ in their recommendations: American guidelines allow allopurinol dose to be increased >300 mg daily even in patients with renal impairment,[139] whereas the European guideline indicates that the maximum allopurinol dosage should be adjusted to CrCl.[127] To minimise the risk of AHS and to reduce the chance of a mobilisation attack of gout, a 'start low, go slow' approach for dosing should be adopted.[106,127,128] A starting dose of allopurinol 50-100 mg daily, increased at monthly intervals, to a maximum daily maintenance dose of about 100-300 mg, based upon the patient's estimated CrCl and serum urate level, is recommended (Table 33.11).[51-53,104,105] In those who have experienced mild rash in reaction to allopurinol, and in whom there is a clear-cut need for allopurinol therapy, slow, cautious reintroduction of allopurinol is possible.[157,158]

Apart from allopurinol dose and renal impairment, other factors also contribute to AHS, including the presence of HLA-B*58:01 in people of Han Chinese, Thai, Japanese, Korean, European, and Portuguese descent,[159-164] and concomitant use of diuretics.[148] It is postulated that the presence of HLA-B*58:01 and high oxipurinol concentration act synergistically to trigger AHS through T-cell sensitisation.[152] The HLA-B*58:01 carrier rate is high among the Han Chinese, reported as 20% in Taiwan[165] and 14% in Hong Kong.[166] Studies in Taiwan showed that HLA-B*58:01 screening for SCAR has a sensitivity of 100% and specificity of 85.2% for prediction of allopurinol-induced SCAR; and a positive predictive value of 2% and a negative predictive value of 100% when estimated from the historical incidence of 0.30% per year.[163,165] A study of Hong Kong Han Chinese patients confirmed the strong association between the HLA-B*58:01 and allopurinol-induced SCAR; the presence of the HLA-B*58:01 allele has a sensitivity of 100% and specificity of 86.7% in predicting allopurinol-induced SCAR.[167] Another study in South Korea showed that in the presence of HLA-B*58:01 and stage 3 or worse chronic kidney disease, the risk of developing SCAR is around 18%.[168] The American College of Rheumatology (ACR) Guideline recommends screening for the presence of the HLA-B*58:01 allele in individuals considered to be at high risk of developing AHS — Koreans with stage 3 or worse chronic kidney disease and all those of Han Chinese and Thai descent.[139]

Febuxostat is a newer oral non-purine selective inhibitor of xanthine oxidase with efficacy demonstrated first in the FACT (Febuxostat versus Allopurinol Controlled Trial)[169] and subsequent studies.[170] Its urate-lowering efficacy at 80-120 mg/day is better than 'standard dose' allopurinol (300 mg/day).[169,170] Febuxostat 80 mg daily appears to be equipotent to allopurinol 450 mg/day. Febuxostat may provide an important option in patients unable to use allopurinol, or refractory to allopurinol. It was approved for use at a daily dose range of 80-120 mg in Europe in 2008; 40-80 mg in the US in 2009; 10-60 mg in Japan in 2010.[170] By 2016, febuxostat had been approved for use in most Asian countries. It is mainly eliminated by glucuronidation in the liver. Its pharmacokinetics and pharmacodynamics are not significantly altered in patients with moderate renal impairment or hepatic impairment. Safety data of this drug in patients with severe renal impairment (estimated glomerular filtration rate <30 ml/min) are lacking. In randomised trials of febuxostat, common adverse reactions were liver function abnormalities, nausea, arthralgia, and rash. There was a higher rate of cardiovascular thromboembolic events (cardiovascular deaths, non-fatal myocardial infarctions, and non-fatal strokes) in patients treated with febuxostat compared with those prescribed allopurinol; and trials are ongoing to evaluate the

Table 33.12 Adjustment of maximum allopurinol dose based on creatinine clearance (CrCl), estimated using the Cockcroft-Gault equation[149]

CrCl (ml/min)	Dose (mg)	Interval
100	300	Daily
80	250	Daily
60	200	Daily
40	150	Daily
20	100	Daily
10	100	Every 2 days
0	100	Every 3 days

cardiovascular safety of febuxostat.[170] Post-marketing surveillance has received reports of hepatic failure (fatal and nonfatal), jaundice, rhabdomyolysis, hypersensitivity skin reactions (including SCAR), and anaphylaxis.[170]

33.5 RA

The prevalence of RA in elderly persons aged ≥60 years in the West is 2%.[171] The prevalence of RA ranged from 0.2-0.37% in mainland China, 0.35% in Hong Kong, 0.93% in urban Taiwan, and 0.26% in rural Taiwan.[172] Two disease patterns can be recognised among elderly patients with RA, depending on their age at onset (Table 33.13).[10,173,174] The young-onset RA patients, most of whom have had long years of disease activity, are left with varying degrees of polyarticular synovitis and deformities. Elderly-onset RA (EORA) patients, with their disease onset after 60-65 years of age, are characterised clinically by disabling morning stiffness and marked pain that affects the large proximal joints.

The interpretation of rheumatological laboratory tests also differs in older versus younger patients. Rheumatoid factor (RF) at low titres may occur in 5-42% of healthy elderly individuals,[175] so a positive RF alone is not diagnostic of RA, though a higher titre of RF positivity is given more weighting in the classification criteria for RA. The common occurrence of comorbidities in elders means that an elevated ESR may also be related to factors other than active synovitis, e.g. infection, heart failure, or malignancy. EORA often runs a milder course because of the lower incidence of RF positivity. The differential diagnoses of RA in elderly patients include polymyalgia rheumatica (PMR), CPPD disease, gouty arthritis, OA, occult malignancy, and thyroid disease.[173] Presence of anti-cyclic citrullinated peptide antibodies in EORA has been shown to be useful in distinguishing the condition from PMR with symptoms similar to EORA.[176]

The management of RA aims to control pain, stop disease progression, and improve functional status. In contrast to OA in which exercise and activity are the mainstay of rehabilitation therapy, joint rest is important for active synovitis in RA. Joint rest is enhanced by splinting the affected joints in positions of function and treatment of synovitis.[177] For elders living alone, adequate rest at home may be impossible necessitating inpatient management of acute RA. Current drug therapies include early aggressive treatment with one or more disease-modifying antirheumatic drugs (DMARD), along with symptomatic therapy with NSAIDs and low-dose prednisolone (5-10 mg per day).[173]

Of the DMARD, hydroxychloroquine and sulfasalazine are generally preferred in elderly patients with comorbidities because of their less toxic profile.[173] While the treat-to-target strategy and role of biological DMARD have been established in middle-aged patients with RA, an evidence-based strategy suitable for patients with EORA or younger-onset elderly RA is difficult to establish because of multi-morbidities, frailty, and treatment-related risks.[178] Tumour necrosis factor (TNF) inhibitors are slightly less or equally effective in reducing disease activity in elderly patients with RA than in younger patients. Evidence of non-TNF biological DMARD use in EORA is limited. With regard to safety, observational studies show increasing age as an independent risk factor for serious infections, and infection is the primary concern in elderly patients with RA, who are initiating treatment with biological DMARD.[178]

Low-dose prednisolone has been advocated for EORA because of its strong favourable effects on disease activity,[179] but chronic use carries the risk of osteoporosis and thus the need for bone-protective agents and regular monitoring to detect possible negative side-effects in time.[173,180]

Table 33.13 Clinical features of rheumatoid arthritis: young onset versus elderly onset[10,173,174]

	Young-onset rheumatoid arthritis	Elderly-onset rheumatoid arthritis
Age at onset (years)	30 to 50, advanced stage	>60 to 65
Gender distribution	Female predominant	Equal sex
Arthritis	Gradual >6 weeks Polyarticular (>6)	Abrupt / gradual Oligoarticular (2-6)
	Small distal	Large proximal (shoulders, wrists / knees); spare hemiplegic side
Rheumatoid nodules	Present if seropositive	Rare
Morning stiffness (hours)	1-2	3-4
Sicca symptoms	None in majority	Reported in >50%
Systemic reaction	-	Fever, fatigue, malaise, weight loss
Erythrocyte sedimentation rate	Normal to increased	Significantly raised
Rheumatoid factor	Seropositive	Seronegative

33.6 'Rarer' Musculoskeletal Disorders in Chinese (Paget's Disease of Bone, GCA, and PMR)

33.6.1 Paget's Disease of Bone

Paget's disease of bone (PDB) presents late in life and is reported to be a common disease among elderly people in the West, but regarded as rare in the East. In the West, PDB is the second most common metabolic bone disease after osteoporosis. PDB is characterised by increased and disorganised bone remodelling at one or more foci that contain large overactive osteoclasts, resulting in bone fragility, enlargement, and deformities. In his original article on the disease, Paget[181] illustrated his first patient, a 48-year-old man who complained of painful thighs and legs for 2 years, with bowed legs, large head, enlarging hat, and drooped spine. Paget described this as 'osteitis deformans' because of the deformities caused and his interpretation of the disease as a 'consequence of an inflammation' based on his autopsy finding of extreme resorption and laying down of new bone in a disorganised manner.[181,182] Currently, the pathogenesis of PDB is thought to arise from an interaction of both environmental (e.g. chronic paramyxovirus infection) and genetic factors that impact on osteoclast differentiation and function.[183,184]

The prevalence of PDB has declined over the past three decades in several countries in the West, particularly in those where the disease was initially common.[185] This secular decline is mostly attributed to altered exposure to environmental factors. The current prevalence of PDB in the West is up to 3% in people aged >55 years with a male predominance, and its prevalence increases with age, exceeding 6% after the age of 80 years.[185] Since the first case report of PDB in Chinese in China in 1941 by Snapper,[186] who remarked that PDB was rare in China, an increasing number of cases have been reported in Chinese from various places of the Asia-Pacific region (Table 33.14). Between 1951 and 2015, there were 120 additional cases of PDB reported in Chinese from mainland China,[187-200] Hong Kong,[201-207] Taiwan,[208-212] Singapore,[213-215] Thailand,[216,217] Malaysia,[218] and New Zealand.[219] For these reported Chinese cases of PDB, there was less male predominance, with the overall male-to-female ratio being 1.22:1. The age ranged from 16 and 91 years, and the mean age was 56.8 years. Although the majority were of older age, (63% aged ≥55 years), a significant proportion were middle-aged (34.8% aged 35-55 years), attributed in some to early detection of asymptomatic cases through the use of screening tests during annual medical checkups.[217] However, an early-onset familial form of PDB with genetic mutation has also been reported from Shanghai.[196] Histological confirmation of PDB was obtained in some of the cases reported because malignancy had been entered into the differential diagnosis or surgery was performed for fractures, decompression, or cosmetic reasons.[186,194,200,201,203,204,206,208,209,211,212,216,217] With more awareness among clinicians of PDB in Chinese and of its diagnostic clinicoradiological features, unnecessary investigations will be minimised before the diagnosis is reached. A New Zealand study has reported an increase in the number of Asian patients (two were Chinese) with PDB in Auckland against a background of an apparently decreasing prevalence of PDB in the population of European descent. On a population basis, the prevalence of PDB in the Asian population in Auckland appears to be similar to that in patients of European descent.[219] The finding supports the view that Asians are not genetically protected from PDB and an environmental determinant to the disease exists.[219]

Table 33.14 Number of cases of Paget's disease of bone reported in Chinese from 1941 to 2015[186-219]

Period reported	Total	Mainland China	Hong Kong	Taiwan	Singapore	Thailand	Malaysia	New Zealand
1941-1950	1	1	-	-	-	-	-	-
1951-1970	13	12	-	-	1	-	-	-
1971-1990	11	10	-	-	-	-	1	-
1991-2010	64	32	20	6	5	1	-	-
2011-2015	32	17	9	-	-	4	-	2
1941-2015	**121**	**72**	**29**	**6**	**6**	**5**	**1**	**2**

Table 33.15 Clinical features of Paget's disease of bone

- Asymptomatic: revealed by
  - o high alkaline phosphatase
  - o abnormal X-ray
  - o abnormal bone scan
- Bone pain (5%)
  - o Paget's disease (deep-seated, warmth)
  - o secondary osteoarthritis
  - o osteosarcomatous change (excruciating, <1%)
- Fractures
- Bone deformities
  - o bowed tibia
  - o enlarged skull
- Spinal stenosis
- High-output heart failure
- Visual impairment
- Deafness
- Mental changes
  - o intracranial steal (internal-to-external carotid shunt)
  - o hydrocephalus (basilar invagination)

Regarded as a rare disease among Chinese, PDB may be underdiagnosed in Chinese because of lack of experience with this disease. Also, some of its clinical features (Table 33.15), like bone and joint pain, impaired vision, hearing and mental state, may be mistaken as changes of old age or attributed to other common diseases found in elderly patients. The threshold for investigating the aetiology of a raised alkaline phosphatase in an elderly person may be high, or when investigated, attention may be focused on excluding hepatobiliary or other bone pathologies. When hot spots on bone scan or sclerotic bone lesions are detected in an elderly person, malignancies instead of PDB may be diagnosed. Nonetheless PDB can cause significant morbidity in elderly patients through complications such as fracture, bone pain, secondary OA, deafness, dental problems, and osteosarcoma, but it is a potentially treatable disease.[183,220] Thus, it deserves more clinical attention.

The author has had experience in the diagnosis and management of 23 patients (22 Chinese, one Caucasian) with PDB from 1994 to 2016 in the geriatric clinic of Princess Margaret Hospital in Hong Kong that serves a catchment population of one million. The age at presentation of the 22 Chinese PDB patients ranged from 44-93 years, the mean age was 73.6 years, and the male-to-female ratio was 1:1.2. With the exception of one patient who presented at the younger age of 44 years, all the others (95.5%) presented at the age of ≥55 years, and 27.3% presented at the age of ≥80 years. The authors' colleague Ko[203] has performed a retrospective study of 13 Chinese PDB patients (10 from our geriatric clinic and three from a medical clinic) diagnosed between 1994 and 2000 in Princess Margaret Hospital. The most common presentation in our local series was an incidental finding of raised alkaline phosphatase. Coexistence of both PDB and biliary tract disorder occurred in four of the 13 patients studied, and thus the possibility of a double aetiology of raised alkaline phosphatase should be borne in mind when a biliary tract disorder cannot fully explain the patient's clinical picture (Case 33.5, Plate 33.8). The supposed rarity of this condition in this locality means that an osteosclerotic lesion is often presumed to be due to malignancy and reported as such rather than as PDB, resulting in a biased management plan for the clinician and an undue psychological impact on the patient. Such malignant interpretation of sclerotic bone lesions will likely be made in an elderly person who has known or suspected cancer. In the authors' series of PDB patients, however, there is a patient with prostatic cancer whose osteosclerotic bone lesions are due to PDB, not prostatic bone secondaries (see the case scenario of Learning Manual of Chapter 13). Occasionally, local radiologists have spotted the classic radiological features of PDB (increase in size of affected bones, changes in bone texture with areas of sclerosis and lucency, skeletal deformity), and alerted the clinician to the diagnosis of PDB (Plate 33.9b). The lack of experience of this condition may mean that signs and symptoms of PDB become obvious only retrospectively after investigations, often done for other reasons, reveal the presence of PDB (Cases 33.6, 33.7).

Case 33.5 Unexplained raised alkaline phosphatase after cholecystectomy

A 68-year-old man was referred to our geriatric clinic by a surgical colleague for unexplained raised alkaline phosphatase of 674 U/L at 10 months after cholecystectomy for acute cholecystitis; post-cholecystectomy ultrasound and endoscopic retrograde cholangiopancreatography did not suggest recurrence of his biliary tract disorder. The heat stability index was 0.259, suggesting a possible bone source for his raised alkaline phosphatase. His history revealed intermittent backache and knee pain for 7 years. Subsequent bone scan and X-ray revealed multiple sites of increased bone uptake (Plate 33.8a) and osteosclerotic lesions (Plate 33.8c) suggestive of polyostotic Paget's disease of bone (PDB). A bone biopsy of his pelvis, done for suspicious malignant change, confirmed PDB (Plate 33.8d). Both his serum alkaline phosphatase level and bone scan improved after intravenous pamidronate infusions (Figure 33.5 patient A, Plate 33.8b).

Case 33.6 Polyostotic Paget's disease of bone in a 78-year-old man with increasing size of skull and neck

A local Chinese man had polyostotic Paget's disease of bone diagnosed in 1985 at the age of 63 years when he presented with neck pain with drooped head, hip pain, and knee pain (Plates 33.9a, 33.9b, 33.9c). Retrospective history checking showed that since the age of 55 years, he needed progressively increasing sizes for his pullovers, underwear and waist belts, and his friends commented on increased warmth over areas of his body with bones affected by Paget's disease. He needed increasingly large pullovers and underwear because his skull and pelvis, which were affected by the Paget's disease process, were growing markedly in size. This was also reflected by the increasing size of his neck collar (Plate 33.9d). He also had knee pain, due to associated degenerative arthritis in joints adjacent to affected bones. Both his serum alkaline phosphatase level and bone scan improved after intravenous pamidronate infusions (Figure 33.5 patient E).

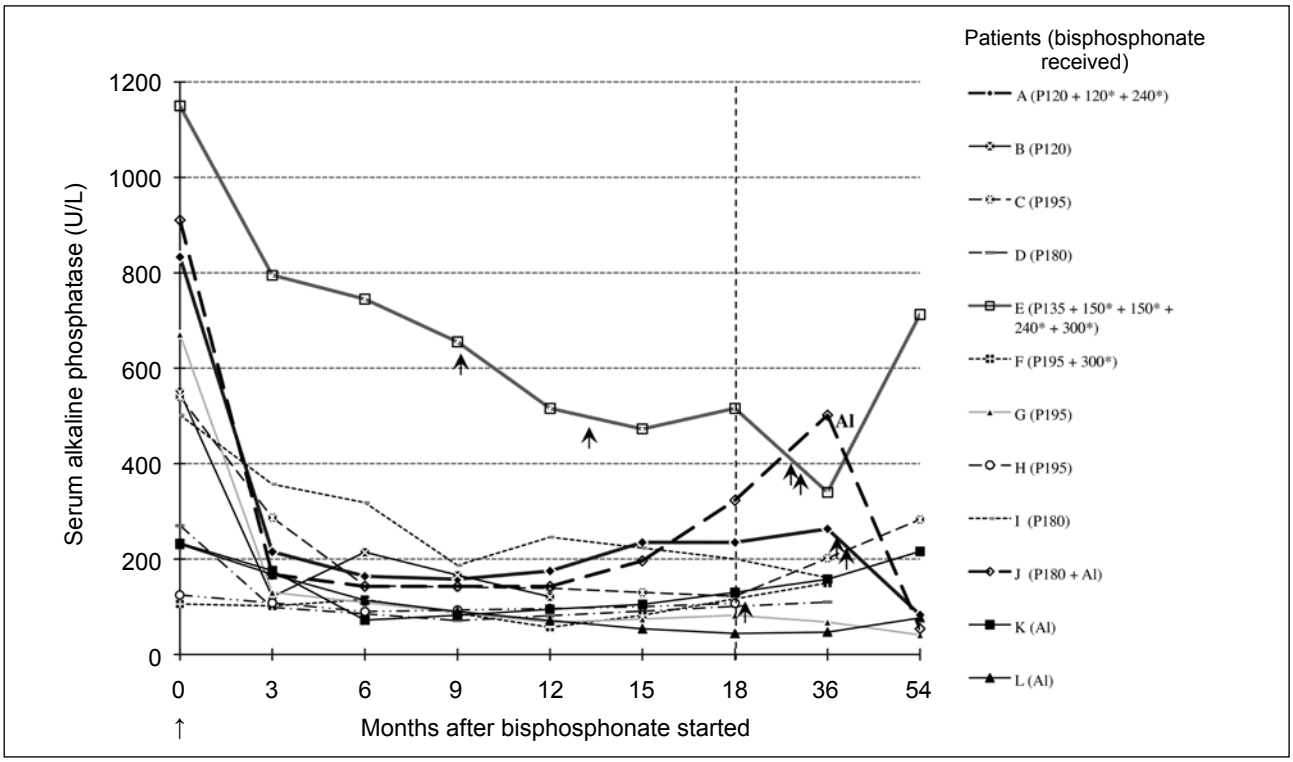

Figure 33.5 Changes in serum alkaline phosphatase in 12 Chinese patients (A to L) with Paget's disease of bone after treatment with bisphosphonates initiated at "↑". Patients A to L were given intravenous infusion of pamidronate (P, number is dose in mg) initially, with additional courses of pamidronate (*) for patients A, E, F indicated at ↟, and additional oral alendronate (AI) course for patients A, E, G, J in the later course of their illnesses (shown for J only). Patients K and L were given oral AI initially; additional AI course at 7 years and intravenous zoledronate at 10 years were given for patient K for relapsed pagetic activity (not shown)

Case 33.7 Incidental finding of raised alkaline phosphatase in a 73-year-old woman with stroke

A 73-year-old woman with a history of diabetes mellitus, hypertension, cholecystectomy, and right nephrectomy was hospitalised in 1994 for stroke. A raised serum alkaline phosphatase of 550 U/L, with normal serum calcium and phosphate, was detected incidentally. Workup (bone scan, X-ray) confirmed Paget's disease of bone affecting her skull. Frontal bossing of her skull was obvious when her serial photos were reviewed (Plate 33.10). It is of interest to speculate in her case whether intracranial steal (internal-to-external carotid shunt) might have played a role in the aetiology of her stroke in addition to her usual vascular risk factors of diabetes and hypertension. Her alkaline phosphatase declined to 107 U/L after she was treated with intravenous pamidronate infusions (Figure 33.5 patient B).

Treatment of PDB is indicated if the symptoms of the patient are due to Paget's disease or one of its complications. Nonetheless pain may be due to the complicating osteoarthritic process and should be treated by conventional measures. Asymptomatic active disease of the skull or vertebrae also warrants treatment to prevent potentially serious neurological complications such as nerve compression radiculopathy, brain stem or spinal cord compression. Treatment of asymptomatic active disease of the long bones is also recommended to reduce the risk of deformity, fracture, and arthritis.[221] Bisphosphonates, via their antiresorptive effect, decrease bone pain, reduce the activity of PDB, improve quality of life, normalise bone turnover, and heal lytic lesions on radiographs.[221] Etidronate, the first bisphosphonate used to treat PDB in the 1970s, has now been replaced by the more effective second-generation bisphosphonates because of its lower efficacy and impaired mineralisation with prolonged use. Pamidronate, given by weekly infusion of 30 mg for 6 weeks, or by three 2-weekly infusions of 60 mg, has produced biochemical remissions lasting 2-3 years.[222] Pamidronate has now been superseded by more potent aminobisphosphonates. Alendronate, given orally 40 mg daily for 6 months, has been widely used in PDB over the last decade, the 6-month courses being repeated as necessary at intervals of 2-5 years. Risedronate, given orally 30 mg daily for 2 months, normalises alkaline phosphatase in 73% of patients. Zoledronate, given as one single intravenous infusion of 5 mg over 15 minutes, can achieve

high rates of biochemical remission and sustain a long duration of suppression, with persistence of a treatment response for up to 6.5 years in 87% of patients.[221,223] Gastrointestinal irritation is a limiting side-effect of the oral bisphosphonates. Intravenous bisphosphonates often cause a transient acute-phase response with bone pain, myalgia, headache, nausea, pyrexia, and fatigue within 1-3 days of infusion. These symptoms can be reduced if paracetamol is given before and for a few days after the infusion. Hypocalcaemia may also occur with intravenous bisphosphonates, particularly in patients at risk of vitamin D deficiency. The risk can be minimised by correcting vitamin D deficiency before treatment and providing calcium and vitamin D supplements after the infusion. Since intravenous bisphosphonates are potentially nephrotoxic, renal function should be assessed before dosing, and zoledronate should not be administered if the glomerular filtration rate is <30 ml/min.

Efficacy of and experience in the use of bisphosphonates in treating PDB among Chinese patients have also been reported; the agents used included clodronate,[208,211] pamidronate,[193,195,198,203-206,217] alendronate,[204,217] ibandronate,[196,199,217] and zoledronate.[197-200,206,217] The author has been treating PDB in local Chinese patients with intravenous pamidronate infusion since 1994, oral alendronate since 2003, and zoledronate infusion since 2014. Improvement in terms of reduced or normalised alkaline phosphatase (Figure 33.5), and reduced disease activity as revealed by bone scan has been observed in all the treated patients without adverse effects other than occasional instances of transient asymptomatic hypocalcaemia. Of the 12 patients treated, additional courses of bisphosphonates have been required in six for relapsed pagetic activity during follow-up.

33.6.2 GCA and PMR

33.6.2.1 Diagnosis and Management of GCA and PMR

PMR and GCA are two common related disorders that affect elderly people in the West. The rarity of these disorders among Chinese may pose a diagnostic challenge to local clinicians, especially when an elder presents with non-classic systemic symptoms (Cases 33.8, 33.9). In an elder with GCA, the single disease underlying the multi-system presentation may be missed initially, and the afflicted elder may be simultaneously under the care of several organ specialists tackling the respective organ affected by the arteritic process (Case 33.10).[224] GCA may be overlooked when the presentation is that of a stroke,[225,226] the aetiology of which may be attributed to the more common atherosclerosis rather than the rarer condition of GCA (Case 33.9). Mulley[227] has rightly said, "If we are to manage our elderly patients effectively we must ensure that we do not overlook those conditions that can be modified or prevented by treatment. If the diagnosis of GCA is delayed or missed the patient may become blind, have severe pain, be subjected to inappropriate investigations, and even undergo unnecessary operations." A correct early diagnosis of PMR / GCA and the institution of appropriate treatment with steroids is thus of utmost importance when elderly people begin to fail mentally or physically.

Case 33.8 Headache, dizziness, and falls with fever of unknown origin and anaemia in a 69-year-old Chinese

A 69-year-old man was admitted to the geriatric department of Princess Margaret Hospital in 1984 via the Accident and Emergency Department with a 1-week history of headache, dizziness and fall, on top of a 2-year history of recurrent dizziness, blurred vision, and frequent falls. He ran a fever of unknown origin for 4 weeks after hospitalisation with negative workup for sepsis and tuberculosis. Investigations revealed normochromic normocytic anaemia (haemoglobin, 8.6 g/dL), a high erythrocyte sedimentation rate (ESR) of 140 mm/h, hyperglobulinaemia, impaired liver function (reversed albumin-globulin ratio, raised alkaline phosphatase), and an aneurysm of the ascending aorta. His temporal arteries were palpable and were neither thickened nor tender. No bruits were detected. Temporal artery biopsy revealed fragmentation of the internal elastic lamina and partial occlusion of the lumen consistent with temporal arteritis. He showed dramatic improvement with prednisolone treatment: his headache and fever subsided, haemoglobin level rose from 8.6 to 12.1 g/dL, ESR fell from 140 to 30 mm/h, alkaline phosphatase decreased from 125 to 88 U/L, albumin improved from 29 to 33 g/L, and globulin declined from 48 to 31 g/L.

Case 33.9 Fever and anorexia in a 72-year-old Chinese with a history of recurrent strokes and headache

A 72-year-old man with a history of hypertension and recurrent ischaemic strokes was admitted to the geriatrics department for fever, reduced appetite, dizziness, and reduced mobility. He ran a fever of unknown origin for 3 weeks after hospitalisation with negative workup for sepsis, immunological / myeloma screen. Investigations revealed normochromic normocytic anaemia (haemoglobin 11.7 g/dL), and a high erythrocyte sedimentation rate of 125 mm/h. Further history revealed intermittent right frontal headache, dizziness, blurred vision, and right-sided hearing impairment

for the past 3 years. Examination revealed left carotid bruit and left hemianopia. A temporal artery biopsy confirmed the presence of giant cell arteritis (GCA). Although he had hypertension as a risk factor for stroke, it was possible that his recurrent strokes were manifestations of GCA.

Case 33.10 Earache, toothache, and diplopia in an 86-year-old Chinese[224]

An 86-year-old man was brought to the geriatric clinic for advice, both the patient and his family being distressed after multiple investigations and interventions over 6 months by specialists (ear, nose and throat surgeon, dental surgeon, ophthalmologist) for fluctuating earache, deafness, toothache, insomnia (because of pain), transient diplopia, dysphagia, hoarseness, dysphonia, cough, constipation, weakness, weight loss, and reduced appetite had yielded no benefit. He underwent excision of a left aural polyp and extraction of seven teeth, but the earache and toothache remained. Giant cell arteritis was suspected because of a history of improvement of earache after computed tomographic scan under contrast (when he was given steroid cover), fluctuating facial pain with nocturnal exacerbation, the possibility of swallowing claudication as the cause of his dysphagia, a raised erythrocyte sedimentation rate (ESR) of 122 mm/h, and anaemia (haemoglobin 11.2 g/dL). Temporal artery biopsy was suggested but declined. Steroid was suggested but not complied with for fear of side-effects. Sixteen months later, he developed right-sided temporal headache, diplopia, and ptosis. Examination revealed right 3rd, 4th, and 6th cranial nerve palsy and right Horner's syndrome. Haemoglobin then dropped to 8.6 g/dL (normochromic normocytic), and ESR was 46 mm/h. Prednisolone was initiated with improvement of headache, earache and ptosis, rise in haemoglobin from 8.6 to 10.6 g/dL, and fall in ESR from 46 to 5 mm/h.

Although the classic myalgic and arteritic presentations of PMR / GCA are well described in textbooks, the diagnosis of PMR / GCA is often overlooked when GCA masquerades as cachectic, febrile, anaemic, aneurysmal (e.g. aortic regurgitation, ruptured aortic aneurysm), or occlusive (e.g. stroke, intermittent claudication, myocardial ischaemia) forms.[228] Failure to recognise these non-classic 'masked' presentations of GCA may result in unnecessary invasive investigations, delayed diagnosis, and consequent morbidity and mortality.[228] On the contrary, other diseases, e.g. neoplasia (particularly lymphomas), may simulate GCA and should therefore be suspected when there is no clinical response to steroid therapy.[229-231]

There is a lack of prospective studies on the applicability of the commonly used criteria for classifying or diagnosing GCA and PMR in elderly patients with multiple aetiologies and pathologies.[232] Diagnosis of GCA is based on clinical, laboratory and imaging findings, and is confirmed by the histology of temporal artery biopsy. When a temporal artery biopsy is negative, there are no independent criteria to determine whether GCA is present. The ACR criteria for classification of GCA[233] may assist in GCA diagnosis (Figure 33.6), but their validity as diagnostic criteria for GCA has been questioned[234] and cautioned[235] when used for diagnosing individual cases. Colour Doppler (or duplex) ultrasonography of superficial arteries, such as the temporal artery, has been suggested as a newer, non-invasive method to diagnose temporal arteritis. A periluminal hypoechoic halo representing vessel wall oedema is said to be diagnostic of GCA,[236] but the results of studies examining its diagnostic value vary considerably.[237-239]

The diagnosis of PMR is based on clinical findings, laboratory evidence of an acute phase reaction, exclusion of other diseases, and response to glucocorticosteroids.[240] There is no diagnostic test for PMR, but diagnostic and classification criteria have been suggested by Bird et al,[241] Jones and Hazleman,[242] and others. Imaging studies such as ultrasonography and magnetic resonance imaging typically show a predominantly periarticular inflammatory process (synovitis, bursitis, and tendinitis) in the shoulders and hips, and lend support to a diagnosis of PMR, although the findings are non-specific and seen also in other inflammatory forms of arthritis.[243] A set of provisional classification criteria for PMR (Figure 33.6) was published in 2012 by the European League Against Rheumatism and the ACR.[244] With these criteria, a score of ≥4 had a sensitivity of 65% and a specificity of 78% for discrimination of PMR from other disorders. Addition of ultrasound improved specificity, with a score of ≥5 providing 66% sensitivity and 81% specificity for classification of PMR.[244] Isolated PMR at presentation may evolve into GCA or RA subsequently.[245-247]

Prospective studies are lacking on the appropriate steroid regimen for PMR / GCA to minimise the long-term steroid side-effects to which elders are particularly vulnerable.[232,248] Based on current evidence, a management algorithm for diagnosis and treatment of PMR / GCA in geriatric patients is summarised in Figure 33.6.[228,232,233,244,248,249] Careful follow-up is required to monitor the patient for any adverse effects of chronic steroid use: infectious complications, cataract (often worsening of an existing cataract), diabetes mellitus (new diabetes or poor glycaemic control in existing disease),

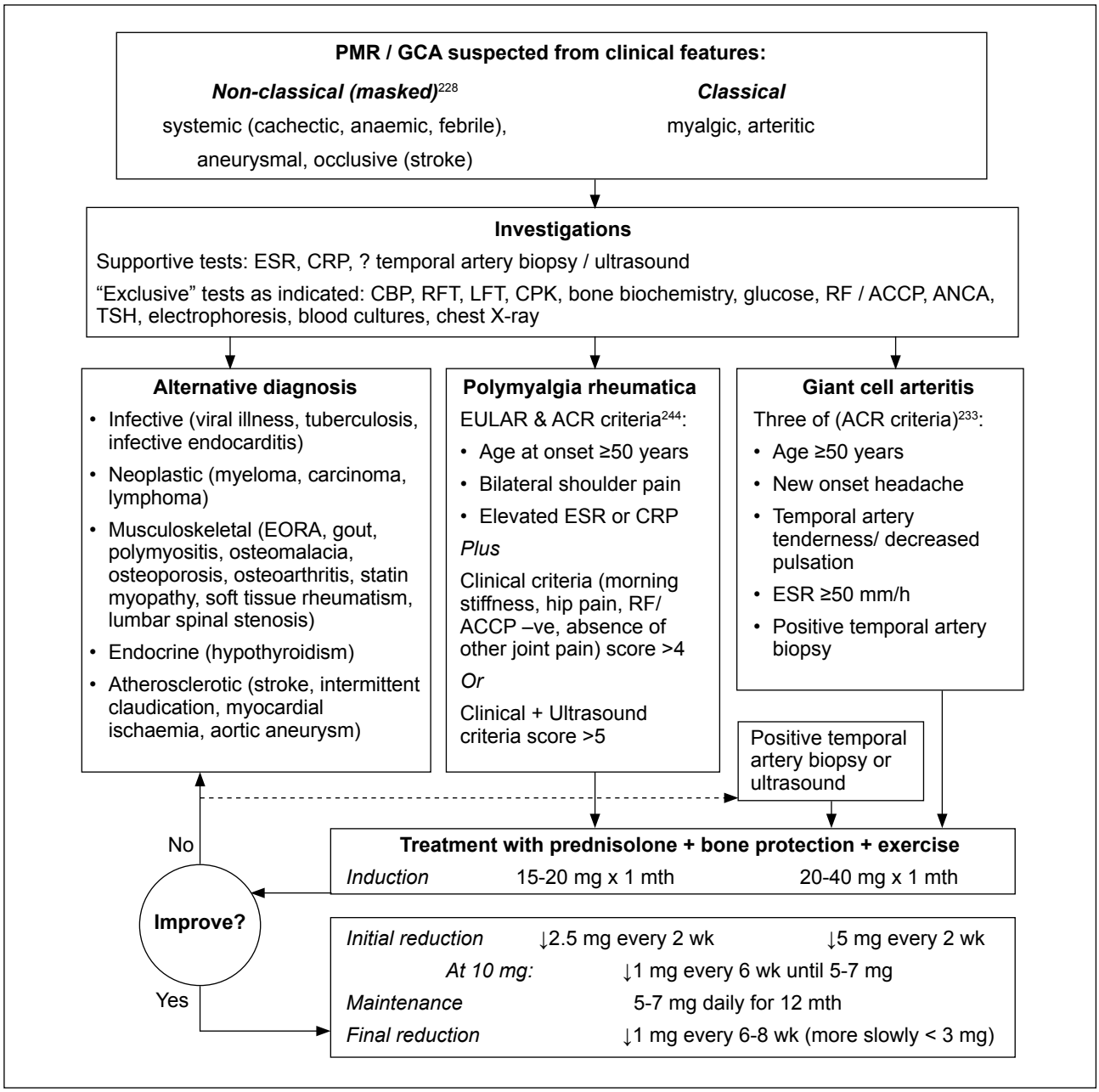

Figure 33.6 Management algorithm for diagnosis and treatment of PMR / GCA in geriatric patients[228,232,233,244,247,249]
Abbreviations: ACCP = anticyclic citrullinated peptide antibodies; ACR = American College of Rheumatology; ANCA = antineutrophil cytoplasmic antibodies; CBP = complete blood picture; CPK = creatine phosphokinase; CRP = C-reactive protein; EORA = elderly-onset rheumatoid arthritis; ESR = erythrocyte sedimentation rate; EULAR = European League Against Rheumatism; GCA = giant cell arteritis; LFT = liver function test; mth = month(s); PMR = polymyalgia rheumatica; RF = rheumatoid factor; RFT = renal function test; TSH = thyroid-stimulating hormone; wk = week(s)

hypertension, myopathy, osteoporosis, and increased risk of falls and fractures.[250] To reduce the adverse effects of prolonged steroid use, adjuvant treatments for steroid-induced osteoporosis, regular exercise, and appropriate immunisations should be part of the management. The efficacy of methotrexate and other immunosuppressants in reducing relapses and steroid use (steroid-sparing effect) has been studied but with conflicting results.[237,243] Low-dose aspirin may be beneficial in reducing ischaemic complications in GCA.[237,240,251]

33.6.2.2 GCA and PMR in Chinese

The incidence rates of GCA per 100 000 people aged >50 years has been quoted as varying between 1 and 30 in the West, with the highest rates from Northern Europe and populations of predominantly Scandinavian descent.[252] Similar to GCA, the highest incidence rates of PMR were observed in Northern Europe; the rate of PMR quoted in the West being 3-110 per 100 000 population older than 50 years.[243]

GCA has been regarded as a rarity in Asians, roughly

estimated as 20 times less common in Asian than Caucasian patients.[253] Among Chinese, there have been a few isolated case reports of GCA from China,[254-257] Hong Kong,[258,259] Singapore,[260] and the US[261,262] in the western literature. In addition, there have been three retrospective studies of GCA in Chinese from China[263] and Hong Kong,[204,264] one prospective study of GCA in China,[230] and two retrospective studies of PMR in China[265] and Hong Kong.[266]

The findings of the four studies on GCA in Chinese[204,230,263,264] are compared with the western study by Hunder[267] in Table 33.16. The study by Sun et al[263] on 70 patients with GCA diagnosed between 1990 and 2014 in Peking Union Medical College Hospital (PUMCH) is the largest series of Chinese GCA patients ever published. Only 70 inpatients were admitted and diagnosed with GCA during the study period of 23 years in PUMCH, compared with 688 admissions for Takayasu's arteritis during the same period, confirming the impression that GCA is a rare disease in Chinese.[263] Tam and Wong[264] have performed a retrospective case record review of Chinese GCA diagnosed in public hospitals in Hong Kong from 1996 to 2000. There were 19 biopsy-proven GCA cases over the 4-year period of study, giving an estimated annual incidence rate of 0.34 biopsy-proven GCA per 100 000 people aged >50 years in Hong Kong.[264] The patients in Hu et al's prospective study[230] were relatively young (mean, 43.13 years; range, 28-60 years; 81.25% under age 50 years) with a marked male predominance (male:female = 15:1), for which no explanation could be offered. It is noteworthy that one 73-year-old man who fulfilled all of the five ACR criteria (including positive temporal artery biopsy) and was originally classified as having GCA, deteriorated despite steroid treatment, and was later diagnosed with lymphoma.[230]

The author has had experience in the diagnosis and management of nine patients (five females, four males) with GCA among local Chinese in the Geriatrics Department of Princess Margaret Hospital in Hong Kong ever since he diagnosed the

Table 33.16 Clinical features of giant cell arteritis: Chinese[204,230,263,264] versus Americans[267]

	Sun et al[263]	Hu et al[230]	Kong[204]	Tam and Wong[264]	Hunder[267]
Years of study	1992-2014	1999-2001	1984-2003	1996-2000	1980s
Setting	Rheumatology department, China	Neurology department, China	Geriatrics department, Hong Kong	Public hospitals, Hong Kong	Rheumatology department, US
Diagnostic / classification criteria	Clinical, biopsy, PET-CT (96% fit ACR[231])	Clinical, biopsy, steroid response (81% fit ACR[231])	Clinical, biopsy (100% fit ACR[231])	Clinical, biopsy (100% fit ACR[231])	-
Positive temporal artery biopsy (%)	45.7	68.75	33	100	-
No. of patients	70	16	9	19	100
Female:male	1:1	0.067:1	1.25:1	2.2:1	2.2:1
Mean age (range / distribution) [years]	65.2 (43-83)	43.13 (28-60)	75.1 (69-85)	84% >70 years, 47% >80 years; (61-89)	
Arteritic (%)					
Headache	68.6	93.75	67	79	68
Facial pain / numbness	-	-	78	-	-
Abnormal / tender temporal artery	30	81.25	11	32	46
Claudication of jaw / tongue / swallow	30	12.50	33	32	45 / 6 / 8
Visual symptoms	38.6	56.25	44	32	16
Myalgic (%)					
Proximal myalgia, stiffness, tenderness	27.1	6.25	56	42	39
Systemic (%)					
Fever	57.1	25	67	26	42
Weight loss / anorexia	50	-	44	} 37	50
Malaise / fatigue / weakness	47.1	12.5	44		40
Anaemia (%)	72.9	-	56	79	-
Mean ESR (range / distribution) [mm/h]	92.9 95.7% >50 35.7% >100	- Raised in 68.75%	118.2 (97-144)	104 89% >50 63% >100	-

Abbreviations: ACR = American College of Rheumatology; ESR = erythrocyte sedimentation rate; PET-CT = positron emission tomography–computed tomography

first case in 1984 (Case 33.7, Table 33.16). The age at diagnosis ranged from 69 to 85 years (mean, 75.1 years). All improved dramatically with the initiation of steroid treatment. Five of the nine patients with GCA consented to temporal artery biopsy, yielding positive results in three. Although the classic combination of new headache (especially when the history suggested this being worse at night and on exposure to coldness) and a markedly raised ESR in an elderly patient alerted the author to the possible diagnosis of GCA, there was quite a high percentage of GCA patients (78%) in the author's series who complained of facial pain (earache, toothache, jaw pain); such patients had been managed initially by other clinicians (ear, nose and throat surgeons, dental surgeons, neurologists) as having problems with their ears, dentition, or as trigeminal neuralgia. It has been emphasised that the absence of headache or its presence beyond or excluding the temple should not eliminate the diagnosis of GCA from consideration.[268] Non-classic presentations such as fever, anaemia, and weight loss were also common in the author's series; such presentations have led to over-investigations for occult infection or malignancy, especially in a locality where GCA is uncommon and thus not the first diagnosis to consider. The diagnosis and treatment of GCA in this locality is also complicated by the prevalence of tuberculosis among elders in Hong Kong. Tuberculous rheumatism (Poncet's disease), a controversial entity described in people of Asian, African and Caribbean descent,[269,270] may mimic PMR / GCA in symptomatology. In a review of rheumatic diseases in China, Chang[254] commented that 15% of patients with primary arteritis of the aorta and its main branches (pathologically considered by some as one variant of GCA) had a history of tuberculosis prior to onset of the arteritis and suggested that the condition in China may be closely related to tuberculosis. In the author's series of nine patients with GCA, two had treated tuberculosis (lung, pancreas) and three had evidence of old tuberculosis on chest X-ray prior to the diagnosis of GCA. Long-term steroid treatment may also increase the chance of reactivation of tuberculosis in elderly people. Of the nine GCA patients treated with steroid, two subsequently developed tuberculosis (one microbiologically confirmed, one of clinical tuberculosis). Thus, careful workup to exclude active tuberculosis before diagnosis of GCA and initiation of steroid treatment is important in this part of the world. Once a patient with GCA is started on steroid therapy, regular monitoring for the emergence of

Table 33.17 Clinical features of polymyalgia rheumatica: Chinese[265,266] versus Europeans[271]

	Tang[265]	Li et al[266]	Bird et al[271]
Years of study	1987-1997	1997-2008	1998-2001
Setting	Rheumatology department, China	2 Regional hospitals, Hong Kong	8 Rheumatology centres in 8 European countries
Diagnostic / classification criteria	Clinical[242]	Clinical	Clinical (99.5% fit in Bird et al's study[241])
No. of patients	18 (5 Biopsy positive GCA)	44 (12 RA)	213
Female:male	2:1	2.4:1	-
Mean age (range / composition) [years]	61.3 (43-86)	75.8 (75% >65)	100% >50 80.2% >65
Arteritic (%)			
Headache	50	-	-
Facial pain / numbness	6	-	-
Abnormal / tender temporal artery	28	-	-
Claudication of jaw / tongue / swallow	6	-	-
Visual symptoms	6	-	-
Myalgic (%)			
Bilateral shoulder pain	61	95.5	90.8
Bilateral shoulder stiffness	56	95.5	90.8
Bilateral tender upper arm	-	11.4	75.5
Stiffness >1 hour	-	68.2	84.9
Onset of illness <2 weeks	-	29.5	75.5
Arthralgia	33	-	-
Systemic (%)			
Fever	61	-	-
Weight loss / anorexia / depression	67	29.5	40
Malaise / fatigue / weakness	100	-	-
Anaemia (%)	67	-	-
Mean ESR (range / composition) [mm/h]	103 (50-160)	100% >40 56.8% >100	95.7% >40

Abbreviations: ESR = erythrocyte sedimentation rate; GCA = giant cell arteritis; RA = rheumatoid arthritis

tuberculosis is essential. In summary, the rarity of GCA among Chinese and the multiple faces of GCA have two important clinical implications locally. First, we should not miss GCA as a possible treatable cause of distressing pain and functional decline in an elder not improving with treatments based on other presumed diagnoses. Second, if an elder is not improving while being treated with the appropriate steroid regimen for GCA, alternative diagnoses should be seriously considered even though a positive temporal artery biopsy has been obtained.

The two studies on PMR in Chinese by Tang[265] in China and Li et al[266] in Hong Kong are compared with a European series[271] of PMR in Table 33.17. Tang's clinical analysis of 18 cases of PMR in China over a 10-year period consisted of five biopsy-proven GCA.[265] The age of onset of PMR in his series ranged from 43 to 86 years (mean, 61.3 years), and there was a female predominance (female:male = 2:1). All showed clinical and laboratory improvement within 1 month of steroid therapy.[265] Li et al's series of Chinese PMR were older with a mean age of 75.8 years. Most patients (39 of 44, 88.6%) were treated with prednisolone at a mean dose of 17.2 mg with a rapid response as shown by a drop in ESR by over 50% from baseline within 2-3 months of treatment. Of the 44 patients, 12 (27.3%) evolved into RA after a median of 2 months (range, 1-24 months) from onset of PMR.[266]

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CHAPTER 34

PARKINSONISM

Francis Chun-keung MOK

OBJECTIVE

To learn the presenting features, diagnosis, differential diagnoses, and management of Parkinson's disease and related disorders, with special geriatric considerations.

KEY POINTS

- Parkinson's disease is a neurodegenerative disorder with four cardinal features; special features of older patients should be noted.
- Early treatment with L-dopa is safe and improves patient's symptoms; education and psychosocial support are important for improving patient outcomes.
- The long-term prognosis of the disease is less pessimistic than before; various pharmaceutical treatments can improve the late-stage symptoms; neurosurgical treatment is also available in local clinical practice.

34.1 Introduction

Parkinson's disease (PD), first described by James Parkinson (1817), is considered the third commonest neurological disorder causing chronic disability in older people, after stroke and dementia. The incidence is estimated to be 20 in 100 000 population. The mean age of onset is 65 years and its prevalence increases with age. The prevalence of PD in China for those aged >65 years is 1.7%,[1] similar to that reported in developed countries, and the age-specific prevalence increases with age for both sexes (Figure 34.1).

PD is a neurodegenerative disorder characterised by four cardinal features:

- tremor (resting, 4-6 Hz);
- rigidity (cogwheel);
- bradykinesia; and
- postural instability (not explained by other causes such as sensory deficits, motor weakness, etc.)

The primary pathology of PD is the progressive loss of cells producing the neurotransmitter dopamine from the substantia nigra in the brainstem. External insults and genetic defects are considered possible causes. There was a theory that all these affect a common pathway, the ubiquitin-proteasome system, leading to the production of Lewy bodies (intracellular cytotoxic proteins) and progressive neuronal death.

34.2 Diagnosis

Some conditions lead to features similar to PD, which are grouped under the name of secondary parkinsonism (Table 34.1). Some other rare neurodegenerative disorders can also mimic PD (Table 34.2). The differential diagnoses of tremors that are commonly seen in elderly people are shown in Table 34.3.[2]

The UK NICE (National Institute for Health and Care Excellence) guideline for PD recommends the use of UK PD Brain Bank diagnostic criteria in routine clinical practice.[3] Parkinsonian syndrome is diagnosed in the presence of bradykinesia and at least one of muscular rigidity; 4-6 Hz resting tremor; or postural instability (not caused by primary visual, vestibular, cerebellar or proprioceptive dysfunction). Exclusion and supportive inclusion criteria are then used to distinguish between primary PD and secondary parkinsonism.

34.2.1 Special Diagnostic Features of PD in Older People

- Initial presentation can be asymmetrical; one may easily misdiagnose an early PD patient as having stroke because of unilateral clumsiness of the hand. The presence of cogwheel rigidity and no definite weakness is a clue for diagnosing

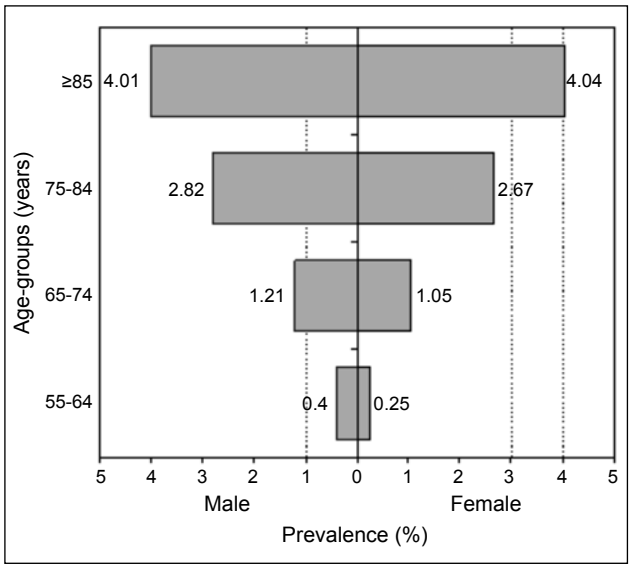

Figure 34.1 Age- and sex-specific prevalence of Parkinson's disease in a community-based cross-sectional study in 29 454 adults aged ≥55 years in three regional centres in China in 1997-1998 (drawn using data from Zhang et al[1])

Table 34.1 Causes of secondary parkinsonism

• Drug-induced parkinsonism (e.g. antipsychotics or neuroleptics, prochlorperazine [stemetil])
• Vascular parkinsonism
• Toxins (e.g. MPTP [1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine], pesticides, herbicides, carbon monoxide poisoning)
• Encephalitis
• Head trauma
• Tumours

Table 34.2 Neurodegenerative diseases mimicking Parkinson's disease

• Dementia with Lewy bodies
• Progressive supranuclear palsy
• Multiple system atrophy (formerly Shy-Drager syndrome; olivopontocerebellar atrophy, striatonigral degeneration syndrome)
• Alzheimer's disease — end stage
• Creutzfeldt-Jakob disease
• Gait dyspraxia
• Normal pressure hydrocephalus

PD. Unilateral PD often progresses to involve the 'asymptomatic' side within 1 to 2 years. Strictly unilateral features (unilateral parkinsonism) after 3 years make primary PD unlikely and may occur in vascular parkinsonism.

- Vascular parkinsonism is an important cause of secondary parkinsonism in elderly people, but its clinical manifestations are heterogeneous, including lower-body parkinsonism (prominent gait disorder, absent or atypical resting tremor, pyramidal signs, poor L-dopa response); parkinsonism indistinguishable from primary PD (reported in basal ganglia lacunar infarcts, with L-dopa responsiveness); unilateral parkinsonism; progressive supranuclear palsy-like syndrome; and overlaps syndrome with coexistent Lewy body and cerebrovascular pathology.[4,5]
- Despite the bradykinesia and postural instability, PD patients seldom present with falls in the early stage. Such patients are more likely to have other neurodegenerative disorders with parkinsonism such as progressive supranuclear palsy. PD should be borne in mind, however, when assessing elderly patients with unexplained injurious falls.
- Patients with essential tremor are easily misdiagnosed as having PD. The higher tremor frequency, absence of rigidity and bradykinesia point to the former, which is very different from PD in terms of management and prognosis.
- Beware of multiple pathologies developing at different times in the same elderly person. For example, an elderly person with pre-existing senile essential tremor or tremor of hyperthyroidism may develop parkinsonian tremor, which may not be recognised as a new problem. Onset of new functional impairment and other associated features of PD should point to the correct diagnosis.
- Dementia is not a common early feature of PD, and this should raise the suspicion of dementia with Lewy bodies.
- Besides the cardinal features of PD, the following features are useful clues to the diagnosis: decrease in arm swing during walking, shuffling gait that worsens on turning, mask-like face, bent posture, constipation, drooling of saliva, and dramatic

Table 34.3 Differential diagnoses of hand tremors in older patients[2]

Type of tremor	Cause	Characteristics
Resting tremor	Parkinsonism	4-6 Hz, pill-rolling of the fingers; can involve other parts of body
Postural tremor (in outstretched hands)	Physiological (normal)	
	Exaggerated physiological (e.g. thyrotoxicosis)	8-12 Hz, usually involve hands
	Essential tremor	4-9 Hz, hands and head, family history present, slowly progressive, usually not at rest, can be disabling
	Parkinsonism	Resting tremor that persists with maintained posture
	Others (e.g. dystonia, Wilson's disease)	
Kinetic tremor	Cerebellar	3-5 Hz, dysmetria

response to L-dopa treatment.
- Sensory symptoms and other non-motor symptoms are common in elderly patients with PD (see Chapter 34.3.3) and should not distract one from the diagnosis of PD. Non-motor symptoms (e.g. hyposmia, constipation) can also occur before the onset of motor symptoms in PD.

34.2.2 Early Recognition of PD

The pathology of PD was mainly shown in the striatum and its connections to the midbrain nuclei, especially, the substantia nigra. It has been demonstrated that over 70-80% of the dopaminergic neurons are already lost when the clinical features of PD occur. Thus, methods for recognition of the disease in its preclinical stage and interventions to arrest the degenerative process are topics under vigorous medical research.

34.3 Management

To date, there is no definite cure for PD. Nearly all treatments serve to reduce symptoms. The natural course of the disease does not appear to be slowed down or reversed with the available drugs. Whether good management can reduce mortality remains controversial. A 10-year longitudinal follow-up community study of 121 PD patients (mean age, 70 years) in the UK[6] showed that the prognosis of PD is heterogeneous: 55% of PD patients died, 46% developed dementia, 68% had postural instability by 10 years from diagnosis but a 23% had good outcome, with preserved mobility and cognition. The mortality was not increased compared with the general population. Most PD death was not directly related to PD.[6] Another 10-year prospective cohort study of 171 Chinese PD patients (mean age, 62.2 years; younger compared with the UK study) in a movement disorder clinic of a regional hospital in Hong Kong[7] showed that after a mean follow-up of 11 years, 29% died, 49% developed dementia, 59% had falls, 34% had postural hypotension, and 60% had dysphagia. The 10-year mortality ratio of the cohort was not significantly different from the general population of Hong Kong. Despite that 27% were institutionalised, 32% still lived and functioned independently. It was found that older onset, postural instability gait disorder type, and dementia had a negative impact on survival.[7] The commonest cause of death in both studies was pneumonia (33% and 64% of deaths in the UK[6] and Hong Kong[7] studies, respectively), likely related to aspiration pneumonia and reduced ability to clear secretions due to weakened cough in PD patients. A nationwide population-based cohort study of 2001 PD patients in Taiwan[8] with a mean follow-up of 5.8 years identified older age (>80 years), male sex, chronic heart failure, and chronic kidney disease as risk factors for hospitalisation with pneumonia in PD patients. However, treated dental caries reduced the risk of hospitalisation with pneumonia in PD patients.[8] Attention should be paid to oral hygiene and dental health in older PD patients in the prevention of pneumonia.

The patient usually responds well to drug treatment in the first few years. Subsequently, there is development of motor complications such as peak-dose dyskinesia, end-of-dose akinesia / freezing, painful dystonia, etc. Consultation with specialists will be needed to manipulate the medication combination and dosing to improve symptom control. The long-term prognosis is now less pessimistic because of better treatment modalities and supportive measures. The different classes of drugs used in PD are discussed below.

34.3.1 Drug Treatment

34.3.1.1 L-dopa (dopamine precursor) Plus Dopa-decarboxylase Inhibitor

This class of drugs is the mainstay of drug treatment (e.g. Sinemet and Madopar).
- These drugs improves all the symptoms of PD, especially the akinetic symptoms.
- Treatment is associated with prolongation of survival of PD patients.
- Previous worries about early treatment with L-dopa leading to worsening of the long-term prognosis of PD is not supported by evidence.
- Elderly patients should be started with smaller doses; slower titration is advised to prevent psychiatric side-effects. For example, start with Sinemet or Madopar 62.5 mg twice a day and gradually step up to Sinemet or Madopar 250 mg 4 times a day.
- Common adverse effects include nausea, anorexia, postural hypotension, confusion, psychosis, and dyskinesia.
- Modified-release preparations such as Sinemet CR or Madopar CR are available, which may reduce the frequency of dosing and cause less dyskinesia.
- L-dopa should not be stopped abruptly to prevent the rare occurrence of parkinsonism-hyperpyrexia syndrome.[9]
- Patients taking higher doses of L-dopa should take it 30 minutes before meals to improve absorption.

34.3.1.2 Dopamine Agonists

- Examples are bromocriptine, pramipexole, ropinirole (Requip), rotigotine (Neupro), and pergolide (withdrawn from market).
- These drugs act directly on the surviving dopamine receptors.
- They have similar efficacy as L-dopa but are more expensive; they have synergistic effects with L-dopa.
- They are sometimes useful in reducing 'off' periods and in ameliorating motor fluctuations in the later

stage of PD; ropinirole (a D2 receptor agonist) is supposed to delay the incidence of dyskinesia and has been recommended for younger patients (age <60 years).[10] However, this early use of dopamine agonists does not necessarily translate into a better outcome in the long term and recognition of the side-effect of impulse control disorders is a major limitation to dopamine agonist therapy.[11]
- Transdermal delivery can be advantageous for patients with dysphagia.
- Common adverse effects include nausea, dyskinesia, confusion, hallucination, and hypotension; pramipexole and ropinirole can cause somnolence and sudden onset of sleep and, therefore, patients should not drive while taking this medication.
- Pergolide use is associated with a risk of valvular heart disease and was withdrawn from market.

34.3.1.3 Anticholinergic Drugs

- An example is Artane.
- This drug is good for tremor and rigidity as well as severe drooling of saliva.
- It is associated with significant adverse effects rendering it not useful in the older or demented patients including those with confusion, memory impairment, sedation, urinary retention, and blurred vision.

34.3.1.4 COMT (catechol-O-methyl transferase) Inhibitors

- An example is entacapone (Comtan).
- These drugs are used as an adjunct with every dose of L-dopa to increase its central nervous system availability; these are very useful when a patient develops end-of-dose akinesia in the later stage of the disease.
- Common side-effects include gastrointestinal symptoms, liver function derangement, confusion, dyskinesia, and urine discoloration.

34.3.1.5 MAO-B (monoamine oxidase B) Inhibitors

- Examples are selegiline and rasagiline.
- These drugs may be useful in early PD but have only modest symptomatic benefits as monotherapy. The value of their neuroprotective effect is unclear. Rasagiline can be used as an add-on drug to L-dopa for PD when the efficacy of L-dopa becomes inconsistent.
- Adverse effects include nausea and headache; selegiline can cause confusion in elderly patients.

34.3.1.6 Others

- Examples are amantadine and apomorphine.
- Some difficult cases of PD may respond to treatment with these drugs. Their use in the routine management of older PD patients is very limited.

34.3.2 Surgical Treatment

Ablation of some deep nuclei in the extrapyramidal system (pallidal, subthalamic, or thalamic) by stereotactic approach or gamma-knife has been tried with success to control symptoms of PD patients. Deep brain stimulation of these nuclei has also been tried with success. Although these modalities are available in Hong Kong, their overall impact on the management of the older PD patients remains small. Transplantation of autologous adult adrenal cells or fetal nigral brain cells remains experimental.

34.3.3 Management of Comorbid Problems Associated with PD

Non-motor comorbid symptoms such as tingling of arms and legs, urinary difficulties, constipation, daytime sleepiness, insomnia, neuropsychiatric symptoms (e.g. psychosis, hallucination, and depression), fatigue, and dementia are common in elderly patients with PD, but often underdiagnosed and ignored in the management of PD. Dysarthria and hypophonia are also common. Speech therapists may be helpful to improve the speech volume.[12] Drooling of saliva is common and the use of anticholinergic drugs and botulinum toxin injection into salivary glands have been shown to be effective in studies. Such non-motor comorbid problems have great impact on both the patients and caregivers. These can be disease-related or treatment-related, and should also be assessed and managed.[13]

34.3.4 Supportive Management

As PD is a chronic disabling disease, supportive care to the patients and caregivers is very important. The Hong Kong Parkinson's Disease Association (香港柏金遜症會; http://www.hkpda.org) is a self-help organisation formed by a group of PD patients, their families, carers, and community members. General health of the patient should be maintained with programmes of regular exercise (e.g. aerobic exercises or tai chi[14]), activity, and rest. Nutritional advice, including dietary manipulation to alleviate constipation and low protein diet to enhance L-dopa absorption, should be carefully planned. Depression is common in these patients and should be appropriately assessed and treated. In the late stage of the illness, good personal care and nursing care are essential. Occupational therapy, physiotherapy, and speech therapy may help. In the terminal stage of the disease, end-of-life care approach should be considered.

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CHAPTER 35

STROKE

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OBJECTIVE

To learn the epidemiology, risk factors, early recognition, clinical evaluation, diagnosis, and management of acute stroke; and discuss the role of stroke rehabilitation, secondary prevention of stroke, the organisation of stroke services, and local stroke resources in the management of acute stroke.

KEY POINTS

- Stroke is a leading cause of morbidity and mortality.
- Stroke is an emergency; timely diagnosis is important to triage stroke patients for appropriate treatments.
- Stroke thrombolysis remains an effective treatment even in patients with advanced age; it improves neurological impairment in selected patients. Stroke thrombolysis should be considered in those aged >80 years and who receives treatment within 4.5 hours of onset of symptoms.
- A structured management in a stroke unit with integration of medical, nursing, and rehabilitation efforts improves patient outcomes.
- Stroke rehabilitation should be intensive, started early, sustained, goal-driven, and matched with good nutrition.
- Secondary prevention measures and management of long-term complications are important in the continuing medical care of a stroke individual after the acute phase.
- Timely, comprehensive, and aggressive management of transient ischaemic attack can reduce the risk of subsequent stroke.

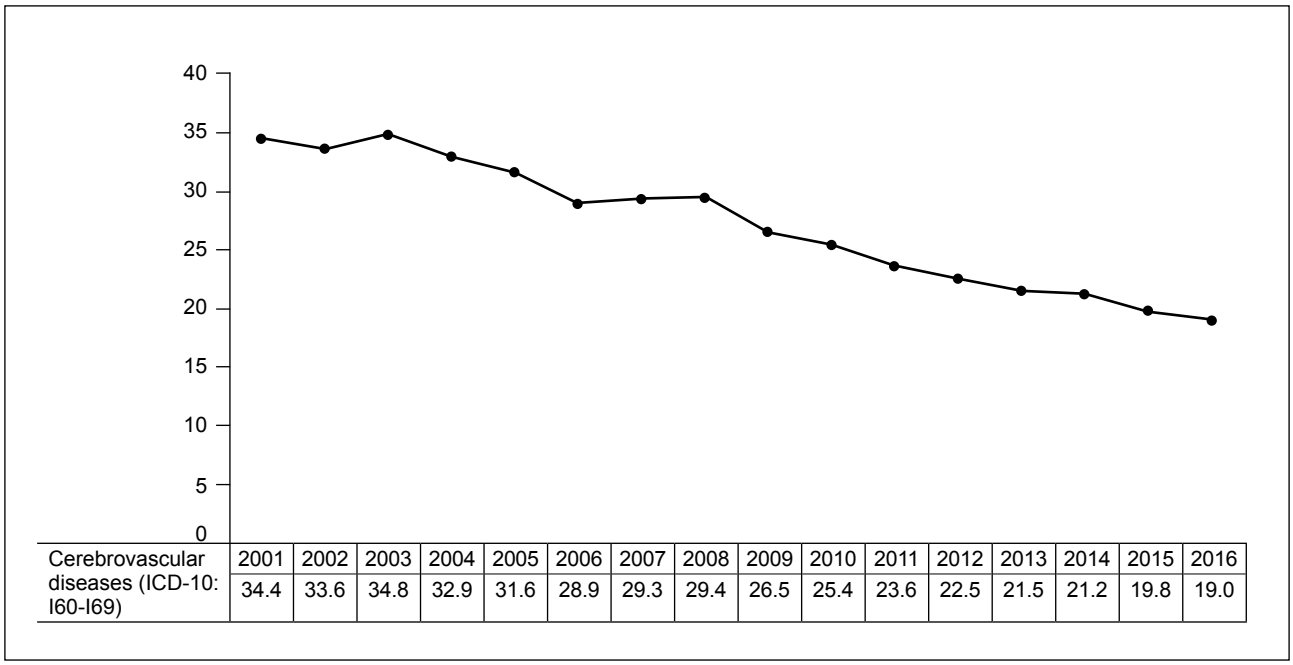
35.1 Introduction

Stroke is the fourth leading cause of death in Hong Kong, after cancer, pneumonia, and heart disease.[1] It is devastating, with a 1-year mortality rate of 22.5%, and a 5-year survival rate after the first stroke of only 60%.[2] More encouraging, however, is a consistent decline in both the crude and age-standardised stroke death rates since 2008, which reflects the overall improvement in stroke services in Hong Kong (Figure 35.1[1]).

35.2 Epidemiology

In China, the provincial stroke incidence varies from 73.1 to 466.9 per 100 000 persons, with a belt of high-stroke incidence rate across provincial regions located in western and northern China, attributed to higher regional prevalence of hypertension and overweight.[3] Chinese populations have relatively more haemorrhagic strokes compared with western populations (17.1-55.4% vs. 6.5-19.6%); however, there was a trend of decreasing incidence of haemorrhagic strokes in the 1990s in China.[4]

The number of strokes in Hong Kong has increased with population growth and ageing. A study of the number of stroke admissions to public hospitals in Hong Kong from 1999 to 2007 revealed that the incidence rates of ischaemic stroke were 348.4 per 100 000 males and 298.3 per 100 000 females, and those of haemorrhagic stroke were 99.1 per 100 000 males and 62.1 per 100 000 females.[5] The incidence rates of both ischaemic and haemorrhagic strokes increased with age and were higher in men than in women (Figure 35.2a and 35.2b[5]). Older people have higher proportions with ischaemic strokes compared with younger adults (Figure 35.2c[5]). From 1999 to 2007, the incidence of ischaemic stroke has declined, while that of haemorrhagic stroke has remained constant, except for a slight increase among those aged 35 to 44 years. Nevertheless, haemorrhagic stroke now accounts for 19.3% of all strokes in Hong Kong, which is close to the value reported for Caucasians; it has decreased substantially since the 1980s, when rates of 27.1-31.1% were reported.[6,7] The observed overall reduction in haemorrhagic stroke rate from 1980s to 2000s in Hong Kong may be attributed to wider use



	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016
Cerebrovascular diseases (ICD-10: I60-I69)	34.4	33.6	34.8	32.9	31.6	28.9	29.3	29.4	26.5	25.4	23.6	22.5	21.5	21.2	19.8	19.0

Figure 35.1 Standardised death rates for stroke in Hong Kong from 2001 to 2016[1]
Abbreviation: ICD-10 = International Classification of Diseases, tenth revision

of computed tomographic (CT) scan that picked up milder ischaemic strokes, ageing population with a higher proportion of ischaemic strokes among older people, or change in lifestyle towards the western pattern. However, the current incidence rates of ischaemic and haemorrhagic stroke in Hong Kong, after standardisation, remain higher than those in most other cities with similar socioeconomic profiles.

35.3 Risk Factors

Age and male gender are important risk factors for stroke (Figure 35.2[5]), but these are not modifiable. Many medical diseases increase stroke risk, and optimal control of these diseases can successfully prevent stroke. The single most important disease in this category is hypertension, which is implicated in 50% of all strokes.[8] Age is a strong risk modifier in stroke, and the attributable risk for all the modifiable diseases decreases with ageing, meaning that treatment of these modifiable diseases becomes less effective for stroke prevention at advanced age. The only exception is atrial fibrillation (AF), where the attributable risk increases with age. Therefore, in an ageing population, we expect more AF-related ischaemic stroke, and anticoagulation is more worthwhile in older subjects (Table 35.1).[9]

35.4 Early Recognition, Clinical Evaluation, and Investigations

A sudden onset of focal neurological deficit requires the assumption of a clinical diagnosis of stroke until proven otherwise. The Face Arm Speech Test (FAST; Table 35.2a) has been developed to encourage rapid recognition of important stroke warning symptoms for early access to organised stroke care.[10] This has been simplified by a group of local medical students in 2013 to the Chinese acronym for FAST「談笑用兵」防中風 (Table 35.2b, http://www.facebook.com/fourstepssurvivestroke) as part of a public campaign to promote early recognition of stroke symptoms for timely access to stroke care.

Clinical evaluation should begin with a detailed history from the patient and eyewitness, noting the exact time of onset of symptoms, its evolution and time course, presence of vascular risk factors, current medications, pre-morbid physical and social function, and support network. Examination should focus on establishing the nature and severity of neurological deficits, the likely size and location of the vascular territory, and probable underlying causes. Impairment, disability, and handicap should be assessed using validated tools to guide acute treatment and rehabilitation, and to gauge progress and outcome, e.g. severity of the neurological impairment measured by National Institutes of Health Stroke Scale (NIHSS) [see Chapter 35.5.1], activities of daily living by Barthel Index (see Chapter 4.5.1), and hemineglect by Albert's line cancellation test[11] (see Chapters 2.6, 2L).

All patients with clinical stroke should undergo

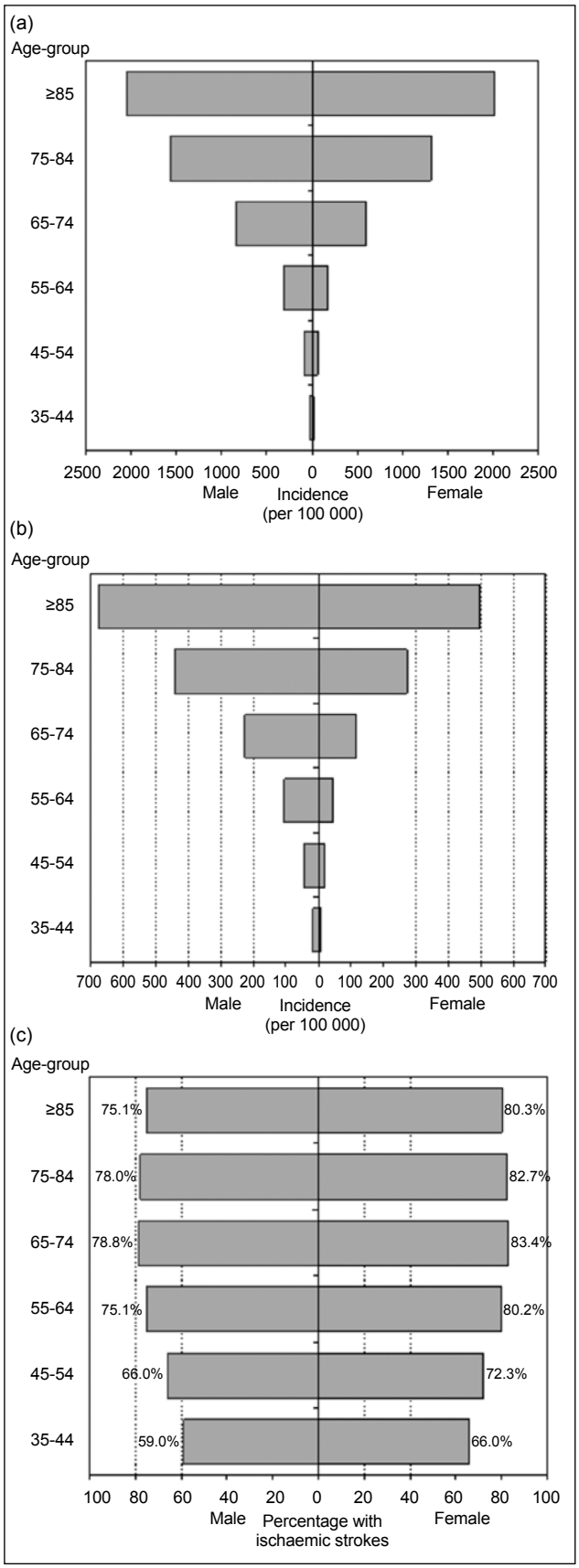


Figure 35.2 (a) Ischaemic stroke incidence in Hong Kong by age-group for the period 2005-2007[5]
(b) Haemorrhagic stroke incidence in Hong Kong by age-group for the period 2005-2007[5]
(c) Proportion of ischaemic strokes in Hong Kong by age-group for the period 2005-2007[5]

neuroimaging as soon as possible to differentiate haemorrhagic from ischaemic stroke because the treatment differs considerably. Plain CT brain, which is now available in all acute care hospitals in Hong Kong, is very sensitive for the detection of acute haemorrhage, but it can be normal within the first few hours of ischaemic stoke. In selected patients, whose clinical presentations are less straightforward, magnetic resonance imaging (MRI) of the brain with a diffusion-weighted sequence would be a better option for distinguishing genuine acute stroke from stroke-mimicking conditions.[12] MRI is better than CT in detecting early infarcts and small infarcts, especially those located in the posterior fossa. MRI of the brain can also be used to estimate the age of acute infarction. A mismatch in visibility of an acute ischaemic lesion between diffusion-weighted MRI (DWI) and fluid-attenuated inversion recovery (FLAIR) MRI (DWI-FLAIR mismatch) is now accepted as a surrogate for hyperacute infarction to consider thrombolytic therapy in patients who cannot ascertain the onset time, including wake-up strokes.[13] MRI is very sensitive for both acute and old haemorrhages, and the new sequences have exaggerated the visibility of these abnormalities to even untrained eyes. These advantages make MRI the neuroimaging modality of choice for stroke in the private sector. While the type of neuroimaging used to characterise a stroke may be determined by local availability, it is important to think of the nature of the information needed and the safety of the patient. Thus, for a patient with a major stroke at risk of hypoxia, especially when lying flat, CT may be favoured over MRI as a rapid imaging procedure.

After the initial brain imaging, the next step in diagnostic evaluation is to determine the aetiology of stroke, which involves the investigation of vascular lesions and potential cardioembolic sources. Transcranial Doppler (TCD) ultrasound is a non-invasive investigative tool for large artery disease, intracranial in particular, and can be performed at the bedside. Intracranial large artery disease is common among local stroke patients, with a prevalence of up to 50% in ischaemic stroke patients.[14] The recurrent risk and prognosis of intracranial large artery disease were comparable to extracranial carotid stenosis.[14] TCD ultrasound has many other uses in stroke, and these are summarised in Table 35.3.

MRI and CT angiograms are alternatives to TCD and carotid duplex for vascular imaging, and both yield good-quality images for both diagnostic and interventional planning purposes.

AF is the most important aetiology of cardioembolic stroke. A normal sinus rhythm in the electrocardiogram (ECG) does not exclude

Table 35.1 Modifiable risk factors for stroke[8,9]

• Hypertension	• Sedentary lifestyle / lack of exercise
• Atrial fibrillation	• Valvular heart disease
• Cigarette smoking	• Ischaemic heart disease
• Hyperlipidaemia	• Hypercoagulable states
• Diabetes mellitus	• Previous stroke / transient ischaemic attack
• Obesity	• Severe carotid stenosis
• Homocystinaemia	• Intracranial arterial stenosis
• Excess alcohol consumption	• Radiotherapy

Table 35.2a The Face Arm Speech Test (FAST)[10]

Facial movements

Ask patient to smile or show teeth:
1. Look for NEW lack of symmetry (e.g. unequal smile or grimace)
2. Note which side does not move well, and mark on the form whether it is the patient's right or left side

Arm movements

Lift the patient's arms together to 90° if sitting, 45° if supine and ask the patient to hold the position for 5 seconds, then let go:
1. Does one arm drift down or fall rapidly?
2. If one arm drifts down or falls, note whether it is the patient's left or right arm

Speech

If the patient attempts a conversation:
1. Look for NEW disturbance of speech
2. Check with companion
3. Look for slurred speech
4. Look for word-finding difficulties. This can be confirmed by asking the patient to name commonplace objects that may be nearby, such as a cup, chair, table, keys, and pen
5. If there is a severe visual disturbance, place an object in the patient's hand and ask him / her to name it

Table 35.2b The Chinese acronym for FAST:「談笑用兵」

「談」: 表達説話有困難	Talk for Speech
「笑」: 笑容表情不對稱	Smile for Face
「用」: 用手用腳軟無力	Use for Arm
「兵」: 致電求助等救兵	SOS for Timely call for help

Table 35.3 Potential applications of transcranial Doppler in stroke

- To investigate intracranial and extracranial large artery stenosis
- To monitor vasospasm in subarachnoid haemorrhage
- To monitor vessel recanalisation after thrombolysis
- Intraoperative monitoring during carotid interventions
- Sonothrombolysis
- Detection of patent foramen ovale
- To monitor microembolic signals from symptomatic intracranial stenosis

paroxysmal AF. Patients who do not have an apparent cause for ischaemic stroke should undertake an ECG test and 24-hour ECG recording to detect a potential cardioembolic source. However, even the 24-hour ECG recording might not be able to capture paroxysmal AF. In a study applying implantable loop recorders to a group of otherwise cryptogenic stroke patients, paroxysmal AF was detected in 25.5% of patients at a median of 48 days of monitoring.[15] The optimal mode and duration of monitoring for paroxysmal AF detection in stroke are yet to be determined. However, the presence of AF does not necessarily mean that a stroke is cardioembolic, and one has to keep an open mind for other aetiologies requiring different treatment approaches (see Chapter 35.10.2). Further investigations to detect causes of unexplained stroke are to be guided by clinical presentations, e.g. inflammatory markers (erythrocyte sedimentation rate, C-reactive protein) for giant cell arteritis or infective endocarditis; vasculitis screen for vasculitis syndromes; MRI angiogram for dissection; genetic studies for CADASIL (cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy); and tumour markers for underlying malignancy (see Chapter 35.10.3).

35.5 Acute Management of Stroke

35.5.1 Stroke Thrombolysis for Hyperacute Ischaemic Stroke

From the basic pathophysiology, the only way to save an endangered neuron is to re-establish the circulation and re-perfuse the ischaemic penumbra. Intravenous alteplase at 0.9 mg/kg to a maximum dose of 90 mg is the only recanalisation strategy with good evidentiary support. The original National Institute

of Neurological Disorders and Stroke rt-PA Stroke trial using the 3-hour treatment window increased the number of patients with good neurological recovery by 12.5%, despite a 6% excess of symptomatic haemorrhagic transformation.[16] This treatment window could be extended to 4.5 hours, which allows more patients to be eligible for the treatment.[17] However, the efficacy of thrombolysis drops rapidly with time, and the benefit becomes marginal at the end of the 4.5-hour window. Advanced age is not an exclusion criterion for thrombolysis; results from the IST-3 (Third International Stroke Trial) demonstrated that alteplase remained affective in those aged >80 years.[18] Nevertheless, the natural course of stroke in advanced age is worse than that in younger patients, and even after thrombolysis, the proportion of aged adults with good recovery is low. The most-feared adverse effect of thrombolysis is haemorrhagic transformation. The most important ways of reducing the risk of haemorrhage are: (a) meticulous examination of the inclusion and exclusion criteria, (b) accurate measurement of the patient's weight to estimate dosage, and (c) meticulous blood pressure (BP) control during and after infusion (e.g. using intravenous glyceryl trinitrate or labetalol). The BP level that should trigger BP-lowering treatment should be below the acceptable recommended threshold of 185/110 mm Hg.

The benefit of stroke thrombolysis is highly dependent on the successful recanalisation of the occluded vessels. It has been shown that intravenous thrombolysis was less successful in patients with terminal internal carotid artery or proximal (M1) middle cerebral artery (MCA) infarcts because of the low recanalisation rate in these proximal occlusions.[19] Since 2015, clinical trials on endovascular thrombectomy, using the newer stent retriever devices, had consistently demonstrated superior recanalisation rates and patient outcomes against the standard intravenous thrombolysis in these high risk stroke patients.[20] Endovascular thrombectomy should be considered in stroke patients with proximal artery occlusion within 6 hours of symptom onset.[21]

The NIHSS was developed in the US to assess neurological deficit in acute stroke patients,[22] and this has been used to measure outcomes of thrombolytic therapy. Overall, 15 items are scored in the NIHSS: level of consciousness (LOC, with scores of 0-3); LOC questions (0-2); LOC commands (0-2); best gaze (0-2); visual (0-3); facial palsy (0-3); motor arm (0-4 each for right and left); motor leg (0-4 each for right and left); limb ataxia (0-2); sensory (0-2); best language (0-3); dysarthria (0-2); and extinction and inattention (0-2). The total score is a summation of the individual scores, giving a range from 0 to 42. A higher score indicates a more severe neurological deficit. The NIHSS is available at: https://www.ninds.nih.gov/sites/default/files/NIH_Stroke_Scale_Booklet.pdf. A Chinese version of the NIHSS has also been developed and validated.[23]

35.5.2 Medical Treatment in Ischaemic Stroke

Ischaemic stroke patients who are not eligible for thrombolysis should receive antiplatelet therapy as soon as possible. Medium-dose aspirin (160 mg/day for 4 weeks in Chinese Acute Stroke Trial, and 300 mg/day for 2 weeks in IST) is recommended for all ischaemic stroke patients without contraindications.[24] In patients with atherosclerotic stroke and lower bleeding risk (i.e. minor stroke or transient ischaemic attack [TIA]), a combination of aspirin and clopidogrel for up to 1 month has been shown to decrease early recurrence risk, as demonstrated in the CHANCE study, and is therefore recommended.[25] A combination of aspirin and clopidogrel can also be considered for symptomatic large artery disease with unstable neurological status or progressive stroke, after carefully balancing the benefit of stroke stabilisation versus the risk of haemorrhagic transformation.[26]

Early anticoagulation should be considered in patients with non-disabling cardioembolic stroke. Immediate anticoagulation with unfractionated or low-molecular-weight heparin, however, did not show convincing benefits in acute treatment and are, therefore, not recommended, except in the context of cerebral venous thrombosis, or in patients with minor stroke or TIA, where the underlying cardiac conditions carry high embolic risk (e.g. AF in valvular heart disease, presence of mural thrombus on echocardiogram, etc).

35.5.3 Medical Treatment in Haemorrhagic Stroke

Medical treatment is the mainstay treatment for haemorrhagic stroke. The recently published INTERACT 2 trial demonstrated that rapid control of systolic blood pressure (SBP) to ≤140 mm Hg reduced death and disability in haemorrhagic stroke.[27] Aggressive BP control is now recommended for all haemorrhagic stroke patients. Surgical treatment for haemorrhagic stroke should be considered in selected patients with cerebellar haemorrhage who have neurological deterioration, hydrocephalus or brainstem compression, as well as in patients with lobar haemorrhage of >30 ml and located within 1 cm of the surface.[28] Clinicians should work closely with neurosurgeons in preparing suitable patients for surgical treatments.

Many patients now receive anticoagulation for

different medical conditions and anticoagulation-related haemorrhagic stroke is becoming a new challenge in stroke medicine. These patients carry high mortality, and their coagulopathy should be reversed as soon as possible once the haemorrhagic stroke is confirmed. There is no universal guideline for a reversal strategy. One reversal regimen is 1000 to 1500 IU prothrombin complex concentrates together with intravenous vitamin K1 (10 mg) in warfarin-related haemorrhagic stroke, unless there is a known international normalised ratio (INR) of <1.4 at the time of diagnosis. Further doses of prothrombin complex concentrates and fresh frozen plasma could be considered according to the subsequent INR levels.

35.6 Complications, Disabilities, and Stroke Rehabilitation

The following are complications of stroke in the acute phase:

- raised intracranial pressure from cerebral oedema or hydrocephalus, leading to reduced conscious level
- haemorrhagic transformation
- convulsion
- hypoventilation
- deep vein thrombosis or pulmonary embolism
- dysphagia
- aspiration
- myocardial infarction
- pneumonia or urinary tract infection
- incontinence
- pressure sore
- recurrent strokes

Prevention, early detection, and appropriate treatment of these complications through medical and skilled nursing care are believed to be the main reasons underlying the success of stroke units in managing stroke.

Only about half of the stroke survivors can remain independent within the community. Many stroke patients have residual neurological impairments and stroke-related complications, which include the following:

- spasticity
- shoulder pain or subluxation
- central post-stroke pain syndrome
- visual problems (hemianopia)
- perceptive deficit (hemineglect)
- dysphasia
- dysphagia
- falls and fractures
- depression
- dementia
- epilepsy
- excessive fatigue
- headaches

To achieve the best possible outcome and to help the patient to reach their own full potential, stroke rehabilitation should be intensive, goal-oriented, matched with good nutrition, holistic, and started as early as possible and sustained for as long as the patient can tolerate. Simultaneous attention to psychosocial, cognitive and sexual needs, as well as family dynamics and expectations, are also important.

Research on stroke rehabilitation has continued to broaden our knowledge on[29,30]:

- evidence-based practice of old treatment strategies in stroke rehabilitation;
- developments in the treatment of 'neglected' impairments, such as hemianopia, hemineglect, and sensory loss;
- evaluation of the use of technology in stroke rehabilitation, e.g. stimulation, robotic therapy, virtual reality, and feedback devices; and
- advances in neurorestorative treatments after stroke

Modern rehabilitation science has introduced many intervention strategies targeting improvements in subacute and chronic phases of stroke[31,32]:

- task-oriented and repetitive training-based interventions
- device-based therapies, e.g. robotics, functional electrical stimulation, etc.
- electromagnetic stimulation e.g. transcranial magnetic stimulation, transcranial direct current stimulation, etc.
- botulinum toxin injection or peripheral nerve blocks for spasticity

Significant advances in the knowledge and skill of stroke rehabilitation have been made. An in-depth discussion of the new developments in stroke rehabilitation is beyond the scope of this chapter. These new therapies have been gradually integrated into stroke rehabilitation in Hong Kong and show promise to further increase the independence level of stroke survivors.

35.7 Secondary Prevention

Patients who have one stroke have an increased risk of recurrence; the cumulative risk is 26% at 5 years and 39% at 10 years after the first stroke.[33] The recurrence risk depends upon the stroke aetiology, with large artery atherosclerosis being the highest risk for early recurrence, compared to relatively benign small artery disease stroke.[34] Therefore, every stroke patient who is suitable for secondary prevention should be investigated for risk factors, and appropriate treatments for secondary prevention should be commenced as soon as possible. This secondary prevention is personalised according to the risk profile of individual patients.

35.7.1 Lifestyle Modifications

The research-based evidence for lifestyle measures should likely be emphasised more in primary prevention, but these measures are no less important in secondary prevention. Secondary prevention requires a long-term adherence to a healthy lifestyle adjustment to earn credits. A healthy lifestyle includes smoking cessation, weight control, regular exercise, and a balanced diet with a high proportion of fruit and vegetables, as well as less saturated fat.[35] Exercise can help to reduce stress, reduce BP, improve weight control and cardiovascular function, and achieve improved balance, muscle strength, and mobility.

35.7.2 BP

Because a transient rise in BP is common in the first 24 hours after the onset of stroke, and cerebral autoregulation may be impaired in the acute phase of stroke, initiation of antihypertensive therapy in acute ischaemic stroke is not recommended unless it is in the setting of thrombolysis, hypertensive emergency, or serious concomitant medical conditions (e.g. hypertensive cardiac failure / myocardial infarction, or aortic dissection).[35,36] Guidelines in the UK and US differ in their recommendation of premorbid antihypertensive medications at the onset of acute ischaemic stroke, with the former recommending continuation[35] (unless patient is dysphagic or has contraindications) and the latter discontinuation or reduction (of beta-blockers to avoid withdrawal syndrome).[36] The optimal time after the onset of acute ischaemic stroke to restart or start long-term antihypertensive therapy has not been established. While guidelines in the US suggest that it is reasonable to initiate or resume long-term antihypertensive therapy after the first several days from stroke onset,[37] guidelines in the UK recommend initiation of antihypertensive therapy prior to hospital discharge or at 2 weeks, whichever is earlier.[35]

In the perindopril protection against recurrent stroke study (PROGRESS) [mean age, 64 years], lowering the BP by 12/5 mm Hg decreased recurrent stroke by 42%, and the benefit extended to both hypertensive and normotensive patients.[38] However, PROGRESS did not indicate the level to which BP should be lowered, and caution is needed in older or frail patients prone to orthostatic hypotension and those with severe, bilateral carotid, or vertebrobasilar occlusive disease with reduced cerebrovascular perfusion pressure.[39,40] In the SPS3 trial (mean age, 63 years) on BP targets in patients with recent lacunar stroke, there was a non-significant reduction in stroke rate in the lower SBP (<130 mm Hg) group compared with the 130-139 mm Hg group.[41] The SPS3 trial was underpowered to exclude confidently an increase in serious complications of hypotension with target SBP <130 mm Hg.[42] In the lower target group of the SPS3 trial, there were more, though non-significant, complications of injurious fall, orthostatic syncope, and stroke associated with hypotension.[41] While the Royal College of Physicians recommends a target BP of <130/80 mm Hg (except for patients with severe bilateral carotid stenosis),[35] the American Heart Association / American Stroke Association acknowledges the uncertainty of an optimum target for BP reduction and recommends that BP management should be individualised according to stroke subtype and comorbidities, but a reasonable goal is a BP of <140/90 mm Hg.[37]

New hypertension guidelines in the US emphasise less stringent thresholds for elderly hypertensive patients. The US 8th Joint National Committee (JNC 8) 2014 Hypertension Guidelines recommend that older adults (aged ≥60 years) should be treated to a BP target of <150/90 mm Hg, rather than 140/90 mm Hg for adults of all ages as recommended a decade ago in its 7th Hypertension Guideline.[43] The conclusion of the panel of the JNC 8 was based on data from randomised controlled trials (RCTs). The early hypertension RCTs generally excluded elderly patients. Later trials that focused specifically on older populations found that treating isolated systolic hypertension was beneficial, yet treatment targets of SBP <160 mm Hg were set in these trials. The JNC 8 also removed the tighter control recommendations in patients with diabetes and renal disease. Nevertheless, the panel emphasised that the recommendations of JNC 8 were not a substitute for clinical judgement, and decisions about care must carefully consider and incorporate the clinical characteristics and circumstances of each individual patient. In addition to reducing mean BP, reducing visit-to-visit variability in SBP, by means of calcium channel blockers and diuretics, rather than beta-blockers, is also likely to reduce stroke risk.[40,44]

35.7.3 Diabetes Mellitus

Diabetes mellitus patients have an increased risk of stroke. Long-term cohorts have demonstrated a decrease in macrovascular events with a target glycated haemoglobin (HbA1c) level of <7%. Further intensive therapy, however, did not improve macrovascular outcomes. Therefore, in diabetic patients who have ischaemic stroke, glycaemic control with HbA1c level of <7% is recommended for secondary prevention.[37] However, glycaemic control should be balanced against hypoglycaemic risk, and an individualised approach should be adopted in setting the management goals in an elderly diabetic with higher HbA1c targets set for frailer diabetics (see Chapter 41.4, Table 41.6).

35.7.4 Lipids

Hypercholesterolaemia increases the incidence of cardiovascular events and stroke. The benefits of

lipid-lowering therapy in reducing stroke have been confirmed in the Heart Protection Study, which demonstrated a 25% risk reduction in stroke with simvastatin 40 mg daily among patients with high cardiovascular risks, and in the SPARCL trial, which found a 15% risk reduction in stroke after atorvastatin (80 mg daily) among patients with recent stroke or TIA.[45,46] This benefit has been shown to be consistent regardless of the baseline cholesterol level. Nevertheless, the absolute risk reduction with intensive lipid lowering in patients with low baseline low-density lipoprotein cholesterol (LDL-C) is small. It is recommended that statins should be started for all patients with ischaemic stroke, and the therapy could be intensified if LDL-C does not reach a level of <2.0 mmol/l with initial therapy.[35] In those with a recent primary intracerebral haemorrhage, treatment with statin therapy should be avoided and only used with caution, if required for other indications.[35] Although statins are currently routinely prescribed in patients >85 years old, the research evidence remains controversial regarding the balance between absolute long-term benefit and potential adverse effects.

35.7.5 Carotid Stenosis

In patients with non-disabling stroke from symptomatic carotid stenosis, carotid endarterectomy is recommended, which results in an absolute stroke risk reduction of 17% compared with medical treatment.[47] Carotid angioplasty with stenting is an alternative, especially in patients with unfavourable operative risk because of comorbidity, post-irradiation carotid stenosis, or high carotid bifurcation.

35.7.6 AF (see also Chapter 36)

All ischaemic stroke patients with AF should receive anticoagulation with either warfarin or one of the non–vitamin K antagonist oral anticoagulants (NOAC) unless contraindicated, and the 2016 European Society of Cardiology Guidelines state that NOAC is the preferred choice for non-valvular AF if eligible.[48] The relative risk reduction is 67% in secondary stroke prevention. Certain categories of patients are particularly suited to NOAC, e.g. those intolerant to warfarin, those who cannot have regular blood tests or with poor venous access, those with maintenance INR treatment therapeutic level of <65% over the last 6 months, and those who require immediate anticoagulation such as high-risk cardioembolic TIAs with AF.[49] In patients with large territory of arterial infarction and / or mass effect, anticoagulation should be delayed for 2 weeks to reduce the risk of secondary haemorrhage. Addition of aspirin, clopidogrel, or both, to warfarin significantly increases haemorrhagic risk, especially in those with high bleeding risk (e.g. hypertensive elderly patients). Risk of haemorrhage with anticoagulant use should be balanced against the risk of stroke from AF. Further discussions on anticoagulation and antithrombotic therapy in AF are detailed in Chapters 36.4.4, 36.5, and 36.6.

35.7.7 Antiplatelet Agents

Antiplatelet agents should be started in all patients with presumed atherosclerotic stroke. Typically, maintenance therapy with low-dose aspirin (e.g. 80 mg daily) is given after initial medium-dose aspirin (160 mg daily for 4 weeks or 300 mg daily for 2 weeks). Aspirin monotherapy achieves a 15% relative risk reduction in stroke, and clopidogrel monotherapy or aspirin plus extended-release dipyridamole are both alternatives with slightly better potency than aspirin monotherapy; they would be preferable in patients with increased vascular risks.[50-52] Cilostazol is another antiplatelet agent that is being studied, mainly in Asian ethnic populations. In a head-to-head comparison, cilostazol was found to be slightly superior to aspirin in secondary stroke prevention, and it had fewer bleeding complications.[53] Cilostazol could be particularly useful in patients with high bleeding risk.

35.8 TIA

Patients whose neurological symptoms resolve completely within 24 hours are said to have TIA. Up to 23% of stroke patients have preceding symptoms of TIA, and TIA patients are particularly vulnerable to develop a subsequent stroke shortly after their TIA episodes.[54] A meta-analysis of 11 studies (n=7000) found that the overall cumulative incidence of stroke after TIA was 3.5% by 2 days, 8.0% by 30 days, and 9.2% by 90 days.[55] More importantly, in three studies with active outcome ascertainment, the rates of cumulative incidence by 2, 30, and 90 days rose to 9.9%, 13.4%, and 17.3%, respectively. Most of the strokes developed in the first month, and in half of the patients, in the next 48 hours. The risk of stroke could be further refined by using the validated ABCD2 score (Table 35.4); any patient with a score of ≥4 needs immediate attention.[56]

Treatment of TIA to prevent stroke could be hugely encouraging. This condition is a perfect wake-up call for immediate medical attention and treatment. As the time window between TIA and the subsequent stroke is short, medical service should be timely, investigations must be rapid and comprehensive, and the treatments must be aggressive. These important elements in a rapid TIA service model have been highly effective, as the EXPRESS study demonstrated, to successfully prevent development of subsequent strokes.[57] Since 2012, the Hong Kong Hospital Authority, which governs the public healthcare services in Hong Kong, started to establish specialist-led and protocol-driven TIA clinics in major hospitals in Hong Kong. The service is expected to reach territory-wide coverage by 2017.

Table 35.4 ABCD2 score and the subsequent stroke risk[56]

Item	Score		
Age ≥60 years	1		
Blood pressure ≥140/90 mm Hg	1		
Clinical features			
Unilateral weakness	2		
Speech disturbance without weakness	1		
Duration of symptoms (mins)			
≥60	2		
10-59	1		
Diabetes mellitus	1		
ABCD2 score and risk category	Risk of stroke (%)		
	2 Days	7 Days	90 Days
Low (0-3)	1.0	1.2	3.1
Moderate (4-5)	4.1	5.9	9.8
High (6-7)	8.1	11.7	17.8

Table 35.5 Main results (outcome at final follow-up, median, 1 year) from Cochrane review on stroke unit care for stroke (from Stroke Unit Trialists' Collaboration 2013[58])

	Stroke unit	General medical wards	Risk ratio (95% confidence interval)
Death	17.7%	23.3%	0.81 (0.69-0.94)
Death or institutional care	35.1%	40.4%	0.78 (0.68-0.89)
Death or dependency	56.2%	61.5%	0.79 (0.68-0.90)

Table 35.6 Structured management in stroke units

- To evaluate acute treatment options for individual patients
- To select appropriate patients for thrombolytic treatment
- Skilled nursing care in medical monitoring and rehabilitation
- Protocol-based strategies for swallowing, nutrition, bowel and bladder management
- Comprehensive vascular evaluation and secondary prevention measures
- Early and intensive rehabilitation
- Multidisciplinary approach and holistic patient care
- Continuous professional training in stroke medicine

35.9 Stroke Services and Resources

35.9.1 Stroke Unit and Stroke Service Components

There is strong evidence to suggest that stroke units, defined as organised inpatient stroke care, save lives and reduce disability (Table 35.5),[58] and appeared to be better in stroke units based in a dedicated stroke ward where a multidisciplinary team cares exclusively for stroke patients in a discrete ward.[58-60] In contrast with thrombolysis for acute stroke, the beneficial outcomes of stroke units were independent of initial stroke severity or stroke type.[58] The treatment benefit applies to both ischaemic or haemorrhagic stroke patients.[61] The Stroke Unit Trialists' Collaboration (SUTC) identified different models of organised stroke unit care: acute intensive; comprehensive (combined acute and rehabilitation); stroke rehabilitation; mixed rehabilitation; mobile team; and integrated traditional Chinese medicine.[58] The SUTC concluded that there was no evidence to suggest that acute intensive stroke units or mobile stroke teams improved patient outcomes, while comprehensive stroke units, rehabilitation stroke units, and mixed rehabilitation units were superior to stroke care in general wards.[62] Hospitals admitting large numbers of stroke patients may not be able to provide all stroke care within one comprehensive stroke unit and have to consider providing some longer-term rehabilitation in a separate stroke rehabilitation unit, which has to be closely coordinated with the other parts of the stroke service for effectiveness. The core characteristic features of organised stroke unit care identified in RCTs by the SUTC are[58]:

- coordinated multidisciplinary rehabilitation incorporating meetings at least once per week;
- multidisciplinary staffing (medical, nursing and therapy staff) including a specialist in stroke or rehabilitation;
- routine involvement of caregivers in the rehabilitation process; and
- regular programmes of education and training.

A national audit of stroke units in the UK, however, revealed that less than half of those hospitals which stated that they had dedicated stroke units reported having all the above organisational characteristics shown to be required for reduction in mortality and morbidity.[63] The same audit also found that just over one-third of stroke patients admitted to those

hospitals with a stroke unit actually spent any time in it.[63] The realisation of the benefit of a stroke unit, thus, requires adequate resources, staffing, and capacity for it to operate based on the core organisational characteristics.[64]

Possible functions of a stroke unit are outlined in Table 35.6. The stroke service components of an integrated stroke service are shown in Figure 35.3.[65]

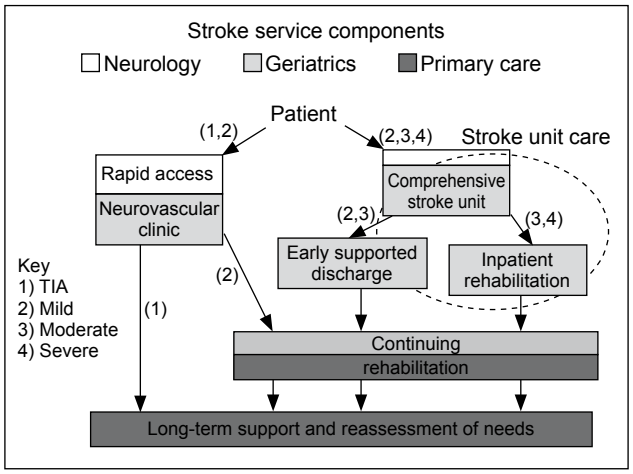


Figure 35.3 Stroke service components[65]
Abbreviation: TIA = transient ischaemic attack

35.9.2 Stroke Services in Hong Kong

Hospitals in Hong Kong have developed stroke units and stroke services to manage stroke across a wide spectrum of care settings (Figure 35.4):

35.9.3 Stroke Resources in Hong Kong

The Hong Kong Stroke Society (http://www.stroke.org.hk/) is a professional society promoting stroke medicine education and service development in Hong Kong. Scientific information on stroke can also be found on other healthcare profession websites, e.g. The Hong Kong Neurological Society 香港腦科學會 (http://www.hkns.org/), The Hong Kong Geriatrics Society香港老人科醫學會(http://www.hkgs.org/), the Hospital Authority 醫院管理局 (http://www21.ha.org.hk/smartpatient/tc/chronicdiseases_zone/details.html?id=97), and the British Association of Stroke Physicians (http://basp.ac.uk/resources/basp-publications/). The Hong Kong Stroke Association 新健社（中風患者及家屬互助社）is a mutual support organisation established for patients of stroke and their caregivers (http://www.hk-stroke-a.org.hk/). The Hong Kong Stroke Association website includes useful webpages on community resources. The Hong Kong Society for Rehabilitation 香港復康會 provides various community based services (http://www.rehabsociety.org.hk/c/main-2.html).

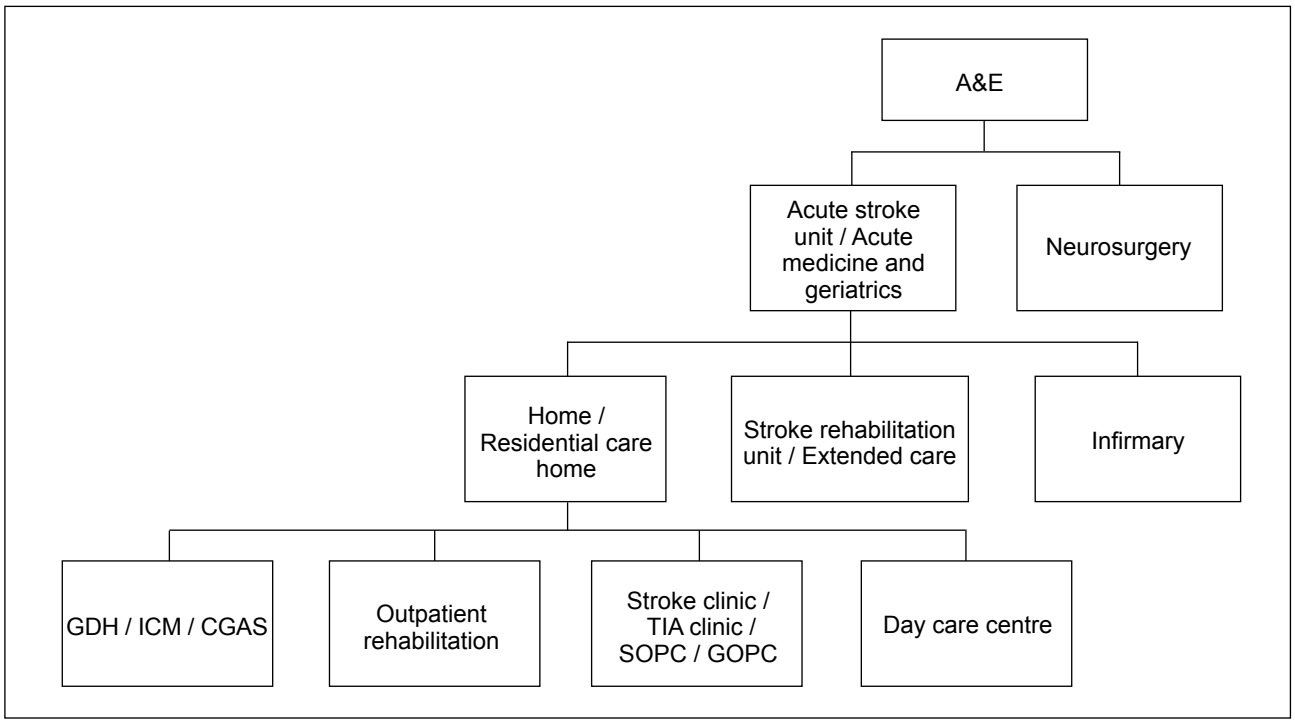


Figure 35.4 Stroke care settings in Hong Kong
Abbreviations: A&E = accident and emergency department; GDH = geriatric day hospital; ICM = Integrated Care Model (transitional care of discharge planning and home support); CGAS = Community Geriatric Assessment Service (outreach to residential care homes); TIA = transient ischaemic attack; SOPC = specialist outpatient clinic; GOPC = general outpatient clinic

35.10 Case Discussions

Case Scenario 1

Ms Law, aged 81 years, had a history of diabetes mellitus, hypertension, heart failure, and acromegaly. She developed sudden left-sided weakness at 13:00 while visiting her husband in a residential care home. Upon arrival to the hospital at 13:54, her left-sided strength was Medical Research Council (MRC) grade 3/5, and the ECG showed AF. The stroke physician was alerted to assess the patient for thrombolysis. When he examined the patient at 14:05, her left-sided strength had already returned and the only residual neurological deficit was left sensory inattention, which yielded an NIHSS score of only 1. The plain CT brain revealed a hyperdense, right MCA (Plate 35.1). The stroke physician decided to proceed with thrombolysis after discussing the scenario with Ms Law, and intravenous alteplase was given at 14:50. In the first 24 hours, the neurological impairment fluctuated, and the NIHSS scores rose to 6 and then returned to 1. Warfarin was started during her hospital stay for secondary stroke prevention. Ms Law was discharged 1 week later. She enjoyed a complete recovery.

Comments

This case illustrates the dynamic nature of an acute stroke, especially how the clinician evaluated the available information to reach a timely treatment decision. Apparently, this patient had a rapidly improving stroke or TIA, which could result in minimal or no neurological deficits. In that sense, any acute treatment would not have affected her outcome in this incident event, and thrombolytic therapy, which carried a certain bleeding risk, should have been avoided. However, from another perspective, this patient presented initially with prominent motor weakness with cortical deficits, which suggested a large initial perfusion deficit. Her transient improvement might also have resulted from partial spontaneous recanalisation or perfusion from collateral circulation, which is often unsustainable for a large perfusion deficit. This observation has been repeatedly reported in the literature. In one survey, among those patients excluded from stroke thrombolysis because of mild symptoms or clinical improvement, up to 50% did not have the expected good recovery, and rapid early improvement in patients with severe neurological deficit, in fact, predicted subsequent deterioration and poor neurological outcomes.[66] In this case, the presence of the radiological hyperdense right MCA sign convinced the stroke physician that this particular patient, indeed, had a high probability of deterioration and that thrombolytic therapy was indicated.

Case Scenario 2

Ms Wu, aged 84 years, with a history of hypertension and hyperlipidaemia, was first admitted for sudden right-sided weakness with power of MRC grade 4/5, which recovered shortly after admission. Her ECG revealed AF, and CT brain was unremarkable. Warfarin was started for secondary prevention in view of her minor stroke and AF. Ten days later, she attended outpatient clinic for her scheduled appointment and reported acute onset of aphasia in the morning, without new limb weakness; her INR was 3.9. Brain CT was repeated and showed an acute infarction in the left internal border-zone region (Plate 35.2). A TCD study revealed a stenotic flow at the left siphon artery through the ophthalmic window (Plate 35.3), while the MCAs could not be insonated because of the poor temporal windows. The ECG and carotid duplex ultrasound were unremarkable. Warfarin was stopped, and substituted with dual antiplatelet therapy with clopidogrel and aspirin. The plan was to continue dual antiplatelet therapy for up to 1 year and then switch back to warfarin.

Comments

This case illustrates the complexity in dealing with more than one possible stroke aetiologies in the same patient. This case is not uncommon, given the high prevalence of intracranial large artery disease in the local population. A local study of consecutive ischaemic stroke patients found the presence of large artery disease in up to 50% of ischaemic stroke patients, and most of these were intracranial large artery diseases.[14] Large artery disease carries the highest early recurrence risk, and pooling results from several stroke cohorts showed that the 3-month recurrence risk was up to 20%.[34] That risk declines with time; in the local study of symptomatic intracranial stenosis, the recurrence risk dropped from 17.1% in the first year to 8.6% in the second year.[14] This reduction in recurrence is supposed to result from the stabilisation of atherosclerotic plaques as well as the development of collaterals after the incident stroke. Anticoagulation was not superior to aspirin in large artery disease, while recent evidence suggests that dual antiplatelet therapy with clopidogrel and aspirin for a short period of time, together with lipid and BP control, could successfully decrease the risk for recurrence. The dual antiplatelet regime also performs better than aspirin alone in AF but is definitely not as effective as anticoagulation.[67] From the bleeding risk perspective, both long-term use of dual antiplatelets and a combination of anticoagulation with antiplatelet drugs in stroke carry high risks and are not supported. In this case, the presence of siphon artery stenosis and a border-zone infarction in its feeding territory suggested large

Table 35.7 Case scenario 3: Baseline blood investigations and tumour marker screening

Blood investigation / tumour marker screening	Data
Haemoglobin (g/dL)	11.5
Platelet count (x 10^9 /L)	157
Sodium (mmol/L)	134
Potassium (mmol/L)	3.5
Urea (mmol/L)	2.7
Creatinine (μmol/L)	62
Albumin (g/L)	36
Globulin (g/L)	37
Bilirubin (μmol/L)	10
Alkaline phosphatase (U/L)	85
Alanine aminotransferase (U/L)	45
Low-density lipoprotein cholesterol (mmol/L)	2.7
CA125 (U/mL)	2642 (Reference range: <35)
Carcinoembryonic antigen (μg/L)	4470 (Reference range: <5)
CA19-9 (U/mL)	261 577 (Reference range: <35)

artery disease as the main aetiology accounting for the rapid sequential stroke. Dual antiplatelet therapy should work better in the short term, but there is no good evidence-based guidance for the duration of treatment. The decision for 1 year of therapy was arbitrarily chosen, based on the risk and benefit assessments.

Case Scenario 3

Ms Wong, aged 63 years, previously healthy, presented with acute confusion without obvious limb weakness. On performing CT brain, multiple acute cerebral infarctions were found over different vascular territories (Plate 35.4). She was afebrile; her chest X-ray, ECG, TCD study, and baseline blood tests were normal. Because this patient developed multiple cerebral infarctions whereas the initial investigations did not identify any significant vascular risk factors, the stroke physician decided to screen for occult malignancy with tumour markers, which were markedly elevated (Table 35.7). Her neurological status deteriorated rapidly and she passed away before a histological diagnosis of her underlying cancer could be confirmed.

Comments

Cancer increases risks for both arterial and venous thrombosis. A local study examining cancer in stroke found that the prevalence of active cancer was 4.8% in acute stroke, and not uncommonly, stroke could be the first presentation of an occult malignancy.[68] Cancer patients tend to have multiple strokes, with fewer background vascular risk factors, and a very high D-dimer.[69] Physicians need to be aware of this association of stroke and cancer, especially in patients with apparently cryptogenic stroke.

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CHAPTER 36

Atrial Fibrillation

Bun SHENG, Tak-kwan KONG

OBJECTIVE

To learn the epidemiology, impact, and management of atrial fibrillation (AF) in old age.

KEY POINTS

- AF is the most common sustained arrhythmia in old age.
- AF in old age is more difficult to manage because of multiple comorbidities, fall risk, and frailty. Older patients have both a higher risk for thromboembolic events from AF and higher major bleeding complications from anticoagulation treatment. They are more sensitive to side-effects of drugs, such as postural hypotension, symptomatic bradycardia, arrhythmia, and organ toxicities. There is also a problem with the underutilisation of oral anticoagulants due to overestimation of fall and bleeding risk.
- The decision to start antithrombotic therapy is based on the assessment of stroke and bleeding risk, and individual patient's preference. Safe and effective antithrombotic therapy in an elderly person with AF requires an individualised approach and comprehensive geriatric assessment (e.g. comorbidities, renal function using Cockcroft-Gault formula, risk of fall, cognition, drug adherence, polypharmacy, and socioeconomic factors) to guide clinical judgement on the benefit-to-risk ratio of anticoagulation in an elderly patient.
- AF significantly increases the risk of stroke, and is associated with heart failure, cognitive impairment, functional decline, and death.
- AF is frequently undiagnosed, and first presentation with complications of AF is common.
- Rate control together with anticoagulation is the mainstay therapeutic strategy in AF.
- Choices of antiarrhythmic agents should take into account the comorbidities of individual patients.
- Anticoagulation with warfarin with a targeted international normalised ratio of 2 to 3 significantly decreases the risk of stroke in AF patients.
- Warfarin dose requirements reduce with age and with impaired renal function.
- Warfarin maintenance doses are lower among Chinese patients compared with Caucasian patients.
- Asians are more prone to bleeding from the use of warfarin.
- Non–vitamin K antagonist oral anticoagulants are now available as alternatives to warfarin for stroke prevention in non-valvular AF.
- Validated assessment tools for stroke and bleeding risks are available to assist decision making in AF.

36.1 Introduction

Atrial fibrillation (AF) is the most common sustained arrhythmia in elderly people. It is associated with an increased risk of stroke (5-fold), heart failure, cognitive impairment, functional decline, and death.[1-4] Yet, AF is frequently undiagnosed in the aged. In the Leiden 85-plus Study conducted in the Netherlands, AF was undetected in 42% of the elders despite the fact that 78% of the elders with undetected AF had had prior contact with general practitioners.[5]

36.1.1 Epidemiology

The prevalence of AF in the US is 1%.[6] It increases significantly with age, from 0.1% in adults aged <55 years to 9.0% in those aged ≥80 years, and is more common in men than women (Figure 36.1[6]). The estimated lifetime risk of developing AF is 26% in men and 23% in women by 80 years of age.[7] In China, two high-quality epidemiological studies reported an age-standardised prevalence of AF at 0.65% and 0.77%.[8,9] A wide range of prevalence estimates was found when comparing more studies in Chinese populations. This variation could be largely explained by the differences in study designs and study populations (Table 36.1).[8-13] Overall, the prevalence of AF appears to be lower in Chinese populations. This observation

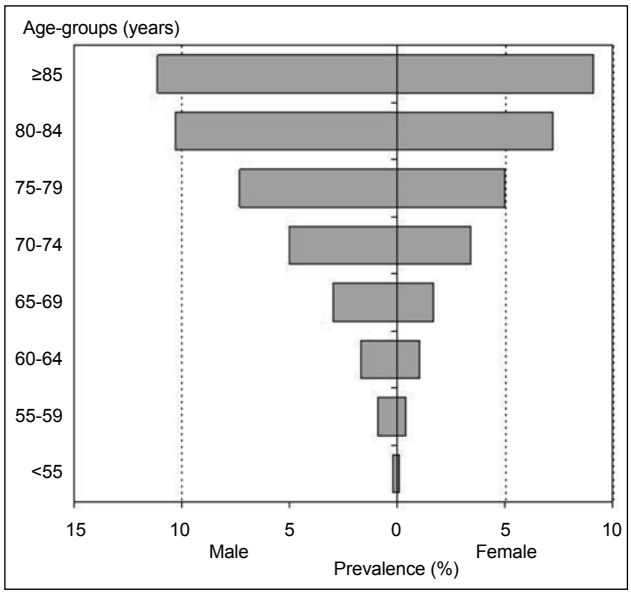

Figure 36.1 Age- and sex-stratified prevalence of atrial fibrillation in a cross-sectional study of 1.89 million adults aged ≥20 years who were enrolled in a large health maintenance organisation in California between July 1996 and December 1997[6]

is also supported by the recently published ASSERT study in which the incidence of AF, detected through an implanted pacemaker, was found to be lower in Chinese patients versus European patients.[14]

36.1.2 Risk Factors and Classification

Apart from age and male gender, the other risk factors for AF include comorbidities such as the components of metabolic syndrome (obesity, hypertension [HT], diabetes mellitus, low high-density lipoprotein cholesterol), obstructive sleep apnoea, hyperthyroidism, valvular heart disease, coronary artery disease, myocardial infarction, cardiomyopathies, heart failure, atrial septal defect, cardiac surgery, chronic obstructive pulmonary disease, chronic kidney disease, excessive alcohol consumption, and strenuous exercise.[15-17] AF can be classified based on either its aetiology, presentation, duration, or intervention (Table 36.2).[17]

36.2 Clinical Presentation

Patients with AF could have a wide variety of

Table 36.1 Summary of studies reporting prevalence of AF in Chinese populations[8-13]

Study	Site	Study design	No. of subjects; age (years)	AF determination	Prevalence of AF
Zhou and Hu, 2008[8]	13 Provinces in China	Population-based epidemiology study with random cluster sampling	29 079; ≥30	12-Lead ECG or known medical record for AF	Overall: 0.65%* M: 0.66%, F: 0.63%
Long et al, 2011[10]	Guangzhou, China	Cross-sectional survey in a community cohort	20 430; ≥50	12-Lead ECG	M: 1.15%, F: 0.66%
Li et al, 2013[9]	10 Populations in China	Population-based epidemiology study with random cluster sampling	19 363; ≥35	12-Lead ECG or known medical record for AF	Overall: 0.77%* M: 0.78%, F: 0.76%
Chien et al, 2010[11]	Taiwan	Cross-sectional survey in a community cohort	3560; ≥35	12-Lead ECG	M: 1.4%, F: 0.7%
Yap et al, 2008[12]	Singapore	Cross-sectional survey in a community cohort	1839; ≥55	12-Lead ECG	Overall: 1.5% M: 2.6%, F: 0.6%
Lok and Lau, 1996[13]	Hong Kong	Cross-sectional survey through a health screening programme in 7 elderly centres	1454; ≥60	12-Lead ECG	1.3%

Abbreviations: AF = atrial fibrillation; ECG = electrocardiogram; F = female; M = male
* Age-standardised prevalence

Table 36.2 Classification of AF[17]

Type	Definition
Based on aetiology	
Valvular AF	With rheumatic heart disease or a prosthetic heart valve
Non-valvular AF	Without rheumatic heart disease or a prosthetic heart valve
Secondary AF	With associated cardiovascular disease or other identifiable causes / risk factors
Lone AF	With a structurally normal heart and without identifiable cause for AF
Based on presentation, duration, and intervention	
First-diagnosed AF	Patient who presents with AF for the first time
Paroxysmal AF	Self-terminating AF, usually within 48 hours
Persistent AF	Sustained AF >7 days, or AF that requires cardioversion
Long-standing persistent AF	AF lasted ≥1 year when a rhythm control strategy is adopted
Permanent AF	Failed cardioversion, or decided not to attempt restoring the sinus rhythm

Abbreviation: AF = atrial fibrillation

symptoms such as palpitations, chest pain, dyspnoea, dizziness, syncope, and fatigue, at some point during the course of AF. Lévy et al[18] reported the prevalence of different symptoms related to AF and noticed that these were more common in paroxysmal AF (Figure 36.2). However, these figures were reported from patients under the care of cardiologists and tended to overestimate the disease burden. Many AF patients are asymptomatic. In the epidemiological study on AF in China, Li et al[9] found that one-third of the AF patients were unrecognised. Similarly, while 69% of patients experienced some symptoms related to AF since diagnosis in the Euro Heart Survey on AF, 54% were asymptomatic at the time of survey.[19] Apart from arrhythmia-related symptoms, patients with AF could also first present with embolic complications such as stroke and peripheral embolism. About one-fifth of AF cases associated with acute stroke were cases of first-diagnosed AF.[20]

The presentation of AF in elderly patients differs from younger patients. Analysis of the EORP-AF general pilot registry[21] revealed that elderly patients (aged ≥75 years) more often had persistent or permanent AF, and had a higher prevalence of comorbidities (coronary artery disease, chronic heart failure with preserved ejection fraction, chronic kidney disease, chronic obstructive pulmonary disease, valvular heart disease, and HT). Previous haemorrhagic events and transient ischaemic attack (TIA) were reported more often in elderly patients. With respective to symptoms, elderly patients reported palpitations less frequently, but reported dyspnoea more often. It is important to recognise that elderly patients with AF may manifest with cognitive impairment or dementia secondary to reduced cerebral perfusion or recurrent thromboembolism.[3] Not only will this identify AF as a potentially treatable cause of cognitive impairment, but also this will alert the clinician to assess the patient's ability to handle medication and the need for family involvement in drug adherence.

Many patients with AF do not consult for AF-related symptoms and remain unrecognised even with regular medical services. AF is suspected clinically from an irregularly irregular pulse, which can be mimicked by multifocal atrial tachycardia or atrial flutter with variable block. The clinical suspicion of AF is readily confirmed with an electrocardiogram (ECG). Regularisation of AF (i.e. regular rhythm on pulse examination and ECG recording) should alert one to the development of complete heart block with escape rhythm in a patient with AF as seen in digoxin overdose. It has been shown that in primary care, a simple opportunistic screening by examining the pulse, and a follow-up ECG if appropriate, can increase the chances of AF detection by more than 50%.[22] Since AF is common in elderly people and is a major risk factor for stroke, the incidence of which could be dramatically reduced by anticoagulation, this case-finding strategy is worthy of consideration.

36.3 Investigations

A 12-lead ECG is usually sufficient to confirm the diagnosis of AF, which typically shows irregular RR intervals, absence of regular P waves, and atrial cycle length (if visible) of <200 ms (>300 beats per minute [bpm]). In patients with symptoms suspicious of

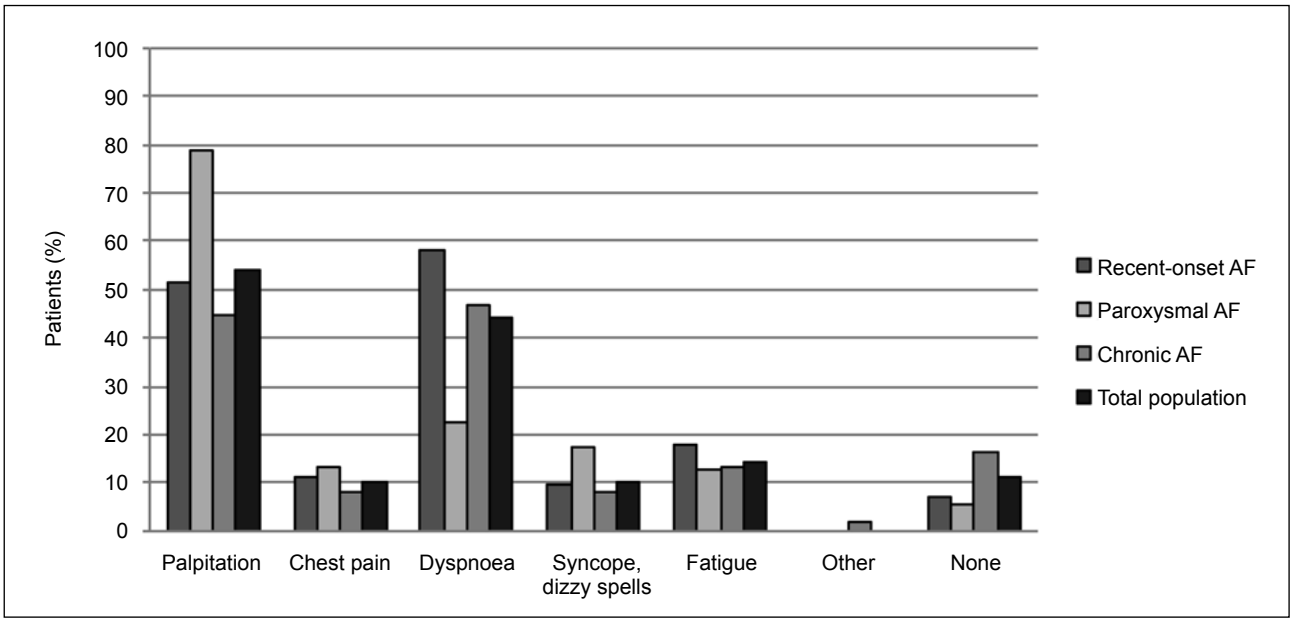

Figure 36.2 Symptoms of atrial fibrillation (AF) in the ALFA study[18]

paroxysmal AF, continuous ECG monitoring is necessary to establish the diagnosis. A 24-hour or 48-hour Holter can pick up an arrhythmia if it is frequent; however, for infrequent events, prolonged monitoring with external or implantable loop recorders may be required.[23] Continuous ECG recording is also useful to guide therapeutic options if there is evidence of both bradyarrhythmia and tachyarrhythmia in the same patient.

An echocardiogram should be arranged to look for evidence of valvular heart disease, and to assess the left atrial size and left ventricular function. An enlarged left atrium predicts AF recurrence, while an impaired left ventricular systolic function is associated with higher risk of stroke, hospitalisation, and mortality; both conditions have prognostic significance.[24]

Both overt and subclinical hyperthyroidism are risk factors for AF, and AF occurs in 10-20% of elderly patients with hyperthyroidism.[25,26] Therefore, serum thyroid-stimulating hormone should be measured in all patients with new-onset AF to rule out thyroid disease. The classic thyrotoxic symptoms seen in younger adults are frequently absent in elderly patients, and AF or heart failure could be the only clinical manifestations of hyperthyroidism in old age.[27,28]

36.4 Management

36.4.1 Rate Control Versus Rhythm Control

AF increases the risk of stroke, hospitalisation, and cardiovascular death. It has once been presumed that these adverse outcomes might be reversed after reverting AF into sinus rhythm. However, in the six completed clinical trials comparing this rhythm-control strategy against rate-control strategy, including the large AFFIRM study which enrolled 4060 patients, failed to demonstrate a reduction in any of these three clinical endpoints in the rhythm-control group (Table 36.3).[29-35] Instead, the AFFIRM study reported a trend towards increased mortality in association with rhythm control (P=0.08).[30]

A study comparing the effect of rate- versus rhythm-control strategies on outcomes in a propensity-matched cohort of 2248 AFFIRM participants of 70-80 years of age concluded that a rate-control strategy using beta-blockers, digoxin, diltiazem, and verapamil was superior in reducing the risk of mortality (18% vs. 23%; P=0.010) and hospitalisation (61% vs. 68%; P<0.001) for patients with AF in the eighth decade of life.[36]

Even in patients with heart failure, as in the AF-CHF (Atrial Fibrillation and Congestive Heart Failure) study, no difference in worsening of symptoms of heart failure and survival was found between the rhythm- and rate-control arms, despite the fact that AF is a well-known cause for heart failure decompensation and a predictor for mortality in this high-risk group.[34]

All these trials had a common important limitation: lack of an effective antiarrhythmic agent to maintain sinus rhythm in the rhythm-control arm (percentage of patients maintained in sinus rhythm was 23-64%).[35] In addition to having limited efficacy, the currently available antiarrhythmic agents also carry many side-effects, and are potentially pro-arrhythmic for ventricular arrhythmia. Therefore, until a new antiarrhythmic agent with better efficacy and lesser toxicity is developed, rate control should be the mainstay treatment strategy for AF. In addition, anticoagulation should be continued, even in patients being restored to sinus rhythm, since the majority of the stroke or embolic events reported in the trials occur in patients who are taken off from anticoagulation, or inadequately anticoagulated.

Table 36.3 Clinical trials on rate control versus rhythm control in AF (adapted from Chinitz et al[35]) [reproduced with permission]

Trial	No. of patients	Mean (± SD) age (years)	Mean follow-up period (years)	% Sinus rhythm in rhythm control	Stroke or thromboembolism	Hospitalisation	Mortality
PIAF,[29] 2000	252	61 ± 10	1	56% (1 Year)	Not reported	24% vs. 69%, P=0.001	Not reported
AFFIRM,[30] 2002	4060	69.7 ± 9.9	3.5	62.6% (5 Years)	5.5% vs. 7.1%, P=0.79	73% vs. 80.1%, P<0.001	25.9% vs. 26.7%, P=0.08
RACE,[31] 2002	522	68 ± 9	2.3	39% (End of follow-up)	5.5% vs. 7.9%, P=NS	Not reported	7% vs. 6.8%, P=NS
STAF,[32] 2003	200	66 ± 8	1.6	23% (3 Years)	2% vs. 5%, P=NS	26% vs. 54%, P<0.001	8% vs. 4%, P=NS
HOT CAFE,[33] 2004	205	61 ± 11	1.7	63.5% (End of follow-up)	1% vs. 2.9%, P=NS	12% vs. 74%, P<0.001	1% vs. 2.9%, P=NS
AF-CHF,[34] 2008	1376	67 ± 1	3.1	42% (End of follow-up)	4% vs. 3%, P=NS	59% vs. 64%, P=0.06	25% vs. 27%, P=0.59

Abbreviations: AF = atrial fibrillation; NS = not significant; SD = standard deviation

Catheter ablation of AF has been reported to be superior to pharmacological therapy in restoring the sinus rhythm.[37] Results from an international multicentre registry with more than 1000 patients revealed that sinus rhythm was maintained in 85% and 72% of patients with paroxysmal AF and persistent AF, respectively, and the stroke and death rates were significantly lower than in the medically treated patients in the Euro Heart Survey cohort.[38] This preliminary suggestion that rhythm control by catheter ablation may reduce stroke risk and death warrants confirmation from prospective studies.

36.4.2 Choices of Antiarrhythmic Agents for Rate Control

Rate control together with adequate anticoagulation is the mainstay treatment in AF. In the RACE II study, strict control of resting heart rate at physiological level (<80 bpm) was compared with lenient rate control at <110 bpm. It was observed that lenient rate control was as effective as strict rate control, was easier to achieve, and required fewer medications.[39] Therefore, in most clinical situations, a lenient rate control strategy is adequate for the symptomatic treatment of AF.

Choice of a pharmacological agent depends on the patient's tolerance to that particular agent, the therapeutic response, and existing comorbidities. Beta-blockers and non-dihydropyridine calcium channel antagonists (verapamil, diltiazem) are commonly used for heart rate control in AF.[17] They are particularly useful in patients with HT and ischaemic heart diseases since these diseases also benefit from the treatment. However, beta-blockers and non-dihydropyridine calcium channel antagonists may not be tolerated by patients with severe heart failure or systolic dysfunction, and in such cases digoxin or amiodarone could be considered.

Digoxin slows ventricular response to AF through vagal tone enhancement, but has limited efficacy in states of high sympathetic tone such as exercise, paroxysmal AF, fever, thyrotoxicosis, acute volume loss, and postoperative state.[40] Digoxin is more suitable for sedentary patients with permanent AF. Digoxin should be used with caution in elderly people, in whom renal impairment and electrolyte disturbance (hypokalaemia) are common and who are predisposed to digoxin toxicity, a condition which can present non-specifically with anorexia, nausea, vomiting, malaise, weight loss, falls, weakness, visual disturbances, delirium and neuropsychiatric symptoms.[40] As digoxin is a drug with a narrow therapeutic window, appropriate dosing, therapeutic drug monitoring and vigilant clinical monitoring are required for its safe and effective use.[38] A digoxin maintenance dose of 125 μg daily is recommended for most elderly people with glomerular filtration rate (GFR) of >50 ml/min, and lower doses (≤62.5 μg daily) are recommended in those with lower GFR and heart failure with multiple risk factors for digoxin accumulation.[40] Based on evidence of maintained clinical efficacy of digoxin at a lower serum digoxin concentration (SDC) and higher risk of toxicity and mortality at higher SDC, the therapeutic range for SDC has been revised to a lower level in heart failure (0.6-1.2 nmol/L).[40-43] If the heart rate has not been controlled adequately with digoxin monotherapy, combination therapy with digoxin and a beta-blocker is preferred to escalating the digoxin dose to a higher level. Enhanced survival with the digoxin-carvedilol combination has been demonstrated in a retrospective analysis of the US Carvedilol Heart Failure Trials Program.[44] Amiodarone raises plasma level of digoxin and increases the risk of digoxin toxicity. Patients on digoxin-amiodarone combination should therefore be monitored closely.

In patients who are very resistant to rate control even with different combinations of pharmacological agents, referral to a cardiologist for atrioventricular nodal ablation and pacemaker implantation can be considered although it is seldom necessary in elderly patients. In rate control of AF, one should watch out for bradycardia secondary to drug treatment. In some patients, the fast heart rate cannot be controlled without causing bradycardia. Under such circumstances, 'back up' cardiac pacing may be necessary. A 24-hour Holter is often necessary for a more detailed assessment.

36.4.3 Paroxysmal AF

Pharmacological therapy is often unsuccessful in maintaining sinus rhythm in paroxysmal AF, a condition which is usually more symptomatic than permanent AF. Antiarrhythmic therapy could be targeted towards patients in whom AF recurs infrequently and who can tolerate the antiarrhythmic therapy.[17] The use of flecainide or propafenone is limited to patients with structurally normal hearts and without coronary heart disease, because they increase mortality in patients with myocardial infarction or other abnormal structural heart diseases.[45] Studies have shown that older age is an independent predictor of adverse events with flecainide and propafenone, including death, with a relative risk of 1.3 per decade of age. Therefore, these drugs should be avoided in elderly patients.[46] Sotalol is primarily excreted renally, and given the higher prevalence of renal dysfunction in older patients, this drug should be either avoided or its doses renally adjusted. Sotalol should be avoided in elderly women and heart failure patients because of a greater risk of developing QT prolongation and torsades de pointes.[47]

Amiodarone could be used both for cardioversion and maintenance of sinus rhythm. Amiodarone has many side-effects, including disruption of normal thyroid function; thus, regular monitoring of thyroid function is mandatory when using amiodarone. In one study, long-term amiodarone was associated with development of hypothyroidism in 15-20% of elderly patients and hyperthyroidism in 1-5% of patients.[48] The most fearful adverse effect of amiodarone, however, was pulmonary toxicity, which occurs in 2% of patients. It is recommended to perform a pretreatment chest X-ray and lung function test that includes diffusing capacity of the lung for carbon monoxide, and then regular annual chest X-ray surveillance as long as a patient is on amiodarone treatment.[49] Since the half-life of amiodarone is very long, its effect may take weeks to wear off after cessation. It has therefore been suggested that the lowest possible maintenance doses, probably 100 mg per day, should be prescribed in elderly patients to reduce the incidence of dose-dependent adverse effects of amiodarone, in particular symptomatic bradycardia.[50] Dronedarone is a newer agent related to amiodarone, with a much improved safety profile and pharmacokinetic properties. However, dronedarone should be avoided in patients with class IV heart failure.

Catheter ablation could be considered in symptomatic paroxysmal AF, particularly those with dilated left atrium and impaired systolic function.[51]

Table 36.4a $CHADS_2$[52] and CHA_2DS_2-VASc[53] stroke / TE risk stratification schemes for non-valvular AF
(Top) Risk factors of stroke and scoring rules for calculating the CHADS2 score.[52] (Bottom) Risk factors of TE and scoring rules for calculating the CHA_2DS_2-VASc score[53] (reproduced with permission)

Item	Points
Congestive heart failure	1
Hypertension	1
Age ≥75 years	1
Diabetes mellitus	1
Stroke / transient ischaemic attack	2

Item	Points
Congestive heart failure	1
Hypertension	1
Age ≥75 years	2
Diabetes mellitus	1
Stroke / transient ischaemic attack	2
Vascular disease (prior myocardial infarction, peripheral vascular disease, aortic plaque)	1
Age 65-74 years	1
Sex category: female	1

Abbreviations: AF = atrial fibrillation; TE = thromboembolism (ischaemic stroke, pulmonary embolism, or peripheral embolism)

36.4.4 Anticoagulation

Approximately 5% of patients with AF develop stroke or systemic embolism each year. Risk stratification scoring systems such as $CHADS_2$ or the more detailed CHA_2DS_2-VASc (Table 36.4a[52,53]) have been widely used to estimate the embolic risk in non-valvular atrial fibrillation (NVAF), and accordingly, to recommend anticoagulation for intermediate- and high-risk patients.[52,53] These two risk stratification schemes have been validated in non-anticoagulated Caucasian and Chinese patients with AF (Table 36.4b).[52-55] A validation study on Chinese AF patients in Beijing[54] observed that the CHA_2DS_2-VASc score performed better than the $CHADS_2$ score for identifying low-

Table 36.4b $CHADS_2$ and CHA_2DS_2-VASc stroke / TE risk stratification schemes for non-valvular AF compared and validated in real world cohorts of non-anticoagulated Caucasian and Chinese AF patients[52-55]

Study		Annual stroke[52,55] / TE[53,54] rate (%)					
		Gage et al[52*]	Lip et al[53*]	Guo et al[54†]		Siu et al[55‡]	
Population		American	European	Chinese (Beijing)		Chinese (Hong Kong)	
No.		1733	1084	885		3881	
Age (mean ± SD or range) [years]		81 (65-95)	66 ± 14	77 (66-84)		77.2 ± 13.3	
Stroke / TIA risk category / score		$CHADS_2$	CHA_2DS_2-VASc	$CHADS_2$	CHA_2DS_2-VASc	$CHADS_2$	CHA_2DS_2-VASc
Low	0	1.9	0	0	0	5.44	2.41
Intermediate	1	2.8	0.7	2.9	0.9	8.59	6.64
High	2	4.0	1.9	4.9	1.7	11.24	7.84
	3	5.9	4.7	3.4	2.7	11.86	9.56
	4	8.5	2.3		1.8		11.58
	5	12.5	3.9	4.9	8.8	14.06	12.69
	6	18.2	4.5				
	7		10.1		9.0		13.18
	8		14.2				
	9		100				

Abbreviations: AF = atrial fibrillation; SD = standard deviation; TE = thromboembolism (ischaemic stroke, pulmonary embolism, or peripheral embolism); TIA = transient ischaemic attack
* Rates adjusted for aspirin prescription
† Rates include antiplatelet prescription
‡ Rates without aspirin prescription

risk subjects, and had a better predictive ability for stroke and thromboembolic events (Table 36.4b). This validation study on Chinese AF patients only included a small number in the low risk range and found a thromboembolic risk comparable to non-Asians. Recently, a large Chinese AF cohort study from Hong Kong[55] with a greater number of low-risk patients (CHA_2DS_2-VASc score 0 and 1) revealed that the stroke risk among them were higher than expected (Table 36.4b). The higher stroke risk in AF patients for Chinese compared with Caucasians, as stratified by the CHA_2DS_2-VASc score, may indicate a missing component risk factor in the score that contributes to stroke in Chinese.[56] Examples may include factors such as age 55 to 64 years, undiagnosed and / or suboptimally treated HT and hyperthyroidism, and high prevalence of noncardioembolic stroke due to intracranial atherosclerosis in Chinese.[56]

A meta-analysis of 29 randomised trials published between 1989 and 2007 showed that warfarin, with a targeted international normalised ratio (INR) between 2 and 3, reduced stroke risk in patients with AF by 64%, with absolute risk reduction of 2.7% for primary prevention and 8.4% for secondary prevention.[57] There have been concerns about whether this benefit can be extrapolated to older age-groups, who have been identified to be at higher risk for warfarin-associated haemorrhage, because patients >75 years were under-represented in early clinical trials. A study in the US among a cohort of elderly patients (aged ≥65 years; mean age, 77 years) with AF initiated on warfarin treatment revealed a relatively high major bleeding rate of 7.2% per 100 person years.[58] However, a UK study (Birmingham Atrial Fibrillation Treatment of the Aged Study [BAFTA]) among elderly patients (aged ≥75 years; mean age, 81.5 years) established the superior efficacy of warfarin over aspirin as a stroke prevention strategy in this age-group of elderly patients with AF; the major haemorrhage rate associated with warfarin (1.9% per year) was similar to that with aspirin (2.0% per year).[59] The lower bleeding rate associated with warfarin in the UK study compared with the US study has been attributed to selection bias of elderly patients who were healthier or were not using antiplatelet concomitantly in the UK study.[60]

36.4.4.1 Warfarin

Until recently, warfarin, a vitamin K antagonist, was the only oral anticoagulant in clinical use. Warfarin has a number of limitations necessitating its cautious use in clinical practice. It has a narrow therapeutic window so that under- and over-anticoagulation may expose an individual to risks of thromboembolism and haemorrhage, respectively. Regular blood taking is, therefore, required to monitor the therapeutic effect of warfarin, and ensure that the INR is within the targeted range of 2 to 3. In order to maintain the targeted INR, dosage adjustment may be required and that could confuse elderly patients who are not accustomed to frequent change of drug regimens. Misunderstanding of the warfarin prescription is a major risk for medication error and iatrogenesis. The dose-response relationship of warfarin cannot be reliably predicted. It has a delayed time to therapeutic onset and offset. The metabolism of warfarin is under the influence of genetic polymorphism in two genes, the cytochrome P450 family 2 subfamily C polypeptide 9 enzyme (*CYP2C9*) gene and the vitamin K epoxide reductase complex 1 (*VKORC1*) gene, resulting in wide interpersonal variability.[61]

As a vitamin K antagonist, the anticoagulation effect of warfarin is also affected by the daily foods rich in vitamin K. Patients taking warfarin have to follow a relatively stable diet, with similar amounts of vitamin K–rich green and leafy vegetables in each meal. Drug-drug interaction is another major concern in the local elderly population. Apart from western medicine, some traditional Chinese medicines and herbal foods may also interact with warfarin. For example, combination of warfarin with ginkgo and garlic may give rise to bleeding. However, the true risks of these interactions and effects are difficult to characterise due to the limited number and nature of existing reports (Table 36.5).[62,63] Since the use of warfarin is associated with an increased risk of bleeding when creatinine clearance (CrCl) is <30 ml/min, dose reduction and close monitoring are required in patients with CrCl <30 ml/min.[64]

Table 36.5 Herbs and supplements that can interact with warfarin[62,63]

Potentially increase the effect of warfarin	Potentially reduce the effect of warfarin
Feverfew (*Tanacetum parthenium*) 甘菊	Coenzyme Q10
Garlic (*Allium sativum*) 大蒜	Ginseng 人蔘
Ginger (*Zingiber officinale*) 薑	Green tea (*Camellia sinensis*)
Ginkgo (*Ginkgo biloba*) 白果	St. John's wort 金絲桃
Dan shen 丹參	
Devil's claw (*Harpagophytum procumbens*) 魔鬼之爪	
Dong quai (*Angelica sinensis*) 當歸	
Vitamin E	
Papain (papaya extract)	

It should be noted that because of increased sensitivity to warfarin with ageing, the warfarin dose requirements reduce with age. A local study confirmed this finding and also revealed that warfarin maintenance doses are lower among Chinese patients compared with Caucasian patients: the average daily warfarin doses required to achieve a mean target INR of 2.2 among Chinese patients in a local hospital was found to be 2 mg, 2.5 mg, and 4 mg for patients aged >75 years, <75 years, and <40 years, respectively.[65] Data analyses have shown that Asians are more prone to bleeding from the use of warfarin, even at a lower INR.[66] The risk of intracranial haemorrhage increased to 4-fold in Asians compared to Caucasians when warfarin was used under a similar intensity of anticoagulation.[67]

36.4.4.2 Non–vitamin K Antagonist Oral Anticoagulants

In the last decade, several non–vitamin K antagonist oral anticoagulants (NOAC) were developed from two drug classes: direct thrombin inhibitor (dabigatran) and factor Xa inhibitor (rivaroxaban, apixaban, edoxaban). A comparison of clinical trials and important clinical end-points involving these NOAC is summarised in Table 36.6.[68-72] These drugs are at least non-inferior to warfarin in their efficacy, and superior to warfarin in terms of fewer incidents of clinically significant bleeding (especially intracerebral haemorrhage), wider therapeutic window, fixed dosage, less complex pharmacodynamics, little need for monitoring, very few drug-drug interactions, and no known drug-food interaction. Counterbalancing these advantages are the challenges of monitoring drug adherence, higher expense, lack of easily available laboratory monitoring tests of anticoagulant activity, lack of antidote in the event of bleeding complications, possible higher rates of gastrointestinal haemorrhage, and lack of long-term experience with these drugs, including the largely unknown risk of drug-drug and drug-food interactions.[73-75] Frail elderly people were not represented in NOAC-investigating trials because of exclusion criteria, and only one-third of patients were aged >75 years.[74] A subgroup analysis from the dabigatran-investigating trial indicated that elderly patients might have a higher risk for extracranial bleeding complications with NOAC than with warfarin. Further concerns about the use of NOAC in elderly people include the high prevalence of renal impairment in AF patients aged >75 years.[74] A clinical audit of bleeding events with dabigatran highlighted the difficulty in extrapolating the trial data into clinical practice, and alerted practitioners to bleeding complications despite the use of reduced drug dose in frail elderly patients with poor renal function and low bodyweight.[76] Reports of life-threatening haemorrhages with the first-licensed NOAC, dabigatran, have led to recommendations for more vigilant monitoring of renal function,[77,78] which is particularly important in elderly people aged >75 years.

NOAC should not be prescribed to patients with moderate-to-severe renal impairment. Based on clinical trial exclusion criteria, the CrCl threshold below which use of dabigatran, rivaroxaban, and edoxaban are to be avoided is 30 ml/min, and that for apixaban is 25 ml/min.[79] Warfarin remains the drug of choice among patients with end-stage kidney disease, including those requiring haemodialysis.

36.4.5 Percutaneous Left Atrial Appendage Occlusion for Stroke Prevention in AF

It has been shown that up to 90% of the thrombi that are derived from the left atrium are formed in the left atrial appendage (LAA). Thus LAA occlusion emerges as an innovative strategy for stroke prevention in AF. Its efficacy was found to be comparable with warfarin in a randomised study.[80] Overall, the incidence of stroke after LAA occlusion was 1.4% in a recent systematic review.[81] Percutaneous LAA occlusion could be an alternative to anticoagulation for stroke prevention.

36.5 Antithrombotic Therapy in AF: Current Recommendations

Decision of antithrombotic therapy in AF is based on the assessment of stroke and bleeding risks, as well as patient's personal preference of treatment choices. The following recommendation is adapted from the guideline published by the European Society of Cardiology (ESC) in 2016 (Figure 36.3).[72]

36.5.1 Valvular AF

All patients with valvular AF (AF in the context of rheumatic mitral stenosis and prosthetic valve) should receive dose-adjusted warfarin, which is the only oral anticoagulant available for this group of patients.

36.5.2 Non-valvular AF

Recommendation for antithrombotic therapy in patients with NVAF is based on their stroke risk. $CHADS_2$ score and CHA_2DS_2-VASc score are the two commonly used validated stroke risk assessment tools, with the latter scoring better in the low-risk category of patients (e.g. in those with $CHADS_2$=0). Stroke, TIA or systemic emboli, and age ≥75 years are the major risk factors for stroke; the other components in CHA_2DS_2-VASc score are non-major risk factors.

36.5.2.1 True Low-risk AF

The true low-risk group includes patients aged <65 years and patients with lone AF, irrespective of

Table 36.6 Summary of clinical trials involving NOAC versus warfarin for stroke prevention in non-valvular AF (adapted from Kirchhof et al[72]) [reproduced with permission]

	Dabigatran (RE-LY, 2009)[68]		
Drug characteristics			
Mechanism	Direct thrombin inhibitor		
Bioavailability (%)	6		
Time to peak levels (hours)	3		
Excretion	80% Renal		
Dose	150 mg bd or 110 mg bd (non-FDA approved)		
Special consideration	Intestinal absorption is pH-dependent, it could be reduced in patients taking proton pump inhibitors		
Study characteristics			
Study design	Randomised, open-label (warfarin)		
No. of patients	18 111		
Follow-up period (years)	2		
Randomised groups	Dose-adjusted warfarin vs. dabigatran (150 mg bd, 110 mg bd)		
Baseline patient characteristics			
Age (years)	71.5 ± 8.7 (mean ± SD)		
Male sex (%)	63.6		
Mean CHADS2	2.1		
Outcomes (% per year)			
	Warfarin	Dabigatran 150	Dabigatran 110
	n=6022	n=6076	n=6015
		(RR, 95% CI; P value)	(RR, 95% CI; P value)
Stroke / systemic embolism	1.69	1.11 (0.66, 0.53-0.82; P for superiority <0.001)	1.53 (0.91, 0.74-1.11; P for non-inferiority <0.001)
Ischaemic stroke	1.2	0.92 (0.76, 0.60-0.98; P=0.03)	1.34 (1.11, 0.89-1.40; P=0.35)
Haemorrhagic stroke	0.38	0.10 (0.26, 0.14-0.49; P<0.001)	0.12 (0.31, 0.17-0.56; P<0.001)
Major bleeding	3.36	3.11 (0.93, 0.8-1.07; P=0.31)	2.71 (0.80, 0.69-0.93; P=0.003)
Gastrointestinal bleeding	1.02	1.51 (1.50, 1.19-1.89; P<0.001)	1.12 (1.10, 0.86-1.41; P=0.43)
Myocardial infarction	0.64	0.81 (1.27, 0.94-1.71; P=0.12)	0.82 (1.29, 0.96-1.75; P=0.09)
Death from any cause	4.13	3.64 (0.88, 0.77-1.00; P=0.051)	3.75 (0.91, 0.80-1.03; P=0.13)

Abbreviations: AF = atrial fibrillation; BW = body weight; CI = confidence interval; CrCl = creatinine clearance; FDA = Food and Drug Administration; HR = hazard ratio; IQR = interquartile range; ITT = intention-to-treat; NOAC = non–vitamin K antagonist oral anticoagulants; RR = risk ratio; SD = standard deviation

gender. No antithrombotic therapy is required in these patients. Therefore, when CHA_2DS_2-VASc score equals 1 and female gender is the only risk factor, no antithrombotic therapy is recommended.

36.5.2.2 AF with One Non-major Risk Factor Other Than Female Gender (i.e. CHA_2DS_2-VASc score=1)

Anticoagulation should be considered, balancing the potential benefit, bleeding risk, and patient preference. Anticoagulation with dose-adjusted warfarin (target INR, 2.0-3.0) or one of the NOAC is the preferred antithrombotic therapy of choice.

36.5.2.3 AF with One Major Risk Factor or More Than One Non-major Risk Factor (i.e. CHA_2DS_2-VASc score ≥2)

Anticoagulation with dose-adjusted warfarin (target INR, 2.0-3.0) or one of the NOAC is the preferred antithrombotic therapy of choice.

Rivaroxaban (ROCKET AF, 2011)[69]		Apixaban (ARISTOTLE, 2011)[70]		Edoxaban (ENGAGE AF, 2013)[71]		
Factor Xa inhibitor		Factor Xa inhibitor		Factor Xa inhibitor		
60-80		50		62		
3		3		1-2		
2/3 Liver, 1/3 renal		25% Renal, 75% faecal		50% Renal		
20 mg Daily (15 mg daily if CrCl 30-49 ml/min)		5 mg bd (2.5 mg bd if 2 out of 3 of the following present: age ≥80 years, BW ≤60 kg, serum creatinine ≥133 µmol/L)		60 mg Daily (30 mg daily if CrCl of 30-50 ml/min or BW <60 kg)		
-		-		-		
Randomised, double-blind		Randomised, double-blind		Randomised, double-blind		
14 264		18 201		21 105		
1.9		1.8		2.8		
Dose-adjusted warfarin vs. rivaroxaban 20 mg daily		Dose-adjusted warfarin vs. apixaban 5 mg bd		Dose-adjusted warfarin vs. edoxaban (60 mg daily, 30 mg daily)		
73 (65-78) [median (IQR)]		70 (63-76) [median (IQR)]		72 (64-78) [median (IQR)]		
61.3		64.5		61.9		
3.5		2.1		2.8		
Warfarin	Rivaroxaban	Warfarin	Apixaban	Warfarin	Edoxaban 60	Edoxaban 30
n=7133	n=7131	n=9081	n=9120	n=7036	n=7035	n=7034
	(HR, 95% CI; P value)		(HR, 95% CI; P value)		(HR, 95% CI; P value)	(HR, 95% CI; P value)
2.4	2.1 (0.88, 0.75-1.03; P for non-inferiority <0.001, P=0.12 for superiority) [ITT]	1.6	1.27 (0.79, 0.66-0.95; P<0.001 for non-inferiority, P=0.01 for superiority)	1.5	1.18 (0.79, 0.63-0.99; P<0.001 for non-inferiority)	1.61 (1.07, 0.87-1.31; P=0.005 for non-inferiority)
1.42	1.34 (0.94; 0.75-1.17; P=0.581)	1.05	0.97 (0.92, 0.74-1.13; P=0.42)	1.25	1.25 (1.00; 0.83-1.19; P=0.97)	1.77 (1.41; 1.19-1.67; P<0.001)
0.44	0.26 (0.59; 0.37-0.93; P=0.024)	0.47	0.24 (0.51, 0.35-0.75; P<0.001)	0.47	0.26 (0.54; 0.38-0.77; P<0.001)	0.16 (0.33; 0.22-0.50; P<0.001)
3.4	3.6 (P=0.58)	3.09	2.13 (0.69, 0.60-0.80; P<0.001)	3.43	2.75 (0.80; 0.71-0.91; P<0.001)	1.61 (0.47; 0.41-0.55; P<0.001)
2.2	3.2 (P<0.001)	0.86	0.76 (0.89, 0.70-1.15; P=0.37)	1.23	1.51 (1.23; 1.02-1.50; P=0.03)	0.82 (0.67; 0.53-0.83; P<0.001)
1.1	0.9 (0.81; 0.63-1.06; P=0.12)	0.61	0.53 (0.88, 0.66-1.17; P=0.37)	0.75	0.70 (0.94; 0.74-1.19; P=0.60)	0.89 (1.19; 0.95-1.49; P=0.13)
2.2	1.9 (0.85; 0.70-1.02; P=0.07)	3.94	3.52 (0.89, 0.80-0.99; P=0.047)	4.35	3.99 (0.92; 0.83-1.01; P=0.08)	3.8 (0.87; 0.79-0.96; P=0.006)

36.5.2.4 Antiplatelets in AF

In AF, anticoagulant therapy prevents stroke better than single or dual antiplatelet therapy with aspirin and clopidogrel. Antiplatelet therapy increases bleeding risk, especially dual antiplatelet therapy, with bleeding rates that are similar to those on anticoagulants. Thus, antiplatelet therapy cannot be recommended for stroke prevention in AF patients.

36.5.2.5 Bleeding Risk Assessment

An assessment of major bleeding is advisable. Among the several assessment tools, HAS-BLED provides a simple calculation of bleeding risk and correlates well with intracerebral haemorrhage (Table 36.7).[82,83] The HAS-BLED score was validated in Chinese AF patients and found to be related to major bleeding events, with a modest predictive value,[54] and had the best predictive and discriminatory ability for major bleeding and intracranial haemorrhage in Chinese AF patients.[84] Bleeding risk assessment should not be used to exclude anticoagulation therapy; an informed assessment assists decision making and helps to alert clinicians about correctable risks of bleeding.

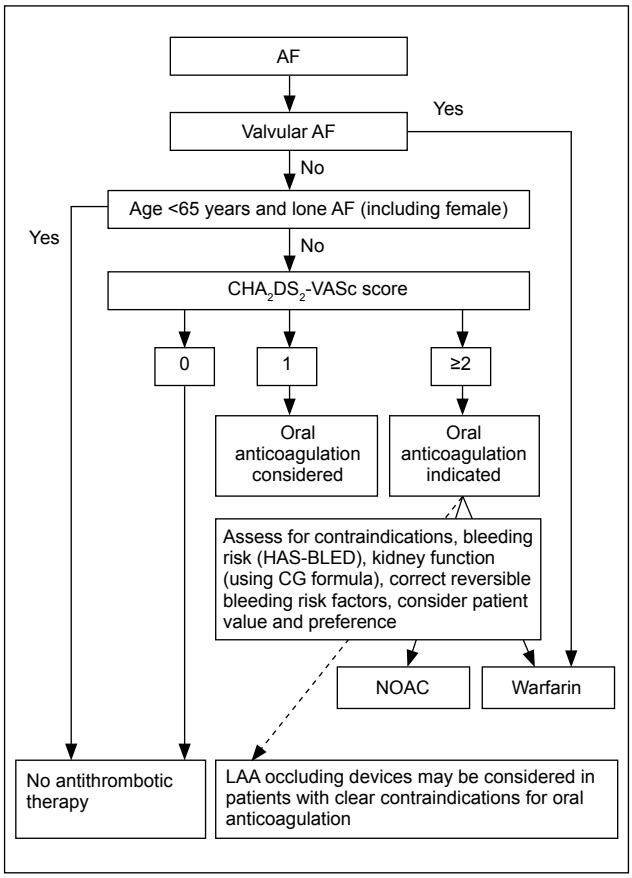

Figure 36.3 Flowchart of choice of antithrombotic therapy in AF (adapted from Kirchhof et al[81])
Abbreviations: AF = atrial fibrillation; CG = Cockcroft-Gault; LAA = left atrial appendage; NOAC = non–vitamin K antagonist oral anticoagulant

36.5.2.6 Special Population Groups

Asian Patients

Current guidelines for the use of warfarin and NOAC in patients with AF are derived mostly using a Caucasian population. The 2016 ESC guideline acknowledged gaps in evidence for non-Caucasian population.[81]

In multinational studies, Asian AF patients, when compared with non-Asian AF patients, were more likely to have a history of prior stroke / TIA, lower body weights, more renal impairment, more intracranial macro- and micro-angiopathy, and at higher risk of warfarin-related intracranial bleeding.[85] Since macro- and micro-angiopathies are more prevalent in Asians than in non-Asians, AF-unrelated stroke / TIA is more likely to occur in Asian AF patients than in non-Asian AF patients.[85] The clinical relevance is that Asian AF patients can have AF-unrelated strokes despite adequate anticoagulation (see 35.10 Case Scenario 2).

NOAC could be particularly appealing to Asian patients because of the higher bleeding complications from warfarin in the Asian population. The superiority of NOAC over warfarin has been demonstrated in an Asian subgroup analysis of NOAC trials.[59] The optimal use of NOAC in Asian patients with AF and stroke has been studied by reviewing available research on NOAC up to December 2015.[85] The relative efficacy and safety of NOAC over warfarin appear to be greater in Asians than in non-Asians, but the bleeding rates with NOAC were higher in Asians

Table 36.7 HAS-BLED score and major bleeding risk while on warfarin[82,83] (reproduced with permission)

Item	Scores
Hypertension (uncontrolled, SBP >160 mm Hg)	1
Abnormal renal function (serum creatinine >200 μmol/L or on dialysis)	1
Abnormal liver function (cirrhosis or ALP / AST / ALT >3x normal)	1
Stroke (history of stroke, particularly lacunar)	1
Bleeding (history of bleeding or predisposition, e.g. anaemia)	1
Labile INR (therapeutic time in range <60%)	1
Elderly (age >65 years)	1
Drug (antiplatelet / NSAID)	1
Alcohol usage	1
Major bleeding while on warfarin according to HAS-BLED score	
HAS-BLED score	Event rate/year (%) in SPORTIF cohort (on warfarin)[75]
0	0.9
1	3.4
2	4.1
3	5.8
4	8.9
5	9.1
>5	Score >5 is too rare to determine the risk

Abbreviations: ALP = alkaline phosphatase; ALT = alanine aminotransferase; AST = aspirate aminotransferase; NSAID = non-steroidal anti-inflammatory drug; SBP = systolic blood pressure; SPORTIF = Stroke Prevention Using an Oral Thrombin Inhibitor in Atrial Fibrillation

than in non-Asians.[85] The results reviewed call for clinical trials to test whether a reduced dose of NOAC is beneficial in Asian patients with AF and stroke. Further researches are needed to establish the safety and efficacy of dose-adjusted NOAC considering both blood levels of NOAC and fragility of patients in Asian patients with AF and stroke.[85]

Elderly Patients

Studies support the use of oral anticoagulants in elderly patients (aged ≥75 years) in AF for prevention of strokes: the BAFTA on warfarin versus aspirin,[59] and the subgroup meta-analysis of NOAC trials on NOAC versus warfarin by Ruff et al.[86] There has been a call for subgroup analysis of NOAC trial data stratified by age ≥80 years and <80 years, but these data are not publicly available for all the trials.[87]

Stroke prevention in elderly AF patients remains a challenge due to high risk of stroke, systemic thromboembolism, and bleeding. Elderly patients may present with multiple comorbidities, cognitive dysfunction, a tendency to falls, polypharmacy, impaired renal and hepatic function, making them vulnerable to drug interactions, adverse drug reactions, and iatrogenesis. Careful adaptation of drug dosing and tighter monitoring in the use of anticoagulants have been proposed by the ESC to reduce the complications of antithrombotic therapy among elderly AF patients.[88]

In the West, INR between 2.0 and 3.0 has been the recommended target for the use of warfarin in preventing stroke in NVAF patients, irrespective of age.[81] In the light of the special features of Asian AF patients discussed in the section "Asian patients" above, the optimal INR for prevention of stroke in Asian AF patients may differ from Caucasians. Studies in Japan have shown that warfarin is beneficial in stroke prevention (both primary and secondary) not only in the old (70-84 years) NVAF patients, but also in very old (≥85 years) NVAF patients if INR is kept between 1.6 and 2.59, suggesting importance of a lower level of anticoagulation in frail patients. These studies give support to a slightly lower INR target of 1.6-2.6 for elderly (≥70 years) NVAF patients recommended in Japanese guidelines.[89,90]

The important issue regarding use of NOAC among elderly patients is the 'fixed dose', but elderly population is characterised by heterogeneity, differing in renal function, comorbidity, and frailty. Safe use of NOAC in elderly patients needs understanding of when to reduce the dose of NOAC[85] and individualised care after comprehensive geriatric assessment (see Chapter 36.6). The NOAC trials have used only the Cockcroft-Gault (CG) formula for drug dose adjustment. Studies showed that the estimated GFR obtained with the Modification of Diet in Renal Disease (MDRD) and the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formulae were consistently higher than that obtained with the CG formula, leading to significantly higher drug doses compared with doses calculated by using CG formula.[91,92] Lack of appropriate renal dosage adjustments for NOAC may result in serious adverse events.[91,92] Thus, the CG equation needs to be used to calculate eligibility for and dosing of NOAC in elderly patients with AF and the MDRD or CKD-EPI–derived estimated GFR should not be relied on for NOAC drug dosing.

36.6 Role of Comprehensive Geriatric Assessment in Antithrombotic Therapy among Elderly People with AF

Despite research evidence showing the net clinical benefit of warfarin in stroke prevention in AF, warfarin has been under-prescribed in elderly people with AF. In a UK cohort that registered new AF patients over a 10-year period, warfarin was prescribed in only 57% of patients aged 60-69 years, and 32% of those aged ≥80 years.[93] Major underuse of anticoagulation in AF has also been reported in China.[94]

Although experience with NOAC remains limited, incorporation of dabigatran into clinical practice has not increased the proportion of individuals treated with anticoagulants in the US.[95] This under-prescription of anticoagulation in AF in old age may be explained not only by the limitations of warfarin anticoagulation mentioned above, but also by a combination of physician-, individual-, and healthcare system–related factors, which include greater risk of bleeding, treatment burden, and diminished cognition in the older age-group, as well as physician perceptions of benefits and risks of anticoagulation.[73]

The provision of safe and effective antithrombotic treatment to an elderly person with AF can be challenging in clinical practice and requires an individualised approach.[73] Age alone should not be used as a criterion to determine anticoagulation decisions; rather, age should act as a reminder on comprehensive geriatric assessment: to actively assess for comorbid illness, renal function, risk of fall, cognition and vision, medication and dietary adherence, polypharmacy, socioeconomic status, and to maintain the INR strictly within the therapeutic range if warfarin is used. Social factors and transport arrangements in medical monitoring are practical issues to consider in the decision process. When relative contraindications, such as gait disturbances, are considered in treatment decision-making processes, it is appropriate to engage patients / family in the process. Listening to the patient's perspective

is important as studies show that many older patients feel that strokes are states "worse than death",[96] and would prefer treatment with anticoagulation therapy, despite the increased risk of bleeding, to the risk of strokes associated with untreated AF. Thus, it is of utmost importance to assess the benefit-to-risk ratio of elderly patients. Furthermore, clinical judgement is required for the appropriate use of anticoagulants in preventing stroke in elderly patients with AF.[97] Once the decision to start anticoagulation for stroke prevention has been made, measures should be taken to minimise bleeding risk in an elderly patient by choosing a drug and dose appropriate for renal function. Regular monitoring should be performed to maintain the lowest effective INR and the renal function reviewed. Dietary advice should be provided if warfarin is used. Gastric complications and renal function should be monitored if dabigatran is used. In addition, education about fall and injury prevention; regular drug review; detection and treatment of any risk factors for bleeding, including uncontrolled HT; and continual evaluation of new problems that may alter the benefit-risk equation of anticoagulant treatment should be undertaken. Of note, overtreatment of HT should be avoided as it may increase the risk of fall risk due to postural hypotension.

A study of hospitalised elderly patients with AF, when compared with non-AF, revealed a higher Charlson Comorbidity Index, greater number of administered drugs, a higher rate of heart failure and chronic kidney disease, and frailty (54% frail, 29% pre-frail).[98] To prescribe the most appropriate anticoagulation with the best efficacy / safety profile and the highest compliance, the scores for thrombotic and haemorrhagic risk stratification should be accompanied by a comprehensive geriatric assessment including baseline kidney function (using CG formula), cognitive status, mobility, fall risk, polypharmacy, body weight, nutritional status assessment, and life expectancy.[98-100]

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CHAPTER 37

Cardiovascular Disorders

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OBJECTIVE

To understand the principles of treating cardiovascular disorders in older adults.

KEY POINTS

- Elevated systolic and / or diastolic blood pressure is detrimental to elderly people.
- Wide variation in blood pressure is prevalent.
- Treating uncomplicated hypertension in very old patients may cause excess mortality.
- The presentation of ischaemic heart disease and heart failure in elderly people can be atypical.
- Selected older patients with acute coronary syndrome may benefit from thrombolytic therapy and coronary intervention.
- Heart failure with reduced ejection fraction and heart failure with preserved ejection fraction are both common in elderly people.
- Heart failure is associated with high morbidity and mortality in elderly people.
- Examination of the lower limbs for trivial foot lesions should be carried out in elderly patients and is mandatory in patients with diabetes mellitus and peripheral vascular disease as most cases are asymptomatic in the early stages.
- Active mobilisation is an essential therapy for elderly patients who present with stasis ulcers of the lower limb.

37.1 Ischaemic Heart Disease

37.1.1 Introduction

Ischaemic heart disease (IHD) is common in the older population. Its high morbidity and mortality call for an aggressive approach to prevention and treatment. Advancing age is a potent and independent predictor of mortality in patients with IHD.[1] Much of the increased risk in elderly patients may also be due to the atypical presentations, such as silent myocardial infarction (MI), advanced coronary disease, multiple comorbid conditions, and underutilisation of medical and interventional therapy. Older patients at high risk of IHD are very likely to experience improved clinical outcomes and functional status with revascularisation. They are, however, frequently excluded from clinical trials and there are consequently fewer data about treatment effectiveness in this age-group. They are also more prone to procedural complications, due to their frailty, physiological changes, and comorbidities. The decision of when to and how to carry out revascularisation in older patients is complex. It is important to assess the risk of older patients to implement the right treatment.[2] A decision to prescribe medical therapy or revascularisation, percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG) depends on multiple factors that include symptoms, severity of coronary heart disease, functional state, comorbidities, and expected lifespan. The views of patients and family members should also be respected.

37.1.2 Epidemiology and Pathophysiology

The incidence and prevalence of acute MI increase with age. In the US, over 60% of acute MIs occur in patients aged ≥65 years, and approximately one third occur in persons aged >75. In addition, mortality rates following acute MI increase exponentially with age, such that approximately 60% of all MI deaths in the US occur in the 6% of the population aged ≥75 years.[3]

The principal pathophysiological process in IHD is coronary atherosclerosis, with plaque formation and narrowing of vessel luminal diameter. Unstable angina and MI is initiated by rupture or fissuring of an atheromatous plaque. This is followed by platelet aggregation, activation of the clotting cascade and

thrombus formation.[4] Complete or partial occlusion of a coronary vessel results in Q-wave (transmural) MI and non–Q-wave MI / unstable angina, respectively.

37.1.3 Clinical Manifestations

In young and middle-aged individuals, exertional chest pain (angina) is usually the initial manifestation of IHD, whereas in elderly patients, myocardial ischaemia is frequently manifested as dyspnoea.[5] Dyspnoea, usually exertional, is a consequence of the transient increase in left ventricular end-diastolic pressure due to ischaemia superimposed on reduced left ventricular compliance. Even when classic angina is present, it tends to be less associated with exertion, less severe, and of shorter duration than in younger patients.[6] This may be related to reduced pain perception in older adults. Silent or unrecognised MI tends to occur more frequently in elderly men and patients with diabetes mellitus.[7] A local study showed that chest pain (79%) was the most common presenting symptom of acute MI in elderly patients, followed by dyspnoea (11.1%), syncope (7.4%), and cardiac arrest (2.5%).[8] When compared with younger patients (<75 years) with acute MI, chest pain was significantly less frequent (79% vs. 92%), while dyspnoea (11% vs. 3%) and syncope (7.4% vs. 0%) were more common in older adults. Confusion, weakness, abdominal pain, nausea, and vomiting are also recognised symptoms of acute MI in elderly patients. The diagnosis is further complicated by the presence of multiple comorbidities in elderly patients including stroke, dementia, and chronic lung disease. A diagnosis of IHD in elderly patients thus requires a high index of suspicion.

Clinical syndromes of IHD in elderly patients include stable angina (angina equivalent) and acute coronary syndrome (ACS), including acute MI. Silent ischaemia, however, has been reported in up to 20-50% of older patients. The angina of ACS, if present, is typically more severe, prolonged (>20 mins) or present at rest. The initial evaluation of an elderly patient with myocardial ischaemia should begin with a thorough history taking, physical examination, and determination of coronary risk factors and associated comorbid illnesses. In a primary care setting, a combination of three variables — sharp or stabbing pain, no history of angina or MI, and pain with pleuritic or positional components or pain that is reproduced by palpation of the chest wall — define a low-risk group with IHD.[9] Common non-cardiac causes of chest pain in the outpatient clinic are musculoskeletal pain (including costochondritis), gastrointestinal, psychiatric, and pulmonary problems.

Ancillary investigations including electrocardiogram (ECG), echocardiogram, and stress testing are useful in confirming a diagnosis and predicting prognosis for patients with suspected or established IHD. Exercise treadmill testing or pharmacological stress testing can be used in the absence of left main coronary artery disease, resting ST-segment and T-wave abnormalities on ECG or left bundle branch block (LBBB). In these conditions, perfusion imaging (e.g. thallium scan) or stress echocardiogram is more useful. Computed tomographic (CT) coronary angiography that also visualises coronary calcifications has a role in investigating selective older patients with IHD symptoms. Coronary angiography is more invasive but is required if we wish to establish the diagnosis, visualise the coronary vascular system directly, and plan for revascularisation.

37.1.4 Management

The specific management of elderly patients with myocardial ischaemia consists of:

- treatment of associated disorders that can precipitate or worsen ischaemia, e.g. anaemia, infection, thyrotoxicosis, and heart failure (HF)
- reduction of coronary risk factors, especially hypertension, hyperlipidaemia, diabetes mellitus, smoking, lack of exercise, and obesity
- specific therapies for IHD

The principles of drug therapy for IHD in elderly patients are similar to those for younger patients. Due to the physiological changes with ageing and the associated comorbid conditions, one should start low and slowly titrate when prescribing drugs to elderly patients. The classes of drug commonly used in myocardial ischaemia include aspirin; beta-blockers; nitrates; calcium channel blockers; and angiotensin-converting enzyme inhibitors (ACEIs). Statins have been proven beneficial in both primary and, especially, secondary prevention of coronary events in older adults. The high prevalence of polypharmacy among older adults with cardiovascular disease, coupled with age-associated physiological changes and comorbidities, provides major challenges to compliance and avoidance of drug-related adverse events.[10]

Aspirin reduces the mortality and incidence of MI in patients with unstable angina and prior MI.[11] The specific dosage is unclear although the usual dose is 75-325 mg daily. Clopidogrel is an alternative for those who are allergic to or intolerant of aspirin.[12] Short-acting (sublingual) and long-acting nitrates are useful to relieve symptoms of angina and decrease recurrent attacks. The major adverse effects of nitrates, headache and hypotension, can be quite bothersome to certain elderly patients. These episodes can be alleviated by the use of a lower dosage, avoidance of immediate standing after medication, and correction of hypovolaemia. Beta-blockers reduce myocardial oxygen demand and

they are effective in preventing ischaemia, reducing MI, sudden death, and total mortality in patients with prior MI. Extreme bradycardia may complicate beta-blocker therapy in older adults with sinus node dysfunction or heart block. Caution should also be exercised in patients with obstructive airway disease, severe peripheral vascular disease, unstable diabetes mellitus, and depression. Calcium channel blockers are generally not used as the first-line treatment in IHD. In older people, they may lead to oedema and constipation, and exacerbate retention of urine by affecting bladder function. Calcium channel blockers may nonetheless be given when beta-blockers are contraindicated. Short-acting dihydropyridines (e.g. nifedipine) should be avoided as they are associated with increased mortality, possibly due to increased myocardial oxygen demand via reflex tachycardia. They are also contraindicated in patients with systolic HF, sinus node dysfunction, and heart block. ACEI (e.g. ramipril) may be useful in reducing MI, stroke, and cardiovascular mortality in elderly patients with myocardial ischaemia, despite a normal left ventricular function.[13]

ACS comprises a spectrum of disorders including[14]:

- unstable angina: acute cardiac ischaemia without ST segment elevation and with normal markers of myocardial necrosis;
- non–ST segment elevation myocardial infarction (NSTEMI): acute cardiac ischaemia without ST segment elevation and with elevated markers of myocardial necrosis; and
- ST segment elevation myocardial infarction (STEMI): acute cardiac ischaemia with ST segment elevation and elevated markers of myocardial necrosis.

Clinically, the differentiation between these last two entities is based on the characteristic ECG changes (ST elevation or new-onset LBBB) and the elevation of biochemical markers (creatine kinase MB or troponin I or T). Cardiac specific troponin I and T are regulatory proteins of muscle contraction and they are highly sensitive and specific markers of myocardial ischaemia.[12] Troponin, however, may not be elevated during the first 6 hours of chest pain and its level remains abnormal after 10 days.[13] Also, elderly individuals have high levels of baseline troponin level. Careful clinical assessment is essential to separate ACS from a variety of acute and chronic conditions also leading to low-level myocardial necrosis (type 2 supply / demand MI and non-ischaemic myocardial injury with necrosis)[15,16] (see Chapter 14.3.5 for interpretation of the troponin test in old age).

One of the underlying reasons for the above ACS classification is to select patients who might benefit from thrombolytic therapy. Patients who are younger than 75 years with acute STEMI, and seen within 12 hours from onset of symptoms, should receive thrombolytic therapy unless there is a contraindication.[17] Intensive medical therapy and monitoring is also indicated in high-risk elderly patients with ACS. High-risk features include fixed or dynamic ST segment depression on resting ECG, persistent or recurrent ischaemic pain, elevated biochemical markers, such as creatine kinase MB or troponin, and findings of left ventricular dysfunction or HF.

Aspirin and beta-blockers should be given to all patients without contraindications.[18] Intravenous nitrates are useful for patients with persistent ischaemia, HF, or hypertension. Low-molecular-weight heparin such as enoxaparin or dalteparin should be given to patients with unstable angina and non-STEMI. In patients aged 65-74 years, early treatment with an ACEI (within 36 hours from MI) is particularly beneficial if there is anterior MI or signs of HF.[19] Older patients (>75 years) are more likely to have contraindications to ACEI such as hypotension (systolic blood pressure [SBP] <100 mm Hg) and renal failure so the efficacy is less apparent in this group. The benefit of ACEI in other MI patients without HF is also less clear. Supportive treatments that include oxygen therapy, diuretics, inotropic agents, and antiarrhythmic drugs should be used where appropriate.

There have been significant advances in adjunctive pharmacotherapy for patients with MI.[20] Routine use of antiplatelet agents such as clopidogrel, prasugrel or ticagrelor, in addition to aspirin, reduces patient morbidity and mortality. Nonetheless the safety and efficacy of 'clopidogrel plus aspirin' in other elderly patients with ACS are not well established. Treatment with direct thrombin inhibitors during PCI is an alternative to unfractionated heparin and low-molecular-weight heparin with similar efficacy and less bleeding complications. The use of a glycoprotein IIb / IIIa antagonist with dual antiplatelet agents and unfractionated heparin in STEMI patients for primary PCI can reduce mortality and morbidity.[21] Clopidogrel has also been shown to reduce early in-stent restenosis in patients undergoing intracoronary stent implantation. Drug-eluting coronary stents are safe and beneficial as primary coronary intervention. It is beyond the scope of this article to describe in detail all the new developments of current IHD management. Readers should refer to cardiology textbooks and the literature for the most up-to-date information.

37.1.4.1 Thrombolysis

Although the benefits of thrombolytic therapy

are well documented in patients younger than 75 years with STEMI, the value of such therapy in patients aged ≥75 years remains controversial and inconclusive. This is due to the conflicting findings of clinical trials and observational studies, as well as the higher risks of major bleeding complications, intracerebral haemorrhage (ICH) in particular, in elderly patients. A meta-analysis that included 3322 patients aged ≥75 years with STEMI revealed that thrombolytic therapy within 12 hours of symptom onset was associated with a 3.4% absolute reduction and 15% relative reduction in mortality at 35 days.[22] On the contrary, several observational studies have suggested that the use of thrombolytic therapy in elderly patients is associated with an increase in mortality, especially early deaths.[23,24] In the Global Utilization of Streptokinase and Tissue plasminogen activator for Occluded coronary arteries I study, a significant reduction in mortality was observed with thrombolysis in patients aged 65-85 years.[25] In addition, in patients >85 years, there was a trend of benefit towards the use of streptokinase. The use of thrombolytic therapy in patients aged ≥75 years with STEMI is considered a class IIa indication in the American College of Cardiology / American Heart Association (ACC / AHA) guidelines. Thrombolytic therapy may be given to selected elderly patients (>75 years) with strict adherence to the indications and contraindications. Combination pharmacological reperfusion with abciximab and half-dose reteplase or tenecteplase to prevent re-infarction should not be given to patients aged >75 years because of an increased risk of ICH.

37.1.4.2 Primary PCI Versus Thrombolysis

Primary PCI in appropriate settings may be useful in selected elderly patients as an alternative to thrombolytic therapy.[26] Studies have shown that there is a lower risk for combined end-point of death and non-fatal stroke in a primary PCI group than with fibrinolysis, due to a higher rate of ICH in the latter group. Recent meta-analysis has shown primary PCI to be associated with lower mortality, risk of repeated MI and stroke in all age-groups compared with fibrinolysis.[27] Hence, in hospitals where direct PCI can be performed rapidly by experienced operators, primary PCI seems to have an advantage over thrombolysis in all age-groups, including older patients.

37.1.4.3 Revascularisation

Older patients at high risk of IHD are very likely to experience improvements in clinical outcomes and functional status with revascularisation but they are also more prone to procedural complications.

In general, early coronary angiogram and revascularisation, either PCI or CABG, is indicated in high-risk elderly patients with unstable angina or NSTEMI.[28] In some patients with old Q-wave MI or chronic angina, non-invasive tests are needed for risk stratification and to select patients who are likely to benefit from revascularisation. The decision to prescribe medical therapy versus revascularisation should not be only based on symptoms, severity of IHD, and left ventricular function. Other factors including comorbidities, lifestyle, functional states, projected life expectancy, cognitive function, social factors, and personal and / or family preferences should be taken into account. Comprehensive geriatric assessment is necessary to detect the factors of frailty before opting for revascularisation.

The optimal timing for revascularisation procedures in the very old is uncertain. In one randomised trial, patients aged ≥75 years with chronic symptomatic IHD were randomised to early invasive revascularisation or optimised medical therapy with rescue intervention.[29] One year later, there was no difference in symptom or quality-of-life improvement, death, or nonfatal MI between the two groups. The invasive approach carries an early intervention risk and excess death, whereas medical management poses an almost 50% chance of later hospitalisation and revascularisation. The use of such procedures in older patients should thus be carefully assessed and individualised.

With regard to the choice between PCI and CABG, it seems that each has its own merits as well as disadvantages. One study showed that CABG patients had a higher mortality and morbidity compared with PCI patients but there was greater angina relief and fewer repeated procedures after CABG.[30] Another study showed that PCI had better in-hospital survival but CABG achieved better long-term survival.[31] When advocating PCI to elderly patients, patients and family members must understand the lower success rate and risks of renal failure, and bleeding. For CABG, the longer duration of ventilator support, higher risk of atrial fibrillation, renal failure, infection, stroke, prolonged hospitalisation, and delirium have to be taken into consideration. As in other operative procedures, the potential need for extended hospitalisation, rehabilitation for deconditioning, home assistance after discharge, and risk of cognitive impairment and depression need to be assessed in older patients.

37.1.5 Rehabilitation

Cardiac rehabilitation should be considered for older patients with recent MI. As older patients have more complex needs, rehabilitation often requires a multidisciplinary approach with protocols and approaches different to those for younger patients. The Cardiac Rehabilitation in Advanced Age study

compared hospital-based versus home-based cardiac rehabilitation in patients without cognitive impairment.[32] The study found similar improvement in work capacity and health-rated quality of life in all age-groups, although the improvement was smaller in older patients of >75 years.

37.1.6 Prevention

Aggressive control of various coronary risk factors is the cornerstone to preventing IHD. This is especially important for elderly patients who are likely to possess multiple risk factors such as diabetes mellitus and hypertension. Smoking should be strongly discouraged. Vigorous attempts to lower cholesterol are indicated for patients with diabetes mellitus and established IHD.[23] The target low-density lipoprotein cholesterol level is 2.6 mmol/L. Regular physical exercise can also prevent heart disease in older individuals. Currently, there is insufficient data to support screening for IHD with ECG, exercise treadmill tests, or electron-beam CT (detection of coronary calcium content) in asymptomatic individuals.[24] The routine use of these tests in low-risk individuals may actually cause more harm than benefit.

37.2 HF in Elders

37.2.1 Classification and Epidemiology

HF is subdivided into two major subtypes: heart failure with reduced ejection fraction (HFrEF), i.e. left ventricular ejection fraction (EF) <40%; and HF with preserved ejection fraction (HFpEF), i.e. left ventricular EF >40-50%. Exercise intolerance is the cardinal feature of HF and can lead to a sedentary lifestyle, deconditioning, and frailty. It is one of the most debilitating chronic illnesses in Hong Kong, especially among elderly people. The annual incidence of HF in Hong Kong was 70 per 100000 population in 1997,[33] and it rose with age, doubling with each decade of age, reaching rates of 1400 per 100000 and 2000 per 100000 for men and women aged ≥85 years, respectively (Figure 37.1).[33] More than 6000 patients were admitted to public hospitals yearly because of HF and 85% were >64 years.[33] A local study in a teaching hospital showed that the annual incidence rate of HF was 300-380 per 100000.[34] HF was the cause of death in 794 patients in Hong Kong in 2009.[35] The 1-year mortality rate in all patients with HF was 32%, and increased to 40% in patients >85 years.[36] Therefore, HF imposes an enormous cost and burden on healthcare resources. Most of the landmark HF clinical trials included subjects with a mean age in the 60s and so the evidence for treating HF in elderly patients is extrapolated from studies that involved subjects up to two decades younger.[37]

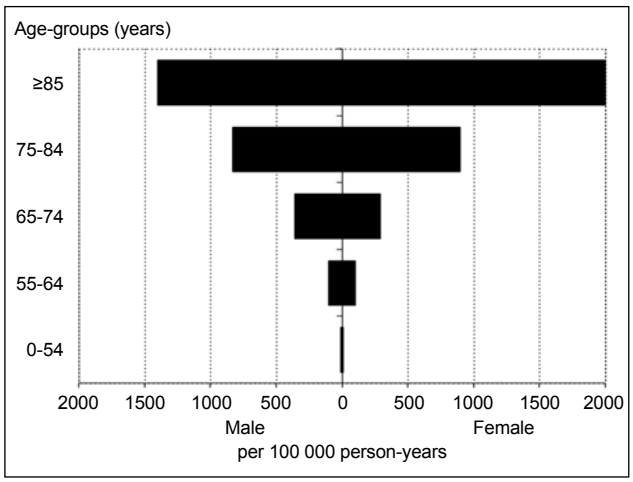

Figure 37.1 Age- and sex-specific incidence rate of heart failure in 1997 for Hong Kong[33]

37.2.2 Cardiovascular Changes and Ageing

With ageing, left ventricular end-diastolic volume, stroke volume, and cardiac output decrease. All these age-related changes begin with the stiffening of arteries and myocardium. Stiffening is a result of the cessation of elastin production in the fourth decade of life and damage to elastin by free radicals over time with a gradual replacement of elastin by the less-flexible collagen protein. The stiffening of arteries results in increased left ventricular afterload, increased workload of the myocardium, myocardial hypertrophy, and left ventricular wall thickening. In addition, there is an extended duration of ventricular contraction and a consequent delay in ventricular relaxation with a decline in early diastolic filling rates by 50% between the second and eighth decades. The heart gradually depends more on atrial filling pressures. Overall there is a predisposition in elderly people to diastolic HF or HFpEF that accounts for the higher prevalence in these patients.[38]

37.2.3 Aetiology of HF

According to a local study, common causes of HF were hypertension (37%), IHD (31%), valvular heart disease (15%), cor pulmonale (27%), and idiopathic dilated cardiomyopathy (4%).[34] Less common causes include hypertrophic cardiomyopathy and restrictive cardiomyopathy.[39] A local study showed that 66% of hospitalised patients with clinical HF in fact had normal EF, suggesting that HFpEF was more common than HFrEF.[40] In another local study involving 73 patients with HFpEF, most subjects were female (69.8%) with a mean age of 76.2 years and average EF of 64.3%.[41] Common comorbidites included hypertension (76.7%), diabetes (46.6%), and renal impairment (16.4%).[41] These findings are similar to those in western populations. Compared with

nearly 90% of HF patients aged <65 years who had HFrEF, 40% of men and two-thirds of women with HF aged >65 years had HFpEF.[39] Approximately 40-60% of hospitalised HF patients have HFpEF and the majority are women.[42] Half of elderly patients with HF aged >75 years have HFpEF and its prognosis appears similar to HFrEF at 1 year.[43] Elderly patients with HF are also likely to have multiple comorbidities: two-thirds with more than two non-cardiac comorbidities and over 25% with more than six.[44] These comorbidities add to the complexity of HF care in elderly patients.

Table 37.1 New York Heart Association classification of heart failure

Class	Symptom of heart failure
I	Only at levels that would limit normal individuals
II	With ordinary exertion
III	On less-than-ordinary exertion
IV	At rest

Table 37.2 American College of Cardiology / American Heart Association Task Force 2013

Stage	Description
A	High risk for heart failure, without structural heart disease or symptoms
B	Heart disease with asymptomatic left ventricular dysfunction
C	Prior or current symptoms of heart failure
D	Advanced heart disease and severely symptomatic or refractory

37.2.4 Clinical Evaluation and Investigations

Symptoms of HF are generally related to fluid retention (dyspnoea, orthopnoea, and oedema) and reduced cardiac output (fatigue and decreased exercise tolerance). Exertional symptoms may be less prominent in elderly patients because of their more sedentary lifestyle.[44,45] Manifestations can also be atypical, especially in frail and demented patients, who may present with lethargy, fatigue, irritability, anorexia, abdominal distension, or confusion.[44,45] Diagnosis is further complicated by the high prevalence of concomitant illnesses or conditions that might produce similar complaints to HF. Renal failure, liver cirrhosis, varicose veins, medications like calcium channel blockers or inactivity can cause oedema. Chronic obstructive pulmonary disease (COPD), anaemia, kyphoscoliosis, and obesity may cause dyspnoea. Factors that might exacerbate HF include myocardial ischaemia / infarction, uncontrolled hypertension, atrial fibrillation, renal failure, viral infections or pneumonia, COPD, anaemia, or drugs (e.g. non-adherence to HF medications or use of non-steroidal anti-inflammatory medications).[44,45] Patients should be classified according to New York Heart Association (NYHA) and ACC / AHA protocols (Tables 37.1 and 37.2).

The diagnosis of HF is mainly based on clinical grounds. Supportive findings on clinical examination include pitting oedema of the legs, elevated jugular venous pressure, displaced apex beat, gallop rhythm, basal crackles, or diminished breath sounds at the lung bases because of bilateral pleural effusion. Basic investigations for HF should include a complete blood picture, renal and liver function tests, thyroid function test, chest X-ray, and ECG. A transthoracic echocardiogram to evaluate left ventricular function should be arranged. Clinicians should look for potential precipitating factors for HF via these investigations. Certain hospitals may be equipped with measurement of B-type natriuretic peptide (BNP) and its precursor N-terminal pro-BNP, both of which are released by cardiac myocytes when the myocardium is stretched.[39] In general, a BNP level of at least 500 pg/mL is suggestive of HF.[39] However, these peptide levels increase with age (especially in female) and degree of renal impairment, and are also lower in obese patients. Thus their specificity in elderly patients decreases.[39] Other potential investigations may include magnetic resonance imaging (MRI) of the heart, radionuclide ventriculography, stress testing (either echocardiogram or radionuclide examination), and coronary angiography.[44]

37.2.5 Management

The goals in management of HF are alleviation of symptoms, and prevention of progression of disease and hospitalisation. Elderly patients with HF should only be regarded as stable when they are able to perform basic daily activities and able to walk at least one block without any symptoms.[44] Clinicians should emphasise the importance of stable fluid balance, maintaining SBP of >100 mm Hg and heart rate range of 60-85 beats/min.[44,46] Clinicians should also optimise the management of other comorbid conditions including hypertension, hyperlipidaemia, diabetes mellitus, COPD, anaemia, and chronic renal failure. Other issues that might need to be addressed include the home environment, caregiver issues, drug compliance (especially for those with cognitive impairment), and emergency response system (e.g. personal emergency link service). HF patients should be given advice about restriction of sodium intake to <2 g per day, fluid restriction to <1.5 L per day in general, and regular monitoring of their own body weight (preferably in the morning without clothes after going to the bathroom but before eating).[44] Low-level intensity exercise (e.g. walking, stationary cycling) should be maintained as far as possible.[44]

37.2.5.1 HFrEF

ACEI, angiotensin receptor blockers (ARB), aldosterone antagonists, and beta-blockers are now the cornerstone of therapy for patients with HFrEF.[44] The latest recommendations for their usage and dosages are summarised in Tables 37.3 and 37.4.[47]

Diuretics (usually loop diuretics like furosemide) are a key component in the management of HF to relieve congestion and fluid overload. In patients with concomitant chronic renal failure or advanced HF, the addition of metolazone (a thiazide diuretic) 2.5-10 mg/day may be needed to maintain euvolaemia. Patients should be monitored for dehydration, worsening of renal function, and electrolytes. Elderly patients are particularly vulnerable to dehydration because of attenuation of the thirst response or decreased oral intake due to other factors.[39,46,47] Spironolactone has been shown to reduce mortality (by 30%) and hospitalisation (by 35%) in patients with NYHA class III-IV HF and EF of ≤35%.[46,47] Gynaecomastia can occur in 10% of patients using it. Eplerenone has also been found to improve outcomes in patients with recent MI complicated by HF or EF <40% as well as in patients with NYHA class II HF and EF of ≤35%.[46,47] Less than 1% of patients prescribed eplerenone develop gynaecomastia. Close monitoring of potassium level and renal function is

Table 37.3 American College of Cardiology / American Heart Association guidelines for the treatment of heart failure with reduced left ventricular ejection fraction[47]

Class	Recommendation
I	• Exercise training (or regular physical activity) is recommended as safe and effective for patients with HF who are able to participate to improve functional status • Diuretics are recommended in patients with HFrEF who have evidence of fluid retention, unless contraindicated, to improve symptoms • ACEIs are recommended in patients with HFrEF and current or prior symptoms, unless contraindicated • ARBs are recommended in patients with HFrEF with current or prior symptoms who are ACEI intolerant, unless contraindicated • Use one of the three β-blockers proven to reduce mortality (bisoprolol, carvedilol, sustained-release metoprolol succinate) unless contraindicated • Aldosterone receptor antagonists are recommended in patients with NYHA class II-IV HF and who have LVEF of ≤35% unless contraindicated • Aldosterone receptor antagonists are recommended to reduce morbidity and mortality following an acute myocardial infarction in patients who have LVEF of ≤40% who develop symptoms of HF or have a history of diabetes mellitus, unless contraindicated
IIA	• Digoxin can be beneficial in patients with HFrEF, unless contraindicated, to decrease hospitalisations for HF • A combination of hydralazine and isosorbide dinitrate can be useful to reduce morbidity or mortality in patients with current or prior symptomatic HFrEF who cannot be given an ACEI or ARB because of drug intolerance, hypotension, or renal insufficiency, unless contraindicated

Abbreviations: ACEIs = angiotensin-converting enzyme inhibitors; ARBs = angiotensin receptor blockers; HF = heart failure; HFrEF = heart failure with reduced ejection fraction; LVEF = left ventricular ejection fraction; NYHA = New York Heart Association

Table 37.4 Initial and maintenance doses of ACEIs, angiotensin II receptor blockers, beta-blockers, and aldosterone receptor antagonists in elderly patients with HFrEF[47]

Agent	Staring dosage	Target dosage
ACEI		
Lisinopril	2.5-5 mg/day	20-40 mg/day
Perindopril	2 mg/day	8-16 mg/day
Ramipril	1.25-2.5 mg/day	10 mg/day
Angiotensin II receptor blocker		
Candesartan	4 mg/day	32 mg/day
Irbesartan	75 mg/day	150-300 mg/day
Losartan	25 mg/day	50-100 mg/day
Olmesartan	20 mg/day	40 mg/day
Telmisartan	20 mg/day	80 mg/day
Valsartan	40 mg BD	160 mg BD
Beta-blockers		
Bisoprolol	1.25 mg/day	10 mg/day
Carvedilol	3.125 mg BD	25-50 mg BD
Metoprolol XL	12.5-25 mg/day	200 mg/day
Aldosterone receptor antagonist		
Spironolactone	12.5-25 mg/day	50 mg/day
Eplerenone	25 mg/day	50 mg/day

Abbreviations: ACEIs = angiotensin-converting enzyme inhibitors; BD = twice daily; HFrEF = heart failure with reduced ejection fraction

needed for patients prescribed spironolactone and eplerenone. Both medications are contraindicated if creatinine level is ≥220 μmol/L.[39,46,47]

ACEI and ARB should be started at a low dosage with gradual upwards titration. Patients should have their renal function and potassium level monitored: a 20% increase in creatinine level may be seen in patients newly prescribed these agents.[46] ARB is usually a substitute for ACEI if patients are intolerant of ACEI side-effects including cough (5-10%) or those with concomitant stress incontinence.[46,47] Combination therapy of ACEI and ARB is not currently recommended.[47]

An effective reduction in mortality (35-65%) has also been achieved with beta-blockers prescribed to patients with NYHA class II-IV HF.[48] The mechanisms were mediated via a combination of antihypertensive, anti-ischaemic, antiarrhythmic, and antiatherogenic effects.[49,50] Beta-blockers can also improve left ventricular function and counteract the chronic activation of the sympathetic nervous system.[46] Nonetheless the reduction in mortality has only been demonstrated for carvedilol, metoprolol XL, and bisoprolol.[47] These medications should be started at their lowest possible dosage with slow upwards titration. The dosage can be doubled every 2-3 weeks to achieve a maintenance dose over 3-4 months. Beta-blockers are contraindicated in decompensated HF, hypotension (SBP <90 mm Hg), second- and third-degree heart block and patients with severe asthma.[46]

Digoxin is indicated for heart rate control in atrial fibrillation and for HF patients with advanced systolic dysfunction in NYHA class III or IV.[46,47] Potential side-effects include anorexia, confusion, visual disturbances, bradyarrhythmias, as well as supraventricular and ventricular tachyarrhythmias.[39,46,47] The therapeutic digoxin serum level should be 0.6-1.2 nmol/L (or 0.5-0.9 ng/mL).[51] The concomitant usage of amiodarone can increase the serum digoxin concentration by 2-fold, thus the dosage of digoxin should be reduced by 50%.[39]

37.2.6 HFpEF

Although HFpEF is more common in elderly patients, there are fewer clinical trials and evidence-based treatments for this condition. To date, no trials have clearly demonstrated that any pharmacological treatment can reduce mortality.[39,46,47] The general guidelines for HFpEF include good control of blood pressure (BP), optimisation of heart rate in patients with atrial fibrillation with beta-blockers, and usage of diuretics in case of congestion (Table 37.5).[46,47] In the latest trial to explore the potential benefits of isosorbide mononitrate in HFpEF (mean age of 69 years, mostly NYHA class II-III), those prescribed isosorbide mononitrate were less active and did not have a better quality of life or submaximal exercise capacity than patients prescribed a placebo.[52]

37.2.7 Adjuvant Therapies

Patients with chronic HF with permanent / persistent / paroxysmal atrial fibrillation and an additional risk factor for cardioembolic stroke should be prescribed chronic anticoagulant therapy.[46] The selection of an anticoagulant (i.e. warfarin vs. non–vitamin K antagonist oral anticoagulant) should be individualised (see Chapter 36.6).[46] Anticoagulation is not recommended in patients with chronic HFrEF without atrial fibrillation, a prior thromboembolic event, or a cardioembolic source.[46] Coronary angiogram and revascularisation should be considered in patients with angina, angina equivalent, such as flash pulmonary oedema, or if there is evidence of myocardial ischaemia on stress testing. Mitral or aortic valve surgery is also indicated in selected patients with valvular dysfunction.

An implantable cardioverter-defibrillator (ICD) device has been shown to reduce mortality associated with sudden cardiac death in patients with ischaemic or non-ischaemic HF and left ventricular EF of ≤35%.[47] Nonetheless there was no impact on symptoms or quality of life.[46] Major complications associated with ICD implantation were 2-fold greater in patients aged ≥80 years compared with younger patients.[39]

Table 37.5 American College of Cardiology / American Heart Association guidelines for the treatment of heart failure with preserved left ventricular ejection fraction[47]

Class	Recommendation
I	• Systolic and diastolic blood pressure should be controlled in patients with HFpEF in accordance with published clinical practice guidelines to prevent morbidity • Diuretics should be used for relief of symptoms due to volume overload in patients with HFpEF
IIA	• Coronary revascularisation is reasonable in patients with CAD in whom symptoms (angina) or demonstrable myocardial ischaemia is judged to be having an adverse effect on symptomatic HFpEF despite guideline directed medical therapy • Management of AF according to published clinical practice guidelines in patients with HFpEF is reasonable to improve symptomatic HF • The use of β-blocking agents, ACEIs, and ARBs in patients with hypertension is reasonable to control blood pressure in patients with HFpEF

Abbreviations: ACEIs = angiotensin-converting enzyme inhibitors; AF = atrial fibrillation; ARBs = angiotensin receptor blockers; CAD = coronary artery disease; HF = heart failure; HFpEF = heart failure with preserved ejection fraction

Up to 20% of the shocks were inappropriate.[39] Also, the current ICD data were derived from patients only up to the age of 75 years.[46] A well-informed decision-making process should be the remit of the cardiologist. Based on the above, prophylactic ICD placement is recommended in patients with NYHA class II or III HF, EF of ≤35%, and life expectancy with good functional status of at least 1 year.[47] ICD implantation should be deferred for at least 40 days after acute MI or at least 90 days after a diagnosis of dilated cardiomyopathy in patients on guideline-directed medical therapy.[47]

Cardiac resynchronisation therapy (CRT) involves the placement of a biventricular pacemaker: one lead within the right ventricle and a second lead inserted into the coronary sinus to stimulate the left ventricle. As the name implies, it can resynchronise left ventricular contraction in HF patients with LBBB (QRS interval ≥150 ms).[39,46] CRT can improve symptoms, quality of life, and survival in patients with medically refractory EF of ≤35% and wider QRS complex (≥150 ms). In general, CRT is indicated for HFrEF patients with EF of ≤35%; sinus rhythm; LBBB with a QRS of ≥150 ms; and NYHA class II, III, or ambulatory IV symptoms on guideline-directed medical therapy.[47] A left ventricular assist device is increasingly being used in elderly patients in western countries, however, it is mainly regarded as a bridge to heart transplantation in Hong Kong and mainly used in younger patients.[39]

37.2.8 Palliative Care, Hospice, and End-of-life Planning

Palliative care involves a holistic approach to improve quality of life in patients who face life-threatening illnesses. A hospice focuses on symptom management in patients with a prognosis of ≤6 months. End-of-life care involves palliative care, hospice care, advance directives, and advance care planning. Patients and family members should be educated about the course and prognosis of HF and decisions for cardiopulmonary resuscitation in patients with advanced HF.[46] A significant proportion of advanced HF patients experience pain (63-80%), anxiety (49%), and breathlessness (60-88%).[53] These symptoms should be managed. Angina pectoris should be managed with nitrates; an electric fan, oxygen supplementation, or opioids should be offered for shortness of breath; opioids might also be offered for pain control; depression and anxiety symptoms should be managed with a selective serotonin reuptake inhibitor or anxiolytics (e.g. benzodiazepines).[46,53] Advance care planning, involving the deactivation of devices (including ICD, CRT, or left ventricular assist device), to allow natural death should involve the cardiologist.[46,53]

37.3 Hypertension in Elderly People

37.3.1 Epidemiology

Hypertension is defined as SBP of at least 140 mm Hg and / or diastolic blood pressure (DBP) of at least 90 mm Hg (Table 37.6).[54] Hypertension is prevalent in Hong Kong. According to the Population Health Survey conducted in 2003-2004 by the Department of Health, around 27% of the population aged ≥15 years had hypertension. The prevalence increased with increasing age and reached 66.3% in the 65-74 years' age-group and 73.3% in those aged ≥75 years.[54] Even more alarming was that 29.9% and 31.8% of the population in the 65-75 and ≥75 years' age-group, respectively had hypertension that remained undiagnosed.[54] The association between hypertension and cardiovascular, cerebrovascular, and renal diseases has been well documented in older adults.[55] An elevated SBP in mid-life has also been associated with cognitive decline and cognitive impairment.[56] There have nonetheless also been many observational studies established that the risk associated with hypertension does not increase with age; the association between hypertension and mortality is weaker in the very old.[55]

37.3.2 Pathophysiology

With increasing age, SBP progressively increases while DBP reaches a peak in the fifth and sixth decade and subsequently declines resulting in elevated pulse pressure. Multiple factors contribute to the elevated BP. First, there is increased arterial stiffness as a result of structural changes in the media including hypertrophy and loss of contractility of the vascular smooth muscle cells, calcification, fibrosis, collagen deposition, and fragmentation of the elastic lamina. Endothelial dysfunction (with decreased nitric oxide production) and atherosclerosis also contribute to arterial stiffness. In addition, there is increased

Table 37.6 Classification of blood pressure in adults aged ≥18 years* [54]

Category	Systolic blood pressure (mm Hg)		Diastolic blood pressure (mm Hg)
Normal	<140	and	<90
Hypertension	Stage 1: 140-159	or	90-99
	Stage 2: Stage 2: ≥160	or	≥100

* Diagnosis of hypertension should be based on at least 3 blood pressure readings taken on 2-3 separate visits over a period of 4-6 weeks

alpha-adrenergic receptor responsiveness resulting in heightened vasoconstriction. Second, there is impaired sensitivity of arterial baroreceptors and hence a greater change in SBP is needed to result in compensatory changes in heart rate and consequent increased sympathetic nervous system activity for any given SBP. The decreased baroreceptor sensitivity has been proposed to underlie the greater variability in BP, white coat hypertension, postprandial and postural hypotension, a relatively higher nocturnal BP, and greater early morning BP rise contributing to increased risk of stroke and MI. Third, with increasing age, there is a 10% per decade decline in renal cortical blood flow with loss of functional glomeruli. The remaining glomeruli are exposed to a greater intraglomerular pressure transmitted from the small arterioles resulting in further loss of glomeruli. Thus there is an overall decline in the ability to handle sodium load. Other potential contributing factors to the development of hypertension include insulin resistance and central obesity.[55]

37.3.3 Diagnosis and Clinical Assessment

An accurate measurement of BP is important in the diagnosis and management of hypertension. Because of the greater variability in BP in elderly patients, the diagnosis of hypertension should be based on at least three BP readings taken on two to three separate visits over a period of 4-6 weeks.[55] The presenting BP should not exceed 180/110 mm Hg. BP should be measured in both arms and the side with higher measurement should be used in subsequent monitoring. Increased arterial stiffness in elderly people may result in an auscultatory gap and thus an underestimation of the true SBP.[55] The auscultatory gap refers to the temporary loss of Korotkoff sounds between phase two and three but can be avoided by inflating the cuff to a pressure of 30-40 mm Hg higher than the pressure that is required to occlude the brachial or radial pulses during rapid cuff inflation; this is followed by a slow deflation of the cuff by 1-2 mm Hg per second to detect the Korotkoff sound.[55] Electronic oscillometric devices are not affected by the auscultatory gap as these machines estimate the SBP and DBP by mean arterial pressure.[55] Clinicians should also be aware of the phenomenon of pseudohypertension caused by extreme rigidity of the brachial arteries that prevents complete occlusion. It should be suspected in elderly patients with resistant hypertension or those with marked hypotension-related symptoms when antihypertensive therapy is started.[57] Other possible suggestive clinical features of pseudohypertension include the presence of calcified arteries on arm radiograph or a positive Osler sign.[57] Osler's manoeuvre is performed by inflating the BP cuff above SBP and carefully palpating the radial or brachial artery. Whenever the artery remains clearly palpable (despite being pulseless), the patient is described as Osler positive.[58] It has been shown that cuff pressure overestimated intra-arterial pressure by 10-54 mm Hg in selected Osler-positive patients.[57] BP should not be lowered aggressively in these individuals.

White coat hypertension should be suspected if there is a marked discrepancy between home and office BP. Careful home BP or 24-hour ambulatory BP monitoring is indicated. The diagnosis can be made in untreated patients who have hospital- / clinic-measured BPs of at least 140/90 mm Hg but 24-hour ambulatory BP of <130/80 mm Hg (awake, <135/85 mm Hg; sleep, <120/70 mm Hg) or home BP of <135/85 mm Hg.[55] During 24-hour ambulatory BP monitoring, nocturnal mean BP should exhibit a 10% drop relative to daytime mean BP; if this is absent, it is associated with higher cardiovascular risk.[57,59]

During the treatment of hypertension, the clinician should also be observant for postural hypotension. To detect postural hypotension, BP should be measured in a supine position and erect / standing posture at 1 minute and 3 minutes. A drop in SBP by at least 20 mm Hg or in DBP by at least 10 mm Hg is diagnostic of postural hypotension. Postural hypotension increases the risk of fall.[60] It was present in around 8% of the subjects recruited in the HYpertension in the Very Elderly Trial (HYVET).[61]

After a diagnosis of hypertension is established, secondary hypertension should be excluded. Most hypertensive elderly patients have essential hypertension, but a secondary form of hypertension should be suspected in patients with resistant hypertension (poorly controlled BP despite ≥3 antihypertensive agents), newly developed uncontrolled BP, or malignant hypertension (BP ≥220/120 mm Hg + grade III / IV fundal changes, i.e. flame haemorrhages, hard exudates, papilloedema). The most common cause of secondary hypertension in elderly people is renovascular disease. The other common causes include usage of non-steroidal anti-inflammatory agents, hyperaldosteronism, and obstructive sleep apnoea.[55,59]

After excluding the possibility of secondary hypertension, patients should undergo risk stratification including identification of target organ damage (e.g. left ventricular hypertrophy) or comorbidities including diabetes mellitus, hyperlipidaemia, renal diseases, or congestive HF. During the clinical assessment, general well-being should also be considered and include functional level, possible life expectancy (e.g. advanced dementia, any end-organ failure, or advanced malignancies) or frailty. Treatment goals and choice of medications should be based on such an assessment.[55,59]

37.3.4 Treatment

Treatment and control of hypertension is well established to improve adverse outcomes related to hypertension. Meta-analyses involving around 55 randomised controlled trials have found that BP lowering by all classes of antihypertensive drugs is accompanied by significant reductions in cardiovascular (e.g. chronic HF) and cerebrovascular (e.g. stroke) morbidity and mortality.[62] The outcome for systolic hypertension among older adults (age ≥60 years) has also been studied in another meta-analysis: treatment reduced total mortality by 13%, cardiovascular mortality by 18%, all cardiovascular complications by 26%, stroke by 30%, and coronary events by 23%.[63] Absolute benefit was greater in men, in patients aged ≥70 years and in those with previous cardiovascular complications or wider pulse pressure.[63] The risks and benefits of antihypertensive therapy in patients aged ≥80 years, predominantly in eastern Europe and China, were studied in HYVET.[61] A total of 3845 participants were randomised to active therapy (extended release indapamide plus an ACEI, perindopril, if needed) or placebo and followed for primary outcomes of fatal or non-fatal stroke. At 2 years, there was a 15/6 mm Hg difference in BP between the two groups; target BP (150/80 mm Hg) was achieved by 20% in the placebo group and by 48% in the active treatment group. The active treatment group showed a 21% reduction in overall mortality, 30% reduction in fatal and non-fatal stroke and 64% reduction in HF.[61] Nonetheless the HYVET population had fewer comorbid illnesses and was generally healthier than the general very-old population, such that the results could not be generalised to older patients with poor functional status, frailty, multiple comorbidities, or dementia.

Evidence-based guidelines for the management of high BP in adults: report from the panel members appointed to the Eighth Joint National Committee (JNC-8) was published in 2014.[64] JNC-8 recommends treating adults aged ≥60 years to a target BP of <150/90 mm Hg, because treatment targets of SBP of <160 mm Hg were set in trials on elderly hypertensives with beneficial outcomes. Other guidelines have recommended target SBP of 140-150 or 150 mm Hg only in those aged ≥80 years or in those aged ≥60 years who are unfit.[65-68] A comparison of the target BP from various guidelines was made (Table 37.7[64-68]). Kidney Disease: Improving Global Outcomes guideline recommends a BP of ≤130/80 mm Hg for patients with chronic kidney disease and moderate-to-severe albuminuria, but they suggest that target BP for patients ≥80 years with chronic kidney disease should be individualised and a higher target is reasonable.[69]

The question of BP targets was further complicated by the results of the Systolic Blood Pressure Intervention Trial (SPRINT).[70] In this trial, 9361 persons with SBP of ≥130 mm Hg and an increased cardiovascular risk but without diabetes were assigned to SBP target of <120 mm Hg or a target of <140 mm Hg; 2636 participants were aged ≥75 years. The intervention was stopped early after a median follow-up of 3.26 years owing to a significantly lower rate of the primary composite outcome in the intensive-treatment group than the standard-treatment group (1.65% per year vs. 2.19% per year; hazard ratio [HR] with intensive treatment = 0.75; 95% confidence interval [CI], 0.64-0.89; P<0.001). All-cause mortality was also significantly lower in the intensive-treatment group (HR = 0.73; 95% CI, 0.60-0.90; P=0.003). Rates of serious adverse events of hypotension, syncope, electrolyte abnormalities, and acute kidney injury or failure, but not of injurious falls, were higher in the intensive-treatment group

Table 37.7 A comparison of target BP across various guidelines[64-68]

	ESH / ESC, 2013[65]	ASH / ISH, 2014[66]	AHA / ACC / CDC, 2013[67]	JNC-8, 2014[64]	TSOC / THS, 2015[68]
Definition of hypertension	≥140/90	≥140/90	≥140/90	Not addressed	≥140/90
BP target (mm Hg)	<140/90	< 140/90	<140/90	<60 Years: <140/90	<140/90
	Elderly <80 years: SBP <140 in fit patients otherwise SBP 140-150	≥80 Years: <150/90		≥60 Years: <150/90	≥ 80 Years: <150/90
	Elderly ≥80 years: SBP 140-150				
BP target in patients with DM (mm Hg)	<140/85	<140/90	<140/90	<140/90	<130/80 (Also for coronary heart disease and chronic kidney disease with proteinuria <130/80)

Abbreviations: AHA / ACC / CDC = American Heart Association, the American College of Cardiology, and the Centers for Disease Control and Prevention; ASH / ISH = American Society of Hypertension and International Society of Hypertension; BP = blood pressure; DM = diabetes mellitus; ESH / ESC = European Society of Hypertension and European Society of Cardiology; JNC-8 = Eighth Joint National Committee; SBP = systolic blood pressure; TSOC / THS = Taiwan Society of Cardiology and the Taiwan Hypertension Society

than in the standard-treatment group. The published results did not specify whether the findings were consistent across various age-groups.[70] Nevertheless, caution has to be exercised in applying the research findings of SPRINT to geriatric practice: many frail elderly patients were excluded from the trial (e.g. older adults residing in nursing homes or assisted living facilities are excluded); higher risk of syncope and acute kidney injury in the intensive-treatment group and thus benefits have to be weighed against risk; thiazide was a preferred drug in the treatment algorithm and this drug is associated with a higher risk of hypokalaemia in elderly patients.

37.3.4.1 Non-pharmacological Management

All hypertensive elderly people should be counselled on lifestyle modifications that should include regular physical exercise and a low-salt diet. In the Trial of Nonpharmacologic Intervention in the Elderly that evaluated the effects of dietary salt restriction (mean, 1.8 g/day) and weight loss (mean, 4 kg), a 30% reduction in the need to reinitiate pharmacological treatment was demonstrated in the intervention group.[71] The effect of dietary sodium restriction on lowering SBP has been studied in a meta-analyses of randomised controlled trials; each decrease of 2.4 g in sodium reduced SBP by 3.7 mm Hg.[72] Increased potassium intake in the form of fruits and vegetables may also lower BP.[73] The initiation of non-pharmacological management can have synergistic effects with antihypertensive agents and further benefits may be obtained in reduction of other cardiovascular risk factors or in improving balance and cognitive function.

37.3.4.2 Pharmacological Management

The initial choice of medications is mainly influenced by the presence of comorbid conditions including diabetes mellitus, coronary artery disease, HF, and benign prostatic hypertrophy (Table 37.8[64-69]). According to JNC-8, thiazide diuretics, calcium channel blockers, ARB, or ACEI are first choice for initial pharmacotherapy.[64] Beta-blockers, centrally acting agents (e.g. methyldopa), and alpha-blockers are not recommended as first choice.[63] In general, a lower initial dose (approximately one half that of younger patients) should be used in older adults. The common side-effects associated with antihypertensive medications are summarised in Table 37.9.[64-69] Reduction in BP should also be gradual to minimise postural hypotension. The importance of home BP monitoring should be explained to the patient and their relatives. If satisfactory BP control cannot be attained despite good compliance with three or more antihypertensives, an evaluation for resistant or refractory hypertension (including renovascular disease, hyperaldosteronism) should be considered. For elderly patients with hypertensive emergencies (i.e. hypertensive encephalopathy, acute HF with pulmonary oedema, unstable angina or aortic dissection), management should be in an acute hospital setting (with consideration of

Table 37.8 Clinical trial and guideline basis for compelling indications for individual drug classes ('✔' refers to preferred items)[64-69]

Comorbid condition	Diuretic	Beta-blocker	ACEI	ARB	CCB	Alpha-blocker	Aldosterone antagonist
Congestive heart failure	✔	✔	✔	✔			✔
Ischaemic heart disease	✔	✔	✔		✔		✔
Diabetes mellitus			✔	✔	✔		
Chronic renal failure	✔		✔	✔			
Prevention of recurrent stroke	✔	✔	✔		✔		
Benign prostatic hypertrophy						✔	

Abbreviations: ACEI = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; CCB = calcium channel blockers

Table 37.9 Common side-effects of individual drug classes[64-69]

Individual drug class	Common side-effect
Diuretics	Hypokalaemia, hyperuricaemia, hypomagnesaemia, hyponatraemia, glucose intolerance, ototoxicity (loop diuretics)
ACEI	Cough, hyperkalaemia, angioedema, renal insufficiency (especially those with renal artery stenosis)
ARB	Hyperkalaemia, renal insufficiency (especially those with renal artery stenosis)
Long-acting dihydropyridine CCB	Ankle oedema, headaches, postural hypotension, constipation
Beta-blocker	Bradycardia
Alpha-blocker	Postural hypotension, higher risk of heart failure
Direct vasodilator (e.g. hydralazine)	Tachycardia, nausea, dizziness
Centrally acting agent (e.g. methyldopa)	Sedation, bradycardia, reflex tachycardia if stopped abruptly, depression

Abbreviations: ACEI = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; CCB = calcium channel blockers

management in a coronary care unit) with parenteral antihypertensives, e.g. labetalol, and with continuous BP monitoring. BP should not be reduced by more than 25% within 2 hours with a goal of 160/100 mm Hg gradually over the first 6 hours of therapy.[57]

37.3.5 Prevention

BP should be measured yearly in otherwise healthy elderly adults.[74] The frequency of monitoring depends on previous measurements. For those with high normal BP (SBP 130-149 mm Hg or DBP 85-89 mm Hg) or a history of high BP, checking should be done annually. In those whose BP remains within an optimal range, monitoring may be less frequent (e.g. every 2 years).

37.4 Aortic Aneurysm

37.4.1 Introduction

An aortic aneurysm is dilation of the aorta to more than 1.5 times of its normal size.[75] It is asymptomatic unless ruptured. They are most commonly found in the abdominal aorta followed by the thoracic aorta. Aortic rupture may occur due to weakness of the aortic wall and result in massive internal bleeding leading to shock or even death unless treated immediately.

Hypertension and atherosclerosis weaken the artery walls. These problems, along with the wear and tear that naturally occurs with ageing, can result in a weak aortic wall that protrudes outwards. An aortic aneurysm in older patients can also occur as a result of trauma or infection.

Aortic aneurysms are classified by their location along the aorta.

- Thoracic aortic aneurysms (TAAs) are found within the chest; these are further classified as ascending, aortic arch, or descending aneurysms.
- Abdominal aortic aneurysm (AAA), or called "triple A", the most common form of aortic aneurysm, involves the part of the aorta within the abdominal cavity. Thoracoabdominal aortic aneurysm, as its name suggests, involves both the thoracic and abdominal aorta.

37.4.2 TAA

TAA is defined as a permanent localised dilation of the thoracic aorta. The dilated aorta has a ≥50% increase in diameter when compared with the normal aortic diameter at that segment. The incidence of TAA is estimated to be 6-10 cases per 100 000 patient-years,[76] but increases with age and is more common in men of 60-70 years. Most TAAs grow slowly over years, with a 0.1-1 cm per year increase in aortic diameter.[77] There is an increasing risk of dissection as aortic size increases.

Patients with thoracic aneurysms are often asymptomatic at the time of presentation. Symptoms are mainly due to compression of adjacent structures and causes chest, back, or abdominal pain. The most serious complications of TAA are dissection and rupture. Patients may present with severe pain and shock.[78] Asymptomatic TAA may be discovered on imaging studies to evaluate an unrelated complaint. On chest X-ray, the aneurysm produces a widening of the mediastinal silhouette, enlargement of the aortic knob, or displacement of the trachea from the midline. CT angiography with intravenous contrast and MRI are the tests of choice to detect a TAA.[79]

Asymptomatic TAA and aneurysms in patients who are not candidates for repair are treated medically with BP control. The goal SBP is <120 mm Hg.[80] Patients should be monitored for any signs and symptoms. Serial imaging should be done with CT angiography or MRI.

For symptomatic patients, repair is an option but morbidity and mortality rates following open TAA repair are high.[81] Most patients have multiple comorbidities that increase the risks associated with surgery. Preoperative risk assessment and implementing strategies to reduce the risk of perioperative morbidity and mortality are important.

37.4.3 AAA

An AAA is a focal dilation of 50% greater than the expected normal diameter of the aorta.[75] It is common but potentially lethal. The segment of aorta below the arteries (infra-renal aorta) is most commonly affected.

AAAs are more common than their thoracic counterpart. One reason for this is that elastin, the main load-bearing protein in the wall of the aorta, is reduced in the abdominal aorta compared with the thoracic aorta. Another is that the abdominal aorta does not possess vasa vasorum, the nutrient-supplying blood vessels within the wall of the aorta. Most AAAs are true aneurysms that involve all three layers (tunica intima, tunica media, and tunica adventitia). The prevalence of AAAs increases with age, with a mean age of 65-70 years at the time of diagnosis. AAAs have been attributed to atherosclerosis although other factors are involved in their formation.

The risk of rupture of an AAA is related to its diameter; once the aneurysm reaches about 5 cm, the yearly risk of rupture may exceed the risks of surgical repair for an average-risk patient. Rupture risk is also related to shape. The so-called 'fusiform' (long) aneurysms are considered less rupture-prone than 'saccular' (shorter, bulbous) aneurysms; the latter having more wall tension in a particular location in

the aneurysm wall. An AAA expands 2-4 mm per year.[82] One that expands ≥0.5 cm over 6 months of follow-up is considered at high risk of rupture.

Approximately 4-8% of older men are determined to have an occult AAA on ultrasound screening.[83] The prevalence of AAA is 4-6 times lower for women but rupture is more common. Before rupture, an AAA may present as a large, pulsatile mass above the umbilicus. A bruit may be heard from the turbulent flow in the aneurysm. Unfortunately, in many cases, rupture may be the first hint of AAA. Once an aneurysm has ruptured, it presents with classic symptoms of abdominal pain that is severe, constant, and radiating to the back.

Most patients with AAA do not have any symptoms.[84] Similar to a TAA, an asymptomatic AAA may be discovered on routine physical examination or on imaging studies to evaluate an unrelated complaint. AAA may become symptomatic if the aneurysm is expanding rapidly and compressing surrounding structures. The most serious complication is rupture. Patients with symptomatic AAA most commonly presents with abdominal, back, or flank pain. The classic triad of ruptured AAA (acute abdominal pain, abdominal distention, and shock) only presents in about 50% of patients.[85]

Abdominal palpation cannot detect an AAA,[86] although sensitivity of palpation increases with increasing AAA diameter and is affected by the abdominal girth of the patient. Abdominal ultrasound and CT of the abdomen are both highly sensitive and specific for diagnosing AAA. Imaging demonstrating focal dilation of the aorta with >1.5 times the expected normal diameter is diagnostic of AAA.

Management of AAA depends on its size and whether it is symptomatic. Asymptomatic small (<5.5 cm in diameter) AAAs can be managed with watchful waiting.[87] Risk of aneurysm rupture does not exceed the risk of repair until the aneurysm diameter reaches 5.5 cm. For a symptomatic or large AAA, elective repair may be considered.[88]

37.5 Leg Ulcers

37.5.1 Introduction

Leg ulcers can affect up to 2% of the population above the age of 80 years, particularly obese and female individuals. They can compromise quality of life and have been shown to have negative physical, financial, and psychological impacts on patients.[89] Venous ulcers are a common type of chronic leg ulcer. Other important pathophysiological causes of lower limb ulcers are arterial insufficiency secondary to peripheral vascular disease, and repeated trauma to insensitive skin tissue due to peripheral neuropathy. The most common secondary leg ulcers occur in diabetes mellitus due to a combination of factors including vascular insufficiency, neuropathy, and infection. Other much rarer causes of non-traumatic leg ulcers include vasculitis, pyoderma gangrenosum and malignancy, particularly squamous cell carcinoma.

37.5.2 Clinical Assessment

A careful and complete medical history and physical examination are the mainstay in identifying the cause of leg ulcers. Comorbid conditions that may contribute to ulcer development must be identified such as diabetes mellitus, peripheral vascular disease, and autoimmune disease. A history of venous thrombosis, orthopaedic surgery, and prolonged bed rest predispose to venous leg ulcers. Heavy smoking contributes to arterial ischaemic ulcers. Medication screening is helpful as concurrent use of immunosuppressive drugs predisposes to infection and delayed ulcer healing. Patients with venous leg ulcers may complain of swelling and heaviness in their lower limbs on standing. Elevation of the leg improves discomfort. Arterial ulcers usually produce severe pain unless there is associated neuropathy. Patients with venous ulcers generally have mild pain over the wound. Intermittent claudication is an important cause of leg ulcers. Venous ulcers are strongly associated with varicose veins and are usually located around the ankles (Plate 37.1). The margin is well defined and a slough may be present. The surrounding skin may be erythematous with scaling, hyperpigmentation, crusting, and weeping. Long-standing oedema and fibrinous exudate lead to extensive fibrosis of the subcutaneous tissue with loss of pigmentation and development of capillary loops in the lower leg. It may lead to lipodermatosclerosis with consequent tapering of legs above the ankles, forming a constricting band that resembles an inverted champagne bottle (champagne bottle legs).

Arterial ulcers are located over a pressure area and are deep and sharply demarcated with little granulation tissue. The base is usually necrotic. Other features of arterial insufficiency may be present (Plate 37.2): loss of hair; atrophy; dry, cold skin; and absent, diminished peripheral pulses. Diabetic foot ulcer is extremely susceptible to infection. Neuropathic ulcers usually develop over weight-bearing areas (Plate 37.3). They are painless and deep, often surrounded by thick callus. Associated peripheral sensory loss and Charcot joints are important diagnostic clues. Clinical features of predisposing disease such as diabetes mellitus, spinal cord injury, and leprosy may be evident.

37.5.3 Management of Leg Ulcers

The principle of any ulcer treatment is to promote

growth of new epidermis across the ulcer to achieve good healing and to prevent / control any adverse factors that may affect this process. Any predisposing, causative systemic diseases such as diabetes mellitus or severe arterial disease must be adequately treated. The approach to treatment of venous ulcers is[90]:

- reduction of oedema by frequent leg elevation, diuretics, avoidance of prolonged standing, and use of compression bandages
- removal of exudates and slough but avoidance of antiseptic solutions that may reduce collagen formation and cause inflammation
- application of an appropriate dressing, e.g. an absorbent dressing for wound exudate
- treatment of clinical infection with systemic broad-spectrum antibiotics. Note that ulcer wounds are frequently colonised by bacteria and thus a positive wound culture does not necessarily imply infection
- topical steroids may be used to treat surrounding eczematous changes to reduce irritation
- skin grafting and corrective surgery may be considered for associated severe venous abnormality
- recent trials with human recombinant epidermal growth factors and the use of bioengineered skin have shown promising results[91-93]

Prevention of sepsis and urgent treatment is vital for all arterial ulcers. Tissue perfusion may be variably improved with aspirin and pentoxifylline. Vascular investigations, including Doppler ultrasound, employing the ankle brachial index can be helpful. An ankle brachial index of <0.6 indicates severe ischaemia although interpretation of the test result in elderly patients with significant sclerotic arteries is difficult. When an ulcer fails to respond to medical treatment, bypass surgery may be indicated to increase circulation to the limb, provided the circulation is adequate below and above the occluded area.

All diabetic patients should be screened regularly for early foot disorders and early foot ulcer care must be sought from a specialist team. To prevent the development of foot ulcers in high-risk patients, footwear should be modified to relieve pressure-sensitive areas. This can be done with padding or other protective devices or with orthotic devices, preferably after assessment by a podiatrist.

A neoplasm, like basal and squamous cell carcinoma, may complicate a long-standing non-healing ulcer. The possibility of a neoplastic transformation should be considered in a non-healing ulcer that shows exuberant granulation and rolling of edges over surrounding skin.

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CHAPTER 38

Chronic Obstructive Pulmonary Disease

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OBJECTIVE

To learn about multi-dimensional management of chronic obstructive pulmonary disease (COPD).

KEY POINTS

To provide individualised assessment and care for COPD patients:

- COPD in older adults is a complex disorder and its therapeutic targets are to alleviate the impact of symptoms, reduce exacerbations, and preserve activities of daily living functions.
- COPD is associated with geriatric syndromes such as falls, malnutrition, cognitive impairment, and functional decline.
- Benefits of pharmacotherapy in elderly patients are comparable with those in younger patients; and combination therapy is frequently required to augment treatment benefits and reduce side-effects.
- Other important interventions in the management of COPD are smoking cessation, vaccination, pulmonary rehabilitation, and oxygen therapy.
- Comorbidities commonly co-exist with COPD. These include cardiovascular disease, metabolic syndrome, skeletal muscle dysfunction, carcinoma of the lung, osteoporosis, depression, and weight loss.
- Frequent exacerbation reflects poor prognosis as it is related to a faster decline in lung function, higher risk of cardiovascular events, repeated hospitalisation, poor quality of life, and increase in mortality.

38.1 Introduction

The worldwide prevalence of chronic obstructive pulmonary disease (COPD) is rising. It is a common disease among elderly populations with a global prevalence of 14.2%.[1] In Hong Kong, the prevalence rate has been reported to be 10% among elderly population.[2] The annual admission rate among the public hospitals is 4% with a total of 65 497 episodes of COPD diagnosed in the years 2006 and 2007.[3]

In 2016, COPD ranked sixth in the top 10 leading causes of death in Hong Kong, with a mortality rate of 22.4 per 100 000 population.[4] A survey by Lai et al[5] revealed that around 10% of community-dwelling elders perceived 'shortness of breath' as one of their chronic illnesses. By the year 2020, COPD will be ranked third among the most important causes of death and fifth among conditions with a high global health burden.[6]

38.2 What is COPD?

According to the 2017 Global Initiative for Chronic Obstructive Lung Disease (GOLD) definition, COPD is a condition characterised by "persistent respiratory symptoms and airflow limitation that is due to airway and / or alveolar abnormalities usually caused by significant exposure to noxious particles or gases".[7] Exacerbations and comorbidities contribute to the overall severity in individual patients.

The chronic and persistent airflow limitation is caused by a combination of small airway disease (obstructive bronchiolitis) and parenchymal destruction (emphysema). Each pathology is mediated by chronic inflammation, and clinically, patient may present with varied mixture of respective pathologies. Previous definitions of COPD had included the terms 'chronic bronchitis' and 'emphysema' but were deliberately excluded by the GOLD working group since 2014.

Chronic bronchitis, defined as the presence of persistent cough and sputum production for at least 3 months in each of two consecutive years is a distinct disease which may present before or after the development of airflow limitation. Emphysema, defined as destruction of the alveoli, is a pathological term that may be used clinically to describe only one of several structural abnormalities. Chronic

bronchitis and emphysema can be found in patients with normal spirometry.

Symptoms of COPD include dyspnoea, chronic cough, and chronic sputum production. Spirometry, an effort dependent test, is essential to diagnose COPD[8] and repeat testing may be warranted to minimise variability in results. However, it has been shown that about four in five of frail elderly patients cannot perform full spirometry. Cognitive screening with Mini-Mental State Examination of <24/30 and / or inability to copy intersecting pentagon is a practical way to screen frail elderly people who are unlikely to perform spirometry reliably.[9] A post-bronchodilator forced expiratory volume in 1 second (FEV_1) / forced vital capacity of <0.70 confirms presence of persistent airflow limitation. A clinical diagnosis of COPD should be considered in any patient who has symptoms (dyspnoea, chronic cough, and / or sputum production), history of exposure to risk factors. Common risk factors include tobacco smoke, indoor air pollution (biomass fuel), occupational dusts and chemicals (vapours, irritants, and fumes), outdoor air pollution (relatively small effect) and alpha-1 antitrypsin deficiency which is uncommon in Hong Kong.

38.3 Age-related Changes in Pulmonary Function

Airflow limitation increases with age. The majority of human beings achieve peak pulmonary function at around 20 years of age. Elastic recoil of the lungs is affected by collagen and elastin. Collagen tends to be more cross-linked as we age despite the content of the collagen remaining unaltered. Elastin tends to undergo degeneration leading to the loss of elastic recoil with age. Reduction of bronchiolar airway size from the age of 40 years and widening of the alveolar ducts and smaller alveolar sacs result in thickening of the alveolar basement membrane (basal laminae). These physiological changes are sometimes known as 'senile emphysema' that can mimic the early stage of COPD.

Others changes are poor chest wall compliance, decreased lateral intercostal muscle mass, inability to perceive air resistance changes, and poor chemosensitivity to carbon dioxide that contribute to the high mortality rates in older adults with pulmonary diseases.[10,11] It has been estimated that FEV_1 decreases by 30 mL/year in men and by 23 mL/year in women who are non-smokers and that the rate of decline is even greater after the age of 65 years.[12]

38.4 Stable COPD and Exacerbations

Core principles in COPD management are immediate symptom relief, and reduction of its short- and long-term impact to patients.

Pharmacological therapies have not been shown to confer any survival benefits or ability to alter the long-term decline in lung function. Medication therapy in older adults is similar to that in younger patients and is mainly used for reduction of symptoms, reduction in exacerbation rate, and improvement in quality of life (QoL). Combination therapy with multiple inhaled agents is preferred as it provides synergistic benefits with fewer side-effects compared with monotherapy.

In past years, pharmacological treatment was largely guided by severity of airflow limitations. The GOLD 2017 report[7] has largely refined the strategy, with much emphasis on severity of respiratory symptoms and number of exacerbations that require hospitalisation instead of spirometric results. Severity of respiratory symptoms can be assessed using the COPD Assessment Test[13] or modified Medical Research Council (mMRC) Questionnaire,[14] both of which are validated. Accordingly, patients are classified into Groups A to D with suggested pharmacological treatments based on these two dimensions (see Chapter 38.4.2). In essence, patients with 0-1 exacerbation in past 1 year are classified as Group A (e.g. mMRC score 0-1) or B (e.g. mMRC score ≥2), and those with two or more exacerbations as Group C (e.g. mMRC score 0-1) or D (e.g. mMRC score ≥2). Spirometric results may give added value in guiding treatment only in Group D patients, of which roflumilast (see Chapter 38.4.1) may be considered.

38.4.1 Update on Pharmacological Treatment of Stable COPD

Bronchodilators (anticholinergic agents [muscarinic antagonists] and beta 2 agonists) remain the cornerstones of COPD therapy for the past two decades[7,15] Regular and as-needed use of short-acting beta agonists (SABAs, e.g. salbutamol) and anticholinergic agents (e.g. ipratropium) as monotherapy or combination therapy improve FEV_1 and symptoms.[16]

The inhaled medication delivery system is preferred to oral therapy. Currently, SABAs are mainly used in subjects with mild COPD symptoms with low risk of exacerbation (Group A). It is also used as an alternative to long-acting bronchodilators ± theophylline when long-acting inhaler is not available or there are cost constraints. SABAs are also used as rescue treatment in moderate-to-severe COPD during an acute exacerbation. The use of high doses of SABAs on an as-needed basis in stable COPD already being regularly treated with long-acting bronchodilators is not recommended.

The twice daily long-acting beta 2 agonists (LABAs) salmeterol and formoterol first became available as

maintenance therapy for COPD more than 15 year ago, while the once-daily long-acting muscarinic antagonist (LAMA) tiotropium has been available for 10 years.[17] Long-acting inhaler (e.g. tiotropium, salmeterol) significantly improves FEV_1, dyspnoea, exercise endurance, health-related QoL, exacerbation rate, lung hyperinflation, and length of stay in hospital.[18-20] Tiotropium has been shown to improve the effectiveness of pulmonary rehabilitation.[21] A variety of LABAs (e.g. indacaterol, olodaterol, vilanterol) and LAMAs (e.g. glycopyrronium, aclidinium) are under development.

The GOLD guidelines recommend the use of long-acting over short-acting bronchodilators, and combining long-acting bronchodilators with different mechanisms of action if symptom relief is not adequate with monotherapy.[22,23] The combination therapy also has the advantage of minimising side-effects.

Older adults usually tolerate the bronchodilators well. Beta 2 agonists can produce resting sinus tachycardia and other cardiac arrhythmia in older patients with concomitant cardiovascular disease, and induce cardiomyopathy. Hand tremors may appear in subjects receiving high doses, and hypokalaemia can occur, especially in the presence of thiazide diuretics. There is no evidence that beta 2 agonists induce accelerated loss of lung function or increase the mortality in COPD as was believed in the past. Older patients may develop tachyphylaxis and tolerance to beta 2 agonists.[24] The most common side-effect associated with anticholinergic drugs is dry mouth. Other rarer symptoms such as cough, urinary retention, and constipation may also occur, especially in the older adults. There were concerns about the occurrence of cognitive impairment with oral anticholinergic agents but ipratropium does not cause cognitive deficits.[25] Pupillary dilatation, blurred vision, and acute glaucoma can occur from escape of droplets from loosely fitting nebuliser masks. Because of the pro-arrhythmic and pro-ischaemic effects of inhaled anticholinergic medications, care should be taken in prescribing inhaled anticholinergics for patients with pre-existing arrhythmias or cardiac disease. The benefit of reduction in dyspnoea and COPD exacerbations should be weighed against the potential risks of serious cardiovascular adverse effects in each individual patient.[26]

The use of inhaled corticosteroids (ICS) in those with FEV_1 of <60% predicted improvements in symptoms, lung function, QoL, and frequency of acute exacerbation.[27-31] Latest GOLD guidelines recommend its use for Group C patients. There is currently no evidence to suggest that regular long-term use of ICS modifies the decline in FEV_1 and reduces mortality. Withdrawal may lead to exacerbations[32] and long-term use as monotherapy both orally and in the inhaled form is not indicated. Patients on long-term use may lead to development of oral candidiasis, skin bruising, cataract, open-angle glaucoma, and increased risk of pneumonia.[27] An association between ICS and fractures has been found in pharmacoepidemiological studies.[7] This is especially relevant for elderly subjects who are prone to falls resulting in debilitation. A meta-analysis including 16 randomised controlled trials and 7 observational studies indicated that ICS was associated with a modest but significant fracture risk, which was dose-dependent (see also chapter 38.6).[33]

Phosphodiesterase 4 inhibitors (PDE-4) is an oral anti-inflammatory agent used in a once-daily dose in subjects with severe and very severe COPD with a history of exacerbation, and chronic bronchitis not adequately controlled by long-acting bronchodilators (Group D patients). Roflumilast is a PDE-4 that reduces exacerbation when used in combination with long-acting bronchodilators alone or with ICS on top of the combination therapy (see Chapter 38.4.2).[34] The only drawback is the common side-effects of nausea, reduced appetite, abdominal pain, diarrhoea, sleep disturbance, and headache leading to withdrawal of the treatment.[34-36] Studies also reveal unexplained weight loss of 2 kg and roflumilast should be avoided in elderly with malnutrition, depression, and those taking theophylline.

It is best to avoid theophylline in elderly COPD due to its narrow therapeutic window. It is less effective as a bronchodilator compared with the newer long-acting inhaled bronchodilators and controversy still exists with regard to the exact effect of theophylline in COPD.[37-39] Adverse effects include palpitation, arrhythmias, myocardial infarction, nausea, vomiting, seizures, and headaches. Theophylline is now considered a third-line agent in COPD therapy that may be added if other better options are suboptimal or the better bronchodilators are not affordable for long-term treatment. It should be noted that elders (because of age-related altered pharmacokinetics and dynamics), those with cognitive impairment / confusional state (e.g. Alzheimer's disease) and patients with chronic hypoxia (e.g. COPD) are more prone to theophylline toxicity on the central nervous system, which can occur even within the 'normal' therapeutic index of the serum drug level. Furthermore, beta 2 agonists and theophylline both have sympathomimetic activities. Overstimulation of the sympathetic system not only results in cardiovascular side-effects, but also increases anxiety that results in worsening of dyspnoea. Moreover, both are catabolic agents that can potentiate muscle wasting and deconditioning and, thus, perpetuate

the dyspnoea-muscle wasting cycle. Many drugs alter theophylline clearance and older persons are more susceptible to adverse drugs reactions from drug-drug interaction.

38.4.2 Combination Therapies

The recent GOLD guidelines[7] recommend various combination therapy options for Group B, Group C, and Group D patients. Combining two bronchodilators with different mechanisms of action may provide a greater degree of bronchodilatation with less side-effects compared with high dosage via monotherapy.

The combination of SABA (salbutamol) and short-acting anticholinergic agent (ipratropium) is commonly prescribed locally and it is more effective than either agent alone.[40-43] Patients who are not adequately controlled with short-acting agents should receive additional long-acting agents in combination therapy. Long-acting formulations are preferred over short-acting bronchodilators for regular maintenance therapy. Combinations of LABA and anticholinergics (LABA / LAMA) are superior in improving symptoms and lung function. Studies have shown better airflow, exercise tolerance, reduction in rates of hospitalisation, hyperinflation, and rescue medication with combination of tiotropium bromide and salmeterol compared with tiotropium monotherapy.[21,23,43-47] ICS combined with either LABA or LAMA is more effective in improving lung function, health status, and exacerbation.[27,45,48] According to GOLD, it is recommended to add ICS to either LABA or LAMA in patients with high-risk COPD who have a history of one or more exacerbation requiring hospitalisation. Dual bronchodilator combination therapy is an alternative choice if the ICS / LABA or ICS / LAMA combination fails to control the symptoms. The use of triple therapy LABA / LAMA / ICS enhances QoL and reduces COPD exacerbations.[19] Triple therapy is, therefore, recommended in those with more severe cases of COPD not adequately relieved by dual bronchodilator combination therapy.

38.4.3 Inhaled Medication Delivery System

The amount of drug delivered directly to the airways is effort dependent. The ideal inhaler for optimal delivery in elderly populations is yet to be established. Elderly subjects may be handicapped with poor manual dexterity, difficulty in coordination, arthritis, muscle weakness, and visual and cognitive impairment.

This may pose a potential barrier against the use of metered-dose inhaler (MDI) alone in an elderly patient with COPD.[49] It has been found that elders with strong evidence of cognitive impairment are usually unable to master any of the inhaler devices. Cognitive function testing is recommended before they are considered for inhaled therapy. It has been shown that elderly subjects with a borderline abbreviated mental test score are significantly more likely to be able to learn to use the less complex turbohaler than an MDI.[50] Nevertheless, evidence has shown that, with the help of spacer (or other assistive devices such as aerochamber), an elder who has underlying cognitive impairment can be taught to better use inhaler drugs.[51,52] Dry-powder inhalers are breath-actuated and most elders find these less challenging to use than the MDI.[53-55] Major consideration in breath-actuated inhaler is the higher level of inspiratory effort required for delivery. MDI usually requires a minimum flow rate of 25 L/min while dry-powder inhaler needs a flow rate up to 30-60 L/min. Older adults with moderate COPD may only be able to achieve a maximum flow rate of 27-44 L/min and, therefore, are not suitable to use a dry-powder inhaler.[53] Inhaler technique should be assessed face-to-face as patients' perception of their inhaler skills correlates poorly with actual performance. It is recommended that reinforcing the correct technique is essential every 6 months.

There has been much controversy as to whether the nebuliser form of delivery is more effective than MDI (with or without assistive device). A study performed in an accident and emergency department among children with acute asthma did not show any superiority of nebulizer over MDI plus spacer.[56] Hospital practice after the severe acute respiratory syndrome (SARS) epidemic has changed and nebulisers were banned from hospital use as these were known to cause spreading of the SARS virus. The European Respiratory Society guidelines[57] and National Health Service[58] recommend the use of nebuliser only after failure of MDI plus assistive device despite adequate education on its use. Patients aged 70 years with COPD receiving long-term nebulisers do encounter difficulties such as cleaning, filling the reservoir, and assembling the device.[59]

38.4.4 Other Pharmacological Treatments

38.4.4.1 Vaccination

Annual influenza vaccination is recommended for all COPD patients. It reduces hospitalisation, pneumonia, all-cost mortality, and cost.[60-64] Pneumococcal polysaccharide vaccination is recommended in COPD patients aged ≥65 years[65-69] and those younger patients with FEV_1 of <40% predicted.[66]

38.4.4.2 Mucolytic Agents

Patients with COPD who have vicious sputum may benefit from mucolytics such as carbocysteine.

The overall effect is minimal. In the 2015 Cochrane systemic review, it was concluded that treatment with mucolytics may produce a small reduction in acute exacerbations and a small effect on overall QoL.[70] However, confidence in the review results is reduced by the fact that effects on exacerbations shown in early trials were larger than those reported by more recent studies, possibly because the earlier smaller trials were at greater risk of selection or publication bias, thus benefits of treatment may not be as great as was suggested by previous evidence.[70]

38.4.4.3 Antitussive and Vasodilators

Antitussive and vasodilators are not recommended.

38.4.5 Other Treatment Options

Besides pharmacological treatment, other treatments include pulmonary rehabilitation, exercise training, oxygen therapy, smoking cessation, ventilator support, and surgery.

38.4.5.1 Pulmonary Rehabilitation Programme

Rehabilitation forms an important component of COPD management. The primary aims of pulmonary rehabilitation programme (PRP) are to reduce symptoms, disability, and handicap; improve functional independence and QoL in people with lung disease.[71-73] The essential components include: smoking cessation; review of medical diagnosis and appropriate medical treatment; bronchial hygiene training; exercise training; energy conservation technique in activities of daily living; patient education on prevention of exacerbation; nutrition therapy; psychosocial adjustment; and self-management intervention. Usually, the service is provided by a multidisciplinary team with involvement of the patient's family and attention to individual needs.

Studies have proven the efficacy of PRP in increasing the peak workload, peak oxygen consumption, and endurance time.[74] PRP is usually conducted in outpatient setting, locally, but it can also be conducted in inpatients and at home.[75,76] The shortest duration of PRP is 6 weeks and the sustainability of the programme relies on patient's self-motivation to continue the exercise training at home. All COPD patients should be candidates for PRP; it should be noted that those with moderate-to-severe muscle weakness and exercise intolerance have more to gain and are probably most suitable for PRP.[77] Studies have suggested that even patients aged 80 years can benefit from pulmonary rehabilitation.[78] A recent study found no correlation between patient demographics, including age, and outcomes in pulmonary rehabilitation.[79] This confirms that older COPD patients should not be excluded from rehabilitation programmes simply because of their age.[79] There is a trend to capture patients earlier and there is strong evidence suggesting that post-discharge pulmonary rehabilitation is safe and importantly associated with a reduction in subsequent hospital admissions.[80,81]

38.4.5.2 Oxygen Therapy

Long-term oxygen therapy (LTOT) should be regarded as one of the medications in COPD management. There is evidence to suggest that LTOT of >15 hours daily can improve long-term survival. Current recommendations for prescribing LTOT (Table 38.1) are based on results from two randomised trials in patients with COPD published almost 30 years ago: the Nocturnal Oxygen Therapy Trial and the Medical Research Council study.[82,83]

Long-term use of supplemental oxygen improves survival in patients with COPD and severe resting hypoxaemia.[84] However, the role of intermittent oxygen in symptomatic patients with COPD who have moderate hypoxaemia at rest and desaturation with activity is unclear, and trials in this area are lacking.[85] Depending on outcome of interests, there is uncertainty with use of LTOT in those without resting hypoxaemia.[86] Short-burst oxygen therapy refers to the intermittent use of supplemental oxygen at home for 10-20 minutes at a time to relieve dyspnoea.

Older people may have limited income and cost may be a concern in terms of electricity bills. Domiciliary oxygen leads to constraints in mobility and there is concern on risk of fire in those who continue to smoke.

38.4.5.3 Exercise Training

Studies have shown that exercise training can improve exercise tolerance, reduce symptoms of dyspnoea, and improve QoL among COPD patients.[7,72,87] Yet, there is wide variation in how to conduct it in terms of several factors including: setting of exercise (inpatient, community-based), exercise intensity (symptom-limiting to high

Table 38.1 Indications for long-term oxygen therapy in COPD

- PaO_2 of ≤7.3 kPa (55 mg Hg) or SaO_2 of ≤88%, with or without hypercapnia confirmed twice over a 3-week period; or
- PaO_2 between 7.3kPa (55 mg Hg) and 8.0 kPa (60 mm Hg), or SaO_2 of 88%, if there is evidence of pulmonary hypertension, peripheral oedema suggesting congestive cardiac failure, or polycythaemia (haematocrit >55%)

Abbreviations: COPD = chronic obstructive pulmonary disease; PaO_2 = partial pressure of oxygen; SaO_2 = oxygen saturation

intensity), frequency (thrice weekly to thrice daily), and types of exercise. The optimal exercise intensity is unknown and it has to be individualised.[88] In general, it is recommended that the higher the intensity and frequency, the more is the gain. High level of exercise intensity (>60% maximal work rate) for 20-60 minutes per session is more potent in terms of inducing a physiological response to training than less intensive exercise (<40% maximal work rate).[89] Yet, this must be balanced against its practicability. Many COPD patients may not be able to train at this level, and substantially lower intensity exercise is another alternative.[90] Recently, more patients opted for interval exercise training, alternating short bouts of high-intensity exercise (30-180 seconds) with either rest or low intensity exercise.[91] The goal is to find ways to ensure sustainability and retain patients in the training programme. Combination of resistance and endurance training resulted in increasing strength in quadriceps and thigh muscles.[92] In the past, exercise training was mainly prescribed rigidly and comprised of cyclical training.[74] However, it is now believed that walking is relatively easy to prescribe, economical, and enjoyable for individuals.[93] More research is expected to focus on walking in the near future.

38.4.5.4 Nutrition Supplementation

Nutrition in COPD has been a topic of extensive scientific research since the landmark paper by Wilson et al in 1985.[94] Malnutrition is well recognised in COPD, with 25-40% of COPD patients in an undernourished state.[95-99] A low body mass index (BMI) has been shown to be an independent predictor of mortality, regardless of the pulmonary status, even in those on LTOT.[98-102] The 2012 Cochrane meta-analysis and other studies have shown that oral nutritional supplementation improves nutritional status in malnourished patients with COPD without compromising the ventilatory system, and even improves respiratory muscle strength and health-related QoL and exercise tolerance.[103,104] This also applies to older patients.[105] A low fat-free mass (FFM) has been shown to be an independent predictor of mortality in COPD, even in those with a normal BMI.[106-108] This may explain why even a normal BMI in patients who have COPD has been shown to be associated with increased mortality. A decline in FFM has also been shown to be associated with increased frequency of COPD exacerbations and the use of corticosteroids.[109]

To improve undernutrition in COPD patients, most experts advocate the consumption of high-calorie foodstuffs (high fat / low carbohydrates),[110] fruits and vegetables, and intake of nutrients such as vitamins, amino acids, and unsaturated fatty acids.[111] Studies have shown that nutritional supplement therapies may not only improve undernutrition in COPD patients, but also provide various other beneficial effects such as prevention of the development, progression and exacerbation of COPD, and suppression of inflammation.[112,113]

Concurrent PRP on top of supplementary caloric intake gives additional benefit. For example, a study with combined regimens consisting of nutritional supplement therapy and concomitant low-intensity respiratory rehabilitation over a period of 12 weeks, found increased body weight and FFM, improvement in QoL and exercise tolerance, and reduced blood levels of inflammatory cytokines.[114]

Further large-scale randomised controlled trials are needed to clearly define the relationship of dietary supplements with various phenotypes and severity of COPD and the effect of hormones such as adipokines on systemic inflammation.[115,116]

38.4.5.5 Smoking Cessation

Smoking cessation is one of the interventions that alters the natural history of COPD. Irrespective of the severity of disease, smoking cessation in older patients improves health and reduces mortality.[117-121] With effect from 1 January 2007, statutory 'no smoking' areas in Hong Kong have been extended to cover the indoor areas of all restaurant premises, indoor workplaces, public indoor places, and some public outdoor places in accordance with the amended Smoking (Public Health) Ordinance (Cap. 371).[122] At present, there are a number of smoking cessation clinics run by the Department of Health, and Smoking Counselling and Cessation Centres run by the Hospital Authority (https://www.ha.org.hk/visitor/ha_visitor_text_index.asp?Content_ID=10098&Lang=ENG&Dimension=100&Parent_ID=10085). Some private doctors and private hospitals also provide smoking cessation services that smokers may join.[123]

A long-term abstinence rate of 25% can be achieved if counselling and resources are dedicated to promote smoking cessation.[124] Most physicians do not recommend smoking cessation as frequently in older compared to younger patients, mainly due to the wrong assumption that it is too late or that cessation rate is low in older patients.[117,121] In fact, studies show that the smoking abstinence rate is comparable between young and old subjects.[124,125] Although multiple safe and fairly effective pharmacological interventions are available such as nicotine replacement, bupropion, and varenicline; concern has been raised by the US Food and Drug Administration about the risk of adverse neuropsychiatric symptoms such as behavioural changes, depression, and agitation.[126] More information on pharmacotherapy for smoking cessation can be obtained from the review by Elrashidi et al in their 2014 updates.[127]

38.5 COPD Exacerbations

According to the GOLD guidelines, COPD 'exacerbation' is defined as an acute worsening of respiratory symptoms that leads to additional therapy.[7] Major cause of exacerbation is tracheobronchial infection although up to one-third of exacerbations has no infective element and the cause is unknown.[128] It should be noted that shortness of breath from COPD 'exacerbations' is multifactorial in older patients. It can be due to 'true' exacerbations related to bacterial or viral infection (fever and elevated white cell count, with or without microbiological or serological proof) or conditions such as congestive heart failure, pneumothorax, pulmonary embolism, and cardiac arrhythmia that can either mimic or aggravate the exacerbation. While exacerbations can also be due to cold weather or air pollution,[129] these precipitating factors can probably be reduced now that the Hong Kong Observatory gives good prediction of temperature and actual measurement of air pollution index. Patients should be educated to use this information to protect themselves in adverse circumstances. There exists a group of patients in whom the exacerbation is due to incomplete medical diagnosis, non-compliance, psychological factors, physiological, and metabolic derangement.

Specific treatment includes oxygen to maintain saturation targeted within 88-92% and short-acting inhaled beta 2 agonists with or without short-acting anticholinergics.[130] Bronchodilators can be administered via MDI with or without spacer and there is no significant difference in FEV_1 with the use of MDI and nebulisers.[131] In fact, nebuliser is no longer recommended in Hong Kong hospitals since the SARS outbreak in 2003. Arterial blood gas analysis should be obtained within 30-60 minutes to rule out carbon dioxide retention. Systemic corticosteroid has been shown to be useful in shortening the recovery time, improving lung function, and reducing relapse and hospital length of stay.[132-134] A dose of prednisolone 40 mg daily for 5 days is recommended.[135] However, given the high prevalence of tuberculosis in Hong Kong, especially among elderly males, the appropriateness and safety of using intermittent courses of oral steroids during acute exacerbations has been questioned.[136] Antibiotics are given to those with severe exacerbation or increased dyspnoea, sputum volume, and purulence.[128,137,138]

Non-invasive positive pressure ventilation (NIPPV) has become both the 'gold standard' and 'standard of care' for most patients with an acute exacerbation of COPD of sufficient severity to require ventilatory support. It can be given to suitable older adults with severe exacerbation,[139] and has been shown to reduce the mortality and intubation rates associated with severe COPD exacerbation. However, up to 30% cannot tolerate NIPPV, especially those with confusion, uncooperative status, excessive amount of sputum, and unstable vital signs.[140-142] In acute exacerbation, special attention should also be given to hydration status, undernutrition, management of comorbidities, and early mobilisation.

38.5.1 Frequent COPD "Exacerbations"

Frequent exacerbation reflects poor prognosis as it is related to a faster decline in lung function, higher risk of cardiovascular events, repeated hospitalisation, poor QoL, and an increase in mortality.[143-146] In those who present with two or more COPD exacerbations per year, nothing is more important than review of the underlying diagnostic labelling, and circumstances leading to or precipitating the 'exacerbation'. If the underlying cause is either chronic asthma or bronchiectasis, it should be treated accordingly and appropriately. Environmental factors such as the local practice of burning of jog stick should be ruled out. One should consider the possibility of gastroesophageal reflux disease in which micro-aspiration of gastric acid precipitates frequent exacerbations. In very frail COPD patients, one should also entertain the possibility of recurrent aspiration pneumonia due to respiratory or swallowing muscle incoordination instead of 'COPD'. Education on swallowing precaution with or without modification of diet can do much to reduce the 'COPD exacerbation'. Patients with intact cognition should be educated about the importance of understanding of their own condition and developing a self-help management plan. Rapid recognition of exacerbation symptoms and early treatment improves recovery and reduces the risk of hospitalisation.[147] There exists a phenotype of patients which is susceptible to frequent exacerbations irrespective of the stages of COPD.[148] There are 'rescue' programmes both locally and overseas that recommend these patients to keep a course of antibiotics and corticosteroids at home. This is part of the self-management programme and patients are educated to consume the medications at early signs of exacerbation.[149,150] It should be noted that in the 2017 GOLD update, patients with one or more exacerbation requiring hospitalisation are classified as the high-risk group.

38.6 COPD Comorbidities

Another key component in managing COPD is identification of comorbidities. These are common in any stage of COPD making differential diagnosis difficult even for the most experienced physician.[151] Some may be independent of the disease while others may be causally related. Commonly encountered comorbidities include cardiovascular diseases such as heart failure, osteoporosis, mental illness, lung cancer, infections, metabolic syndrome, and bronchiectasis.

Cardiovascular diseases are widely prevalent in COPD patients with the rate being 2-3 times higher than in those without COPD. The use of selective beta 1 blockers is considered safe but high doses should be avoided. Majority of the young COPD subjects die of cardiovascular disease rather than the disease itself. Heart failure is also common with a prevalence of about 30% in COPD patients.[152,153] Heart failure, COPD, and asthma with common cardinal symptom of breathlessness could coexist making diagnosis and management a great challenge. In general, cardiovascular disease should be treated as usual according to the various clinical guidelines but patient has to be observed closely to avoid adverse side-effects.

Anxiety and depression are major comorbidities easily overlooked. About 42-46% of older COPD patients have depression. They are generally associated with a poorer prognosis. Attention to risk factors such as age, social isolation, poverty, severe symptoms, immobility, and insomnia is important.[154]

The risk of osteoporosis increases in older adults with COPD and it is often underdiagnosed. The risk is greater with moderate-to-severe COPD.[155] The prevalence is up to 33% in older women and 11% in older men. Systemic corticosteroid use markedly increases the risk of osteoporosis and should not be used on a long-term basis. As mentioned in Chapter 38.4.1, meta-analysis revealed a dose-dependent higher fracture risk in those exposed to ICS.[33] However, a study of 251 COPD males showed that low-dose ICS reduced annual bone mineral density loss in bronchitic patients.[156] It was hypothesised that low-dose ICS downregulate the pulmonary and systemic inflammatory profile of COPD patients (thus reducing the COPD osteoporosis comorbidity risk) while at the same time would not aggravate the bone mineral loss, because of their lower circulating levels from systemic absorption.[156] Another retrospective cohort and population-based study in Taiwan showed that female patients with COPD using ICS had a dose-response protective effect for osteoporosis.[157] Other important clinical risk factors of osteoporosis that have been described in COPD patients included older age, emaciation, physical inactivity, and vitamin D deficiency.[158] Because osteoporotic fractures have significant impacts on QoL and even respiratory function in COPD patients, early detection and treatment of comorbid osteoporosis is warranted in those with risk factors of osteoporosis.[158]

38.7 Summary

COPD is widely prevalent in older adults and has a progressive nature that worsens with advancing age. Spirometry and symptomatology are essential for diagnosis and staging, even in older adults. Early diagnosis is advocated and current treatment is aimed at relieving the impact of symptoms and reducing adverse events such as exacerbations. There is a trend to use long-acting bronchodilators in early stage of the disease and older patients benefit the same as younger patients do, even with combination therapy. In addition, treatment of COPD in older adults involves effective management of the comorbidities and preserving functional abilities. Attention should be given to vaccination, nutrition, smoking cessation, inhaler technique and the use of pulmonary rehabilitation and home oxygen therapy.

For those with frequent 'exacerbation', nothing is more important than a detailed history on circumstances leading to recurrent 'exacerbation'; appropriate management should be initiated accordingly. Patients should be educated on chronic disease management approach and clinicians should empower caregivers and patients about disease control.

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CHAPTER 39

Anaemia in Old Age

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OBJECTIVE

To learn the most common causes of anaemia in elderly people and to perform an initial evaluation, interpret serum iron, total iron-binding capacity, and ferritin results in differentiating anaemia of chronic disease from iron deficiency and to recommend treatment for common anaemia.

KEY POINTS

- Anaemia in elderly people is not a normal age-related change and symptoms of anaemia may be erroneously attributed to ageing.
- Chronic disease and iron deficiency are the most common causes of anaemia in old age.
- The mean corpuscular volume may not be representative of the classic causes of anaemia. Investigations may have to be expanded if a classic cause is not found.
- Iron-deficiency anaemia most often results from chronic gastrointestinal blood loss and should prompt an evaluation of the gastrointestinal tract to look for bleeding.
- Vitamin B_{12} deficiency may or may not result in anaemia.
- The causes of anaemia in some elderly patients remain unexplained even after extensive evaluation.
- Anaemia is commonly associated with heart failure that is linked to chronic kidney disease, inflammatory cytokines, haemodilution, and use of heart failure medication.

39.1 Special Consideration in Anaemia in Old Age

The World Health Organization (WHO) defines anaemia as serum haemoglobin of <12 g/dL in women and <13 g/dL in men. Many studies have shown that low normal haemoglobin level by WHO criteria is associated with increased mortality, hospitalisation, and functional decline in elderly people.[1] Thus, the WHO threshold has been widely criticised but data have shown that important causes of anaemia can be identified if evaluation is carried out for anaemia as defined by the WHO threshold and it remains a useful reference when comparing anaemia prevalence in different studies.

In studies of community-dwelling older adults from the US and Europe, the prevalence of anaemia ranged from 8-25%. In the NHANES III (third US National Health and Nutrition Examination Survey) that involved a community-dwelling population aged ≥65 years, the prevalence of anaemia was 11% in men and 10.2% in women.[2] The prevalence also increased with increasing age, reaching 26% in men and 20% in women aged ≥85 years.[2] The severity of anaemia reported in these studies tended to be mild, with haemoglobin usually above 10 g/dL.[2] Anaemia was generally more prevalent and severe among institutionalised elderly people.

Anaemia is associated with increased mortality. In a study of 686 community-dwelling women aged ≥65 years, a serum haemoglobin of <11 g/dL was associated with increasing all-cause mortality.[3] In a Dutch study of 755 community-dwelling people aged ≥85 years, a haemoglobin level of <13 g/dL in men and 12 g/dL in women was associated with increased relative risk of mortality of 1.6 in men and 2.3 in women.[4] Disorders such as malignancy, peptic ulcer, and infection were more common in the anaemic population.

Anaemia is associated with decreasing physical and mental performance in older people. Many studies have confirmed the association between anaemia and impaired mobility, frailty,[5] muscle weakness,[6,7] and falls.[8] There is also an association with impaired cognitive performance, depressive symptoms, and reduced quality of life.[6,8,9]

Anaemia in old age is easy to miss because elderly people may subconsciously decrease their activity level to compensate for the effects of anaemia, thus

delaying recognition. Anaemia may also develop slowly, causing few symptoms and signs until late in the course of disease. Symptoms such as fatigue, weakness, and lack of stamina may be erroneously attributed to ageing.

There are very few symptoms specific to anaemia in elderly people. It may be difficult to separate the effect of shortness of breath from congestive heart failure that has been exacerbated by anaemia. The physical signs may be overshadowed by other disorders aggravated by anaemia, e.g. congestive heart failure, cognitive impairment, and apathy. It is also more difficult to detect conjunctival pallor in elderly patients.[10]

39.2 Aetiology

Many conditions can cause anaemia in elderly people and there may be more than one underlying factor (Table 39.1). In practice, the aetiology can usually be determined. The most common causes of anaemia in elderly persons include iron deficiency (with or without blood loss), chronic disease / inflammation, and chronic kidney disease.[2,11-13]

Table 39.1 Prevalence of different causes of anaemia in elderly people[11-13]

Cause	Prevalence (%)
Iron deficiency	15-23
Chronic disease / inflammation	15-35
Chronic kidney disease	8
Endocrine problems	<5
Vitamin B_{12} or folate deficiency	0-14
Myelodysplastic syndromes	0-5
Unexplained anaemia	17-45

Other aetiologies in elderly patients include vitamin B_{12} or folate deficiency, diseases of the bone marrow (e.g. myelodysplastic syndrome [MDS], aplastic anaemia, myeloproliferative syndrome, acute or chronic leukaemia, myeloma), hypothyroidism, hypersplenism, and haemolytic anaemia. Despite a complete evaluation, in a significant number of cases no apparent aetiology will be identified, despite thorough investigations, i.e. unexplained anaemia.[11,12]

39.3 Effect of Age on Erythropoiesis

The bone marrow is capable of continuous replication even in elderly people. The short-term response to haemopoietic growth factors seems to be intact and anaemia tends to occur as a result of a reduced reserve of haemopoietic progenitors with advancing age. Anatomical studies have suggested that bone marrow cellularity decreases by approximately one-third during adult life, and bone marrow scans using iron labelling of erythroid elements have also indicated a decline in marrow activity with advancing age.[14] Magnetic resonance imaging studies in humans have also confirmed a shift from red to yellow marrow at all sites with age, with an age-associated shift of red marrow from the appendicular to the axial skeleton.[15]

Human studies have shown no age-related reduction in committed haematopoietic stem cells or differentiated haematopoietic cell numbers.[16] Nonetheless the ageing individual has a decreased reserve capacity and may not respond to infection with the same degree of leukocytosis or to bleeding with the same degree of reticulocytosis as a younger person. These blunted haematopoietic responses may be caused by age-related defects in the marrow progenitor cells, changes to the marrow microenvironment, decreased production of

Table 39.2 Classification of anaemia in elderly people

Hypoproliferative	Ineffective haematopoiesis	Haemolytic

Intrinsic marrow lesion
• Stem cell dysfunction
  o Aplastic anaemia
  o Red blood cell aplasia
• Marrow replacement
  o Leukaemia
  o Myelofibrosis
  o Tumour infiltration
• Myeloma
• Mild marrow failure (aetiology unknown)
 	
Insufficient erythropoietin
• Renal disease
• Nutritional
• Endocrine, e.g. thyroid
 	
Iron-deficient erythropoiesis
• Iron-deficiency anaemia
• Anaemia of chronic disease / inflammation
	Megaloblastic
	• Vitamin B_{12} deficiency
	• Folate deficiency
	
	Microcytic / normocytic
	• Thalassaemia
	• Myelodysplastic syndrome (usually macrocytic)
	• Sideroblastic anaemia (congenital, drug-induced)
		Immunologic
		• Idiopathic
		• Secondary
		  o Drugs
		  o Lymphoproliferative disease
		  o Collagen vascular disease
		
		Intrinsic
		• Metabolic
		• Haemoglobinopathy
		
		Extrinsic
		• Mechanical
		• Lytic agents

regulatory growth factors, or a combination of these factors. A lot of these data, however, are conflicting and remain to be rigorously studied.

39.4 Classification of Anaemia

Anaemia can be classified into three types as shown in Table 39.2.

39.5 Initial Work-up

The work-up for anaemia in elderly people begins with a thorough history and physical examination to look for symptoms and signs of blood loss, malignancy, renal or hepatic failure, nutritional deficiencies, haemolysis, and chronic infection. A history of recent hospitalisation may result in anaemia due to an acute illness itself, or rarely multiple phlebotomies. A history of recent surgery would suggest peri-operative blood loss while end-stage renal disease uniformly causes anaemia. The symptoms of anaemia include fatigue, weakness, dyspnoea on exertion, and angina. They are non-specific and may relate more to the rapidity of the anaemia or the depth of fall in haemoglobin or to associated medical conditions. It is also useful to enquire about use of prescribed and over-the-counter medications as well as use of traditional Chinese medicine.

The initial laboratory evaluation should include complete blood count, red blood cell (RBC) indices, reticulocyte count or reticulocyte production index, peripheral blood smear, and serum biochemistry. It is useful to compare previous blood counts if available (Figure 39.1).

Most anaemia algorithms are based on the mean corpuscular volume (MCV) with separate pathways for macrocytosis, microcytosis, and normochromic anaemia.[17] These may be less helpful in elderly people because classic erythrocyte size does not always accompany anaemia in this age-group.

Usually, normocytic, normochromic anaemia predominates in elderly people. Occasionally, the MCV may point away from the true cause,[18] e.g. classic causes of macrocytosis can present with microcytosis and vice versa. Therefore, initial evaluation of anaemia should follow the algorithm for MCV, but

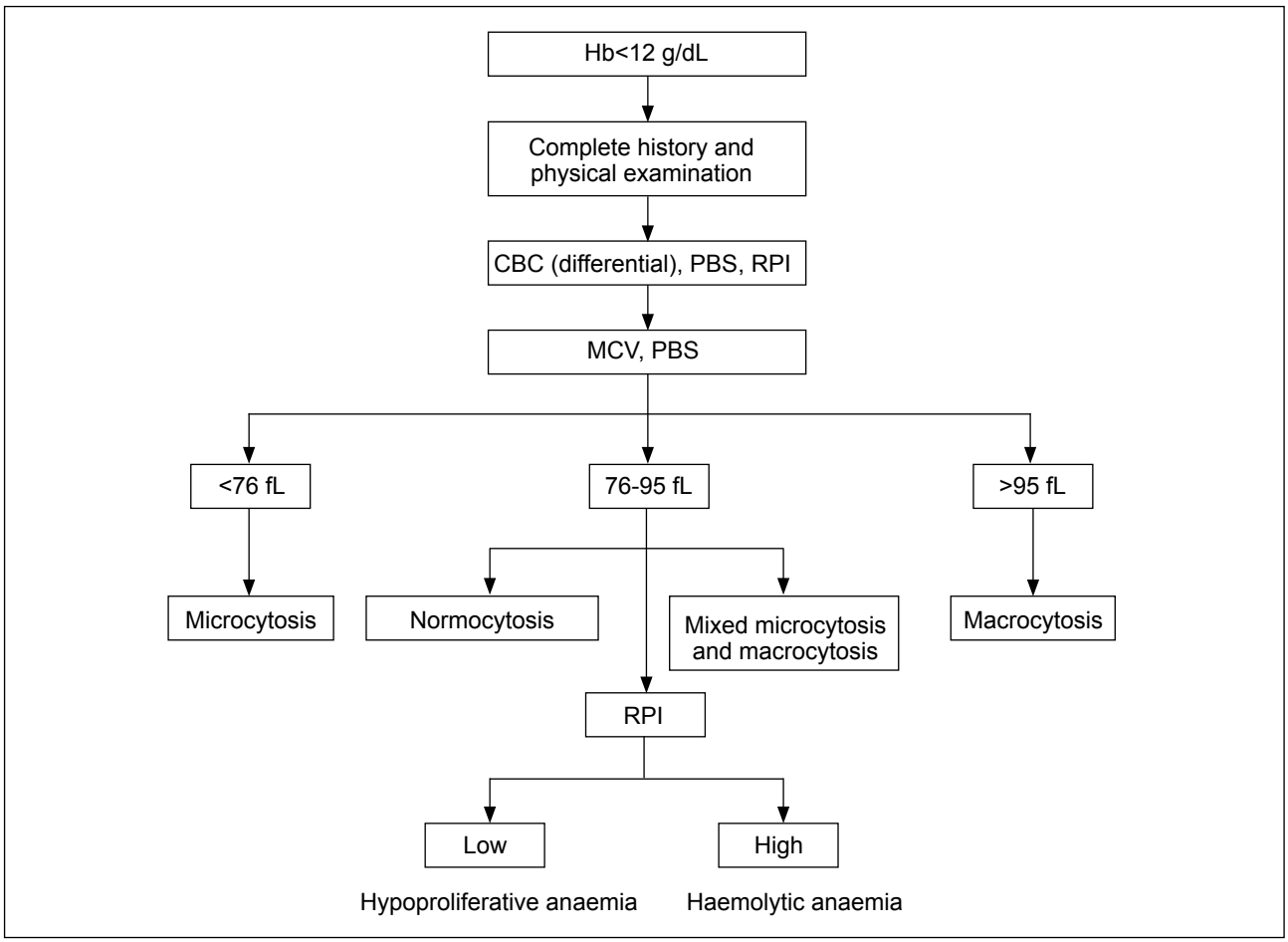

Figure 39.1 A practical approach to anaemia in old age
Abbreviations: CBC = complete blood count; Hb = haemoglobin; MCV = mean corpuscular volume; PBS = peripheral blood smear; RPI = reticulocyte production index

be expanded if no obvious classic cause can be found. If the patient is unstable, blood transfusion should be arranged after checking iron studies, ferritin, vitamin B_{12}, and folate level.

39.6 Anaemia of Chronic Disease

Anaemia of chronic disease (ACD) is by far the most common form of anaemia in elderly people.[19,20] Anaemia forms part of the clinical picture of many acute and chronic diseases, including inflammatory processes, malignant disease, and infection.

Several mechanisms are involved in the development of ACD: failure of erythropoiesis, lack of iron for haemoglobin synthesis (iron block); and decreased RBC survival. There is abnormal iron metabolism with reduced absorption of iron from the gastrointestinal tract and trapping of iron in macrophages. This results in reduced plasma iron level, making iron unavailable for new haemoglobin synthesis.

Inflammatory markers implicated in ACD include tumour necrosis factor–alpha (TNF-alpha),[21,22] interleukin-1 (IL-1),[23,24] interleukin-6 (IL-6),[25,26] and interferon gamma.[27]

Hepcidin is a 25 amino acid peptide synthesised by the liver, and serves as a primary regulator of iron homeostasis. Hepcidin inhibits ferroportin that mediates iron absorption from the gastrointestinal tract and iron release from the reticuloendothelial system. Inflammation upregulates hepcidin expression,[28,29] and thereby suppresses ferroportin expression leading to impaired iron absorption and iron block.

There is a haemolytic component to anaemia in ACD as RBC survival is mildly decreased to 80-90 days due to increased phagocytic capacity of the macrophages.[30,31] There is a relative decrease in erythropoietin (EPO) production and patients with ACD have lower levels of EPO compared with patients with iron deficiency and a similar degree of anaemia.[32]

Typically, ACD is of mild-to-moderate severity, non-progressive, usually appears 1-2 months after onset of chronic disease and correlates with the severity of underlying disease. Rarely the haemoglobin falls below 10 g/dL. Classically the anaemia is normocytic and normochromic but in <25% of patients it can be hypochromic and microcytic with MCV of <70 fL.[33]

Both the serum iron concentration and total iron-binding capacity are usually low but a usually normal transferrin saturation helps distinguish ACD from iron-deficiency anaemia.[34] A reduced ability to release iron from macrophages is presumably responsible for the low serum iron level and low transferrin saturation. Ferritin is an acute-phase reactant and is usually normal or elevated in ACD but cannot represent the iron store in the presence of chronic inflammatory diseases. Bone marrow examination in general reveals a normal number of erythroid precursors but erythroid iron is decreased while the storage iron is increased, i.e. iron block.

There is no specific treatment for the ACD. Rather, optimal management and treatment of the underlying disease is warranted. Despite a low iron level, iron therapy does not help. The use of erythropoiesis-stimulating agents (ESA) has been shown to improve haemoglobin level in ACD.

39.7 Iron-deficiency Anaemia

Iron-deficiency anaemia is the second most common cause of anaemia in elderly people and is usually a consequence of chronic gastrointestinal blood loss. Causes of such blood loss include non-steroidal anti-inflammatory drug–related gastritis, peptic ulcer disease, gastroesophageal reflux disease with oesophagitis, stomach and colon cancer, diverticular disease, as well as angiodysplasia. Less commonly, chronic blood loss from the genitourinary or pulmonary tract can also lead to iron-deficiency anaemia. Iatrogenic anaemia can be due to repeated phlebotomy during hospital admission with consequent iron-deficiency anaemia. Malabsorption or dietary deficiency are uncommon causes of iron deficiency in the older adult.

Classically iron deficiency causes microcytosis. Iron deficiency is diagnosed when the serum ferritin is decreased or a decreased serum iron is accompanied by a normal or increased total iron-binding capacity, with the transferrin saturation being ≤15%.

Iron-binding capacity decreases with ageing and is affected by factors such as malnutrition and chronic disease, both of which have a higher prevalence in elderly people. Ferritin is an acute-phase reactant and its level increases with ageing and may be elevated by acute and chronic inflammatory conditions. It is estimated that acute inflammation can triple the serum ferritin level.[35] A study carried out in hospitalised elderly patients showed that a higher serum ferritin level ≤110 pmol/L (50 μg/L) is a more reliable predictor of iron deficiency in elderly patients than the traditional cut-off value of serum ferritin ≤33 pmol/L (15 μg/L) commonly used for defining iron deficiency in younger adults.[36]

The anaemia of iron-deficiency in elderly patients may not present with the classical microcytic picture. MCV reduction in elderly patients is often lacking in early stages or blunted by other concomitant folic acid / vitamin B_{12} deficiencies. Nevertheless, only 6%

of patients with MCV of >95 fL have iron-deficiency anaemia, thus a very large MCV virtually excludes iron deficiency anaemia. A serum ferritin level of >220 pmol/L (100 μg/L) also makes iron deficiency highly unlikely.

The diagnosis of iron-deficiency anaemia prompts evaluation of the gastrointestinal tract for any source of blood loss. Upper gastrointestinal endoscopy is perhaps the most useful investigation and a bleeding source, e.g. peptic ulcer, gastritis, oesophagitis or gastric cancer can be identified in 20-40% of cases.[37-39] Colonoscopy should be considered if no upper gastrointestinal cause can be found since a pathology e.g. colon cancer, angiodysplasia, polyps or colitis, or diverticular disease can be identified in the lower gastrointestinal tract in 15% of cases.[37-39] In 10-40% of cases, no identifiable cause can be found despite intensive investigations.

39.7.1 Treatment of Iron-deficiency Anaemia

The cornerstone of management of iron-deficiency anaemia, in addition to iron supplementation, is identification and correction of a source of bleeding. Oral iron is a safe, cheap, and effective means of supplementation. There are a few simple principles governing the use of oral iron.

- Iron is absorbed best from the duodenum and proximal jejunum. The more expensive enteric-coated or sustained-release capsules, which release iron further down the gastrointestinal tract, are not good choices.
- Iron salts should not be given with food because the phosphates, phytates, and tannates in food bind the iron and impair its absorption.
- Iron should be given 2 hours before, or 4 hours after, ingestion of antacids.
- The cheapest preparation available is oral ferrous sulphate, containing 300 mg of iron salts, of which 60 mg is elemental iron.

A single 300-mg ferrous sulphate tablet taken orally 3 times daily between meals provides 180 mg of elemental iron per day. This regimen should lead to a modest reticulocytosis after 5-7 days and a rise in the haemoglobin concentration of approximately 2 g/dL over 3 weeks. It is necessary to treat for about 6 months after the haemoglobin normalises in order to completely replenish iron stores.

Up to 50% of elderly people may experience gastrointestinal side-effects such as nausea, constipation, diarrhoea, epigastric discomfort, or vomiting and these often lead to poor compliance.[40] In this case, it is possible to initiate treatment at a lower oral dose with slow dose escalation, or to take it with meals, although this will impair absorption.

Parenteral iron is rarely indicated in a patient who is unable to tolerate even a modest dose of oral iron, or in patients whose level of continued gastrointestinal bleeding exceeds the ability of the gastrointestinal tract to absorb iron.

39.8 Megaloblastic Anaemia

Megaloblastic anaemia refers to a group of disorders with abnormal haematopoiesis characterised by the appearance of megaloblasts in the bone marrow, and characterised by dyssynchronous nuclear and cytoplasmic maturation in all cell lineages. This is usually associated with peripheral macrocytosis and hypersegmentation of neutrophils in the peripheral blood. The most common cause of megaloblastic anaemia is isolated or combined deficiency of cobalamin or folate. Megaloblastic anaemia occurs most frequently in patients older than 60 years.

As macrocytosis occurs prior to anaemia in nearly all subjects with megaloblastic anaemia, a MCV of >95 fL should prompt the physician to institute further diagnostic investigations. A normal MCV may nonetheless be encountered in patients with megaloblastic anaemia and coexisting iron deficiency or thalassaemia.

39.8.1 Vitamin B_{12} Deficiency

Vitamin B_{12} (cobalamin) deficiency is the cause of anaemia in 5-10% of elderly people.[41,42] It can be difficult to detect in elderly people who do not always show the typical symptoms and signs. Anaemia is only present in about 60% of older patients found to be vitamin B_{12} deficient.[43] Anaemia need not be present in a patient who has complications of vitamin B_{12} deficiency.[43] In addition to haematological changes, cobalamin deficiency can cause neurological deficits, such as spinal cord degeneration and changes to mental state. Left untreated, it is estimated that 80-90% of deficient individuals will develop neurological disorders with symptoms such as fatigue, ataxia, muscle aches, irritability, paranoia, dementia, and depression. If therapy can be started within 3-6 months of the onset of symptoms, most neurological deficits will resolve.

Even when anaemia from vitamin B_{12} deficiency is present, it is not always the classic macrocytic and megaloblastic anaemia, but can be normocytic or even microcytic. Diagnosing vitamin B_{12} deficiency can be difficult, because serum B_{12} levels do not always reflect tissue B_{12}. As many as 30% of patients with low-normal serum B_{12} level will have anaemia and neurological disease,[42] with normal serum level of vitamin B_{12} defined as 148-590 pmol/L (200-800 ng/L). This finding has prompted a search for more reliable ways to detect vitamin B_{12} deficiency.

Methylmalonic acid and homocysteine are two metabolites that accumulate in vitamin B_{12}–deficient states. Elevated serum methylmalonic acid and homocysteine levels are found in subclinical vitamin B_{12} deficiency, and essentially exclude vitamin B_{12} deficiency when the levels are normal.[44] Some authorities recommend that when vitamin B_{12} deficiency is suspected and the B_{12} level is <258 pmol/L (350 ng/L), methylmalonic acid and homocysteine level should be determined. These tests, however, are not yet generally available.

Another approach for diagnosing vitamin B_{12} deficiency is immunoassay of active B_{12} (also known as holotranscobalamin) that comprises 10-30% of total vitamin B_{12} and is the biologically active form of vitamin B_{12} bound to transcobalamin that can be taken up from the blood into the cells of the body. A locally derived diagnostic cut-off has been quoted as "Active B_{12} ≤28 pmol/L detects vitamin B_{12} deficiency with a specificity of 95%." Determination of active B_{12} concentration can be of value in the presence of a high titre of autoantibodies that interfere with total vitamin B_{12} assay, e.g. high titre of anti–intrinsic factor (IF) antibodies that falsely elevate serum vitamin B_{12} level.

39.8.1.1 Causes

Vitamin B_{12} deficiency more often results from inadequate intestinal absorption than from inadequate intake. Only persons who follow strict dietary restrictions are at risk of an inadequate intake. In a study of Chinese vegetarian women in Hong Kong with a mean age of 81 years,[45] the mean haemoglobin level was lower in vegetarians, the prevalence of anaemia being 30%, with deficiencies in B_{12} and / or iron accounting for 64% of the anaemia, compared with only 30% in non-vegetarians. Serum B_{12} concentration below the reference range occurred in 54% of the vegetarian subjects.

Pernicious anaemia is the classic example of inadequate intestinal absorption of vitamin B_{12}. Patients develop autoimmune antibodies that destroy their gastric parietal cells (GPCs) leaving them without IF. Up to 2% of otherwise healthy older persons may have undiagnosed pernicious anaemia.[46]

Another frequent cause of vitamin B_{12} deficiency in elderly people is food-cobalamin malabsoprtion[47]; an inability to free B_{12} from its binding to dietary proteins due to hypochlorhydria or pancreatic insufficiency, e.g. post-gastrectomy, use of proton pump inhibitors, H_2 receptor antagonists, or even antacids.[48-50] Of patients who had a partial gastrectomy, 10-30% had inadequate absorption of vitamin B_{12}.[51] Inadequate absorption also occurs in patients with small bowel disorders and bacterial overgrowth.[52] Metformin, a commonly prescribed oral hypoglycaemic drug, may also reduce ileal B_{12} absorption due to calcium dependent ileal membrane antagonism.[53] This effect can be corrected by administration of oral calcium supplements.[54] The prevalence of many of these conditions increases with age, and in an elderly person, the cause of B_{12} deficiency may be multifactorial.

The Schilling test, once used to identify the cause of inadequate vitamin B_{12} absorption, is no longer available. Caucasian studies revealed that the anti-IF antibody is detected in up to 70% of patients with pernicious anaemia with a specificity reaching 100% and sensitivity of 50-70%.[55] The anti-GPC antibody is more sensitive but less specific for pernicious anaemia.[55] Nonetheless the relative prevalence of antibodies to GPC and to IF in patients with pernicious anaemia differs between Caucasians and Chinese: among Caucasians the frequency of serum antibodies to GPC and IF is reported to be >90% and 50-70% respectively, whereas among Chinese with pernicious anaemia, serum antibody to IF (73%) occurs slightly more frequently than serum antibody to GPC (65-70%).[56] An upper endoscopy is usually recommended in patients with pernicious anaemia to exclude underlying stomach carcinoma.

39.8.1.2 Treatment

Vitamin B_{12} can be given both parenterally and orally. The traditional treatment of vitamin B_{12} deficiency and pernicious anaemia has been to administer a daily intramuscular injection of 1 mg for a week, then 4-weekly injections of 1 mg, followed by lifelong maintenance injections of 1 mg of cyanocobalamin monthly (or 1 mg of hydroxocobalamin 3-monthy). There is an alternative lower-efficiency transport system of cobalamin absorption that does not require IF or a functioning terminal ileum and oral therapy with 1-2 mg of vitamin B_{12} has been shown to be as effective, and in some ways, may be superior in patients with food cobalamin malabsorption.[57] Oral replacement should be monitored for the desired response since absorption can be variable and may be insufficient in some patients. It may be more practical to initially administer parenteral cobalamin and then to continue with oral cobalamin. An increase in reticulocytosis usually occurs within a few days of starting vitamin B_{12} replacement. Serum potassium levels may fall during therapy for severe cobalamin or folate deficiency and also require replacement.

39.8.2 Folate-deficiency Anaemia

Unlike vitamin B_{12} deficiency, folate deficiency usually develops as a result of inadequate dietary intake. The body stores very little folate, enough to last only 4-6 months. Folate-deficiency anaemia is

usually macrocytic, but can be normocytic in 25% of patients.[18] Red cell folate is frequently believed to be a better indicator of tissue stores of folate although evidence for this is lacking.[58] Overall, serum folate is cheaper and faster to measure than red cell folate, is influenced by fewer analytical variables and provides an assessment of folate status that may be superior to red cell folate.[58] Also, vitamin B_{12} deficiency may falsely lower red cell folate results in the absence of genuine folate deficiency.[58] 90% of patients with folate deficiency will have an elevated homocysteine level[44] that can be helpful in detecting folate deficiency when folate level is low-normal. If methylmalonic acid level is also elevated, vitamin B_{12} deficiency should be considered.[43] It is important to recognise concomitant vitamin B_{12} deficiency because administration of folate in patients with vitamin B_{12} deficiency will worsen the neurological complications. The recommended dietary allowance or adequate intake of folate for elderly persons is 0.4 mg daily, while the tolerable upper intake level is 1 mg daily.

39.9 MDS

MDS is a common haematological malignancy in elderly patients. It represents a heterogeneous group of closely related clonal haematopoietic disorders characterised by a dysplastic hypercellular or hypocellular bone marrow and peripheral blood cytopenias due to ineffective blood cell production.[59,60] The disorder presents with macrocytosis or a dimorphic red cell morphological pattern (a microcytic subpopulation of red cells with the remaining cells being normocytic). Cases of vitamin B_{12}–responsive pancytopenia mimicking MDS have been described.[61]

The median age at which MDS is diagnosed is 60-75 years. Signs and symptoms are non-specific and generally relate to the cytopenias.[62,63] Fatigue, weakness, and malaise are common. A small percentage of patients have splenomegaly, most commonly those with chronic myelomonocytic leukaemia. Hepatomegaly is rare. The features generally used to define MDS include various combinations of blood cytopenias, ineffective haematopoiesis, dyserythropoiesis, dysgranulopoiesis, dysmegakaryopoiesis, and increased blast count. Bone marrow is usually hypercellular but can occasionally be normocellular or even hypocellular. The most important finding in the bone marrow is the dysplastic changes and increased blasts count. Cytogenetic study may reveal characteristic changes and a number of recurrent cytogenetic abnormalities are diagnostic of MDS. Patients can be stratified into different risk groups according to the severity of cytopenia, blast count, and cytogenetic abnormality.

Approximately 5-15% of older adults with unexplained anaemia have MDS. The treatment of MDS in such patients differs to that of younger patients as they are unlikely to be candidates for bone marrow transplantation and do not tolerate intensive cytotoxic treatment. Treatment should be individualised and tailored to the patient's clinical situation and risk group. Supportive care should be offered to patients with low-risk MDS or poor performance status. Demethylating agents are an option for patients with reasonable physical condition and high-risk MDS.

39.10 Unexplained Anaemia

It has been increasingly recognised that approximately one-third of older adults may not have an obvious cause for their anaemia even after extensive evaluation.[11] This anaemia is generally mild with serum haemoglobin of 9-12 g/dL, normocytic and hypoproliferative (low reticulocyte count). The potential underlying mechanisms for this anaemia include low testosterone,[64] occult inflammation,[65] reduced haematopoietic reserve with ageing, inappropriately low serum EPO secretion[66,67] and some so-called 'pre-MDS'.[63]

39.11 Anaemia in Heart Failure

Anaemia occurs commonly in patients with chronic heart failure and is associated with increased risk of hospitalisation and all-cause mortality. Estimates of the prevalence of anaemia in patients with heart failure range from 4-61%.[68] Most studies indicate an increased prevalence of anaemia in heart failure populations with comorbid kidney disease, advanced age, and more severe symptoms. The majority of anaemia seen in heart failure patients is normocytic, often classified as ACD. Chronic kidney disease is a strong predictor of anaemia in heart failure patients.[69] For patients with cardiac cachexia, their level of proinflammatory cytokines including TNF-alpha, IL-1, and IL-6 is usually increased.[70] Inhibition of the renin-angiotensin system with angiotensin-converting enzyme inhibitors or angiotensin receptor blockers is associated with decreased EPO production and reduced haemoglobin level.[71,72] The expanded plasma volume and consequent haemodilution also contributes to the low haematocrit but the red cell mass can be normal.[73] Red cell transfusion can be considered an acute treatment for severe anaemia on an individualised basis only. Treatment with ESA and supplemental iron can be considered in those with moderate-to-severe anaemia and concomitant moderate-to-severe chronic kidney disease.

39.12 Anaemia in Multiple Myeloma

Most patients with myeloma present with anaemia, second only to skeletal lytic lesions. Approximately

70% of patients have anaemia at diagnosis and 97% at some time during the course of the disease.[74] The anaemia is mostly normocytic and normochromic and is due to ACD and shortened red cell survival due to failure of bone marrow compensation. There is also reduced red cell production from impaired iron storage, inadequate EPO response, or overproduction of cytokines that inhibit erythropoiesis.

Depressed red cell production by the bone marrow is the main mechanism causing anaemia in myeloma. Marrow replacement by myeloma cells and cumulative marrow suppression by chemotherapy are the main reasons. Defective production of EPO due to renal impairment or impaired erythroid marrow response is the underlying mechanism of reduced erythropoiesis.

Treatment options for anaemic myeloma patients include either blood transfusion or recombinant human EPO.

39.13 Summary

Anaemia is a common condition in elderly people. Elderly anaemic patients should undergo a standard evaluation for anaemia to find a treatable cause. ACD and iron-deficiency anaemia can both present with low serum iron and can be differentiated using serum ferritin level.

Vitamin B_{12} deficiency, folate deficiency, and gastrointestinal bleeding are other common causes of anaemia in elderly patients. Oral vitamin B_{12} can be used to continue replacement after initial intramuscular administration.

For those with unexplained, mild and asymptomatic anaemia, treatment is not indicated. In those who are symptomatic, options include red cell transfusions or ESA. Bone marrow examination may be considered in those patients who need to be investigated for unexplained anaemia in order to exclude MDS.

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CHAPTER 40

Common Skin Problems

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OBJECTIVE

To learn the common skin conditions encountered in old age including pruritus, scabies, and herpes zoster.

KEY POINTS

Pruritus

- Pruritus can be caused by both dermatological conditions and systemic diseases.
- For conditions without recognisable dermatological causes, further investigations are recommended to look for underlying systemic diseases.
- Treatment should start with advice on general skin care and avoidance of scratching.
- Oral antihistamines are useful in pruritus due to systemic diseases.
- Successful treatment of underlying diseases is often curative for pruritus.

Scabies

- Scabies is common in elderly people, especially among those with dementia, neurological disorders, malnutrition, infectious diseases, haematological malignancy, and immunocompromised states.
- The mode of transmission is by direct skin-to-skin contact with infected individuals.
- Scabies is suspected when there are burrows or when patients have typical symptoms with characteristic skin lesions and distribution. Scabies, however, may present atypically in elderly people.
- Various topical preparations can be used to treat scabies effectively, including benzyl benzoate, permethrin, lindane, malathion, and crotamiton. Oral ivermectin has been used (off-label) for treatment of scabies, especially Norwegian scabies, however its effectiveness and safety have not been fully established.
- Control of scabies outbreak depends on improvement in recognition of scabies, and awareness and prompt treatment of the disease.

Herpes zoster

- Herpes zoster is a common vesicular eruption due to reactivation of latent varicella-zoster viral infection.
- Vesicular lesions can be seen developing in one dermatome. Occasionally, it can involve up to three dermatomes. Patients with underlying diseases such as lymphoma may develop disseminated zoster.
- The commonest dermatomes are those in the thoracic region followed by trigeminal nerve distribution.
- Complications include postherpetic neuralgia, ocular involvement, and cutaneous complications. Complications in the nervous system, heart and lung have also been reported.
- Treatment includes symptom control and specific antiviral agent.
- Prevention is possible with herpes zoster vaccine.

40.1 The Ageing Skin: Structural and Physiological Changes

In elderly people, the dermis is markedly thinned leading to paper-thin skin.[1] The thin dermis layer is relatively avascular and acellular.[2] Typically, there is flattening of the dermoepidermal junction, with, histologically, less villous cytoplasmic projections of the epidermal basal cells into the dermis. These

result in decrease in nutrient transfer and resistance to shearing force. Attenuation of epidermal turnover rate of elderly skin leads to decreased repair rate of the injured skin. Decrease in sensory perception as well as vitamin D synthesis has also been described. In addition, thermoregulation is rendered inefficient due to reduction in sweat glands.

40.2 Pruritus

Both localised and generalised pruritus may be experienced by the older people.[3] The diagnosis is easily made if pathognomonic skin lesions and associated clinical features are present. However, it is often challenging to establish a specific diagnosis among elderly patients with pruritus who do not have skin lesions.

40.2.1 Causes of Pruritus

Pruritus can be caused by both dermatological conditions as well as systemic diseases.[4] Indeed, most, if not all, dermatological ailments associate with a certain degree of pruritus. Pruritus is often worse at night. This is especially so in scabies infestation. Apparently, because of activities and distractions during daytime, elderly patients often report less-severe itching during the day. However, the lack of distraction during sleep leads to overwhelming sensation of pruritus. The scratching of the skin leads to the release of more histamine and more itchiness, thus, leading to the development of an itch-scratch vicious cycle.

The common systemic diseases associated with generalised pruritus are listed in Table 40.1. The sufferers usually have no evidence of cutaneous inflammation and tend to develop generalised pruritus that also affects the scalp and face. Below are the salient features concerning pruritus.

- Although chronic renal failure is a common systemic cause of pruritus among older patients, there is no apparent correlation between severity of pruritus and degree of renal failure.
- Iron-deficiency anaemia is associated with pruritus.[5]

Table 40.1 Systemic diseases associated with pruritus without diagnostic skin lesions (often generalised pruritus affecting the scalp and face)

Chronic renal failure
Hepatobiliary disease
Haematological diseases: polycythaemia rubra vera, Hodgkin's disease, myelomatosis, iron-deficiency anaemia
Endocrine diseases: thyrotoxicosis, hypothyroidism
Drugs, e.g. opiates
Psychosis
AIDS

Abbreviation: AIDS = acquired immunodeficiency syndrome

- Endocrine causes of pruritus are not uncommon. Diabetes mellitus usually presents with localised itching such as pruritus ani or vulva. Thyroid disorders have been shown to be associated with pruritus. Thyrotoxic patients can present with pruritus which responds to treatment of the thyroid diseases. On the other hand, hypothyroidism may be associated with pruritus usually secondary to xerosis.
- Differential diagnoses for generalised pruritus in elderly people include dermatographism, scabies, and asteatotic eczema.
- Some elderly patients have pruritus without apparent cause. Hence, there is a group of patients having idiopathic pruritus. Psychological factors may contribute to this entity.[6]

40.2.2 Investigations for Pruritus

As usual, complete history and physical examination are mandatory in the diagnosis of pruritus.[7] For practical purposes, patients with pruritus are divided into one of the following three categories:

1. Itching due to a readily recognisable dermatological disease;
2. Itching due to a primary dermatological disease with the main diagnostic lesions masked by secondary changes of eczematisations and excoriations; and
3. Itching with no detectable primary lesions or secondary changes

With careful history taking and physical examination, patients in categories 1 and 2 can be identified. For category 3, some authors suggest the empirical use of an intensive 2-week therapy for xerosis. If symptoms persist, a minimal outpatient workup is recommended. Tests should include renal function test, liver function test, thyroid function test, complete and differential blood counts, and chest X-ray.[7] Further investigations should be carried out, as indicated, in individual patients. Occasionally, judicious psychiatric assessment may be needed in selected patients with no apparent causes of pruritus despite exhaustive investigations.

40.2.3 Treatment of Pruritus

Treatment should start with advice about general skin care to avoid xerosis. Patients should be educated about avoiding scratching as far as possible as it can cause more itching through release of histamine. The use of topical ice therapy or emollient can be helpful. Topical steroids are useful for specific dermatological conditions such as eczema. However, in pruritus secondary to systemic diseases, steroids are usually ineffective. Oral antihistamines (anti–H_1-receptor antagonists) are particularly useful; chlorpheniramine (Piriton) is commonly used in Hong Kong. On the other hand, cyproheptadine is useful as it has both

antihistamine and antiserotonin properties. Low-dose doxepin, a tricyclic antidepressant drug with a strong anti–H_1 histamine receptor effect, is also useful in treating pruritus.

Obviously, if the systemic cause of the pruritus can be identified, successful treatment of the underlying diseases is often curative for itching. For instance, treatment of iron deficiency with iron replacement and the therapeutic interventions for neoplastic diseases such as polycythaemia rubra vera can lead to cessation of pruritus.[8] Narrow-band ultraviolet B can also be effective for chronic pruritus of unknown cause.

40.3 Scabies

Among older people, scabies is usually associated with conditions such as dementia, neurological or mental disorders, malnutrition, infectious diseases, haematological malignancy, and immunocompromised states. Unlike other infectious diseases, scabies is not a statutorily notifiable disease in Hong Kong, though any suspected institutional outbreak of scabies can be reported to the Central Notification Office of the Centre for Health Protection for arrangement of investigation and control, as appropriate.[9] According to the Hong Kong Department of Health, the proportion of new cases of scabies in dermatology clinics was between 2% and 3% of the total number of dermatology cases in 2008 to 2009.[10] These figures might not reflect the true incidence as many older patients with scabies are treated in other settings including Community Geriatric Assessment Service, geriatric clinics, or during hospitalisation. On the other hand, the prevalence of scabies in local residential care homes has been reported to be 5.8%.[11]

The impact of scabies in the older population is often underestimated. While infestation with the scabies mite is not a life-threatening condition, the persistent generalised itchiness often results in much debility and suffering in elderly patients, and may even lead to depressive illnesses. Scabies is common in elderly populations and effective treatment is available.

40.3.1 Biology and Life Cycle of *Sarcoptes scabiei*

Scabies is caused by the mite *Sarcoptes scabiei* (Figure 40.1). Infestation starts as a fertilised female mite arrives on the skin surface and excavates a burrow in the stratum corneum. Eggs and faecal pellets laid down in the burrows then act as allergens which lead to itching. The larvae hatch after 50-72 hours and start to make new burrows. After maturation, they mate and the cycle is repeated in 10-17 days. The mites can survive for up to 2 months.

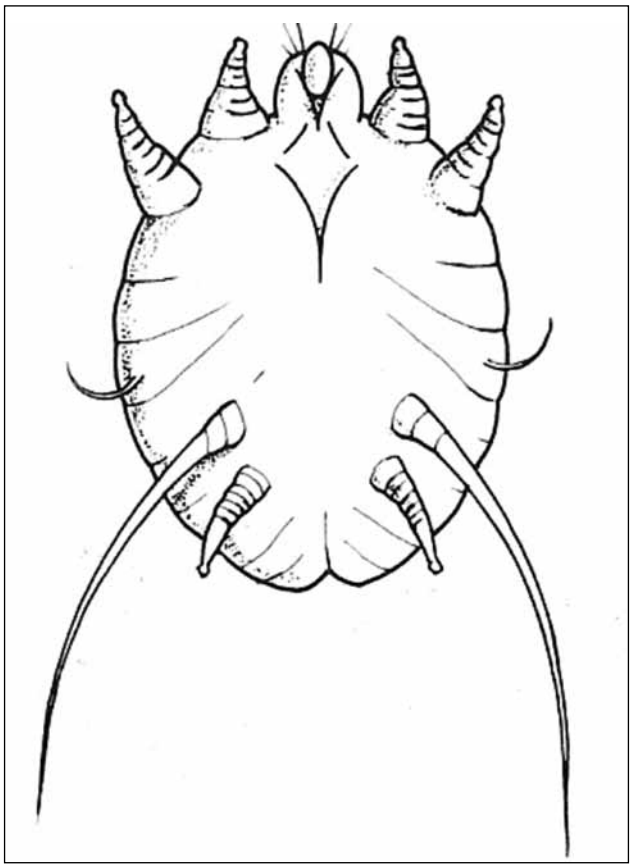

Figure 40.1 Sarcoptes scabiei

40.3.2 Mode of Transmission

The mode of transmission is by direct skin-to-skin contact. The mites can spread rapidly between residents of the same home, nursing home, or hospitals. Scabies tends to spread in homes and institutions where a high frequency of intimate personal contact or sharing of inanimate objects occurs. It confirms that fomite transmission is a major factor in household and nosocomial transmission of scabies.

40.3.3 Clinical Features

There are several clinical characteristics that help to identify scabies. First, generalised pruritus is present. The itchiness is felt even in areas with no apparent skin lesions while in many skin conditions such as eczema, the pruritus only occurs in areas with skin lesions. The pruritus usually does not occur on the face except in patients with Norwegian scabies (see below), and the itchiness is worse at night, when the patient is warm in bed.

The classical distribution of the rash includes the hands and fingers, anterior parts of the wrists, ankles, elbows, anterior axillary folds, buttocks, surrounding

the areolar region in females, and external genitalia in males. Erythematous papules and nodules in the same areas are also seen. Overall, the most common sites are the hands and wrists while the scalp, face, and neck are usually spared, except in patients with Norwegian scabies.

Primary lesions such as burrows are quite diagnostic and these are usually tunnels in the stratum corneum created by the mites. However, it is not unusual not to find burrows in many patients. One should not hesitate to diagnose scabies when other compatible clinical features, excluding burrows, are present. Vesicles are also primary lesions that are associated with scabies. Secondary lesions can result from infestation or due to scratching. All forms of eczematous reactions can occur including urticaria, eczematous papules, and scratch marks. Pustules, impetigo, furuncles, and cellulitis are features of secondary bacterial infection.

Scabies, like other diseases, may present atypically in elderly people. Elderly patients may have few cutaneous lesions but experience severe itching. One should beware of atypical forms of scabies among institutionalised elders: diffuse, nonpruritic, erythematous maculopapular lesions, discrete crusted lesions with surrounding erythematous papules, distributed on the patient's back / trunk and absent over peripheries and without any finger web lesions or burrows.[12] Elders in hospitals and residential care homes might be physically restrained, which may hinder proper examination of the skin condition. A high index of suspicion of scabies should be raised if an elder becomes restless and is seen wriggling all the time, especially those who cannot communicate verbally. Scabies can mimic various types of skin diseases such as eczema, bullous disease, and even erythroderma. Physicians should, therefore, include scabies in the differential diagnosis of eczema, drug eruptions, and fungal skin infections in elderly patients with skin lesions, and, more importantly, the presence of one skin diagnosis does not exclude the coexistence of scabies. Generalised lymphadenopathy may also be present in some severe cases.

The extent of the skin lesions varies among patients. The most severe one is Norwegian scabies which was first described in 1848 in patients with Hansen's disease who had overwhelming scabies infestation.[12,13] This condition is also called crusted scabies, as the lesions tend to involve hands and feet with crusting (Plate 40.1) instead of the classical inflammatory papules and vesicles. Scales and thick crusts can be found all over the body. In contrast to non-Norwegian scabies in which there are only 5-15 female mites in an infested host, the number of mites can be hundreds or even millions in those with Norwegian scabies. In this condition, the face and head may be involved with facial desquamation and shedding of the hair (Plate 40.2). Nail dystrophy is also a common feature in the fingers and toes. Pruritus varies from severe to absent. Immunocompromised patients are particularly prone to develop Norwegian scabies as they cannot develop an allergic cutaneous reaction which leads to pruritus and scratching; in contrast, scratching may help to keep the mites at a low number in patients with non-Norwegian scabies. In addition, frail elderly patients who have poor awareness and upper limb function such as those having stroke and dementia are at risk of developing Norwegian scabies because of their inability to scratch.

40.3.4 Establishing Diagnosis

Scabies is suspected whenever there are burrows or when patients have typical symptoms with characteristic lesions and distribution. Identification of burrows can be facilitated by touching the lesions with a dark fountain or felt tip pen. The ink will be absorbed by the burrow and highlighted as a dark line. Definite diagnosis of scabies requires the demonstration of mites, eggs, egg casings or faeces in the burrows or vesicles. To obtain diagnostic materials, a commonly used method is to scrape with a surgical blade and transfer the material to a microscope slide for examination in mineral oil or potassium hydroxide wet mount. However, the sensitivity of skin scrapping for diagnosis of scabies is only 46%.[14] Other methods including dermoscopy and adhesive tape test have also been described.[15]

As such, the dermatological investigation is not satisfactory. In practice, even in older patients with widespread excoriations, only few mites are present and these may be difficult, if not impossible, to find. Thus, treatment is often based on clinical findings. Delayed diagnosis and treatment could lead to spreading and persistence of scabies.

40.3.5 Treatment

40.3.5.1 Topical Preparations

The topical preparations for scabies treatment include benzyl benzoate, crotamiton, lindane, malathion, and permethrin. According to the 2007 Cochrane review, permethrin is the most effective topical treatment for scabies.[16] All the preparations are available in Hong Kong. Permethrin, lindane, and malathion have the advantage of needing a single application in most cases. Benzyl benzoate needs to be applied twice in 24 hours, while crotamiton needs to be applied once daily for 5 consecutive days for adequate effectiveness.

The usual recommendation is to apply the topical scabicide to the skin surface below the neck. The scabicide is allowed to dry and the same clothes can

be worn. On the evening of the second day, a hot bath is taken and clean clothes should be worn.

The traditional advice of taking a hot bath before application of a topical scabicide is considered obsolete as this may actually increase absorption of the drugs into the bloodstream leading to reduction in the effectiveness of the scabicide as it is removed quickly from the skin. In addition, it may increase the systemic toxicity of the scabicide.

Occasionally, in older persons who are immunocompromised with Norwegian scabies or treatment failure, application of the scabicide to the scalp, neck, face, and ears may be needed. The general principle is to avoid applying the topical preparations on mucous membranes. It is important to apply the scabicide to every part of the body, not just where the rash is present. Judicious skin preparation should be done to improve the success of topical treatment. Nails should be trimmed and thick crust loosened mechanically or with keratolytic agents such as half-strength Whitfield ointment. Many specialists recommend retreatment after 1-2 weeks to ensure all the mites are killed.

Benzyl Benzoate
It is commonly used in Hong Kong with a cure rate of 86-96%.[17] It is a safe, cheap, and effective scabicide for classical scabies. However, it has been replaced by permethrin in western countries due to high treatment failure rate and the need for two applications for each benzyl benzoate treatment. The compound itself is an irritant, and repeated application may cause dermatitis, especially in the genital area and on the face.

Permethrin
Permethrin has been shown to be more effective than lindane and crotamiton with a cure rate of >90%.[18] However, head-to-head comparison between permethrin and benzyl benzoate has not been performed. The drug has a favourable safety profile and is associated with insignificant systemic absorption.[19] Permethrin is now considered to be the first-line treatment in many western countries.

Lindane (Gamma Benzene Hexachloride)
This preparation is available in Hong Kong as well as in the US. However, it is no longer available in the UK and Australian markets. Lindane is a central nervous system stimulant and it leads to seizures and death in the scabies mite. Around 10% of lindane is absorbed via intact skin. It can accumulate in fat and bind to the brain tissue. In addition to minor reactions such as rash, papules, and diarrhoea after lindane application, severe adverse effects such as aplastic anaemia and increased risk of neurotoxicity with numbness, anxiety, tremors, or convulsions have been reported.[20] Hence, it is recommended to be avoided in patients with central nervous system disorders including epilepsy and those with extensive skin excoriations which may lead to significant systemic absorption of the drug. One application is usually adequate for successful treatment; mucous membranes should be avoided.

Malathion
It is an effective treatment and is recommended in the UK for treatment of scabies. It may lead to irritation of the skin but major side-effects are rare. This preparation is commercially available in Hong Kong.

Crotamiton
Reported cure rates with 2-5 consecutive days of single crotamiton application are between 50% and 100% with good tolerability.[21] It also has antipruritic properties. Because of its possible lower efficacy as compared to other scabicides, and the need for continuous application over a period of time, crotamiton is not recommended as a first-line agent for scabies treatment; however, it can be used as a complimentary treatment as well as follow-up treatment for post-scabietic pruritus.

40.3.5.2 Oral Treatment

Ivermectin is the only oral drug for scabies treatment.[22] It has not been licensed by the US Food and Drug Administration (FDA) for human scabies treatment. It has been used as 'off-label' treatment for patients who fail to respond to topical treatment or those who cannot comply with topical therapy. For crusted scabies, three to seven doses of the drug are recommended alongside a topical scabicide. However, there has been concern about its safety, especially in older persons, as increased risk of death due to neurotoxicity in a group of elderly patients with scabies in a long-term care facility has been reported.[23] More studies about its safety profile are needed before it can be widely used in the older populations. Since safe and effective topical scabicides are available, oral ivermectin is not recommended as the treatment of choice for scabies in older people at this moment.

40.3.5.3 Persistent Itchiness and Rash

Scabies infestation usually becomes non-infectious within a day after effective treatment has been given. However, itchiness and rash can persist for weeks to months. Itchiness usually decreases within 1-2 days after treatment, and then, gradually reduces during the following 1-2 weeks. Doctors should avoid unnecessary retreatment as it may lead to side-effects such as contact dermatitis, or even systemic toxicity (e.g. with reapplication of lindane).

Adjunctive treatment with antihistamines and calamine lotion can relieve pruritus. Judicious use of topical steroids helps to alleviate the itchiness that lingers after treatment with scabicides. Occasionally, antibiotics are needed if bacterial superinfection is present. Potent topical steroids under occlusion are recommended for scabies nodules that may persist for months if left untreated.

40.3.5.4 Control of Scabies Outbreak

As in most diseases, the principle of 'prevention is better than cure' should be upheld in scabies management. In institutionalised settings, effective control of the epidemic is achieved by improvement in scabies recognition, awareness, and prompt treatment of the disease.[24] When an infested case is identified in the institution, laborious screening for scabies is needed in all the older residents, staff, and family members of the index case.[25] If there is an outbreak, all the older residents, staff, and family members of the index cases should receive prophylactic treatment for scabies.

40.4 Herpes Zoster

Herpes zoster or shingles is a common vesicular eruption due to reactivation of latent varicella-zoster virus in the dorsal sensory ganglia. The causative agent is a DNA virus belonging to the herpes group of virus.

40.4.1 Epidemiology

Herpes zoster occurs during the lifetime of 10-20% of all people.[26] In one study, the incidence increased 10-fold in elderly people aged 80-89 years compared with children younger than 10 years.[27] The risk of postherpetic neuralgia (PHN) also increases with age.[28] To date, no epidemiological data for herpes zoster in Hong Kong are available. Predisposing conditions are summarised in Table 40.2.

40.4.2 Clinical Features

40.4.2.1 Latency Phase

Following a primary varicella infection in childhood, the virus may remain dormant in the dorsal sensory root ganglia. Reactivation can occur when there is decreased host immunity and increased physical stress.

Table 40.2 Conditions which predispose to the development of herpes zoster

Advanced age
Malignancy
Immunosuppression
AIDS

Abbreviation: AIDS = acquired immunodeficiency syndrome

40.4.2.2 Prodromal Phase

The prodromal period is usually 1 to 4 days before development of skin lesions but can be unusually long.[29] There may be pain in the affected dermatome, described as burning, throbbing, stabbing, or electric shock like. Dysesthesia at the site of future lesion can also occur; this may include increased sensitivity to touch (paraesthesia), increased response to external stimuli (hyperaesthesia), or pain triggered by trivial stimuli (allodynia). The longer the duration of pain before development of rash, the more likely other causes of rash will be explored, resulting in many expensive and invasive investigations as well as treatments.

40.4.2.3 Active Phase

Vesicular lesions can be seen developing in one dermatome of a single sensory ganglion and the lesions are arranged in a herpetiform manner (like a bunch of grapes). However, adjacent dermatomes may be involved in 20% of the cases. Some patients may have a few scattered lesions at some distance from the involved dermatome. By day 3 to 5, the vesicles become pustular and, sometimes, haemorrhagic. New lesions continue to appear within a few days. They finally form crusts in the second week and resolve within 1 month. Patients may complain of pain and dysaesthesia at the site of eruption. The commonest dermatomes are those in the thoracic region (>50%) followed by those affecting the trigeminal nerve distribution (up to 20%).[30] Among the three trigeminal nerves, the ophthalmic branch is involved more commonly. The lumbar and cervical dermatomes are less frequently affected by the zoster. Immunocompromised patients are prone to develop disseminated zoster with generalised vesicular lesions affecting several dermatomes and, frequently, crossing the midline, rendering the diagnosis more difficult.[31] During the eruption phase, pain may be associated with some burning and itching sensation. Systemic symptoms including fever, headache, and / or malaise may be present in less than one-fifth of the patients.

40.4.3 Complications of Herpes Zoster

40.4.3.1 PHN

It is defined as pain along the course of a nerve after the rash of herpes zoster has healed, usually in 1 month. Some authors use 2, 3, or 6 months to define this condition.[32] It is the most common complication of herpes zoster, affecting nearly 74% of patients of >60 years when using the definition of 30 days of pain.[33] Persistent pain is more common in trigeminal involvement with an incidence of 15% in reported cases.

40.4.3.2 Ocular Complications

Ophthalmic zoster, involving the ophthalmic branch of the trigeminal nerve, is found in 20-70% of patients

with herpes zoster. Different pathologies can occur including keratitis, iridocylitis, optic neuritis, retrobulbar neuritis, secondary glaucoma, and panophthalmitis.[34]

40.4.3.3 Cutaneous Complications

These include hyperpigmentation of the skin, bacterial superinfection, scarring, and cutaneous dissemination in immunocompromised patients.

40.4.3.4 Other Complications

Myelitis, encephalitis, and meningomyelitis have been reported in patients with herpes zoster. Motor zoster with weakness or paralysis can occur in zoster involving the anterior horn cells in the same segment of the spinal cord.[35] Lumbosacral herpes zoster can present with neurogenic bladder and retention of urine. Pneumonitis, oesophagitis, enterocolitis, myocarditis, pancreatitis, and hepatitis can also complicate herpes zoster. In fact, pneumonitis, albeit uncommon, is the leading cause of mortality in herpes zoster patients. Cerebrovascular complications have also been reported, especially in immunocompromised patients, presenting as contralateral hemiparesis (i.e. paralysis on the side opposite to the site of herpes zoster lesions). Thrombosis and vasculitis affecting arteries, arterioles, and venules have also been observed. Last but not the least, Ramsay Hunt syndrome, a lower motor neuron lesion of the facial nerve with deafness and vertigo, may also develop.

40.4.4 Diagnosis and Treatment

40.4.4.1 Diagnosis of Herpes Zoster

The diagnosis of herpes zoster is, usually, easily made on the basis of history, morphology, and distribution of the lesions. In some situations, however, confirmation of infection may be needed. The most sensitive and specific test is the polymerase chain reaction for viral DNA.[36] This can be performed on fluid from skin lesions, blood, plasma, and cerebrospinal fluid. Direct florescent antibody testing can be performed on scrapings from active vesicular lesions. Viral culture is not recommended and serological testing is usually not useful for diagnosing acute infections. Viral antigen detection is rapid and the most commonly used test in clinical practice if diagnosis is in doubt or in disseminated cases.

40.4.4.2 Analgesics and Antipruritic Agents

Pain should be controlled by adequate analgesics; sometimes, opiates are needed. Itchiness can be relieved by antipruritic medications to prevent scratching. Gentle cleansing and drying should be adequate in most cases with skin lesions.

40.4.4.3 Antiviral Agents

The major justification for antiviral treatment of herpes zoster in immunocompetent patients is to shorten and reduce the clinical course and, probably, decrease the complications of herpes zoster, in particular, PHN.[37,38] It is especially important in elderly patients who have higher rates of PHN. Optimal duration of treatment is 7 days and therapy should be commenced within 72 hours of the onset of lesions.[39] Evidence so far suggests that acyclovir, valacyclovir, and famciclovir can limit the duration of zoster-associated pain. In addition, antiviral agents can hasten the healing of cutaneous lesions by a mean of 2 days. The choices of antiviral drugs are determined by cost and dosing schedules rather than efficacy. Regimens recommended for those with normal renal function are: acyclovir 800 mg orally 5x/day for 7 days; famciclovir 500 mg orally 3x/day for 7 days and; valacyclovir 1000 mg orally 3x/day for 7 days. Doses have to be adjusted in patients with renal insufficiency.

Elderly patients are at higher risk of PHN, and should be offered antiviral agents, if possible. Patients with ophthalmic zoster should be treated with antiviral agents as it can reduce the chances of keratitis. For immunocompromised patients, intravenous acyclovir is recommended to prevent cutaneous dissemination of the varicella virus.

40.4.4.4 The Use of Corticosteroids in Acute Herpes Zoster

The use of steroids in uncomplicated herpes zoster is controversial. Oral corticosteroids have been shown to reduce pain and provide an improvement in quality-of-life measures when combined with acyclovir rather than using acyclovir alone.[40] Oral steroids have not been studied with valacyclovir or famciclovir, so the benefit is unknown. The use of oral steroids has no effect on the development or duration of PHN. Because of the potential adverse effects of and contraindications to corticosteroid use, current expert opinion does not recommend the routine use of steroids in herpes zoster patients.

40.4.4.5 Zoster Vaccine in Elderly People

As the risk of developing herpes zoster increases significantly as one ages, the US FDA approved a zoster vaccine in 2006 for prevention of herpes zoster among those aged ≥60 years. It was based on a large-scale randomised control trial called Shingles Prevention Study involving more than 38 000 adults aged ≥60 years.[41] The study showed that the zoster vaccine could reduce the incidence of herpes zoster by half and PHN by two-thirds. The zoster vaccine is a live, attenuated vaccine containing the same virus as the varicella zoster, but at a higher potency. In the US, the Advisory Committee on Immunization Practices recommends zoster vaccination for elderly people aged ≥60 years, regardless of their history of

zoster. Since 2013, the zoster vaccine is available in Hong Kong for use in the private sector, and elderly people can receive the vaccine at their own cost.

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CHAPTER 41

Diabetes Mellitus

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OBJECTIVE

To learn the diagnosis, recognise the altered presentation, and perform accurate assessment of elderly diabetic patients, and to devise an individualised management plan in terms of food planning, physical activities, drug administration, and prioritising of metabolic targets. To appreciate the importance of identifying microvascular complications and hypoglycaemia in elderly diabetics.

KEY POINTS

- The prevalence of diabetes mellitus is >20% for elderly people aged >65 years in Hong Kong.
- Presenting symptoms of hyperglycaemia may be atypical in elderly people.
- Food planning, physical activity, and drugs are the mainstay of treatment.
- Self-monitoring of glucose at home by the patient or caregiver using a glucometer should be encouraged.
- Assessments should be performed before formulating a management plan for each individual. These include assessment of function, vascular risk, metabolic parameters, presence or absence of complications, and self-care ability.
- Although guidelines for ideal metabolic targeting for diabetics based on a single disease model are appropriate for younger diabetic patients, they should be modified in elderly diabetic patients to take account of comorbidities, dependency, frailty, and susceptibility to hypoglycaemia.
- Sulphonylureas, biguanides, meglitinides, dipeptidyl peptidase 4 inhibitors, thiazolidinediones, α-glucosidase inhibitors, sodium glucose cotransporter 2 inhibitors, and insulin are the main drugs of choice.
- Diabetic foot is an important complication. Proper footwear, dedicated foot care, and regular examination are important preventive measures.
- Retinopathy and nephropathy should be regularly and routinely screened for. Early and timely interventions are essential.
- Hypoglycaemia in elderly diabetic patients presents with fewer sympathetic and more neuroglycopaenic symptoms.
- Referral to a hospital specialist is recommended for uncontrolled metabolic state, presence of microvascular or macrovascular complications, or multiple problems that require multidisciplinary intervention.

41.1 Introduction

One in 10 people in Hong Kong have diabetes mellitus; the prevalence ranges from 2% in people aged <35 years to >20% in those >65 years.[1] The increased prevalence of diabetes mellitus with increasing age is related to lifestyle factors, obesity, physical inactivity, and ageing. Diabetes mellitus is the leading cause of kidney failure, blindness, leg amputations, heart disease, and stroke. Diabetic patients often have multiple risk factors such as hypertension, dyslipidaemia, and albuminuria; all of which increase the risk of developing complications. The optimal control of blood glucose, blood pressure, and hyperlipidaemia in diabetic patients has been proven to reduce complication rates in randomised controlled trials and is cost-effective.

41.2 Definition, Diagnosis, and Risk Factors

Diabetes mellitus is a metabolic disorder of multiple aetiologies characterised by chronic hyperglycaemia with disturbed carbohydrate, protein, and fat metabolism resulting from defects in insulin secretion, insulin action, or both. Cardinal symptoms include polyuria, polydipsia, and unexplained weight loss with confirmation by abnormal blood glucose levels

(Table 41.1).

The oral glucose tolerance test can be inconvenient and impractical. The American Diabetes Association[2] has recommended glycated haemoglobin (HbA1c), which is an estimate of mean plasma glucose levels over the previous 3 to 4 months, as a diagnostic test for type 2 diabetes mellitus with a cut-off level of 6.5%. The presence of comorbid diseases such as anaemia, chronic renal failure and rheumatoid arthritis, however, can alter HbA1c value and change the ability of HbA1c to diagnose diabetes.[3] A study in Singapore showed that HbA1c has a low sensitivity to diagnose diabetes in older Asian subjects.[4]

41.2.1 Clinical Features of Diabetes Mellitus in Elderly People

It should be noted that due to the high renal threshold for glucose, the symptoms of polydipsia and polyuria may not be present in an elderly diabetic patient. Elderly diabetic patients may present with the "Is" of geriatrics as listed in Table 41.2.[5]

41.2.2 Risk Factors for Diabetics

When any of the following risk factors are present, doctors should maintain increased vigilance and consider screening patients for glucose intolerance and other cardiovascular risk factors[1]:

- family history (first-degree relatives) of diabetics;
- overweight (body mass index of ≥23 kg/m^2);
- dyslipidaemia (especially with a high level of triglycerides);
- hypertension;
- history of gestational diabetes; and
- age ≥45 years

Other predisposing factors shown below may also contribute to the development of diabetes in elderly people.[5]

- Decreased physical activity: associated with increased insulin resistance at postreceptor levels, an effect independent of weight or body composition
- Drugs: use of steroids, thiazide diuretics, tricyclic antidepressants, and risperidone
- Age-related decreased insulin secretion: lean elderly people have profoundly impaired pancreatic insulin secretion whereas obese ones have relatively preserved glucose-induced insulin secretion

41.3 Lifestyle Management

Effective management of type 2 diabetes in elderly people cannot be achieved without proper attention to lifestyle management. All elderly diabetic patients should be offered lifestyle interventions. Food planning, physical activity, and drug therapies are partners in the management to achieve good metabolic control, namely glucose, lipid, and blood pressure control. Multidisciplinary lifestyle intervention programmes should be considered. Educational information produced by a group of diabetologists in Hong Kong is available for patients (www.diabetes-hk.org/).

Below is the rationale for quality care of older diabetics.[5]

- Early diagnosis may prevent progression of undetected complications.
- Better glucose control may reduce complications.
- Better screening for cataract and maculopathy

Table 41.1 Diagnosis of diabetes based on venous plasma glucose*

Medical reason	Fasting glucose (mmol/L)		OGTT glucose (2 hours post oral glucose) [mmol/L]		Random glucose (mmol/L)
Diabetes mellitus	≥7.0	**or**	≥11.1	**or**	≥11.1
Impaired glucose tolerance			≥7.8 but <11.1		-
Impaired fasting glycaemia	5.6-6.9				-

Abbreviation: OGTT = oral glucose tolerance test

* For epidemiological or population screening purposes, the fasting or OGTT results may be used alone. For clinical purposes, the diagnosis of diabetes should always be confirmed by repeating the test on another day unless there is unequivocal hyperglycaemia with acute metabolic decompensation or obvious symptoms

Table 41.2 Hyperglycaemic consequences and the "Is" of geriatrics[5]

Hyperglycaemic consequence	The "Is" of geriatrics / geriatric syndromes
Osmotic diuresis	Incontinence, insomnia
Fluctuating refraction	Impaired vision and instability / falls
Increased red cell deformability and platelet adhesiveness	Infarction of brain and heart, intermittent claudication
Impaired immunity	Recurrent infections
Impaired repair	Poor wound healing
Alteration in mentation	Impairment of cognition and poor compliance
Decreased pain threshold	Increased painful symptoms
Hyperosmolar non-ketotic hyperglycaemia	Dehydration, delirium

will reduce the number of elderly people with blindness.
- Better management and prevention of peripheral vascular disease and foot problems will reduce complication rates.
- Reduce escalating costs and use of health service resources.

41.3.1 Food Planning

Healthy eating has beneficial effects on weight, metabolic control, and well-being. Caloric restriction in those who are overweight with diabetes is associated with improved glycaemic control and blood pressure control. Below are the essential points although referral to a dietitian is required for detailed meal planning.
- 55-65% of total daily energy intake should come from complex carbohydrates.
- 25-30% of total daily energy intake should come from fat. Less than one-third of this should come from saturated fats.
- 15-20% of total daily energy intake should come from protein.
- Distribution of food intake should be as even as possible throughout the day for patients on oral hypoglycaemic agents or insulin.
- Alcohol intake should be restricted, especially in obese, hypertensive, or hypertriglyceridaemic patients. Alcohol may cause hypoglycaemia in patients prescribed sulphonylurea or insulin. The blood glucose lowering effect of alcohol is greater if alcohol is combined with exercise.
- Salt intake should be restricted, especially in hypertensive patients.

Undernutrition is quite common in elderly patients with type 2 diabetes, especially those living in residential care homes, and should be avoided. Malnutrition is associated with pressure ulcers and increased mortality.[6] In this subgroup, one should carefully evaluate nutritional status and recommend appropriate caloric consumption. Elderly subjects also have more difficulties with acquisition, preparation, and ingestion or swallowing of food, and this again can affect their nutritional status and increase the risk of hypoglycaemic episodes. It is therefore prudent to ensure a support system that can provide appropriate meals in a timely manner.[5]

41.3.2 Physical Activity

Physical activity plays an important role in the management of diabetes. Exercise improves insulin sensitivity and thus improves glycaemic control. It helps in weight reduction, blood pressure control, and improvement of dyslipidaemia. In older people, exercise is associated with benefits of improving balance, reduced fall risk, increased muscle strength, and enhanced quality of life.

In general, diabetic patients should perform 30 minutes of moderate physical activity at least 5 times per week. This includes brisk walking, tai chi, or cycling. More vigorous activities such as swimming, jogging, or dancing should also be encouraged. Older people should continue to be as active as their health and functional status allow. A low-intensity home-based exercise programme may be suggested to maintain mobility and physical strength in more frail patients.

It should be noted that adjustment of insulin or oral hypoglycaemic agents is needed to avoid hypoglycaemia, especially if physical activity is sudden or vigorous. Injecting insulin into exercising areas increases its absorption and the risk of hypoglycaemia and should thus be avoided if physical activity is anticipated. High temperature also increases insulin absorption.

It is also important to bear in mind the risks of exercise for diabetic patients in special circumstances.
- Hyperglycaemia may occur after very strenuous exercise.
- Hyperglycaemia and ketosis may occur in insulin-deficient patients.
- Precipitation or exacerbation of cardiovascular disease, e.g. angina pectoris
- Worsening of long-term complications of diabetes:
  - Proliferative retinopathy, with consequent vitreous haemorrhage / retinal detachment
  - Nephropathy, with consequent increased proteinuria
  - Peripheral neuropathy, with consequent soft tissue and joint injuries
  - Autonomic neuropathy, with consequent decreased cardiovascular response to exercise, decreased maximum aerobic capacity, and postural hypotension

Advice about physical activity should also take account of other coexistent medical conditions that may limit ability to exercise, e.g. osteoarthritis.

41.3.3 Smoking Cessation

It should be emphasised that it is never too late to stop smoking (Table 41.3).

41.3.4 Self-monitoring of Glucose Levels

Home blood glucose monitoring is recommended, and should be encouraged to be performed either by the patient or caregivers. Testing can be performed before each meal, 2 hours after meal, or at bedtime. Extra tests should be performed during illness, prior to strenuous activity, post-activity, and on commencement of enteral feeding or drugs that may affect glucose level such as corticosteroids. Hyperglycaemia or hypoglycaemia in those with

dementia may lead to altered mental performance and consequent confusional state or delirium. Where indicated, blood glucose monitoring should be undertaken by caregivers taking into account special individual circumstances.

Urine glucose testing has its limitations in elderly diabetic patients as their renal threshold may be elevated giving a false sense of good control. Urine ketone testing should be performed during illness or when blood glucose level exceeds 20 mmol/L.

41.4 Setting Treatment Objectives for Elderly Diabetic Patients

It is important to note the following special characteristics of elderly diabetic patients[5]:

- high levels of comorbidities;
- age-related impairment of functional ability;
- increased vulnerability to hypoglycaemia;
- overlapping and often limited medical follow-up by primary care physicians, diabetic specialists, and geriatricians; and
- a different management system that involves spouses and informal caregivers to a great extent

They represent a population of diverse heterogeneity, and most trials have not been designed to evaluate patients aged ≥75 years with poor general health. Treatment goals should therefore be individualised and take account of functional status, cognitive status, comorbidity especially the presence of established cardiovascular disease, presence of microvascular complications, vulnerability to hypoglycaemia, and life expectancy. Glycaemic targets should be individualised and based on the benefit-to-risk ratio. The five-step approach for prioritising diabetes care in elderly people include[5]:

- functional assessment including cognitive testing and screening for depression;
- vascular risk assessment with advice on lifestyle modifications and vascular prophylaxis;
- metabolic targeting (individualised): a single disease model versus frailty model;
- consider specific interventions for diabetes-related complications; and
- assess suitability for self-care versus caregiver assistance

The recommended HbA1c target for elderly diabetic patients in various international guidelines are outlined in Table 41.4.[7-9] In general, a strict glycaemic goal of ≤7%,[10,11] or 7-7.5%[7] is appropriate for elderly people who are independent and without major comorbidities to minimise vascular complications. A more lenient HbA1c target of up to 8-8.5%[9] is advisable for those who are frail, demented, or have a life expectancy of <5 years, as avoidance of hypoglycaemia is of paramount importance, even though long-term benefits of intensive glycaemic control may not appear for 6-8 years. Data have suggested a U-shaped association between HbA1c and mortality where HbA1c of about 7.5% had the lowest hazard ratio, supporting a slightly higher targeted goal in the elderly population.[12] Raising HbA1c goal to >9% is not recommended as the risk of developing acute hyperglycaemic complications will increase. In addition, studies suggest that despite the higher HbA1c level, episodes of hypoglycaemia may still be frequent.[9]

41.5 Pharmacotherapy

41.5.1 Sulphonylureas

The mechanisms of action of sulphonylureas are to increase pancreatic secretion of insulin, decrease

Table 41.3 What smoking cessation means

Duration of not smoking	Benefit
1 Year	Risk of death from heart attack decreases to half that of a lifelong smoker's risk
5 Years	Risk of death from lung cancer decreases to half that of a lifelong smoker's risk Risk of stroke same as non-smoker
10 Years	Risk of death from lung cancer almost same as non-smoker Risk of other cancers, e.g. mouth, larynx, oesophagus, bladder, kidney, and pancreas decreases

Table 41.4 Recommended HbA1c targets for elderly diabetic patients in various international guidelines7-9

Patient characteristics	HbA1c		
	International Diabetes Federation (2013)[7]	European Diabetes Working Party (2011)[8]	American Geriatrics Society (2012)[9]
Independent relatively healthy elderly patient	7.0-7.5%	7-7.5% (fasting glucose 6.5-7.5 mmol/L)	≤7.5%
Functionally dependent (frail, dementia) or with life expectancy of <5 years	7.0-8.5%	7.6-8.5% (fasting glucose 7.6-9.0 mmol/L)	8-8.5%

Abbreviation: HbA1c = glycated haemoglobin

hepatic glucose output, and reverse postreceptor effect at adipose tissue and muscle. The characteristics of specific drugs in this group are summarised in Table 41.5.

Sulphonylureas are effective oral hypoglycaemic agents and are capable of lowering HbA1c by 1.5-2%. They are inexpensive, widely available, and can be used as first-line therapy. The risk of hypoglycaemia may be problematic for older patients, however, and risk of weight gain is a drawback for obese adults. Use of glibenclamide in elderly diabetic patients is not advised as it has the highest hypoglycaemic risk[13] with a protracted hypoglycaemic effect due to the prolonged presence of active metabolites dependent on renal elimination.[14] Initiation of drugs with anorexic side-effects in elderly diabetic patients under stable control with sulphonylureas may also result in hypoglycaemic attacks, e.g. prescription of clarithromycin as part of *Helicobacter pylori* eradication therapy.[15] Risk of hypoglycaemia is the lowest with gliclazide.[7] The ADVANCE trial demonstrated improved outcomes with gliclazide MR without weight gain and with low rates of hypoglycaemia.[16] Severe hypoglycaemia, although uncommon in the ADVANCE trial, was more common in the intensive-control group (2.7%, vs. 1.5% in the standard-control group; hazard ratio, 1.86; 95% confidence interval, 1.42-2.40; P<0.001). Sulfonylureas are nonetheless a good choice for older adults who eat consistently, and are able to recognise and treat hypoglycaemia appropriately. They can be used safely in chronic kidney disease, although shorter-acting ones may be preferred, and glibenclamide should be avoided.

41.5.2 Biguanides

The mechanisms of action of biguanides are the inhibition of lipolysis in adipose tissue, increased insulin-mediated glucose uptake in muscle, and decreased gastrointestinal absorption of glucose. Biguanides have no effect on pancreatic insulin secretion.

Metformin has a plasma half-life of 1.5-4 hours and is not metabolised in the body. It is an effective agent and achieves a HbA1c reduction of 1.5-2%. Its low potential for hypoglycaemia, low cost, wide availability, and lack of weight gain side-effect make it a preferred agent as first-line therapy. Its higher gastrointestinal intolerance (diarrhoea, abdominal discomfort or anorexia) and unintended weight loss, however, may be undesirable in frail elderly diabetic patients.

Dosage reduction with regular monitoring of renal function is necessary if estimated glomerular filtration rate (eGFR) is 30-60 mL/min/1.73 m^2. Metformin should be used with caution in those at risk of a sudden deterioration in kidney function and it should be avoided if eGFR is <30 mL/min/1.73 m^2.[2,17,18] Lactic acidosis is exceedingly rare if used carefully with adjustment according to the eGFR.[17] Lactic acidosis occurs in about nine per 100 000 patient treatment years.[19] Metformin should be withdrawn promptly in conditions that predispose to lactic acidosis, such as acute onset of dehydration, congestive heart failure, renal failure, liver failure, hypoxaemia, sepsis, or the use of contrast media.[5] It can lower vitamin B_{12} level so should be used with caution in patients with pre-existing and untreated vitamin B_{12} deficiency.

41.5.3 Meglitinide Analogues

These are rapid-acting insulin secretagogues that stimulate pancreatic insulin release, similar to sulphonylureas. Repaglinide (NovoNorm) and nateglinide have a short half-life of 60-90 minutes. They should be taken just before meals, and can be skipped if meals are skipped, making this drug class particularly convenient for the dementia patients with erratic eating habits, thus avoiding hypoglycaemia. Disadvantages of the glinides include multiple dose administration and high cost. They are metabolised in the liver, and should be used with caution in patients with liver failure. HbA1c reductions of about 1-1.5% and 0.5-1.0% can be achieved for repaglinide and nateglinide, respectively.

41.5.4 Thiazolidinediones

These drugs are agonists of the peroxisome proliferator-activated receptor in muscle, fat, and liver. They enhance the sensitivity of peripheral glucose uptake and possibly reduce hepatic glucose output. They lower HbA1c by 1-2%, and have a

Table 41.5 Characteristics of specific sulphonylurea drugs

Drug	Dose range (mg)	Peak level (hours)	Half-life (hours)
Tolbutamide	500-3000	3-5	4.5-6.5
Glibenclamide	2.5-20	4	10
Gliclazide	40-320	2-4	8-12
Glimepiride	1-8	2-3	5-9
Glipizide	2.5-20	1-3	2-4
Gliclazide MR	30-120	4-6	16

low risk of hypoglycaemia. Adverse effects include weight gain, fluid retention, peripheral oedema, and aggravation of congestive heart failure. It is important to remember the associated risk of upper limb fracture, particularly in older females, if this class of drug is being considered in elderly people with fall risk or established osteoporosis. There are also recent concerns about an increased risk of bladder cancer with pioglitazone, although the association has not been proven. Thiazolidinediones are metabolised by the liver, and not excreted by the kidneys. In view of the potential risk of fluid retention, they should not be used in chronic kidney disease or advanced heart failure. When used in conjunction with insulin, dosage should be kept at half the maximum.

41.5.5 α-Glucosidase Inhibitors

Acarbose acts on the gastrointestinal tract to inhibit the breakdown of complex carbohydrates in the small bowel. Hence the absorption of glucose is delayed and postprandial glucose levels reduced. Acarbose is only minimally absorbed from the gastrointestinal tract and is metabolised by intestinal bacteria and digestive enzyme degradation. Its plasma half-life is about 2 hours. It produces an HbA1c lowering of 0.5-1.0%. It has a low risk of hypoglycaemia, but its high cost and gastrointestinal side-effects such as flatulence and diarrhoea are major limiting factors in its use.

41.5.6 Dipeptidyl Peptidase 4 Inhibitors

DPP4 inhibitors work by blocking the action of dipeptidyl peptidase 4 (DPP4), an enzyme that degrades the incretin hormones. Incretins (e.g. glucagon-like peptide 1 and glucose-dependent insulinotropic peptide) are gut hormones that are released in response to a meal. They stimulate insulin secretion and inhibit glucagon release.

These agents are useful for postprandial hyperglycaemia, an important feature in older people. Other potential benefits are low risk of hypoglycaemia, weight neutral, lack of gastrointestinal side-effects, and their safe use in patients with renal insufficiency. The three most commonly reported adverse reactions in clinical trials were nasopharyngitis, upper respiratory tract infection, and headache. The first DPP4 inhibitor sitagliptin was approved in 2006, followed by vildagliptin, saxagliptin, alogliptin, and linagliptin (Table 41.6). They have an efficacy of HbA1c reduction of 0.6-0.8% compared with other agents, and are of higher cost.

41.5.7 Glucagon-like Peptide 1 Receptor Agonists

They are incretin mimetic agents that require subcutaneous injection. Similar to DPP4 inhibitors, they reduce postprandial hyperglycaemia, with an additional benefit of weight reduction by 1.5-3 kg. They are, however, associated with nausea and vomiting that may be intolerable. Examples of glucagon-like peptide 1 receptor agonists (GLP-1RA) include exenatide, exenatide extended-release, and liraglutide. They reduce HbA1c by 0.8-1.5%. Exenatide is not recommended in patients with eGFR of <30 mL/min/1.73 m^2, and liraglutide with eGFR of <60 mL/min/1.73 m^2.[20]

Postmarketing surveillance studies and case reports have linked incretin-based therapies (GLP-1RA and DDP4 inhibitors) with acute pancreatitis. The European Medicines Agency and the US Food and Drug Administration have called for additional studies to assess this potential risk. To date, both positive[21] and negative[22,23] studies on the association of DDP4 inhibitors with acute pancreatitis have been published, but a causative relationship has not been established.

41.5.8 Sodium Glucose Cotransporter 2 Inhibitors

These are new agents that produce a glucose-lowering effect by inhibition of renal glucose reabsorption. Examples of sodium glucose cotransporter 2 (SGLT2) inhibitors are dapagliflozin, canagliflozin, and empagliflozin. They lower HbA1c by 0.7-0.8%. The risk of hypoglycaemia is low, but the associated increased risk of genital and urinary tract infection, hypovolaemia, postural hypotension, and weight loss (2-3 kg) may be problematic in some older individuals. Recent study on empaglifozin demonstrated significant reduction

Table 41.6 Characteristics of DPP4 inhibitors

	Linagliptin	Saxagliptin	Sitagliptin	Vildagliptin
Dosage	5 mg Daily	5 mg Daily	100 mg Daily	50 mg Twice daily
Primary route of excretion	None	Liver	None	Liver
Share of renal excretion	Bile and gut	Kidney	Kidney	Kidney
Dose adjustment in renal insufficiency	No	Yes	Yes	Yes
GFR (mL/min/1.73 m^2)		≤50: 2.5 mg daily	30-50: 50 mg daily <30: 25 mg daily	<50: 50 mg daily
Drug-related monitoring	No	RFT	RFT	RFT, LFT

Abbreviations: DPP4 = dipeptidyl peptidase 4; GFR = glomerular filtration rate; LFT = liver function test; RFT = renal function test

in primary outcome (cardiovascular death, non-fatal myocardial infarction, and non-fatal stroke) by 14%, cardiovascular mortality by 38%, and hospitalisation for heart failure by 35%.[24] Renal function should be evaluated prior to initiation of SGLT2 inhibitors and monitored periodically thereafter. More frequent renal function monitoring is recommended in patients with an eGFR of <60 mL/min/1.73 m^2. Use of SGLT2 inhibitors is not recommended when eGFR is <45 mL/min/1.73 m^2.

41.5.9 Insulin

Insulin therapy should be considered if glycaemic control is unsatisfactory, and should not be precluded in advanced age. Elderly people, however, do need the following special considerations.

- Visual disturbances and impaired dexterity may result in inadequate or overdosage of insulin. This requires predrawn syringes by a caregiver or use of a syringe magnifier or use of injection pens or even injection by a caregiver.
- Inability to prepare meals or decreased food intake (e.g. from concurrent illness) may result in hypoglycaemia.
- Worsening renal and hepatic function may affect clearance of insulin and result in hypoglycaemia.
- The need for more blood glucose monitoring may increase treatment burden.
- Hypoglycaemia risk, especially nocturnal, is somewhat lower with insulin analogues compared with human insulin but the former are more expensive.

Referral to a diabetes specialist nurse is strongly advised for an elderly diabetic patient if insulin therapy is being considered or commenced.

41.5.10 Choice of Drugs

When making drug choices, therapy should be individualised based on the following:

- knowledge of the underlying pathophysiology, e.g. consider metformin as first-line therapy for obese patients;
- degree of hyperglycaemia, e.g. metformin, sulphonylurea, and insulin have a greater glucose-lowering effect;
- risk of hypoglycaemia, e.g. DPP4 inhibitor, GLP-1RA, acarbose, and thiazolidnediones have minimal risk of hypoglycaemia;
- side-effect profile in relation to patient's medical conditions, such as weight gain, weight loss, or gastrointestinal symptoms;
- renal function of the patient, e.g. metformin should be avoided if eGFR is <30 mL/min/1.73 m^2;
- functional, cognitive status, and need for caregiver involvement;
- personal preferences and compliance of patient; and
- cost and availability

Generally, metformin is recommended as initial treatment for older people after lifestyle modifications, just as for all adults.[7,8] The blood glucose treatment algorithm for older people with diabetes taken from the International Diabetes Federation global guidelines for managing older people with type 2 diabetes is listed in Table 41.7.[7]

41.6 Complications with Special Relevance to Elderly Diabetic Patients

41.6.1 The Diabetic Foot

Diabetic foot problems are a common complication of diabetes and lower limb amputation is more common in elders, usually type 2 diabetic patients. A number of causes contribute to foot problems, including:

- peripheral sensorimotor neuropathy, with symptoms of numbness, 'pins and needles', burning pain, and hyperaesthesia;
- autonomic neuropathy, with dry, atrophic skin, and dilated dorsal veins;
- peripheral vascular disease;
- limited joint mobility, especially at the subtalar joint or great toe extension;
- high foot pressure, with callus formation at sites of high pressure; and
- reduced resistance to infection

The above factors can result in foot ulceration, and if coupled with infection, can lead to amputation. In

Table 41.7 International Diabetes Federation guideline on blood glucose treatment for older diabetic patients[7]

	Usual approach	Alternative approach	Other options (in alphabetical order)
First-line therapy	Metformin	Sulfonylurea or DPP4 inhibitor	Acarbose, glinides, insulin, SGLT2 inhibitor, or thiazolidinedione
Second-line therapy: dual therapy by adding to first line	Sulfonylurea or DPP4 inhibitor	Metformin (if not used as first line)	Acarbose, glinides, GLP-1RA, insulin, SGLT2 inhibitor, or thiazolidinedione
Third-line therapy: triple oral therapy, insulin, or GLP-1RA	DPP4 inhibitor or sulfonylurea or basal insulin or pre-mix insulin	GLP-1RA	Acarbose, glinides, SGLT2 inhibitor, or thiazolidinedione

Abbreviations: DPP4 = dipeptidyl peptidase 4; GLP-1RA = glucagon-like peptide 1 receptor agonists; SGLT2 = sodium glucose cotransporter 2

particular, an elderly individual who lives alone, has impaired vision, or a history of foot ulceration with the above factors should be identified as high risk and referred to a podiatrist for regular review.

Daily foot examination should be encouraged for elderly diabetic patients. The help of caregivers should be sought for those who have problems with vision or joint immobility that hinder self-examination of feet. New shoes should be correctly measured and fitted. A square toe box and lace type are preferred. Elderly people should wear socks and not be barefoot. Nails should be cut straight across instead of rounded. Water temperature should be checked before bathing and feet kept away from excessive heat.

Ischaemia or infection can deteriorate quickly. Any signs of critical ischaemia (rest or night pain, pale mottled feet, dependent rubor, ischaemic ulceration, gangrene) or severe infection (cellulitis, abscess) may warrant emergency referral to hospital specialists. Intravenous antibiotics are required for infection control if systemic signs are present, e.g. fever or worsening of infection observed after 48 hours of initiation of oral antibiotics. Polymicrobial organisms are usually involved in diabetic ulcer infection. Antibiotics should cover Gram-negative organisms. Delay in management will result in undesirable amputation.

41.6.2 Vision Loss

41.6.2.1 Retinopathy

Up to 30-40% of patients have diabetic retinopathy at presentation. Diabetic retinopathy is a microvascular complication of diabetes and may be associated with maculopathy. Diabetic retinopathy can result in blindness. Delay in treatment for >2 years from diagnosis of sight-threatening diabetic retinopathy is associated with poor outcome and severe vision loss. Systematic annual screening for diabetic retinal disease should be provided for all diabetic patients.

The following factors have been shown to affect the development and progression of diabetic retinal disease and intervention should be initiated for modifiable factors to halt or slow down progression:
- poor glucose control;
- high blood pressure;
- microalbuminuria and proteinuria;
- raise triglyceride and lower haematocrit;
- duration of diabetes; and
- increasing number of microaneurysms

Referral to an ophthalmologist should be considered in the following circumstances:
- unexplained drop in visual acuity;
- preproliferative or proliferative retinopathy;
- macular oedema; and
- non-proliferative retinopathy that is severe, of new onset or progressive

41.6.2.2 Cataract

Vision outcome following cataract surgery is closely related to age and severity of retinopathy prior to surgery. Cataract extraction should not be delayed in patients with diabetes. It is also advised when sight-threatening retinopathy cannot be excluded.

41.6.3 Nephropathy

Diabetic nephropathy is the recent most common cause for commencement of renal dialysis in Hong Kong. It accounts for one-third of all patients who require dialysis and is the single most critical determinant of overall prognosis, particularly in Asians. It is also associated with an increased risk of macrovascular disease. The pathogenesis of nephropathy is as follows.
- Microalbuminuria (30-300 mg/day) → macro-albuminuria (>300 mg/day) → proteinuria → increase in creatinine concentration
- 20-40% of microalbuminuria → overt nephropathy

Diabetic patients should have annual screening for microalbuminuria, the earliest sign of diabetic nephropathy. A first morning urine sample test with measurement of urinary albumin-creatinine ratio of >2.5 mg/mmoL in men and >3.5 mg/mmoL in women; or a urinary albumin concentration of >20 mg/L in two out of three samples measured within 6 months will establish the diagnosis. A positive urine albustix already signifies the presence of proteinuria.

Risk factors for the development of diabetic nephropathy include:
- poor glucose control;
- high blood pressure;
- long duration of diabetes;
- presence of retinopathy;
- smoking;
- increasing age;
- genetic factors;
- male gender;
- increased cholesterol and triglyceride; and
- increased serum homocysteine level

With early and aggressive interventions, diabetic nephropathy is now regarded as a potentially preventable complication. The mainstay of treatment includes:
- tight control of hypertension, target should be <130/80 mm Hg;
- tight control of glucose;
- use of angiotensin-converting enzyme inhibitors or angiotensin II receptor antagonists for their renoprotective effect, independent to that of blood pressure lowering;

- good control of hyperlipidaemia; and
- cessation of smoking

Patients should be referred for assessment by specialists and discussion of need for dialysis if creatinine level is >200-300 mmol/L. Work-up to identify alternative causes of proteinuria or renal impairment is advised if causes other than diabetic nephropathy are suspected in the presence of features that are not typical of diabetic nephropathy, e.g. absence of retinopathy, short duration of diabetes, or presence of red blood cells or casts in urine specimens.

41.6.4 Hypoglycaemia

Hypoglycaemia is the most serious and disruptive side-effect of the treatment of diabetes in elderly people. It can result in adverse consequences such as falls, fractures, seizures, coma, poor quality of life, prolonged arrhythmias, and death.[25] Ageing may impair counter-regulatory hormonal responses to hypoglycaemia,[26] thus there is an increased risk of hypoglycaemia going unnoticed. Moreover, a lower blood glucose level is required to initiate hypoglycaemic symptoms and the intensity of symptoms is also lower. Elderly patients have fewer sympathetic symptoms, e.g. palpitation, tremor at the time of hypoglycaemia, and neuroglycopaenic symptoms, e.g. weakness, poor concentration, malaise, poor concentration, dizziness and confusion, predominate. Cognitive dysfunction in elderly patients affects the perception of hypoglycaemic symptoms. Severe hypoglycaemic episodes are associated with an increased risk of dementia.[27]

Elderly patients with type 2 diabetes mellitus may be more prone to hypoglycaemia due to the following risk factors[25,26]:

- declining renal function;
- use of long-acting sulphonylurea, e.g. glibenclamide;
- poor drug clearance;
- polypharmacy;
- medications that alter autonomic warning symptoms of hypoglycaemia such as angiotensin-converting enzyme inhibitor and beta blockers;
- malnutrition / malabsorption;
- reduced food intake / erratic meals;
- swallowing problems;
- unusual activity such as wandering about during the night; and
- blocked feeding tubes

Elders should be educated about the management of hypoglycaemia whenever oral hypoglycaemic agents are prescribed. Caregivers of frail elders should be educated as well. Tight control is advocated for independent elderly person with good functional and cognitive status. Nonetheless less tight control is advised for frail elderly people, e.g. with dementia or stroke that results in them being chair-bound, to avoid the risk of hypoglycaemia. An HbA1c of <7% should be used as a warning of possible overtreatment in this group, and glucose level of <6.0 mmol/L should be avoided.[7]

41.6.5 Hyperglycaemia

Although emphasis has been placed on the risks of hypoglycaemia in elderly diabetic patients, untreated or undertreated hyperglycaemia also has risks, even in patients with a limited life expectancy. Chronic hyperglycaemia increases the risks for dehydration, electrolyte abnormalities, urinary incontinence, dizziness, and falls. Hyperglycaemic hyperosmolar syndrome is a severe complication of hyperglycaemia with a risk of mortality, hence severe hyperglycaemia should be minimised in all patients.

41.7 Organisation of Diabetes Services for Elders

The St Vincent Declaration in 1989 called attention to the need for regular review and structured care for diabetic patients, something that is of particular importance for elderly people with diabetes who are often missing out.[28,29] The subsequent Paris Declaration in 2000 identified primary areas of concern for elderly diabetic patients, leading to the release of the clinical guidelines for type 2 diabetes mellitus by the European Diabetes Working Party for Older People in 2004,[30] subsequently updated in 2011.[8] They include:

- importance of functional and vascular risk assessment;
- relationship between functional outcome and metabolic control;
- management of diabetes in primary care;
- detection of cognitive impairment and depression;
- management of specific major complications, e.g. foot disease, visual loss, hypoglycaemia, pain;
- care home diabetes; and
- ethical and moral aspects of treatment

Modern diabetes care systems for elderly people require integrated care between general practitioners, hospital specialists, and other members of the healthcare team; with an emphasis on prevention of diabetes and its complications, early intervention for vascular disease, and assessment of disability due to limb problems, eye disease, and stroke.

A structured diabetic care include the following[5]:

- design a protocol with which all staff members agree;
- make a register of all patients diagnosed with diabetes;
- undertake a survey and record of every diabetic patient;

- organise an appointment and recall system;
- organise a monitoring system;
- organise referrals to paramedics and hospital specialists;
- initiate patient education; and
- set up a regular evaluation / audit of care

Areas to be reviewed at diagnosis include the following.[5]

- Existing disease?
- On medication?
- Complications (examine fundus and feet, and cardiovascular diseases and risk factors)
- Blood: glucose, HbA1c, serum creatinine, lipids
- Review goals of care
- Education
- Monitoring

The following are essential elements of an annual review.[5]

- Body mass index
- Blood pressure: lying and standing
- HbA1c
- Creatinine
- Lipids
- Smoking habit
- Foot examination
- Retinal examination and visual acuity
- Pharmacotherapy
- Coexistent diseases

Below are the criteria for referral to hospital specialists.[5]

- Commencement of insulin
- Uncontrolled severe hyperglycaemia and hypertension
- Severe hypoglycaemia
- Presence of diabetic complications that require hospital support, e.g. stroke, ischaemic heart disease, renal impairment, peripheral vascular disease, retinopathy
- Multiple problems requiring multidisciplinary intervention, e.g. foot ulcer in an elderly person living alone, increasing dependency, and immobility following a stroke

Elderly patients with type 2 diabetes who fulfil the following criteria will benefit from comprehensive geriatric assessment (CGA) covering global / physical function, cognitive / affective function, nutritional status, and social networks / support[30]:

- presence of a 'geriatric syndrome': confusional state, depression, falls, incontinence, immobility, pressure sores;
- those with evidence of functional decline;
- those with several coexisting comorbidities apart from diabetes with complex drug regimens;
- those with disabilities due to lower limb vascular disease or neuropathy requiring a rehabilitation programme; and
- absence of a terminal illness or advanced dementia syndrome

CGA targeted at these frail elderly people is crucial at the initial assessment, and helpful in planning care, and rehabilitation and monitoring progress.

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CHAPTER 42

Thyroid Disorders

Tin-wai WONG

OBJECTIVES

- To learn the effects of ageing on thyroid hormone synthesis, secretion, degradation, and feedback.
- To learn the presentation, diagnosis, and assessment of elderly patients with various types of thyroid disorder.
- To appreciate the interaction of drugs and polypharmacy in relation to diagnosis and management of thyroid disorders in elderly patients.

KEY POINTS

- The ageing process affects the thyroid gland and serum thyroid parameters.
- Interpretation of thyroid function in elderly patients is difficult due to coexistent chronic illness, polypharmacy, and ageing.
- The prevalence of thyroid disease increases in elderly people and is associated with significant morbidity if left untreated.
- Classic symptoms of hyper / hypothyroidism are usually absent, and may mimic signs of ageing or masquerade as diseases of the cardiovascular, gastrointestinal, or nervous system.
- Treatment of overt hyper / hypothyroidism is essentially the same as that for younger adults.
- The decision on whether to treat subclinical thyroid disease is based on thyroid-stimulating hormone / free T4 levels, symptoms, and risk factors of the individual patient.
- Thyroxine replacement in elderly patients should be initiated at a low dose followed by gradual upward titration every 4 to 6 weeks until adequate replacement is achieved.
- The incidence of thyroid cancer increases with age, as well as being more aggressive in nature.

42.1 Iodine Nutrition Status and Its Impact on Thyroid Function

Iodine is an essential micronutrient required for the synthesis of thyroid hormones. Approximately 50-75 μg of iodine is required per day to maintain an adequate level of thyroid hormone. The World Health Organization (WHO) recommends a daily iodine intake of 150 μg for adults and 250 μg for pregnant or lactating women.[1]

Iodine deficiency remains the most common cause of hypothyroidism worldwide. Global iodine status has improved markedly over the past decade, however, largely due to strengthened salt iodisation programmes and improved monitoring. Iodine nutrition status in different countries can be reviewed at www.iccidd.org. A local study in 1995 showed that 55.3% of elderly people in Hong Kong had a fasting urinary iodine concentration below 100 μg/L (0.8 μmol/L), the WHO cut-off value for iodine insufficiency; and 20.0% had a level of <50 μg/L (0.4 μmol/L), the WHO cut-off value for severe iodine insufficiency.[2]

The following can ensure sufficient iodine intake in older adults:

- eat a variety of iodine-rich foods (seaweed, seafood, milk, milk products, eggs, egg products) as part of a healthy balanced diet
- replace non-iodised salt with iodised salt, adhering to the WHO recommendation of <5 g salt per day
- steam or stir-fry with little oil to retain the maximum amount of iodine in food
- add iodised salt just before serving

42.1.1 Iodine-induced Hyperthyroidism

Transient hyperthyroidism can occur after excessive iodine exposure in patients with underlying thyroid conditions.[3] Iodine-induced hyperthyroidism is most commonly found in iodine-deficient areas, and in elderly patients with longstanding nodular goitre.[4] Increased availability of iodine as substrate leads to an increase in thyroid hormone synthesis and release by autonomous thyroid tissue. In normal thyroid tissues

and following excess iodine administration, the Wolff-Chaikoff effect protects against iodine-induced hyperthyroidism by inhibiting iodide organification in the thyroid gland. Nonetheless in patients with underlying thyroid autonomy, the Wolff-Chaikoff effect may be absent.

42.1.2 Iodine-induced Hypothyroidism

Transient hypothyroidism occurs as a result of the Wolff-Chaikoff response to excess iodine exposure. The Wolff-Chaikoff effect usually lasts several days, after which it is followed by an escape phenomenon, and normal thyroid iodide organification resumes. However, prolonged hypothyroidism can occur in patients with underlying organification defects such as Hashimoto thyroiditis, and also in patients with a failure to halt the Wolff-Chaikoff effect, for example, in some patients prescribed amiodarone therapy.[5]

42.2 Thyroid Hormones, Thyrotropin, Antibodies, and Ageing

Thyroid hormone secretion, metabolism and action all change with advancing age. In general, ageing is often associated with increased serum thyroid-stimulating hormone (TSH) and reverse triiodothyronine (rT3), decreased serum free T3/total T3 (fT3/T3), and usually unchanged serum free T4/total T4 (fT4/T4). These may vary across studies and populations, however, and are influenced by ethnicity, genetic background, iodine status, and prevalence of thyroid autoimmunity.

Early reports observed a decrease in serum TSH,[6] common in iodine-deficient or iodine borderline-sufficient populations.[7,8] However, increasing evidence now indicates that serum TSH tends to rise in elderly people, up to values above the upper limit of the traditional reference range.[9] Recent cross-sectional studies in the US including the National Health and Nutrition Examination Survey, as well as large longitudinal studies have shown a positive correlation between ageing and increasing serum TSH.[10-13] This is particularly true in iodine-sufficient areas, and in populations where the dominant abnormality is hypothyroidism secondary to Hashimoto thyroiditis. The mechanism attributed to the change in serum TSH level in elderly people is unclear. It is postulated that the negative feedback loop between fT4 and TSH is altered, and TSH glycosylation, hence its bioactivity is affected.[9]

Serum fT4 level in elderly people tends to remain normal, as supported by cross-sectional and recent longitudinal studies.[6,11,12] It is not just thyroid hormone synthesis and secretion that decline with age (particularly in subjects aged >60 years). Peripheral T4 degradation by outer ring deiodination also diminishes.[14] As a result, the decrease in degradation equals the decrease in thyroidal secretion, so fT4 concentrations in elderly people appear unaffected.

Serum fT3 and T3 levels decrease with advancing age, and this finding is consistent in all studies.[14] The decline in serum T3 is likely explained by a decrease in peripheral T4 to T3 conversion, due to decreased deiodinase type 1 (D1) and deiodinase type 2 (D2) activity.[15] It is postulated that the reduced T3 level serves as an adaptive response to ageing. Decreasing T3 will lower basal metabolic rate, and consequently lower the production of reactive oxygen species and damage to biomolecules (e.g. DNA), and hence slow down the ageing process.

D1 and D2 are also responsible for clearance of the inactive metabolite rT3. The net result of this decreased deiodination with advancing age is an age-dependent decline in total and fT3 levels and an increase in serum rT3 level.[15]

The prevalence of thyroperoxidase antibodies (TPOAb) and antithyroglobulin antibodies increases during life, especially in females, and plateaus between the sixth and eighth decade.[15] Nonetheless, positivity for thyroid antibodies in old age does not predict future development of a thyroid disorder, nor does it affect morbidity and mortality.[12] Routine checking in elderly people is not recommended.

42.3 Factors and Drugs Influencing Thyroid Function Tests

When interpreting abnormal thyroid function tests in elderly people, it is important to consider concurrent medical illness, nutritional status, and prescribed drugs. Non-thyroidal illness (NTI), also known as "low T3 syndrome", is characterised by diminished T3 and elevated rT3 levels, and is caused by acute and chronic diseases in the absence of genuine thyroid disease. NTI occurs in up to 32% of critically ill hospitalised patients, and is more common among elderly patients.[16] The underlying mechanism is believed to be decreased D1 and increased deiodinase type 3 activity. The net result of a low T3 is thought to be a beneficial adaption to stress, as the reduced catabolic state may help to avoid further damage. Fasting, caloric restriction, and malnutrition may also produce a similar picture to NTI.

It is well known that certain medications may influence thyroid function tests, and elderly patients are more susceptible as they use more drugs. Drugs may interfere with thyroid function by the following mechanisms[17]:

- suppress TSH secretion (e.g. glucocorticoids, dopamine, octreotide, bexarotene)

- decrease thyroid hormone secretion (e.g. iodide, amiodarone, lithium)
- increase thyroid hormone secretion (e.g. iodide, amiodarone)
- decrease T4 absorption (e.g. ferrous sulfate, aluminium hydroxide, calcium carbonate, cholestyramine, colestipol, sucralfate, omeprazole)
- alter T4 and T3 metabolism (e.g. phenytoin, carbamazepine, phenobarbital, rifampicin)
- increase serum thyroxine-binding globulin (TBG) [e.g. oestrogens, heroin, methadone, mitotane, fluorouracil]
- decrease serum TBG (e.g. androgens, anabolic steroids)
- displacement from protein-binding sites (e.g. furosemide, salicylates, heparin, meclofenamate)
- others such as cytokine mediated (e.g. interferon-alpha)

Drugs that suppress serum TSH to a subnormal level rarely suppress to an undetectable level (e.g. <0.01 mIU/L). Dopamine infusions at >1 μg/kg/min, dexamethasone at 0.5 mg/day, and hydrocortisone at 100 mg/day are known to inhibit TSH release.[17] Bexarotene, a retinoid X receptor ligand used in the treatment of lymphoma, suppresses the pituitary TSH-beta promoter, leading to central hypothyroidism.

Medications that alter TBG concentration cause a change in total T4 and total T3, but do not affect fT4 or fT3 concentration. However, drugs that inhibit binding of thyroid hormones to TBG may cause a transient increase in free thyroid hormone level. Examples of such drugs include furosemide (>80 mg/day), salicylates (>2.0 g/day), heparin and several non-steroidal anti-inflammatory drugs.[17]

Amiodarone, a class III antiarrhythmic agent, is commonly used in elderly patients to treat ventricular arrhythmia. Its use may induce both hypothyroidism and hyperthyroidism. Amiodarone-induced hypothyroidism typically occurs between 6 and 12 months after commencing amiodarone treatment. If amiodarone cannot be discontinued, levothyroxine therapy can be initiated. There are two types of amiodarone-induced hyperthyroidism. Type 1 is related to iodine-induced hyperthyroidism as amiodarone has a high iodine content, and it typically occurs in individuals with underlying non-toxic multinodular goitre (MNG) or Graves' disease. Type 2 is caused by drug-induced destructive thyroiditis. Many patients, however, have overlapping type 1 and type 2 amiodarone-induced hyperthyroidism. Treatment for type 1 disease is with large doses of antithyroid drugs, such as methimazole 40-80 mg per day, or propylthiouracil 400-800 mg per day. For type 2 disease, prednisolone 40-60 mg daily is considered the treatment of choice.[18]

Lithium can cause goitre, hypothyroidism, chronic autoimmune thyroiditis, and possibly hyperthyroidism. Lithium inhibits thyroid hormone secretion and synthesis, although the underlying mechanism is not well understood. Goitre may develop as a result of an increased pituitary TSH secretion in response to the inhibition of thyroid hormone. Lithium-induced hypothyroidism usually occurs within the first 2 years of therapy and in 5-20% of patients; many of whom have underlying chronic autoimmune thyroiditis. De-novo autoimmune thyroiditis may also be induced by lithium. Treatment is by simply adding levothyroxine if lithium is continued.

In patients who are prescribed thyroxine replacement therapy, it is important to be aware of drugs that impair its absorption and or affect its metabolism (see above). In such cases, levothyroxine should be separated from these drugs, or a higher dose of levothyroxine may be needed.

42.4 Hyperthyroidism

The prevalence of hyperthyroidism in older adults is estimated to be 0.5-4%.[9] Although Graves' disease remains the most common cause, the frequency of toxic MNG also increases with age. Toxic MNG occurs more frequently in areas with a low iodine intake, whereas Graves' disease is more common in areas with a relatively high iodine intake such as the US and Northern Europe.[6,19,20] Single toxic adenoma is rare. Subacute thyroiditis can also occur with advancing age. Other aetiologies of hyperthyroidism are listed in Table 42.1.[21]

Compared with younger patients, elderly hyperthyroid patients display fewer clinical symptoms and signs.[3] In elderly patients, classic features of sympathetic overdrive such as hand tremor, heat intolerance, nervousness, and sinus tachycardia may be absent; palpable goitre is less common while atrial fibrillation is more common. Symptoms may be non-specific and include

Table 42.1 Causes of hyperthyroidism in the older adults[21]

- Graves' disease
- Toxic multinodular goitre
- Toxic adenomas
- Painless (silent) thyroiditis
- Subacute (granulomatous, de Quervain's) thyroiditis
- Acute thyroiditis
- Drugs (e.g. amiodarone-induced thyroiditis)
- Iatrogenic thyrotoxicosis
- Factious ingestion of thyroid hormone
- Struma ovarii
- Extensive metastases from follicular thyroid cancer
- Thyroid-stimulating hormone–producing pituitary adenomas
- Resistance to thyroid hormone

unexplained weight loss, loss of appetite, muscle weakness, diarrhoea, constipation, and persistent vomiting. One-third of hyperthyroid elderly patients may even present with apathetic hyperthyroidism.[19] Psychiatric manifestation as depression or mania can also occur.[22]

Despite fewer thyrotoxic features, the frequency of atrial fibrillation, heart failure, and cardiovascular mortality is higher in older adults.[23] Atrial fibrillation is estimated to be present in up to 20-35% of older hyperthyroid patients, and sometimes may be the only clinical sign.[24] Because of the degeneration of sinus nodes and frequent use of beta-blockers in elderly patients, tachycardia or palpitations may be absent.

Low bone mineral density and osteoporotic fracture are also known complications of hyperthyroidism. A diagnosis of thyrotoxicosis should therefore be considered in the presence of hypercalcaemia or worsening osteoporosis, especially in this older age-group.

The preferred treatment in most cases of hyperthyroidism in elderly patients is radioactive iodine (RAI) ablation, because of its high efficacy, safety, and cost-effectiveness. The most common side-effect is the development of subsequent hypothyroidism, and long-term thyroid hormone replacement therapy is required. One potential concern is that elderly patients are at increased risk of complications of worsening hyperthyroidism from transient RAI-related thyroiditis. Hence, post-treatment with beta-adrenergic blockade, and resuming antithyroid drug 3-7 days after RAI should be considered. In patients with toxic MNG or toxic adenoma, RAI should be the mainstay of treatment, because remission is very unlikely to occur otherwise. Alternatively lifelong low-dose antithyroid drugs could be an option for frail or institutionalised elderly patients.

Antithyroid drugs are used when RAI is contraindicated, or due to personal preference. Carbimazole or methimazole is now the drug of choice. Because of (fatal) fulminant hepatic necrosis that has been reported with use of propylthiouracil, use is generally reserved for the treatment of thyroid storm. Mild adverse side-effects of antithyroid drugs include abnormal sense of taste, pruritus, arthralgia, and urticaria. Hepatotoxicity is a serious side-effect and reported in 0.1-0.2% of treated patients.[25] The most serious adverse drug event is agranulocytosis that often presents with fever and a sore throat and affects 0.2-0.5% of patients.[26] Older adults may be at greater risk of side-effects. They are also at a higher risk of relapse after drug therapy.

Thyroidectomy is generally reserved for large goitres with obstructive symptoms, or associated malignancy.

Beta-blockers (i.e. propranolol, atenolol, metoprolol) should be given to elderly patients with symptomatic thyrotoxicosis. Beta-adrenergic blockade helps to reduce heart rate, systolic blood pressure, muscle weakness, tremor, irritability, emotional lability, and to improve exercise tolerance.[21] Anticoagulation should be considered for atrial fibrillation in the absence of contraindications, as hyperthyroid patients are at high risk of embolic stroke.

42.5 Subclinical Hyperthyroidism

Subclinical hyperthyroidism is defined as low serum TSH level with normal fT4 and fT3. The prevalence of subclinical hyperthyroidism increases in older populations, especially those older than 70 years, and in women.[15,27] The estimated prevalence is 1-2.5% in iodine-sufficient populations,[10,28] and up to 9% in iodine-deficient populations.[29]

The aetiologies of subclinical hyperthyroidism are essentially the same as those for overt hyperthyroidism. Nonetheless it is important to note that elderly patients are more prone to overtreatment with levothyroxine because its requirement decreases with age. Prior to making a diagnosis of subclinical hyperthyroidism, it is essential to exclude drugs (e.g. glucocorticoid), NTI, and malnutrition as an underlying cause. In addition, a second measurement of serum TSH at 3 to 6 months is necessary to exclude a transient low level.[30] Progression from subclinical to overt hyperthyroidism is uncommon in elderly patients, approximately 1% per year.[31] Risk factors include a lower serum TSH level (i.e. <0.1 mIU/L), and those with toxic MNG.[32]

Even without progression to overt hyperthyroidism, subclinical hyperthyroidism by itself is not without risks. Subclinical hyperthyroidism is associated with a 2.8-fold increased risk of atrial fibrillation in persons over the age of 60 years,[33] as well as decreased bone mineral density and osteoporosis. An increased fracture rate has been found in postmenopausal women even with only mildly suppressed serum TSH level.[34]

Treatment of subclinical hyperthyroidism is controversial. The choice between treatment and adopting a wait-and-see approach depends on the TSH level, clinical risk factors (atrial fibrillation, osteoporosis), and the underlying aetiologies. The American Thyroid Association (ATA) recommends that patients aged ≥65 years be treated if their serum TSH level is persistently <0.1 mIU/L and that treatment can be considered if their serum TSH

level is below the lower limit of normal but above 0.1 mIU/L.[30] A summary of factors recommended by ATA to consider when deciding whether to initiate treatment is listed in Table 42.2.[30] If subclinical hyperthyroidism is to be treated, the treatment is similar to that for overt hyperthyroidism.

42.6 Hypothyroidism

The prevalence of overt hypothyroidism is variable depending on the dietary iodine intake and environmental factors of the population, but it steadily increases with advancing age, and affects up to 5% of subjects older than 60 years.[35] Hypothyroidism has a higher prevalence in iodine-sufficient regions than in mild iodine-deficient areas,[6] and is 5 to 8 times more common in women than men. The most frequent cause is chronic autoimmune (Hashimoto) thyroiditis that increases with age. Other common causes are iatrogenic (post-thyroidectomy, post-radioiodine, drug-induced).[35] Less common causes are secondary to pituitary or hypothalamic disorders.

Elderly patients with hypothyroidism differ to younger patients. They lack classic symptoms and have a more insidious onset and atypical presentation. In addition, several signs and symptoms such as fatigue, reduced appetite, cold intolerance, constipation, dry skin, and bradycardia may often be attributed to the process of ageing itself. Hence, a high index of suspicion is required for the diagnosis of hypothyroidism in older adults.

Severe hypothyroidism may mimic depression and dementia, particularly in this age-group. Overt hypothyroidism is associated with cognitive impairment, affecting attention, concentration, memory, language, executive function, as well as perceptual and visuospatial function.[36] These neuropsychiatric symptoms are usually reversible upon restoration of a euthyroid state. Hypothyroidism should also be considered in the presence of persisting hyperlipidaemia (hypertriglyceridaemia), congestive heart failure, and macrocytic anaemia.

The most severe complication of hypothyroidism is myxoedema coma. It occurs almost exclusively in older adults with longstanding primary hypothyroidism, and is usually precipitated by a concomitant medical illness. The hallmarks include localised neurological signs, hypothermia, bradycardia, and hypomania. Patients may develop stupor, seizures, and rapid progression to respiratory depression. Myxoedema coma is a life-threatening clinical state with a mortality as high as 40%.[37]

The treatment of hypothyroidism at all ages is thyroxine replacement. The average replacement dose is 1600 μg/day.[38] However, it is recommended that elderly patients should be given a dose 20-30% lower or 200-400 μg lower than younger adults. This lower requirement may be related to declining metabolic clearance, lower lean body mass, and physiological age–associated reduction in thyroid hormone production.[39]

Several important points should be noted when prescribing thyroxine treatment to elderly patients:

- begin levothyroxine at a lower dose, and augment dose at a lower rate, as thyroid hormone increases myocardial oxygen demand, and may thereby induce angina, myocardial infarction, or cardiac arrhythmia
- start levothyroxine at 25 μg daily, and increase by 12.5-25 μg every 4-6 weeks until TSH returns to normal[40]
- a larger levothyroxine dose may be used in the absence of cardiovascular or mental disease
- in cases of angina, congestive heart failure or mental changes, reduce dose of levothyroxine and gradually increase over several months
- target serum TSH level at 1.0-4.0 mIU/L,[40] as this avoids overtreatment that can trigger atrial fibrillation or increase the risk of fractures
- note that several drugs increase thyroxine clearance rate or decrease intestinal absorption (see earlier section), leading to increased requirements for levothyroxine

42.7 Subclinical Hypothyroidism

Subclinical hypothyroidism is defined as an elevated

Table 42.2 When to treat subclinical hyperthyroidism[30] (reproduced with permission)

Factor (age in years)	TSH (<0.1 mIU/L)	TSH (0.1-0.4 mIU/L)*
>65	Yes	Consider treating
<65 With comorbidities		
Heart disease	Yes	Consider treating
Osteoporosis	Yes	Considering treating
Menopause, not on oestrogens or bisphosphonates	Yes	Consider treating
Hyperthyroid symptoms	Yes	Consider treating
<65 Asymptomatic	Consider treating	Observe

Abbreviation: TSH = thyroid-stimulating hormone
* Where 0.4 mIU/L is the lower limit of the normal range

TSH combined with a normal fT4. The frequency varies from 4% to 10% in different populations, and is more prevalent in iodine-sufficient countries.[14,15] As with hypothyroidism, its frequency increases with age and especially in females. It is estimated that subclinical hypothyroidism may be present in up to 20% of elderly women and 8% of elderly men.

The rate of progression to overt hypothyroidism is approximately 8% per year.[41] The magnitude of TSH elevation is the most powerful predictor of progression. Presence of goitre, clinical symptoms of hypothyroidism, and a low normal fT4 are also related to an increased risk of progression. However, it should be noted that serum TSH level may normalise in around 50% of patients.[42] Serum TSH should be re-measured after 3 to 6 months to exclude a temporary increase in TSH.

There have been conflicting results regarding the association of subclinical hypothyroidism with cardiovascular risk and mortality. Hence, it has been a matter of debate whether or not subclinical hypothyroidism should be treated. In a recent participant-based meta-analysis, an increased risk of coronary heart disease and cardiovascular mortality, but not all-cause mortality, was noted in patients with serum TSH level of >10 mIU/L.[43] Two recent observational studies, looking at subjects aged >73 years and aged >85 years, respectively showed that high TSH and / or low fT4 level were associated with a lower mortality rate.[44,45] Furthermore, age-related subtle thyroid hypofunction has been related to longevity.[46]

Based on the available evidence, the American Association of Clinical Endocrinologists recommends consideration of treatment of patients with serum TSH level of >10 mIU/L.[47] Treatment should also be considered in patients with serum TSH levels of 5-10 mIU/L in the presence of hypothyroid symptoms, positive TPOAb, evidence of atherosclerotic cardiovascular disease or heart failure.[47]

The European Thyroid Association (ETA) tailored the recommendations according to patient age.[48] For patients with serum TSH of ≥10 mIU/L, ETA recommended treating younger patients (<65-70 years) even if asymptomatic, and consideration of treatment in older patients (>70 years) if symptomatic or at high vascular risk. For patients with serum TSH of <10 mIU/L, ETA recommended a trial period of treatment in younger patients (<65-70 years) if symptomatic, and a wait-and-see approach with careful follow-up for older patients (especially >80-85 years).

In a recent multicentre, double-blind, randomised, placebo-controlled, parallel-group trial in Europe involving 737 older patients (mean age 74.4 years, range 65-93 years) with subclinical hypothyroidism, treatment with levothyroxine targeting at a serum TSH of 0.40-4.60 mIU/L provided no symptomatic benefits.[49] However, the trial was underpowered to detect any effect of levothyroxine on the incidence of cardiovascular events or mortality. It cannot address if treatment benefits might occur with a more aggressive target (e.g. serum TSH of 0.40-2.50 mIU/L), in the subgroup with baseline serum TSH of >10 mIU/L, in those with more marked symptoms, and in antibody-positive patients. Also, dementia patients were excluded from the study.

42.8 Thyroid Nodules and Thyroid Carcinomas

The prevalence of thyroid nodules and thyroid cancers increases with age. In iodine-sufficient areas, the prevalence of thyroid nodules detected by ultrasonography at the age of 65 years is 50%.[50] In adults aged 50-70 years, the prevalence of clinically apparent thyroid cancer is estimated to be 0.1%.[50] Although women are 2 to 3 times more often affected than men, the ratio declines with advancing age. Aggressiveness of thyroid cancer, mortality rates, and recurrence rates all increase with age.

In the 2015 ATA guidelines,[51] when TSH is found to be normal or high, fine-needle aspiration (FNA) is suggested in nodules with suspicious ultrasound features of malignancy (solid hypoechoic nodule, irregular margins, microcalcification, taller than wide shape, intranodular vascularity, evidence of extrathyroidal extension) and are ≥1 cm in diameter. For nodules of ≥2 cm without suspicious features, both FNA or observation are reasonable.

Similar to their younger counterparts, papillary thyroid cancer (PTC) is still the most common thyroid cancer in older adults, and its incidence increases disproportionally in those older than 45 years.[52] PTCs are slow growing and patients usually present with an asymptomatic painless neck mass. Mortality is low, but increases in patients older than 60 years due to increased mitotic activity of the tumour, increased likelihood of distant metastasis, and associated mutated *BRAF* gene in advancing age.[53]

Follicular thyroid cancer (FTC) is the second most common after PTC, and has a peak incidence in the sixth decade. An asymptomatic neck mass is the most common presentation. Haematological spread to distant sites (most commonly bone and lung) is more likely and thus results in a worse prognosis than PTC.

Medullary thyroid cancer (MTC), which derives from parafollicular C cells, represents 5% of all thyroid

malignancies. In the older age-group, the sporadic form rather than the hereditary form is more likely, and is associated with a worse prognosis.[54] Patients present with a palpable neck mass with or without symptoms of hormone (calcitonin) hypersecretion (diarrhoea, flushing, and bronchospasm).

Anaplastic thyroid cancer, although rare, is aggressive, and has a peak incidence in the seventh decade of life. It presents as a rapidly growing neck mass, often with widespread local invasion, lymph node involvement, and distant metastasis.

For most cases of PTC and FTC, total thyroidectomy is the treatment modality. Postoperative RAI ablation should be followed in patients with incomplete tumour resection, extrathyroidal extension, vascular invasion, more than five positive lymph nodes, any lymph node of >3 cm, aggressive histology, and iodine-avid distant metastases.[51] Thyroxine-suppression therapy is used as an adjunctive therapy to surgery to reduce the recurrence rate. The target serum TSH levels should be <0.1 mIU/L, 0.1-0.5 mIU/L, and 0.5-2.0 mIU/L in high-risk, intermediate-risk, and low-risk diseases, respectively.[51]

Total thyroidectomy is a treatment option in most cases of MTC. Treatment of anaplastic thyroid cancer is mostly of palliative intent with surgery, radiotherapy or chemotherapy, in view of its aggressive nature and high recurrence rate.

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CHAPTER 43

Gastrointestinal Disorders

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OBJECTIVE

To define age-related changes in the gastrointestinal (GI) tract, discuss common GI problems in elderly people, and discuss the use of percutaneous endoscopic gastrostomy (PEG) feeding.

KEY POINTS

Dysphagia

- Dysphagia is common in elderly patients with neurodegenerative disease.
- Dysphagia with silent aspiration should be considered in elderly patients with recurrent pneumonia.
- Assessment by bedside water swallow test, videofluoroscopic swallowing study or fiberoptic endoscopic evaluation of swallowing may guide further management.
- Tube feeding may be avoided by careful hand feeding using foods of an appropriate consistency.

Gastroesophageal reflux disease (GERD)

- GERD classically presents with heartburn but other presentations include extra-oesophageal symptoms.
- Erosive oesophagitis is found in only one-third of patients with GERD symptoms.
- In frail elderly patients who cannot tolerate endoscopy, a therapeutic trial of proton pump inhibitor (PPI) and close monitoring of symptoms may be the most practical option.

Peptic ulcer disease

- Elderly people are predisposed to peptic ulcers and ulcer complications may result in poor outcomes.
- Reasons for poor outcome include atypical presentation, delayed diagnosis, and comorbidity.
- Prompt endoscopy is indicated for those with alarming features.
- Important causes of peptic ulcers are *Helicobacter pylori* and drugs, such as non-steroidal anti-inflammatory drugs (NSAIDs) and aspirin.
- *H. pylori* eradication markedly reduces the rate of ulcer recurrence.
- High-risk patients prescribed aspirin and NSAID should be given prophylaxis with PPIs.

Chronic diarrhoea

- New-onset diarrhoea with alarming features requires exclusion of serious underlying disease such as malignancy. Diagnosis of irritable bowel syndrome in elderly people is considered only after excluding other causes.
- Medication history is necessary to rule out drug-induced causes. Antibiotic-associated diarrhoea such as *Clostridium difficile* infection should be considered in the presence of recent antibiotic intake.

Diverticular disease

- Diverticulosis is largely asymptomatic but may cause abdominal pain and constipation.
- Consider diverticulitis, perforation, and abscess formation in elderly patients who present with acute abdomen or unexplained fever.

Small bowel bacterial overgrowth

- Bacterial overgrowth in the small bowel is not uncommon in elderly people and presents with weight loss, diarrhoea, and malnutrition in the presence of predisposing conditions.

Hepatobiliary disorders

- Chronic liver disease such as hepatitis B is frequent in elderly people who show good tolerance to antiviral treatment. Non-alcoholic steatohepatitis is a consequence of diabetes and obesity.
- Acute biliary sepsis such as cholangitis and liver abscess present atypically without the classic triad of fever, abdominal pain, and jaundice.

43.1 Introduction

43.1.1 Burden of Gastrointestinal Diseases

Gastrointestinal (GI) problems are common in elderly people. One-third of geriatric patients have GI symptoms, commonly due to dysmotility, peptic ulceration, and carcinoma.[1] Dysmotility problems such as dysphagia, gastroesophageal reflux disease (GERD), and constipation are frequent. Peptic ulcer disease is partly related to widespread use of antiplatelet agents and anticoagulants. Colorectal cancer (CRC) is responsible for the second highest cancer mortality in Hong Kong, predominantly affecting elderly people.[2]

Existing guidelines are based on studies in the young and exclude frail elderly people. Hence, it is important to individualise management, based on a comprehensive geriatric assessment. As the spectrum of elderly people range from the healthy fit to the cognitively and physically frail, management strategies should be individualised. This chapter will discuss practical approaches to common GI problems in elderly people.

43.1.2 Age-related Changes

Age-related changes reduce the functional reserve of elderly people. With comorbidity, the impairments become overt and contribute to the atypical presentation and delayed diagnosis.

There is age-related prolongation of the oropharyngeal swallowing phase and delayed opening of the upper oesophageal sphincter, accentuated in Parkinsonism. Additionally, reduced protective reflexes lead to pulmonary aspiration.

Concomitant diabetes blunts the visceral response to inflammation, due to autonomic and sensory neuropathy. Typically, there is a lack of abdominal guarding or rigidity in peritonitis.

Ageing impairs gastroprotective mucosal function resulting in the propensity for peptic ulceration. Elderly patients have higher incidence of atrophic gastritis, and this may be due to changes in gastric acid production. There is also a gradual decline in protein and fat absorption that may become evident as malnutrition in the face of sickness.

Changes to the commensal intestinal microflora may occur in the older patient and predispose the critically ill elderly patient to *Clostridium difficile*–related pseudomembranous colitis and diarrhoea.

Altered cellular ageing mechanisms predispose to colon carcinoma.

43.2 Common GI Problems in Elderly People

43.2.1 Dysphagia

Dysphagia is common in elderly persons with an increased prevalence of neurodegenerative diseases such as stroke, Parkinson's disease, and dementia. Half of institutionalised demented elderly people have swallowing difficulties due to a slowed swallowing process and oromotor apraxia. These prolong the time needed for meals and assistance to ensure adequate intake, adding to carer burden. Inadequate support for carers leads to initiation of tube feeding, often inappropriately as evidence does not show it can prevent aspiration pneumonia or prolong survival in advanced dementia.

Consequences of dysphagia include aspiration, malnutrition, dehydration, and need for institutionalised care. Dysphagia with silent aspiration should always be considered in elderly patients with recurrent pneumonia. The triad of dysphagia, malnutrition, and pneumonia is seen in advanced dementia and other neurodegenerative diseases. Dysphagia is classified into two types.[3]

- Oropharyngeal dysphagia is due to impaired transfer of food from mouth to oesophagus. It occurs in half of all nursing home residents, resulting in a high frequency of aspiration pneumonia.
- Oesophageal dysphagia is due to impaired transfer of food through the oesophagus into the stomach. In elderly people, it is often referred to as "presbyoesophagus" resulting from diminished peristaltic amplitude and secondary peristaltic contractions. It is associated with motility disorders, mechanical obstruction, and medications.

Medications can act centrally or peripherally to impair the swallowing mechanism. Drugs that increase sedation (e.g. sedatives), with extrapyramidal side-effects (e.g. neuroleptics) can precipitate dysphagia. Potassium syrup, bisphosphonates, ferrous sulphate, and non-steroidal anti-inflammatory drugs (NSAIDs) can cause local oesophageal mucosa damage.

43.2.1.1 Clinical Features

A detailed history from an elderly patient and caregiver will lead to a diagnosis in the majority of cases. Bedside assessment takes note of the level of consciousness, neck posture, cognition, speech, and cough. A water swallow trial provides useful information.

Symptoms of oropharyngeal dysphagia include food sticking in the throat, difficulty initiating a swallow, nasal regurgitation, and coughing or choking during

swallowing. In acute-onset dysphagia, a stroke must be excluded. History of stroke and nasopharyngeal carcinoma are relevant.

For oesophageal dysphagia, symptoms include food or liquid sticking in the chest, or chest pain. In motility disorders, there is difficulty in swallowing both solids and liquids whereas in mechanical obstruction such as oesophageal carcinoma, there is difficulty with solids initially.

Signs of primary disease (ear, nose and throat and neurological problems) or aspiration may be present.

43.2.1.2 Investigations

A bedside water swallow test of swallowing 30-90 ml of water while in an upright position, observing for coughing, drooling, and dysphonia is commonly performed to detect oropharyngeal dysphagia. Further evaluation with fiberoptic endoscopic evaluation of swallowing (FEES) and videofluoroscopic swallowing study (VFSS) can be performed, usually after assessment by a speech therapist. If local causes are suspected, then a computed tomographic (CT) scan or oesophagogastroduodenoscopy is indicated.

VFSS is a modified barium swallow, using different consistencies of foods or liquids, which is videotaped to evaluate swallow delay, residue, and aspiration.

FEES involves passing a thin endoscope with transnasal approach to directly visualise the larynx and pharynx during swallowing of different liquid and meal consistencies and effectiveness of compensatory measures. Compared with VFSS, it takes less time, does not need imaging, and can be performed at the bedside. This is preferred for a frail elderly patient but has limitations since the oesophagus cannot be viewed.

43.2.1.3 Management

Treatment depends on the cause. Obstructive causes such as carcinoma and strictures require endoscopic treatment or surgery. Neurodegenerative causes are managed by swallowing rehabilitation and compensatory strategies by speech therapists. These include modifying food and liquid consistency, and positioning or changing the mode of feeding to prevent aspiration and achieve adequate nutritional intake. Thickened fluids may be unpalatable for some elderly patients so it is important to monitor intake and hydration.

There are often complex decisions to be made about feeding in the care of terminally ill patients. Promoting advance care planning at an early stage can help ensure the patient's best interests are met.

43.2.2 GERD

GERD is defined by symptoms with or without endoscopic evidence of oesophagitis caused by reflux of the gastric contents into the oesophagus. Its prevalence increases with age. Age-related impaired clearance of refluxed acid is due to decreased lower oesophageal sphincter pressure and reduced secondary peristaltic waves. Comorbid disease, presence of a hiatus hernia, and medications contribute to GERD in elderly patients.

43.2.2.1 Clinical Features

GERD classically presents with heartburn but other presentations include extra-oesophageal symptoms such as chronic cough, asthma, sore throat, hoarseness, and chest pain. Acid reflux causes laryngitis and reflex bronchoconstriction (Table 43.1[4]). Red flags such as weight loss, dysphagia, and anaemia indicate an urgent need for further investigations.

43.2.2.2 Investigations

Upper GI endoscopy can help exclude malignancy and stricture formation. It can detect erosive oesophagitis, present in some patients with GERD symptoms. In a frail elderly patient who cannot tolerate endoscopy, a therapeutic trial of proton pump inhibitor (PPI) for symptomatic relief may be the most practical option.

43.2.2.3 Management

The goals are to control symptoms, heal erosive oesophagitis, and prevent complications. Most patients have a recurrent but non-progressive form of GERD. Less frequently, progressive disease may result in severe complications, such as strictures or Barrett's oesophagus.

Measures consist of lifestyle modifications such as weight loss for the obese, waiting 3 hours after meals before lying down, elevating the head of the bed in those who are bedbound or at night, and avoiding large meals. Treatment with PPIs using a once-daily dose before breakfast for 8 weeks is effective for symptomatic relief and healing of oesophagitis. H_2 receptor antagonists and antacids are less effective alternatives. Anti-reflux surgery is rarely performed in elderly patients.

Table 43.1 GERD symptoms[4]

Typical	Atypical (extra-oesophageal)
• Heartburn • Belching • Epigastric pain • Worse post-prandial • Worse supine • Relieved by antacids	• Chronic cough, shortness of breath • Hoarse voice • Non-cardiac chest pain • Dental erosions

Maintenance PPI therapy is indicated for severe complicated oesophagitis and when relapses are frequent. Whenever possible, the lowest effective PPI dose, 'on demand' or as intermittent therapy is used. Although PPIs are well tolerated with <2% of users complaining of headache and diarrhoea, there have been reported associations of long-term use of PPI with the following conditions.[5]

- Osteoporosis and hip fractures
- Vitamin B_{12} deficiency in institutionalised elderly people
- Rebound acid hyper-secretion
- Interactions with clopidogrel
- Sepsis (enteric infections, food-borne bacterial infections, *C. difficile* colitis, spontaneous bacterial peritonitis in cirrhotics, pneumonia)

43.2.3 Peptic Ulcer Disease

43.2.3.1 Aetiology

The pathogenesis of a peptic ulcer involves acid and pepsin secretion, and mucosal defence mechanisms. *Helicobacter pylori* and NSAIDs, both of which are common in elderly people, disrupt mucosal defences, and are the two major causes of peptic ulcers. Predisposing factors include alcohol, tobacco, and physiological stress such as trauma, sepsis, surgery, or intensive care unit admission.

43.2.3.2 Peptic Ulcers in Elderly People

Age-associated changes, such as decreased gastric mucosal prostaglandins, result in reduced mucosal protection and predispose elderly people to development of gastric ulcers as a consequence of NSAID prescription. Elderly people are also more prone to ulcer complications, such as GI bleeding, perforation, and gastric outlet obstruction. The complication rate is 50% with a mortality rate of 30%.[6] Poor outcome is due to atypical presentations (Table 43.2[7,8]), delayed diagnosis, and the frequent presence of comorbid conditions.

43.2.3.3 Investigations

Investigations include a complete blood count, faecal occult blood testing (FOBT), and endoscopy. Endoscopy is the diagnostic modality of choice with proven safety and tolerability in the older population[9] whereas barium meal has lesser sensitivity and a false-negative rate of 8%.[10] Alarm features for which prompt endoscopy is indicated in elderly patients are malaena, significant weight loss of >10% of body weight, anaemia, and dysphagia.[11]

Table 43.2 Clinical features of geriatric peptic ulcers[7,8]

Atypical presentations
• Abdominal pain frequently absent, especially if on non-steroidal anti-inflammatory drugs
• May mimic angina
• Anorexia, weight loss, and vomiting may be the only presentation
Complications
• High complication rate compared to the young
• Index manifestation may be with complications (bleeding, perforation, or obstruction)
• Ulcer bleeding may initially present as cardiac or cerebral ischaemia
Malignancy must be excluded

43.2.3.4 Treatment

Management consists of *H. pylori* eradication, ulcer healing drugs, stopping NSAIDs and aspirin if possible, and repeating endoscopy until gastric ulcers are healed.

- Eradicate *H. pylori* — In uncomplicated small duodenal ulcers, *H. pylori* eradication therapy need not be followed by anti-secretory treatment.[11] Eradication of *H. pylori* heals ulcers and prevents recurrence. Hong Kong has a relatively low prevalence of clarithromycin resistance so clarithromycin-based triple therapy remains the preferred therapy in clinical practice.[12]
- Lifestyle factors — Alcohol, NSAIDs and aspirin, and tobacco should be avoided.
- Acid control measures — H_2 blockers and PPIs are all effective in treating peptic ulcers when given for 6-8 weeks. H_2 blocker healing rates for duodenal ulcer and gastric ulcer are satisfactory at duration of 8 weeks compared to 4 weeks. However, PPIs remain superior to H_2 blockers.
- Caveats — Reduce famotidine dose by 50% if creatinine clearance is <50 ml/min.[13] For nasogastric (NG) feeding, use esomeprazole and lansoprazole for similar bioavailability as oral dosing.[14,15]
- Caveats — Recent therapy with H_2 receptor antagonists or a PPI can cause false-negative biopsy urease test by suppressing the organism. Therapy should be stopped for 2 weeks in advance of testing.

Aspirin and NSAID-induced Peptic Ulcers

- Low-dose aspirin and other antiplatelet drugs lead to a beneficial reduction in cardiovascular mortality. However, aspirin doubles the risk of a major upper GI bleed.
- NSAIDs are widely prescribed drugs. They inhibit gastric synthesis of prostaglandins, necessary for protective gastric mucus and bicarbonate production. The GI-event–related hospitalisation rate in institutionalised elderly people increases 3-fold.[16] NSAIDs cause a 3-4 fold increase in peptic ulcers, triple the risk of bleeding, and increase perforation 6-fold.[17]
- The first month of treatment is the most dangerous period. Intermittent users of NSAIDs are not spared from the adverse effects.[18]
- Many aspirin and NSAID-related ulcers are

painless.[19] Because they are potent analgesics, there is poor correlation of dyspeptic symptoms with ulcers.
- Once peptic ulcer has developed, treatment consists of stopping NSAIDs and treating with H_2 receptor antagonists or PPI.
- If a NSAID must be continued, co-prescription with PPIs is indicated. In this case, use the lowest possible dose of NSAID as ulcerogenic potential is dose-related.[20]
- Additionally, select NSAIDs with less GI side-effects, e.g. ibuprofen, etodolac[20]
- Consider selective COX-2 inhibitors
- Primary prophylaxis using concomitant PPIs can prevent ulcer complications in high-risk elderly patients prescribed NSAIDs as well as aspirin.[21]

43.2.4 Chronic Diarrhoea

Diarrhoea is defined by the World Health Organization as three or more unformed bowel movements within 24 hours.[22] New-onset diarrhoea that is stained with blood and mucus and associated with weight loss warrants exclusion of colonic malignancy. A careful drug history is needed to elicit use of antibiotics, metformin, cholinesterase inhibitors, serotonin reuptake inhibitors, and digoxin.

Chronic diarrhoea may cause social isolation and reduced activity outside. For chronic diarrhoea where initial tests are negative, it is important to consider systemic disease such as diabetes, thyrotoxicosis, inflammatory bowel disorders, malabsorptive conditions, small bowel bacterial overgrowth and, importantly, drugs. The cause is often multifactorial.

Although the diagnosis of irritable bowel syndrome should not be made in elderly patients without first excluding other underlying causes, it is a common disorder. Decreased rectal compliance occurs with ageing and may contribute to sensations of faecal urgency in these patients. Other symptoms include abdominal pain, cramping, distention, and altered stool consistency (loose or hard). Treatment guidelines for younger patients are used in the geriatric population, because there are little data to guide treatment in this older cohort.

43.2.4.1 Drug-induced Diarrhoea

Diarrhoea accounts for 7% of all drug adverse effects. Antibiotics account for 25% of drug-induced diarrhoea.[19] Antibiotic-associated diarrhoea (AAD) refers to otherwise unexplained diarrhoea associated with the administration of broad-spectrum antibiotics such as amoxicillin-clavulanate (Augmentin). Most cases are self-limiting upon discontinuation of the antibiotic. Onset of diarrhoea may occur after 5-10 days of antibiotics. The clinical features of AAD range from potentially serious colitis to uncomplicated 'nuisance diarrhoea'.

C. difficile diarrhoea is most prevalent in elderly people, related to frequent antibiotic use and hospitalisations. Presentation varies but includes asymptomatic colonisation, severe diarrhoea, pseudomembranous colitis, and toxic megacolon. It accounts for 20% of AAD. Broad-spectrum antibiotics such as the cephalosporins, clindamycin, and amoxicillin-clavulanate disrupt the normal gut flora, leading to permissive growth of *C. difficile*, probably acquired from a nosocomial source. *C. difficile* itself is noninvasive. Its pathogenesis is linked to toxins A and B — toxin A is a potent enterotoxin primarily responsible for the clinical symptoms associated with *C. difficile* infection, whereas toxin B forms the basis of the cytotoxin stool assay but has less of a pathogenic role than toxin A.

The mainstay of management is supportive after stopping the culprit antibiotic. One-quarter of patients improve within 24-48 hours, and diarrhoea resolves after 7-10 days. Fluids and electrolytes should be replenished. Conservative treatment suffices in most cases. Antibiotic treatment should be oral, since *C. difficile* is restricted to the lumen of the colon. Oral vancomycin and metronidazole lead to resolution of fever within 1 day and diarrhoea in 4-5 days.[23]

If patients cannot take oral antibiotics, the options are intravenous metronidazole 500 mg every 8 hours, vancomycin (500 mg every 6 hours) by NG tube or vancomycin (500 mg every 4 hours) retention enemas.[23]

The relapse rate is 20%, indicated by symptom recurrence that occurs around 3-21 days after discontinuation of treatment. Half of the patients will respond to another course of therapy.

Prevention is the most important strategy with avoidance of unnecessary antibiotics and infection control measures such as hand washing to limit spread.

43.2.5 Diverticular Disease

Colonic diverticulosis affects up to 50% of those older than 60 years.[24] Increased intra-abdominal pressure related to a low-fibre diet is linked to diverticular disease. Most elderly people are asymptomatic but complications include diverticulitis, perforation, abscess, and lower GI bleeding. Diverticulitis presents with abdominal pain, fever, raised white cell count, and typically with constipation. Chronic manifestations can include chronic pain and irritable bowel syndrome–like presentations. CT (contrast) scan of the abdomen or pelvis is the gold standard for

diagnosis of acute diverticulitis. Management is with broad-spectrum antibiotics such as ciprofloxacin and metronidazole in milder cases.

43.2.6 Small Bowel Bacterial Overgrowth

Bacterial overgrowth in the small bowel is much more prevalent in older community-dwelling adults than in younger subjects. This is a syndrome of weight loss, diarrhoea, and malnutrition as a consequence of malabsorption of macronutrients such as fat, carbohydrate, and micronutrients. Bacteria digest carbohydrates leading to fermentation and deconjugated bile acids with consequent malabsorption of fat and fat soluble vitamins A, D, and K. In addition, bacteria utilise vitamin B_{12}.

Presentation is non-specific with weight loss, malaise, abdominal pain, bloating, and anaemia. There is B_{12} deficiency anaemia, hypocalcaemia and osteomalacia due to vitamin D deficiency, with high serum folate levels.

Small bowel bacterial overgrowth should be considered a cause of malnutrition in the presence of risk factors. These include small bowel diverticulosis, strictures, surgical blind loops, and reduced motility disorders such as scleroderma, autonomic neuropathy, intestinal pseudo-obstruction, or medication-induced. Diagnosis can be obtained by a small bowel aspirate for bacterial counts or bile acid breath tests. Treatment of bacterial overgrowth improves diarrhoea and promotes weight gain.

43.2.7 Hepatobiliary Disorders

Liver disease is increasing in older people, in part due to improved management of conditions such as chronic hepatitis. There are no chronic liver diseases specific to elderly people. Antivirals for hepatitis B are well tolerated and management is similar to that for younger age-groups, although with implications for polypharmacy and increased drug interactions. As the population ages and becomes more obese, the number of geriatric patients referred for complications of obesity and diabetes will increase significantly. These include liver dysfunction due to nonalcoholic steatohepatitis.

The prevalence of cholelithiasis increases with age. Bile seems to become increasingly lithogenic as a function of ageing, with precipitation of supersaturated bile and concomitant crystallisation of cholesterol or calcium bilirubinate. Acute biliary sepsis such as cholangitis and liver abscess is prevalent in elderly people. Atypical presentation without the classic triad of fever, abdominal pain, and jaundice is common. The presentation may be subtle with anorexia, vomiting, and low-grade fever, with altered liver biochemistry providing the only clue for further tests such as ultrasonography or CT scan of the hepatobiliary system.

43.2.8 Screening for Colon Cancer

The incidence of CRC rises markedly with age, regardless of sex and racial background. Elderly people constitute the greatest proportion of newly diagnosed CRC in the US, with 24.4% occurring in persons aged 64-74 years, 26.8% in those aged 75-84 years, and 12.2% in those aged ≥85 years.[25]

The US Preventive Services Task Force recommends CRC screening starting at the age of 50 years and continuing until age 75 years.[26] For patients aged 76-85 years, CRC screening should be an individualised decision, taking into account the patient's overall health and his / her screening history. The Task Force does not recommend routine screening for CRC for persons aged ≥86 years. The frequency recommended for CRC screening is FOBT every year, colonoscopy every 10 years, or flexible sigmoidoscopy every 5 years.[26]

Elderly patients have more comorbid medical conditions, potential cognitive impairment resulting in difficulty obtaining informed consent, shorter life expectancy compared with younger patients, and potentially more risks for adverse events. While the US Preventative Services Task Force recommends an age at which CRC screening should no longer be offered, most agree that the health status, life expectancy, and functional status should be considered in deciding when to discontinue screening. The American Geriatrics Society does not recommend CRC screening in those unlikely to live more than 5 years or who have significant comorbidity that would preclude treatment.[27]

FOBT is the most widely used screening modality. A positive test is followed by a colonoscopy and has resulted in 15-33% reductions in CRC mortality.[28,29] It is worth noting that guaiac-based FOBT may have false-positive results as it is affected by red meat intake. Immunochemical-based FOBT is more specific but less sensitive than guaiac-based FOBT.

Colonoscopy is advocated as a 'preferred' screening method by some groups as elderly patients have more right-sided carcinomas. Non-polypoidal (flat or depressed) lesions and colorectal neoplasms that arise without preceding adenoma (de-novo cancers) are more common in Asian than in other populations.

Previous studies of colonoscopy have consistently shown a high prevalence of colorectal neoplasia. Although elderly people have lower caecal intubation rates (approximately 85%), longer procedure time, higher perforation risk and suboptimal bowel

preparation, colonoscopic complication rates remain low (about 0.1-0.5%). Nonetheless the complications are greater in octogenarians than in those younger than 80 years.[26]

The role of CT colonography is uncertain, given its lower sensitivity for detecting small polyps of <1 cm in size. No study has demonstrated the efficacy of CRC in reducing CRC incidence and mortality, although given its less-invasive nature, it may be considered as an adjunctive screening modality.

43.2.9 Percutaneous Endoscopic Gastrostomy Feeding

Percutaneous endoscopic gastrostomy (PEG) has been increasingly used since the 1980s as an alternative to NG feeding to provide long-term enteral nutrition. It is safe and well tolerated by elderly patients.[30] The typical PEG patient is frail and dependent. Half are bedridden with neurodegenerative conditions such as stroke, motor neuron disease (MND), and advanced Parkinson's disease.[31] Careful selection of patients who will benefit from this procedure is through close communication with patients and carers by a multidisciplinary team involving geriatricians, gastroenterologists, and speech therapists to align the aims of the procedure and its context in a wider advance care plan, particularly for those with end-stage neurodegenerative disease.

43.2.9.1 The Procedure

The most commonly used 'pull-on-string' method takes 20 minutes and involves two operators. The patient is sedated after fasting for >8 hours. A prophylactic antibiotic (e.g. intravenous amoxicillin / clavulanate or second- / third-generation cephalosporins)[32] is given before the procedure. Patients should also rinse their mouth with an antibacterial solution.

The endoscope is inserted into the stomach and a suitable site (preferably in the left upper quadrant, at least 2 cm below the costal margin) is selected by external indentation of the finger and transillumination. Within the stomach, the gastrostomy site should be in the middle to distal third of the corpus. The assistant makes a 1-cm skin incision through which a cannula and a guidewire is passed percutaneously. The endoscopist snares the guidewire and removes this together with the endoscope through the mouth. The 'mouth end' of the guidewire is attached to the PEG tube. The assistant pulls on the 'abdominal end' of the guidewire so that the PEG tube is passed from the mouth, down into the stomach, and out through the abdominal wall. An inner bumper or balloon retains the PEG tube in the gastric lumen. Gentle traction is applied on the tube and external fixation secured.[33]

43.2.9.2 Choosing between PEG and NG Feeding

Common indications for PEG feeding are similar to those for NG feeding, with a predominance of neurodegenerative conditions such as stroke, advanced dementia, parkinsonism, and MND.

In Hong Kong, NG feeding is far more prevalent than PEG feeding, as the PEG insertion procedure is considered invasive and consent for the procedure is not obtainable from mentally incompetent patients with severe dementia or cerebrovascular accident. The main reason for selecting PEG is its better cosmetic appearance, less frequent need to change the tube, and perceived less discomfort compared with a NG tube. Neither PEG nor a NG tube prevent aspiration pneumonia. PEG tubes have a 1% procedure-related mortality rate and 3-13% complication rate with skin infection, leakage, and tube migration and fistula formation.[34-37]

It has been reported that 38% of elderly people survive 1 year after PEG.[38] Most deaths are due to underlying comorbidity. For the demented patient, PEG feeding does not prolong life or improve comfort but for patients with MND or stroke, it improves nutritional indices and functional outcomes.

Removal of a PEG tube can be percutaneous or endoscopic, depending on the type of tube. Percutaneous removal is used if the internal fixation device is a balloon that can be deflated externally. Complications after PEG included granuloma formation at the PEG exit site (Plate 43.1) and the usual treatment is by silver nitrite pen application.

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CHAPTER 44

Pressure Ulcers

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OBJECTIVE

To learn the pathophysiology, prevention, and management of pressure ulcers.

KEY POINTS

- Pressure ulcers are common in frail older patients and are a cause of substantial morbidity and mortality in the sufferers as well as burden to the healthcare system.
- These commonly occur over bony prominences such as the sacrum, buttock, and heels.
- Pathomechanical aetiological factors include prolonged pressure, shear forces, friction, and excessive moisture.
- Identifying high-risk patients for preventing development of pressure ulcers is of utmost importance for preventing associated morbidity and mortality.
- Pressure ulcers are slow and difficult to heal and need meticulous nursing care.
- Management of pressure ulcers is an important component in 'end-of-life care'.

44.1 Introduction

There are many different names to describe pressure ulcer, including bed sore, pressure sore, and decubitus ulcer. Recently "pressure injury" replaces "pressure ulcer" in the National Pressure Ulcer Advisory Panel Pressure Injury Staging System. The change in terminology more accurately describes pressure injuries to both intact and ulcerated skin.

A pressure injury is localised damage to the skin and / or underlying soft tissue usually over a bony prominence or related to a medical or other device. The injury can present as intact skin or an open ulcer and may be painful. The injury occurs as a result of intense and / or prolonged pressure or pressure in combination with shear. The tolerance of soft tissue for pressure and shear may also be affected by microclimate, nutrition, perfusion, comorbidities, and condition of the soft tissue.[1]

Pressure ulcers are common in immobilised older patients. It leads to much suffering in patients as well as increased consumption of nursing care services and medical resources. Thus, it is important to prevent the occurrence of pressure ulcers.

44.2 Staging of Pressure Ulcers

Pressure injury can be staged according to the National Pressure Ulcer Advisory Panel.[1]

- Stage 1 Pressure Injury: Non-blanchable erythema of intact skin
- Stage 2 Pressure Injury: Partial-thickness skin loss with exposed dermis
- Stage 3 Pressure Injury: Full-thickness skin loss
- Stage 4 Pressure Injury: Full-thickness skin and tissue loss
- Unstageable Pressure Injury: Obscured full-thickness skin and tissue loss (Plate 44.1)
- Deep Tissue Pressure Injury: Persistent non-blanchable deep red, maroon or purple discolouration

44.3 Epidemiology

According to international prevalence audits (n=447 930 patients), the prevalence of pressure ulcer was 9.2-27.3% during 2008-2009.[2] The incidence of hospital-acquired pressure ulcer under Hospital Authority of Hong Kong is about 0.45 per 1000 bed days. Reports from Hong Kong suggest that 8.9% of older patients admitted to a geriatric extended care hospital have a pre-existing pressure ulcer.[3] Another local study found that the prevalence of pressure ulcers was 5.4% in acute wards and 21.5% in subacute wards; 19% and 38% of patients in the respective settings had a high risk of developing pressure ulcer.[4] In local residential care home for the elderly setting, 17% of residents with advanced dementia had a pressure ulcer.[5]

44.4 Sites of Pressure Ulcers

Pressure ulcers commonly occur over bony prominences where external pressure is high. These sites include the ischium (28%), the sacrum (17-27%), the trochanter (12-19%), and the heel (9-18%).[6]

44.5 Morbidity and Mortality

Patients with pressure ulcers are at an increased risk of morbidity and mortality. Infection is the most common major complication of pressure ulcers. Aerobic pathogens are commonly present in all pressure ulcers, whereas anaerobes tend to be present more often in larger wounds (65% in grade III and above). Development of pressure ulcers has been associated with a 4.5-fold greater risk of death compared with that in persons without pressure ulcers.[7]

44.6 Pathomechanical Factors (Extrinsic or Primary)

44.6.1 Prolonged Pressure

Pressure ulcers arise from prolonged tissue ischaemia caused by pressure that exceeds the tissue capillary pressure. Prolonged pressure deprives tissues of oxygen and essential nutrients, which then leads to necrosis and ulceration. Interface pressure, defined as "perpendicular force per unit area between the body and support surface", is affected by the stiffness and composition of the body tissue and by the geometric shape of the body being supported. While interface pressure of <32 mm Hg is assumed by many clinicians to be safe, pressure of ≥32 mm Hg is believed to lead to closure of capillary beds and tissue ischaemia.[8] However, these speculations remain to be challenged. The duration of pressure also plays an important role in tissue injury and Kosiak[9] found that intense pressure for a short duration was as injurious to tissues as lower pressure applied for longer periods of time.

44.6.2 Shear

Shear is mechanical stress directed parallel to the plane of interest. When patients are in the semi-upright position in bed or sliding in chair, shear force tends to distort the tissues and blood vessels near the sacrum, placing this region at risk for tissue breakdown. Defloor and De Schuijmer[10] concluded that elevation of the top end of the operating table to 30 degrees might cause high pressure and shearing force on the sacrum during head and neck surgery.

44.6.3 Friction

Friction is the force between two surfaces moving across one another. Friction and the increased drag coefficient that occurs while moving patients across bed sheets and other support surfaces can cause microscopic or macroscopic tissue trauma.

44.6.4 Moisture and Maceration

Moisture increases the surface tension of the skin and the support surface. Moisture from sensible fluid loss or incontinence leads to maceration of tissues, which in turn makes the tissues more susceptible to pressure, shear, and friction damage.

44.7 Pathophysiological Factors (Intrinsic or Secondary)

Pathophysiological factors that predispose to formation of pressure ulcers include fever, anaemia, infection, ischaemia, hypoxaemia, hypotension, malnutrition, spinal cord injury, neurological disease, decreased lean body mass, and increased metabolic demands. Nutrition and correction of anaemia are important factors in the healing and prevention of pressure ulcers.

44.8 Prevention of Pressure Ulcers

Pressure ulcers are better prevented than treated. It has been shown that early assessment and preventive measures using a multidisciplinary team approach is effective in reducing the incidence of pressure ulcers in acute hospital settings. Prevention strategy starts with identification of high-risk patients for prevention and early detection of skin breakdown to prevent further deterioration.

44.8.1 Risk assessment

Every newly admitted patient should be assessed by a validated risk assessment tool. The assessment should be repeated periodically (usually weekly) in the hospital setting. Both the Norton Score[11] and the Braden Scale[12] are commonly used. Once these high-risk patients are identified, a standardised preventive protocol should be implemented immediately.[1]

The Norton Score[11] can be found by adding up the score of each parameter: general condition, mental condition, activity level, mobility level, and status of incontinence. With a total score of 20, a score of ≤12 means very high risk, and 13-14 means high risk.

The Braden Scale[12] can be found by adding up the score of each parameter: sensory perception, moisture exposure, activity level, mobility level, nutrition status, and effect of friction and shear. With a total score of 23, a score of ≤9 means very high risk, and 10-12 means high risk.

44.8.2 Pressure and Shear

The key for prevention and management of pressure ulcers is reducing the forces of pressure and shear that damage skin in the deep tissue compartments, particularly subcutaneous fat and muscle. To

minimise shear, do not raise the head of the bed to >30 degrees and avoid slipping or sliding with transfers or in various types of seating.

Recommended positioning and arrangement of support surfaces when laying at-risk individuals.[13]
- Re-position bed-bound persons every 2 hours or at least every 4 hours
- Use a pressure-relieving mattress for patients who are able to reposition themselves
- Use a pressure redistribution mattress for patients who are not able to reposition themselves
- Use devices to eliminate pressure on the heels and other bony prominences
- Avoid direct positioning on the trochanter; use the 30-degree lateral inclined position
- Avoid elevation of the bed above 30 degrees as far as possible
- Use lifting / transfer devices to minimise shear and friction during transfer

Recommended positioning and arrangement of support surfaces when seating at-risk individuals.[13]
- Consider postural alignment, distribution of weight, balance and stability, and pressure redistribution when positioning persons in chair or wheelchair
- Teach chair-bound persons to shift weight every 15 minutes. Reposition chair-bound persons every 2 hours if they fail to do so
- Use a pressure-relieving chair cushion
- Avoid using donut-type devices for pressure redistribution
- Refer high-risk patients to occupational therapist for proper sitting device

44.8.3 Nutrition

The nutritional assessment should include measuring the body mass index, and haemoglobin and serum albumin levels. Nutritional support should include adequate intake of energy (30-35 kcal/kg/day), protein (1.25-1.5 g/kg/day), vitamins, and minerals. Zinc deficiency is not uncommon in malnourished older adults and additional supplementation is needed if deficiency is detected.[1]

44.8.4 Moisture

Excess moisture may occur due to sweat but is more often associated with urinary or faecal incontinence. Faecal incontinence in the sacral area is very harmful, and a bowel routine or external collection device should be considered, as well as prompt changing of wet underwear or diapers. Urinary incontinence may be controlled with intermittent catheterisation, a condom catheter, or an indwelling catheter; however, their use is associated with other complications such as infections.[1]

44.8.5 Skin Care

Judicious skin care is important and includes regular bathing with a mild cleansing agent, regular use of moisturisers for dry skin, and avoidance of hot water, excessive rubbing, and massage over the bony prominence.[1]

44.9 Treatment of Pressure Ulcers

Apart from local wound management, holistic assessment, and treatment to address the causes of immobility, continence status, nutrition, and hydration state will benefit patient management.[1]

44.9.1 Wound Assessment

The wound should be assessed at least weekly and documented in a standard wound chart. The assessment should include noting the location, size, stage, presence of undermining, wound bed tissue, amount of exudate, odour, and condition of the surrounding skin and wound edge. A decrease in wound size, exudate, amount of necrotic tissue and improved appearance of granulation tissue as well as the surrounding skin are signs of improvement in the pressure ulcer.

44.9.2 Local Wound Treatment

The aim of local wound treatment is to remove bacteria, clear non-viable tissue, absorb excessive wound exudates, protect peri-wound skin, and provide a moist environment for the growth of granulation tissue to facilitate healing. It includes wound cleansing, debridement, and dressing.

44.9.2.1 Wound Cleansing

The major purpose is to decrease its bioburden. Do not use skin cleansers or antiseptic agents to clean the wound as healthy tissue will be destroyed by their use. Normal saline or drinking water can be used when no germicidal action is required. Consider using cleansing solutions with surfactants and / or antimicrobials to clean pressure ulcers that are filled with debris, confirmed infection, suspected infection, or suspected high levels of bacterial colonisation.

44.9.2.2 Wound Debridement

Removal of necrotic tissue is a vital step in reducing the bacterial concentration of the wound and facilitating healing. However, do not debride stable, hard, dry eschar in ischaemic limbs.

Non-mechanical debridement is preferred when there is no urgent clinical need for drainage or removal of necrotic tissue. Enzymatic debridement entails using proteolytic enzymes that act by attacking collagen and liquefying necrotic wound debris without damaging granulation tissue. Autolytic debridement

entails using the body's enzymes to degrade dead tissues by placing an occlusive dressing.

Mechanical debridement is quick but bears the risk of damaging underlying healthy tissues and incomplete debridement. Mechanical non-selective debridement, in which necrotic tissue is loosened and removed, is generally accomplished by whirlpool treatments, forceful irrigation, or use of wet-to-dry dressings. Selective sharp debridement includes surgical removal of the eschar and any devitalised tissue. It is done in cases with advancing cellulitis, crepitus, fluctuance, and / or sepsis secondary to ulcer-related infection. It is contraindicated in patients who cannot withstand the loss of blood. A great deal of clinical skill and judgement are needed for performing surgical debridement.

44.9.2.3 Wound Dressing

Wound dressing is a central component of pressure ulcer care. Selection of the dressing should be determined by the tissue in the ulcer bed, condition of the skin around the ulcer bed, and the goals of the person with the ulcer. Generally, maintaining a moist ulcer bed is ideal when the ulcer bed is clean and granulating to promote healing or closure. Several moisture-retentive dressings are available. However, selection of dressing may change over time as the ulcer heals or deteriorates.

44.10 Infection

Bacteria are present on all skin surfaces and wound surfaces. When the bacteria (by number or virulence) cause damage to the body, infection is said to be present. The number of bacteria and their effect on the host can be categorised as contamination, colonisation, critical colonisation, or infection. Infection is uncommon in stage I or II ulcers, and assessment of infection should focus on stage III and IV ulcers. Infection may spread beyond the pressure ulcer, resulting in serious systemic infections.[1]

44.10.1 Local Infection

This should be suspected if there are no signs of healing for 2 weeks, or when there is presence of friable granulation tissue, foul odour, increased pain in the ulcer, increased heat in the tissue around the ulcer, increased drainage from the wound, an ominous change in the nature of the wound drainage, increased necrotic tissue in the wound bed, pocketing, or bridging. Surface swabs should not be used for diagnosis as this will only detect the colonising organism. Tissue biopsy or quantitative swab technique can be used to reflect deeper tissue infection. Tissue biopsy or quantitative swab technique are seldom required in clinical practice, however, as wound infection is usually diagnosed clinically without further need for bacteriological diagnosis.

44.10.2 Treatment of Local Infection

It includes cleansing and debridement to remove bacteria, debris, slough, and biofilms. Biofilms tend to redevelop after debris is removed. Antimicrobials may help slow the rate of biofilm redevelopment. Silver, iodine (cadexomer iodine or povidone iodine), or medical-grade honey dressings can be considered. Topical antiseptics can be used for pressure ulcers that are not expected to heal and are critically colonised. They should be properly diluted and used for a limited time period to reduce the risks of toxicity and adverse reactions. Topical antibiotics are generally not recommended for pressure ulcers. However, short courses of topical metronidazole can be useful in the treatment of malodour in fungating wounds or wounds with anaerobic infection.

44.10.3 Systemic infection

This should be suspected in individuals with clinical evidence of systemic infection such as positive blood cultures, cellulitis, fasciitis, osteomyelitis, systemic inflammatory response syndrome, or sepsis. Broad-spectrum systemic antibiotics should be used for treating these cases.

44.11 Adjunctive Therapies

44.11.1 Biophysical Energy

Several forms of energy have been studied in the management of pressure ulcers. These include infrared (thermal) radiation, ultraviolet light (invisible light), laser (coherent and monochromatic light), and acoustic energy (ultrasound). The efficacy of these agents needs further examination in future randomised controlled trials.[1]

44.11.2 Negative-pressure Wound Therapy

It can be considered as an early adjuvant for the treatment of stage III and IV pressure ulcers. The pressure ulcer should be debrided of necrotic tissue prior to the use of negative-pressure wound therapy. It enhances wound healing by reducing oedema, increasing the rate of granulation tissue formation, and stimulating circulation.[1]

44.12 Surgical Treatment

Plastic surgical treatment for pressure ulcers includes skin grafts, skin flaps, and musculocutaneous flaps. However, successful surgery requires complete control of sepsis such as wound infection prior to the operation. Any ongoing septic process may cause future wound infection. Bowel and urinary continence control is important both before and after

surgery. It is not surprising to see that many older patients are not suitable candidates for the plastic surgical treatment.[1]

44.13 End-of-life Care

Pressure ulcers are common in frail, immobilised older patients in the last stage of their lives. It is difficult for their pressure ulcers to heal as these patients are ill, with negative protein balance, or inadequate blood supply. We need to balance between comfort and treatment of these patients. Wound care should be accompanied with pain management, odour control, and exudate control to give the best comfort to the patient. Conservative debridement of slough can, however, be undertaken to control the bioburden. Local, topical analgesic treatment including ibuprofen-impregnated dressings, lidocaine preparations and diamorphine hydrogel can be used. Systemic analgesics like opioids and / or non-steroidal anti-inflammatory drugs can be used 30 minutes prior to dressing changes and afterwards.[1]

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Colour Plates

Unless otherwise stated, the sources of colour plates originate from the authors of the corresponding chapters.

Plate 1.1 Distribution of elderly population (age ≥65 years) by country: **(a)** 2008 and **(b)** 2040

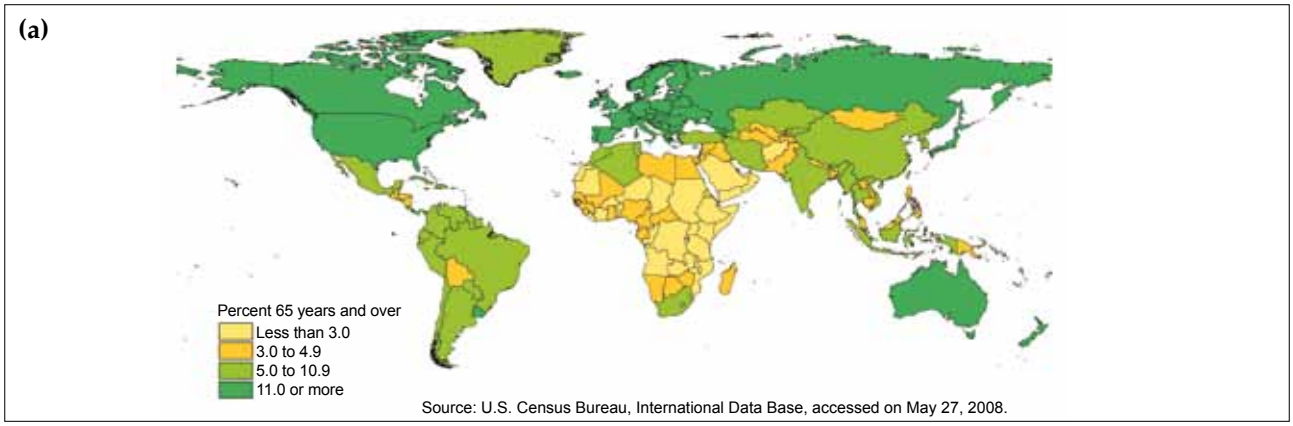


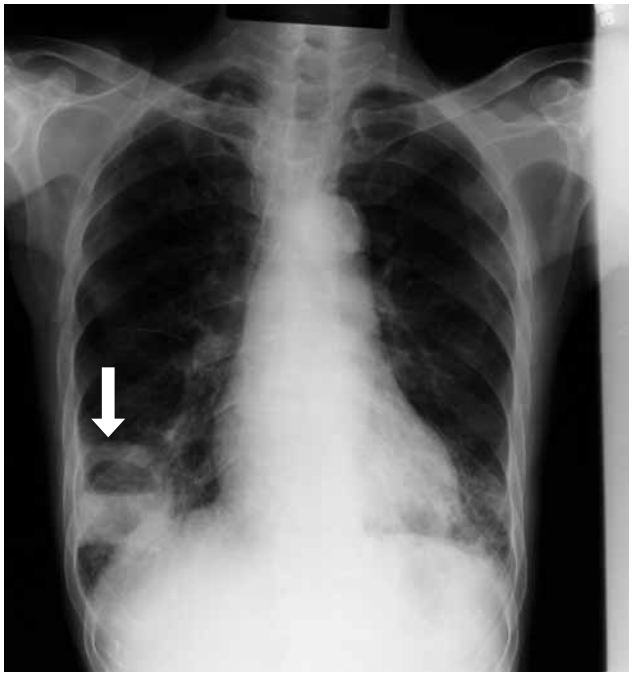
Plate 2.1 A patient with post-stroke left hemineglect with characteristic sitting posture, trunk and head rotated away from the side of weakness, and conjugate eyes deviation away from the side of weakness. Attentional deficit is evidenced from half of the plate being untouched on the side of his neglect

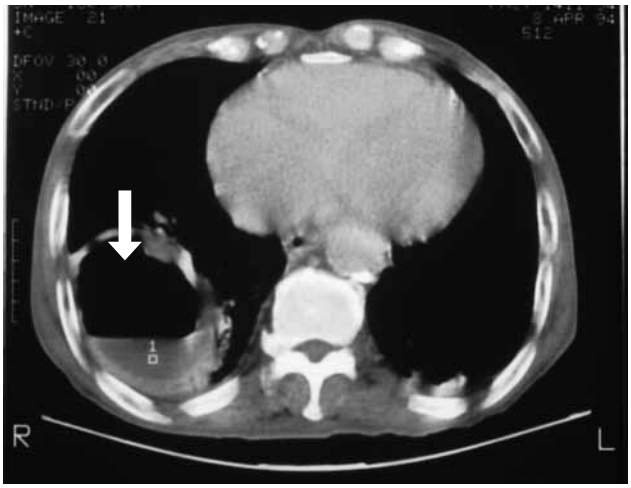


Plate 13.1 Cancer mimic in old age: tuberculosis of the knee in a 99-year-old woman with right knee pain. Imaging shows an osteolytic lesion with increased uptake reported as suspected malignancy



Plate 13.2 Multiple pathologies and aetiologies in old age. An 82-year-old man with right lower lobe lung abscess; sputum cytology revealed squamous cell carcinoma; pathology of the resected right lower lobe revealed squamous cell carcinoma over the periphery and tuberculous caseation in the centre (arrows)





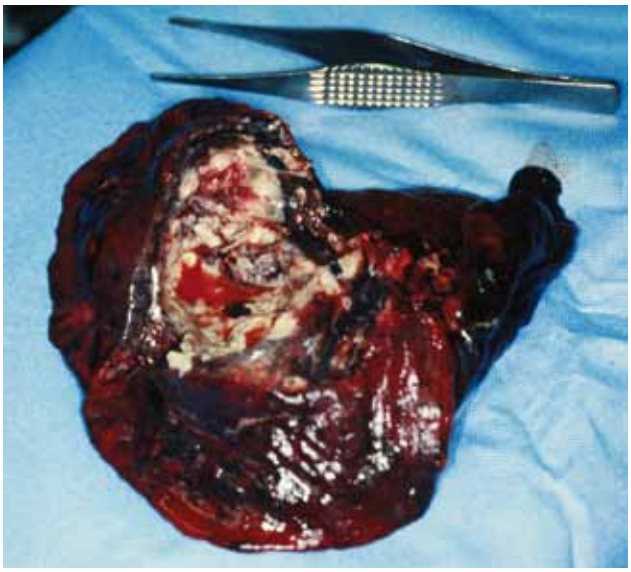


Plate 29.1 Digital gangrene of right foot in a 67-year-old man with fungal (*Candida parapsilosis*) infective endocarditis, predisposed by chronic use of steroid for gouty arthritis



Plate 29.2 Chest X-ray showing miliary tuberculosis in a 65-year-old man, predisposed by chronic use of steroid for polyarticular gout. He presented as weakness and fall with fractured patella, and the miliary tuberculosis was apparent only later during work-up for 'postoperative pneumonia'

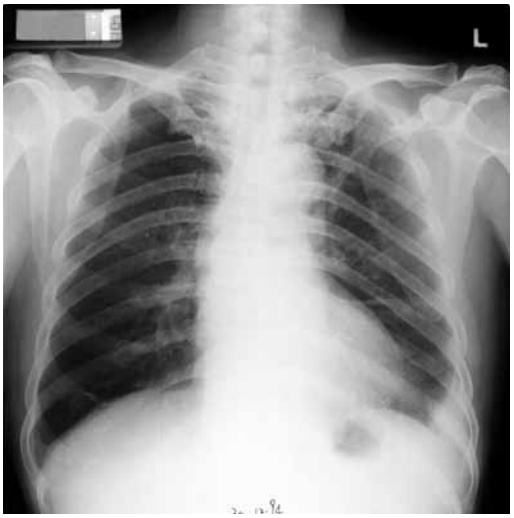


Plate 33.1 Case 33.1: Periventricular hypodensity (arrows) in the computed tomographic brain of a 68-year-old woman with postoperative confusion after total knee replacement. Her impaired cognition persisted and she developed vascular dementia

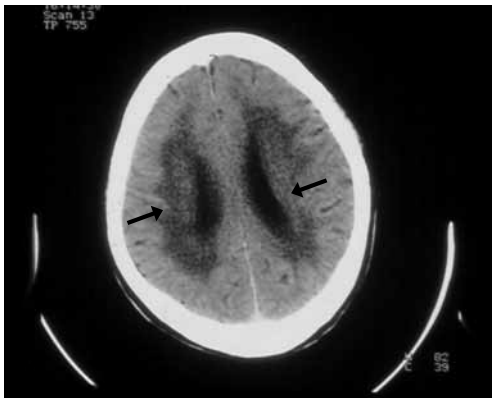


Plate 33.2 Case 33.3: **(a, b)** Osteoporotic collapse of T7 and L2 vertebral bodies, and **(c)** faecal loading revealed in X-rays of spine and abdomen in an 80-year-old man with acute subcostal pain

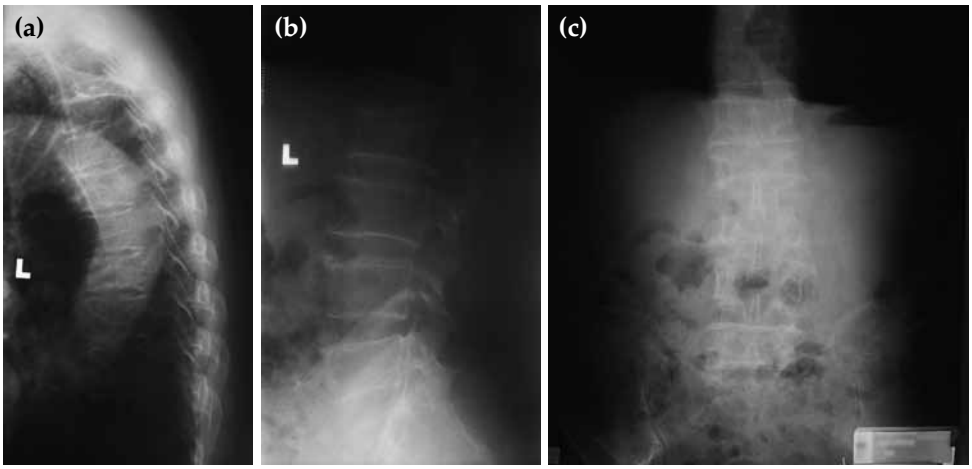


Plate 33.3 Typical gout with acutely inflamed first metatarsophalangeal joint (podagra)



Plate 33.4 Polyarticular gout in an 82-year-old woman on diuretics for hypertension and heart failure, involving finger joints of **(a)** hand, **(b)** foot, and **(c)** knee. Note tophaceous deposits in **(a)** Heberden's nodes and **(b)** bunion bursa

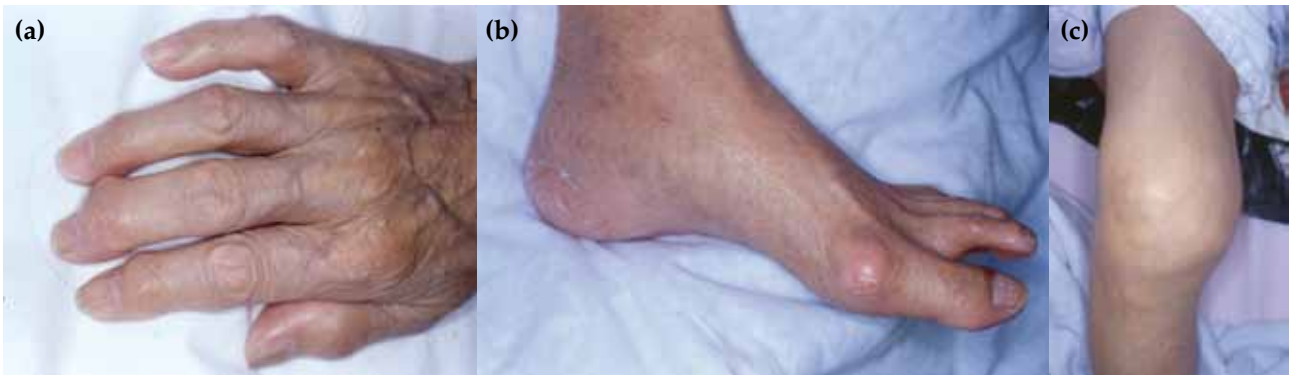
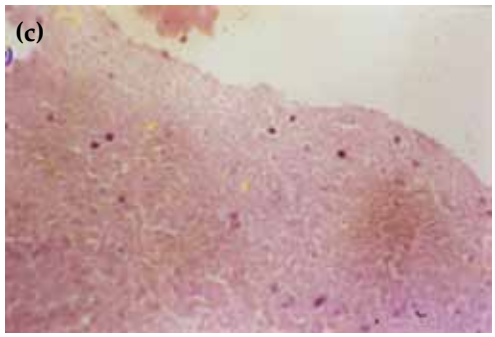


Plate 33.5 Axial gout (spinal gout) affecting the lumbar spine simulating infective spondylitis in an 80-year-old man with fever, backache, a high erythrocyte sedimentation rate of 100 mm/h, and known history of polyarticular gout. **(a)** X-ray and **(b)** computed tomographic lumbar spine reveal changes suspicious of infective spondylitis / tuberculosis of the spine with end-plate destruction. **(c)** Needle biopsy of L2 revealed needle-shaped, strongly negatively birefringent monosodium urate crystals. When viewed under a light microscope with a polariser and compensator added, the crystal appears yellow when its axis is parallel to the plane of the compensator and blue when the axes are perpendicular (plate c provided by Dr Kam-cheong Lee, Department of Pathology, Princess Margaret Hospital)







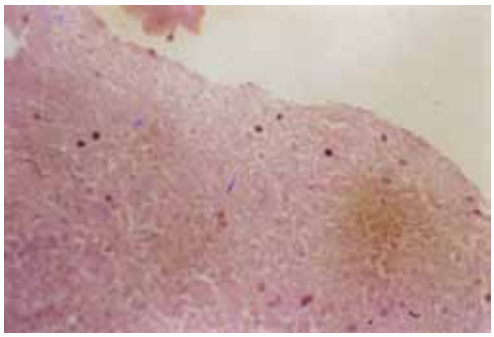
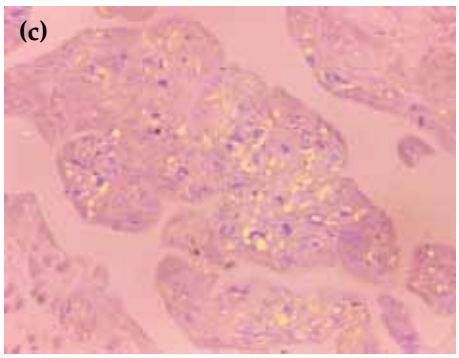


Plate 33.6 Pseudogout in a 65-year-old woman: **(a)** acutely inflamed right knee with effusion; previous total knee replacement of left knee for disabling arthritis; **(b)** chondrocalcinosis (calcified articular cartilage) shown in X-ray of her right knee; **(c)** synovial biopsy of her left knee showing rhomboid-shaped, weakly positively birefringent calcium pyrophosphate dihydrate crystals. When viewed under a light microscope with a polariser and compensator added, the crystal appears blue when its axis is parallel to the plane of the compensator and yellow when the axes are perpendicular (plate c provided by Dr Kam-cheong Lee, Department of Pathology, Princess Margaret Hospital)







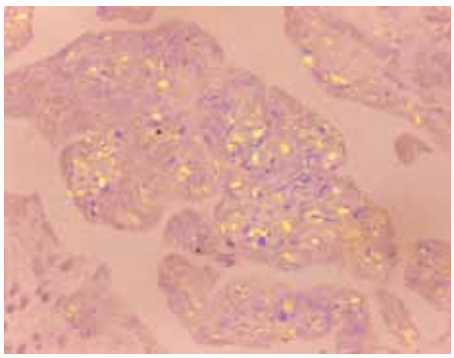


Plate 33.7 Allopurinol hypersensitivity syndrome in a 68-year-old man: **(a)** initial erythematous papules and target lesions **(b)** progressing to toxic epidermal necrolysis, renal failure, and hepatitis



Plate 33.8 Case 33.5: Polyostotic Paget's disease in a 68-year-old man referred for raised alkaline phosphatase (674 U/L) after cholecystectomy. **(a)** Bone scan reveals multiple sites of increased bone uptake over T-L spine, right hemipelvis, left clavicle, and right skull base. **(c)** X-ray pelvis shows loss of some trabeculae with coarsening of the remaining trabeculae, thickened iliopectineal line, and marked deformity of the pelvic inlet and enlargement of the right pubis and ischium. **(d)** A bone biopsy of his pelvis, done for suspicious malignant change, shows increased osteoclastic and osteoblastic activity with presence of giant cells and fibrosis, thick bone with mosaic cement line characteristic of Paget's disease of bone. **(b)** A follow-up bone scan shows improvement after intravenous pamidronate infusions (plate d provided by Dr Kam-cheong Lee, Department of Pathology, Princess Margaret Hospital)

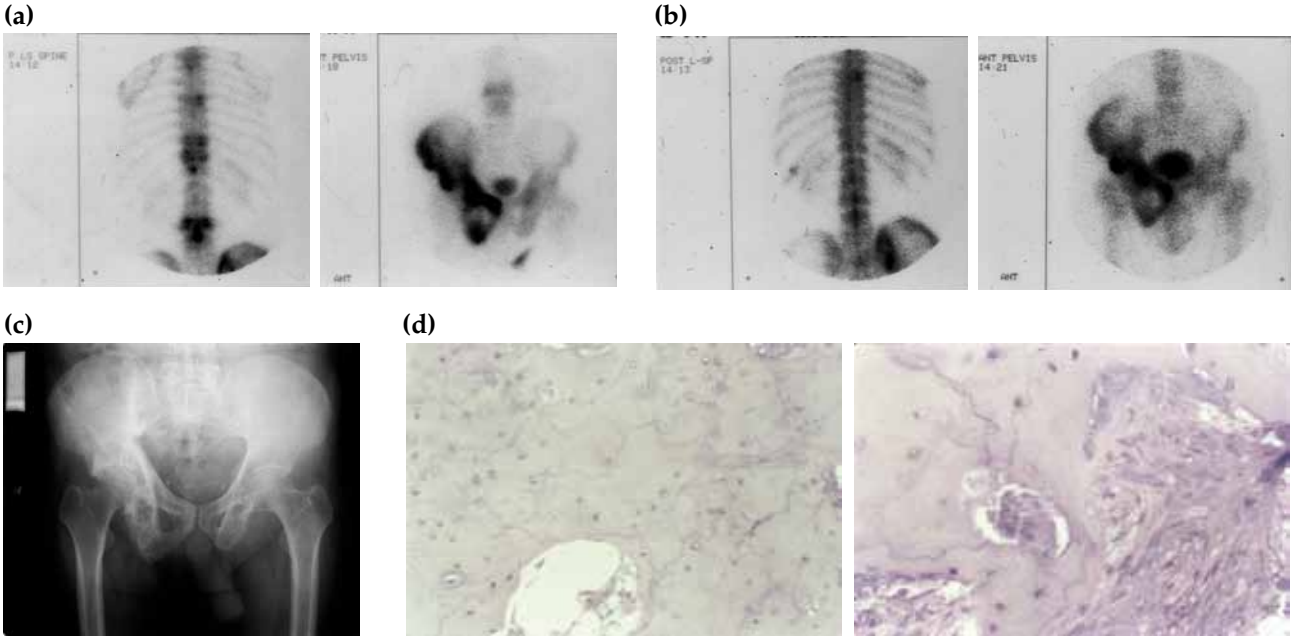


Plate 33.9 Case 33.6: Polyostotic Paget's disease in a 78-year-old man with increasing size of skull and neck: **(a)** lateral view of head and neck; **(b)** X-ray skull showing skull vault thickening with patchy sclerosis; **(c)** X-ray neck showing enlarged C2 with loss of trabeculations; **(d)** size of neck collars has increased over 10 years

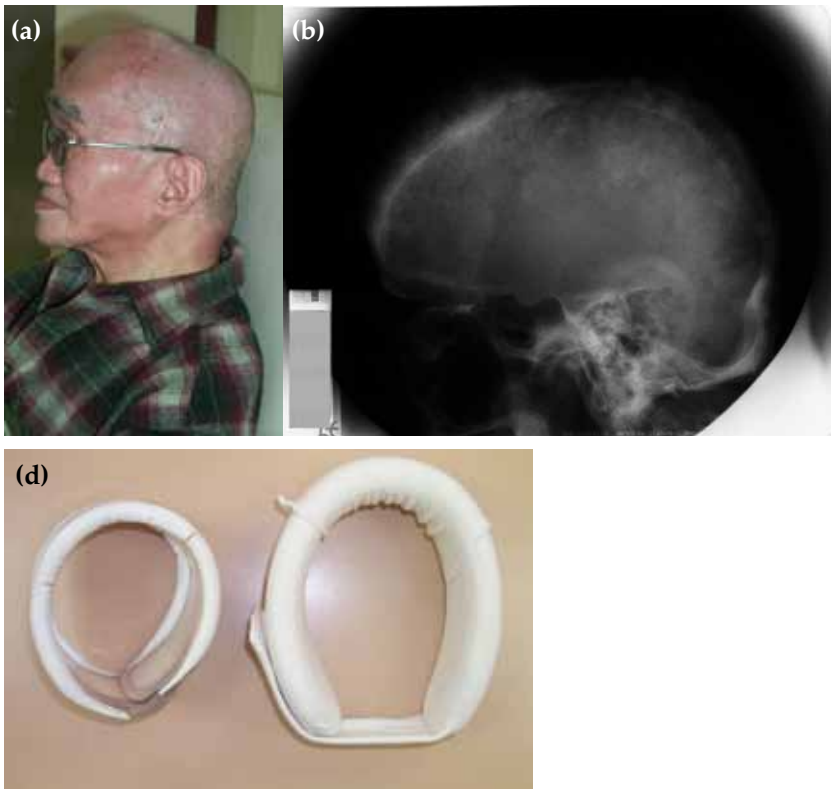


Plate 33.10 Case 33.7: Serial photos of a patient with Paget's disease of bone diagnosed at age 73 years, showing frontal bossing since the age of 56 years

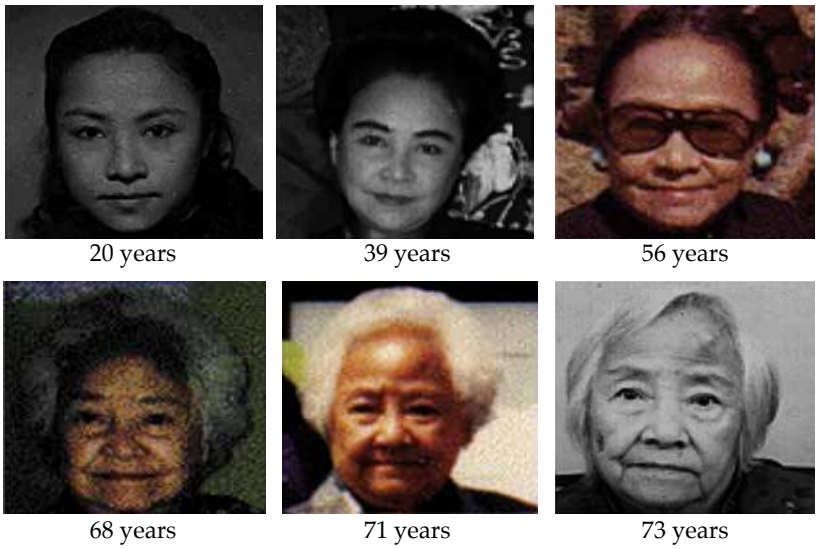


Plate 35.1 Case scenario 1: computed tomography of the brain showing hyperdense right middle cerebral artery sign (arrows)

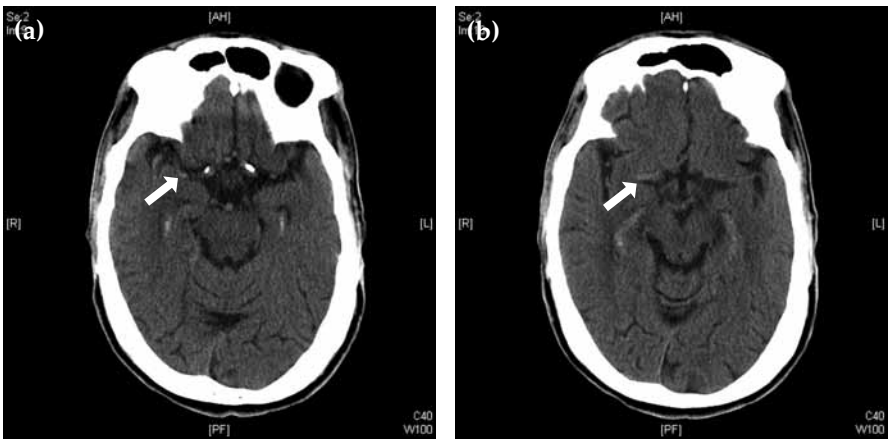


Plate 35.2 Case scenario 2: computed tomography of the brain showing left internal border-zone infarction (arrow)



Plate 35.3 Case scenario 2: transcranial Doppler revealing turbulence flow at left siphon artery, the peak velocity was 158 cm/s

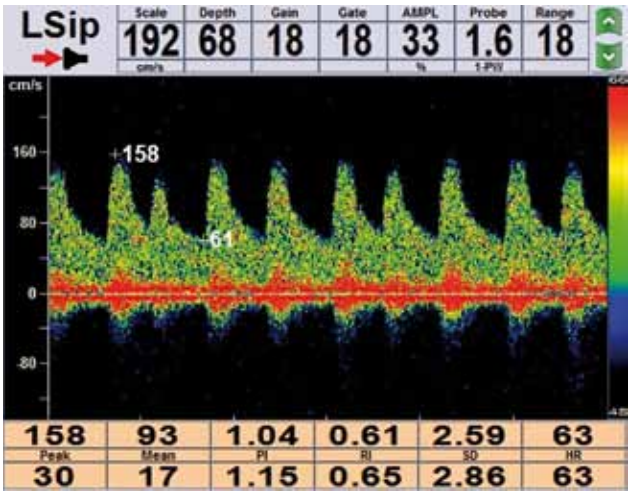


Plate 35.4 Case scenario 3: computed tomography of the brain showing multiple cerebral infarctions (arrows)

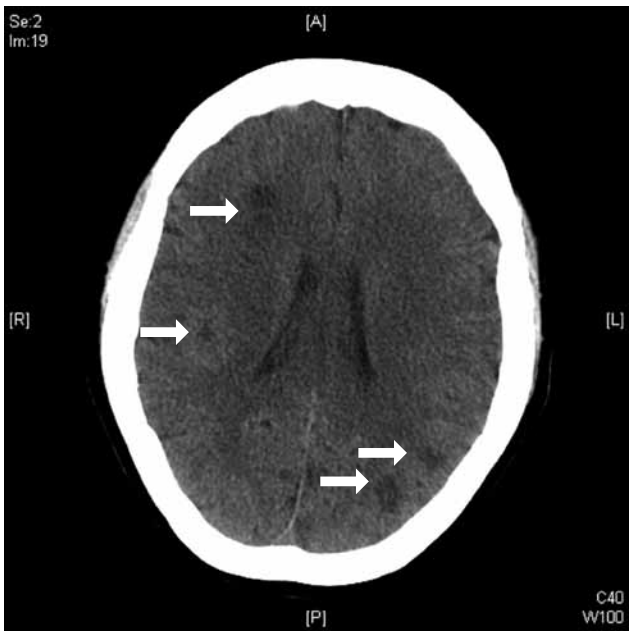


Plate 37.1 Venous ulcer at left ankle complicating varicose veins (plate provided by Dr Tak-kwan Kong, Department of Medicine and Geriatrics, Princess Margaret Hospital)



Plate 37.2 Arterial ulcers at left foot and leg in a diabetic patient with peripheral vascular disease and dilated cardiomyopathy (plate provided by Dr Tak-kwan Kong, Department of Medicine and Geriatrics, Princess Margaret Hospital)



Plate 37.3 Neuropathic ulcer at sole of left foot in a diabetic patient with autonomic neuropathy and Charcot foot (plate provided by Dr Tak-kwan Kong, Department of Medicine and Geriatrics, Princess Margaret Hospital)



Plate 40.1 Scabies affecting the hand **(a)** before and **(b)** after treatment

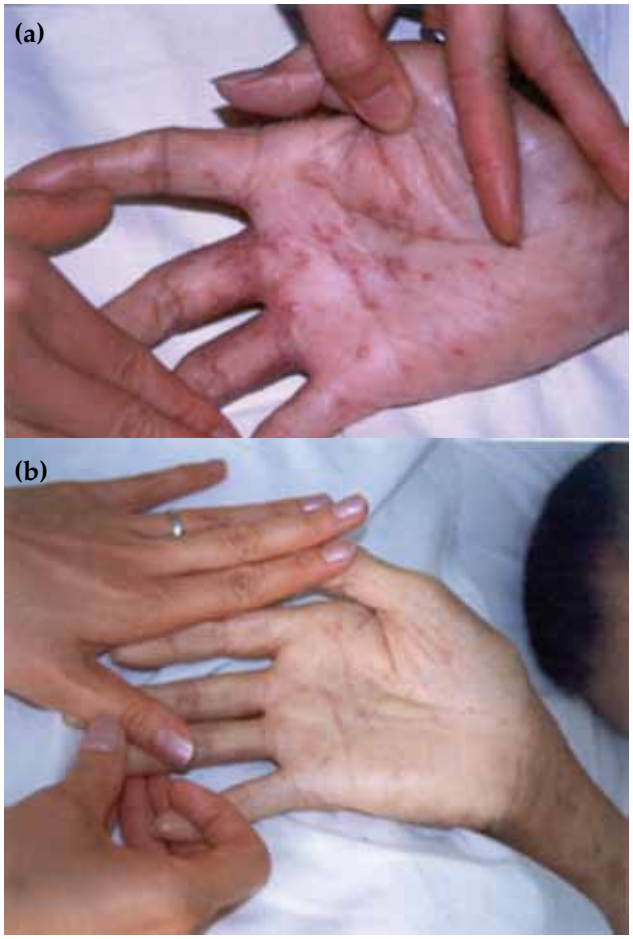


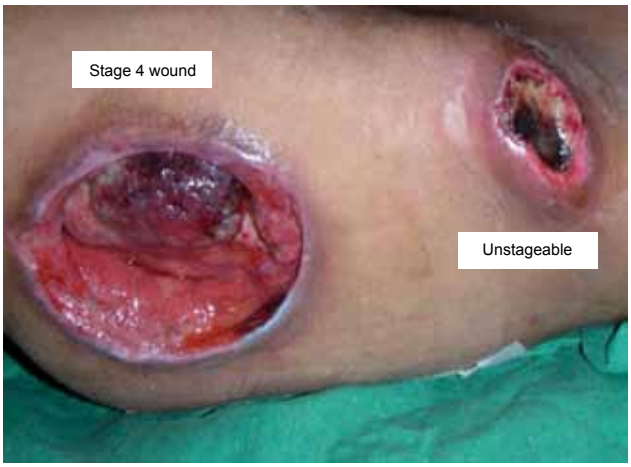
Plate 40.2 Patient with Norwegian scabies affecting the face and pinna



Plate 43.1 Complications after percutaneous endoscopic gastrostomy (PEG): granuloma
This elderly man complained of pain at the PEG tube–exit site. The pain and granuloma resolved after repeated topical treatment with silver nitrite pen applications



Plate 44.1 Stage 4 and unstageable pressure ulcers





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ISBN 978-988-78347-0-0