eighth edition

MURTAGH'S GENERAL PRACTICE


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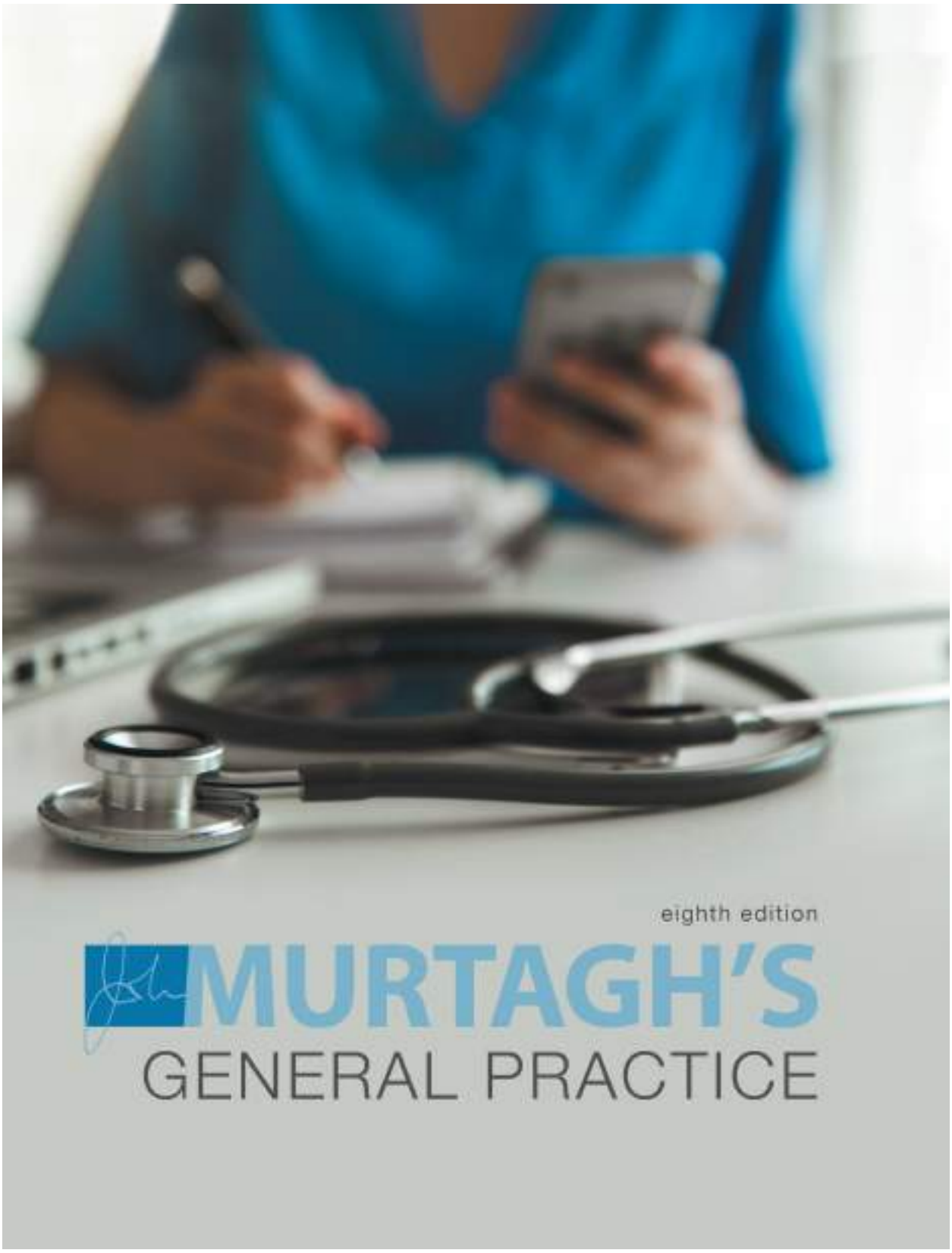
eBook Murtagh General Practice, 8th Edition

Cover



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NOTICE
Medicine is an ever-changing science. As new research and clinical experience broaden our knowledge, changes in treatment and drug therapy are required. The editors and the publisher of this work have checked with sources believed to be reliable in their efforts to provide information that is complete and generally in accord with the standards accepted at the time of publication. However, in view of the possibility of human error or changes in medical sciences, neither the editors, nor the publisher, nor any other party who has been involved in the preparation or publication of this work warrants that the information contained herein is in every respect accurate or complete. Readers are encouraged to confirm the information contained herein with other sources. For example, and in particular, readers are advised to check the product information sheet included in the package of each drug they plan to administer to be certain that the information contained in this book is accurate and that changes have not been made in the recommended dose or in the contraindications for administration. This recommendation is of particular importance in connection with new or infrequently used drugs.

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MBBS, MD, BSc, BEd, FRACGP, DipObstRCOG

Emeritus Professor in General Practice, School of Primary Health, Monash University, Melbourne

Professorial Fellow, Department of General Practice, University of Melbourne

Adjunct Clinical Professor, Graduate School of Medicine, University of Notre Dame, Fremantle, Western Australia

John Murtagh was a science master teaching chemistry, biology and physics in Victorian secondary schools when he was admitted to the first intake of the newly established Medical School at Monash University, graduating in 1966. Following a comprehensive postgraduate training program, which included surgical registrarship, he practised in partnership with his wife, Dr Jill Rosenblatt, for 10 years in the rural community of Neerim South, Victoria.

He was appointed Senior Lecturer (part-time) in the Department of Community Medicine at Monash University and eventually returned to Melbourne as a full-time Senior Lecturer. He was appointed to a professorial chair in Community Medicine at Box Hill Hospital in 1988 and subsequently as chairman of the extended department and Professor of General Practice in 1993 until retirement from this position in 2010. He now holds teaching positions as Emeritus Professor in General Practice at Monash University, Adjunct Clinical Professor, University of Notre Dame and Professorial Fellow, University of Melbourne. He achieved the Doctor of Medicine degree in 1988 for his thesis ‘The management of back pain in general practice’.

He was appointed Associate Medical Editor of *Australian Family Physician* in 1980 and Medical Editor in 1986, a position he held until 1995. In 1995 he was awarded the Officer of the Order of Australia for services to medicine and to medical education in the field of general practice and to professional groups.

One of his numerous publications, *Practice Tips,* was named as the British Medical

Association’s Best Primary Care Book Award in 2005. In the same year John Murtagh was awarded the inaugural David de Kretser medal from Monash University for his exceptional contribution to the Faculty of Medicine, Nursing and Health Sciences over a significant period of time. Members of the Royal Australian -College of General Practitioners may know that the honour of the namesake of the College library was bestowed upon him. In 2018 he was awarded the Australian Medical Association’s Gold Medal for exceptional and long-standing commitment and contribution to general practice and advancing the profession through medical education.

Today John Murtagh continues to enjoy active participation in medical education activities. His vast experience with all medical groups has provided him with tremendous insights into their needs, which is reflected in the culminated experience and wisdom of *John Murtagh’s General Practice.*

Dr Jill Rosenblatt

MBBS, FRACGP, DipObstRCOG, GradDipAppSci

Jill Rosenblatt graduated in medicine from the University of Melbourne in 1968. Following terms as a resident medical officer she entered rural practice in Neerim South, Victoria, in partnership with her husband John Murtagh. She was responsible for inpatient hospital care in the Neerim District Bush Nursing Hospital and in the West Gippsland Base Hospital. Her special interests were obstetrics, paediatrics and anaesthetics. Jill has also had a special interest in Indigenous culture and health since she lived at Koonibba Mission in South Australia, where her father was Superintendent.

After leaving rural life she came to Melbourne and joined the Ashwood Medical Group, where she practised comprehensive general medicine, and care of the elderly in particular. She was appointed Adjunct Senior Lecturer in the Department of General Practice at Monash University in 1980 and a teacher in the GP registrar program.

She gained a Diploma of Sports Medicine (RACGP) in 1985 and a Graduate Diploma of Applied Science in Nutritional and Environmental Medicine from Swinburne University of Technology in 2001.

Jill Rosenblatt brings a wealth of diverse experience to the compilation of this textbook. This is based on 50 years of experience in rural and metropolitan general practice. In addition, she has served as clinical assistant to the Shepherd Foundation, the Menopause Clinics at Prince Henry’s

Hospital and Box Hill Hospital and the Department of Anaesthetics at Prince Henry's Hospital. Jill has served as an examiner for the RACGP for 39 years and for the Australian Medical Council for 16 years. She was awarded a life membership of the Royal Australian College of General Practitioners in 2010 and a Distinguished Service award of the College in 2014.

Dr Justin Coleman

MBBS, FRACGP, MPH

General Practitioner, Julanimawu Health Service, Wurrumiyanga, NT

Senior Lecturer, Flinders University Medical School

Medical Educator, NTGPE

Editorial Advisory Board, Diabetes Management Journal

Board Director, GP Supervisors Association Governance Board, ALIVE National Centre for Mental Health Research Translation

Justin Coleman graduated from Melbourne University Medical School in 1992 and has subsequently worked as a rural GP in Victoria, the remote NT and Brisbane, primarily in Aboriginal and Torres Strait Islander health.

Soon after graduating, Justin began writing for the GP newspaper *Medical Observer* and hasn't stopped since. One of his weekly columns, 'Handy Hints for GPs', ran for 13 years. He writes regular humorous opinion columns.

Justin is a prolific writer for medical and non-medical readerships; he has published well over 1500 medical articles in around 50 different newspapers, magazines, books and journals. For five years he served as President of the Australasian Medical Writers Association and he regularly runs writing workshops for medical writers and academics.

Since completing a Master of Public Health (UQ 2011, first class hons), Justin has dedicated much of his career to educating other GPs about how to improve various aspects of medical practice. His interests include evidence-based medicine, the rational use of medical tests and treatments, and dealing with uncertainty during a GP consultation. He represents the RACGP on matters pertaining to conflicts of interest and fiercely guards his own independence, never having accepted payment from a pharmaceutical or medical device company.

Over three decades, Justin has supervised hundreds of medical students and GP registrars. He has taught in the medical schools of four universities and for a dozen medical education

organisations.

Justin edited his first medical book 25 years ago and has remained a medical editor ever since. He completed a Writing and Editing program in 2010 (UQ, first class hons). He was editor of the *Diabetes Management Journal*, writes and does peer reviews for the MJA, AJGP (formerly AFP) and BMJ, and is a member of the Australasian Health and Medical journal Editors' Network (AHMEN).

Justin was honoured to be invited by Professor John Murtagh to help edit Australia's seminal textbook on general practice. This represents the grand intersection of every one of his aforementioned interests.

Dr Clare Murtagh

MBBS, FRACGP

General Practitioner, Sydney

Clare Murtagh completed her medical studies at Monash University in 2007 and spent her early career working in hospitals in Geelong and rural Victoria. Following experience as a medical officer for trekkers in Nepal, she moved to Sydney where she completed her General Practice training in 2013.

A passionate generalist, Clare has special interest in dermatology, women's health and paediatrics. She holds a Diploma of Dermatology and Certificates in Sexual and Reproductive Health, and Medical Education. While practising at Your Doctors in Sydney's inner west, she has cared for a wide variety of patients and is an antenatal shared care provider.

In recent years, Clare has gained increasing experience in medical education as a supervisor of training GPs and as an examiner for the RACGP. She has worked as a medical educator at GP Synergy and is a lecturer on dermatology.

Clare has been an enthusiastic contributor to the 'Women's health', 'Sexual health' and 'Problems of the skin' sections of the last three edition of *Murtagh's General Practice.* As the daughter of co-authors John Murtagh and Jill Rosenblatt, she has benefited from their mentorship and appreciates the genesis and philosophy of the editorial direction of the textbook.

Foreword

In 1960 a young schoolmaster, then teaching biology and chemistry in a secondary school in rural Victoria, decided to become a country doctor. He was part of the first intake of students into the Medical School of the newly established Monash University, and at the end of his six-year undergraduate medical course and subsequent intern and resident appointments his resolve to practise community medicine remained firm. After more than a decade in country practice with his life partner, Dr Jill Rosenblatt, during which he meticulously documented the cases he treated, in 1977 John Murtagh took up an academic position in the new Department of General Practice at Monash University. He subsequently moved through the ranks of Senior Lecturer, Associate Professor and Professor, now enjoying the title of Emeritus Professor.

Through his writing, pedagogy and research, John Murtagh became a national and international authority on the content and teaching of primary care medicine. It was during his tenure as Medical Editor of *Australian Family Physician* from 1986 to 1995 that the journal became the most widely read medical journal in Australia.

This textbook provides a distillate of the vast experience gained by a once rural doctor, whose career has embraced teaching; whose abiding interest is in ensuring that disease, whether minor or life-threatening, is recognised quickly; and whose concern is that strategies to match each contingency are well understood.

The first edition of this book, published in 1994, achieved remarkable success on both the national and international scene. The second and third editions built on this initial success and the book has become known as the ‘bible of general practice’ in Australia. In addition to being widely used by practising doctors, it has become a popular and standard textbook in several medical schools and also in the teaching institutions for alternative health practitioners, such as chiropractic, naturopathy and osteopathy. In particular, medical undergraduates and graduates struggling to learn English have found the book relatively comprehensible. The fourth and fifth editions were updated and expanded, retaining the successful, user-friendly format, including clinical photography and illustrations in colour. Dr Jill Rosenblatt joined John in authoring and editing the fifth, sixth and seventh editions. Two new author/editors in Dr Justin Coleman and Dr Clare Murtagh subsequently joined the panel.

Having known John and worked with him for more than three decades, I feel privileged to write this foreword to the eighth edition, adding to earlier forewords by the late Professor Schofield. During this 27-year period I have watched each edition blossom, only to be superseded by a bigger and better replacement. John Murtagh has become a legend nationally and internationally, and in a 2012 *Medical Observer* survey he was voted the most revered Australian doctor, ahead of Fred Hollows and Victor Chang. Most recently, in 2018 John was awarded the Australian Medical Association’s highest honour, the AMA Gold Medal for his ‘contribution to medicine and general practice as a doctor and educator’. In addition, in 2019 he became an Officer of the Order of Australia (AO) for his contribution to scholarship in General Practice, superseding his

award of Member of the Order of Australia (AM) awarded in 1996.

This edition retains the time-honoured framework that has made it the seminal text for GPs, GP registrars and students of general practice worldwide. It is to general practice what ‘Harrisons’ is to internal medicine.

Although this edition retains the same format, it has a number of significant changes and additions, including a strong emphasis on viral infections including the coronaviruses. Reflecting John’s lifelong commitment to medical education, he has included more visual material, more practical tips for day-to-day clinical practice and importantly, more on therapeutics supported by references to *Therapeutic Guidelines.*

The expanded volume has necessitated a significant increase in references to original sources to substantiate the evidence base within this text. As expected in contemporary texts, there is also an abundance of online resources.

John Murtagh’s works, including this text, have been translated into Italian by McGraw-Hill Libri Italia s.r.l., Portuguese by McGraw-Hill Nova Iorque and Spanish by McGraw-Hill Interamericana Mexico and also into Chinese, Greek, Polish and Russian. In 2009 *John Murtagh’s General Practice* was chosen by the Chinese Ministry of Health as the textbook to aid the development of general practice in China. Now, 27 years since its beginning, the text is available in 13 languages, most recently adding Farsi and Turkish translations. A truly remarkable achievement.

GC SCHOFIELD
OBE, MD, ChB(NZ), DPhil(Oxon), FRACP, FRACMA, FAMA
Emeritus Professor of Anatomy
Dean of Medicine
Monash University, 1977–88

Leon Piterman AM (Foreword to the sixth, seventh and eighth editions)
MBBS, MD, MMed, MEdSt, FRCP(Edin), FRACGP
Professor of General Practice,
Monash University

Contents

Acknowledgments

Professor Murtagh would like to thank the Publication Division of the Royal Australian College of General Practitioners for supporting his past role as Medical Editor of *Australian Family Physician*, which provided an excellent opportunity to gather material for this book. Acknowledgment is also due to those medical organisations that have given permission to use selected information from their publications. They include the Preventive and Community Medicine committee of the RACGP (Guidelines for Preventive Activities in General Practice), Therapeutic Guidelines Limited, the Hypertension Guideline Committee: Research Unit RACGP (South Australian Faculty) and the *Medical Observer*, publishers of *A Manual for Primary Health Care*, for permitting reproduction of Appendices I–IV.

For decades, *Therapeutic Guidelines* (TG) has set the gold standard for practice guidelines, beginning with the benchmark antibiotic guidelines. The panels for the various disciplines include experts from many fields whose collective wisdom and evidence base in their deliberations inspires confidence and authority for treatment decisions. General practitioners also have input in the panels. The authors of *Murtagh's General Practice* wish to thank Therapeutic Guidelines Limited for the outstanding information which provides an authoritative framework for our publication. *Therapeutic Guidelines* is the ultimate therapeutic reference across all categories, from analgesics and antibiotics to ulcers and wound management.

Special thanks to the late Chris Sorrell for his art illustration, and to Nicki Cooper, Jenny Green and Caroline Menara for their skill and patience in typing the manuscript.

Many of the quotations at the beginning of chapters appear in either Robert Wilkins (ed), *The Doctor's Quotation Book*, Robert Hale Ltd, London, 1991, or Maurice B. Strauss (ed), *Familiar Medical Quotations*, Little, Brown & Co., New York, 1958.

Thanks also to Professor Roger Pepperell, Dr Bruce Mugford, Dr Lucie Stanford, Dr Mohammad Shafeeq Lone, Dr Brian Bedkobar for content advice and Professor Chris White for technical support, Dr Ebrahim Pishan, Dr Joseph Turner and Lesley Rowe for reviewing the manuscript, and to the publishing and production team at McGraw-Hill Education (Australia) for their patience and assistance in so many ways.

Finally, thanks to Dr Ndidi Victor Ikealumba for his expert review of *General Practice* sixth edition, tropical medicine and his subsequent contribution.

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Dr Richard P Usatine, 3e: Figure 8.3, p. 68; Figure 25.5, p. 284; Figure 30.13, p. 362; Figure 39.12, p. 478; Figure 61.6, p. 752; Figures 84.6 and 84.7, p. 971; Figure 84.8, p. 972; Figure 93.5, p. 1071; Figure 98.5, p. 1119; Figure 99.1, p. 1124; Figure 109.5, p. 1214; Figure 113.11, p. 1258; Figure 117.2, p. 1310; Figures 118.5 and 118.6, p. 1324; Figure 119.7 (photo), p. 1330.

Dr William Clark: Figure 39.3, p. 471; Figure 39.6, p. 472; Figure 39.7, p. 473; Figure 48.1, p. 580.

Frontline Medical Communications: Figure 88.4, p. 1019; Figure 115.6, p. 1288.

Paul D. Comeau: Figure 40.6, p. 489.

DEA: Figure 12.6, p. 117.

Dr Nicolette Deveneau: Figure 93.2, p. 1069.

Javier La Fontaine DPM: Figure 115.5, p. 1287.

Dr Michelle Rowe: Figure 12.5, p. 117.

Dr C. Blake Simpson: Figure 46.1, p. 557.

Dr Marc Solioz: Figure 8.1, p. 66.

Dr Eric Kraus: Figure 112.5, p. 1240.

Dr Hugh Newton-John: Figure 9.1, p. 73; Figure 20.4, p. 200; Figure 50.3, p. 59.7; Figure 86.3, p. 991.

Professor Barry Firkin and Professor Hatem Salem: Figure 9.4, p. 77.

Dr Peter Ryan: Figure 21.4, p. 209.

Dr Marissa Lassere: Figure 21.5, p. 209.

Professor John Masterton: Figure 25.12, p. 292; Figure 26.2, p. 300; Figure 26.3, p. 302.

Bruce Black: Figure 39.8, p. 474; Figure 39.10, p. 476.

John Colvin and Joseph Reith: Figure 40.2, p. 487; Figure 40.3, p. 487; Figure 40.4, p. 488; Figure 40.5, p. 489; Figure 40.8, p. 491; Figure 40.12, p. 493; Figure 40.14, p. 493.

Robin Marks: Figure 57.13, p. 710; Figure 112.10, p. 1243; Figure 116.17, p. 1300.

Dr Peter Couran: Figure 113.15, p. 1260.

Dr John Troller: Figure 116.9, p. 1297.

Preface

The discipline of general practice has become complex, expansive and challenging, but nevertheless remains manageable, fascinating and rewarding. *John Murtagh's General Practice* attempts to address the issue of the base of knowledge and skills required in modern general practice. Some of the basics of primary healthcare remain the same. In fact, there is an everlasting identity about many of the medical problems that affect human beings, be it a splinter under a nail, a stye of the eyelid, a terminal illness or simply stress-related anxiety. Many of the treatments and approaches to caring management are universal and timeless.

This text covers a mix of traditional and modern practice with an emphasis on the importance of clinical reasoning, early diagnosis, strategies for solving common presenting problems, continuing care, holistic management and 'tricks of the trade'. One feature of our discipline is the patient who presents with undifferentiated problems featuring an overlap of organic and psychosocial components. There is the constant challenge to make an early diagnosis and identify the ever-lurking, life-threatening illness. Hence the 'must not be missed' catch cry throughout the text. To reinforce this awareness, 'red flag pointers' to serious disease are included where appropriate. The general practice diagnostic model, which pervades all the chapters on problem solving, is based on the authors' experience, but readers can draw on their own experience to make the model work effectively for themselves.

This eighth edition expands on the challenging initiative of diagnostic triads (or tetrads), which act as a brief *aide-memoire* to assist in identifying a disorder from three (or four) key symptoms or signs. A particular challenge in the preparation of the text was to identify as much appropriate and credible evidence-based information as relevant. This material, which still has its limitations, has been combined with considerable collective wisdom from experts, especially from the *Therapeutic Guidelines* series. A key objective of this publication is to achieve a balance between science and the art of general practice. To provide updated accuracy and credibility, the authors have had the relevant chapters peer reviewed by independent experts in the respective disciplines. These consultants are acknowledged in the reviewers section. The revised editions also have the advantage of co-authorship from experienced general practitioner Dr Jill Rosenblatt. Additional authors include Dr Clare Murtagh, a general practitioner with experience in medical education, and Dr Justin Coleman, past president of the Australasian Medical Writers Association with special interests in 'Choosing wisely' programs and evidence-based medicine.

A comprehensive book such as this one, which presents a basic overview of primary medicine, cannot possibly cover all the medical problems likely to be encountered. An attempt has been made, however, to focus on problems that are common, significant, preventable and treatable. Recent content includes expanded material on genetic disorders and infectious diseases, particularly coronaviruses and acute respiratory distress syndrome.

John Murtagh's General Practice is written as a user-friendly text with the recent graduate, the

international medical graduate and the medical student in mind. However, all primary-care practitioners will gain useful information from the book’s content.

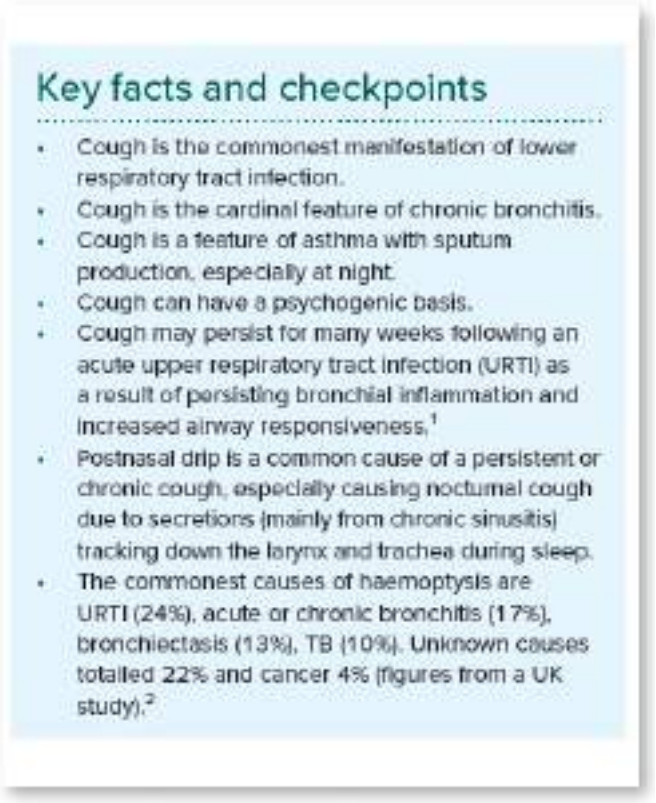
Making the most of your book

Diagnostic strategy models

Diagnostic strategy models for common presenting problems form the backbone of this book. *General Practice* is renowned for this unique and powerful learning feature, which was introduced in the first edition.

Key facts and checkpoints

Key facts and checkpoints provide accurate statistics and local and global contexts.

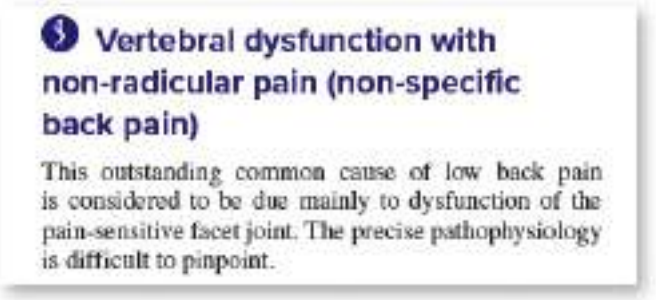


Key facts and checkpoints

- Cough is the commonest manifestation of lower respiratory tract infection.
- Cough is the cardinal feature of chronic bronchitis.
- Cough is a feature of asthma with sputum production, especially at night.
- Cough can have a psychogenic basis.
- Cough may persist for many weeks following an acute upper respiratory tract infection (URTI) as a result of persisting bronchial inflammation and increased airway responsiveness.[1]
- Postnasal drip is a common cause of a persistent or chronic cough, especially causing nocturnal cough due to secretions (mainly from chronic sinusitis) tracking down the larynx and trachea during sleep.
- The commonest causes of haemoptysis are URTI (24%), acute or chronic bronchitis (17%), bronchiectasis (13%), TB (10%). Unknown causes totalled 22% and cancer 4% (figures from a UK study).[2]

The staff of Asclepius

The staff of Asclepius icon highlights diseases for when you are specifically searching for information on a particular disease.



Vertebral dysfunction with non-radicular pain (non-specific back pain)

This outstanding common cause of low back pain is considered to be due mainly to dysfunction of the pain-sensitive facet joint. The precise pathophysiology is difficult to pinpoint.

Red and yellow flags

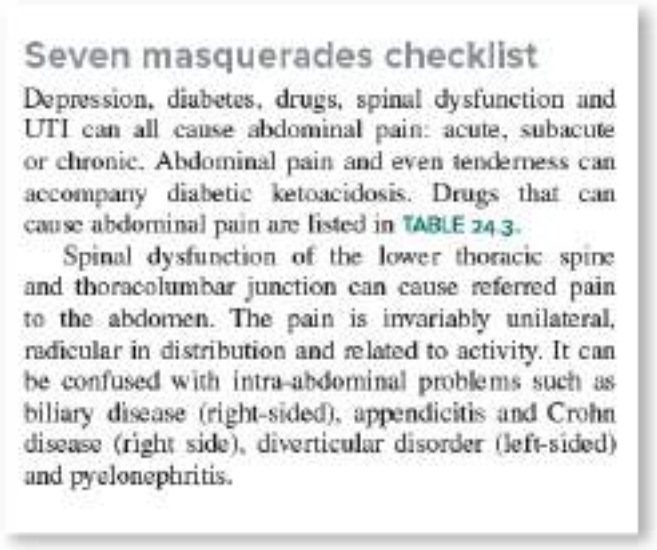
Red and yellow flags alert you to potential dangers. Red is the most urgent, but yellow also requires careful consideration.

Clinical framework

Clinical framework based on major steps of clinical features, investigations, diagnosis, management and treatment reflects the key activities in the daily tasks of general practitioners.

Seven masquerades checklist

This unique feature of the book reminds you of potential and hidden dangers underlying patient presentations.



Seven masquerades checklist

Depression, diabetes, drugs, spinal dysfunction and UTI can all cause abdominal pain: acute, subacute or chronic. Abdominal pain and even tenderness can accompany diabetic ketoacidosis. Drugs that can cause abdominal pain are listed in TABLE 24.3.

Spinal dysfunction of the lower thoracic spine and thoracolumbar junction can cause referred pain to the abdomen. The pain is invariably unilateral, radicular in distribution and related to activity. It can be confused with intra-abdominal problems such as biliary disease (right-sided), appendicitis and Crohn disease (right side), diverticular disorder (left-sided) and pyelonephritis.

Diagnostic triads

Key features that may discriminate between one disease and another are clearly presented.

DxT light-brown skin patches + skin tumours + axillary freckles ➜ NF1

Evidence-based research

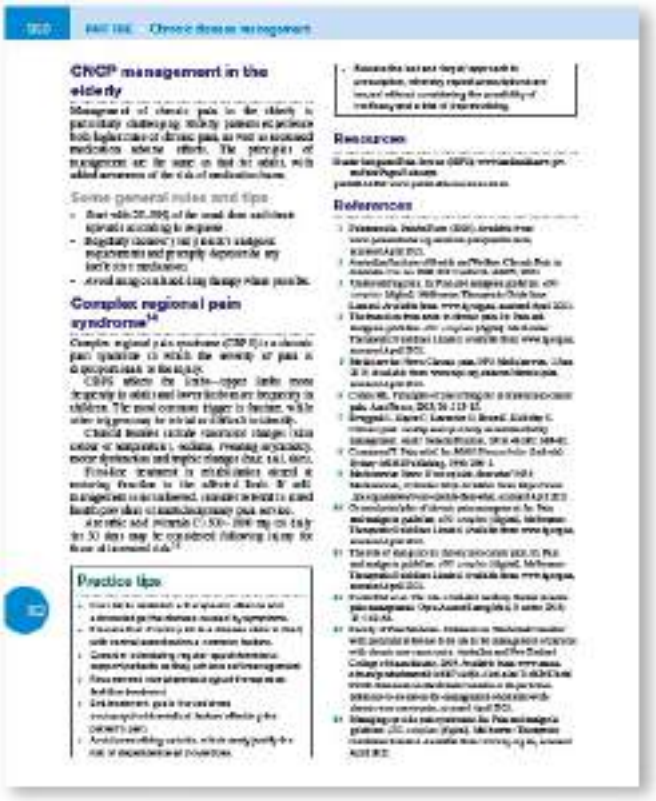
Evidence-based research is recognised with a full chapter on research in general practice and evidence base, including more on qualitative models. In addition, substantial references are provided for every chapter.


7

Research and evidence-based medicine

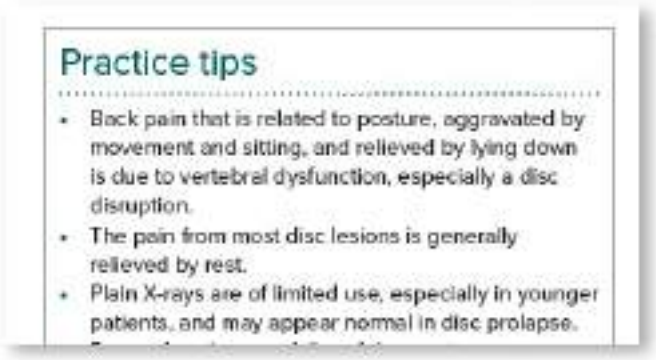
Extensive coverage of paediatric and geriatric care, pregnancy and complementary therapies

Extensive coverage of paediatric and geriatric care, pregnancy and complementary therapies is integrated throughout, as well as devoted chapter content providing more comprehensive information in these areas.



Practice tips

Practice tips consist of key points that are of use in the clinical setting.



Clinical photos

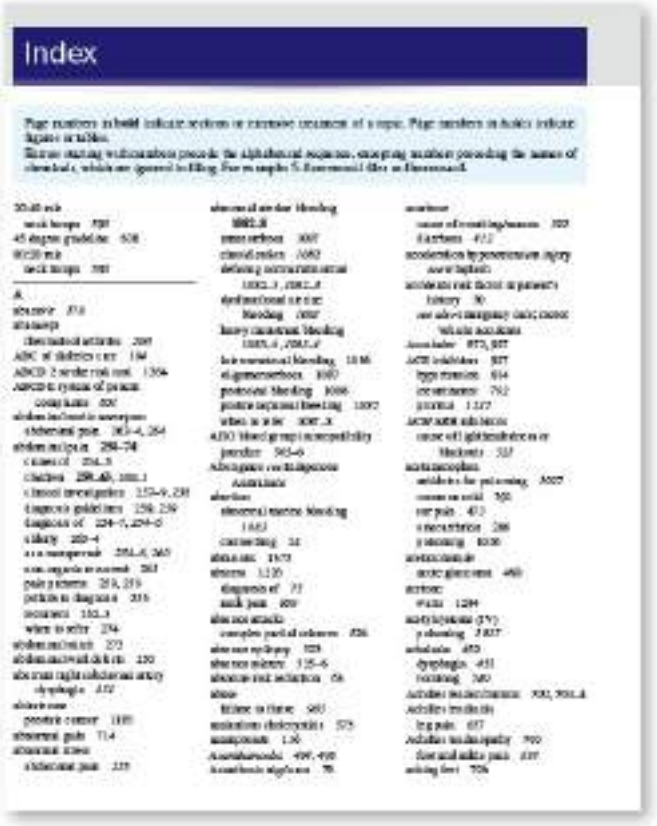
Clinical photos provide authentic, visual examples of many conditions and serve as either a valuable introduction or confirmation of diagnosis.

Full colour illustrations

Full colour illustrations are provided, with more than 600 diagrams in the clean, simple style that has proved so popular.

Significantly enhanced index

The index has more sub-categories with bold page numbers indicating the main treatment of a topic, enabling you to quickly pinpoint the most relevant information. Page numbers in italics refer to figures and tables. Entries with '*see also*' have cross-references to related, but more specific information on the topic.


Index

Patient education resources

Indicates where you can find relevant information from *Murtagh's Patient Education*, eighth edition, to photocopy and hand out to patients.


Patient education resources

Hand-out sheets from *Murtagh's Patient Education* 8th edition:

- Backache
- Exercises for your lower back
- Sciatica
- Spondylosis

Reviewers

Content consultants

The authors are indebted to the many consultants who reviewed parts of the manuscript relevant to their areas of expertise and provided help and advice.

Dr Marion Bailes, Dr Joanne Gardiner and Dr Kate Walker	refugee health
Associate Professor Deborah Bateson	family planning
Dr Roy Beran	epilepsy; neurological dilemmas
Dr James Best	depression, anxiety, male health, child and adolescent health, communication skills
Dr Clare Boema	family planning
Dr John Boxall	palpitations
Dr Penny Burns	disaster medicine, pandemics
Dr Jill Cargnello	hair disorders
Dr Belinda Chan	breast disorders
Dr Paul Coughlin and Professor Hatem Salem	bruising and bleeding; thrombosis and thromboembolism
Mr Rod Dalziel	shoulder pain
Dr David Dilley	pain in the arm and hand
Dr David Dunn and Dr Hung The Nguyen	the health of Indigenous peoples
Dr Robert Dunne	common skin wounds and foreign bodies
Associate Professor John Eden	the menopause
Professor Jon Emery	genetic disorders, malignant disease
Dr Fiona Fargie	sexually transmitted infections
Genetic Health Services, Victoria	genetic disorders
Dr Lindsay Grayson and Associate Professor Joseph Torresi	travel medicine, the returned traveller and tropical medicine
Mr John Griffiths	pain in the hip and buttock
Professor Michael Grigg	pain in the leg

Dr Gary Grossbard	the painful knee
Dr Eliza Hannam	postnatal care
Dr Peter Hardy-Smith	the red and tender eye; visual failure
Associate Professor Peter Holmes	cough; dyspnoea; asthma; COPD
Professor Michael Kidd, Dr Ron McCoy and Dr Alex Welborn	human immunodeficiency virus infection
Professor Gab Kovacs	abnormal uterine bleeding; the subfertile couple
Professor Even Laerum	research in general practice
Mr Peter Lawson (deceased), Dr Sanjiva Wijesinha and Dr Andrew Pattison	men’s health, disorders of the penis, prostatic disorders, scrotal pain, inguinoscrotal lumps
Dr Jessica Lowe	cervical cancer screening
Dr Peter Lowthian	arthritis
Mr Frank Lyons	common fractures and dislocations
Dr John Mackellar	child abuse and domestic violence
Dr Linda Mann	basic antenatal care
Professor Barry McGrath	hypertension
Dr Joe McKendrick	malignant disease
Dr Kim Matthews	the subfertile couple
Dr Luke Murtagh	pain and its management
Professor Robyn O’Hehir	allergic disorders, including hayfever
Dr Michael Oldmeadow	tiredness
Dr Frank Panetta	chest pain
Dr Geoff Quail	pain in the face, sore mouth and tongue
Mr Ronald Quirk	pain in the foot and ankle
Dr Ian Rogers	emergency care
Professor Avni Sali	abdominal pain, lumps in the breast, jaundice, constipation, dyspepsia, nutrition
Dr Stanley Santiagu and Dr Jemma Dalrymple	abnormal uterine bleeding
Dr Ronald Schweitzer	intimate partner violence and sexual assault
Dr Deshan Sebaratnam and Dr Margit Polcz	problems of the skin
Dr Heidi Spillane	sexual health
Dr Hugo Standish	urinary tract infection, chronic kidney failure

Dr Richard Stark	neurological diagnostic triads
Dr Liz Sturgiss	obesity
Professor Geoff Sussman	skin ulcers
Dr Paul Tallman	stroke and transient ischaemic attacks
Dr Alison Walsh	breastfeeding, postnatal breast disorders
Professor Greg Whelan	alcohol problems, drug problems
Dr Lynne Wray	vaginal discharge, vulvar disorders
Dr Alan Yung	fever and chills, sore throat
Dr Ronnie Yuen	diabetes mellitus, thyroid and other endocrine disorders

Laboratory reference values

These reference values and ranges are given in the system of international units (SI) and may vary from laboratory to laboratory.

An asterisk (*) indicates that paediatric reference ranges differ from the adult range given.

Electrolytes/renal	
Sodium	135–145 mmol/L
Potassium*	3.5–5.0 mmol/L
Chloride	95–110 mmol/L
Bicarbonate	23–32 mmol/L
Urea	3–8.0 mmol/L
Creatinine	♀ 50–110; ♂ 60–120 µmol/L
eGFR	>60 mL/min/1.72 m2
Calcium*	2.10–2.60 mmol/L (total)
Phosphate	0.90–1.35 mmol/L
Magnesium*	0.65–1.00 mmol/L
Uric acid*	♀ 0.12–0.40; ♂ 0.15–0.45 mmol/L

Liver function/pancreas	
Bilirubin*	<20 µmol/L (total) <3 µmol/L (direct)
AST*	<40 U/L
GGT*	♀ <30; ♂ <50 U/L
Alkaline phosphatase (ALP)*	25–100 U/L
Total protein	60–80 g/L
Albumin	38–50 g/L
Amylase	30–110 U/L
Lipase	<100 U/L

Glucose	
Glucose fasting	3–5.4 mmol/L
Glucose random	3–7.7 mmol/L
HbA1c	4.7–6.1%

Haematology	
Hb*	♀ 115–165; ♂ 130–180 g/L
PCV*	♀ 37–47; ♂ 40–54%
MCV*	80–100 fL
Reticulocytes	0.5–2.0%
White cells	4.0–11.0 × 109/L
Platelets	150–400 × 109/L
ESR	<20 mm; <35mm if >70 years
Band neutrophils*	(0.05 × 109/L)
Mature neutrophils*	(2.0–7.5 × 109/L)
Lymphocytes*	(1.0–4.0 × 109/L)
Monocytes*	(0.2–0.8 × 109/L)
Eosinophils*	(0.0–0.4 × 109/L)
Folate	serum 7–45 nmol/L, red cell 360–1400 nmol/L
s Vitamin B12	(150–700 pmol/L)

Coagulation	
Bleeding time	2.0–8.5 min
Fibrinogen	2.0–4.0 g/L
Prothrombin time	sec.
Prothrombin ratio INR	1.0–1.2
APTT	25–35 sec
D-dimer	<500 mg/mL

Others	
s Creatine phospho kinase	<90 U/L
s Lead	<2 µmol/L
s C–reactive protein	<10 mg/L
Vitamin D	>75 mmol/L

Cardiac/lipids	
Troponin I or T	<0.1 ug/L
CK total	♀ <200; ♂ <220 U/L
CK-MB	<25 U/L
Cholesterol*	<5.5 mmol/L
Triglycerides*	<1.7 mmol/L
HDL cholesterol	♀ 1–2.2; ♂ 0.9–2.0 mmol/L

LDL cholesterol	2–3.4 mmol/L

Thyroid tests	
Free T_4	10.0–25.0 pmol/L
Ultra–sensitive TSH*	0.4–5.0 mU/L
Free T_3	3.3–8.2 pmol/L

Other endocrine tests	
s Cortisol	8 am 130–700 nmol/L
	4 pm 80–350 nmol/L
FSH	1–9 IU/L (adult ♀) 10–30 IU/L (ovulation) 4–200 IU/L (postmenopausal)
Oestradiol menopausal	<200 pmol/L
Testosterone	♀ <3.5; ♂ 10–35 nmol/L

Tumour markers	
PSA	0–1.0 mcg/L
CEA	<7.5 mcg/L
AFT	<10 mcg/mL
CA–125	<35 U/mL

Iron studies	
Ferritin	♀ 15–200; ♂ 30–300 mcg/L
Iron	10–30 µmol/L
Iron–binding capacity	45–80 µmol/L
Transferrin	2–3.5 g/L
Transferrin saturation	♀ 15–45%; ♂ 15–55%

Blood gases/arterial	
pH*	7.38–7.43
P_aO_2*	85–105 mmHg
P_aCO_2*	36–44 mmHg
Bicarbonate*	20–28 mmol/L
Base excess*	–3 to +3 mmol/L

Normal values: worth knowing by heart

The following is a checklist that can be used as a template to memorise normal quantitative values for basic medical conditions and management.

Vital signs (average)	< 6 months	6 months – 3 years	3 – 12 years	Adult
Pulse	120–140	110	80 – 100	60 – 100
Respiratory rate	45	30	20	14
BP (mmHg)	90/60	90/60	100/70	≤ 130/85

Children's weight	1–10 years
Rule of thumb:	Wt = (age + 4) × 2 kg

Fever—temperature (morning)[(a)] (a) There is considerable diurnal variation in temperature so that it is higher in the evening (0.5–1°C). I would recommend the definition given by Yung et al. in *Infectious Diseases: a Clinical Approach*: 'Fever can be defined as an early morning oral temperature > 37.2°C or a temperature > 37.8°C at other times of the day'. Dangerous ≥ 41.5°C.	
Oral	> 37.2°C
Rectal	> 37.7°C
Diabetes mellitus—Diagnostic criteria: blood sugar	
Random 1 reading if symptomatic 2 readings if asymptomatic	> 11.1 mmol/L
Fasting	> 7.0 mmol/L
or	the 2 values from an oral GTT
Hypokalaemia	
Serum potassium	< 3.5 mmol/L
Jaundice	
Serum bilirubin	> 19 µmol/L
Hyperkalaemia	
Serum potassium	> 5.0 mmol/L
Hypertension	
BP	> 140/90 mmHg
Alcohol excessive drinking	
Males	> 4 standard drinks/day
Females	> 2 standard drinks/day
Alcohol health guidelines (NHMRC)	
Males and females	≤ 10 standard drinks/week < 4 standard drinks/occasion

Anaemia—haemoglobin	
Males	< 130 g/L
Females	< 120 g/L
Body mass index	**Wt (kg)/Ht (m2)**
Normal	20–25
Overweight	> 25
Obesity	> 30

Abbreviations

AAA	abdominal aortic aneurysm
AAFP	American Academy of Family Physicians
ABA	Australian Breastfeeding Association
ABC	airway, breathing, circulation
ABCD	airway, breathing, circulation, dextrose
ABFP	American Board of Family Practice
ABI	ankle brachial index
ABO	A, B and O blood groups
AC	air conduction
AC	acromioclavicular
ACAH	autoimmune chronic active hepatitis
ACE	angiotensin-converting enzyme
ACL	anterior cruciate ligament
ACR	albumin creatine ratio
ACTH	adrenocorticotrophic hormone
AD	aortic dissection
AD	autosomal dominant
ADHD	attention deficit hyperactivity disorder
ADLs	activities of daily living
ADT	adult diphtheria vaccine
AF	atrial fibrillation
AFI	amniotic fluid index
AFP	alpha-fetoprotein
AI	aortic incompetence
AICD	automatic implantable cardiac defibrillator
AIDS	acquired immunodeficiency syndrome
AIIRA	angiotension II(2) reuptake antagonist
AKF	acute kidney failure
ALE	average life expectancy
ALL	acute lymphocytic leukaemia
ALP	alkaline phosphatase
ALT	alanine aminotransferase
ALTE	apparent life-threatening episode
AMI	acute myocardial infarction
AML	acute myeloid leukaemia
ANA	antinuclear antibody
ANCA	antineutrophil cytoplasmic antibody
ANF	antinuclear factor
a/n/v	anorexia/nausea/vomiting
AP	anterior–posterior
APF	Australian pharmaceutical formulary
APH	ante-partum haemorrhage
APRI	AST to platelet ratio index
aPTT	activated partial thromboplastin time
AR	autosomal recessive
ARB	angiotension II receptor blocker
ARC	AIDS-related complex
ARDS	adult respiratory distress syndrome

ARR	absolute risk reduction
ART	anti-retroviral therapy
ASD	atrial septal defect
ASIS	anterior superior iliac spine
ASOT	antistreptolysin 0 titre
AST	aspartate aminotransferase
ATFL	anterior talofibular ligament
AV	atrioventricular
AVM	arteriovenous malformation
AZT	azidothymidine

BC	bone conduction
BCC	basal cell carcinoma
BCG	bacille Calmette–Guérin
bDMARDs	biological disease modifying antirheumatic drugs
BMD	bone mass density
BMI	body mass index
BNP	B-type natriuretic peptide
BOO	bladder outlet obstruction
BP	blood pressure
BPH	benign prostatic hyperplasia
bpm	beats per minute
BPPV	benign paroxysmal positional vertigo
BSE	breast self-examination

Ca	carcinoma
CABG	coronary artery bypass grafting
CAD	coronary artery disease
CAP	community-acquired pneumonia
CBE	clinical breast examination
CBT	cognitive behaviour therapy
CCB	calcium-channel blocker
CCF	congestive cardiac failure
CCP	cyclic citrinullated peptide
CCT	controlled clinical trial
CCU	coronary care unit
CD4	T helper cell
CD8	T suppressor cell
CDT	combined diphtheria/tetanus vaccine
CEA	carcinoembryonic antigen
CFL	calcaneofibular ligament
CFS	chronic fatigue syndrome
cfu	colony forming unit
CHC	combined hormonal contraception
CHD	coronary heart disease
CHF	chronic heart failure
CI	confidence interval
CIN	cervical intraepithelial neoplasia
CJD	Creutzfeldt–Jakob disease
CK	creatinine kinase
CK–MB	creatinine kinase–myocardial bound fraction
CKD	chronic kidney disease
CKF	chronic kidney failure

CMC	carpometacarpal
CML	chronic myeloid leukaemia
CMV	cytomegalovirus
CNS	central nervous system
co	compound
COAD	chronic obstructive airways disease
COC	combined oral contraceptive
COCP	combined oral contraceptive pill
COMT	catechol-O-methyl transferase
COPD	chronic obstructive pulmonary disease
COX	cyclooxygenase
CPA	cardiopulmonary arrest
CPAP	continuous positive airways pressure
CPK	creatine phosphokinase
CPPD	calcium pyrophosphate dihydrate
CPR	cardiopulmonary resuscitation
CPS	complex partial seizures
CR	controlled release
CRD	computerised reference database system
CREST	calcinosis cutis; Raynaud phenomenon; oesophageal involvement; sclerodactyly; telangiectasia
CRF	chronic renal failure
CRFM	chloroquine-resistant falciparum malaria
CRH	corticotrophin-releasing hormone
CR(K)F	chronic renal (kidney) failure
CRP	C-reactive protein
CSF	cerebrospinal fluid
CSFM	chloroquine-sensitive falciparum malaria
CSIs	COX-2 specific inhibitors
CSU	catheter specimen of urine
CT	computerised tomography
CTD	connective tissue disorder
CTG	cardiotocograph
CTS	carpal tunnel syndrome
CVA	cerebrovascular accident
CVS	cardiovascular system
CXR	chest X-ray

DAA	direct-acting antivirals
DBP	diastolic blood pressure
DC	direct current
DDAVP	desmopressin acetate
DDH	developmental dysplasia of the hip
DDP	dipeptidyl peptidase
DEXA	dual energy X-ray absorptiometry
DHA	docosahexaenoic acid
DHEA	dihydroepiandrosterone
DI	diabetes insipidus
DIC	disseminated intravascular coagulation
DIDA	di-imino diacetic acid
DIMS	disorders of initiating and maintaining sleep
DIP	distal interphalangeal
dL	decilitre
DMARDs	disease modifying antirheumatic drugs
DNA	deoxyribose-nucleic acid

DOACs	direct acting anti-coagulants
DOM	direction of movement
DRE	digital rectal examination
DRABC	defibrillation, resuscitation, airway, breathing, circulation
drug dosage	bd—twice daily; tid, tds—three times daily; qid—four times daily
ds	double strand
DS	double strength
DSM	diagnostic and statistical manual (of mental disorders)
DU	duodenal ulcer
DUB	dysfunctional uterine bleeding
DVT	deep venous thrombosis
DxT	diagnostic triad

EAR	expired air resuscitation
EBM	Epstein–Barr mononucleosis (glandular fever)
EBNA	Epstein–Barr nuclear antigen
EBV	Epstein–Barr virus
ECC	external chest compression
ECG	electrocardiogram
ECT	electroconvulsive therapy
ED	emergency department
EDD	expected due date
EEG	electroencephalogram
ELISA	enzyme-linked immunosorbent assay
EMG	electromyogram
ENA	extractable nuclear antigen
EO	ethinyloestradiol
EPA	eicosapentaenoic acid
EPL	extensor pollicis longus
EPS	expressed prostatic secretions
ER	external rotation
ESRF	end-stage renal failure
ESR(K)F	end-stage renal (kidney) failure
ERCP	endoscopic retrograde cholangiopancreatography
esp.	especially
ESR	erythrocyte sedimentation rate
ET	embryo transfer
ETT	endotracheal tube

FAD	familial Alzheimer disease
FAI	femeroacetabular impingement
FAP	familial adenomatous polyposis
FB	foreign body
FBE	full blood count
FDIU	fetal death in utero
FDL	flexor digitorum longus
FEV_1	forced expiratory volume in 1 second
FHL	flexor hallucis longus
fL	femto-litre (10–15)
FOBT	faecal occult blood test
FRAX	fracture risk assessment tool
FRC	functional residual capacity
FSH	follicle stimulating hormone

FTA–ABS	fluorescent treponemal antibody absorption test
FTT	failure to thrive
FUO	fever of undetermined origin
FVC	forced vital capacity
FXS	fragile X syndrome

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g	gram
GA	general anaesthetic
GABHS	group A beta-haemolytic streptococcus
GBS	Guillain–Barré syndrome
GCA	giant cell arteritis
GESA	Gastroenterological Society of Australia
GFR	glomerular filtration rate
GGT	gamma-glutamyl transferase
GHJ	glenohumeral joint
GI	glycaemic index
GIFT	gamete intrafallopian transfer
GIT	gastrointestinal tract
GLP	glucagon-like peptide
GnRH	gonadotrophin-releasing hormone
GO	gastro-oesophageal
GORD	gastro-oesophageal reflux disease
GP	general practitioner
G-6-PD	glucose-6-phosphate dehydrogenase
GSI	genuine stress incontinence
GU	gastric ulcer
GV	growth velocity

HAV	hepatitis A virus
anti-HAV	hepatitis A antibody
Hb	haemoglobin
HbA	haemoglobin A
anti-HBc	hepatitis B core antibody
HBeAg	hepatitis Be antigen
anti-HBs	hepatits B surface antibody
HBsAg	hepatitis B surface antigen
HBV	hepatitis B virus
HCG	human chorionic gonadotropin
HCV	hepatitis C virus
anti-HCV	hepatitis C virus antibody
HDL	high-density lipoprotein
HDV	hepatitis D (Delta) virus
HEV	hepatitis E virus
HFA	hydrofluoro alkane
HFM	hand, foot and mouth
HFV	hepatitis F virus
HGV	hepatitis G virus
HHC	hereditary haemochromatosis
HIDA	hydroxy iminodiacetic acid
HIV	human immunodeficiency virus
HLA-B27	human leucocyte antigen
HMGCoA	hydroxymethylglutaryl CoA
HNPCC	hereditary non-polyposis colorectal cancer
HPV	human papilloma virus

HRT	hormone replacement therapy
HSIL	high-grade squamous intraepithelial lesion
HSP	Henoch–Schönlein purpura
HSV	herpes simplex viral infection
H	hypertension

IBS	irritable bowel syndrome
ICE	ice, compression, elevation
ICHPPC	International Classification of Health Problems in Primary Care
ICS	inhaled corticosteroid
ICS	intercondylar separation
ICSI	intracytoplasmic sperm injection
ICT	immunochromatographic test
IDDM	insulin dependent diabetes mellitus
IDU	injecting drug user
IgA	immunoglobulin A
IgE	immunoglobulin E
IgG	immunoglobulin G
IgM	immunoglobulin M
IGRA	interferon gamma release assay
IHD	ischaemic heart disease
IHS	International Headache Society
IM, IMI	intramuscular injection
IMS	intermalleolar separation
inc.	including
INCS	intranasal corticosteroids
INR	international normalised ratio
IOC	International Olympic Committee
IOFB	intraocular foreign body
IP	interphalangeal
IPPV	intermittent positive pressure variation
IR	internal rotation *or* immediate release
ITP	idiopathic (or immune) thrombocytopenia purpura
IUCD	intrauterine contraceptive device
IUGR	intrauterine growth retardation
IV	intravenous
IVF	in-vitro fertilisation
IVI	intravenous injection
IVP	intravenous pyelogram
IVU	intravenous urogram

JIA	juvenile idiopathic arthritis
JVP	jugular venous pulse

KA	keratoacanthoma
KFT	kidney function test
kg	kilogram
KOH	potassium hydroxide
KS	Kaposi sarcoma
KUB-CT	kidney ureter bladder scan

LA	local anaesthetic

LABA	long-acting beta agonist
LBBB	left branch bundle block
LBO	large bowel obstruction
LBP	low back pain
LCR	ligase chain reaction
LDH/LH	lactic dehydrogenase
LDL	low-density lipoprotein
LFTs	liver function tests
LH	luteinising hormone
LHRH	luteinising hormone releasing hormone
LIF	left iliac fossa
LMN	lower motor neurone
LNG	levonorgestrel
LPC	liquor picis carbonis
LRTI	lower respiratory tract infection
LSD	lysergic acid
LSIL	low-grade squamous intraepithelial lesion
LSS	lumbar spinal canal stenosis
LUQ	left upper quadrant
LUT	lower urinary tract
LUTS	lower urinary tract symptoms
LV	left ventricular
LVH	left ventricular hypertrophy

MAIS	*Mycobacterium avium intracellulare* or *M. sacrofulaceum*
mane	in morning
MAOI	monoamine oxidase inhibitor
MAST	medical anti-shock trousers
MB	myocardial base
mcg	micrograms (also µg)
MCL	medial collateral ligament
MCP	metacarpal phalangeal
MCU	microscopy and culture of urine
MCV	mean corpuscular volume
MDI	metered dose inhaler
MDMA	methylenedioxymethamphetamine
MDR	multi-drug resistant TB
MG	myaesthenia gravis
MHT	menopause hormone therapy
MI	myocardial infarction
MIC	mitral incompetence
MID	minor intervertebral derangement
MMSE	mini mental state examination
MND	motor neurone disease
MRCP	magnetic resonance cholangiopancreatography
MRI	magnetic resonance imaging
MRSA	methicillin-resistant *staphylococcus aureus*
MS	multiple sclerosis
MSM	men who have sex with men
MSST	maternal serum screening test
MSU	midstream urine
MTP	metatarsophalangeal
MVA	motor vehicle accident

N	normal
N saline	normal saline
NAAT	nucleic acid amplification technology
NAD	no abnormality detected
NCDs	non-communicable diseases
NET	norethisterone
NF	neurofibromatosis
NGU	non-gonococcal urethritis
NHL	non-Hodgkin lymphoma
NH&MRC	National Health and Medical Research Council
NIDDM	non-insulin dependent diabetes mellitus
NNT	numbers needed to treat
nocte	at night
NR	normal range
NRT	nicotine replacement therapy
NSAIDs	non-steroidal anti-inflammatory drugs
NSCLC	non-small cell lung cancer
NSTEACS	non-ST segment elevation acute coronary syndrome
NSU	non-specific urethritis
NTT	nuchal translucency test
NVDPA	National Vascular Disease Prevention Alliance

(o)	taken orally
OA	osteoarthritis
OCP	oral contraceptive pill
OGTT	oral glucose tolerance test
OSA	obstructive sleep apnoea
OSD	Osgood–Schlatter disorder
OT	occupational therapist
OTC	over the counter

PA	posterior–anterior
PAD	peripheral arterial disease
PAN	polyarteritis nodosa
Pap	Papanicolaou
PBG	porphobilinogen
PBS	Pharmaceutical Benefits Scheme
pc	after meals
PCA	percutaneous continuous analgesia
PCB	post coital bleeding
PCI	percutaneous coronary intervention
PCL	posterior cruciate ligament
PCOS	polycystic ovarian syndrome
PCP	pneumocystitis pneumonia
PCR	polymerase chain reaction
PCV	packed cell volume
PD	Parkinson disease
PDA	patent ductus arteriosus
PDD	pervasive development disorders
PEF	peak expiratory flow
PEFR	peak expiratory flow rate
PET	pre-eclamptic toxaemia
PET	positron emission tomography
PFO	patent foramen ovale

PFT	pulmonary function test
PGL	persistent generalised lymphadenopathy
PH	past history
PHR	personal health record
PID	pelvic inflammatory disease
PIP	proximal interphalangeal
PJP	pneumocystis jirovecii pneumonia
PKU	phenylketonuria
PLISSIT	permission: limited information: specific suggestion: intensive therapy
PLMs	periodic limb movements
PMDD	premenstrual dysphoric disorder
PMS	premenstrual syndrome
PMT	premenstrual tension
PaO2	partial pressure oxygen (arterial blood)
POP	plaster of Paris
POP	progestogen-only pill
PPI	proton-pump inhibitor
PPROM	preterm premature rupture of membranes
PR	per rectum
prn	as and when needed
PRNG	penicillin-resistant gonococci
PROM	premature rupture of membranes
PSA	prostate specific antigen
PSGN	post streptococcal glomerulonephritis
PSIS	posterior superior iliac spine
PSVT	paroxysmal supraventricular tachycardia
PT	prothrombin time
PTC	percutaneous transhepatic cholangiography
PTCA	percutaneous transluminal coronary angioplasty
PTFL	posterior talofibular ligament
PU	peptic ulcer
PUO	pyrexia of undetermined origin
PUVA	psoralen + UVA
pv	per vagina
PVC	polyvinyl chloride
PVD	peripheral vascular disease

RA	rheumatoid arthritis
RACGP	Royal Australian College of General Practitioners
RAP	recurrent abdominal pain
RBBB	right branch bundle block
RBC	red blood cell
RCT	randomised controlled trial
RF	rheumatic fever
Rh	rhesus
RIB	rest in bed
RICE	rest, ice, compression, elevation
RIF	right iliac fossa
RPR	rapid plasma reagin
RR	relative risk
RRR	relative risk reduction
RSD	reflex sympathetic dystrophy

RSI	repetition strain injury
RSV	respiratory syncytial virus
RT	reverse transcriptase
rtPA	recombinant tissue plasminogen activator
RUQ	right upper quadrant

s	serum
SABA	short-acting beta agonist
SAH	subarachnoid haemorrhage
SARS	severe acute respiratory distress syndrome
SBE	subacute bacterial endocarditis
SBO	small bowel obstruction
SBP	systolic blood pressure
SC/SCI	subcutaneous/subcutaneous injection
SCC	squamous cell carcinoma
SCFE	slipped capital femoral epiphysis
SCG	sodium cromoglycate
SCLC	small cell lung cancer
SERM	selective estrogen receptor modulator
SIADH	syndrome of secretion of inappropriate antidiuretic hormone
SIDS	sudden infant death syndrome
SIJ	sacroiliac joint
SL	sublingual
SLD	specific learning disability
SLE	systemic lupus erythematosus
SLR	straight leg raising
SND	sensorineural deafness
SNHL	sensorineural hearing loss
SNPs	single nucleotide polymorphisms
SNRI	serotonin noradrenaline reuptake inhibitor
SOB	shortness of breath
SLS	salt-losing state
sp	species
SPA	suprapubic aspirate of urine
SPECT	single photon emission computerised tomography
SPF	sun penetration factor
SR	sustained release
SSRI	selective serotonin reuptake inhibitor
SSS	sick sinus syndrome
statim	at once
STEMI	ST segment elevation myocardial infarction
STI	sexually transmitted infection
STS	sodium tetradecyl sulfate
SUFE	slipped upper femoral epiphysis
SVC	superior vena cava
SVT	supraventricular tachycardia

T_3	tri-iodothyronine
T_4	thyroxine
TA	temporal arteritis
TB	tuberculosis
TCA	tricyclic antidepressant
tds, tid	three times daily
TENS	transcutaneous electrical nerve stimulation

TFTs	thyroid function tests
TG	triglyceride
TIA	transient ischaemic attack
TIBC	total iron binding capacity
TM	tympanic membrane
TMJ	temporomandibular joint
TNF	tissue necrosis factor
TOE	transoesophageal echocardiography
TOF	tracheo-oesophageal fistula
TORCH	toxoplasmosis, rubella, cytomegalovirus, herpes virus
TPHA	Treponema pallidum haemaglutination test
TSE	testicular self-examination
TSH	thyroid-stimulating hormone
TT	thrombin time
TUE	therapeutic use exemption
TUIP	transurethral incision of prostate
TURP	transurethral resection of prostate
TV	tidal volume

U	units
UC	ulcerative colitis
U & E	urea and electrolytes
UGIB	upper gastrointestinal bleeding
µg	microgram
UMN	upper motor neurone
URT	upper respiratory tract
URTI	upper respiratory tract infection
US	ultrasound
UTI	urinary tract infection
U	ultraviolet

VAD	voluntary assisted dying
VAS	visual analogue scale
VBI	vertebrobasilar insufficiency
VC	vital capacity
VDRL	Venereal Disease Reference Laboratory
VF	ventricular fibrillation
VMA	vanillylmandelic acid
VPG	venous plasma glucose
VRE	vancomycin-resistant enterococci
VSD	ventricular septal defect
VT	ventricular tachycardia
VUR	vesicoureteric reflux
VVS	vulvar vestibular syndrome
vWD	von Willebrand disease

WBC	white blood cells
WBR	white → blue → red
WCC	white cell count
WHO	World Health Organization
WPW	Wolff–Parkinson–White

XL sex linked

Part 1 The basis of general practice

1 The nature, scope and content of general practice

Medical practice is not knitting and weaving and the labour of the hands, but it must be inspired with soul and be filled with understanding and equipped with the gift of keen observation; these together with accurate scientific knowledge are the indispensable requisites for proficient medical practice.

MOSES BEN MAIMON (1135–1204)

General practice is a traditional method of bringing primary health care to the community. It is a medical discipline in its own right, linking the vast amount of accumulated medical knowledge with the art of communication.

Definitions

General practice can be defined as that medical discipline which provides 'community-based, continuing, comprehensive, preventive primary care', sometimes referred to as the CCCP model. It is regarded as synonymous with primary care and family practice.

The Royal Australian College of General Practitioners (RACGP) uses the following definitions of general practice and primary care:

> General practice is that component of the health care system which provides initial, continuing, comprehensive and coordinated medical care for all individuals, families and communities and which integrates current biomedical, psychological and social understandings of health.
>
> A general practitioner is a medical practitioner with recognised generalist training, experience and skills, who provides and coordinates comprehensive medical care for individuals, families and communities.
>
> General/family practice is the point of first contact for the majority of people seeking health care. In the provision of primary care, much ill-defined illness is seen; the general/family practitioner often deals with problem complexes rather than with established diseases.

The RACGP has defined five domains of general practice:

- communication skills and the doctor–patient relationship
- applied professional knowledge and skills
- population health and the context of general practice
- professional and ethical role
- organisational and legal dimensions

Furthermore the RACGP has identified eight core characteristics of general practice:

1. whole person care
2. person centredness
3. continuity of care
4. comprehensiveness
5. diagnostic and therapeutic skills
6. a command of complexity and uncertainty
7. coordinated clinical teamwork
8. leadership, advocacy and equity

Additional functions of primary health care promoted by the American College of Family Physicians (AAFP).[1,2]

- First contact care including the early diagnosis of acute disease
- Continuity of care for the individual patient, their family and his/her environment
- Highly personalised care
- Care of chronic disease
- Gatekeeper care or co-ordinating role drawing on traditional major disciplines
- Community health awareness

General practice is fundamentally relational, based on the doctor having a deep understanding of the whole person and the ability to manage complex conditions and circumstances. The general practitioner functions as a physician, counsellor, advocate and agent of change for individuals, families and their communities.[3]

General practice is not the summation of specialties practised at a superficial level and we must avoid the temptation to become ‘specialoids’. In the current climate, where medicine is often fragmented, there is a greater than ever need for the generalist. The patient requires a trusted focal point in the often bewildering health service jungle. Who is to do this

better than the caring family doctor taking full responsibility for the welfare of the patient and intervening on his or her behalf? Specialists also need highly competent generalists to whom they can entrust ongoing care.

Unique features of general practice

Anderson, Bridges-Webb and Chancellor[4] emphasise that ‘the unique and important work of the general practitioner is to provide availability and continuity of care, competence in the realm of diagnosis, care of acute and chronic illness, prompt treatment of emergencies and a preventive approach to health care’.

The features that make general practice different from hospital- or specialist-based medical practices include:

- first contact
- compassion
- diagnostic methodology
- early diagnosis of life-threatening and serious disease
- continuity and availability of care
- personalised care
- care of acute and chronic illness
- domiciliary care
- emergency care (prompt treatment at home or in the community)
- family care
- palliative care (at home)
- preventive care
- scope for health promotion and patient education
- holistic approach to management
- health care coordination

The GP has to be prepared for any problem that comes in the door (see FIG. 1.1).

FIGURE 1.1 Degrees of care of health

Apart from these processes the GP has to manage very common problems including a whole variety of problems not normally taught in medical school or in postgraduate programs. Many of these problems are unusual yet common and can be regarded as the ‘nitty gritty’ or ‘bread and butter’ problems of primary health care.

In considering the level of care of symptoms, 25% of patients abandon self-care for a visit to the GP. Ninety per cent of these visits are managed entirely within primary care. Levels of care are represented in FIGURE 1.1 .[5]

Holistic approach to management

The management of the whole person, or the holistic approach, is an important approach to patient care in general practice. Whole-person diagnosis is based on two components:

1. the disease-centred diagnosis
2. the patient-centred diagnosis

The disease-centred consultation is the traditional medical model based on the history, examination and special investigations, with the emphasis on making a diagnosis and treating the disease. The disease-centred diagnosis, which is typical of hospital-based medicine, is defined in terms of pathology and does not focus significantly on the feelings or circumstances of the person suffering from the disease.

> Whole-person care—mind and body—is the hallmark of the good GP.

The patient-centred consultation not only takes into account the diagnosed disease and its management but also adds another dimension—that of the psychosocial hallmarks of the patient, including details about:

- the patient as a person
- emotional reactions to the illness
- the family
- the effect on relationships
- work and leisure

- lifestyle
- the environment

Taylor and colleagues, in their patient-centred model of health care, emphasise six interactive components of the patient-centred process:[6]

1. exploring both the disease and the illness experience
2. understanding the whole person
3. finding common grounds regarding management
4. incorporating prevention and health promotion
5. enhancing the doctor–patient relationship
6. being realistic regarding time and resources

Contemporary general practice focuses on patient-centred medicine, which, in alliance with evidence-based medicine, benefits both patient and doctor.

Continuing care

The essence of general practice is continuity of care. The doctor–patient relationship is unique in general practice in the sense that it covers a span of time that is not restricted to a specific major illness. The continuing relationship involving many separate episodes of illness provides an opportunity for the doctor to develop considerable knowledge and understanding of the patient, the family and its stresses, and the patient's work and recreational environment.

The epidemiological work of Barbara Starfield and others demonstrates that most of the important population health outcomes are more associated with access to primary health care than they are to specialist services. In other words, if a society (rich or poor) wishes to reduce the number of heart attacks, cancer deaths or infant mortality, it is more effective to improve access to GPs than it is to cardiologists, oncology centres or neonatal units. According to Starfield and her colleagues, 'The evidence also shows that primary care (in contrast to specialty care) is associated with a more equitable distribution of health in populations.'[7]

In 2008 the World Health Organization (WHO) reaffirmed the global importance of primary health care with its landmark report *Primary Health Care*: *Now More Than Ever*. WHO[8] highlighted the evidence that continuity of care through general practice contributed to the following better outcomes:

- lower all-cause morbidity
- better access to care
- fewer rehospitalisations
- fewer consultations with specialists
- less use of emergency services
- better detection of adverse effects of medication interventions

Home visits

'You don't know your patient until you have seen them in their home.' Home visits are a goldmine of information about intrafamily dynamics. They should cement the doctor–patient relationship if used appropriately. GPs are the only doctors who practise domiciliary care.

Common presenting problems

Common presenting symptoms in Australian general practices (BEACH study, 2013) are presented in TABLE 1.1 [9] (note that the top 15 problems represent only one-third of all encounters).

Table 1.1 Most frequent presenting problems in Australian general practice

		Per cent of problems
1	Hypertension	5.7
2	Immunisation	4.2
3	URTI	3.3
4	Depression	2.9
5	Diabetes	2.3
6	Lipid disorders	2.1
7	General check-up	1.9
8	Osteoarthritis	1.7
9	Back pain	1.7
10	Prescription	1.6
11	Oesophageal (inc. GORD)	1.6
12	Female genital check-up	1.5
13	Acute bronchitis/bronchiolitis	1.5
14	Asthma	1.3
15	Anxiety	1.2
Cumulative top 15		34.6

Source: Cooke et al.[9]

To cover 75% of presenting problems, GPs must be able to diagnose and manage more than 100 different problems, and to cover 85% requires a good working knowledge of 167 problems.[9] The breadth of knowledge required to become a proficient GP is tremendous.

The content of this book reflects what is fundamental to the nature and content of general practice—that which is common but is significant, relevant, preventable and treatable.

Symptoms and conditions related to litigation

Medical defence organisations have highlighted the following areas as being those most vulnerable for management mishaps:

- acute abdominal pain
- acute chest pain
- breast lumps

- children’s problems, especially the sick febrile child <2 years, groin pain and lumps
- dyspnoea ± cough (?heart failure, cancer, TB)
- headache

The most common reasons for claims against GPs are:

- diagnostic error 38%
- procedural complications 18%
- treatment issues 16%
- general duty of care 14%
- medication-related issues 9%
- legal issues 2%
- consent issues 1%
- medicolegal reports 1%
- anaesthesia 1%

Source: S Bird, MDA National

Chronic disease management

A study of international target conditions[10,11] in chronic disease management has highlighted the importance of the following (as common themes):

- ischaemic heart disease
- chronic heart failure
- cerebrovascular disease
- hypertension
- type 2 diabetes
- chronic obstructive pulmonary disease
- asthma
- obesity

- epilepsy
- hypothyroidism
- chronic mental illness, especially depression
- medication monitoring
- arthritis

The family

Working with families is the basis of family practice, and families living in relative harmony provide the basis for the good mental health of their members and also for social stability.

Families take many shapes and forms, among them single-parent households, de facto partnerships, three-generational households, same-sex couples and families formed by a partnership between two separated parents and their children. Psychosocial problems may occur in almost any family arrangement and family doctors need to know how to address such problems.

Family therapy is ideally undertaken by GPs, who are in a unique position as providers of continuing care and family care. It is important for them to work together with families in the counselling process and to avoid the common pitfalls of working in isolation and assuming personal responsibility for changing the family. We should understand that definitions of family vary greatly across cultures.

Bader[12] summarises working with families succinctly:

> From the perspective of family therapy, working with families means avoiding the trap of being too directive, too responsible for the family's welfare, with the result that the family becomes overly dependent on the general practitioner for its health and development. From the perspective of family education, working with families means developing the skills of anticipating guidance, helping families to prepare, not only for the normal changes occurring as the family develops, but also for the impact of illness on the family system.

Families in crisis

Doctors are closely involved with families who experience unexpected crises, which include illnesses, accidents, divorce, separation, unemployment, death of a family member and financial disasters.

The effect of illness

Serious illness often precipitates crises in individual members of the family, crises that have not previously surfaced in the apparently balanced family system. For example, bereavement over the unexpected loss of a child may lead to marital breakdown.

In the long term, other family members may be affected more than the patient. This may apply particularly to children and manifest as school underachievement and behaviour disturbances.

During the crisis the obvious priority of the doctor is to the patient, but the less obvious needs of the family should not be ignored.

Guidelines for the doctor

- Include the family as much as possible, starting early in the acute phase of the illness. This may necessitate family conferences.
- Include the family on a continuing basis, especially if a long-term illness is anticipated. It is helpful to be alert for changes in attitudes, such as anger and resentment towards the sick member.

Page 6

- Include the family in hospital discharge planning.
- If a serious change in family dynamics is observed, the use of experts may be needed.
- Offer a family conference at critical times.

Significant presentations of family dysfunction

The following presentations may be indicators that all is not well, and the doctor needs to ‘think family’:

- relationship or sexual difficulties
- multiple presentations by multiple family members
- abnormal behaviour in a child
- the ‘difficult patient’
- inappropriate behaviour in the antenatal and/or postpartum period
- drug or alcohol abuse in a family member
- evidence of physical or sexual abuse in one of the partners (male or female) or a child
- psychiatric disorders, especially depression and psychosis
- increased stress/anxiety
- complaints of chronic fatigue or insomnia

It is important that the family doctor remains alert to the diversity of presentations and takes the

responsibility for identifying an underlying family-based problem.

The patient and family dynamics

Family doctors see many people who present with physical symptoms that have primarily an emotional or psychosocial basis with either little or no organic pathology. In order to understand the clinical manifestations of the sick role of patients, family doctors should first understand the individual's response to stress stimuli, which may come from external (family, work or sexual behaviour) or internal (personality trait or psychosocial) sources (see FIG. 1.2 and TABLE 1.2).

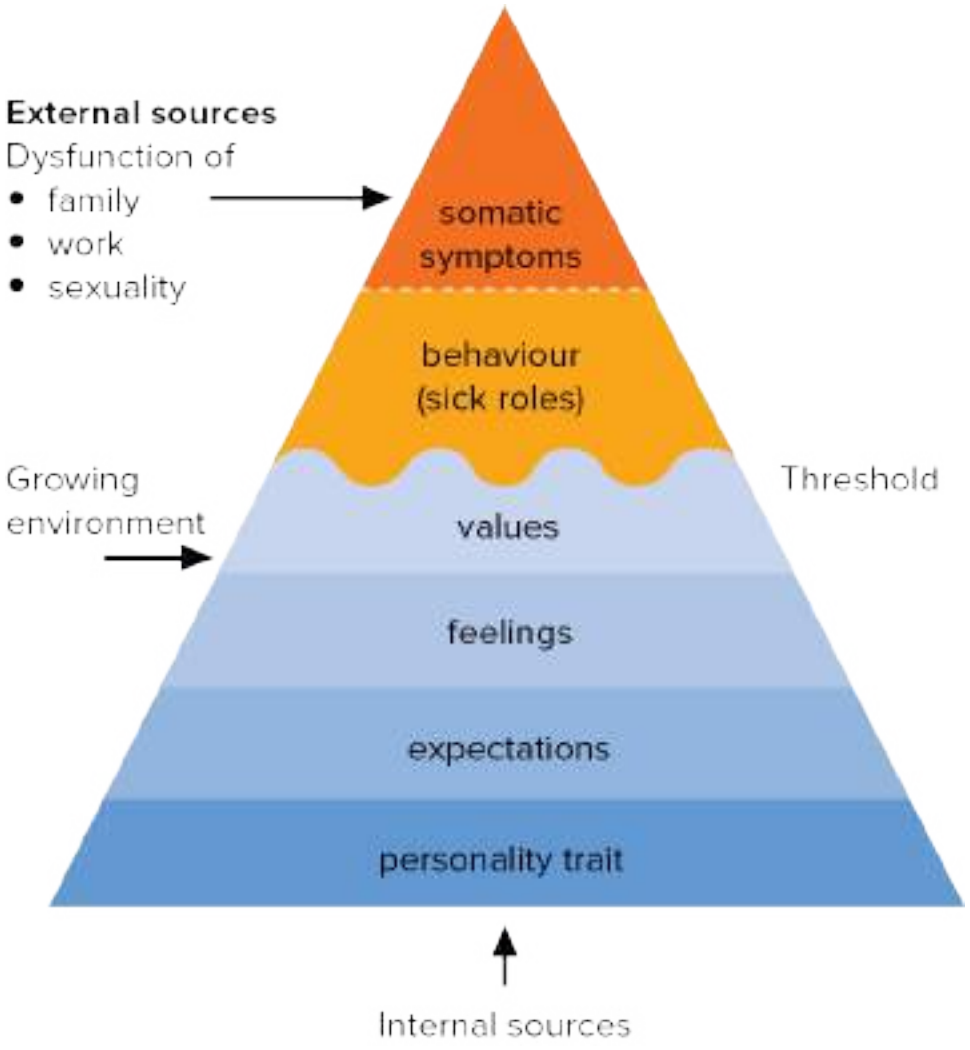

FIGURE 1.2 Family dynamics and psychosomatic illness iceberg

Table 1.2 Areas of possible biopsychosocial dysfunction

Work	Family	Sex
Type of work Workload Work environment Goals Work satisfaction	Present family (change of structure and function) Extended family (parents and relatives) Growing environment (family tree)	Sexual dysfunction Disharmony Deprivation Guilt

How to evaluate the family dynamics

- Carefully observe family members interacting.
- Invite the whole family to a counselling session (if possible).
- Visit the home: a visit on the way home from work may be very revealing. This will be appropriate in some but not all family practice settings.
- Prepare a genogram (see FIG. 23.1 , CHAPTER 23): family dynamics and behaviour can be understood by drawing a family map or genogram (a diagrammatic representation of family structure and relationships).[13,14]

The genogram

The genogram is a very valuable pedigree chart that usually covers three generations of a family tree.[13] Such a visual framework helps the general practice consultation as you can continue to build on that basic framework. Copies can be given to patients and families to reflect on at home and return to their GP for further insights.[15] Genograms are a useful strategy for involving family members who may have been reluctant to be involved in discussions on family matters.[14] An example, including the use of symbols, is shown in FIGURE 23.1 (refer to CHAPTER 23).

The family life cycle

Helpful in understanding the dynamics of the family is the concept of the family life cycle,[16] which identifies several clearly defined stages of development (see TABLE 1.3). Such an understanding can help the doctor form appropriate hypotheses about the problems patients are experiencing at a particular stage. Each stage brings its own tasks, happiness, crises and difficulties. This cycle is also well represented in FIGURE 1.3 , which indicates the approximate length of time on each of the stages.

Table 1.3 The family life cycle[12]

1.	Leaving home	Establishing personal independence. Beginning the emotional separation from parent(s).
2.	Getting married, entering de facto	Establishing an intimate relationship with spouse/partner. Developing further the emotional separation from parent(s).
3.	Learning to live together	Dividing the various relationship roles in an equitable way. Establishing a new, more independent relationship with family.
4.	Parenting the first child	Opening the family to include a new member. Dividing the parenting roles.
5.	Living with the adolescent	Increasing the flexibility of the family boundaries to allow the adolescent(s) to move in and out of the family system.
6.	Launching children: the empty-nest phase	Accepting the multitude of exits from and entries into the family system. Adjusting to the ending of parenting roles.
7.	Retirement	Adjusting to the ending of the wage-earning roles. Developing new relationships with children, grandchildren and each other.
8.	Old age	Dealing with lessening abilities and greater dependence on others. Dealing with losses of friends, family members and, eventually, each other.

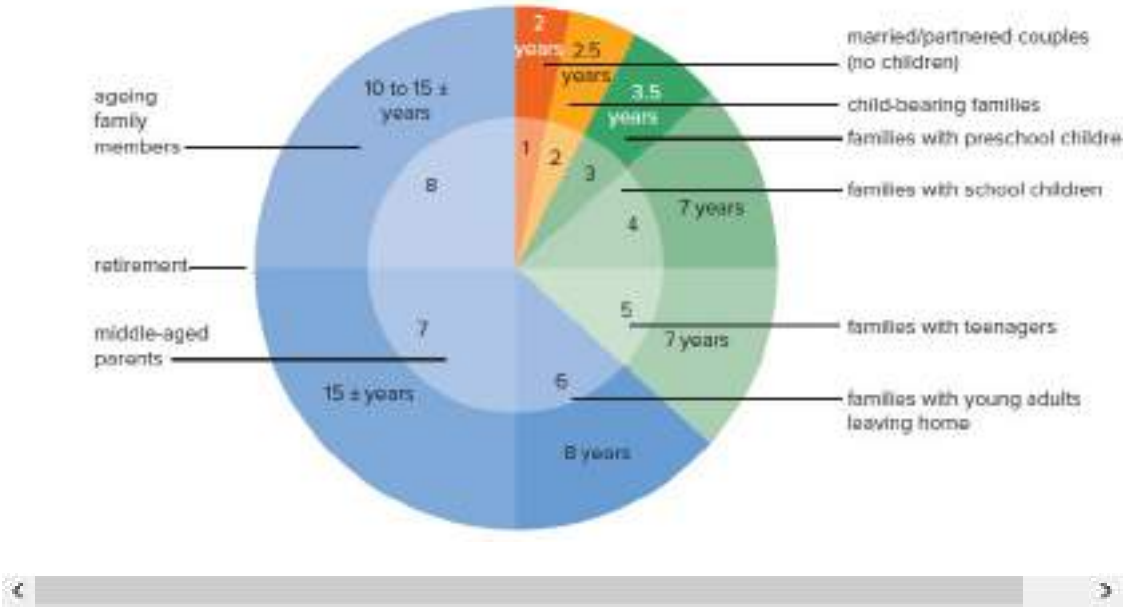

FIGURE 1.3 The family life cycle (approximate time in each stage)[17, 18]

Resources

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2 Consulting skills

The essential unit of medical practice is the occasion when in the intimacy of the consulting room the person who is ill or believes himself (or herself) to be ill, seeks the advice of a doctor whom he (she) trusts. This is the consultation and all else in the practice of medicine derives from it.

SIR JAMES SPENCE 1960

The *objectives of the consultation* are to:

- determine the exact reason for the presentation
- achieve a good therapeutic outcome for the patient
- develop a strong doctor–patient relationship

The skills of general practice

A successful outcome to the medical consultation depends on a whole array of skills required by the GP. Although interrelated, these skills, which can be collectively termed ‘consulting skills’, include interviewing skills, clinical skills, diagnostic skills, management skills, communication skills, educative skills, therapeutic skills, manual skills and counselling skills.

Communication skills, which are fundamental to consulting skills, are the key to the effectiveness of the doctor as a professional, and expertise with these skills is fundamental to the doctor–patient relationship. Communication skills are essential in obtaining a good history and constitute one of the cornerstones of therapy (see CHAPTER 3).

A skilled interviewer will succeed in transmitting his or her findings to the patient so that they are clearly understood, are not unduly disturbing, and inspire trust and confidence in the physician.

Models of the consultation

Several models that formalise the general practice consultation can be very useful for developing

an understanding of the process of the consultation. Two classic models are those by Pendleton and colleagues,[1] and by Stott and Davis.[2] Pendleton and colleagues, in their landmark book *The Consultation: An Approach to Learning and Teaching*,[1] defined seven key tasks to the consultation, which serve as helpful guidelines:

1. To define the reason for the patient's attendance, including:
    - the nature and history of problems
    - their aetiology
    - the patient's ideas, concerns and expectations
    - the effect of the problems
2. To consider other issues:
    - continuing problems
    - risk factors
3. To choose, with the patient, an appropriate action for each problem
4. To achieve a shared understanding of the problems with the patient
5. To involve the patient in the management and encourage him or her to accept appropriate responsibility
6. To use time and resources efficiently and appropriately:
    - in the consultation
    - in the long term
7. To establish or maintain a relationship with the patient that helps to achieve the other tasks

The exceptional potential in each primary care consultation described by Stott and Davis,[2] which is presented in TABLE 2.1 , also acts as an excellent aide-mémoire to achieve maximal benefit from the consultation.

Table 2.1 The potential in each primary care consultation

A	B
Management of presenting problems	Modification of health-seeking behaviour
C	**D**

Source: Stott & Davis[2]

Phases of the consultation

The consultation can be considered in three phases, as follows:

1. Establishment of rapport (see FIG. 2.1)
2. Diagnostic phase
   - the interview and history
   - the physical and mental examination
   - investigations
3. Management phase
   - explanation and education
   - prescribing medication
   - procedural–therapeutic or extended diagnostic
   - referral
   - follow-up

FIGURE 2.1 The consultation: establishment of good rapport is the foundation to successful consulting skills

Practice tip

Remembering the patient's preferred name and their basic past history creates powerful rapport.

The history

The doctor has four basic tasks to perform during the history-taking phase of the consultation. These are to determine:

1. the patient's stated reason for attending
2. why the patient is attending today, or at this particular time in the course of this illness
3. a list of problems or supplementary symptoms
4. any other initially unspoken or hidden reason for attending (e.g. the fear of cancer)

The old medical cliché that 'a good history is the basis of the clinical examination' is as relevant

as always. The art of history taking, which is based on good communication, is the most fundamental skill in general practice and requires a disciplined approach.

An interesting approach is that used by Professor Rita Charon of Columbia University: ‘I will be your doctor, and so I need to know a great deal about your body, health and your life. Please tell me what you think I should know about your situation.’[3]

Guidelines include:[4]

- Commence by eliciting the presenting complaint.
- Permit an uninterrupted history.
- Use appropriate language—keep the questions simple.
- Use specific questions to clarify the presenting complaint.
- Write notes or use the keyboard to record information but maintain as much eye contact as possible.
- Enquire about general symptoms, such as fatigue, weight changes, fever, headache, sleep and coping ability (see TABLE 2.2). These are important since they uncover ‘red flags’ for serious, life-threatening disorders.
- Undertake a relevant systems review.
- A historical checklist includes past medical history, complete medication history, drug habits and sensitivities, family history, psychosocial history and preventive care history.
- Give feedback to the patient about your understanding of the problems and agenda, and correct any misconceptions.

Table 2.2 Important general questions

Fatigue, tiredness or malaise
Fever, sweating, shakes
Weight change, especially loss
Pain or discomfort anywhere
Any unusual lumps or bumps
Any unusual bleeding
Skin problems—rash or itching

Good questions

In order to determine any underlying agenda or significant psychosocial problems, it is very helpful to use analytical questions. Such opening questions and inviting statements could include:

- Why have you come to see me today?
- Do you have any particular concern about your health?
- That really interests me—tell me more—it seems important.

- What were you hoping I could do for you today?
- Where would you put your real feelings between 0 and 100%?
- What is it that's really upsetting or bothering you?
- What do you really think deep down is the cause of your problem?
- Are you basically satisfied with your life?
- Is there anything that I haven't asked you and that you should tell me about?
- Tell me about things at home.
- Tell me about things at work.
- Do you experience any bullying?
- Are you afraid that something bad is going to happen to you?
- Is your relationship with any particular loved one/person causing you stress? (This may lead to information about sensitive issues such as domestic violence or sexual problems.)
- Is there anything in your life that you would like to change?
- I'm concerned about what you are not telling me.

Basic interviewing techniques

A number of basic interviewing techniques[5] encourage communication. It is important to use the least controlling interview techniques before embarking on direct questioning.

Questions

When the patient is asked a question, the doctor tends to take control of the interview, and so directs it along the lines of his or her own thinking or hypothesis generation. The problem is that if questions are used too early in the interview, the amount of desirable information is restricted and may disrupt the true priorities of the patient's concerns.

Open-ended questions and direct questions are very useful at appropriate times, while other questions are very restrictive. Examples, using pain as the ‘problem’, are:

- Open-ended question: ‘Tell me about the pain.’
- Direct question: ‘Where is the pain?’
- Closed question: ‘Is the pain severe?’
- Leading question: ‘The pain is severe?’
- Reflective question: ‘You want to know the cause of the pain?’

The open-ended question

The open-ended question is essential in initiating the interview. A question such as ‘What kind of troubles have you been having?’ says to the patient ‘I’m interested in anything you may feel is important enough for you to tell me’.

The open-ended question gives the patient an opportunity to take temporary control of the consultation and to outline problems and concerns.

Ongoing interview strategies of listening and silence, facilitation and summarisation are outlined in CHAPTER 3 (section on Communication in the consultation).

Information from other sources

Sometimes it is important to obtain information from other sources, especially friends or relatives. Off-hand comments from others may be loaded with ‘cues’ and one should be listening intently.

Problem definition

Part of the diagnostic process is defining the patient’s problem or problems. The more complex the presentation, the more necessary it is to have an orderly approach. It is clearly important to list the problems in a priority order. These problems may have been ‘offered’ by the patient, ‘observed’ by the doctor, ‘derived’ during the interview or ‘known’ from the past history. Problems can be conveniently considered as organic or physiological, and intrapersonal or social.[6]

Touching the patient

Sometimes a natural response is to touch the distressed patient as a reassuring gesture. It is best to adopt a caring-and-support gesture, such as offering tissues to the weeping patient, but it may be quite acceptable for most patients to give a reassuring, momentary touch somewhere between the shoulder and wrist on the arm nearest to you. Touching should be a natural gesture that is

comfortable for both the doctor and patient. Touch elsewhere should generally be avoided.

The physical and mental examination

If a diagnostic hypothesis based on the history is being tested, the examination may be confined to one system or to one anatomical region. However, other regions, systems or a general examination may be undertaken for medicolegal or preventive reasons. Patients tend to feel vulnerable during the physical examination, so their sensitivity and modesty have to be respected. Generally, the examination is conducted in relative silence, with the doctor instructing the patient what to do.

Patients need to be warned of possible discomfort or pain that may accompany certain examinations, of the reason for the examination and of its immediate results, particularly if normal. Continued silence on the doctor's part is often interpreted by patients as being indicative of something serious or unusual being found. For the same reason, the doctor's non-verbal behaviour is important.

Medicolegal guidelines for examinations[7,8]

The following guidelines have been recommended by the NSW Medical Board for consultations and physical examinations:

- Carefully explain the nature and purpose of the physical examination before you start. Take particular care with explanations before rectal, vaginal, breast and genital examination.
- Indicate when an examination may be uncomfortable and ask the patient to advise if you are causing pain.
- If a patient is required to disrobe, explain to what extent undressing is required and why.
- A patient's modesty should be preserved when undressing and dressing before and after a physical examination. Privacy screens, sheets and gowns should be provided as a matter of course. Clinic staff should not interrupt physical examinations.
- If the patient requests the presence of a chaperone or a friend, this should be respected.
- Do not lock the door of the consultation room. The setting should allow the patient confidence to terminate the consultation at any time if he or she is uncomfortable.
- Ask yourself, 'Is what I am doing part of accepted medical practice?'.[9]

Investigations

It is often necessary to arrange for special tests to assist in the diagnostic process or to monitor the progress of certain illnesses or response to treatment. The informed consent of patients must

be obtained. A collaborative decision for or against certain tests may be negotiated.

GPs have a responsibility (clinical and economic) to be very discerning and selective in the investigations that they choose. The questions that should be asked in decision making include:

- Is this investigation necessary?
- Will it change my management?

Richard Asher (1954) listed the questions a clinician should ask before requesting an investigation:[10]

- Why am I ordering this test?
- What am I going to look for in the result?
- If I find it, will it affect my diagnosis?
- How will this affect my management of the case?
- Will this ultimately benefit the patient?

In general, investigations should be performed only when the following criteria are satisfied:[10]

- The consequence of the result of the investigation could not be obtained by a cheaper, less intrusive method (e.g. taking a better history or using time).
- The risks of the investigation should relate to the value of the information likely to be gained.
- The result will directly assist in the diagnosis or have an effect on subsequent management.

The three strikes and you're out rule

A very useful rule is to bail out of the diagnosis and refer to a colleague if you have failed to make a diagnosis after three consultations.

Management phase of the consultation

The management phase of the consultation may immediately follow the information-gathering interview, or it may take place on review, after diagnostic tests or referral. It should be remembered that there are at least two people concerned in management: the doctor *and* the patient. Poor patient compliance with any proposed therapy can be a result of a poorly conducted management phase. It is necessary not only for the doctor to make statements concerning therapy and the reasons for the chosen therapy, but also for the information to be conveyed in a language

appropriate to each patient's understanding. Negotiate a management plan.

Management includes immediate care, prevention and long-term care. Doctors generally tend to be authoritarian in their management proposals. Whole-person management, however, implies that the patient's views are listened to, explanations are offered where necessary by the doctor and an educative approach is adopted to encourage the patient to actively participate in management and preventive behaviour, where possible.

The objectives of the management phase of the consultation are summarised in TABLE 2.3 .

Table 2.3 Objectives of the management phase of the consultation[5]

To make use of the doctor–patient relationship in therapy
To involve the patient as far as possible in the management of his or her own problem
To educate the patient about the illness
To promote rational prescribing
To achieve compliance in therapy
To emphasise preventive opportunities
To provide appropriate reassurance
To encourage continuity of ongoing care

The sequence of the management interview[5]

The following, which represents an excellent teaching strategy, is a suggested *10-point plan* or sequence for conducting a management interview. These guidelines will not always need to be applied in their entirety, and may need to be staged over a number of consultations. The use of this sequence should ensure identification of all the patient's problems by the doctor (including fears, feelings and expectations), adequate patient understanding of his or her problems, an acceptable and appropriate treatment plan being defined for each problem, preventive opportunities being addressed, and the patient being satisfied with the consultation and being clear about follow-up arrangements.

The sequence is as follows.

1 Tell the patient the diagnosis

2 Establish the patient's knowledge of the diagnosis

3 Establish the patient's attitude to the diagnosis and management

4 Educate the patient about diagnosis

- Correct any incorrect health beliefs recognised in point 2.
- Supplement the patient's existing knowledge to a level appropriate to the needs of the patient and the doctor.

5 Develop a management plan for the presenting problem

Develop precise instructions using three headings:

- *Immediate:* always included, even if no action is proposed
- *Long term:* for chronic, long-term or recurrent illnesses
- *Preventive:* sometimes specific measures apply—often patient education is the method required

The patient should be encouraged at this stage to participate in decision making regarding management and to make a commitment to the plans.

6 Explore other preventive opportunities

7 Reinforce the information

- Use the patient's own results (e.g. X-rays and ECGs).
- Encourage the patient to participate in the decision making and in accepting some degree of responsibility for his or her own management.

8 Provide take-away information

- Examples of this important strategy include patient instruction leaflets and resource contacts.

9 Evaluate the consultation

10 Arrange follow-up

Closing the session

Good closure is an important strategy; ask 'Has this visit helped you and your problems—is there anything more I can do?'

A patient management strategy

Brian McAvoy, writing in Fraser's excellent book *Clinical Method: A General Practice Approach*, presents a helpful aide-mémoire in the approach to patient management:[10]

1. reassurance and/or explanation

2. advice
3. prescription
4. referral
5. investigation
6. observation (follow-up)
7. prevention

Prescriptions

It is worth emphasising that prescribing medicine is a relatively complex skill that requires considerable knowledge of the disease, patient's expectations, the drugs prescribed, their interactions and their adverse reactions. Part of this skill is making a decision not to prescribe medication when it is not absolutely necessary and then explaining the reasons and including non-pharmacological measures. This decision may be made in the context of a patient expecting a biochemical solution for his or her problem. As McAvoy points out, 'If in doubt whether or not to give a drug—don't'.[10]

Rational prescribing applies particularly to opioids, antibiotics and tranquillisers.

Antimicrobial stewardship

This positive strategy describes a systematic approach to optimising the use of antimicrobials, with a view to improve outcomes and reduce adverse consequences, especially the development of resistant strains.

General guidelines for antibiotic prescribing

Choose the agent with the:

- narrowest spectrum that will cover the likely pathogens (based on culture/sensitivity)
- lowest cost if efficacy and safety are otherwise equal
- indications should be evidence based
- ensure oral therapy is used where clinically appropriate
- dosage individualised to the patient
- fewest serious side effects
- duration as short as possible

- proven microbiological guidelines to guide therapy

Avoid wherever possible:

- prescribing antibacterial antibiotics for viral respiratory infections
- combinations if a single drug is likely to be effective
- topical antibiotics, as resistance is much more likely to develop (exceptions include eye infections and vaginitis)
- antibiotic combinations, except in proven clinical circumstances or when coverage is difficult with a single drug
- prophylactic antibiotics, unless they are of proven benefit (in general only in some elective surgery or dental procedures)

The common respiratory infections such as acute otitis media, pharyngitis, tonsillitis, acute bronchitis, bronchiolitis and influenza have commonly a viral cause and it is appropriate to treat symptomatically with a 'wait and see' surveillance.[11]

Referral

The decision to refer a patient is also another important skill. It is often difficult to find the right balance. Some practitioners refer excessively—others cling to their patients inappropriately. It is a mistake not to refer a patient with a serious chronic or life-threatening disease. Apart from consultants and hospitals, referral should be considered to GP colleagues or partners with special interests or expertise, support groups and other members of the primary health care team, such as physiotherapists, dietitians, chiropodists and social workers. At all times the GP should act as the focal reference point and maintain control of patient management.

The 'gatekeeper' role of the GP

A patient's GP is the obvious and ideal linchpin in the health care system to take responsibility for the patient's health concerns and management. The patient may become confused with the system, especially if his or her problems are many and complex. The patient's GP has a vital role in acting as a 'gatekeeper' between primary and secondary care, and between paramedical services. The GP should always act in the patient's best interests and intervene, if necessary, to ensure that the patient is getting the best possible care.

The healing art of the doctor

The counselling process in general practice is based on the therapeutic effect of the doctor. This well-recorded feature is reinforced if the doctor has a certain professional charisma, and is caring and competent. We cannot underestimate the dependency of our patients on this healing factor, especially where significant psychic factors are involved.

Key points on patient management[12]

- The diagnostic process begins at the door of the waiting room when you invite the patient in.
- It is difficult, perhaps impossible, to reassure patients in the absence of an appropriate physical examination and certain investigations.
- Reassurance must always be appropriate and therefore based on a substantial foundation: inappropriate reassurance damages the credibility of both the doctor and his or her profession.
- The two key characteristics of the doctor in establishing the basis of a successful outcome for the doctor–patient relationship are caring and responsibility.
- Vital factors included in this relationship are good communication, genuine interest and trust.
- Listen to what the patient is saying and not saying.

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3 Communication skills

Most people have a furious itch to talk about themselves and are restrained only by the disinclination of others to listen. Reserve is an artificial quality that is developed in most of us as a result of innumerable rebuffs. The doctor is discreet. It is his business to listen and no details are too intimate for his ears.

W SOMERSET MAUGHAM (1874–1965), *THE SUMMING UP*

Hippocrates wrote:

> In the art of medicine there are three factors—the disease, the patient and the doctor . . . It is not easy for the ordinary people to understand why they are ill or why they get better or worse, but if it is explained by someone else, it can seem quite a simple matter—if the doctor fails to make himself understood he may miss the truth of the illness.[1]

Francis Macnab, Doctor of Divinity and patient, wrote: 'The style of the doctor, the communication of the doctor and the person of the doctor at the level of primary contact and primary care can be crucial in a person's life.'[2]

Much of the art of general practice lies in the ability to communicate. Listening is generally regarded as the most important skill.

Research continues to focus the 'blame' for communication breakdown on the doctor, ignoring the role of the patient.[3]

Communication

Communication can be defined as 'the successful passing of a message from one person to another'.

There are five basic **elements** in the communication process:

- the communicator
- the message
- the method of communicating

- the recipient
- the response

Important **principles** facilitating the communication process are:

- the rapport between the people involved
- the time factor, facilitated by devoting more time
- the message, which needs to be clear, correct, concise, unambiguous and in context
- the attitudes of both the communicator and the recipient

These elements and principles can be seen emerging in various phases through the consultation, as illustrated in FIGURE 3.1 .

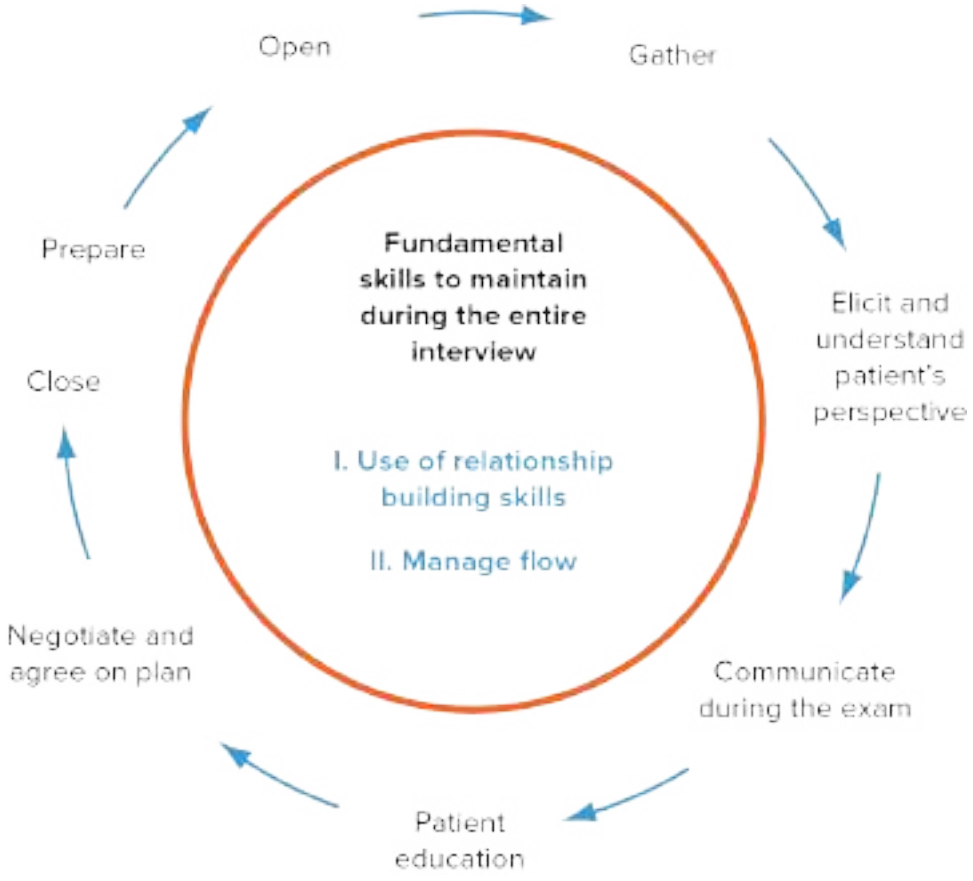

FIGURE 3.1 The sequence of communication in the consultation

Source: Reproduced with permission from Macy Initiative on Health Communication, New York University School of Medicine.

Communication in the consultation[3,4]

Communication in the consultation can be considered in the following sequence:

The doctor requires appropriate communication skills for complete diagnosis (physical, emotional and social) and competent management. It is important to be aware of the patient's cultural background and educational level, and allow for these factors. The majority of interaction between doctor and patient occurs in the traditional consultation. This involves both verbal and non-verbal communication.

Prepare

The 'prepare' phase includes preparation done both well before the consultation and then just prior to the consultation. Well before consulting, the doctor should think about and prepare the physical environment. Comfort and privacy should be maximised, and distractions and interruptions minimised. The patient should be physically positioned to feel empowered (e.g. avoid talking across a desk or talking down to a patient on a bed).

As well as reviewing the environment, doctors should review themselves. They should do some self-reflection to consider what personal qualities, assumptions and values they have that may influence a consultation.

Just prior to the consultation, a review of the patient's health record will improve the doctor's awareness of important facts about the patient. Opening the file is actually when the consultation starts. Here, crucial clues can be found, such as:

- what happened at the last consultation
- the important medical issues for this patient
- any recent test results or correspondence that have arrived
- the names of partners, parents or children who may come into the room with the patient
- brief notes on personal characteristics, likes/dislikes (e.g. has needle phobia)

When a record is examined well, the reasons for the consultation can often be anticipated prior to the formal start of the consultation, giving the doctor a wonderful opportunity to improve communication.

Increasingly in general practice we see patients as part of a team. The patient may previously have been seen by a practice nurse or a more junior doctor or medical student. This 'teamlet model of primary care'[5,6] has been shown to help elucidate patients' concerns, as have 'patient agenda forms', which patients fill out prior to the consultation, prompting them to list what is on their agenda for the consultation.[6,7]

At first contact, we usually call a patient from the waiting room into the consulting room. Having your eyes and ears ready and focused here can give you invaluable information. What is the person wearing? What is the significance of any badges, necklaces, rings or tattoos? What does

his or her body language suggest? Who is accompanying the patient and how are they interacting with each other? Clues about their interests (e.g. a child's T-shirt reflecting a favourite TV character), their cultural and social backgrounds (e.g. dress and appearance) and even their medical issues at hand (e.g. a limp, a bandage or carrying an X-ray folder or hospital letter) abound in this 'pre-opener space'. Picking up on these clues early helps the doctor anticipate and reflect on issues before they emerge in the consultation, avoids communication breakdown, makes the patient feel that the doctor is interested in him or her and can make the doctor appear switched on and observant.

Open

When we get to the 'opener' (e.g. 'What can I do for you today?' or 'Why have you come to see me today?') and beyond, we should:[8]

- greet and address the patient by his or her preferred name (and anyone else entering the room)
- try to make the patient feel comfortable
- try to appear 'unhurried' and relaxed
- focus firmly on the patient
- use open-ended questions where possible
- make appropriate reassuring gestures

Listen and gather

It is in the early stages of the consultation that silence (on the doctor's part) can be golden. In what is termed *active listening*, described by Egan[9] as follows:

> One does not listen with just his ears: he listens with his eyes and with his sense of touch. He listens by becoming aware of the feelings and emotions that arise within himself because of his contact with others (that is, his own emotional resonance is another 'ear'), he listens with his mind, his heart, and his imagination. He listens to the words of others, but he also listens to the messages that are buried in the words or encoded in all the cues that surround the words. He listens to the voice, the demeanour, the vocabulary, and the gestures of the other. He listens to the context, verbal messages and linguistic pattern, and the bodily movements of others. He listens to the sounds, and to the silences.

Allowing the patient to talk (without interruption), and even leaving a slightly prolonged pause, often provides enough space for the patient's concerns to emerge. This is especially the case with psychosocial issues.[10]

Listening includes four essential elements:

- checking facts
- checking feelings
- encouragement
- reflection

Listen with understanding, in a relaxed, attentive silence. Use reflective questions, such as:

- ‘You se‘em very sad today.’
- ‘You seem upset about your husband.’
- ‘It seems you’re having trouble coping.’
- ‘You seem to be telling me that ...’
- ‘Your main concern seems to me to ...’

Three techniques that have been demonstrated[8,11] to improve how we elicit patient concerns are:

- facilitation
- the open-to-closed cone
- summarisation

Facilitation refers to comments or behaviours by the doctor that encourage the patient to keep talking. This could include a head-nod, a ‘hmm’ at the right time, or ‘Tell me more about that’. The open-to-closed cone is a gradual narrowing of focus from an indirect non-directive exploration to a more direct exploration. It is often difficult to resist the urge to ‘dive in’ and explore the initial concern raised and narrow the cone too quickly.[6,12]

After each problem or concern is elicited, the doctor should continue to explore to ensure there are not any more. Using a patient-centred approach leads to improved patient trust and satisfaction, more appropriate prescribing and more efficient practice.[6]

Summarisation is when the doctor provides the patient with an explicit verbal summary of the information gathered thus far in the consultation.[11] This helps to orientate the patient, acknowledging to him or her that the doctor has taken on board what they have said, and reflecting back to the patient the doctor’s understanding of it.

Non-verbal communication

Non-verbal communication or body language is a vital feature of the communication process. Human communication takes place through the use of gestures, postures, position and distances (non-verbal communication or *body language*) more than by any other method. Non-verbal cues comprise the majority of the impact of any communicated message (see TABLE 3.1).[13]

Table 3.1 Impact of the message

Cue	%
Words alone	7
Tone of voice	38

Recognition of non-verbal cues in our communication is important, especially in a doctor–patient relationship. Charles Darwin in his *Expression of the Emotions in Man and Animals* (1872) concluded that there is a unique pattern of non-verbal actions for each emotion, such as snarling as a sign of aggression. The ability to identify non-verbal cues improves communication, rapport and understanding of the patient's fears and concerns. Recognising body language can allow doctors to modify their behaviour, thus promoting optimum communication.

Interpreting body language

The interpretation of body language, which differs between cultures, is a special study in its own right, but there are certain cues and gestures that can be readily understood. Examples illustrated include: the depressed patient (see FIG. 3.2); barrier-type signals, often used as a defensive mechanism to provide comfort or indicate a negative attitude (see FIG. 3.3); and a readiness gesture, indicating a desire to terminate the communication (see FIG. 3.4).

FIGURE 3.2 Posture of a depressed person: head down, slumped, inanimate; position of desk and people correct

FIGURE 3.3 Body language barrier signals: **(a)** arms folded, **(b)** legs crossed, **(c)** ‘ankle lock’ pose

FIGURE 3.4 Body language: ‘readiness to go’ gestures

Having noted the non-verbal communication, the doctor must then deal with it. This may require confrontation—that is, diplomatically bringing these cues to the patient’s attention and exploring the associated feeling further.

The patient’s perspective

To be truly patient-centred, it is not enough merely to find out the patient’s concerns and needs

—we also need to explore his or her ideas, beliefs and expectations.[8] What is the patient's *perspective* on his or her concerns and needs? What does the patient expect of you and from the consultation? What are the priorities?

To best meet this challenge, doctors should be vigilant for verbal and non-verbal cues that suggest frustrations, fears and anxieties.[3] A shuffle in a chair or a stiffening in posture may give a clue as to the emotional context a patient places on a particular issue. The issue is therefore not only whether we have listened, but whether we have understood.[10]

Discovering the patient's beliefs about illness will allow us to make statements to them that are congruent with those beliefs. This is especially important in mental health, where illness beliefs are often emotionally laden and unpredictable.

Understanding the patient's perspective also needs to be considered in a cultural context. Culture can have many dimensions, including ethnicity, age, gender, sexuality, community and religious beliefs. Being *culturally competent* by showing an interest in, respect for and sensitivity to that culture will help us achieve a shared understanding of where the patient is coming from and how he or she is seeing things.[8]

Communicating during the physical examination or procedure

In the same way that we obtain consent for surgical procedures, we should also fully inform the patient of what we plan to do and obtain consent in any physical interaction with the patient. Physical examination can be very confrontational for some patients and this can be underestimated by the doctor. There may be factors of which the doctor is unaware that may make examination particularly difficult for the patient, such as unpleasant previous experiences, cultural, gender or sexual issues surrounding touch, or phobias about medical procedures or needles.

As well as preparing the patient, explaining during the examination or procedure what is happening and what we are observing and finding will help the patient feel valued and respected. We should also continue to keep an ear out for any further patient concerns being raised.[3]

If we are fully eliciting the patient's concerns and needs and are consulting using a patient-centred approach, a point is reached in the consultation where the information flow will need to go in the opposite direction—from doctor back to patient (in fact, in most consultations this flow often moves repeatedly back and forth). How we deliver this information is critical to patient communication.

Four techniques that will help maximise patient understanding are:

- signposting
- 'chunk and check'

- avoiding jargon
- using visual and physical techniques to communicate[8]

Signposting is a technique whereby the doctor explicitly states what he or she has done and/or is about to do (e.g. ‘Andrew, I have finished examining you, now I would like to explain what I think the issues are’ or ‘Mrs Jones, I have two matters I would like to discuss: first ...’). Signposting helps orientate the patient, which further helps him or her to relax and focus better on what you are saying.

Chunk and check is where the doctor provides a chunk of information to the patient and then immediately checks the patient’s understanding of what has been said. Chunk and check works best when the chunks are small, as this information is often new to the patient and best digested in small grabs.

Jargon is a barrier to communication in many professions (think accountants or IT technicians) and medicine is rife with jargon.

When dealing with patients, using jargon not only impairs the patient’s understanding, but can also be alienating and intimidating. The patient needs to have the cognitive and communicative capacity to understand the message.[14]

Visual and physical methods of conveying information given (or plans made) can include diagrams, models, patient hand-outs or information sheets.[8] Having ready access to electronic visual materials or websites on a desktop computer can also help. Videos on websites such as YouTube can be used to illustrate how the body functions, how a disease manifests or a particular medical procedure, and directing patients to reputable and reliable information sources on the internet or elsewhere (before they find unreliable information themselves) will help avoid misinformation and extend the communication beyond the consultation.

Negotiate and agree on a plan

Looking beyond patient-centred communication, we can then think about planning: what do we intend to do, how we are going to decide this, who is going to do it? The preferred technique for this is *shared or collaborative decision making.*[14,15] The aim should be to have such collaboration at all stages of the consultation. But because patients can often feel intimidated, it is a challenge for the doctor to make the patient feel comfortable enough to do so.[16]

To enable this collaboration, the doctor and patient should treat each other’s concerns with respect; this will lead to a shared responsibility for agenda setting.[10] Such collaboration, when done well, can lead to a coming-together of thinking that has been called a *shared mind.*[14] ‘This is what I would suggest, what do you think?’ As a way of thinking, a shared mind involves a doctor being mindful of the patient’s values, thoughts and feelings (as well as those of his or her own), and seeing where the two connect.[17]

This mindfulness of each other's position can help the negotiation of what happens in the consultation and also avoid communication breakdowns. For instance, what shall we deal with today, and what should be delayed or rolled over to another consultation?[10] It can also help repair a communication breakdown.

One technique that uses this principle is called an *empathic bridge*.[10] This is where we anchor the conversation in the patient's experience by reflecting or paraphrasing. From this anchor, we then manoeuvre the conversation back to where it needs to be.

Another aspect of the doctor–patient relationship that enhances collaborative decision making, particularly in general practice, is *shared experiences*.[18] GP–patient relationships evolve over time, and a shared experience such as helping a patient through a difficult pregnancy, a major illness or even doing a home visit can enrich the relationship, deepen the connection and trust between doctor and patient, and lead to greater collaborative decision making.

Close

How should we close a consultation? If we follow the principles of patient-centred communication and remember that we should keep our focus on the patient's concerns and needs, it soon becomes apparent.

First, is the patient aware of the imminent closure? Anxious and distressed patients may have no idea how long they have been ruminating about their concerns through the consultation, and letting them know in advance that closure is being planned (and why) will allow them to not feel pushed out of the room.

Secondly, making sure that there are no further disclosures of concerns or needs to come (yet again) will reduce the risk of what has been termed the 'doorknob presentation'—the raising of a patient concern that happens as the doctor puts his or her hand on the doorknob to leave the room (this has also been called the 'Oh, by the way doctor' syndrome in the USA, the 'à propos, docteur' in France and 'tussen haakjes' in Denmark, which translates to 'between two brackets' or, as we may say, 'parenthetically').[10]

Thirdly, summarising the critical points of the consultation and planned actions and expectations will provide a final opportunity to identify gaps between what the doctor and patient are respectively thinking. We should also prepare a safety net by considering any possible unexpected outcomes to what is being planned (e.g. what a parent should watch out for and what to do if things worsen with the febrile infant patient).

Finally, we should thank and say farewell to the patient with an appropriate parting statement. Does this include a handshake? This may be determined by your style, the patient's style and cultural issues.

Use of relationship-building skills

During the consultation and throughout a doctor–patient relationship over many consultations

(and potentially, in general practice, over decades), effective communication is underpinned by using skills that develop the interpersonal relationship between doctor and patient.[3,8]

These skills include the doctor paying attention to non-verbal behaviour on display, as mentioned above, such as appropriate eye contact, posture, position and movement. Verbal cues such as the speed of speech, volume and tone can also be used. If using a computer or taking written notes, the doctor should do so in a fashion that does not interfere with dialogue or rapport. Also, given that patients are often highly emotionally invested in what we say, consulting in a manner that reflects confidence (without stepping over into arrogance) will help build trust.

Rapport, which originates from an old French word that literally means ‘to carry back’, can be engendered by fostering connections back and forth with the patient. Displaying empathy for the patient’s situation or feelings, acknowledging his or her view or efforts, and dealing sensitively with embarrassing or disturbing topics such as pain or grief will engender rapport. We can show we are willing to provide emotional support by overtly expressing our concern or understanding, or a willingness to help or offer partnership.[8,16] An offer such as ‘I am really keen to help you with this situation’ can go a long way.

Connections that build rapport can also happen away from medical issues, and are often more powerful. This is where the ‘clues’ that we were looking for at the beginning of the consultation can come into play. If a short interplay can happen between doctor and patient about something the patient is passionate about or interested in, which has nothing to do with the medical issues at hand, the patient will feel that he or she is respected as a person, not just as a medical presentation to be solved. Examples of such clues that could be picked up and explored by the doctor could be a favourite toy being held by a child, a book that a patient carries into the room or the doctor noticing that the occupation of the patient is something he or she is interested in asking about. It comes down to patients feeling that the doctor is actually interested in them. When such connections are made, any tension in the consultation room can be seen to evaporate.

Other rapport-building techniques

A person can develop a rapport with another by mimicking his or her body language, speech, posture, pace and other characteristics. Such techniques can be used to help the doctor communicate better with a patient and also to improve the patient’s attitude by changing the patient’s body language position.

Mirroring

Mirroring is a useful technique whereby the limb positions and body angles of the person you are talking to can be copied. A mirror image is formed of their position so that when they look at you they see themselves as in a mirror. It is not necessary to copy uncomfortable gestures or unusual limb positions, such as hands behind the head. A partial mirror is often sufficient.

Pacing

People exhibit a certain rhythm or pace that can be revealed through their breathing, talking and

movements of the head, hands or feet. If you can copy the pace of another person, it will establish a sense of oneness or rapport with them. Once this pace is established, you can change their pace by changing yours. This is called *leading*.

Vocal copying

Vocal copying is another way to develop rapport with people. It involves copying intonation, pitch, volume, pace, rhythm, breathing and length of the sentence before pausing.

Manage flow

At the end of the day, the doctor has a professional responsibility to appropriately meet the needs of the patient but also to keep control of a consultation so that it does not affect other consultations which follow. In most consultations, this is not difficult, but with a small proportion of patients, particularly those who have mental health and/or psychosocial issues to deal with, maintaining control and managing time can be challenging.[19]

A balance must be struck between maintaining control and not undermining the doctor–patient relationship.

The doctor having a ‘wide-angle lens’ on the consultation, so that he or she is mindful of where they are up to in the consultation and how much time has been taken up already, as well as the actual medical issues being discussed, will help anticipate a problem. Doing so subtly (e.g. not looking at a watch!) will help. It can be useful to have a wall clock situated behind the patient’s chair or to be aware of where the consultation timer is on the computer screen. Naturally, even if the time is way over, it may be entirely appropriate to carry on if it is an important issue, such as with a suicidal or distressed patient.

If a consultation’s flow is becoming problematic, the doctor should employ *appropriate use of power*.[18] This can be done with techniques such as setting rules in advance for patients where this is a problem (e.g. time limits for the consultation or limits on the number of concerns to be addressed). We can also use, if required, *blocking behaviours*, which can be verbal or non-verbal. These are behaviours that consciously block the flow of a consultation that is not being appropriately controlled. Examples include the doctor using body language that suggests he or she has something to say, or purposefully focusing on the (sometimes very short) space between a hyper-verbal patient’s sentences to enable him or her to ’jump in’ and take control of the consultation. On the other hand, it is important to avoid blocking approaches to effective patient communication. These are highlighted in the following negative dozen ‘road blocks’.[20]

‘Road blocks’ to good communication

Judging

1. Criticising: ‘You didn’t bother to follow up that test’
2. Name-calling: ‘You are becoming a worrisome drug addict’

3. Diagnosing: ‘I can read you like a book’
4. Praising evaluative: ‘You’re a good patient—I know you can manage this ...’

Sending solutions

1. Ordering: ‘You must stop smoking’
2. Threatening: ‘If you don’t change, you will be in dire circumstances’
3. Moralising: ‘I cannot condone that sort of behaviour—it’s wrong and won’t help you’
4. Excessive/inappropriate questioning
5. Advising/patronising: ‘When you’re overseas, be on your best behaviour’

Avoiding the other’s concerns

1. Diverting/changing the subject: ‘What did you think of the election result?’
2. Logical argument: ‘This wouldn’t have happened if you ...’
3. Reassuring: ‘What are you worrying about? Hundreds of people have to face up to that ...’

Practice tips

- Using a patient-centred approach leads to improved patient trust and satisfaction, more appropriate prescribing and more efficient practice.
- Undertaking the strategies of facilitation, the open-to-closed cone and summarisation will help us effectively elicit patient concerns.
- Associated with listening, observe non-verbal language, which may in many instances be the most significant part of the communication process.
- Techniques that will help maximise patient understanding are signposting, ‘chunk and check’, avoiding jargon and using visual and physical techniques to communicate.
- Collaborative decision making helps the negotiation of what happens in the consultation and also avoids communication breakdowns.

Key features of good communication[21]

- A patient-centred approach
- Eliciting all of the patient's concerns
- Exploring the patient's ideas, beliefs and expectations
- Listening
- Showing empathy and respect
- Summarising
- Avoiding jargon
- Collaborative decision making
- Good closure

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4 Counselling skills

The doctor should have a kind disposition, great patience, self-possession, meticulous freedom from prejudice, an understanding of human nature resulting from an abundant knowledge of the world, adroitness in conversation and a special love of his calling.

G GRIESINGER 1840

The term counselling refers to the giving of advice to help direct or influence the decisions or actions of another. In the clinical context counselling can be defined as the therapeutic process of helping a patient to explore the nature of his or her problem in such a way that he or she determines his or her decisions about what to do, without direct advice or reassurance from the counsellor.

The counselling process in general practice is based on the therapeutic effect of the doctor. There is an enormous and ever-increasing need for people in the community to have many of their emotional and social problems addressed by the health profession. Modern medicine has acquired a much more scientific face over recent years at the expense of its once respected humanistic one. Medicine is primarily a humanitarian pursuit, not an economic or scientific one, and uses science as a tool. Many feel that medicine is losing sight of this, at the considerable expense of its standing in the community.[1]

The public perceives that GPs can and do counsel people because more people go to their GP for counselling than to any other group of health workers, including psychologists, psychiatrists, social workers, marriage guidance counsellors and clergy.[1] People do not generally tell the doctor or even realise that counselling is exactly what led them to come to the doctor in the first place. The GP is, therefore, ideally placed in the community to make the most significant contribution to fill the community's needs in this area.

The GP as an effective counsellor

GPs can be effective counsellors for the following reasons:[2]

- They have the opportunity to observe and understand patients and their environment.
- They are ideally placed to treat the whole patient.

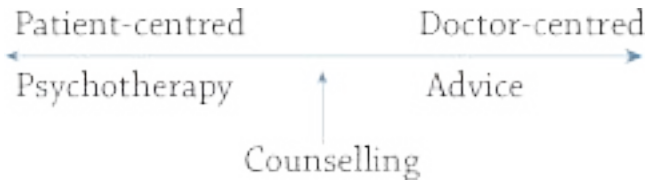
- Their generalist skills and holistic approach permit GPs to have a broad grasp of a patient's problems and a multifaceted approach to treatment.
- They can provide treatment in comfortable and familiar surroundings, including the doctor's rooms and the patient's home.
- They are skilled at working as a member of a professional team and directing patients to more expert members of the team as necessary.
- They can readily organise 'contracts' with the patient.
- They often have an intimate knowledge of the family and the family dynamics.
- They fit comfortably into continuing patient care with appropriate follow-up treatment programs.

To be an effective counsellor the GP must prepare for this role, first by making a commitment to its importance, then by acquiring the knowledge and skills for basic counselling by reading, attending workshops and discussing cases with colleagues who are skilled in counselling.[2] Appropriate workshops are those based on the seminal model of therapy by Balint,[3] which aim to teach the patient new coping skills and so alleviate symptoms and improve the patient's functioning in social and occupational roles. Well-developed interviewing skills are essential, as is self-discipline to appreciate one's strengths and limitations.

Features of counselling

Doctors can respond to patients' problems and distress by a spectrum of behaviours from doctor-centred, directive behaviour (directed psychotherapy) or advice at one end, to patient-centred, non-directive behaviour at the other, where the patient does most of the talking (expressive psychotherapy).[2] In handling psychosocial problems, advice-giving is at one end of the spectrum and psychotherapy at the other.

Counselling, as an activity in general practice, can be represented by a moving point between these two extremes.[1]



Counselling can be seen as having the following features:[1]

- It is a clear-cut treatment option, like a course of antibiotics.
- It is a cooperative problem-solving process.

- It is an educational venture where patients learn new information and new activities.
- It is a developmental process for patients.
- It is a change process—often moving a patient from a ‘stuck state’.
- It is a goal-directed activity.
- It is a process of energising patients and lifting their morale.
- It is a sensitive response to problems within a caring relationship.

A problem-solving approach

Defining the problem (what the matter really is) is the most important step in the process of patient care. The following outline is one approach to counselling that is applicable to a general practice context.[1]

1. Listen to the problem of first presentation: this involves listening not only to issues, events and experiences, but also to the patient’s feelings and distress. The emphasis here is more on the communication skills of facilitation, silence, clarification, reflection, paraphrasing, confrontation and summary, than on questioning. In many cases this phase of the counselling constitutes the major part of the therapy; for example, in grief or bereavement counselling, the doctor supports the patient through a natural but distressing process.
2. Define the problem, if possible, in behavioural terms:

   > Beneath the feeling is the experience, beneath the experience is the event, the event is related to a problem.[4]

3. Establish a contract for counselling, with an agreed number of visits initially (e.g. weekly half-hour or hour appointments for 4 to 6 weeks).
4. Define short-term and long-term goals for action.
5. Decide on one option—‘experimental action’.
6. Build an action program with the patient—negotiate ‘homework’ for the patient between visits.
7. Evaluate progress.
8. Continue action or select another option.
9. Evaluate progress.
10. Terminate or refer.

Counselling models

The PLISSIT model

The PLISSIT model, developed by Annon (1974)[5] as an aid in therapy for sexual problems, is a very useful model for problems presented as feelings where there is limited scope for intervention by the therapist.

The mnemonic PLISSIT stands for the following:

- P is for permission giving
- LI is for limited information
- SS is for specific suggestion
- IT is for intensive therapy

Annon emphasises that every primary care practitioner should be competent to offer ‘permission giving’ and ‘limited information’.

The Colagiuri and Craig model

The medical counselling model was developed by Colagiuri and Craig (see FIG. 4.1)[6] as a useful tool for teaching contraceptive, abortion and sterilisation counselling. It can be applied in most situations as it empowers the patients to make their own decisions through facilitation, as opposed to the directive and advisory learning model.

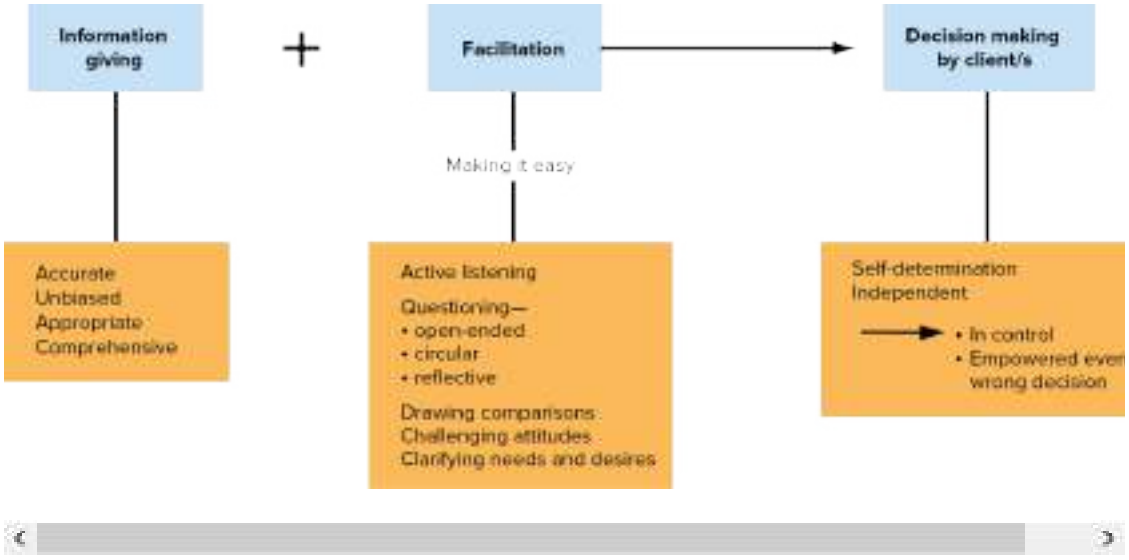


FIGURE 4.1 Medical counselling model

Source: Colagiuri and Craig[6]

The value of patient-centred counselling

There is evidence that the use of non-directive counselling techniques leads to more accurate diagnosis and therefore to more appropriate management and an improved outcome.[7]

Jerome Frank wrote in 1967: 'The field of counselling and psychotherapy has for years presented the puzzling spectacle of unabating enthusiasm for forms of treatment where effectiveness could not be objectively demonstrated'.[8] Traux and Carkhuff[9] measured important aspects of the psychotherapeutic relationship and demonstrated what had long been recognised: the outcome was enhanced if practitioners had such qualities as accurate and sensitive awareness of the patient's feelings, deep concern for the patient's welfare (without attempting to dominate) and openness about their own reactions.

The essential feature of the patient-centred approach is that the counsellor is more like a facilitator; that is, by the asking of well-directed questions it is hoped that patients can realise their own solutions for their problems.[1] This encourages patients to attain understanding and personal growth themselves rather than just put their personal affairs in the hands of someone else. This does not mean to say that the facilitator is passive in the process of assessing the relative merit of various solutions produced by the patient. The doctor-centred approach is most applicable for patients who are so confused or distraught that their ability to reflect usefully is temporarily or permanently inaccessible. Here, taking a more active and authoritarian role may be just what is required. It is therefore important to be flexible and move between the two ends of the spectrum as needed.

Basics of counselling and psychotherapy

- Listening and empathy are the beginning of counselling.
- Good communication is the basis of counselling.
- The therapist must really care about the patient.
- Always be aware of the family context.
- It is important for therapists to handle and monitor their own feelings and emotions.
- Maintain eye contact.
- The therapist must tolerate and be comfortable with what the patient says.
- Confidentiality is essential.
- Counselling is easier if there is a good rapport with the patient, especially if a long-standing relationship exists.

- Counselling is difficult if a social relationship is present.
- Don’t say to the patient ‘I’m counselling you’ or ‘I’m giving you psychotherapy’—make it a natural communication process.
- The therapist must be versatile and adapt a counselling style to the clinical occasion.
- Characteristics of the effective counsellor have been demonstrated to be genuineness, non-possessive warmth for the patient, and accurate and empathetic understanding.

A fundamental feature of counselling is reflective listening to direct patients to think about and then resolve their problems.

Some useful interviewing skills used in counselling are summarised in TABLE 4.1 .

Table 4.1 Interview skills used in counselling

Use reflective statements.
Use silence.
Allow expressions of emotion.
Offer supportive comments.
Paraphrase and summarise.
Allow patients to correct your interpretations of their feelings.
Observe lack of congruence.
Try to understand what the patient is feeling: • anger • hostility • fear • manipulation • seduction • insecurity
Make intelligent guesses to prompt patient to continue.
Don’t reassure too soon.

Counselling strategies[4,7]

- The therapy should be patient-centred.

- Use gentle, clever, probing questions.
- Facilitate the discussion to draw out relevant areas.
- It is important to be non-judgmental.
- Counsel through intuition and base it on common sense.
- Do not tell the patient what to do.
- Do not try to rush patients into achieving a happy ending.

- Provide guidance to allow the patient to gain insight.
- Wherever possible, make therapy non-authoritarian and non-directional.
- Use appropriate ‘gentle’ confrontation to allow self-examination.
- Help patients to explore their own situation and express emotions, such as anxiety, guilt, fear, anger, hope, sadness, self-hate, hostility to others and hurt feelings.
- Explore possible feelings of insecurity and allow free expression of such feelings.
- Explore patients’ belief systems and consider and respect their spiritual aspirations and conflicts.
- Ask key searching questions, such as:

  ‘What would be different in your life if you were well?’

  ‘Who are you mad at?’

  ‘If I understand you correctly you are telling me that ...’

  ‘You seem to be telling me that ...’

  ‘Correct me if I’m on the wrong track, but you are saying that ...’

  ‘What do you think deep down is the cause of your problem?’

  ‘What does your illness do to you?’

  ‘Do you really worry about any things in particular?’

  ‘How do you think your problem should be treated?’

  ‘If you could change anything in your life, what would it be?’

- Empower your patient. Better outcomes are likely if your patients are active participants in

their care and take responsibility for change.

Avoid:

- telling patients what they must do/offering solutions
- giving advice based on your own personal experiences and beliefs
- bringing up problems that the patient does not produce voluntarily

What counselling is not

- Giving information
- Giving advice
- Being judgmental
- Imposing one's own values, behaviour and practices
- The same as interviewing
- Handing out patient education material

Cautions[1]

- Individual doctors cannot be useful to all patients, so be selective.
- We cannot solve patients' problems for them.
- Patients' problems belong to them and not to their counsellors.
- Patients often have to change by only an inch in order to move a mile.
- If a counselling relationship is no longer productive, then terminate and refer.
- Most patients in primary care need information, support and a lift in morale, not long-term psychotherapy.

Patients unlikely to benefit

The following groups of patients are not likely to benefit from counselling therapy[1] (i.e. relative contraindications):

- psychotic patients
- patients who have had an unrewarding experience with psychiatrists and other

psychotherapists

- people who are antagonistic to the notion of a psychosocial diagnosis, subsequently found to be organic
- patients with little awareness or language to express emotional difficulty
- patients who do not believe doctors can treat psychosocial problems
- patients with a vested interest in remaining unwell who are therefore resistant to change (e.g. patients with work-related disabilities awaiting legal settlement)

- patients with chronic psychosomatic tendencies who are willing to do almost anything to maintain the relationship
- those in an intractable life situation who are unable or unwilling to change

Types of psychotherapy[2,10]

- Interpersonal psychotherapy/counselling, ideal for primary care and the treatment of depression, is therapy for interpersonal problems.
- Mindfulness based on intervention therapy.
- Behaviour therapy (BT) is where the patient is directed to do something.
- Cognitive therapy (CT) is based on the identification of automatic thoughts that are invariably negative. Cognitions are thoughts, beliefs and perceptions.
- Cognitive behaviour therapy (CBT), which combines BT and CT.

The psychotherapy continuum is illustrated in FIGURE 4.2 .[2]

FIGURE 4.2 Psychotherapy continuum

Source: Selzer R, Ellen S. *Psych-lite: Psychiatry That's Easy to Read*. Sydney: McGraw Hill, 2010: 70–3

Cognitive behaviour therapy

CBT is a form of non-pharmacologic treatment emphasising self-help and aiming to change perceptions and behaviour that may perpetuate symptoms and disability. It is basically a system of graded exposure (systematic desensitisation). It can be applied in any area of medical practice as a form of psychotherapy and is suitable in general practice for the treatment of depression, insomnia, eating disorders, delusions and hallucinations in psychotic disorders and anxiety in all forms, especially social anxiety disorder and phobias—in all of which CBT has proved to be better than a placebo.[11]

It is a relatively brief, active, directive and practical form of therapy. However, not all therapists or patients are suited to CBT.

The basic processes of CBT are to:

- define specific and concrete goals for functional activities and moods, e.g. pain control
- educate the patient
- teach basic skills for symptom control, relaxation and breathing control (especially for hyperventilation)
- identify, challenge and change maladaptive thoughts, feelings, perceptions and behaviour

Some basic principles and objectives of CBT are to:

- aim to bring about a desired change in patients' lives, i.e. aim to achieve desired goals
- assess, monitor and attempt to modify thoughts and behaviour
- reinforce positive behaviour and discourage negative behaviour
- educate about any misconceptions about a patient's illness
- encourage the patient to be an active participant (not a passive recipient)
- get patients to establish a problem list and hierarchy of problems
- aim for more realistic thinking and more adaptive responses

Mindfulness

Mindfulness, which is based on meditation, is the process about raising awareness and a special way of paying attention to help the person cope with the daily events in their life.

The basic processes of mindfulness are to:

- train people to focus on the positive aspects of their lives and shut out distraction and negativity, i.e. attention training
- learn to concentrate in a restful way and cope with stress

- emphasise attentiveness to the present moment
- voluntarily reign in a wandering attention time and again

Specific areas of counselling

Opportunities for basic counselling by the GP are ubiquitous in medical practice. Complex problems require referral but, even then, the GP still has an important role in continuing management.

Areas demanding counselling include:

- any crisis situation—breaking bad news
- bereavement or grief
- terminal illness/palliative care (CHAPTER 126)
- marital problems (CHAPTER 1)
- family problems (CHAPTER 1)
- sexual dysfunction (CHAPTER 108)
- chronic pain
- anxiety and stress (CHAPTER 70)
- depression (CHAPTER 10)
- intellectual handicap in a child
- infertility (CHAPTER 107)
- any disease or illness, especially severe illness
- sexual abuse/child abuse (CHAPTER 88)
- intimate partner violence (CHAPTER 110)
- insomnia and other sleep disturbances (CHAPTER 63)

Crisis management

Crisis situations are not uncommon in general practice and people in crisis are usually highly aroused and demanding. Examples include tragic deaths, such as children drowning or sudden infant death syndrome (SIDS), unexpected marital break-up and breaking bad news.

FIGURE 4.3 Counselling skills: these skills include good eye contact, listening, empathy and appropriate communication skills

Aims of crisis intervention

- Resolve the crisis and restore psychological equilibrium as quickly and constructively as possible.
- Encourage the person in crisis to regain control and take appropriate action.

Principles of management

- Intervene early—actively and directly.
- Establish an empathetic alliance.
- Be accessible.
- Attend to family and social supports.
- Be prepared for the difficult phase of 24–48 hours.
- Do not carry the burden of crisis.
- Aim for brief, time-limited intervention (no more than six interviews over 6 weeks).
- When necessary, be prepared to provide short-term use of psychotropic drugs (e.g. a hypnotic) for two or three nights of good sleep.

Ten rules to help those in distress

The following rules are given to those in crisis (personal explanation followed by a take-home hand-out):

1. *Give expression to your emotions.* You need to accept your reactions as normal and not be afraid to cry or call out. Try not to bottle up feelings.

2. *Talk things over with your friends.* Try not to overburden them but seek their advice and listen to them. Do not avoid talking about what has happened.

3. *Focus on things as they are now—at this moment.* Aim not to brood on the past and your misfortune. Concentrate on the future in a positive way.

4. *Consider your problems one at a time.* Try not to allow your mind to race wildly over a wide range of problems. You can cope with one problem at a time.

5. *Act firmly and promptly to solve a problem.* Once you have worked out a way to tackle a problem, go for it. Taking positive action is a step in allowing you to get on with life.

6. *Occupy yourself and your mind as much as possible.* Any social activity—sports, theatre, cards, discussion groups, club activity—is better than sitting around alone. Many people find benefit from a holiday visit to an understanding friend or relative. Religious people usually find their faith and prayer life a great source of strength at this time.

7. *Try not to nurse grudges or blame other people.* This is not easy but you need to avoid getting hostile. In particular, endeavour not to get angry with yourself and your family, especially your spouse.

8. *Set aside some time every day for physical relaxation.* Make a point of doing something physical, such as going for a walk, swimming or enjoying an easy exercise routine.

9. *Stick to your daily routine as much as possible.* At times of crisis a familiar pattern of regular meals and chores can bring a sense of order and security. Avoid taking your problems to bed and thus ensuring sleepless nights. Try to ‘switch off’ after 8 pm. Taking sleeping tablets for those few bad nights will help.

10. *Consult your family doctor when you need help.* Your doctor will clearly understand your problem because stress and crisis problems are probably the commonest he or she handles. Consult your doctor sooner rather than later.

- Remember that there are many community resources to help you cope (e.g. your religious leader, social workers, community nurses, crisis centres and organisers within churches and other religious centres).

- Take care: do drive carefully and avoid accidents, which are more common at this time.

Bereavement

Bereavement or grief may be defined as deep or intense sorrow or distress following loss.[12] Raphael uses the term to connote 'the emotional response to loss: the complex amalgam of painful affects including sadness, anger, helplessness, guilt, despair'.[13]

The GP will see grief in all its forms over a wide variety of losses. Although the nature of loss and patient reaction to it varies enormously, the principles of management are similar.

Stages of normal bereavement

1. *Shock or disbelief.* Feelings include numbness and emptiness, searching, anxiety, disbelief ('I don't believe it'), fear and suicidal ideation. Concentration is difficult and spontaneous emotions, such as crying, screaming or laughing, tend to occur. There may be a sense of the deceased's presence, and hallucinations (visual and auditory) may occur.

2. *Grief and despair.* Feelings include anger, 'Why me?', guilt and self-blame, and yearning. Social withdrawal and memory impairment may occur. The feeling of intense grief usually lasts about 6 weeks and the overall stage of grief and despair for about 6 months, but it can resurface occasionally for a few years. The last few months involve feelings of sadness and helplessness.

3. *Adaptation and acceptance.* Features of the third stage include significant feelings of apathy and depression. This phase takes a year or more. Physical illness is common and includes problems such as insomnia, asthma, bowel dysfunction, headache and appetite disturbances.

Pathological bereavement

Pathological bereavement can occur and may manifest as intense emotion, particularly anger, and multiple visits with somatic complaints; the patient often gets around to long dissertations about the deceased and the circumstances surrounding death. Extreme anger is likely when the sense of rejection is great, as with divorce or sudden death. Guilt can also be intense.[12]

Raphael's classification of the patterns of pathological grief and its various resolutions are presented in TABLE 4.2 .[13]

Table 4.2 Patterns and resolution of pathological grief

Morbid or pathological patterns
Absence, inhibition or delay of bereavement
Distorted bereavement
Chronic grief (intense anguish continues unabated)

Outcomes

- Normal resolution, satisfactory adjustment; reintegration in life, satisfying attachments
- General symptomatology (leading to increased care-eliciting behaviour)
- Depression, suicidal behaviour
- Other psychiatric disorders (anxiety state; phobia; mania; alcoholism; criminal activity, such as shoplifting)
- Altered relationship patterns
- Vulnerability to loss
- Anniversary phenomenon
- Death (more likely in the first 12 months)

The GP as counsellor[1]

Important rules to bear in mind:

- The bereaved may be feeling very guilty.
- They may be angry towards their doctor or the medical profession in general.
- They need a clear explanation as to the exact cause and manner of death. Autopsy reports should be obtained and discussed.
- The bereaved tend to view an apparent lack of concern and support as disinterest or guilt.[12]
- Early intervention averts pathological grief.

The GP probably had a close relationship with the deceased and the family. The GP will have a special awareness of those at risk and the nature of the relationships within the family. The family is likely to maintain the relationship with the GP, expressing the physical and psychological effects of grief and consulting about intercurrent problems.[12]

Working through the stages of grief with patients will allow GPs to reach some acceptance of their own emotions, as well as ensure that patients feel supported and cared for, rather than distanced by embarrassment.

Help from religious sources is highly valued as it can meet both spiritual and personal needs. Other resources include funeral directors, hospice (and other) counsellors and support groups, such as those for SIDS.[12]

At least 30 minutes should be allowed for consultations.

Long-term counselling

Normal bereavement can persist for years. Ongoing counselling is indicated if it continues unabated, or psychiatric referral sought if grief is extreme. Regular enquiries during routine consultations or meetings are important if the patient appears to be coping.

Breaking bad news

Good communication skills are fundamental to giving bad news appropriately. When bad news is broken insensitively or inadequately the impact can be distressing for both giver and recipient, leaving lasting scars for the latter. For the doctor it may represent professional failure, fear of people's reaction and feelings of guilt. Doctors should have a plan for this difficult process and learn how to cope with the recipient's reaction. Most of the circumstances described apply to unexpected death or anticipated death.[14]

Sharing bad news with a patient

This difficult task is based on sound communication skills and good dialogue. The meeting should be face to face, not over the phone or internet.

Basic guidelines

- Plan the consultation, review clinical data, set aside ample time.
- Meet in an appropriate room with privacy and no interruption.
- Ask the patient if they would like company (e.g. a relative or friend).
- Make good eye contact and be alert for non-verbal responses.
- Use simple, understandable language; avoid medical jargon, speak clearly and sensitively.
- Be honest and diplomatically to the point (don't cover up the issue).
- Allow time, silence, tears or anger.
- Avoid inappropriate methods (refer to 'avoid' in TABLE 4.4) and don't give precise predictions about life expectancy.
- Write down significant points and use sketches or diagrams as appropriate.

Management

Follow the 10 basic steps of the management interview (see CHAPTER 2) with the emphasis on the patient's understanding of the message and his/her feelings about it (see TABLE 4.3). Offer ongoing support and arrangements for continuing involvement, including allied health professionals.

Table 4.3 Seven-step protocol for breaking bad news

1. Assess the patient's interest in, and capacity for, detailed information.
2. Establish the patient's beliefs about the illness, and what he or she wants to know.
3. Provide accurate information in small doses, checking regularly what has been understood.
4. Monitor how the patient feels about the problem and what has been said.
5. Repeat the messages as the illness progresses, especially after each new step of management and/or deterioration.
6. Involve family members as much as the patient wants.
7. Plan for continued involvement. An assurance of continuing contact between doctor and patient is important.

Source: Buchanan[15]

Coping with patient responses

- The responses cover a wide range—stunned silence, disbelief, acute distress, anger, extreme guilt.
- Be prepared for any of these responses.
- Appropriate training using simulated patients, video replays and skilled feedback improves communication skills.
- Give permission and encouragement for reactions, such as crying and screaming.
- Have a box of tissues available.
- A comforting hand on the shoulder or arm or holding a hand is an acceptable comfort zone.
- Offer a cup of tea or a cool drink if available.
- Ask the patients or relatives how they feel, what they would like to do and if they want you to contact anyone.
- Arrange follow-up.
- Give appropriate patient education material.
- Provide information about support services.

Children

Remember that there are two ‘patients’—child and family. The same ‘bad news’ principles apply. Talk in age-appropriate terms to the child with the aim of establishing their understanding of their illness and feelings.

Unexpected death

Some basic initial rules:[16]

- If relatives have to be contacted it is preferable for the doctor (if at all possible) or a sympathetic police officer to make the contact personally, rather than a relatively matter-of-fact telephone call from the hospital or elsewhere.
- If a telephone message is necessary, it should be given by an experienced person.
- The relatives or close friends should not drive to the clinic alone.

The setting for the interview:

- Use a suitable quiet private room if possible.
- See the recipients of the news alone in the room.
- Advise that the meeting should be undisturbed.

Guidelines for the doctor

- Be well prepared: check the facts and plan your approach.
- Always ask those involved if they have heard any news or know the reasons for the consultation.
- Always assess their understanding.
- Give information in an unhurried, honest, balanced, empathetic manner.[17]
- Look directly at the person you are talking to, be honest and direct, and keep information simple (avoid technical language).
- The sad news must be accompanied by positive support, understanding and encouragement.
- Give recipients time to react (offer time and moments of silence to allow the facts to sink in) and opportunities to ask questions.
- Avoid false reassurance.
- Remember that relatives appreciate the truth and genuine empathy.
- In the event of death, relatives should be given a clear explanation of the cause of death.

A list of guidelines for the interview is summarised in TABLE 4.4 .[16]

Table 4.4 Breaking bad news for unexpected death: recommended actions during the interview

Allow
Time
Opportunities to react
Silence
Touching
Free expression of emotions
Questions
Viewing of a dead or injured body
Avoid
Rushing
Bluntness
Withholding the truth
Platitudes
Protecting own inadequacies
Euphemisms
The notion ‘nothing more can be done’
Using medical jargon
Meeting anger with anger
Leaving the patient or loved one without a follow-on contact

Source: McLauchlan[16]

The depressed patient

Studies have emphasised the importance and therapeutic efficacy of counselling in the management of the depressed patient.[18] The most practical approach by the GP to the depressed patient is empathy, support and a logical explanation of their malaise. The author gives the following explanation to the patient.

> Depression is a very real illness that affects the entire mind and body. It seriously dampens the five basic activities of humans, namely their energy for activity, sex drive, sleep, appetite and ability to cope with life. They cannot seem to lift themselves out of their misery or fight it themselves. Superficial advice to ‘snap out of it’ is unhelpful because the person has no control over it.
>
> The cause is somewhat mysterious but it has been found that an important chemical is present in smaller

amounts than usual in the nervous system. It is rather like a person low in iron becoming anaemic.

Depression can follow a severe loss such as the death of a loved one, a marital separation or financial loss. On the other hand, it can develop for no apparent reason, although it may follow an illness such as glandular fever or influenza, an operation or childbirth.

Emphasising the ‘missing chemical’ theory really helps patients and family come to terms with an illness that tends to have socially embarrassing connotations. It also helps compliance with therapy when antidepressant medication is prescribed.

Ongoing contact, support and availability are an important component of counselling, with appropriate referral to someone with more expertise, should that be required. CBT is a most effective and important approach to the management of depressive illness.

Chronic pain

Patients suffering from long-term pain are a special problem, especially those with back pain who seem to be on a merry-go-round of failed multiple treatments and complex psychosocial problems. These patients are frequently treated in pain clinics. As family doctors, we often observe an apparently normal, pleasant person transformed into a person who seems neurotic, pain-driven and doctor-dependent. The problem is very frustrating to the practitioner, often provoking feelings of suspicion, uncertainty and discomfort.

De Vaul and colleagues[19] list five subgroups of patients where perplexing pain presents as the major symptom:

1. pain as a symptom of depression
2. pain as a delusional symptom of psychosis
3. pain as a conversion symptom of hysterical neurosis
4. pain as a symptom of an unresolved bereavement reaction
5. pain as a symptom of a ‘need to suffer’

Patients who somatise their symptoms present one of the most difficult challenges to our skills and usually require a multidisciplinary team approach.

Management involves:

- thorough medical assessment
- psychological assessment
- detailed explanations to the patient and family about treatment
- rational explanations about the cause of the pain

- management of associated problems (e.g. depression, sexual dysfunction)
- behavioural modification to encourage increased activity and a gradual return to normality

Problem gambling

Problem or pathological gambling is a persistent and recurrent behaviour despite its detrimental effect (disruption of personal, family or work life). It is undoubtedly a dependence disorder similar to alcohol and other drugs, with a similar approach to management. Refer to DSM-5 criteria for pathological gambling. Prevalence: 0.5–1.5% adult population.

Dangers

- Suicide risk (high)
- Major depression (up to 75%)
- Stress-related problems
- Domestic violence

Key warning

- Gambling >$200 week
- Chasing losses

Other telltale signs

- Spending many hours gambling
- Placing larger, more frequent bets
- Lying about behaviour
- Being secretive
- Promising cutting back, but not doing it
- Impulsive activity
- Mood swings
- Gambling at the expense of other pleasant social activities
- Growing debts

- Excessive drinking

First-line management

- Ask (as part of social history).
- Consider South Oaks Gambling screen to support provisional diagnosis.[20]
- Confront firmly if suspected.
- Consider using the Prochaska and DiClemente model of change (CHAPTER 12).
- Provide education material.
- Look into the family (domestic violence?) and provide support.
- Advise the family not to provide ‘rescue money’.
- There is no recommended pharmacological treatment.

Counselling approach

Problem gambling is a treatable condition and GPs can provide a central role in management. As for smoking and alcohol dependence, a brief intervention and education consultation session about the impact of excessive behaviour can be most effective. CBT is a very effective treatment for gambling. It combines systematic discussion and carefully structured behavioural assignments to help patients modify problematic thinking patterns and behaviours. ‘CBT directed towards correcting erroneous perceptions, irrational beliefs and misunderstanding of concepts of randomness and independence of chance events is a fundamental element of any therapeutic approach.’[21]

It is appropriate to use specialist gambling counsellors when one’s intervention is not proving effective or where there is evidence of disturbing gambling problems. (Refer to: www.gaaustralia.org.au)

Family counselling

Relationship disharmony

Family doctors often have to provide counselling for one or both partners in a relationship. The problems may be resolved quite simply or be so complex that relationship breakdown is inevitable despite optimal opportunities for counselling.

Opportunities for prevention, including anticipatory guidance about relationship problems, do exist and the wise practitioner will offer appropriate advice and counselling. Examples include an accident to a child attributable to neglect by a parent, or similar situation in which that parent

may be the focus of blame, leading to resentment and tension. The practitioner could intervene from the outset to alleviate possible feelings of guilt and anger in that relationship.

Some common causes of relationship disharmony are:

- selfishness, jealousy
- unrealistic expectations
- financial problems/meanness
- poor communication, not listening to each other
- sickness (e.g. depression)
- drug or alcohol excess
- fault finding, 'playing games' with each other
- driving ambition
- immaturity

Basic counselling of couples/families

The following text on basic counselling of couples[22] can also be regarded as a patient education sheet:

> The two big secrets of relationship success are caring and responsibility.

Some important aspects

- Research has shown that we tend to choose partners who are similar to our parents and that we may take our childish and selfish attitudes into our relationships.
- We often expect our partner to change and meet our needs.
- If we take proper care and responsibility, we can keep these problems to a minimum.
- Physical passion is not enough to hold a relationship together—'when it burns out, only ashes will be left'.
- While a good sexual relationship is great, most experts agree that what goes on *out* of bed counts for more.
- When we do something wrong, it is most important that we feel forgiven by our partner.

Positive guidelines for success (summary)[22]

1. Know yourself.
2. Share interests and goals.
3. Make love, not war.
4. Cherish your partner.
5. Prepare yourself for parenthood, where relevant.
6. Seek proper help when necessary.
7. Do unto your partner as you would have your partner do unto you.

The BE attitudes (virtues to help achieve success)	
BE honest	**BE** loyal
BE loving	**BE** desiring
BE patient	**BE** fun to live with
BE forgiving	**BE** one
BE generous	**BE** caring

Making lists—a practical task

Make lists for each other to compare and discuss.

- List qualities (desirable and undesirable) of your parents.
- List qualities of each other.
- List examples of behaviour each would like the other to change.
- List things you would like the other to do for you.

Put aside special quiet times each week to share these things.

Pitfalls[23]

The GP who is too closely attached to one member of a couple can easily become trapped in the role of the ‘rescuer’ or ‘saviour’ of that person. The best defence against this trap is to respect the couple’s autonomy and work with them to achieve the goals they set for themselves, thus avoiding three major pitfalls for the GP in treating couples or families:

1. assuming personal responsibility for changing the family
2. working alone, neglecting the assistance of the family
3. becoming a ‘rescuer’ or ‘saviour’

Other pitfalls

- Conducting family therapy in the absence of a significant family member
- Breaching confidentiality of individuals within a relationship or family
- Failing to recognise the ‘ganging-up effect’
- Taking sides
- Failing to use available resources
- Overrelating to your own experiences

Possible solutions to avoid pitfalls[23]

- Let the couple do the work.
- Ensure that the goals for therapy are realistic.
- Point out that all family members have to work together and that therapy works best when there is openness on all sides.
- Identify any tendency to look for scapegoats within the family.
- Look out for vulnerable family members—the ‘hidden patient’.
- Avoid trying to achieve quick solutions.
- Obtain clear-cut agreements on confidential matters and record this in the history.
- Keep an open mind and avoid forcing your own values on to the family.
- Share the burden with a colleague or other resources.

Summary: counselling skills strategies

- Provide guidance and facilitation to allow the patient to gain insight.
- Use appropriate ‘gentle’ confrontation to allow self-examination.
- Help patients to explore their own situation and express emotions such as anxiety,

guilt, fear, anger, hostility and hurt feelings.

- Explore possible feelings of insecurity and allow free expression of such feelings.
- Ask key searching questions, such as:

  What do you think deep down is the cause of your problem?

  How do you think your problem should be treated?

- Provide 'okay' specific suggestions, such as:

  I wonder if your basic problem is that you are a perfectionist?

  Many people in your situation feel guilty about something that may be trivial and need to feel forgiven.

Effective counselling comes from commitment, experience and a genuine caring and compassionate feeling for patients and their ethos.

Key rules to counselling

- The patient must leave feeling better.
- Provide *insight* into their illness and/or behaviour.
- Address any feelings of *guilt* (people must feel okay or forgiven about any perceived transgression).

If one feels out of one's depth, then immediate referral to an expert is important. CBT is an appropriate therapy for most conditions.

Patient education resources

Hand-out sheets from *Murtagh's Patient Education* 8th edition:

- Depression
- Coping with a crisis
- Bereavement
- Gambling: problem gambling

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5 Health promotion and patient education

Whoever is to acquire a competent knowledge of medicine, ought to be possessed of the advantage of instruction.

HIPPOCRATES (460–370 BC)

Health promotion

Health promotion is the motivation and encouragement of individuals and the community to see good health as a desirable state that should be maintained by the adoption of healthy practices. It is also the process of helping people to increase control over and improve their health (WHO definition 2009).

For those who feel healthy, the message may have little meaning, but it is reinforced by contact with others who become ill, particularly within the family.

The Lalonde report[1] highlighted the notion that all causes of death and disease had four contributing elements:

- inadequacies of the existing health care system
- environmental hazards
- behavioural factors and unhealthy lifestyles
- human biological factors

Health education

Health education is the provision of information about how to maintain or attain good health.

There are many methods, including the advertising of health practices, the provision of written information (e.g. about diet and exercise, immunisation, accident prevention and the symptoms

of disease) and information about methods to avoid disease (e.g. sexually transmitted infection).

Illness education

A lot of so-called ‘health’ education is, in reality, information about the cause of particular illnesses. Clearly, the medical practitioner is in a pre-eminent position to provide his or her patients with specific information about the cause of an illness at the time, either individually or to the family. This educative strategy has a preventive objective that is often the modification of help-seeking behaviour.

Every consultation is an opportunity to provide information about the condition under care and this can be reinforced in written, diagrammatic or printed form. Patients’ own X-rays can be similarly used to illustrate the nature of the problem.

Health promotion in general practice

GPs are ideally placed to undertake health promotion and prevention, mainly due to opportunity.

There are several reasons for this health promotion role:

- Population access: over 80% of the population visits a GP at least once a year.[2]
- On average, people visit a GP about five times each year.
- GPs have a knowledge of the patient’s personal and family health history.
- The GP can act as leader or coordinator of preventive health services in his or her local area.
- The GP can participate in community education programs.
- GPs should undertake opportunistic health promotion—the ordinary consultation can be used not just to treat the presenting problem, but also to manage ongoing problems, coordinate care with other health professionals, check whether health services are being used appropriately and undertake preventive health activities.[2]

Opportunistic health promotion

The classic model by Stott and Davis (see TABLE 2.1 in CHAPTER 2) highlights the opportunities for health promotion in each consultation.[3] Since the consultation is patient-initiated, it is the doctor who needs to be the initiator of preventive health care. The potential in the consultation involves reactive and proactive behaviour by the doctor (see FIG. 5.1).[4]

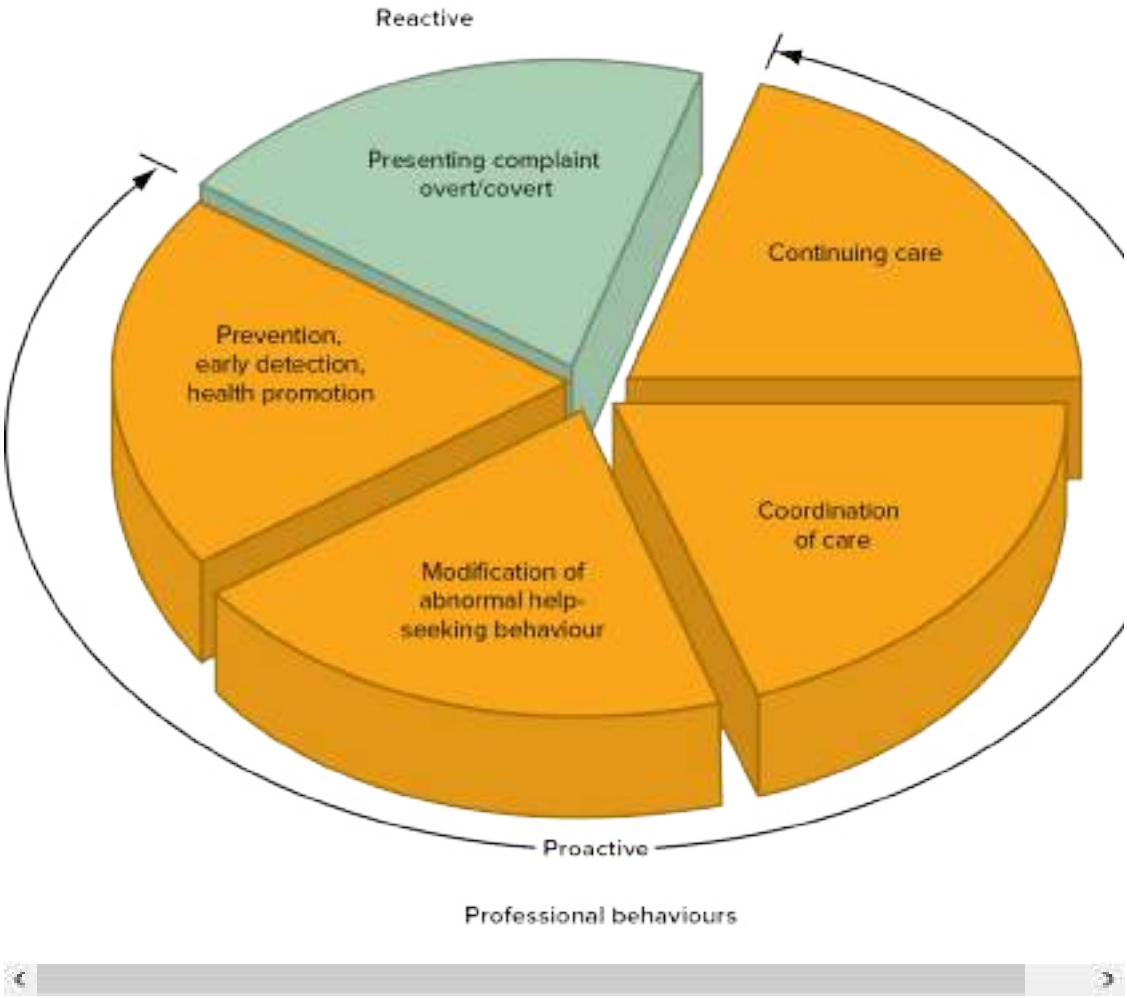

FIGURE 5.1 The potential in every general practice consultation

Source: Reproduced with permission from M Sales[4]

Reactive professional behaviour deals only with the presenting complaint. It may be performed with skill but if the practitioner is only trained to perform reactively then the opportunity for preventive and promotive health care will be lost.

Proactive behaviour is defined as professional behaviour that is necessary for the patient's well-being, but it is performed not merely as a response to the presenting problem and it is initiated by the doctor.[4] It includes health promotion, preventive care and screening and the early detection of disease, before it becomes symptomatic. Other aspects of proactive care are seen in FIGURE 5.1 .

Proactive behaviour also includes:[4]

- continuing care of a previously treated problem (e.g. rechecking blood pressure, checking diabetic control, follow-up bereavement counselling)

- coordination of care by organising referral to appropriate agencies or specialists and maintaining adequate medical records
- the modification of abnormal or inappropriate help-seeking behaviour (e.g. the person who never attends is at risk from ‘silent disease’; the too-frequent attender wastes resources and serious illness may be overlooked)

This mix of reactive and proactive behaviour is not appropriate in every consultation. It requires counselling skills and training in the delivery of quality general practice.

Methods

- Being informed and updated by maintaining continuing medical education, especially in preventive roles.
- Using health promotional material for patient education:

  hand-outs

  waiting room posters

  waiting room video systems
- Having an efficient medical record system.
- Operating a patient register and recall system.
- Encouraging regular health checks for at-risk groups.
- Providing regular advice on:

  nutrition

  exercise

  stress management

  weight control
- Providing personal health records to the parents of newborn babies.

Health goals and targets

Health goals and targets as determined by the Health Targets and Implementation Committee[5] were set in three areas—population groups, major causes of illness and death, and risk factors (see TABLE 5.1).

Table 5.1 Established health promotion goals and targets[5]

Population groups

- Socioeconomically disadvantaged
- Indigenous Australians
- International arrivals—migrants, refugees
- Older people
- Children and adolescents
- Vulnerable women and men

Major causes of morbidity and mortality

- Cardiovascular and cerebrovascular disease
- Cancer esp. lung, breast, cervical, prostate, skin
- Diabetes mellitus
- Respiratory disease esp. asthma, COPD
- Communicable diseases
- Mental illness
- Musculoskeletal disease
- Disability
- Accidents and injury

Risk factors

- Hypertension, high blood lipids, obesity
- Drugs—smoking, alcohol, illicit drugs and substance abuse, pharmaceutical misuse/abuse
- Physical inactivity
- Occupational and environmental health hazards
- Inappropriate nutrition
- Unprotected and unsafe sexual activity

The seven priority health areas leading to reduction of morbidity and mortality rates for Australians are:[5]

- asthma
- cancer control
- cardiovascular health
- obesity control

- diabetes mellitus
- injury prevention and control
- mental health

Promoting healthy lifestyle in general practice

GPs can provide a simple framework to encourage patients to adopt a healthy lifestyle whether they have a particular disorder or not. The acronyms act as a good aide-mémoire for practitioners for opportunistic health promotion.

The SNAP guide[6]

The SNAP guide was developed by the Royal Australian College of General Practitioners to address important risk factors with patients with a view to encouraging change if appropriate. The guide comes as a comprehensive booklet and includes the 'Estimation of absolute 5-year risk of cardiovascular events' tables.

The SNAP guide can be summarised by the following risk factors.

S = **S**moking
N = **N**utrition
A = **A**lcohol
P = **P**hysical activity

The guide emphasises that there are *health inequalities* in the community because the risk factors are far more prevalent in people from low-socioeconomic-status backgrounds and Indigenous Australians.

The guide focuses on the '5 As' as stages of change theory to promote change of lifestyle where appropriate (see TABLE 5.2).

Table 5.2 The '5 As'

1	**Ask**	Identify patients with risk factors
2	**Assess**	Level of risk factor and its relevance to the individual in terms of health Readiness to change/motivation
3	**Advise**	Provide written information Provide a lifestyle prescription
4	**Assist**	Pharmacotherapies Support for self-monitoring

5	**Arrange**	Referral to special services Social support groups Phone information/counselling services Follow-up with the GP

This information can be accessed at: http://www.racgp.org.au/guidelines/snap.

The NEAT guide

The NEAT guide (see TABLE 5.3) is similar to the SNAP guide, but with a greater emphasis on counselling the patient about lifestyle and the importance of stress management.

Table 5.3 The NEAT guide

N	=	**N**utrition: optimal diet
E	=	**E**xercise/physical activity
A	=	**A**voidance or moderation of potential harmful substances (CATS): • caffeine • alcohol • tobacco • sugar, salt and social drugs
T	=	**T**ranquillity and promotion of recreation, relaxation techniques, meditation

Psychosocial health promotion

There is a tendency for health goals and targets to focus mainly on physical illness and not emphasise mental health. However, this area represents an enormous opportunity for anticipatory guidance. It includes the important problems of stress and anxiety, chronic pain, depression, crisis and bereavement, sexual problems, adolescent problems, bullying, domestic abuse, child behavioural problems, psychotic disorders and several other psychosocial problems.

Time spent in counselling, giving advice and stressing ways of coping with potential problems such as suicide and deterioration in relationships is rewarding. GPs need to pay more attention to promoting health in this area, which at times can be quite complex.

Nutrition for health

Good nutrition is fundamental to good health. It influences management in all branches of medicine. Modern people's health varies from the excesses of inappropriate nutrition, resulting in obesity and various degenerative disorders, to malnutrition and other deficiency states seen in those unfortunates deprived of nutrients.

The essential components of nutrition[10] can be classified as:

- macronutrients—proteins, fats and carbohydrates, which are interchangeable sources of energy and also water
- macrominerals—sodium, chloride, potassium, calcium, phosphate and magnesium
- micronutrients—water-soluble vitamins (e.g. C, B); fat-soluble vitamins (e.g. A, E, K); essential trace elements (e.g. copper, iodine, iron, zinc)

Nutritional factors may play a vital role in the causation of several of the major diseases, such as coronary artery disease, hypertension, diabetes and cancer. Where weight loss is a goal, see CHAPTER 80.

Special diets are important in the management of many hereditary metabolic disorders, such as phenylketonuria and galactosaemia, and several other disorders such as coeliac disease.

Protein[11]

Proteins are composed of carbon, hydrogen, oxygen, nitrogen, phosphorus, sulphur and iron. They make up the greater part of plant and animal tissue and provide the amino acids essential for the growth and repair of tissue. Protein in the body in muscle, connective tissue and enzymes is constantly being broken down, while dietary protein is hydrolysed to amino acids that are both essential and non-essential. A complete protein is one that contains all the nine indispensable amino acids, namely, histidine, isoleucine, leucine, lysine, methionine, phenylalanine, threonine, tryptophan and valine.

Protein in animal products (fish, meat and milk) is of high quality and that in vegetable products is lower because of a limited supply of lysine (in cereals) and methionine and cysteine (in legumes).[12] Vegetarian diets are usually adequate in protein, especially if the combining vegetable groups complement each other in basic amino acid groups. Diets that exclude all animal products may be inadequate, especially in children. Infants and children require 2–2.2 g protein/kg/day.

- High protein content foods—lean beef and lamb, chicken, fish, eggs, milk, cheese, soy beans
- Medium protein content foods—bread, spaghetti, corn, potatoes (cooked), rice (cooked), cabbage, cauliflower

Energy malnutrition

This is a deficiency syndrome with a reduction in all macronutrients, energy (kilojoules) and many micronutrients due to an inadequate intake of protein and energy foodstuffs.

It is commonly found in infants and children in developing countries but can occur in persons of any age in any country.

Clinically, protein-energy malnutrition has three forms:

1. dry (thin, desiccated)—*marasmus*
2. wet (oedematous, swollen)—*kwashiorkor*
3. combined—*marasmic kwashiorkor*

Marasmus

Clinical features:

- grossly underweight
- gross muscle wasting
- abdominal distension
- no fat
- hungry
- ‘old man’s’ face
- no oedema
- normal hair

Caused by a diet low in protein and calories.

Kwashiorkor

Clinical features:

- oedema (feet first, then generalised)
- ‘moon’ face
- anorexia
- hair pale and thinned
- apathetic

- skin changes

Caused by a diet low in protein with some carbohydrate, leading to hypoalbuminaemia.

Carbohydrates

Dietary carbohydrates include simple sugars, complex carbohydrates (starches) and indigestible carbohydrate (dietary fibre). Carbohydrates are the main source of dietary energy. The two most important crops feeding the world are rice and wheat, which are rich in starch. Starch and sucrose account for the majority of carbohydrates consumed in all diets. Carbohydrates that are available in food are:

- sugars—sucrose, lactose, maltose, glucose, fructose
- polyols—sorbitol, xylitol, maltitol, lactitol
- starch—amylose, amylopectin
- dextrose

As long as adequate energy and protein are provided in the diet, there is no specific requirement for dietary carbohydrate. A small amount—100 g/day—is necessary to prevent ketosis.[12]

The glycaemic index (GI)

The GI, which applies to carbohydrate foods, is a numerical index based on a reference point of 100. It is a measure of the capacity to increase postprandial glucose levels compared to a glucose load. The standard food is glucose, which is given an arbitrary level of 100.

The higher the GI, then the higher the rise in blood glucose level and thus the greater the insulin response. Low-GI foods may improve glycaemic control.

Low-GI foods (<55) include dense wholegrain breads, porridge, most fresh fruits, yoghurt and low-fat milk. High-GI foods (>70) include sweet breakfast cereals, potatoes, white bread, watermelon, ripe bananas, dates, white rice and biscuits.

Fat

Dietary fat, which is composed mainly of fatty acids and dietary cholesterol, is the most concentrated source of food energy.[11]

Fatty acids are classified according to the number of unsaturated double bonds:

- nil—saturated (e.g. butyric and stearic acids)
- one—monounsaturated (e.g. oleic acid)

- more than one—polyunsaturated (e.g. linoleic acid, eicosapentanoic acid [EPA], docosahexanoic acid [DHA])

Polyunsaturated fatty acids (two or more unsaturated bonds) can be subdivided into:

- n-6 (e.g. linoleic acid, 2 unsaturated bonds; arachidonic acid, 4 unsaturated bonds)
- n-3—omega-3 fatty acids (e.g. alpha-linolenic acid, 3 unsaturated bonds; EPA, 5 unsaturated bonds; DHA, 6 unsaturated bonds)

The n-3 and n-6 polyunsaturated fatty acids with chain lengths of 18 or more are called essential fatty acids because they are required for vital body functions and animals, including humans, are unable to synthesise them.[11]

The proportions of saturated, monounsaturated and polyunsaturated fatty acids in the diet are important determinants of health and disease.[11] The current strategy is to reduce total fat intake and reduce saturated fats and increase unsaturated fats, especially n-3 polyunsaturated fats.

Fish oil contains omega-3 fatty acids (EPA and DHA), which are considered more potent than the omega-3 fatty acids found in plants. The value of omega-3 fatty acids in preventing cardiovascular mortality has been well proven. They have no effect on cholesterol levels but have a well-documented potent hypotriglyceridaemic effect.[13]

Omega-3 rich foods include salmon, trout, halibut and tuna. Suitable plants include avocado, walnuts, other nuts and seeds, and legumes (beans and lentils). Our diet should include these fats.

Trans fats, which are unsaturated fats usually generated industrially by a hydrogenation process, should be avoided.

Cholesterol, which is a major constituent of cell membranes, is synthesised by the body and is not an essential nutrient. The plasma cholesterol level, and hence the amount of cholesterol in the diet, has been related to the development of atherosclerosis.

Nutritional assessment

The first step in nutritional assessment is to identify the high-risk patient.[14] Those at high risk of nutritional insufficiency include those with a history of obesity, eating disorders, chronic illness, psychological disorders, the elderly, the institutionalised, trauma victims and those with long periods of hospitalisation, including major surgery. Of particular interest is the rate of growth and development in infants and children and the body composition in children and adults.

When taking the history it is appropriate to include a 24-hour recall of foods eaten and ideally get the patient to complete a symptom questionnaire that can then be linked to a computerised nutritional evaluation program, such as Nutricheck.[14] Evaluate sunlight exposure.

A nutritionally focused physical examination should be performed on each patient at risk, with

the emphasis on body weight, waist size, muscle wasting, fat stores and signs of micronutrient deficiencies. Examples of the latter include zinc deficiency, which affects taste, smell and the skin. Deficiencies of vitamins B6 and B12 cause neurological disorders, such as peripheral neuropathy.[11] Alcoholism and malnutrition affect many systems, including the gastrointestinal system. The oral cavity, especially the gums, teeth and buccal mucosa, are affected by vitamin B complex and vitamin C deficiencies. Bones and joints are affected in scurvy, rickets, osteomalacia and osteoporosis. The important anthropometric measurements include height and weight, skinfold thickness and waist:hip circumference ratio (refer to CHAPTER 67). Laboratory investigations depend on the clinical examination and should be selective.

The general principles of optimal nutrition

In order to help people make healthy choices, the health foundations of several countries have developed recommendations for eating a healthy diet.

The heart-healthy eating pyramid of the Australian Nutrition Foundation (2012)[11] has a simplified system, namely:

- Eat most—vegetables, dried peas, beans and lentils, cereals, bread, fruit and nuts
- Eat moderately—healthy protein foods such as lean meat, eggs, fish, chicken (without skin), milk, yoghurt, cheese
- Eat in small amounts—oil, margarine, reduced-fat spreads, butter, sugar and salt, and avoid trans fats

The RACGP in its red book[15] outlines dietary recommendations based on national guidelines. For adults this is summarised as follows.

Enjoy a wide variety of foods each day:

- five serves of vegetables and two serves of fruit
- lean meat, fish, poultry, eggs, tofu, nuts and seeds and/or alternatives
- grains and cereals, mostly wholegrain or high-fibre varieties such as bread, rice, pasta, oats and couscous
- drink plenty of water

Take care to:

- limit saturated fat
- limit salt intake
- limit alcohol intake

- limit sugars and foods containing added sugars, including soft drinks and commercial juice
- limit red meat to 3–4 serves per week and limit or avoid processed meat
- care for food: prepare and store it safely
- encourage and support breastfeeding

See CHAPTER 80 for further detail on the dietary management of a person who wants to lose weight.

Antioxidants

The antioxidant issue is still controversial and unclear. Empirical observation of healthy communities over the years indicates good health outcomes, especially with cardiovascular status, in people having an optimal diet containing high levels of vitamins and minerals (especially from fruit and vegetables).

Food antioxidants (see TABLE 5.4) appear to protect against free radicals, which can suppress immunity.[16]

Table 5.4 Food antioxidants

Vitamin A, especially beta-carotene
Vitamin C
Vitamin E
Ubidecarenone (co-enzyme Q10)
Selenium, zinc, manganese and copper (nutrient cofactors)

Source: Sali[16]

Prime sources of antioxidants in food[17]

- Vitamin C—citrus fruits, berries, papaya, green leafy vegetables
- Vitamin E—seed-like cereal grains, nuts and oils (plants), eggs
- Beta-carotene—orange-coloured and dark-green leafy vegetables
- Selenium—grains, meats, Brazil nuts, fish
- Copper—cocoa, wheat bran, yeast

- Ubiquinone—meats, fish, peanuts
- Phytochemicals—soy, tea, green tea, herbs, apples, onions, cocoa

Folate-containing foods

- Green leafy vegetables—broccoli, spinach
- Wheat grain
- Wholegrain cereals
- Starchy beans—kidney and butter
- Peas, corn, cauliflower
- Nuts
- Avocado
- Liver
- Folic acid fortified foods (e.g. breakfast cereals)

Vitamin deficiency disorders[18]

These are rare in our society but can occur sporadically and are not rare in children in some third world countries or in refugees from these countries. Deficiencies tend to be seen as a specific disorder or as a multivitamin effect.

- *Vitamin A (beta-carotene/retinol)* deficiency causes night blindness and eye disease, as well as dryness with keratinisation of the conjunctivae and cornea. It causes growth retardation in children. Toxicity from overdosage of vitamin A is a serious problem.
- *Vitamin B complex*

  Vitamin B1 (thiamine) deficiency causes **beriberi** (dry or wet), heart failure, neuropathy and also Wernicke–Korsakoff syndrome (typically in alcoholics).

  Vitamin B2 (riboflavin) deficiency causes growth retardation, dry scaly skin and angular cheilitis.

  Vitamin B3 (niacin, nicotinic acid) deficiency causes **pellagra**, the classic ‘d’ triad—diarrhoea, dementia, dermatitis ± neuropathy.

  Vitamin B6 (pyridoxine) deficiency may cause oral soreness, anaemia and CNS dysfunction.

Vitamin B12 (cobalamin) deficiency causes **pernicious anaemia**, neuropathy, glossitis and memory dysfunction.

- *Vitamin C (ascorbic acid)* deficiency is responsible for **scurvy**. Clinical features: muscle weakness, malaise, fatigue, bleeding swollen inflamed gums, atraumatic haemarthrosis, cachexia, oedema, impaired wound healing, impaired bone growth. One sign is the hyperkeratotic hair follicle with surrounding hyperaemia. Diagnosis by decreased plasma ascorbic acid and X-rays of bones and joints.

- *Vitamin D (calciferol)* deficiency causes **rickets** in children and **osteomalacia** in adults. Clinical features (rickets): impaired growth, skeletal deformities (bow legs, pelvis, 'rachitic rosary'), inability to walk, bone pain (arms, legs, spine, pelvis), dental deformities, muscle weakness. In adults: muscle weakness, bone pain, bowing of long bones. Diagnosis: low plasma 25(OH)D_3 and phosphate; elevated PTH and alkaline phosphatase; X-rays of joints and long bones of leg.

- *Vitamin E (tocopherol*) deficiency causes no specific disease but may result in vague, undifferentiated symptoms and anaemia.

- *Vitamin K (phylloquinone*) deficiency is rare and can lead to an increased bleeding tendency.

- *Folic acid* deficiency is responsible for pernicious anaemia and neural tube defects in the fetus.

Anaemia and iron

Iron-deficiency anaemia is a common problem in our society, particularly in children from 6 months to 2 years who have been given a lot of cow's milk. In such cases it is important to educate people about iron-rich foods and the quantities they need (see CHAPTER 13).

Guidelines for safe consumption of alcohol (current NHMRC recommendations)[19]

Healthy males and females

- No more than ten standard drinks per week

- No more than four standard drinks on any single occasion, then don't drink at all for 2–3 other days

Young people

- People aged under 18 years should not drink alcohol

Pregnancy and breastfeeding

- No alcohol is the safest option

Refer to CHAPTERS 12 and 100 for alcohol guidelines.

Iodine deficiency

The body needs small amounts of iodine to maintain normal function of the thyroid gland—crucial for normal growth and development. In iodine-deficient areas (in soil and water) there is a high rate of stillbirths, congenital hypothyroidism and cretinism. In adults, deficiency causes goitre and hypothyroidism. The usual intake of iodine in healthy persons is 100–200 mcg/day, mostly from iodised salt. An adequate intake is 150 mcg/day with higher requirements for pregnancy (220 mcg) and breastfeeding (290 mcg).[20]

Australian dietary guidelines[19]

(See: www.nhmrc.gov.au)

1. To achieve and maintain a healthy weight, be physically active and choose amounts of nutritious food and drinks to meet your energy needs
2. Enjoy a wide variety of nutritious foods from these five groups every day

- plenty of vegetables, including different types and colours, and legumes/beans
- fruits
- grain (cereal) foods, mostly wholegrain and/or high cereal fibre varieties, such as breads, cereals, rice, pasta, noodles, polenta, oats, quinoa and barley
- lean meat and poultry, fish, eggs, tofu, nuts and seeds, and legumes/beans
- milk, yoghurt, cheese and/or alternatives, mostly reduced fat (reduced fat and milk not suitable for children <2 years)
- drink plenty of water

3. Limit intake of foods containing saturated fat, added salt, added sugars and alcohol

- limit intake of foods high in saturated fat, such as many biscuits, cakes, pastries, pies, processed meats, commercial burgers, pizza, fried foods, potato chips, crisps and other savoury fats (e.g. butter, cream, cooking margarine, coconut and palm oil) with foods that contain predominantly polyunsaturated and monounsaturated fats, e.g. oils, nut butters/pastes and avocado (low fat diets are unsuitable for children <2 years)
- limit intake of food and drinks containing added salt; do not add salt to foods in cooking or at

the table

- limit intake of food and drinks containing added sugar, such as confectionery, sugar-sweetened soft drinks and cordials, fruit drinks, vitamin waters, and energy and sports drinks
- if you choose to drink alcohol, limit intake and follow NHMRC guidelines

4. Encourage, support and promote breastfeeding
5. Care for your food, and prepare and store it safely

Summary of general dietary guidelines for good health

- Keep to an ideal weight (BMI: adults 20–25).
- Eat a high-fibre diet.
- Eat more fruits and vegetables, least-processed breads and cereals, preferably wholegrain.
- Eat fish at least twice a week (daily if possible).
- Choose a nutritious diet.
- Eat less saturated fat, refined sugar and salt.
- Use low-fat dairy products—milk and yoghurt.
- Avoid fast foods and deep-fried foods.
- Do not eat animal meat every day, and then only in small portions. Note that processed meats, such as sausages, have a very high fat content.
- Use monounsaturated (olive) oils and perhaps margarine instead of butter.
- Use olive oil for cooking rather than polyunsaturated oils.
- Always trim fat off meat.
- Limit alcohol intake to 10 standard drinks per week.
- Drink more water.
- Limit salt intake; pepper is okay.
- Limit caffeine intake (0–3 drinks a day maximum).
- Check plasma cholesterol level and, if it is elevated, aim to reduce it with diet.

- The appropriate diet is a low-carbohydrate, healthy-fat, Mediterranean-based diet.

Patient education resources

Evidence has shown that intervention by GPs can have a significant effect on patients' attitudes to a change to a healthier lifestyle. If we are to have an impact on improving the health of the community, we must encourage our patients to take responsibility for their own health and thus change to a healthier lifestyle. They must be supported, however, by a caring doctor who follows the same guidelines and maintains a continuing interest. Examples include modifying diet, nutrition, cessation of smoking, reduction of alcohol intake, undertaking exercise, depression and anxiety.

Patient education materials have been shown to have a beneficial effect. Giving patients a hand-out about tetanus increased the rate of immunisation against tetanus among adults threefold.[7] An education booklet on back pain for patients reduced the number of consultations made by patients over the following year and 84% said that they found it useful.[8] Providing systematic patient education on cough significantly changed the behaviour of patients to follow practice guidelines and did not result in patients delaying consultation when they had a cough lasting longer than 3 weeks or one with 'serious' symptoms.[9]

One form of patient education is giving hand-outs (either prepared or printed from a computer at the time of the consultation) to the patient as an adjunct to the verbal explanation which, it must be emphasised, is more important than the printed hand-out.

Murtagh's Patient Education 8th edition:

- Diet guidelines for good health

Resource

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6 Prevention in general practice

When meditating over a disease, I never think of finding a remedy for it, but, instead, a means of preventing it.

LOUIS PASTEUR 1884

Definitions[1]

Prevention may be defined as the means of promoting and maintaining health or averting illness.

It is concerned with removal or reduction of risks; early diagnosis; early treatment; limitation of complications, including those of iatrogenic origin; and maximum adaptation to disability.

The promotion of health concerns helping well people to learn healthy behaviours and to accept responsibility for their own well-being.

A preventive attitude implies that the doctor understands and can utilise the preventive potential in each primary care consultation by an 'opportunistic approach'. In addition to the traditional management of both presenting and continuing problems, the doctor takes the opportunity to modify the patient's health-seeking behaviour, to provide education about the illness and to promote health by relating the patient's present condition to previous unhealthy behaviour.

A general reference for prevention is the RACGP red book—*Guidelines for Preventive Activities in General Practice* (9th edn). See: https://www.racgp.org.au/clinical-resources/clinical-guidelines/key-racgp-guidelines/.

Primary prevention

Primary prevention includes action taken to avert the occurrence of disease. As a result there is no disease. Primary preventive strategies include:

1. education to bring about changes in lifestyle factors known to be associated with diseases (e.g. smoking cessation, healthy balanced diets, reduction in alcohol intake, exercise)
2. sterilisation of surgical instruments and other medical equipment

3. eradication, as with vector control of mosquitoes to prevent malaria
4. immunisation against infective diseases
5. sanitation, keeping our water supplies clean and disposing efficiently of sewage and industrial wastes
6. legislation to ensure that some of these primary preventive measures are carried out

Secondary prevention

Secondary prevention includes actions taken to stop or delay the progression of disease.

The term is usually applied to measures for the detection of disease at its earliest stage, i.e. in the presymptomatic phase, so that treatment can be started before irreversible pathology is present. The early recognition of hypertension through routine testing (screening) of patients allows treatment during the presymptomatic phase of the illness process. Screening for cervical cancer allows the treatment of cervical dysplasia, a premalignant condition. Other examples include mammography and endoscopy for polyps of the large bowel.

Tertiary prevention

Tertiary prevention includes the management of established disease so as to minimise disability.

The term is usually applied to the rehabilitation process necessary to restore the patient to the best level of adaptation possible when there has been damage of an irreversible nature. A patient who has suffered a stroke because of hypertension may be restored to a useful lifestyle with appropriate rehabilitation.

Relationship between types of prevention

It can be seen that there is a clearer demarcation between primary and secondary prevention than between secondary and tertiary prevention, although the latter term is particularly useful in dealing with the elderly and the handicapped. Conceptually, curative medicine falls within the definitions of secondary and tertiary prevention while public health measures are mainly concerned with primary prevention. Prevention is really wider than medical practice but because of the success of public health practices in the past, more attention is now being focused on prevention by doctors (see FIG. 6.1).[2]

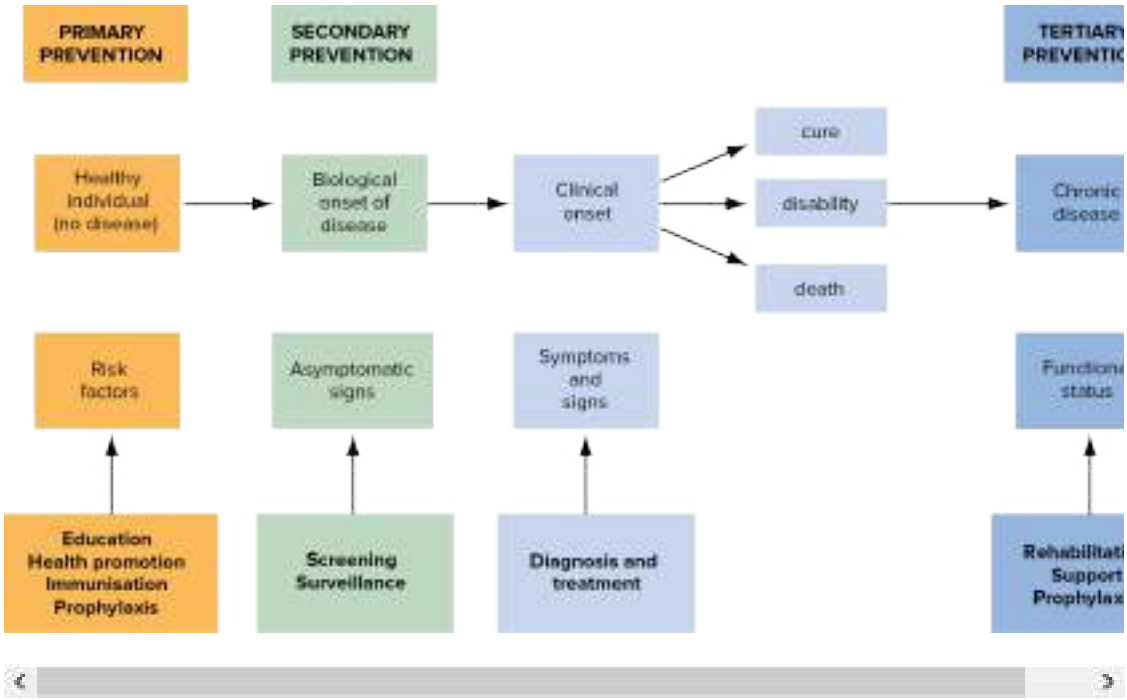

FIGURE 6.1 The phases of prevention in relation to the natural history of disease

As GPs our role in prevention is twofold.

1. First, we can recognise the preventable factors that are involved in an illness process and determine appropriate interventions.

2. Second, we can act to implement the preventive measure. In cases where the responsibility rests with the individual or the community, doctors can support prevention through education, applying political pressure or working with community agencies.

The practice of preventive medicine

What is preventable?

The first step in the implementation of prevention is to define which specific diseases can be prevented and to what extent, given certain restraints such as human resources, technology and the cost to the community. All diseases have a potential preventability but it may be unrealistic to try to achieve this.

Diseases that can be prevented can be grouped according to their aetiology. They fall into the following broad categories:

- genetic disorders

- conditions occurring during pregnancy and the puerperium
- developmental disorders
- accidents
- infections
- addictions
- behavioural disorders
- occupational disorders
- premature vascular disease
- neoplasms
- handicap in the disabled
- certain ‘other’ diseases (e.g. diverticular disease)

Mortality is the only reliable index by which the outcome of preventive activities can be judged. Conditions can be ranked in importance as causes of premature death according to the ‘years of potential life lost before 78 years [of age]’ as follows:[1]

- Accidents, poisoning, suicide and violence
- Neoplasms
- Circulatory diseases
- Perinatal conditions
- Congenital conditions
- Alzheimer disease

This gives quite a different perspective to prevention and explains why the efforts of public health authorities and practising doctors do not always coincide.

The interventions available to us in medical practice are as follows:

1. educational—health promotion, health education and illness education
2. screening
3. surveillance

4. interventional care—immunisation, behaviour modification and drug prophylaxis
5. rehabilitation

Optimal opportunities for prevention

Primary prevention par excellence can be practised in general practice under the opportunities provided by the following clinical circumstances:

- antenatal care
- postnatal care
- advising people travelling overseas
- visits by infants with their parents
- times of crisis or potential crisis
- pregnancy planning

The Royal College of General Practitioners (UK) has identified the seven most important opportunities for prevention as:

1. family planning
2. antenatal care
3. immunisation
4. fostering the bonds between mother and child
5. discouragement of smoking
6. detection and management of raised blood pressure
7. helping the bereaved

Mortality and morbidity considerations

An understanding of the mortality and morbidity patterns in the modern human being is essential to the planning of preventive programs. The great infectious diseases of the past, such as tuberculosis, syphilis, smallpox, influenza, diphtheria and streptococcal infections, have been largely contained but other diseases have become prominent as life expectancy increases. The great modern diseases are atherosclerosis (hardening of the arteries), malignant disease (cancer), HIV infection and iatrogenesis (doctor-induced illness). The two most common causes of death in Australia are cancer and cardiovascular disease, each accounting for approximately 20% of all

deaths.[3]

By comparison, the 10 leading causes of death in the world are (in order): ischaemic heart disease; stroke; COPD; lower respiratory tract infection; neonatal conditions; trachea, bronchus and lung cancers; Alzheimer disease and dementia; diarrhoeal diseases; diabetes mellitus; and kidney disease (WHO: Fact sheet: The top 10 causes of death; December 2020).

These diseases and the common causes of mortality (see TABLE 6.1) act as a focus for our energies in addressing preventive programs.

Table 6.1 Common causes of deaths in Australia in 2019 (in rank order)

1 Ischaemic heart disease
2 Dementia/Alzheimer disease
3 Cerebrovascular disease
4 Tracheal, bronchial and lung cancer
5 Chronic lower respiratory disease
6 Colorectal cancer
7 Diabetes mellitus
8 Blood and lymph cancer
9 Influenza and pneumonia
10 Diseases of the urinary system
11 Heart failure
12 Prostate cancer
13 Suicide, intentional self-harm
*Cancer is the no. 1 cause if grouped together

Source: Australia's leading causes of death. Causes of death, Australia, 2019. Australian Bureau of Statistics. Available from: https://www.abs.gov.au/statistics/health/causes-death/causes-death-australia/latest-release#australia-s-leading-causes-of-death-2019

It is worth focusing on the changes in disease indices during the past generation in order to evaluate the effect of preventive and health promotion programs during this period (see TABLE 6.2).[3] The messages are to harness and promote with renewed vigour those strategies that are working, such as prevention of death from coronary artery disease and motor vehicle accidents, and to re-evaluate those important areas, such as Aboriginal mortality, HIV infection, cancer, suicide and asthma, which are bad news! On the positive side, the overall life expectancy in Australia has risen to 82.5 years.

Table 6.2 Major changes in public health in Australia, with trends in the [4]

two decades up to 2012[4]

Improvements	Deterioration
Overall mortality	Alcohol-related diseases
Heart disease	Drug abuse
Stroke	Dementia
Smoking	Depression, mental health
Road safety	Health inequalities
Diseases controlled by immunisation	Obesity
Dental health	Diabetes
Cancer, overall especially	Arthritis/musculoskeletal problems
• cervix	Fall injuries
• stomach	Environmental pollution problems
• breast	
• testis	
• colorectal	
Pregnancy complications	
Congenital abnormalities	
HIV/AIDS	

A global strategy for good health

The World Health Organization (WHO) defines good health as ‘a state of dynamic harmony between the body, mind and spirit of a person and the social and cultural influences which make up his or her environment’.

A considerable amount of epidemiological information has emerged to support what GPs have known for a long time—that a commonsense, healthy lifestyle not only promotes good health but also reduces the risk of the main causes of mortality and morbidity in this country, including cardiovascular disease and cancer.

The common theme for virtually all disease is to follow the nutrition and lifestyle guidelines presented in CHAPTER 5 .

Behaviour modification

Lifestyle habits that have developed over many years can be very difficult to change even when the individual is well motivated to change. A variety of instructional, motivational and behavioural techniques can be used to initiate a lifestyle change program; GPs should be aware of these and use the resources of a multidisciplinary team to give support to motivated people who as a rule find behaviour modification difficult.

Vascular disease

Risk factors for vascular disease (atherosclerosis) are:

- hypertension
- smoking
- high cholesterol
- diabetes
- obesity
- sedentary lifestyle
- stress
- alcohol excess
- poor diet
- family history

The guidelines for good health given in CHAPTER 5 , if followed, will help prevent the development of cardiovascular and cerebrovascular disease.

It is worth noting that the death rate from coronary heart disease is about 70% higher for smokers than for non-smokers, and for very heavy smokers the risk is almost 200% higher. It has been shown that the incidence of heart disease falls in those who have ceased smoking.

GPs can estimate the absolute 5-year risks of cardiovascular events in their patients by referring to the Absolute CVD risk clinical guidelines set out by the Heart Foundation (www.heartfoundation.org.au); see CHAPTER 75 .

The parameters used are:

- gender and age
- smoking status
- diabetes status

- blood pressure
- total cholesterol/HDL ratio

Malignant disease

Primary prevention of cancer is an important objective and there is a need to focus on this vital factor as much as on secondary prevention.

Updated important facts about cancer in Australia* (current and immediate future)

- It continues to be more common in men than women overall; in 2019 more than half (54%) of all diagnosed cases were expected to be in men.
- Breast cancer (in women) is expected to be the most common type of cancer diagnosed, followed by colorectal cancer and prostate cancer.*
- The death rate from all cancers has fallen from 209 deaths per 100 000 people in 1982 to an estimated 161 per 100 000.
- Survival rates from all cancers have improved substantially, with 5-year survival increasing from 48% in 1984–1988 to 69% in 2011–2015.
- Survival varied by cancer type, with the largest survival improvements seen in prostate cancer, non-Hodgkin lymphoma, kidney cancer and multiple myeloma.
- Pancreatic cancer and lung cancer showed only small improvements, while bladder cancer and cancer of the larynx actually had lower survival rates.
- Lung cancer is likely to be the leading cause of cancer death, followed by colorectal cancer, prostate cancer, breast cancer in women and pancreatic cancer.

**Excluding basal and squamous cell carcinoma*

Source: Cancer Council of Australia (www.cancer.org.au/cancer-information); and Australian Institute of Health and Welfare (AIHW), Cancer in Australia, Cancer series no 101. Cat no. CAN 100. 2019. Canberra.

That environmental factors are involved in the aetiology of colorectal cancer and other cancers is indicated by wide variations in incidence between different countries.

Suspicion falls on diet and there is epidemiological evidence implicating diets high in animal fats and low in insoluble fibre, fruits and vegetables, and also high alcohol consumption. It is noted that there are higher incidence rates in people migrating from low- to high-risk countries, such as Japanese to Hawaii and Greeks and Italians to Australia.[5]

Studies in the US indicate that at least 35% of all cancer deaths are related to diet. Obese

individuals have an increased risk of colon, breast and uterine cancers. High-fat diets are a risk factor for prostate, breast and colon cancers. Salt-cured, smoked and nitrate-cured foods increase the risk of upper GIT cancers. Foods rich in vitamin A and folate (dark green and deep yellow vegetables and fruits) and vitamin C and cruciferous vegetables (cabbage, Brussels sprouts, broccoli and cauliflower) are all considered to have protective effects for various cancers.[6,7] Phytochemicals (plant chemicals) exist in these foods and in other vegetables and fruit that have a cancer-protective effect.[8]

Overall, diet, smoking, alcohol and occupational exposures (5%) appear to account for over 73% of all cancer mortality.[7]

Doll and Peto[9] considered that environmental factors were responsible for 80–90% of cancers and estimated that diet was a major factor in the cause of cancer in 40% of men and 60% of women.

The role of immunity in cancer

The development of a number of cancers appears to be related to a depression of the individual's immune system, particularly in relation to cellular immunity, in a similar way (albeit on a different scale) to the effect of HIV infection. Studies have shown that the immune system is adversely influenced by:[10]

- stress, especially bereavement
- depression
- ageing
- drugs
- pollutants
- cigarette smoke
- inappropriate diet
- alcohol
- radiation

On the other hand, a protective effect on the immune system may be provided by:

- food antioxidants (see TABLE 5.4 in CHAPTER 5)
- tranquillity
- meditation

In some instances malignancies appear to undergo unpredictable remissions with patients following an optimal diet, taking antioxidants, changing their lifestyle and practising meditation. However, an Australian study indicated that the enthusiasm for the value of antioxidants may be unjustified.[11]

Diet certainly appears to be a most important factor in the primary prevention of disease. If immune-deficient diseases can respond in such a way, imagine what a powerful primary preventive force such a lifestyle represents for all disease.

Asthma and other respiratory diseases

The death rate and morbidity rate for asthma and other respiratory diseases is unacceptable and much of it can be prevented.[12] A report on the cost of asthma claimed that there is evidence that a significant proportion of diagnosed asthmatics are currently receiving treatment that does not provide the best possible control of the disease.[12]

Prevention means being better informed and treating such an 'irritable' disease as bronchial asthma aggressively. It means focusing on better assessment and monitoring (e.g. home use of the mini peak flow meter), better delivery of medication to the airways (e.g. use of spacers attached to inhalers and/or use of pumps and nebulisers) and appropriate management of the cause (inflammation of the bronchial tree) by the use of inhaled corticosteroids or sodium cromoglycate as the first-line treatment for significant asthma. An appropriate strategy is to follow the six-step asthma management plan (see TABLE 6.3) of the National Asthma Campaign.

Table 6.3 The six-step asthma management plan

1. Establish the severity of the asthma.
2. Achieve best lung function.
3. Maintain best lung function—identify and avoid trigger factors.
4. Maintain best lung function—follow an optimal medication program.
5. Develop a written, accessible action plan.
6. Educate and review regularly.

Source: National Asthma Campaign: Australia, 2008

The protective effect for asthma and COPD of vitamin C, fish oils, a low-salt diet and other natural antioxidants is highlighted by Sridhar.[13]

Periodic health examination

Since 86% of the population visit a GP at some stage of the year,[3] and these people visit about five times each year (on average), GPs are in an ideal position to develop strategies for a periodic health examination. An emphasis should be placed on the history in addition to the physical examination and related basic investigations.

As for any smooth-running quality professional program, it is important to be organised with prepared practice staff, checklists and record systems. The Royal Australian College of General Practitioners (RACGP) has developed a College Record System, which has several leaflets covering all approaches to the patient 'check-up'.[14]

The following guidelines for the periodic health examination are adapted from those recommended by the Preventive and Community Medicine Committee of the RACGP.[14] This represents appropriate screening at the front line of primary health care.

Aims of screening

In practice, screening is not only to detect disease in asymptomatic people at its earliest stage in order to classify them as likely or unlikely to have a disease, but also to find individuals at risk or those with established disease who are not receiving adequate care. There are three levels at which screening practice can be applied in general practice:

1. 'well' individuals with risk factors that predispose to disease (e.g. obesity, uncomplicated essential hypertension, hyperlipidaemia)
2. asymptomatic individuals with signs of early disease or illness (e.g. developmental dysplasia of the hip, ectopic testis, glaucoma, bacteriuria of pregnancy, carcinoma in situ of cervix)
3. symptomatic individuals whose irreversible abnormalities are unreported but the effects can be controlled or assisted (e.g. visual defects, deafness, mental handicap)

The history[14]

An appropriate history will allow the recognition of certain risk factors that may foreshadow future disease. Though established patients will have a previously acquired database, their history should be reviewed and updated. It is recommended that the following items be included in history taking in the appropriate age groups.

Family history. In particular, cardiovascular disease, some cancers (breast, bowel, melanoma with dysplastic naevi), diabetes, asthma, genetic disorders and bowel disease will alert the doctor to specific risk factors (and psychological factors) for these patients.

Suicide and accidents. Consider the risk factors predisposing to suicide and accidents, which are the major preventable causes of death in children and young adults.

Substance abuse. Tobacco and alcohol are the major causes of preventable death in adults, although other drugs contribute to a lesser extent. Counselling by GPs, about smoking in

particular, has been shown to be effective.

Exercise and nutrition. These factors have a role to play in preventing cardiovascular disease and to a lesser extent in blood pressure control, cancer, diabetes and constipation. They have an even greater role to play in improving general well-being and preventing morbidity.

Occupational health hazards. Consider these in working adults, as occupational health hazards can significantly contribute to morbidity and mortality (e.g. exposure to toxic substances, unsafe work practices). Specific examples include:

- coal miners—pneumoconiosis
- gold, copper and tin miners—silicosis
- asbestos workers and builders—asbestosis, mesothelioma
- veterinarians, farmers, abattoir workers—zoonoses
- aniline dye workers—bladder cancer
- health care providers—hepatitis B

Physical functioning, home conditions and social supports. Consider these in elderly people, as physical function and social supports are of crucial importance in determining whether they can care for themselves—intervention can prevent accidents and death.

Sexuality/contraception. Sexually transmitted infections are all preventable, as are unwanted pregnancies. Opportunities should be sought to ask young people, in particular, about their sexuality, and to counsel them. The question ‘Do you have any concerns about sex?’ is very useful in this context.

Osteoporosis. Osteoporosis affects nearly a third of all postmenopausal women, most of whom suffer osteoporotic fractures. Fractures of the femoral neck have a particularly poor prognosis, with up to a third of these women dying within 6 months, and many more requiring continuing nursing home care. Bone loss accelerates at the time of the menopause, and can be reduced by hormone replacement therapy.

Women at risk of osteoporosis are short, slim, Caucasian; they drink coffee and alcohol, smoke, eat a high-protein and high-salt diet, and don’t exercise.

Masquerades in general practice. It is worth considering the ‘masquerades’ (see CHAPTER 9 , TABLES 9.4 and 9.5), which may present as undifferentiated illness, as a means of following the important medical principle of early detection of disease: engendering a certain awareness.

Primary masquerades to consider are:

- depression
- diabetes mellitus

- drug problems
- anaemia
- thyroid disorders, especially hypothyroidism
- urinary tract infection
- vertebral (spinal) dysfunction

Hypothyroidism has been estimated to exist in up to 15% of women aged 60 and above, and searching for clues may elicit subtle symptoms and signs previously attributed to ageing.

Relationships and psychosocial health. Consider the mental health of patients, particularly the elderly, by enquiring about how they are coping with life, how they are coping financially, about their peace of mind and how things are at home. Focus on the quality of their close relationships (e.g. husband–wife, father–son, mother–daughter, employer–employee). Enquire about losses in their life, especially family bereavements.

Screening for children[14]

Childhood health record books provide an excellent opportunity for communication between different health care givers; parents should be provided with the record books and encouraged to bring them to every visit. Various recommendations for screening are made under the following headings.

Height/weight/head circumference. Record length/height, weight and head circumference at regular intervals. Head circumference can be recorded until 2 years and should increase by 1 cm per month in the first 3 months, then 0.5 cm per month from 3–6 months. It provides further data about a child's growth. It is important to check the fontanelles. Calculate the BMI from 2 years. The adequacy of a child's growth cannot be assessed on one measurement and serial recordings on growth charts are recommended.

Hips. Screen for congenital dislocation at birth, 6–8 weeks, 6–9 months and 12–24 months (see CHAPTER 54).

The flexed hips are abducted, checking for movement and a 'clunk' of the femoral head forwards (the test is most likely to be positive at 3–6 weeks and usually negative after 8 weeks). Shortening or limited abduction is also abnormal. Ultrasound examination is more sensitive than the clinical examination, especially up to 3–4 months. Observe gait when starting to walk.

Strabismus. Strabismus should be sought in all infants and toddlers by occlusion testing (not very sensitive), examining light reflexes and questioning parents, which must be taken very seriously. Amblyopia can be prevented by early recognition and treatment of strabismus by occlusion and surgery. Early referral is essential.

Visual acuity. At birth and 2 months, eyes should be inspected and examined with an

ophthalmoscope with a 3+ lens at a distance of 20–30 cm to detect cataracts and red reflexes. At 9 months gross vision should be determined by assessing ability to see common objects. Visual acuity should be formally assessed at school entry using Sheridan Gardiner charts.

Hearing. Hearing should be tested by distraction at 9 months or earlier; also by pure tone audiometry at 1000 and 4000 hertz when a child is 4 years (preschool entry) and 12 years.

Note: Formal audiological evaluation should be carried out at any time if there is clinical suspicion or parental concern. No simple screening test is very reliable for sensorineural or conductive deafness.

Testes. Screen at birth, and 6–8 weeks, 6–9 months and 3 years for absence or maldescent. Those who have been treated for maldescent have a higher risk of neoplastic development in adolescence.

Oral health/dental assessment/fluoride. Advise daily fluoride drops or tablets, if water supply is not fluoridated. Children's teeth should be checked regularly, particularly if a school dental service is not available. Advice should be given on sugar consumption, especially night-time bottles, and tooth cleaning with fluoride toothpaste to prevent plaque.

Scoliosis. Screening of females by the forward flexion test, which is carried out around 12 years of age, is of questionable value because of poor sensitivity and specificity.

Congenital heart disease. The heart should be auscultated at birth, in the first few days, at 6–8 weeks and on school entry.

Femoral pulses. Testing for absence of femoral pulses or delay between brachial/femoral pulses at birth and 8 weeks will exclude coarctation of the aorta. Refer the child immediately if concerned.

Speech and language. A child's speech should be intelligible to strangers by 3 years. It is related to hearing.

General development

Apart from speech and language, check social and emotional development, toilet habits, behaviour and mood.

This includes 'parents' evaluation of developmental status' (PEDS) (see: www.pedstest.com; www.rch.org.au/ccch).

For general checklists, see www.cdc.gov/actearly. Be alert to the 'red flags' of autism spectrum disorder (see CHAPTER 87).

Screening in the elderly

Refer to CHAPTER 125 .

Screening for adults[14]

The following recommendations apply for adults.

Weight. Weight should be recorded at least every few years. Obesity is a major reversible health risk for adults, contributing to many diseases (e.g. heart disease, diabetes, arthritis). Body mass index (BMI) should ideally be between 20 and 25.

$$\text{BMI} = \text{weight (kg)} \div \text{height (m}^2\text{)}$$

Abdominal obesity is a major risk factor for adults. The waist:hip circumference ratio is regarded as a useful predictor of cardiac disease. Recommended waist:hip ratios are:

- males <0.9
- females <0.8

Blood pressure. Blood pressure should be recorded at least every 1–2 years on all people 16 years and over. There is no dispute that control of blood pressure results in reduced mortality from cerebrovascular accidents and, to a lesser extent, heart disease, kidney failure and retinopathy.

Cholesterol. All adults aged 45 and over should have a 5-yearly estimation of serum cholesterol. Total cholesterol is adequate for screening purposes. HDL levels give additional information. The National Heart Foundation recommends keeping cholesterol levels below 4.0 mmol/L. For most, dietary modification is sufficient to achieve these levels; some may require drug treatment.

Fasting blood glucose. Screen every 3 years for all patients >40 years of age.

Cervical cancer. From age 25 (to age 74), women who have ever been sexually active should commence cervical screening with a HPV test 2 years after their last Pap test. If negative, this can be performed every 5 years. If positive, cervical cytology will be checked with the sample and follow-up is determined according to the national guidelines. Women aged 70–74 should be offered a final ‘exit test’ and can cease screening if negative.

Breast cancer. Mammography should be performed at least every 2 years on women aged 50–74 years. It is not useful for screening prior to age 40 years due to difficulty in discriminating malignant lesions from dense tissue. Women aged 40–49 years may also choose to have a mammogram.[14] Mammography must not be used alone to exclude cancer if a lump is palpable. Such lesions require a complete appraisal since, even in the best hands, mammography still has a false-negative rate of at least 10%. Genetic testing should be considered in those at risk.

Colorectal cancer (CRC). A history should be taken, with specific enquiry as to family history of adenomas or colorectal cancer, past history of inflammatory bowel disease and rectal bleeding. Rectal examination should be performed as part of an examination. Immunochemical faecal occult blood testing (FOBT) every 2 years is now recommended for screening for people over 50 years (continuing to 74 years) without symptoms and with average or slightly above average

risk.

Should a positive history be elicited, then the following are recommended:

- past history of large bowel cancer or colonic adenomas—colonoscopy
- past or present history of ulcerative colitis—colonoscopy with biopsies
- familial polyposis, Gardner syndrome—sigmoidoscopy or colonoscopy

Prophylactic colectomy needs consideration in some individuals.

Apart from FOBT screening, the National Health and Medical Research Council (NHMRC) currently recommends:

- for people at moderate risk (family history category 2)—2-yearly FOBT for people age 40–49, then colonoscopy every 5 years for people age 50–74
- for high risk (family history category 3)—2-yearly FOBT for people age 35–44, then colonoscopy every 5 years for people age 45–74

Refer to the RACGP *Guidelines for Preventive Activities in General Practice*[14] for further information. Genetic testing should be considered in those at risk.

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Prostate cancer. Screening is controversial. The RACGP guidelines do not recommend routine screening with DRE, PSA or transabdominal ultrasound. Patients should make their own decision after being fully informed of the potential benefits, risks and uncertainties of testing. Doctors should also use their clinical judgment for their individual male patients.

Skin cancer. All patients should be informed regularly about the need for protection of the skin and eyes from ultraviolet (UV) radiation, using hats, clothing, sunglasses and sunscreens, and avoiding exposure during peak UV periods (10 am to 3 pm).

Skin cancer, which is increasing in incidence, is common in Australia, particularly in more northern areas. Squamous cell carcinoma, and melanoma in particular, may be lethal. Detection and treatment of early lesions prevents mortality and morbidity. Prevention of skin cancer by reduction of sun exposure should be taught to all patients.

Oral hygiene/cancer. Patients should be counselled about cessation of smoking and alcohol consumption, and dental hygiene should be taught. The oral cavity should be inspected annually in patients over the age of 40 years.

Although oral cancer has a relatively low incidence, premalignant lesions may be detected by inspection of the oral cavity. Its incidence is highest in elderly people with a history of heavy smoking or drinking. Poor dental hygiene may result in poor nutrition, particularly among the elderly.

Cancer screening in summary:[14]

- Screen for breast, cervical and colorectal cancer.
- Routine population-based screening is at this stage of evidence not recommended for lung, melanoma, ovarian, prostate and testicular cancers.

Immunisation

Children and adolescents should be immunised according to the current NHMRC recommended standard vaccination schedule (see: www.health.gov.au/health-topics/immunisation).

The NHMRC advises administering intramuscular and subcutaneous vaccines to the anterolateral thighs for children under 12 months, and to the deltoid region in older children and adults. Do not postpone immunisation for minor illnesses such as mild URTI.

All adults should receive an adult diphtheria and tetanus (ADT) booster every 10 years.

All women of child-bearing years should have their rubella antibody status reviewed. During pregnancy, it is recommended to have the seasonal influenza vaccination any time in the pregnancy and a pertussis booster in the final trimester.

Table 6.4 National Immunisation Program Schedule[15] (as from 2018)

Age	Immunisation
Birth	Hepatitis B
2 months	DTP, Hib, hepatitis B, polio, pneumococcus, rotavirus
4 months	DTP, Hib, hepatitis B, polio, pneumococcus, rotavirus
6 months	DTP, polio, Hib and hepatitis B
12 months	Measles/mumps/rubella (MMR), pneumococcus, meningococcal ACWY
18 months	DTP, varicella, measles, mumps, rubella (MMR), Hib
4 years	DTP, polio
School programs	HPV, DTP (adult), meningococcal ACWY, hepatitis B (if no first course)

12–16 years (contact state authorities)	
Pregnant women	Influenza, pertussis
From 65 years	Influenza (yearly), pneumococcal polysaccharide (23vPPV)
70–79 years	Herpes zoster

Hib = Haemophilas influenzae type b
HPV = human papilloma virus
DTP (triple antigen) = diphtheria, tetanus, pertussis

Note: Aboriginal and Torres Strait Islanders (ATSIP) and other at-risk groups have further recommended immunisations, including influenza, pneumococcus and hepatitis A.

Adverse effects of vaccination

Common adverse effects are irritability, malaise, fever and a local reaction to the injection. There is a very small risk of anaphylaxis. It is advisable to wait 15–30 minutes for observation after vaccination. Paracetamol is recommended for fever and local pain; however, routine use at the time of or immediately after vaccination is not recommended.[15]

Fever and illness. Children with minor illness (providing the temperature is <38.0°C) may be vaccinated safely. Otherwise it should be delayed. A simple past febrile convulsion or pre-existing neurological disease is not a contraindication to pertussis vaccination. Absolute contraindications include encephalopathy within 7 days of a previous DTP or an immediate severe or anaphylactic reaction to DTP.

The case for vaccines

Influenza. The population needs to be immunised against this ever severe and constantly mutating endemic infection. Influenza immunisation is recommended on an annual basis for persons of all ages with chronic debilitating diseases, especially chronic cardiac, pulmonary, kidney and metabolic diseases, persons over 65 years of age, all Aboriginal and Torres Strait Islander people over 12 months of age, especially adults over 50 years of age, and persons receiving immunosuppressant therapy. Health care personnel may wish to consider it for their own use.

Pneumococcal disease. This should be considered for the same risk groups as influenza vaccine. Those at higher risk of fatal pneumococcal infection (e.g. post-splenectomy or Hodgkin lymphoma) should receive a booster every 5 years. This is currently provided for all children.

Hepatitis A. Immunisation is recommended for:

- certain occupational groups at risk (e.g. health workers, child care workers, sewage workers)
- non-immune homosexual men

- those with chronic liver disease
- recipients of blood products
- travellers to hepatitis A-endemic areas

FIGURE 6.2 Immunisation of an older child: important continuing preventive care

Hepatitis B. Immunisation is recommended routinely for all children at birth, 2 months, 4 months and at either 6 or 12 months, and for individuals of all ages who, through work or lifestyle, may be exposed to hepatitis B and have been shown to be susceptible. Such groups would include health care personnel, personnel and residents of institutions, prisoners and prison staff, persons with frequent and/or close contact with high-risk groups, and persons at increased risk due to their sexual practices. Household contacts of any of the above groups should be considered for immunisation. Booster doses are not recommended for immunocompetent people but are recommended for immunosuppressed individuals. Universal vaccination represents a preventive step against hepatocellular cancer.

Haemophilus influenzae type b. Hib immunisation is recommended for all children, especially those in child care. It is ideal to achieve immunity by the age of 18 months and preferably commencing at 2 months. Risk factors for Hib disease include day care attendance, presence of ill siblings under 6 years of age in the home and household crowding.

Q fever. People at reasonable risk from Q fever, particularly abattoir workers, should be given this vaccine, which is virtually 100% effective.

Measles-mumps-rubella. Both females and males should be immunised against measles, mumps and rubella at the age of 12 months and 18 months using the trivalent vaccine. All non-immune women who are postpartum or of child-bearing age should be immunised.

Varicella vaccine. This is available and one dose is given at 18 months. Those over 12 years have a course of two injections.

Meningococcal vaccine. Meningococcal disease is caused by *Neisseria meningitides*, which has 13 serogroups of which A, B and C account for over 90% of isolated cases, with serogroup B responsible for most cases. A vaccine against serogroup B is available but is not yet part of the recommended schedule, as evaluation studies are being assessed. The main vaccine that is available is a quadrivalent polysaccharide vaccine against serogroups A, C, Y and W125 for use in individuals over 2 years as a single injection. Universal prevention by immunisation remains unsatisfactory. It is most useful when a community outbreak due to proven serogroup C occurs.

Rotavirus. A course of two (usually) or three oral live attenuated rotavirus vaccines is given to children to cover a common cause of childhood gastroenteritis. Inform parents of the risk of intussusception with the first dose.

Human papillomavirus. A course of two injections is provided by schools to all pupils aged 11–12 years.

Neonatal screening

Blood from a heel prick at 48–72 hours following birth checks 25 conditions including cystic fibrosis, phenylketonuria and hypothyroidism.

Genetic screening

Genetic screening programs are presented in CHAPTER 23 .

Key checkpoints

- Leading risk factors contributing to the burden of disease in Australia are tobacco, hypertension, excess weight/obesity, physical inactivity, hypercholesterolaemia, alcohol, and low fruit and vegetable consumption.
- Studies in the US conclude that a healthy lifestyle consists of not smoking, BMI <30 kg/m^2, five servings of fruit and vegetables a day and about 150 minutes of exercise each week.

Resource

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7 Research and evidence-based medicine

Not the possession of truth, but the effort of struggling to attain it brings joy to the researcher.

GOTTHOLD LASSING (1729–1781)

Effective research is the trademark of the medical profession. When confronted with the great responsibility of understanding and treating human beings, we need as much scientific evidence as possible to render our decision making valid, credible and justifiable.

Research can be defined as 'a systematic method in which the truth of evidence is based on observing and testing the soundness of conclusions according to consistent rules'[1] or, to put it more simply, 'research is organised curiosity',[2] the end point being new and improved knowledge.

In the medical context the term 'research' tends to conjecture bench-type laboratory research. However, the discipline of general practice provides a fertile research area in which to evaluate the morbidity patterns and the nature of common problems in addition to the processes specific to primary health care.

There has been an excellent tradition of research conducted by GPs. Tim Murrell in his paper 'Nineteenth century masters of general practice'[3] describes the contributions of Edward Jenner, Caleb Parry, John Snow, Robert Koch and James MacKenzie, and notes that 'among the characteristics they shared was their capacity to observe and record natural phenomena, breaking new frontiers of discovery in medicine using an ecological paradigm'.

This tradition was carried into the 20th century by Australian GPs such as Clifford Jungfer, Alan Chancellor, Charles Bridges-Webb, Kevin Cullen and Trevor Beard,[4] and now the research activities of the new generation of GPs, academic-based or practice-based, have been taken to a higher level with the development of evidence-based medicine (EBM).

Based on the work of the Cochrane Collaboration and the initiatives of Chris Silagy, Paul Glasziou and Chris Del Mar in particular, research has moved from the relatively 'pure' hospital environment to 'real world' scenarios which better reflect the circumstances of patients living in the community. Plenty of interventions which seem to work well in sponsored clinical trials are

less impressive when measured in general practice. Some other interventions such as lifestyle management turn out to be surprisingly effective once they are accurately measured.

The focus of EBM has been to improve health care and health economics. Its development has gone hand in hand with improved information technology. EBM is inextricably linked to research.

The aim of this chapter is to present a brief overview of research and EBM and, in particular, to encourage GPs, either singly or collectively, to undertake research—simple or sophisticated—and also to publish their work. The benefits of such are well outlined in John Howie's classic text *Research in General Practice*.[5]

Why do research?

The basic objective of research is to acquire new knowledge and to justify decision making in medical practice. Research provides a basis for the acquisition of many skills, particularly those of critical thinking and scientific methodology. The discipline of general practice is special to us with its core content of continuing, comprehensive, community-based primary care, family care, domiciliary care, whole-person care and preventive care. To achieve credibility and parity with our specialist colleagues, we need to research this area with appropriate methodology and to define the discipline clearly. There is no area of medicine that involves such a diverse range and quantity of decisions each day as general practice, and therefore patient management needs as much evidence-based rigour as possible.

Our own patch, be it an isolated rural practice or an industrial suburban practice, has its own micro-epidemiological fascination. Thus, it provides a unique opportunity to find answers to questions and make observations about that particular community.

Increasingly, GPs are expected to be able to sift through mountains of information in order to reliably offer advice about how the evidence applies to the individual sitting in front of them. Much of the information available to patients and doctors is either dubious opinion or skewed towards the interests of an entity providing the intervention, rather than to the patient. An understanding of how research is produced provides a basis for the ability to critically appraise medical evidence.

There are also personal reasons to undertake research. The process assists professional development, encourages clear and critical thinking, improves knowledge and offers the satisfaction of developing new skills and opening horizons.

The author undertook many small studies on common, everyday problems during 10 years in country practice to determine the most effective treatments for which no or minimal evidence in the literature could be found. Many of these recommendations—for problems such as tennis elbow, cold sores, aphthous ulcers, ingrown toenails, hiccoughs, back pain, nightmares, temporomandibular dysfunction and warts—appear in this text. Although the numbers were relatively small, it was useful to compare treatments for about 10 or 20 cases to test hypotheses and allow trends to emerge. Subsequent results from a large controlled trial would, of course,

take precedence over these recommendations if they differed. However, the exercise, albeit limited, added immense interest to one's practice, which at times can be tedious without such scholarly challenges.

An important reason to undertake research is to conform with quality assurance processes that are now being expected of practitioners. The significant processes evaluating our accountability for quality control include audits of our own records, studies of critical incidents and morbidity studies.

Who should do research?

Any GP searching for answers to questions and who has the opportunity should undertake research. Research is largely opportunistic; for some it may be an impulsive reaction to a fascinating observation, for others a carefully conceived plan.

The research question should be answerable, and that answer, whether positive or negative, should be useful to patients.

Research can be collaborative, and in fact a group practice is an excellent way to get started. Ideally, start with the 'apprentice' model, where one's first attempt is a collaboration alongside a more experienced researcher.

Many GPs who have started 'small' have progressed to great heights of research activity, especially if they enjoy analysing the raw data once it has been produced. In the process of posing questions and eventually finding the answers, they frequently refer to the experience as 'good fun'.

The Royal Australian College of General Practitioners (RACGP) promotes and supports general practice research (visit: www.racgp.org.au/support/research or email: research@racgp.org.au).

Asking questions

We often ask questions during the course of managing patients and such questions can form the basis of a research project, however simple.

Typical questions might be:

- Is suicide or attempted suicide in adolescent males precipitated by sexual-orientation issues?
- Is recurrent migraine caused by cervical dysfunction?
- Should we use antibiotics to treat otitis media in children presenting in general practice?
- Does the distribution of leaflets by the receptionist in the waiting room lead to increased immunisation rates or cervical smears?
- Are my patients satisfied with the services they receive?

- Does the provision of patient information leaflets for the management of hypertension lead to better adherence?

Research on what?

General practice has its own unique characteristics including illness content, processes, epidemiology, health services, quality assurance and doctor–patient relationships. The special contact with patients provides opportunities to evaluate their perspectives on health service delivery, psychosocial issues and communication skills. The old saying ‘dig where you are’ is relevant to all of us. GPs invariably develop their own special interests and this is a logical area in which to conduct research. Conducting a morbidity and prescribing survey in a practice is a simple and fascinating study. If the results are added to a wider study, invaluable information about the nature of general practice is obtained.[5,6]

The World Organization of National Colleges and Assemblies of General Practice (WONCA) produced the first *International Classification of Primary Care* (ICPC) in 1987, since ratified by the WHO. This classification of presenting symptoms, diagnoses and treatments has greatly assisted the process of morbidity studies.[7]

Research in general practice obviously covers many clinical areas studied by other groups but we may ask different types of questions, study different populations and use different methodologies, especially qualitative methods.

It would be logical to conduct research on those common problems requiring continuing care by the GP. These include:

- alcohol problems
- anxiety and depression
- arthritis

- chronic back pain and neck pain
- cancer
- cardiovascular disorders
- diabetes
- epilepsy
- common acute infections
- migraine and other headache
- women’s health

Special opportunities, such as the observation that certain diseases or conditions are linked with specific circumstances, present frequently in primary care. An example is the observation that a group of farmers who presented to their rural practitioner over a period of time with lymphosarcoma were all exposed to a specific herbicide to control blackberry growth on their farms. This led to further, statewide investigations of this association, which indicated a significant link between the agent and the disease.

Research in primary care populations often overturns long-held beliefs based on specialist research in high-risk populations, which was wrongly assumed to apply to general practice. Examples include screening for prostate cancer using the PSA test and the use of antibiotics in otitis media.

Understanding terminology

Validity and reliability

- An ideal method of collecting research material is one that is valid.
- A valid method is one that measures what it claims to measure.
- A reliable method is one that produces repeatable results.

Validity refers to the 'true' answer, which must be relevant, complete and accurate. Three significant questions that evaluate validity are:[1]

- Is the study useful or is the result inconclusive?
- Do you accept the results of this study as applied to the source population?
- Do the results apply to the population in which you would be interested?

Internal validity refers to the adequacy of the study methods in reference to the study population, while *external validity* refers to the generalisability of the results to the general population (or, more specifically, to the patient sitting in front of you).

Reliability refers to the stability of question-and-answer response and is most successfully measured by testing and then retesting (repeatedly).

Sensitivity, specificity and predictive values

Sensitivity and specificity, which are integral to validity, are important considerations in decision making in medicine, particularly in choosing appropriate investigations for disease diagnoses. The method of calculation of sensitivity, specificity and predictive values is summarised in FIGURE 7.1 .

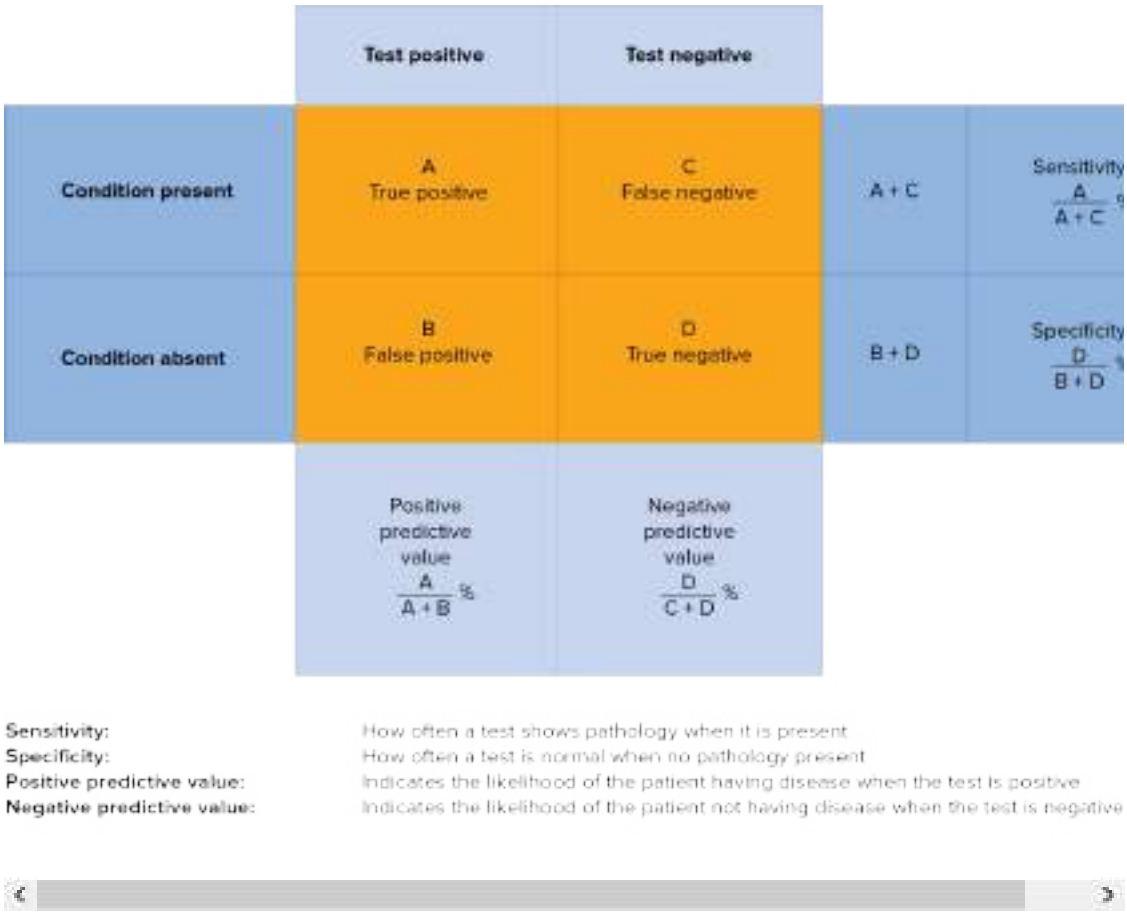

FIGURE 7.1 Definitions of sensitivity, specificity and predictive values

The *sensitivity* of a test depends on the proportion of people with the characteristic (disease) in whom the test is positive (i.e. percentage positive with disease). For example, if the sensitivity for a sign is 90%, then the sign will be detected in 90% of the people but not in 10%. The ultimate sensitive test is one that detects all true positive cases.

The *specificity* of a test depends on the proportion of people without the characteristic (disease) in whom the test is negative (i.e. percentage negative of healthy people). The ultimate specific test is one that detects all the truly negative (disease-free) cases. A *gold standard* test is one that is as close to 100% specificity and 100% sensitivity as possible.

Aide-mémoire tip

SPIN—Specific tests where a positive helps rule *IN* disease

SNOUT—Sensitive tests where a negative helps rule *OUT* disease

A clinical example of sensitivity and specificity is presented in TABLE 7.1 .

Table 7.1 The predictability of signs and symptoms for carpal tunnel syndrome[9]

	Sensitivity (%)	Specificity (%)
Paraesthesia	97	4
Waking at night	91	14
Anaesthesia	57	61
Phalen test	58	54
Tinel test	42	63
Two-point discrimination test	6	98

Predictive values express the proportion of positives that are ‘true positives’ and negatives that are ‘true negatives’. They are markedly affected by the underlying risk of the disease in the population being studied. Because community patients usually have considerably lower rates of disease than hospitalised patients, positive predictive values (PPVs) are generally lower in general practice for any given test or symptom. This reduces the usefulness of ordering the test or relying on the symptom for a diagnosis.

For example, the presence of haematuria in a general practice patient gives a PPV of less than 5% for carcinoma being the cause, but the PPV is about 50% in the inpatient hospital setting.

Incidence and prevalence

The meanings of these two terms are easily confused:

- *Incidence* refers to the number of new cases of a disease (or factor of interest) occurring in a defined population within a specified period of time.
- *Prevalence* refers to the total number of individuals who have the disease (or factor of interest) at a particular time in a population. The number is divided by the number of people in the population at that time.

  Examples: The prevalence of multiple sclerosis in temperate climates is 1 in 1000–2000 compared with 1 in 10 000 in the tropics. The incidence of multiple sclerosis in the Australian state of Victoria (population 5.8 million) is 8 per 100 000 per year.

Bias

This is any effect occurring during the research that tends to produce results that depart systematically from the true values. Varieties of bias include *measurement bias* (e.g. fault with a

sphygmomanometer recording blood pressure), *confounding bias* (e.g. investigating the association between stress and hypertension without considering the influence of alcohol), *selection bias* (e.g. using hospital outpatients in a community-based study) and *publication bias* (around half of all research is not published, which skews the pool of evidence available for reading and analysis).

Confounding

This is a situation in which a measure of the effect of exposure on risk is distorted by the association of exposure with other (known or unknown) factors that influence the outcome.[1] A confounder is a factor that distorts the apparent magnitude of the effect of a study on risk.

Chance

One must question the probability that the results favouring the experimental intervention could have occurred by chance; therefore, we resort to statistical help in the form of a probability statement or significance level.

How is the research undertaken?

'Getting started' can be quite difficult for the beginner. However, assistance is available from several sources, including individual GPs with research experience, university departments of general practice and the RACGP research committee. Seek out a suitable supervisor for the study. A chronological method follows.

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1. *The idea.* Start with an idea or question, which needs to be interesting, relevant, significant and answerable.[8] It may be appropriate to develop a hypothesis at this stage.
2. *Float the idea.* Next, discuss the idea with colleagues or an appropriate accessible authority.
3. *Do a literature search.* Review the literature: for example, a Medline search or check with a central research 'bank'. Undertake a critical review. Don't waste effort researching a question someone else has satisfactorily answered already.
4. *Prepare a plan.* This can be a short written plan outlining the methodology for the study.
5. *Evaluate the plan.* Then, contact a supervisor or appropriate authority to evaluate the study plan, which may be referred to a reference group or research committee.
6. *Methodology: develop a protocol.*
    - Prepare background, outline objectives and develop a hypothesis.
    - Select target population using clear criteria and appropriate numbers.
    - Design the research:

qualitative or quantitative?

questionnaire(s)

- Assess internal validity.
- Consider statistical implications early:

  number of patients

  method for data analysis

- Recruit subjects and assistants.
- Assess the timeframe.
- Assess the ethical considerations → ethics approval committee.

7. *Consider a preliminary pilot study and project timetable.*
8. *Seek funding.* Solicit advice from appropriate funding bodies.
9. *Conduct the study.*
10. *Analyse the data/statistics.*
11. *Undertake interpretation and conclusions.*
12. *Prepare for publication.*

Research design

Hypothesis development

The reasoning process of the researcher is based on the null hypothesis—the default assumption that an experimental group does not differ from a control 'normal' group in outcome. One question to consider is: 'What is the probability that results from the experimental intervention would have occurred by chance?'

Selecting a representative sample of appropriate size

Two basic components of subject selection are sample size and sample representativeness. The latter should be selected in a well-controlled manner.

A common question is: 'What is the ideal size of the sample?' There is no fixed answer but it must be adequate to produce statistically meaningful results, without being so large as to make the research impractical or unaffordable.

Recruitment of patients is a particular skill and often hard work, but it is easier if the researcher has a large pool of patients with whom he or she enjoys a good relationship. A useful rule is to aim to approach 3*n* patients if you wish to work with a sample size of *n*.

Some guidelines for choosing the sample size are:[9]

- the more the individuals in the population differ, the larger the required sample
- the more planned comparisons, the larger the size
- larger sample sizes allow detection of smaller differences

Types of research[1]

The two broad categories of research in general practice are qualitative research, which is based on observation and talking with people, and quantitative research, which is based on measurement and analysis of data collection.

Research can also be classified as primary research, which includes both qualitative and quantitative methods, and secondary research, which involves systematic reviews and meta-analysis.

Qualitative research[10]

This research is basically concerned with evaluating human behaviour from the subject's perspective. It is based on close observation and is expressed in a descriptive way. It addresses questions that begin 'Why?', 'How?', 'In what way does?', e.g. 'Why have so many people been dropping out of this fitness program?'

Common qualitative approaches

- Phenomenology
- Ethnography
- Grounded theory
- Biography (life story, narrative enquiry)
- Case study

The methods used are:

- interviews (open-ended, semi-structured)
- focus groups

- participation observation
- document analysis

Qualitative research is an excellent method for generating hypotheses, and can lead to quantitative research.

Phenomenology

The central focus of this philosophy/method is the lived experience of the world of everyday life. It describes events, situations, experiences and concepts. It provides:

- detailed descriptions of an experience or event as it is lived
- deeper understandings and sensitivities
- improved thoughtful provision of care

Examples:

- effects of Viagra (and other agents) on marital/sexual relationships
- experience of carers in Alzheimer disease
- effects of workplace bullying on absenteeism

Ethnography (*ethnos* = a nation)

This examines cultures, peoples and societies including subgroups, e.g. adolescents. It is the basis of anthropology. The investigator usually identifies a number of key witnesses (informants) and interviews them to clarify observations.

Grounded theory

This is the development of new theory through the collection and analysis of data. It seeks to identify the core social processes within a given context in order to build theory that is grounded in the reality of those being studied.

Quantitative research

Quantitative research is research based on the collection of data in numerical quantities and asks a specific narrow question, such as ‘What is the chance that . . .?’ or ‘What proportion of ...?’. It is concerned with hypothesis testing, reliability and validity, and is the cornerstone of epidemiology. It can be classified broadly as *observational*, which includes case control, cross-sectional and cohort studies, and *experimental*, which includes the classic controlled trial.[10]

- *Case control (or retrospective) study* is an observational study in which people with a disease

(cases) are compared with those without it (control group). It is often used to determine a statistical association between an exposure and an outcome, particularly a rare outcome.

  Examples: Patients with mesothelioma were investigated for exposure to asbestos or other agents; the mothers of children born with birth defects were investigated for an association with drug intake during pregnancy.

- *Cross-sectional or prevalence study* follows a correlation approach using existing databases. It is a survey of the frequency of disease, risk factors or other characteristics in a defined population at one particular time.

  Example: The prevalence of type 2 diabetes mellitus (diagnosed and undiagnosed) was investigated in an Aboriginal community living in a particular area of metropolitan Sydney.

- *Cohort (or prospective) study* is also referred to as 'follow-up'. The study follows a group (cohort) of individuals with a specified characteristic or disease over a period of time. Comparisons may be made with a control group.

  Example: 120 people with chronic sciatica were followed over 10 years to determine the outcome of their pain and neurological deficit. These were compared with a matched group who had undergone laminectomy.

- *Randomised controlled trial* (RCT) is an experimental study that tests for hypothesised outcomes. Participants in the trial are randomly allocated to either receive the specific intervention or not receive it (the control group). The objective is to establish a causal relationship between the intervention and the hypothesised outcome. The ideal scientific trial is a **double-blind trial** where neither staff nor the participating patient are aware whether the participant is in the intervention or control group. An RCT is the typical study when assessing the effect of a drug compared to a placebo.

Meta-analysis

Meta-analysis is the process that systematically assesses compatible randomised controlled trials by merging the data (usually from smaller and inconclusive trials) to draw a 'firmer' conclusion from larger numbers of subjects.

Evidence-based medicine

Evidence-based medicine (EBM) is a process of basing clinical practice on validated information. According to one of its modern architects, David Sackett, it is 'the explicit, judicious and conscientious use of current best available evidence in making decisions about the care of individual patients'.[11] According to Silagy and Haines, 'EBM is the integration of the best available scientific evidence with your clinical expertise and knowledge, your intuition, your wisdom'.[12]

The process of using EBM should be very comfortable for GPs because scientific methodology

and evidence is second nature to us and has been the basis of our clinical decision making prior to and subsequent to graduation.

The proposed five steps of applying EBM are similar to basic research methodology:[12]

1. Construct a clinical question or define the problem.
2. Search for the evidence.
3. Appraise the quality and relevance of the evidence for this particular circumstance.
4. Apply it to the care of an individual patient.
5. Evaluate how effective it is.

The statistical methodologies used in EBM cover the traditional research methods but there is an emphasis on the methods of risk reduction, absolute and relative risk reduction, and number needed to treat (NNT). These definitions are included in the glossary of terms later in this chapter.

GPs have a responsibility to their patients to be well versed with the best evidence when making decisions about management (see TABLE 7.2), whether it be for a minor surgical procedure, selection of drugs, selection of an investigation or referral to the most appropriate consultant. If the best evidence reveals that a certain practice we are using is of no value or is less efficacious than another method, then we should be prepared to change. On the other hand, if we find that a certain method works for us and there is no current evidence that it is inappropriate, or the evidence is equivocal, then there is no compelling reason to change.

Table 7.2 Levels of evidence

I	Evidence obtained from a systematic review of all relevant randomised trials.
II	Evidence obtained from at least one properly designed randomised controlled trial.
III	Evidence from well-controlled trials that are not randomised, or from well-designed cohort or case-control studies, or multiple time series (with and without the intervention).
IV	Opinions of respected authorities; opinion based on clinical experience; descriptive studies; or reports of expert committees.

Source: Modified from the NHMRC

GPs need a healthy scepticism about what is best evidence and claims for treatment in addition to the skill of critical appraisal of research/evidence. We tend to be impressed by the perception that

evidence is a numbers game. However, the great work of James Lind shows that facts do not necessarily involve large numbers.

For EBM to be accepted by GPs the information needs to be readily accessible, user friendly, significant, relevant and, perhaps, believable.

The strength of EBM is that it can provide the answers to very important everyday decisions, especially in screening and preventive medicine, where guidelines have fluctuated over the decades. The most recent RACGP guidelines for preventive activities in general practice (the red book)[13] highlight the value of current evidence (see: www.racgp.org.au/your-practice/guidelines).

GPs are currently faced with important decisions about the effectiveness of complementary therapies, which are very tempting to embrace or trial when searching for ways to manage difficult problems, such as chronic fatigue syndrome, fibromyalgia, chronic asthma, chronic pain syndromes and other difficult-to-treat diseases. We are hopeful that EBM can provide the answers to best practice in addition to evaluating individual therapies.

Remember, however, that Bayes' theorem tells us that a positive trial result means less if the pre-existing chances of the treatment working were low to begin with. In other words, 'extraordinary claims require extraordinary evidence'. Thus, it is poor practice to justify a new, unusual treatment by using the reasoning that 'no one knows, so it might be true', as if sceptics of that treatment need to produce proof it doesn't work before discrediting it. Treatments that are inherently very unlikely to work (such as homeopathic products or crystal healing) will remain very unlikely unless a large body of independent evidence suggests otherwise. For the vast majority of marketed 'unlikely' therapies, such evidence will never be produced because the null hypothesis is actually true.

While it is important to ensure one's medical practice is based on—or at least heavily informed by—evidence, there are concerns that EBM will be seized by bureaucrats to develop 'cook book' guidelines, Holy Writ or economic rationalisation. Others are concerned about the perceived lack of flexibility. An interesting critical review, especially affecting psychiatry, was presented by John Ellard in his paper 'What exactly is evidence-based medicine?'[14] He questioned the validity of the evidence underpinning EBM and the biases of both the proponents of 'science' and 'art' with the caution of Louis Pasteur: 'The greatest derangement of the mind is to believe in something because one wishes it to be so'.[15]

Glossary of terms[16,17]

Apart from the terms and definitions used in preceding pages, it is important to highlight the following terms used in EBM/research.

Absolute risk reduction (ARR) The absolute difference in event rates between two intervention or treatment groups. It gives an indication of the baseline risk and treatment effect. An *ARR* of 0 means no difference and thus the treatment has no

effect.

Example: The *ARR* for prophylactic ciprofloxacin in the case cited is 10 – 2 = 8 per 100 (0.08) or 8%.

Accessing the evidence

- The Cochrane Library is free to access in Australia. It includes:

  Database of Systematic Reviews

  Database of Abstracts of Reviews of Effectiveness

- www.thecochranelibrary.com

  or

  www.cochrane.org

- The TRIP database searches for all relevant research articles that answer your question (typed in plain English). See: www.tripdatabase.com

Analysis of variance This allows comparisons between the means of two samples of similar populations with a normal distribution. The contribution to variance for each variable can be determined and tested for statistical significance.

Clinical significance Whether the benefit to people receiving an intervention compared to the control group is great enough to warrant the intervention. It is based on measure of effect.

Confidence interval A measure of the imprecision of the data results. The statistically derived range of values around a trial result in which the probability is that the true result will be within the range.

A 95% (standard) confidence interval for a sample indicates that there is a 95% chance that the interval includes the true population proportion whose circumstances comply with the evidence.

Control event rate (CER) The percentage of subjects in the control group that experienced the event of interest.

Experimental event rate (EER) The percentage of subjects in the intervention group that experienced the event of interest.

Kappa Cohen's *kappa* measures the agreement between the evaluations of two raters when both are rating the same object. A value of 1 indicates perfect agreement. A value of 0 indicates that agreement is no better than chance. It is an appropriate statistic for tables that have the same categories in the columns as in the rows (e.g. when measuring agreement between two raters).

Number needed to treat (NNT) The number of people who must be treated over a given period of time with the experimental therapy (specific intervention) to achieve one good outcome or prevent one adverse outcome. This incorporates the duration of treatment. It is a measure of the absolute relative risk. Obviously the lower the *NNT,* the better the treatment. It is calculated as 100/*ARR* (%); that is, the reciprocal of the *ARR.*

Note: The *NNT* will be different for different patient populations depending on their baseline risk for developing the outcome of interest.

Odds ratio The probability of the occurrence of an event compared to its non-occurrence.

Publications

- Clinical evidence: BMJ Publishing Group, refer to: www.clinicalevidence.org
- Evidence-based medicine. BMJ Publishing Group

Probability (p) value A deceptively complex measure to understand. It is a statistical summary of the incompatibility between the observed data and what we would have expected to see if the treatment did not work in the slightest (i.e. if the 'null hypothesis' was true). The lower the p value, the less consistent it is that the experiment results can be explained by the null hypothesis. Confusingly, a p=0.05 does not at all equate to saying the treatment is therefore 95% likely to work, nor that the result had a 5% probability of occurring by chance. For the curious, a 2016 article offers a 14-page explanation.[18]

Relative risk (RR) The ratio of the risk of the outcome (e.g. disease or death) in the treatment/exposure group compared with the control/unexposed group. *RR* informs us how many times more likely an event is to occur in the treatment group compared with the control group.

Calculation: *RR = EER/CER*

RR = 1 means no difference, so treatment has no effect

RR >1 means the treatment increases the risk of disease/death

RR <1 means the treatment decreases the risk

Example: If the risk of death from people exposed to inhalation of anthrax spores is reduced from 10 in 100 cases to 2 in 100 cases with 60 days of prophylactic ciprofloxacin, the *RR* of death in this group is 0.20 or 20%.

Relative risk reduction (RRR) The proportional reduction of adverse events between the treatment/experimental and the control groups in a trial (i.e. *RRR* is the ratio of the absolute risk reduction to the risk of the outcome in the control

group). An alternative way to calculate the *RRR* is to subtract the *RR* from 1 (i.e. *RRR* = 1 − *RR*).

In the example it is: 1 − 0.2 = 0.80 or 80% or:

$$RRR = \frac{\text{APR}}{10} = \frac{8}{10} = 0.80 \text{ or } 80\%$$

RRR is probably the most commonly reported measure of treatment effects, particularly when trying to emphasise the usefulness of a treatment, but the *ARR* gives a more realistic picture.

Risk (R) The probability that an event (death or disease) will occur.

Statistical significance The likelihood of a difference between two groups being real, based on the possibility that the difference may have occurred by chance alone. It is based on confidence intervals and p values.

Type I error A type I error occurs when a study concludes that there is a difference between two groups when there is no difference.

Type II error A type II error occurs when a study concludes that no difference exists between groups when there is a true difference.

Critical appraisal of published research

The objective of critically appraising a paper is to determine whether the methods and results of the research have significant validity to produce useful information. The appraisal starts with a careful review of the abstract, which ideally should be presented in a structured format.

1. What were the objectives of the study?
2. What was the study design?
3. Were there any potential problems associated with the design?
4. Were all the patients who entered the study properly accounted for at its conclusion?
5. What were the important results?
6. How would you interpret and explain these results?

Many studies, quite legitimately, are sponsored by a company that may benefit from a positive finding, and/or are authored by researchers who have received industry funding. This does not

negate any finding, but does probably warrant a more thorough critical appraisal before applying that finding to your patients. A sponsored study that supports an intervention is more likely to be published and widely disseminated than an unsponsored study that does not support the intervention.

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8 Inspection as a clinical skill

More mistakes, many more, are made by not looking than by not knowing.

SIR WILLIAM JENNER (1815–1898)

GPs have an ideal opportunity to practise the art of careful observation and to notice all the signs and features characteristic of a patient from the time seen in the waiting room until the physical examination. We should be ‘like Sherlock Holmes’ in our analysis of the patient and accept the challenge of being astute diagnosticians and proud members of a noble profession.

It is important to stand back (so to speak) and look at the patient’s general appearance and demeanour. We should be assessing their mood and affect as much as their physical appearance. The first assessment to make is ‘Does the patient look sick?’.

First impressions

The first impression of the patient is always striking in some way and we should discipline ourselves to be as analytical as possible.

A rapid inspection from a trained observer may be all that is necessary to allow the observer to pinpoint specific disorders, such as anaemia, hyperthyroidism, jaundice, acromegaly and alcohol abuse. Such ‘spot’ diagnosis is not justifiable unless the original signs are supported by further examination, which must be comprehensive.

The following observations should therefore be made:

- facial characteristics
- abnormalities of the head and neck
- examination of the mouth
- character and distribution of hair
- examination of the skin (in general)

- height and weight
- posture and gait
- genitalia
- examination of extremities (hands, feet, nails, etc.)

Physiognomy

Physiognomy, from the Greek *physiognomonia*, meaning the judging of one's nature, is the art of judging character from the features of the face. It flourished during the Middle Ages. According to Addison,[1] 'everyone is in some degree a master of that art which is physiognomy; and naturally forms to themselves the character of a stranger from the features of the face'. In reality, all doctors use a physiognomical approach to diagnose many medical conditions, although we may not be as expert at the art as we should be.

The face is a person's most immediate means of communicating with others; it is a shield and banner, a mask and a mirror. It reveals mental faculties and emotional turmoil. It is the first perspective gained of patients as they walk into the consulting room.

The face as a mirror of disease

A fascinating aspect of the art of clinical medicine is the clinical interpretation of the patient's facies. The term 'facies' refers to the appearance, form and character of the face. Not only are specific skin lesions common on the face but the face may also mirror endocrine disorders and organ failure such as respiratory, cardiac, kidney and liver failure.

Jaundice may be masked by the natural colour of the cheeks but the yellow conjunctivae will be distinctive. A marked plethoric complexion may be seen in chronic alcoholics (alcohol may produce a pseudo-Cushing syndrome), in Cushing disease or in polycythaemia. Thickening of the subcutaneous tissues may be seen in chronic alcoholism, acromegaly and myxoedema, and the puffiness of the eyelids in the latter condition may simulate the true subcutaneous oedema of kidney disease.

An individual's personality and mood rarely fail to leave an impression on the facial characteristics.[2] This is partly due to the alteration in facial lines and wrinkles, which may become modified in anger, irritability, anxiety and stress. More profound changes occur with mental disease. Various CNS diseases, such as Parkinson disease and myopathies, can affect facial expression (e.g. the immobile face of the patient with Parkinson disease).

The appearance of the eyes can also be very significant and may reflect underlying systemic disease (see FIG. 8.1).

FIGURE 8.1 Kayser–Fleischer ring around the cornea in a patient with Wilson disease

Diagnostic facies[3]

Acromegalic

The enlarged characteristic face is due to a large supra-orbital ridge that causes frontal bossing, a broad nose and a prominent broad and square lower jaw. Other features include an enlarged tongue and soft tissue swelling of the nose, lips and ears (CHAPTER 14).

Adenoidal

Due to mouth breathing in children: a narrow nose/nares, a high-arched palate (the 'Gothic' palate), prominent incisor teeth, undershot jaw with a perpetually open mouth and 'stupid' expression.

Alcoholic (due to chronic use)

It is important to recognise the characteristic changes as early as possible—a plethoric face, thickened 'greasy' skin, telangiectasia, suffused conjunctivae and rosacea. Other features may include rhinophyma, parotid swelling and characteristic changes to the lips and corners of the mouth.

Children born with fetal alcohol spectrum disorder have specific facial features (see

CHAPTER 23).

Bird-like (systemic sclerosis: CREST syndrome)

The bird-like features—beaking of the nose, limitation of mouth opening, puckering or furrowing of the lips and a fixed facial expression—are due to binding down of facial skin. Other features include telangiectasia on the face and hands.

Chipmunk (thalassaemia major)

There is bossing of the skull, hypertrophy of the maxillae (which tends to expose the upper teeth), prominent malar eminences and depression of the bridge of the nose. The major haemoglobinopathies cause hyperplasia of the skull and facial bones because of an increase in the bone marrow cavity.

Choleric

The patient with cholera has a pale face with cold clammy skin, sunken eyes, hollow cheeks and a forlorn, apathetic look (similar to the Hippocratic facies).

Congenital syphilis

Sometimes referred to as ‘bulldog’ face, it is characterised by a saddle nose due to the sunken bridge of the nose and interstitial keratitis.

Cushingoid

The face has a typical ‘moon shape’, plethora (rubicund), hirsutism (more obvious in women) and acne (CHAPTER 14).

Facial nerve palsy

Features include unilateral drooping of the corner of the mouth and flattening of the nasolabial fold (CHAPTER 22).

- Upper motor neurone (UMN) type: the forehead movement is spared
- Lower motor neurone (LMN) type (e.g. Bell palsy, Ramsay–Hunt syndrome): lack of forehead muscle tone

Hippocratic

This describes the deathly, mask-like features of advanced peritonitis or streptococcal septicaemia—sunken eyes; ‘gaunt’ face; ‘collapsed’ temples; dry, crusty lips; and clammy

forehead.

Horner syndrome

Due to dysfunction of cervical sympathetic output, the main abnormalities are ocular—ipsilateral partial ptosis, miosis (constricted pupil) and enophthalmos; also associated anhidrosis.

Marfanoid (Marfan syndrome)

The typical tall stature, arachnodactyly and chest deformities, combined with the facial features of a subluxation of the lens of the eye and high-arched palate, help to pinpoint the diagnosis (CHAPTER 23).

Mitral (mitral valve disease, especially mitral stenosis)

This is typically shown in flushed or rosy cheeks with a bluish tinge due to dilatation of the malar capillaries. It is associated with pulmonary hypertension.

Mongoloid (Down syndrome)

The facial features include a flat profile, with crowded features, a round head, dysplastic lowset ears, protruding tongue, mongoloid slant of the eyes with epicanthic folds, mouth hanging open and peripheral silver iris spots (Brushfield spots) (CHAPTER 23).

Myopathic (myopathy/myasthenia gravis)

Facial characteristics include an expressionless, ‘tired’-looking face with bilateral ptosis.

Myotonic (dystrophia myotonia)

Typical features include frontal baldness, expressionless triangular facies, partial ptosis, cataracts and temporal muscle atrophy.

Myxoedemic (hypothyroidism)

The face usually has an apathetic look and is ‘puffy’ with possible periorbital oedema. There is broadening of the lower part of the face. The skin (not the sclera) may appear yellow (due to hypercarotenaemia) and is generally dry and coarse. Other features may include thin, coarse, listless hair and loss or thinning of the outer third of the eyebrows. The tongue is usually enlarged and the patient speaks with a ‘thickened’, croaking, slow speech (CHAPTER 14).

Obese

The distinguishing feature from the ‘moon face’ of Cushing disease is the general roundness and

uniform fatness of the face.

Pagetic (Paget disease)

The main feature is skull enlargement, notably of the frontal and parietal areas (the head circumference is usually greater than 55 cm, which is abnormal)—the ‘hat doesn’t fit any more’ hallmark. Other features include increased bony warmth and deafness (CHAPTER 58).

Parkinsonian

Characteristic is the mask-like facies with lack of facial expression and fixed unblinking stare. There is immobility of the facial muscles (CHAPTER 22).

Peutz–Jeghers

Pigmented macules (1–5 mm in diameter) occur on lips, buccal mucosa and fingers.

Risus sardonicus

A grin-like appearance of hypertonic facial muscles (typical of tetanus).

Smoker’s

A face older than the years with premature gross wrinkling of the skin, stained teeth, deep raspy voice, ‘loose’ cough, smell of tobacco.

Thyrotoxic (hyperthyroidism)

The prominent eyes (sclera may not be covered by the lower eyelid) and conjunctivitis are features of the thyrotoxic patient (CHAPTER 14). The thyroid stare (a frightened expression) may also be present (see FIG. 8.2).

FIGURE 8.2 Thyrotoxicosis illustrating a typical thyroid stare

Turner syndrome

The facial characteristics include ptosis—'fish-like' mouth, small chin (micrognathia), low-set ears and deafness. Cardiac lesions include coarctation of the aorta and pulmonary stenosis. Webbing of the neck is the classic sign (CHAPTER 23).

Uraemic

A sallow 'muddy' complexion with uraemic fetor—an ammoniacal halitosis.

Other classic facies (similes)

Bulldog	Congenital syphilis
Chipmunk	Thalassaemia major
Death mask	Peritonitis; cholera
Elfin	William syndrome
Fish-like mouth	Turner syndrome
Hatchet	Dystrophia myotonia
Long face	Fragile X syndrome
Mask	Parkinson; Wilson disease
Monkey	Hypopituitarism
Moon	Cushing syndrome
Old man in child	Marasmus

Raccoon	Basilar skull fracture
Rodent	Beta thalassaemia; Pierre Robin sequence
Toad	Hypothyroidism

Specific characteristics

Various facial signs may be present. The causes of these signs are listed below.

Butterfly ‘rash’

- **SLE**

  Erythema, scaling with a discrete red advancing edge on the cheeks and bridge of the nose; the sharp border, lack of pustules and adherent scale make it differ from rosacea

- **Rosacea**

  Papules, pustules and telangiectasia on an erythematous background on cheeks, forehead and chin

- Erysipelas

  Painful, erythematous, indurated skin infection with a well-defined raised edge

- **Seborrhoeic dermatitis**

  Red and scaly rash involving eyebrows, eyelids, nasolabial folds

- **Photosensitivity eruptions**

  Erythematous on areas that are exposed to the sun

Chloasma/melasma

Increased browning pigmentation, usually confined to symmetrical areas of the cheeks (see FIG. 8.3). Common in pregnancy and caused by drugs:

- combined oral contraceptive pill
- hydroxychloroquine (Plaquenil)
- diphenylhydrazine

FIGURE 8.3 Melasma (chloasma) in the typical distribution soon after the birth of the second child. Sometimes called the mask of pregnancy.

Malar flush

- Mitral stenosis
- Pulmonary stenosis
- Rosacea
- SLE
- Mesenteric adenitis

Spider naevi

- Pregnancy
- Liver disease
- Vitamin B deficiency in normal people

Enlarged tongue

- Acromegaly
- Hypothyroidism
- Amyloidosis
- Down syndrome

Cataracts: risk factors

- Ageing
- Senility
- Corticosteroid therapy
- Diabetes
- Hypoparathyroidism
- Dystrophia myotonia
- Trauma (may be delayed)
- Ocular disease (e.g. glaucoma)
- Smoking

Telangiectasia

- Systemic sclerosis
- CREST syndrome
- Liver disease (e.g. alcoholism)

Cyanosis

Cyanosis is a bluish discolouration of the skin and mucous membranes due to deoxygenated haemoglobin concentrated in the superficial blood vessels. The arterial oxygen saturation is 80–85% before it is clinically apparent. It is classified as central or peripheral.

Central

Cyanosis is present in parts of the body with good circulation, such as the lips and tongue. The areas feel warm. The main causes are pulmonary disease, pulmonary oedema, cyanotic congenital heart disease (right to left shunt), respiratory depression and polycythaemia (see FIG. 8.4).

FIGURE 8.4 Adolescent patient with central cyanotic heart disease and associated clubbing of the fingers

Peripheral

Cyanosis is in the extremities, such as the outer surface of the lips, finger tips, nose and ears. The areas feel cold. The main causes are peripheral vascular disease, cardiac failure, ‘shock’, exposure to cold, left ventricular failure and all causes of central cyanosis.

Clubbing of fingers

Features

- Loss of usual angle between base of nail and nail fold
- Curvature in two planes
- Increased sponginess in base of nail
- Increased convexity of nail
- Mainly caused by respiratory disease

Causes

1. Lung disease:
   - carcinoma
   - bronchiectasis
   - cystic fibrosis
   - abscess/empyema
   - pulmonary fibrosis
2. Heart disease:
   - infective endocarditis
   - cyanotic congenital heart disease (see FIG. 8.4)
3. Liver disease:
   - cirrhosis
4. Gastrointestinal disease:
   - ulcerative colitis
   - Crohn disease

- coeliac disease

5. Congenital

Increased general pigmentation

Increased pigmentation is not common but if obvious in areas exposed to the sun, look for ‘hidden’ areas, such as the inner aspect of the forearms. Causes include those listed below.

Increased melanocyte-stimulating hormone (MSH)

- Addison disease (see CHAPTER 14)
- Cushing syndrome
- Ectopic ACTH syndrome
- AIDS

Metabolic

- Hyperthyroidism
- Haemochromatosis (see FIG. 8.5)
- Cirrhosis of the liver
- Porphyria cutanea tarda
- Chronic kidney failure
- Malnutrition/malabsorption
- Pregnancy

FIGURE 8.5 Patient showing pigmentation of primary haemochromatosis and associated arthritis of fingers

Drugs

- Amiodarone
- Antibiotics (busulphan, bleomycin, minocycline)
- Antimalarials (chloroquine/hydroxychloroquine)
- Arsenic, gold, silver
- Chemotherapy
- Dapsone
- Oral contraceptive pill (OCP)
- Phenothiazines
- Photochemotherapy (PUVA)
- Psoralens
- Thiazides

Tumours

- Lymphomas
- Acanthosis nigricans
- Metastatic melanoma

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Part 2 Diagnostic perspective in general practice

9 A safe diagnostic model

For most diagnoses all that is needed is an ounce of knowledge, an ounce of intelligence, and a pound of thoroughness.

ANON (1951), *LANCET*

The discipline of general practice is probably the most difficult, complex and challenging of all the healing arts. Our field of endeavour is at the very front line of medicine and as practitioners we shoulder the responsibility of the early diagnosis of very serious, perhaps life-threatening, illness in addition to the recognition of anxiety traits in our patients.

The teaching of our craft is also an exciting challenge and presupposes that we have a profound comprehension of our discipline.

Our area is characterised by a wide kaleidoscope of presenting problems, often foreign to the classic textbook presentation and sometimes embellished by a ‘shopping list’ of seemingly unconnected problems or vague symptoms—the so-called undifferentiated illness syndrome.[1] Common undifferentiated symptoms include tiredness or fatigue, sleeping problems, anxiety and stress, dizziness, headache, indigestion, anorexia and nausea, sexual dysfunction, weight loss, loss of interest, flatulence, abdominal discomfort and chest discomfort.[2] It is important, especially in a busy practice, to adopt a fail-safe strategy to analyse such presenting problems. Such an approach is even more important in a world of increasing medical litigation and specialisation.

To help bring order to the jungle of general practice problems, the principal author has developed a simple model to facilitate diagnosis and reduce the margin of error.

The concept of diagnostic triads

A most useful guide to learning or memorising diagnoses, especially of elusive and uncommon conditions, is to remember three key points to the condition. The cognitive process of learning these so-called ‘triads’ and even ‘tetrads’ provides a useful template for the diagnostic

methodology required in general practice. Some simple examples are shown in the following box.

Examples such as these are interspersed throughout the text, especially in this chapter, and are prefixed by the symbol DxT.

Examples of diagnostic triads

DxT angina + dyspnoea + blackouts → aortic stenosis

DxT menstrual dysfunction + obesity + hirsutism → polycystic ovarian syndrome

DxT malaise + night sweats + pruritus → Hodgkin lymphoma

DxT abdominal pain + diarrhoea + fever → Crohn disease

DxT vertigo + vomiting + tinnitus → Ménière syndrome

DxT dizziness + hearing loss + tinnitus → acoustic neuroma

DxT fatigue + muscle weakness + cramps → hypokalaemia

The basic model

The use of the diagnostic model requires a disciplined approach to the problem with the medical practitioner quickly answering five self-posed questions. The questions are shown in TABLE 9.1 .

Table 9.1 The diagnostic model for a presenting problem

1	What is the probability diagnosis?
2	What serious disorders must not be missed?
3	What conditions are often missed (the pitfalls)?
4	Could this patient have one of the ‘masquerades’ in medical practice?
5	Is this patient trying to tell me something else?

This approach, which is based on considerable experience, requires the learning of a predetermined plan which, naturally, would vary in different parts of the world but would have a

certain universal application in the so-called developed world.

Each of the above five questions will be expanded.

An excellent acronym on this theme, ‘PROMPT’, was devised by a reader, Dr Kelly Teagle:

P **P**robability
R **R**ed flag
O **O**ften missed
M **M**asquerades
P **P**atient wants to
T **T**ell me something

Another contribution is by Flinders University medical student, Judah:

Things are not always ‘cut and dried’:

C **C**onnective tissue disorders
U **U**TIs, particularly in very old and very young
T **T**hyroid disease
and
D **D**epression
R remember to **R**ule out serious and **R**are causes
I **I**atrogenic causes
E **E**motional needs
D **D**iabetes

1 The probability diagnosis

The probability diagnosis is based on the doctor’s perspective and experience with regard to prevalence, incidence and the natural history of disease. GPs acquire first-hand epidemiological knowledge about the patterns of illness apparent in individuals and in the community, which enables them to view illness from a perspective that is not available to doctors in any other disciplines. Thus, during the medical interview, the doctor not only is gathering information, allocating priorities and making hypotheses, but also is developing a probability diagnosis based on acquired epidemiological knowledge.

2 What serious disorders must not be missed?

While epidemiological knowledge is a great asset to the GP, it can be a disadvantage in that he or she is so familiar with what is common that the all-important rare cause of a presenting symptom may be overlooked. On the other hand, the doctor in the specialist clinic, where a different spectrum of disease is encountered, is more likely to focus on the rare at the expense of the

common cause. However, it is vital, especially working in the modern framework of a litigation-conscious society, not to miss serious, life-threatening disorders.

To achieve early recognition of serious illness, the GP needs to develop a ‘high index of suspicion’. This is generally regarded as largely intuitive, but this is probably not the case, and it would be more accurate to say that it comes with experience.

The serious disorders that should always be considered ‘until proven otherwise’ include malignant disease, acquired immunodeficiency syndrome (AIDS), coronary disease and life-threatening infections such as meningitis, meningococcal infection (see FIG. 9.1), *Haemophilus influenzae* b infections, septicaemia and infective endocarditis (see TABLE 9.2).

FIGURE 9.1 Meningococcal infection: complications of infarction (DIC) including gangrene from meningococcaemia

Table 9.2 Serious ‘not to be missed’ conditions

Neoplasia, especially malignancy

HIV infection/AIDS

Asthma/anaphylaxis

Severe infections, especially:

- meningoencephalitis
- septicaemia
- meningococcal infection (see FIG. 9.1)
- epiglottitis
- infective endocarditis
- pneumonia/influenza/SARS
- clostridia infections, e.g. tetanus, botulism, gas gangrene

Coronary artery disease:

- myocardial infarction
- unstable angina
- arrhythmias

Imminent or potential suicide

Intracerebral lesions (e.g. subarachnoid haemorrhage)

Ectopic pregnancy

Myocardial infarction or ischaemia is extremely important to consider because it is so potentially lethal and at times can be overlooked by the busy practitioner. It does not always manifest as the classic presentation of crushing central pain but can present as pain of varying severity and quality in a wide variety of sites. These sites include the jaw, neck, ear, arm, epigastrium and interscapular region. Coronary artery disease may manifest as life-threatening arrhythmias that may present as palpitations and/or dizziness. A high index of suspicion is necessary to diagnose arrhythmias.

Diagnostic triads for life-threatening conditions (examples)

DxT fever + rigors + hypotension → septicaemia

DxT fever + vomiting + headache → meningitis

DxT fatigue + dizziness ± syncope → cardiac arrhythmia

DxT fever + drooling + stridor (child) → epiglottitis

DxT headache + vomiting + altered consciousness → subarachnoid haemorrhage (SAH)

DxT abdominal pain + amenorrhoea + abnormal vaginal bleeding → ectopic pregnancy

DxT fatigue + dyspnoea on exertion + dizziness → cardiomyopathy

Consider M^2I^2

A traditional way of classifying serious diseases is the pathology aide-mémoire:

- Malignancy
- Metabolic
- Infarction
- Infection

Danger: think VIC

V = **V**ascular

I = **I**nfection (severe)

C = **C**ancer

Red flags

Red flags (alarm bells) are symptoms or signs that alert us to the likelihood of significant harm. Such underlying disease *must not be missed* and demands careful investigation. Examples include weight loss, vomiting, altered cognition, fever >38°C, dizziness, and/or syncope at the toilet and pallor. Red flags will be outlined under presenting symptoms throughout the text.

3 What conditions are often missed?

This question refers to the common ‘pitfalls’ so often encountered in general practice. This area is definitely related to the experience factor and includes rather simple, non-life-threatening problems that can be so easily overlooked unless doctors are prepared to include them in their diagnostic framework.

Classic examples include smoking or dental caries as a cause of abdominal pain, allergies to a whole variety of unsuspected everyday contacts, foreign bodies, occupational or environmental hazards as a cause of headache, respiratory discomfort or malaise, and faecal impaction as a cause of diarrhoea. We have all experienced the ‘red face syndrome’ from a urinary tract

infection, whether it is the cause of fever in a child, lumbar pain in a pregnant woman or malaise in an older person. The dermatomal pain pattern caused by herpes zoster prior to the eruption of the rash (or if only a few sparse vesicles erupt) is a common trap.

A typical pitfall is Addison disease, where some patients can wait up to 15 years before being diagnosed. The absence of subdued classic pigmentation (see FIG. 9.2) can mask the early diagnosis.

FIGURE 9.2 Woman with Addison disease showing facial pigmentation

Haemochromatosis can be a surprise diagnosis, often discovered by serendipity following a screening blood test for unexplained fatigue. Coeliac disease is a classic master of disguise in both children and adults. It now ranks as one of the most common, widespread and undiagnosed illnesses affecting humans. In Australia, 1.5% of the population are affected but 80% remain

undiagnosed.[3] Research by dermatologists[4] has highlighted that it can present in a number of ways that can affect the skin and hair. Apart from typical gastrointestinal symptoms, such as chronic diarrhoea, steatorrhoea, weight loss, anorexia and abdominal distension, the following atypical symptoms have been described:

- nutritional presentations, including folate, zinc or iron (in particular) deficiency
- grouped blisters around the knees, elbows and buttocks (dermatitis herpetiformis)
- hair loss and mouth ulcers

Menopausal symptoms can also be overlooked as we focus on a particular symptom. Some important pitfalls are given in TABLE 9.3 .

Table 9.3 Classic pitfalls

Abscess (hidden)
Addison disease
Allergies
Candida infection
Chronic fatigue syndrome
Coeliac disease
Domestic abuse, including child abuse
Drugs (see TABLE 9.4)
Endometriosis
Faecal impaction
Foreign bodies
Giardiasis
Haemochromatosis
Herpes zoster
Lead poisoning
Malnutrition (unsuspected)
Menopause syndrome
Migraine (atypical variants)
Paget disease
Pregnancy (early)
Sarcoidosis
Seizure disorders
Tourette syndrome
Urinary infection

Diagnostic triads for some ‘pitfalls’

DxT fatigue + weight loss + diarrhoea → coeliac disease

DxT anorexia/nausea + faecal leaking + abdominal bloating → faecal impaction

DxT abdominal cramps + flatulence + profuse diarrhoea → giardiasis

DxT lethargy + tiredness + arthralgia → haemochromatosis

DxT lethargy + abdominal pains + irritability (in child) → lead poisoning

DxT aching bones + waddling gait + deafness → Paget disease

DxT malaise + cough + fever (± erythema nodosum) → sarcoidosis

DxT (male child) snorting, blinking + oral noises (e.g. grunts) ± loud expletives → Tourette syndrome

4 The masquerades (‘chameleons’)

It is important to utilise a type of fail-safe mechanism to avoid missing the diagnosis of these disorders. Some practitioners refer to consultations that make their ‘head spin’ in confusion and bewilderment, with patients presenting with a ‘shopping list’ of problems. It is in these patients that a checklist is useful. Consider the apparently neurotic patient who presents with headache, lethargy, tiredness, constipation, anorexia, indigestion, shortness of breath on exertion, pruritus, flatulence, sore tongue and backache. In such a patient we must consider a diagnosis that links all these symptoms, especially if the physical examination is inconclusive; this includes iron deficiency anaemia, depression, diabetes mellitus, hypothyroidism (see FIG. 9.3) and drug abuse.

FIGURE 9.3 Hypothyroidism in a 60-year-old woman, a classic masquerade, with a slow subtle onset of facial changes

A century ago it was important to consider diseases such as syphilis and tuberculosis as

the great common masquerades, but these infections have been replaced by iatrogenesis, malignant disease, alcoholism, endocrine disorders and the various manifestations of atherosclerosis, particularly coronary insufficiency and cerebrovascular insufficiency.

If the patient has pain anywhere it is possible that it could originate from the spine, so the possibility of spinal pain (radicular or referred) should be considered as the cause for various pain syndromes, such as headache, arm pain, leg pain, chest pain, pelvic pain and even abdominal pain. The author's experience is that spondylogenic pain is one of the most underdiagnosed problems in general practice.

A checklist that has been divided into two groups of seven disorders is presented in TABLES 9.4 and 9.5 . The first list, 'the seven primary masquerades', represents the more common disorders encountered in general practice; the second list includes less common masquerades—although some, such as Epstein–Barr mononucleosis, can be very common masquerades in general practice.

Table 9.4 The seven primary masquerades

1. Depression
2. Diabetes mellitus
3. Drugs
   - iatrogenic
   - self-abuse
     - alcohol
     - narcotics
     - nicotine
     - others
4. Anaemia
5. Thyroid and other endocrine or metabolic disorders
   - hyperthyroidism
   - hypothyroidism
   - Addison disease
6. Spinal dysfunction
7. Urinary tract infection (UTI)

Table 9.5 The seven other masquerades

1. Chronic kidney failure
2. Malignant disease
   - lymphomas
   - lung
   - caecum/colon
   - kidney
   - multiple myeloma
   - ovary
   - pancreas
   - metastasis
3. HIV infection/AIDS
4. Baffling bacterial infections
   - syphilis
   - tuberculosis
   - infective endocarditis
   - the zoonoses
   - *Chlamydia* infections
   - atypical pneumonias (e.g. Legionnaire disease)
   - others
5. Baffling viral (and protozoal) infections
   - Epstein–Barr mononucleosis
   - TORCH organisms (e.g. cytomegalovirus)
   - hepatitis A, B, C, D, E
   - mosquito-borne infections
     - malaria
     - Ross River fever
     - dengue fever
     - others
6. Neurological dilemmas
   - Parkinson disease
   - Guillain–Barré syndrome
   - seizure disorders
   - multiple sclerosis
   - myasthenia gravis
   - space-occupying lesion of skull
   - migraine and its variants
   - others

7 Connective tissue disorders and the vasculitides

- connective tissue disorders
  - systemic lupus erythematosus (SLE)
  - systemic sclerosis
  - dermatomyositis
  - overlap syndrome
- vasculitides
  - polyarteritis nodosa
  - giant cell arteritis/polymyalgia rheumatica
  - granulomatous disorders

Neoplasia, especially malignancy of the so-called ‘silent areas’, can be an elusive diagnostic problem. Typical examples are carcinoma of the nasopharynx and sinuses, ovary, caecum, kidney and lymphopoietic tissue. Sarcoidosis is another disease that can be a real masquerade (see CHAPTER 38).

Systemic lupus erythematosus (SLE) has been described as ‘the great pretender’.[5] The two most common symptoms are joint pain and fatigue but it is a multisystem disease that may present with involvement of any of these organ systems and may not initially be recognised as such.

As a practical diagnostic ploy, the author has both lists strategically placed on the surgery wall immediately behind the patient. The lists are rapidly perused for inspiration should the diagnosis for a particular patient prove elusive.

5 Is the patient trying to tell me something?

The doctor has to consider, especially in the case of undifferentiated illness, whether the patient has a ‘hidden agenda’ for the presentation.[6] Of course, the patient may be depressed (overt or masked) or may have a true anxiety state. However, a presenting symptom such as tiredness may represent a ‘ticket of entry’ to the consulting room.[7] It may represent a plea for help in a stressed or anxious patient. We should be sensitive to patients’ needs and feelings and, as listening, caring, empathetic practitioners, provide the right opportunity for the patient to communicate freely.

Deep sexual anxieties and problems, poor self-esteem, and fear of malignancy or some other medical catastrophe are just some of the reasons patients present to doctors.

The patient with a self-induced bruising (see FIG. 9.4) was a health professional who was deeply attracted to an inpatient haematologist (Munchausen syndrome).

FIGURE 9.4 Artefactual purpura showing an unusually symmetrical distribution in sites that can be reached by the patient (a 'ticket of entry')—Munchausen syndrome

The author has another checklist (see TABLE 9.6) to help identify the psychosocial reasons for a patient's malaise.

Table 9.6 Underlying fears or image problems that cause stress and anxiety

1	Interpersonal conflict in the family
2	Identification with sick or deceased friends
3	Fear of malignancy
4	STIs, especially AIDS
5	Impending 'coronary' or 'stroke'
6	Sexual problem
7	Drug-related problem
8	Crippling arthritis
9	Financial woes
10	Other abnormal stresses

In the author's experience of counselling patients and families, the number of problems caused by interpersonal conflict is quite amazing and makes it worthwhile to specifically explore the quality of close relationships, such as those of husband–wife, mother–daughter and father–son.

Diagnostic triads for some 'masquerades'

DxT malaise + fever + cough (± erythema nodosa) → TB *or* sarcoidosis

DxT fever + sore throat + cervical lymphadenopathy → EB mononucleosis

DxT fatigue + a/n/v + sallow skin → chronic kidney failure

DxT polyuria + polydipsia + skin/orifice infections → diabetes mellitus

DxT FUO + cardiac murmur + embolic phenomena → infective endocarditis

DxT fatigue + polyarthritis + fever or skin lesions → SLE

DxT loin pain + haematuria + palpable loin mass → kidney carcinoma

DxT malaise + weight loss + cough → lung carcinoma

DxT fever + myalgia/headache + non-productive cough → atypical pneumonia

DxT malaise + night sweats + painless lymphadenopathy → non-Hodgkin lymphoma

DxT arthralgia + Raynaud phenomenon + GORD (± skin changes) → systemic sclerosis

DxT fatigue + headache + jaw claudication → temporal arteritis

DxT weakness + back pain + weight loss → multiple myeloma

DxT lethargy + physical/mental slowing + constipation → hypothyroidism

Note: Diagnostic triads for neurological dilemmas are included in CHAPTER 22 .

Another common yet overlooked stressor is bullying,[8] whether it is in the workplace, school, university, home, internet or elsewhere. It is a significant public health issue. The current

fashion for tough, dynamic, ‘macho’ management styles has created a culture in which bullying can thrive. As GPs, we should be more aware of the possibility that workplace bullying may be contributing to the stresses with which many patients present. A simple, direct, routine question such as ‘How are things at work?’ can create an opportunity to raise the issue.

Identification and transference of illness, symptoms and death, in particular, are important areas of anxiety to consider. Patients often identify their problems with relatives, friends or public personalities who have malignant disease. Other somatoform disorders and the factitious disorders, including the fascinating Munchausen syndrome, may be obvious or extremely complex and difficult to recognise. Consider also ‘Munchausen by proxy’ where carers intentionally produce or feign symptoms in the person (child or elderly patient) in their care. These subtle psychosocial issues are usually termed ‘yellow flags’.

Yellow flags[7]

Yellow flags are signs or behaviours that flag or indicate a psychosocial barrier to recovery. They have been described originally within the framework of chronic pain and disability, especially chronic back pain, and require a shift in our focus of care. Conditions to consider are anxiety, depression, adjustment disorder and personality disorder. Typical yellow flags are presented in TABLE 9.7 .

Table 9.7 Yellow flags: examples

Abnormal illness behaviour
Devious behaviour
Cancelling appointments
Treatment non-compliance/refusal
Somatisation
Absenteeism from work
Poor work performance
Personal neglect
Relationship breakdown
Law and order incidents

A survey by researchers at Melbourne’s Centre for Behavioural Research[9] revealed that the three most feared diseases are cancer (81%), heart disease (32%) and HIV/AIDS (21%).

The bottom line is that patients are often desperately searching for security and we have an important role to play in helping them.

Some examples of application of the model

Hiccough

1	**Probability diagnosis**
	Food and alcohol excess Psychogenic/functional Postoperative • gastric distension • phrenic nerve irritation
2	**Serious disorders not to be missed**
	Neoplasia • CNS • neck • oesophagus • lung Subphrenic abscess Myocardial infarction/pericarditis CNS disorders (e.g. CVA infection) Chronic kidney failure
3	**Pitfalls**
	Alcohol excess Smoking Aerophagy Gastrointestinal disorders • oesophagitis • peptic ulcer • hiatus hernia • cholecystitis • hepatomegaly *Rarities:* • sudden temperature change • neck cysts and vascular abnormalities
4	**Seven masquerades checklist**
	Drugs

5	**Is the patient trying to tell me something?**
	Emotional causes always to be considered

Halitosis

(see CHAPTER 61)[10]

1	**Probability diagnosis**
	Dietary habits—odour-causing foods, e.g. garlic
	Poor oral hygiene
	Orodental disease, e.g. gingivitis, dental abscess
	Dry mouth (e.g. on waking)
	Smoking/alcohol
2	**Serious disorders not to be missed**
	Malignancy: lung, oropharynx, larynx, stomach, nose, leukaemia
	Pulmonary tuberculosis
	Quinsy
	Oral candidiasis
	Lung abscess
	Blood dyscrasias/leukaemia
	Uraemia
	Hepatic failure
3	**Pitfalls**
	Nasal and sinus infection
	Systemic infection
	Appendicitis
	Bronchiectasis
	Hiatus hernia
	Starvation
	Rarities
	Pharyngeal and oesophageal diverticula
	Sjögren syndrome
	Scurvy
4	**Seven masquerades checklist**
	Depression
	Diabetes: acidosis (acetone)

	Drugs
5	**Is the patient trying to tell me something?** Possible manifestation of psychogenic disorder Halitophobia

Practice tips

- Follow the ‘baseball rule’ for a delayed or puzzling diagnosis: ‘Three strikes and you’re out’.
- Infarction—think fast!

  acute coronary events: 60–90 minutes

  stroke: 3–4 hours

  femoral artery: 4 hours

  torsion of testis: 4–6 hours

Patient education resources

Hand-out sheets from *Murtagh’s Patient Education* 8th edition:

- Bullying of children
- Bullying in the workplace

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10 Depression

I am ignorant and impotent and yet, somehow or other, here I am, unhappy, no doubt, profoundly dissatisfied... In spite of everything I survive.

ALDOUS HUXLEY (1894–1964)

Depressive illness, which is probably the greatest masquerade in general practice, is one of the commonest illnesses in medicine and often confused with other illness. Untreated, depression can result in disability and death.[1] The most feared outcome is suicide. It is present in at least 17% of patients who present to GPs[2] and has a 12-month prevalence of 5% and a lifetime risk of 15%.[3] It is often unrecognised,[1] yet moderate to severe depression is as disabling as congestive heart failure[1] and with a morbidity comparable to coronary heart disease. Further, depression is the *leading* cause of disability for all conditions among both sexes, both in Australia and worldwide.[4,5] The lifetime risk of suicide in patients diagnosed with depression is 6% and treatment halves this risk.[1]

Despite being treatable, 60% of sufferers have not used any form of health service in the previous months.[4] Lack of awareness, stigma and shame on behalf of the patient contribute to this. Of those receiving treatment, three-quarters will be managed in general practice.[5,6] As Whiteford[4] notes:

> It is clear that the main focus of activity aimed at reducing the burden of common mental health disorders in Australia is in primary care. Specialist mental health services play a supporting, but not central, role.

Depression is a chronic relapsing organic brain disease. Its mean onset is at 27 years of age. However, 40% of sufferers present by 20 years of age.[7] The average duration of episodes is 3–4 months and 40% of patients will relapse within a 12-month period.[7]

The cause of depression is multifactorial, having biological, psychological and social factors. Mood disorders in general have a strong familial tendency, and the risk of developing a depressive disorder can be thought of in terms of a ‘stress-vulnerability model’. That is, an individual may have a genetically determined vulnerability and if enough stress is endured a mood disorder may result. Those who are more genetically vulnerable require less stress, but if enough stress is applied, any individual can develop a mood disorder. A significant characteristic is that it impairs thinking, leading to pessimism with negativity and a loss of drive and productivity.

There are six clusters of depressive symptoms:

- mood, e.g. sadness, anhedonia, irritability
- vegetative, e.g. sleep, appetite, sexual drive
- cognitive, e.g. attention, memory, self-worth
- impulse control, e.g. suicide, anger, homicide
- behavioural, e.g. motivation, interests, tiredness
- physical, e.g. headaches, constipation

A useful working rule is to consider depression as an illness that dampens the five basic innate activities of humans:

- energy for activity
- sex
- sleep
- appetite and thirst
- elimination of waste

Classification

- The DSM-5 classification divides depressive disorders into major depressive disorder (MDD), disruptive mood dysregulation disorder, persistent depressive disorder (PDD) and premenstrual dysphoric disorder. Other ‘specified’ and ‘unspecified’ disorder categories allow for diagnosis of those patients who fall short of the various diagnostic criteria.
- MDD includes those disorders with one or more major depressive episodes. Excluding criteria include any previous mania or hypomania, and the episode not being attributable to a psychotic disorder, or a substance or medical condition.

  MDD is subclassified with coded course and severity specifiers. These include mild, moderate and severe (see TABLE 68.1),[8] with psychotic features, in partial remission and in full remission.

- A significant subtype of depression is dysthymia, which is a chronic mild depression lasting at least 2 years but not fulfilling the criteria for MDD. For other subtypes, refer to CHAPTER 68 .

The diagnostic approach

The diagnosis is based on the history and the mini mental state examination.

The two key criteria for major depressive disorder (MDD) in the DSM-5 are a pervasive depressed mood and marked loss of interest or pleasure (otherwise referred to as anhedonia) persisting for at least 2 weeks. Other criteria (listed below) include sleep issues, fatigue, lack of energy, poor concentration and feelings of worthlessness.

In a general practice setting, having a checklist of these symptoms to work through with a patient can be a useful part of the assessment of the depressed patient.

Questions to assess level of depression

- What do you think is the matter with you?
- Do you think that your feelings are possibly caused by nerves, anxiety or depression?
- Can you think of any reason why you feel this way?
- Do you feel down in the dumps?
- Do you feel that you are coping well?
- Do you have any good times?
- Has anything changed in your life?
- How do you sleep? Do you wake early?
- What time of the day do you feel at your worst?
- Where would you put yourself between 0% and 100% (a visual analogue scale is useful here)?
- Have you felt hopeless?
- Do you brood about the past?
- What is your energy like?
- What is your appetite like?
- Are you as interested in sex as before?
- Do you feel guilty about anything?
- Do you feel that life is worthwhile?
- Has the thought of ending your life occurred to you?
- Do you cry when no one is around (especially useful for children)?

Two particularly good questions are:

- In the past month, have you been bothered by the fact that you feel down, depressed or hopeless?
- In the past month, have you often been bothered by the fact that you have little interest or pleasure in doing things?

Rule out other mental disorders

Enquire about substance use and abuse, anxiety, psychosis, manic/hypermanic episodes, intimate partner violence, bereavement and postpartum depression.

Important differentiated diagnoses (organic disease)

Important differential diagnoses (organic disease) to consider are malignancy (especially of lung, brain, pancreas and blood/lymphatics), early dementia, CCF, endocrine disorders (e.g. thyroid disease), menopause, liver and renal failure, infections (e.g. mononucleosis), neurological (e.g. MS, Parkinson), adverse effects of medication, anaemia, SLE and cerebrovascular disease.

Diagnostic guidelines for major depressive disorder[9]

At least 5 of the following 9 symptoms with anhedonia and/or depressed mood for ≥ 2 weeks:

- Depressed mood
- Anhedonia (decreased interest/pleasure)
- Sleep change: increased/decreased
- Guilt/worthlessness
- Decreased energy
- Impaired/increased concentration
- Changer of appetite and weight
- Psychomotor retardation or agitation
- Suicidal ideation

Screening investigations to consider

FBE; TFTs; U&Es; vitamins B, D, folate; blood glucose; urine toxicology; CT or MRI.

Depression scales

Depression scales are useful both to detect potential mood disorders (i.e. screening) and to monitor an individual patient over time. Scales commonly used include K10 (a distress score), DASS 21 or 42 (for depression and anxiety symptoms) and PHQ 2.

Somatisation

Another issue with depression in the primary care setting is recognising somatising patients who present without obvious psychological symptoms. Non-specific symptoms such as insomnia, prolonged fatigue, headache, nausea and musculoskeletal pain are common presentations in people with depression.[10] Studies suggest GPs find it challenging to identify these patients, and that using self-reporting patient-screening tools can help with identification.[3]

Key facts and checkpoints

- Depression is common, serious and treatable.
- Depression is a chronic relapsing organic brain disease.
- It may coexist with anxiety disorders, stress (physical and mental) and substance abuse disorders.
- 1 million Australians live with depression (compared with anxiety, which is 2 million) and 8 lives are taken every day, of which 6 are men.[11]
- The cause of depression is multifactorial, and can be thought of in terms of a ‘stress-vulnerability model’.
- Somatising patients without obvious psychopathology are common and difficult to recognise and manage.
- It is strongly associated with an increased risk of suicide, a fact that demands risk assessment.

Depression in the elderly

The rate of antidepressant prescribing for Australians over 80 years of age is higher than for any other age group.[12] Despite this, depression in the elderly is underdiagnosed.[13] Depression can have bizarre features in the elderly and may be misdiagnosed as dementia or psychosis. Agitated

depression is the most frequent type of depression in the aged. Features may include histrionic behaviour, delusions and disordered thinking.

Depression is often missed in the elderly because it is atypical and less expressive, and patients tend to be ashamed and reluctant to admit it.

A useful clue can be a change in sleep pattern. Medical illness is also an important precipitant of depression in the elderly.

Older patients may have more side effects from medications (especially nausea, dizziness, falls and hyponatraemia).[13] They also tend to have only a modest response to antidepressants, and, if medication is used, a low initial dose and slow increase is recommended.[1] Psychological therapies can be useful, but tend to be underused.[13]

Depression in children and adolescents

Sadness is common in children, but depression, although not as common, does occur and is characterised by feelings of helplessness, worthlessness and despair. Parents and doctors both tend to be unaware of depression in children.[14]

Major depression in children and adolescents may be diagnosed using the same criteria as for adults, namely loss of interest in usual activities and the presence of a sad or irritable mood, persisting for 2 weeks or more. In children, irritability may be more prominent than sadness.[15] The other constellation of depressive symptoms, including somatic complaints, may be present. Examples include difficulty in getting to sleep, not enjoying meals, poor concentration and low self-esteem. Poor motor skills and family instability are an association. Depression can present as antisocial behaviour or as a separation anxiety (e.g. school refusal). Although suicidal thoughts are common, suicide is rare before adolescence.

Depressed adolescents are a difficult challenge for the general practitioner. Effective engagement and establishing rapport in a ‘youth-friendly’ environment is critical.[16]

See CHAPTER 90 for the evaluation and treatment of depression in adolescents.

Perinatal depression

This term refers to depression occurring either in the antenatal period or in the 12 months after delivery. It affects 9% of women during pregnancy and 16% after the birth[17] and affects the well-being of the woman, the baby and significant others. Anxiety is likely to be as or more common.[17] Women at risk of perinatal depression include those with previous mental health problems, those who do not have support and those who have been through difficult times (e.g. family problems, abuse or loss) or who feel isolated either by distance or culture or both.

Because it is so prevalent, routine screening is recommended by the Beyond Blue guidelines. This involves implementing the use of the Edinburgh Postnatal Depression Scale (EPNDS), a

validated screening tool, at least once, preferably twice, both antenatally and postnatally. Asking permission and explaining the process before implementing the screening is helpful. Women at higher risk will require more intense screening and monitoring.

If perinatal depression is identified, the GP should take into account the individual woman's context, her family and cultural setting, and use a family-centred approach. Because of the intense emotions involved in having a baby, establishing a strong therapeutic relationship, using an open collaborative approach and active listening techniques, will help to develop trust, confidence, mutual respect and empowerment. Psycho-education should be provided and appropriate follow-up and continuity of care, with (if appropriate) a coordinated team approach.

If a woman or a baby is at risk, urgent referral is recommended. Pharmacological therapies can be used in pregnancy, but the benefits need to be balanced against the risks to both mother and fetus. Psychosis in perinatal depression is fortunately rare but does occur, and requires urgent psychiatric assessment. Refer to CHAPTER 101 .

Management of depression

Important considerations from the outset are:

- Is the patient a suicide risk?
- Does the patient require inpatient assessment?
- Is referral to a specialist psychiatrist indicated?

Suicide assessment

Data from the Australian Bureau of Statistics illustrate the levels of suicide in Australia over the 10 years to 2019:[18]

- Intentional self-harm (suicide) is the 13th leading cause of death in Australia, but the 10th leading cause in males
- 75% of all suicide deaths in Australia were males
- The national rate is 12.9 suicides per 100 000 people
- The rate of suicide in men has risen over this period from 17.5 per 100 000 to 19.8, and in women, from 5.0 to 6.3
- The median age of people who suicide is in their early to mid-40s

If the symptoms are major and the patient appears in poor health or is a suicide risk, referral is appropriate.

The importance of putting these safety issues at the beginning of the management process is reflected in the SET A PACE[7] model of treatment, proposed by Mahli et al.

To clarify the risk of suicide and appropriate response, ask about:[16]

- suicidal thoughts
- plan
- lethality
- means
- past history
- suicide of family member or peer

Low risk (fleeting thoughts of self-harm or suicide but no current plan or means):

- Discuss availability of support and treatment options.
- Arrange follow-up consultation (timing of this will be based on clinical judgment).
- Identify relevant community resources and provide contact details.

Medium risk (suicidal thoughts and intent but no current plan or immediate means):

- Discuss availability of support and treatment options.
- Organise reassessment within 1 week.
- Have contingency plan in place for rapid reassessment if distress or symptoms escalate.
- Develop a safety plan (a prioritised written list of coping strategies and sources of support to use when experiencing suicidal thinking).

High risk (continual/specific suicidal thoughts, intent, plan and means):

- Ensure that the person is in an appropriately safe and secure environment.
- Organise reassessment within 24 hours and monitoring for this period.
- Follow-up outcome of assessment.

A useful suicide risk assessment is the SAD PERSONS (mnemonic) index (see TABLE 10.1). A score greater than 7 represents a very high risk that demands careful attention, including referral to an acute psychiatric service. The suicide rates in Australia, which demonstrate two peaks in males, are illustrated in FIGURE 10.1 .

Table 10.1 SAD PERSONS index: suicide risk assessment

Risk factor	Criteria	Score
Sex	Male	1
Age	<20 years; >45 years	1
Depression	Major (e.g. depressed mood)	2
Psychiatric history	Previous attempts	1
Excessive drug use	Ethanol or other drug use	1
Rationality loss	Psychosis, severe depression	2
Separated	Loss of spouse or other single	1
Organised plan	Determined suicide plan	2
No supports	No community back-up; generally isolated	1
Sickness	Chronic illness	1

Score >7 = high suicide risk

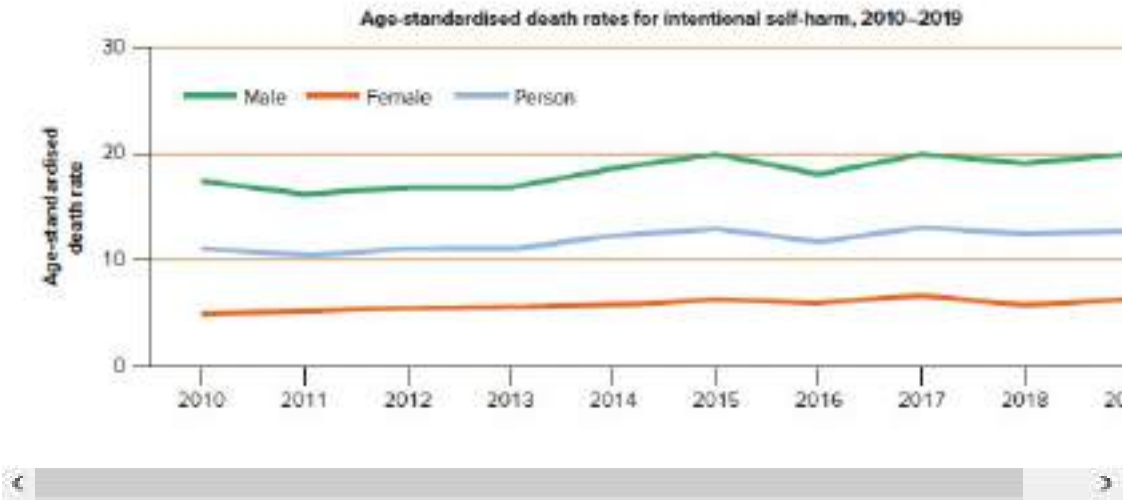


FIGURE 10.1 Death from intentional self-harm (suicides) in Australia, 2010–2019, as a whole and by sex

Source: Australian Bureau of Statistics[17]

Notes: Age-standardised death rate. Death rate per 100 000 estimated resident population as at 30 June (mid year). See the Data quality section of the methodology for further information on specific issues related to interpreting time-series and 2019 data. Care needs to be taken when interpreting data derived from Victorian coroner-referred deaths including suicide. Changes in coding processes have been applied to 2019 data.

If there is concern about suicide risk and treatment is supervised outside hospital,

provide closer supervision and considerable support, and prescribe drugs that are less toxic in overdosage (e.g. mianserin or fluoxetine). If tricyclics are prescribed, useful guidelines are that dangerous medical complications occur with an equivalent dosage of 1000 mg (40 tablets) or imipramine and a high risk of death with 2000 mg (80 tablets).[15]

After safety is established (and this will need to be continually reassessed at each consultation), the two other aspects that need to be developed early in the process (and also continue on through the long-term management of the patient) are **educating** the patient about his or her condition and individual situation, and establishing a **therapeutic relationship.**

Assessment includes:

- **characterising** the symptom profile
- **calibrating** the severity and chronicity—rating scales can be employed here
- **corroborating** (if possible and appropriate) medical and psychiatric comorbidities and context. Significant psychiatric, physical and social comorbidities of depression are common. These include 49% suffering an anxiety disorder, 40% reporting child sexual abuse, 57% child physical abuse, 42% having been at some stage afraid of their partner and 72% reporting a chronic physical condition.[20] Putting the patient's condition into his or her individual psychosocial and medical context will improve the assessment
- **considering** coping styles, and the social, financial and occupational consequences of the patient's condition and situation

This assessment is often spread over multiple consultations.

Treatment

The basic treatments are outlined by the acronym **PACE** (which purposefully places priority on the psychological treatments—see FIG. 10.2). These are:

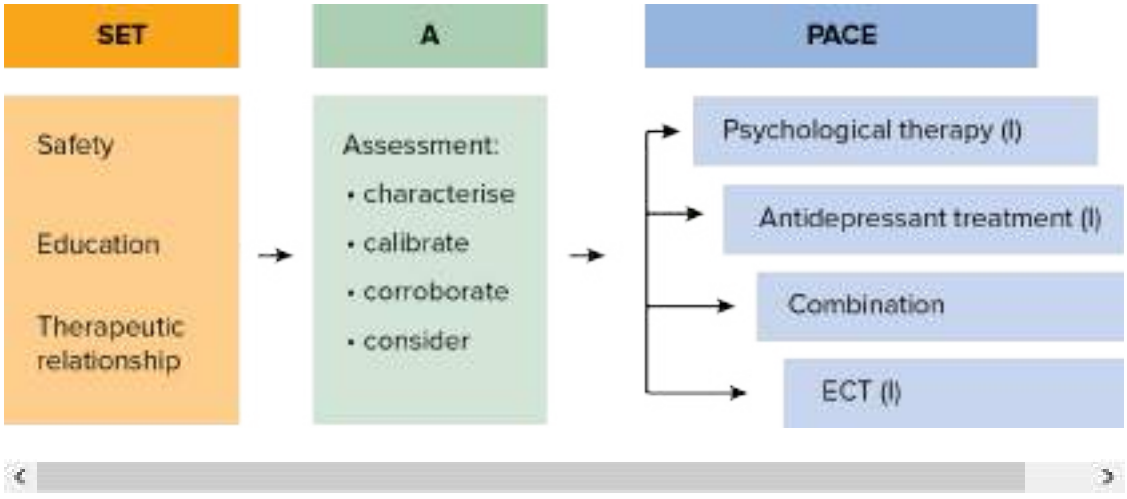
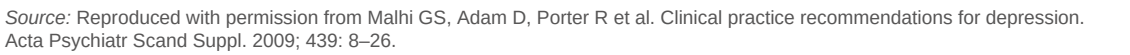


FIGURE 10.2 The SET A PACE model of treatment of depression



Source: Reproduced with permission from Malhi GS, Adam D, Porter R et al. Clinical practice recommendations for depression. Acta Psychiatr Scand Suppl. 2009; 439: 8–26.

- **Psychological**, including basic psychological treatments, such as advice on lifestyle changes, problem solving, guided self-help, structured supervised exercise and supportive counselling.[1,6] All patients with depression should be offered these types of support.[1,16] More sophisticated techniques, such as cognitive behaviour therapy (CBT) or interpersonal therapy (IPT), may be used for selected patients[7] and should be undertaken only by appropriately trained doctors or therapists.[6] Another option that some patients may prefer is computer-based cognitive behaviour therapy (CBT) programs.[13] CBT involves teaching patients new ways of positive thinking, which have to be relevant and achievable for the patient (see CHAPTER 4). Patients need to be able to recognise their own negative cognitions, including their anxieties and worries.

- **Antidepressants:** antidepressant medication is useful in moderate to severe depression (see TABLE 10.2), or when depression has an anxiety disorder codiagnosis.[13] Antidepressant therapy should be avoided if bipolar disorder is suspected, and screening should be actively conducted for symptoms of past or previous mania.[13]

In terms of which drug to use, there is no single drug that is preferred. Most antidepressant agents are approximately equal in efficacy, although individual patient response may vary considerably.[21] However, selective serotonin reuptake inhibitors (SSRIs) are considered to have the most favourable balance of benefit to harm in moderate to severe depression.[13] Sexual dysfunction and gastrointestinal side effects are common.[7] The most toxic agents are the tricyclic antidepressants (TCAs) and the monoamine oxidase inhibitors (MAOIs). Other suitable first-line agents are reboxetine (common side effects include hypersomnia, fatigue and nausea) and mirtazapine (which can cause weight gain and drowsiness).[7] SSRIs have a relatively flat dose–response curve, but dose increase within the recommended range is reasonable if there is a partial response at a lower dose and no troublesome side effects.

Combining different antidepressants or augmentation with lithium or antipsychotics should be done with psychiatrist supervision.[7]

Serotonin and noradrenaline reuptake inhibitors (SNRIs) appear to be more effective in treating severe depression symptoms (and may be a suitable first-line option here) but otherwise adverse effects may limit them to second-line treatment.[7] TCAs and MAOIs are considered second-line because of their side effect profiles.[14]

Table 10.2 First-line pharmacological treatment options for depression[15]

Drug	Usual initial dose	Maximum dose

SSRIs		
citalopram	20 mg (10 mg >65 years)	40 mg (20 mg >65 years)
escitalopram	10 mg	20 mg
fluoxetine	20 mg	80 mg
fluvoxamine	50 mg (at night), then 100 mg after 5–7 days	300 mg
paroxetine	20 mg	60 mg
sertraline	50 mg, then 100 mg after 5–7 days	200 mg
SNRIs		
desvenlafaxine (controlled-release)	50 mg	200 mg
duloxetine	60 mg	120 mg
venlafaxine (controlled-release)	75 mg	375 mg
Other		
mirtazapine	15–30 mg (at night)	60 mg
agometaline	25 mg (at night)	50 mg
reboxetine	2–4 mg (bd)	10 mg

The benefit of medication in moderate depression is equivalent to psychological therapies such as CBT/IPT and both of them are around 20% more likely to achieve remission than placebo.[1] In severe depression, medication is more effective than psychological therapies (see FIG. 10.3), though the latter in addition may help reduce relapse rates once remission is achieved by medication.[1,6]

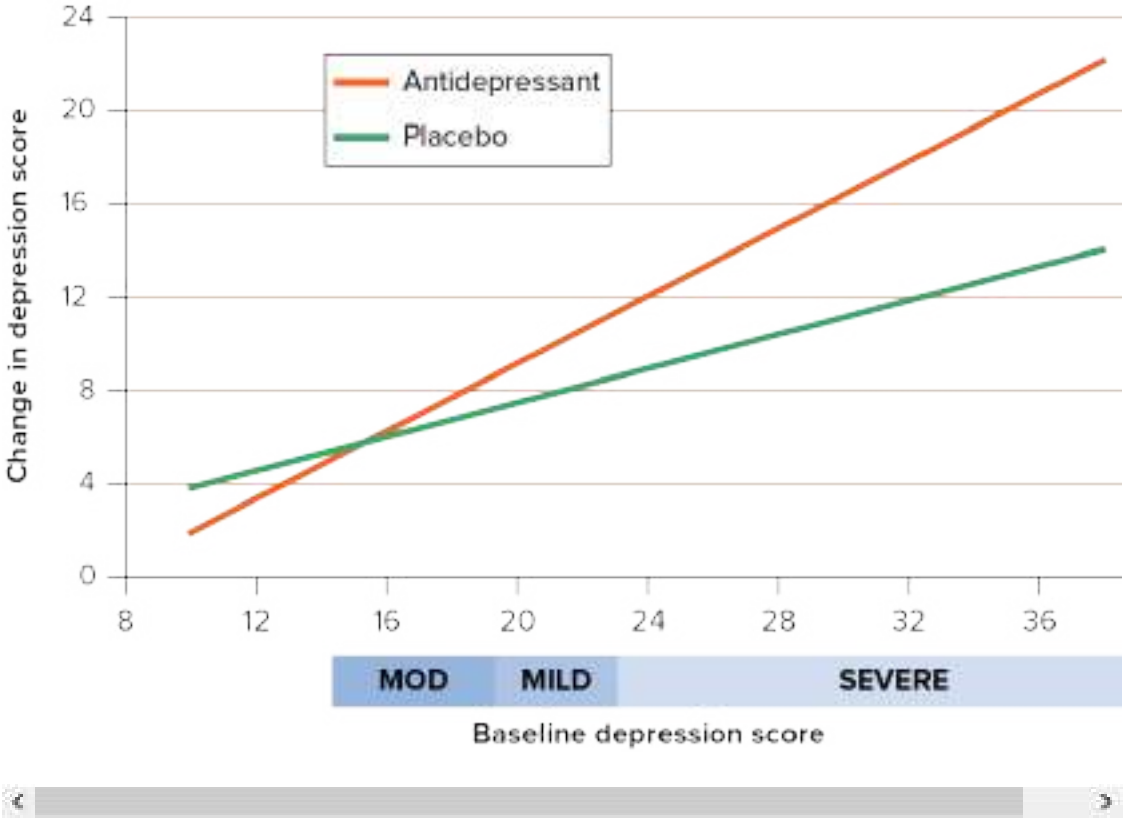


FIGURE 10.3 Antidepressant vs placebo drug effect on depression[19]

The aim of treatment of depression is to achieve and maintain remission.[1] Remission is defined as having minimal or no symptoms of depression,[7] and a good way of asking patients about this is to ask 'Do you think you are back to your normal self?'.

When using antidepressants, if a response is not evident in the first 2 weeks or there is an inadequate response in 6 weeks, then it is unlikely that this medication will work for this patient, and a treatment change is recommended.[7] A washout period will be required before a second medication is tried. Patients need close monitoring early on in the course of treatment, and weekly monitoring may be helpful.[13] If remission is not achieved in 3 months, then consider a second opinion and continue active treatment.[7]

- **A combination** of antidepressants and psychological therapy can be considered if there is an inadequate response to either therapy alone. Combining medication with psychological therapy is more effective than either therapy alone in moderate or severe depression.[7]

- **ECT (electroconvulsive therapy)** is a relatively safe and effective therapy for severe or resistant depression. There is some risk of transient short-term cognitive impairment and long-term memory impairment, and this therapy is reserved for severe depression when pharmacotherapy has failed;[7] it is administered under the supervision of a psychiatrist.

  Possible indications include:

psychotic depression (e.g. delusions, hallucinations)

melancholic depression unresponsive to antidepressants

severe postnatal depression and psychosis

substantial suicide risk

ineffective antidepressant medication and/or previous response to ECT

severe psychomotor depression: refusal to eat or drink, depressive stupor, severe personal neglect

Immediate referral for hospital admission is necessary in most of these circumstances. The course, which is highly variable and individually tailored, is about 8–12 sessions given at 1–3 sessions a week. The most common initial ECT method is unilateral therapy.[1]

Transcranial direct current electro-magnetic stimulation, where no anaesthetic is required, is a procedure being explored as a less invasive alternative to ECT.[22]

Useful management guidelines

- **Mild depression:** psychological therapy
- **Moderate depression:** psychological therapy and/or antidepressants
- **Severe depression:** antidepressants, and consider addition of psychological therapy to maintain remission. Consider psychiatric review, ECT.

When to refer

- Uncertainty about diagnosis
- Inpatient care obviously necessary
- Severe depression
- Inability to cope at home
- Psychotically depressed (with delusions or hallucinations)
- Substantial suicide risk
- Failure of response to routine antidepressant therapy

- Associated psychiatric or physical disorders
- Depression in the elderly can be a difficult problem—where diagnosis including dementia is doubtful
- Children with apparent major depression

Choice of treatment

The choice of treatment should be determined together by doctor and patient, and the best outcomes are likely when a good therapeutic alliance is formed[6] and patient preferences are taken into account.[13] To quote the RANZCP guidelines: 'Of greatest benefit is the therapeutic relationship, which enables agreement on treatment selection and continuation.'[1] A strong determining factor on patient preference may be the patient's own previous experience of treatments, or his or her perceptions of treatment results in other people they know or potential adverse effects of the proposed treatment.[13]

Whatever treatment is chosen by the doctor and patient working together, the choice is less important than persisting with treatment. As the Beyond Blue guidelines state, it is 'not so much what you do but that you keep doing it'.[6] This is why consistent follow-up and monitoring is so beneficial.

Complementary and alternative therapies

Complementary and alternative therapies are widely used for depression. It is estimated that only 50% of Australians who are depressed receive an evidence-based professional intervention.[23] A large driver for this is the public's belief in complementary and alternative therapies. One study[24] showed 57% regarded vitamins, minerals, tonics or herbal medicines as likely to be helpful for treating depression, compared with 29% who regarded antidepressants as likely to be helpful.

Despite this, none of these therapies is supported by evidence, though some warrant further evaluation.[25] Because of their common usage (nearly half of Australians have used a complementary medicine in the previous 12 months),[25] actively enquiring about use of any complementary and alternative therapies in patients with depression is advised.

One of the more commonly used and extensively researched alternative therapies is St John's wort (*Hypericum perforatum*), which has had mixed results in the research on its effectiveness. One review of the literature suggests it is effective in mild to moderate depression,[26] though two others suggest it is not.[27,28]

Regardless of its effectiveness (or not), it has a lot of medication interactions. These include HIV medicines, warfarin, digoxin, anticonvulsants, oral contraceptives and triptans.[21] Because of this, care needs to be taken with using St John's wort, including when switching between different preparations that may contain different amounts of active compound.[19]

Serotonin syndrome

- While rare, serotonin syndrome is a serious adverse reaction to the use of SSRIs and other serotonergic medications, including St John's wort.
- Symptoms must coincide with the introduction or dose increase of a serotonergic agent. Drugs to be considered here include antidepressants, opioids (especially tramadol), stimulant drugs, illicit drugs, anti-emetics, lithium and selegiline.
- Other causes, such as infection, substance abuse or withdrawal, must be excluded.
- At least three of the symptoms or signs attributed to the syndrome must be present, e.g.:

  mental status/behaviour changes (e.g. agitation, confusion, hypomania, seizures)

  altered muscle tone (e.g. tremor, shivering, myoclonus, hyper-reflexia)

  autonomic instability (e.g. hypertension or hypotension, tachycardia, fever, diarrhoea)

The offending agents should be withdrawn immediately and supportive therapy initiated; refer to an emergency department.

Continuing treatment

If antidepressant medication is used and remission achieved, it is recommended that it be continued for a minimum of 12 months for an initial episode, and for 2–3 years in subsequent episodes or in those at high risk of relapse.[1,13] Risk factors for relapse include:[7,13]

- residual depressive symptoms
- 2 or more prior episodes in the past 5 years
- 3 or more prior episodes
- history of severe or prolonged depression (especially with psychosis or attempted suicide)
- comorbid medical problems
- life stressors

When remission is achieved, and the treatment is in the maintenance phase, ongoing monitoring of treatment effectiveness, tolerability and adherence is recommended.[7] Encouragement to persist with the treatment will improve compliance.[1] When ceasing medication, withdrawal reactions are common, so a gradual withdrawal by halving the dose each week may help reduce these.[1]

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11 Diabetes mellitus

Those labouring with this Disease, piss a great deal more than they drink. Authors who affirm the drink to be little or nothing changed are very far from the truth, because the urine very much differed both from the drink taken in and also in being wonderfully sweet as if it were imbued with honey or sugar.

THOMAS WILLIS (1621–1675), *THE PISSING EVIL*

Diabetes comes from a Greek word meaning 'to pass or flow through' (i.e. excessive urination) and *mellitus* means 'sweet'. It is a disease caused by a relative or absolute deficiency of insulin.

There are two main types of diabetes (see TABLE 11.1).

Table 11.1 Clinical differentiation between type 1 and type 2 diabetes

	Type 1	Type 2
Relative frequency (approx.)	10%	85–90%
Peak age incidence	10–30 years	>40 years
Age of onset	Usually young <20	Usually middle-aged >40
Onset	Rapid	Insidious/slow
Presentation	Polyuria, polydipsia weight loss	Milder symptoms, often asymptomatic
Weight at onset	Low (thin)	High (obese)
Ketoacidosis	Yes	Rare
Familial	Weak	Strong
Insulin status	Deficient	Resistant

Note: These are generalisations and the clinical features may vary (e.g. patients with type 2 diabetes may be thin and have a rapid onset, or present at an earlier age).

- Type 1 is also known as juvenile onset diabetes or insulin dependent diabetes mellitus (IDDM).
- Type 2 is also known as maturity onset diabetes or non-insulin dependent diabetes mellitus (NIDDM).

Type 1 has an autoimmune causation which is also responsible for a late-onset form known as late onset autoimmune diabetes in adults (LADA).

Diabetes: a real masquerade

The onset of type 2 diabetes can be subtle and by stealth. In 2014–15, around 1.2 million (5%) Australians had diagnosed type 2 diabetes[1] and another 500 000 (2.1%) were estimated to have type 2 diabetes but were not yet diagnosed.[2] A further 2 million (8.4%) had impaired fasting glucose or impaired glucose tolerance. Around half of those with type 2 diabetes have complications (when microalbuminuria is included), many of whom already have a complication at the time of diagnosis. The challenge for GPs is to be on constant lookout for these individuals, especially those at risk. Type 2 diabetes is becoming more prevalent in industrial countries—due to the ageing population, broadened diagnostic definitions and because our lifestyle encourages us to 'eat more and walk less'.[3] Furthermore, roughly 60% of our population are overweight or obese.

Complications occur in both type 1 and type 2 diabetes.

- Several causes of secondary diabetes are uncommon (pancreatic disease; approx. 2.5%) or very uncommon (see TABLE 11.2).
- Asymptomatic people at high risk of undiagnosed diabetes should be screened by blood glucose or HbA1c measurement.

Table 11.2 Causes of secondary diabetes

Pancreatic disorders (sometimes called 'Type 3c diabetes')
Chronic pancreatitis
Endocrine disorders
Cushing syndrome
Acromegaly
Phaeochromocytoma
Polycystic ovarian syndrome
Haemochromatosis
Drug-induced diabetes (transient)
Thiazide diuretics

Oestrogen therapy (high dose—not with low-dose HRT)
Corticosteroids
Other transient causes
Gestational diabetes
Medical or surgical stress

Key facts and checkpoints

- In Australians older than 25 years the prevalence of diabetes is 7.5%, with another 10.6% having impaired glucose tolerance.[1]
- About 30% of those with impaired glucose tolerance will develop clinical diabetes within 10 years.[3]
- Many people with type 2 diabetes are asymptomatic.
- Diabetes can exist for years before detection and complications may be evident.
- Blood glucose may be temporarily elevated during acute illness, after trauma or surgery.

(a)

(b)

(c)

FIGURE 11.1 Skin signs of diabetes: **(a)** Recurrent staphylococcus folliculitis, **(b)** *Candida albicans* erosio interdigitalis, **(c)** *Candida albicans* balanitis

Clinical features

The classic symptoms of uncontrolled diabetes are:

- polyuria

- polydipsia
- loss of weight (type 1)
- tiredness and fatigue
- propensity for infections, especially of the skin and genitals (vaginal thrush)

The young person with type 1 diabetes typically presents with a brief 2–10 week history of the classic triad of symptoms:

DxT thirst + polyuria + weight loss → type 1 diabetes

The first presentation of type 1 diabetes (typically an unwell child with a high finger-prick blood glucose) is a medical emergency, requiring hospital assessment. Other possible symptoms are:

- vulvovaginitis
- pruritus vulvae
- balanitis

} due to *Candida albicans*

- nocturnal enuresis (type 1)
- blurred vision/visual changes

Symptoms of complications (may be presenting feature) include:

- staphylococcal skin infections
- polyneuropathy: tingling or numbness in feet, pain (can be severe if present)
- impotence

- arterial disease: myocardial ischaemia, peripheral vascular disease

History

The history from a person with suspected or known diabetes should cover the following features, including assessment of cardiovascular risks and end-organ damage.

- Specific symptoms:

    polyuria

    polydipsia

    loss of weight

polyphagia

tiredness/malaise/fatigue

nocturia

- Related general symptom review:

  cardiovascular (e.g. chest pain, dyspnoea)

  urinary function

  sexual function

  neurological (e.g. tingling in feet/hands)

  vision (e.g. blurred)

  infection tendency (e.g. skin, urine, genital)

  genital itching

- General:

  family history

  medication

  smoking and alcohol

  obstetric history (where applicable)

  physical activity

  nutrition/eating habits

Examination

The physical examination should ideally follow the protocol for annual review.

Initial screening for suspected diabetes should include:

- general inspection, including skin
- BMI (weight/height)
- waist circumference
- visual acuity

- blood pressure—lying and standing
- test for peripheral neuropathy: tendon reflexes, sensation (e.g. cotton wool, 10 g monofilament, Neurotips)
- urinalysis: glucose, albumin, ketones, nitrites

Investigations

- Initial: fasting or random blood sugar, follow-up oral glucose tolerance test (OGTT) or glycated haemoglobin (HbA1c) if indicated
- Other tests according to clinical assessment (e.g. lipids, kidney function, urine albumin–creatinine ratio (ACR), ECG)

Risk factors

- Age >40 years
- Family history
- Overweight/obesity
- Sedentary lifestyle
- History of gestational diabetes, pancreatitis
- Women with polycystic ovarian syndrome (PCOS)
- Hypertension/ischaemic heart disease
- Medication causing hyperglycaemia
- Ethnic/cultural groups: Aboriginal and Torres Strait Islanders, Pacific Islanders, people from Indian subcontinent, Chinese, Afro-Caribbeans

Screening (type 2)[4]

- People with known impaired fasting glucose/glucose tolerance (‘prediabetes’)
- Age >40 years, or younger age (e.g. >30 years) with: family history (first-degree relative with T2D), obesity (BMI >30), high-prevalence ethnic groups
- Age >18 years in Aboriginal and Torres Strait Islander people
- Previous gestational diabetes
- People on long-term steroids or antipsychotics

- Polycystic ovarian syndrome, especially if overweight
- Previous cardiovascular event

The optimal frequency is every 3 years from age 40 years using AUSDRISK (www.diabetesaustralia.com.au/are-you-at-risk-type-2). If score ≥12, do fasting blood glucose or HbA1c. Screen annually in very high-risk groups, including Aboriginal and Torres Strait Islander people and those with 'prediabetes'.[5]

Diagnosis

Diabetes is diagnosed as follows:[3,4]

1. If symptomatic (at least two of polydipsia, polyuria, frequent skin infections or frequent genital thrush):
   - fasting venous blood glucose (VBG) ≥7.0 mmol/L

     or

   - random VBG (at least 2 hours after last eating) ≥11.1 mmol/L

     or

   - HbAIc >6.5% (>48 mmol/mol)
2. If asymptomatic:
   - at least two separate elevated values, either fasting, 2 or more hours postprandial, or the two values from an oral glucose tolerance test (OGTT)

Note: If random or fasting VBG lies in an uncertain range (5.5–11.0 mmol/L) in either a symptomatic patient or a patient with risk factors (over 50 years, overweight, first-degree relative with T2D), perform an OGTT. The cut-off point for further testing is 5.5 mmol/L.[4,6]

The 2-hour blood sugar on an OGTT is still the gold standard for the diagnosis of uncertain diabetes, i.e. >11.1 mmol/L.

The OGTT should be reserved for true borderline cases and for diagnosing gestational diabetes, where a 75 mg OGTT is recommended at 24–28 weeks' gestation.[7]

Prediabetes

This is the condition where the VPG is elevated above the normal range (i.e. 6.1–6.9) but does not satisfy the type 2 diagnostic criteria. It includes two states:

- impaired fasting glucose (IFG)

- impaired glucose tolerance (IGT)

A diagnosis of prediabetes is not a call to start medication, but it increases the urgency of promoting lifestyle changes such as weight reduction and increased physical activity.

Urinalysis is unreliable in diagnosis since glycosuria occurs at different plasma glucose values in patients with different kidney thresholds.

For a summary of diagnosis of diabetic states, refer to FIGURE 11.2 .

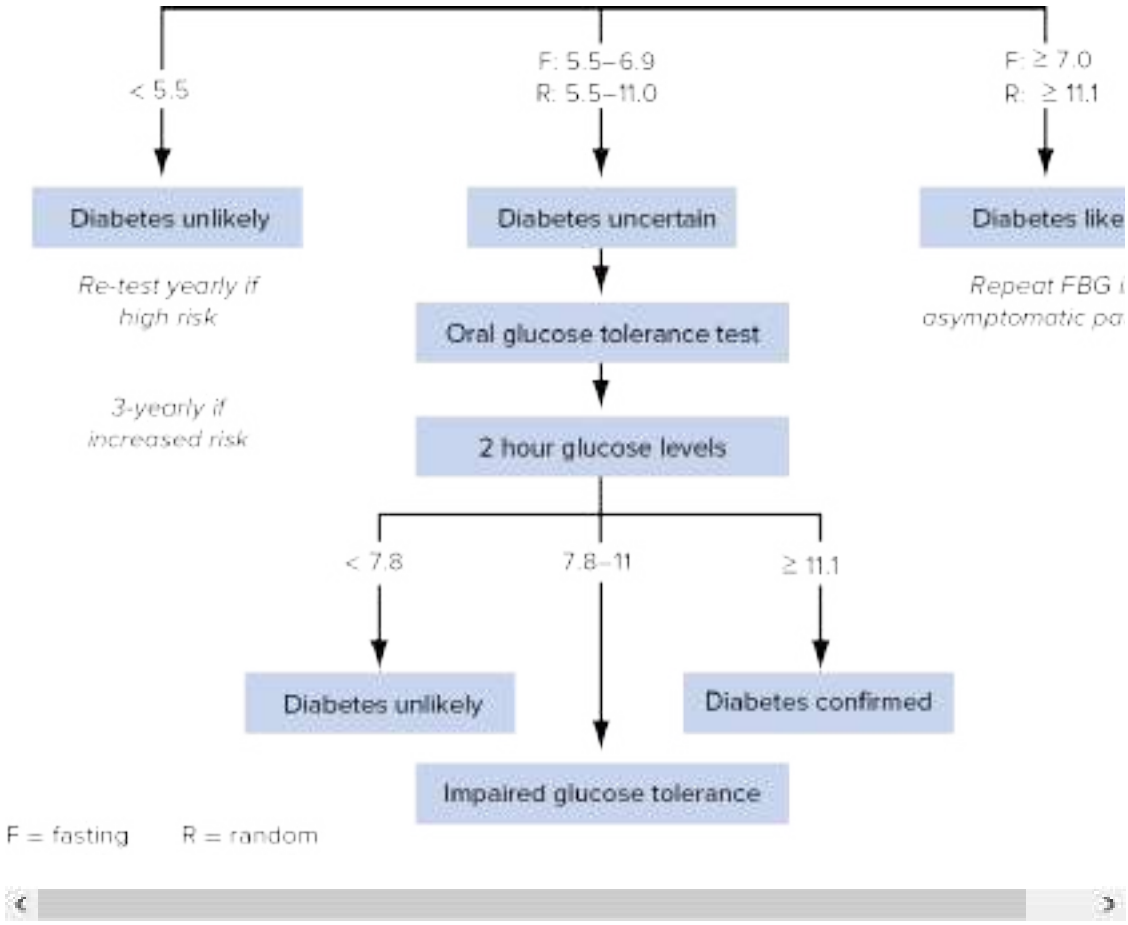

FIGURE 11.2 Blood glucose levels: venous plasma (mmol/L)

Source: Reproduced with permission from RACGP. General Practice Management of Type 2 Diabetes: 2016–18. East Melbourne, 2016 (book available from: http://www.diabetesaustralia.com.au or http://www.racgp.org.au).

Table 11.3 Interpreting diagnostic tests for diabetes[8]

Test	Normal	Intermediate hyperglycaemia	Diabetes

Venous blood glucose concentration	fasting	up to 6 mmol/L	6.1–6.9 mmol/L	≥7 mmol/L
	random	up to 6 mmol/L		≥11.1 mmol/L
Oral glucose tolerance test	2-hour venous blood glucose concentration	up to 7.7 mmol/L	7.8–11 mmol/L	≥11.1 mmol/L
HbA1c				≥48 mmol/mol

Diabetes in children

A study by Sinah and colleagues detected impaired glucose tolerance in 25% of 55 obese children (4 to 10 years of age) and 21% of 112 obese adolescents (11 to 18 years of age).[9] Type 2 diabetes was identified in 4% of obese adolescents. However, over 30% of newly diagnosed diabetes in children and adolescents is upon presentation with diabetic ketoacidosis. Children with type 1 diabetes usually exhibit the classic features of polyuria, polydipsia, weight loss and lethargy. Be aware of unusual presentations such as urinary disorders including enuresis or daytime wetting accidents when a misdiagnosis of urinary infection or some other condition is sometimes forthcoming. The diagnosis can be made by an elevated random or fasting blood sugar. Oral glucose tolerance tests are inappropriate in the very young. Upon diagnosis it is appropriate to refer the child or adolescent to a multidisciplinary diabetes team. The sick child with a high blood glucose is an emergency presentation of type 1 diabetes until proven otherwise.

Gestational diabetes

Gestational diabetes is the new onset of abnormal glucose tolerance during pregnancy. Pregnancy is diabetogenic for those with a genetic predisposition. All pregnant women should be screened at 24–28 weeks with a 75 g oral glucose tolerance test (OGTT). The definition of gestational diabetes mellitus (GDM) by the Australasian Diabetes in Pregnancy Society has widened considerably in the past two decades, and far more women are captured by the lower thresholds.[10] The 2014 consensus definition of gestational diabetes is a fasting plasma glucose of ≥5.1 mmol/L, or a post-75 g oral glucose load at 1 hour ≥10.0, or at 2 hours 8.5–11.0. Refer to CHAPTER 100 .

Diabetes in the elderly

The incidence of diabetes rises with age. The elderly have increased mortality and morbidity from the disease, but also are at increased risk from aggressive treatment regimens. Careful monitoring is required, especially with adverse drug effects aggravated by polypharmacy and comorbidities. Special issues include diet, foot care and postural hypotension.

Complications of diabetes

Complications may occur in patients with both type 1 and type 2 diabetes, even despite early diagnosis and treatment (see FIG. 11.3).

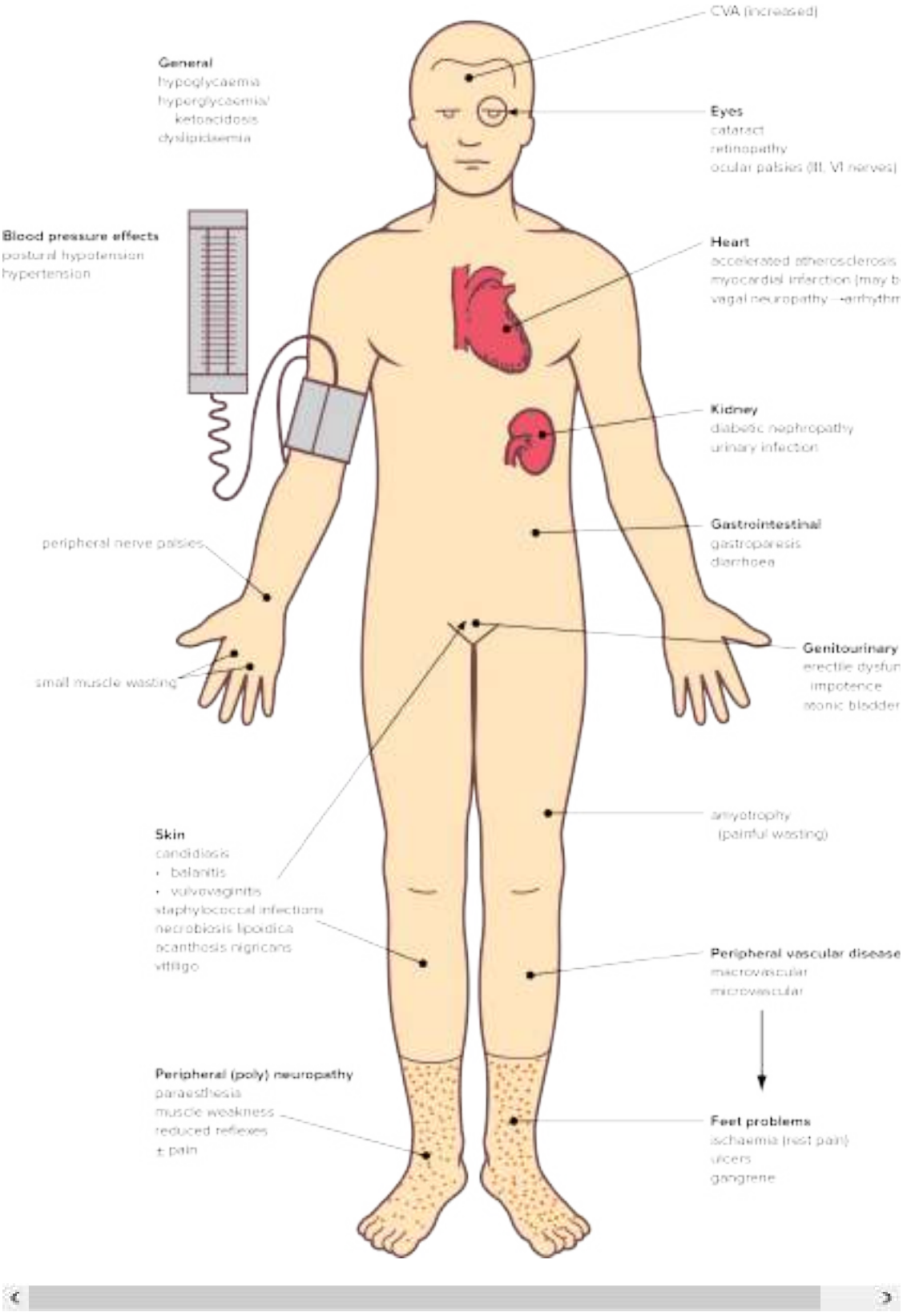

FIGURE 11.3 The complications of diabetes

People with type 1 diabetes still have a significantly reduced life expectancy. The main causes of death are diabetic nephropathy and vascular disease (myocardial infarction and stroke).

Diabetes causes both macrovascular and microvascular complications but microvascular disease is specific to diabetes. Special attention should be paid to the association of type 2 diabetes with obesity, hypertension and dyslipidaemia—the ‘deadly quartet’.[6,11]

Macrovascular complications include:

- ischaemic/coronary heart disease
- cerebrovascular disease
- peripheral vascular disease

An analysis of type 2 diabetes in the HOPE study[12,13] showed a benefit of ramipril to reduce the risk of:

- death (24%)
- myocardial infarction (22%)
- stroke (33%)
- cardiovascular death (37%)
- overt nephropathy (24%)

Consider organs/problems affected by diabetes under the mnemonic ‘KNIVES’:

- **K**idney
- **N**erves
- **I**nfection
- **V**essels
- **E**yes
- **S**kin

Microvascular disease

The small vessels most affected from a clinical viewpoint are the retina, nerve sheath and kidney glomerulus. In younger people it takes about 10 to 20 years after diagnosis for the problems of diabetic retinopathy, neuropathy and nephropathy to manifest.

Nephropathy

Prevention of diabetic nephropathy is an essential goal of treatment. Early detection of the yardstick, which is microalbuminuria, is important as the process can be reversed with optimal control, particularly of blood pressure. The dipstick method is unreliable and the preferred hospital method of 24-hour urine collection is considered impractical in general practice. Screening is done simply by a first morning urine sample to determine the albumin–creatinine ratio (see CHAPTER 79).

ACE inhibitors (or angiotensin II receptor blockers if a cough develops) should be used for evidence of hypertension.

Retinopathy and maculopathy

Retinopathy develops as a consequence of microvascular disease of the retina. Its prevalence is related to the duration of illness but up to 20% of people with type 2 diabetes have diabetic retinopathy at the time of diagnosis. The European multicentre study[14,15] showed that diabetes is the single most common cause of blindness in European adults in the 16–64 years age groups. Assessment of the fundus by an expert is recommended every 1–2 years, via direct ophthalmoscopy (with dilated pupils), retinal photography or, if necessary, fluorescein angiography. Early diagnosis of serious retinopathy is vital since the early use of laser photocoagulation may delay and prevent visual loss.

Neuropathy

The following types of neuropathy may occur:

- radiculopathy (diabetic lumbosacral radiculoplexopathy)
- sensory polyneuropathy
- isolated or multiple mononeuropathy

    isolated peripheral nerve lesions (e.g. median nerve)

    cranial nerve palsies (e.g. III, VI)

    amyotrophy
- autonomic neuropathy, which may lead to:

    erectile dysfunction

    postural hypotension and syncope

    impaired gastric emptying (gastroparesis)

diarrhoea

delayed or incomplete bladder emptying

loss of cardiac pain → ‘silent’ ischaemia

hypoglycaemic ‘unawareness’

sudden arrest, especially under anaesthetic

Infections

People with poorly controlled diabetes are prone to infections, especially:

- skin: mucocutaneous candidiasis (e.g. balanitis, vulvovaginitis), staphylococcal infections (e.g. folliculitis)
- urinary tract: cystitis (women), pyelonephritis and perinephric abscess
- lungs: pneumonia (staphylococcal, streptococcal pneumonia), tuberculosis

Diabetic metabolic complications

- Hypoglycaemia
- Diabetic ketoacidosis
- Hyperosmolar hyperglycaemia
- Lactic acidosis

Other complications

- Cataracts
- Refractive errors of eye
- Sleep apnoea
- Depression
- Musculoskeletal: neuropathic joint damage (Charcot-type arthropathy), tendon rupture
- Foot ulcers (related to neuropathy)

Prevention of diabetes

Several large studies have demonstrated it is possible to prevent or delay the onset of type 2 diabetes in those at risk.[14,15] This involves intensive lifestyle intervention in individuals who are overweight with impaired glucose tolerance or raised fasting blood glucose. The ongoing DiRECT trial demonstrates that even once type 2 diabetes has been present for a few years, remission of diabetes is a realistic aim in general practice, using intense dietary measures for 3 months followed by structured support for weight loss management.[16]

The primary strategy is to follow the SNAP guidelines (Smoking, Nutrition, Alcohol, Physical activity), particularly with a view to weight loss.[17] The essentials are healthy eating, weight loss and physical activity. This represents an important approach that GPs can recommend to their patients at risk. The enormous health gains that can be made in this prediabetic population by concentrating on SNAP outweigh any later health gains from diabetes medication (see CHAPTER 80).

Management of diabetes

The main objectives for the GP are to prevent the development of cardiovascular disease and other complications. Aim to achieve:[4]

1. reduction of 'lifestyle' risks—weight, smoking, low physical activity
2. strict glycaemic control as measured by HbA1c (target varies with circumstance, but usually ≤7%)
3. blood pressure control (≤140/90 mmHg, lower if tolerated)[18]
4. control of blood lipid levels

Note: Refer to the estimations of cardiovascular risk (see FIGS. 75.1 and 75.2 in CHAPTER 75).

Management principles

- Provide detailed and comprehensive patient education, support and reassurance.
- Achieve control of presenting symptoms.
- Achieve blood pressure control (huge impact on mortality risk).
- Develop a diabetes care plan.
- Emphasise the importance of the diet: good nutrition, adequate protein, complex carbohydrates (low carb diets are an option), restrict fats and sugars.
- Promptly diagnose and treat urinary tract infection.

- Treat and prevent life-threatening complications of ketoacidosis or hyperosmolar coma.
- Treat and prevent hypoglycaemia in those taking insulin and oral hypoglycaemic agents.
- Organise self-testing of blood glucose for those on insulin.
- Detect and treat complications of diabetes—neuropathy, nephropathy, retinopathy, vascular disease.
- Ensure immunisation schedule, including influenza and pneumococcus, is updated.

Monitoring techniques

- Blood glucose estimation (mainly useful if on insulin; fasting and postprandial)
- Urine glucose (of limited usefulness)
- Urine or blood ketones (for type 1 diabetes)
- Glycated haemoglobin (HbA1c) (3-monthly)
- Microalbuminuria (usually urine ACR, regarded as an early and reversible indicator of nephropathy)
- Blood pressure
- Serum lipid levels
- Kidney function (serum urea/creatinine eGFR)
- ECG

Control guidelines are summarised in FIGURE 11.4 and TABLE 11.4 .

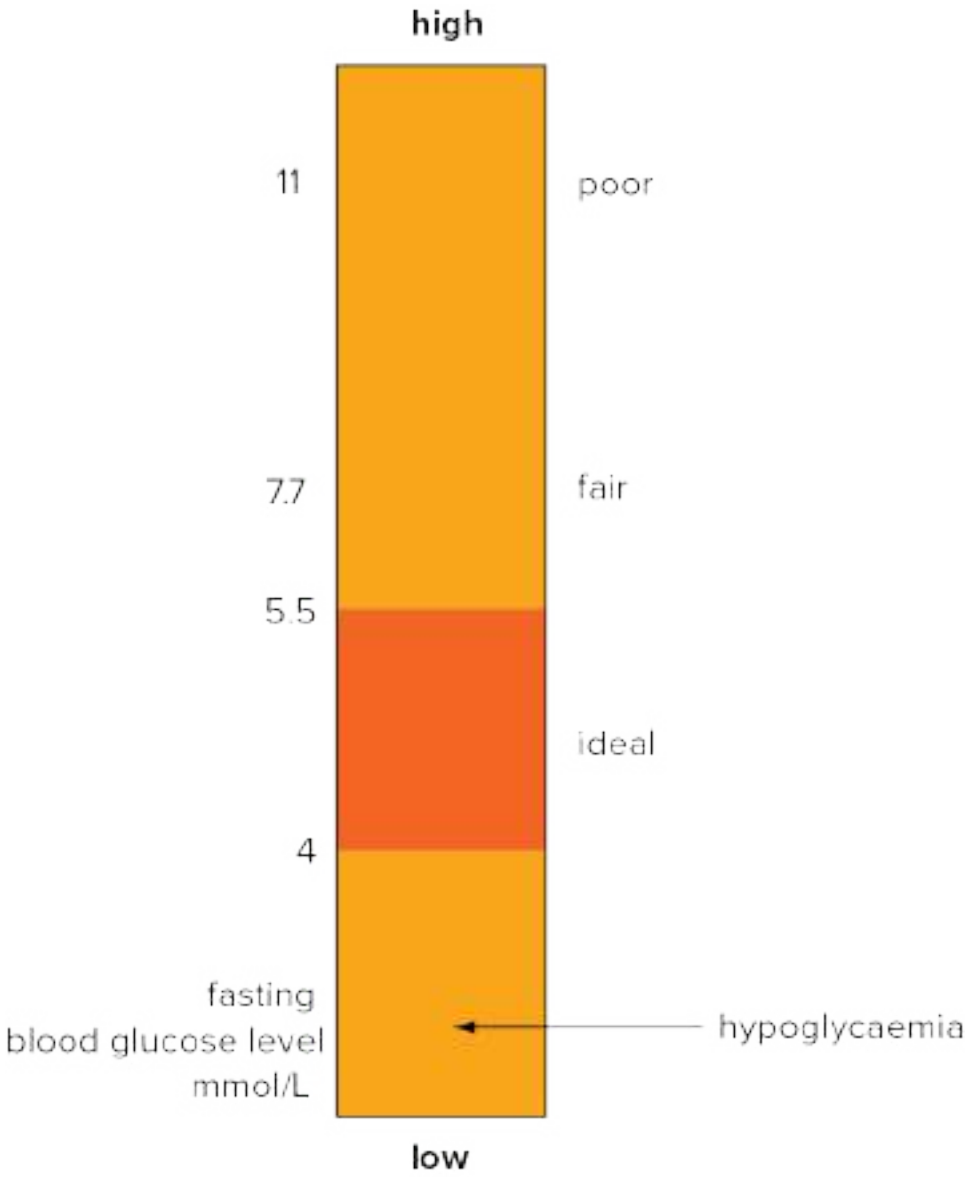

FIGURE 11.4 Blood glucose control guidelines for diabetes management

Table 11.4 Suggested guidelines for glycaemic control (blood glucose mmol/L)[4,5,19]

	Ideal	Suboptimal or unacceptable
Before meals (fasting)	<5.5	>7.7
After meals (2 hours postprandial)	<7	>11
HbA1c %*	≤7	>8

*HbA1c is an index reflecting the mean plasma glucose levels over the preceding 2–3 months (assume a reference range of 4.5–8%).

Blood glucose monitoring at home

This is done using a glucose meter (glucometer). A wide variety of meters and smart phone apps are available: patients will require advice about what suits them.

How often and when?

- Type 1 diabetes:

  four times a day (before meals and before bedtime) at first and for problems

  twice a day (at least once)

  may settle for 1–2 times a week (if good control)

- Type 2 diabetes:

  important for those on insulin, not routinely recommended for oral medication (monitor with HbA1c instead, in most circumstances)

  more useful for pregnant women, frail elderly, heavy machinery operators or symptomatic hypoglycaemia

Goals of management[4,5]

All people with diabetes should be encouraged to maintain the following goals for optimum management:

- Blood glucose (fasting) — ideal 4–6 mmol/L NHMRC 6–8 mmol/L
- Blood glucose (postprandial) — 8–10 mmol/L
- HbA1c — ≤7% (53 mmol/mol)
- Total cholesterol — <4.0 mmol/L
- LDL cholesterol — <2.0 mmol/L
- HDL cholesterol — ≥1.0 mmol/L
- Non-HDL-C — <2.5 mmol/L
- Triglycerides — <2.0 mmol/L
- Blood pressure

	<140/90 mmHg, lower if tolerated, esp. stroke risk ≤125/80 mmHg with proteinuria (1 g/day)
• BMI	18–25 where practicable
• Urinary albumin excretion	<20 mcg/min timed overnight collection <20 mg/L spot collection
• Albumin–creatinine ratio	<2.5 mg/mmol—men <3.5 mg/mmol—women
• Cigarette consumption	zero
• Alcohol intake	≤2 standard drinks, 20 g/day (men and women)
• Exercise	at least 30 minutes walking (or equivalent) 5 or more days/week (total 150 minutes/week)

Note:

- Capillary blood glucose is approximately 7% higher than venous blood.
- Glucometer error is usually ±5%.

Glycated haemoglobin

HbA1c, which normally comprises 4–6% of the total haemoglobin, is abnormally abundant in those with persistent hyperglycaemia, reflecting suboptimal metabolic control. Glycohaemoglobins have a long half-life and their measure reflects the mean plasma glucose levels over the past 2–3 months and hence provides a good method of assessing overall diabetes management. HbA1c should be checked every 3–6 months.

Type 1 diabetes

The three main objectives of the treatment of type 1 diabetes are:

- maintain good health, free from the problems of hyperglycaemia and hypoglycaemia
- achieve proper growth and maturation for children and protect the fetus and mother in a mother with type 1 diabetes

- prevent, arrest or delay long-term macrovascular and microvascular complications

Insulin regimens for type 1 diabetes[4,20]

The most commonly used insulin injection preparations are the 'artificial' human insulins. Insulins are classified according to their time course of action:

- rapid-acting and short duration (ultra-short)—insulin lispro, insulin aspart
- short-acting—neutral (regular, soluble)
- intermediate-acting—isophane (NPH) or lente
- long-acting—ultralente, insulin detemir, insulin glargine
- pre-mixed short/intermediate—biphasic (neutral + isophane)

Also: continuous subcutaneous insulin infusion.

Starting insulin[20]

For less experienced GPs, shared care with an endocrinologist is recommended.

It is important to use the simplest regimen for the individual and to provide optimal education about its administration and monitoring. Full replacement of insulin is achieved by using 2, 3 or 4 injections per day. However, automated glucose monitoring linked to 'smart' insulin pumps can make an injection decision every five minutes. See TABLE 11.5 for available insulins.

Table 11.5 Available insulins[4,20]

Type	Brand name
Ultra-short-acting (peak 1 hour, duration 3.5–4.5 hours)	
Insulin lispro	Humalog*
Insulin aspart	NovoRapid**
Insulin glulisine	Apidra*
Short-acting (peak 2–5 hours, duration 6–8 hours)	
Neutral (regular)	Actrapid**
	Humulin R*
	Hypurin Neutral*
Intermediate-acting (duration 12–24 hours)	

Isophane (NPH)	Humulin NPH*
	Protaphane**
	Hypurin Isophane
Long-acting (analogues)	
Insulin glargine (duration 24–36 hours)	Optisulin (Lantus)
Insulin detemir (duration up to 24 hours)	Levemir
Pre-mixed (short- and intermediate- or long-acting)	
Lispro 25%/Protamine 75%	Humalog Mix 25*
Lispro 50%/Protamine 50%	Humalog Mix 50*
Insulin aspart 30%/Protamine 70%	NovoMix 30**
Neutral 30%/Isophane 70%	Humulin 30/70*
	Mixtard 30/70**
Neutral 50%/Isophane 50%	Mixtard 50/50**

*Available in cartridges for use in pen injectors
**Available in cartridges for use in pen injectors or in disposable insulin pens

1. *The pre-mixed 2 injection (biphasic) system*
   Give twice daily, 30 minutes before breakfast and before evening meal (e.g. Mixtard 30/70, Humulin 30/70—the most common)

   - Typical starting dose: 0.3 IU/kg/day—for a 70 kg person use 10 units bd

2. *3 injections per day*

   - Short-acting insulin before breakfast and lunch
   - Intermediate- or long-acting insulin before evening meal

3. *4 injections (basal-bolus) system*

   - Short-acting insulin before breakfast, lunch and dinner (bolus)
   - Intermediate- or long-acting insulin at bedtime (basal)

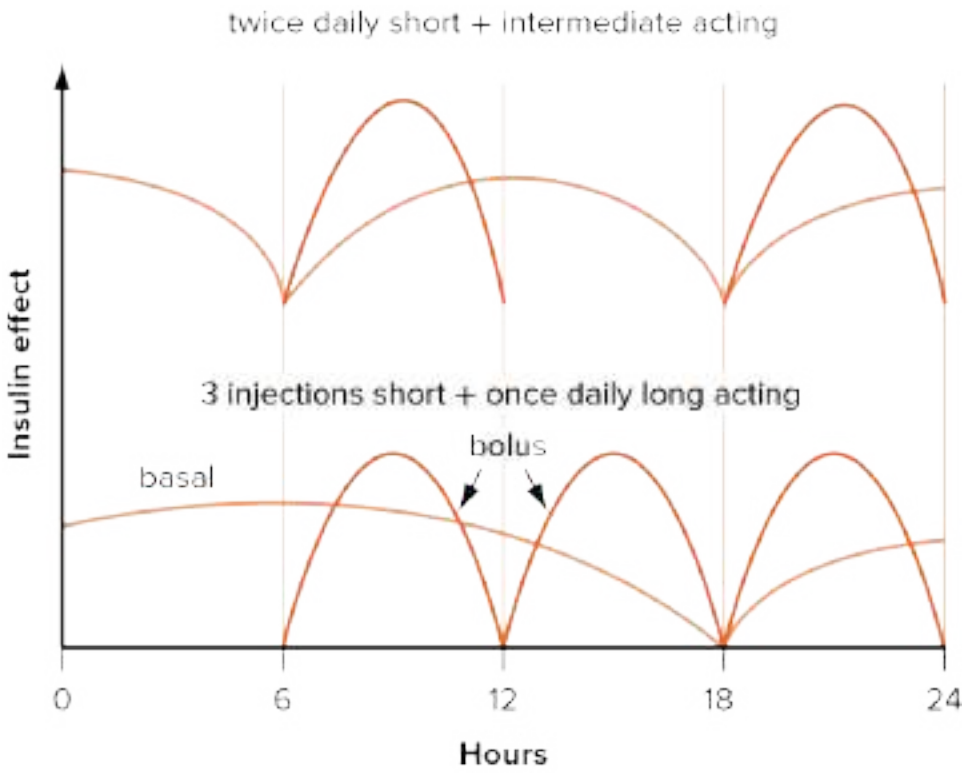

FIGURE 11.5 Illustration of time course of insulin injection regimens

Insulin requirements often vary significantly even in the same individual under different lifestyle conditions. The rapid-acting analogues can be taken with meals.

Methods of giving insulin injections

When

Suggest the patient develops a set routine, such as eating meals on time and giving the injection about 30 minutes before the meal.

Where

Into subcutaneous tissue—the best place is the abdomen. The leg is also acceptable. It is advisable to keep to one area (usually abdomen) and avoid injections into the arms, near joints and the groin. The injection should be given at a different place each time, at least 3 cm from the previous injection. This reduces the risk of the development of lipodystrophy. The means of delivery is the insulin syringe or the insulin delivery pen.

How

Pinch a large area of skin on the abdomen between the thumb and fingers and insert the needle

straight in. After withdrawing the needle, press down firmly (do not rub or massage) over the injection site for 30 seconds. Alcohol swabs are unnecessary.

Guidelines for the patient[4]

- Take your insulin every day, even if you feel ill.
- Do not change your dose unless instructed by your doctor or you are competent to do so yourself.

Problems

Injection sites should be inspected regularly because lipohypertrophy or lipoatrophy can occur.

Sick days

Have a prearranged action plan.

Never omit the insulin dose even if the illness is accompanied by nausea, vomiting or marked anorexia. More top-up insulin is usually required (rapid/fast acting).

Maintain glucose. Keep regular blood glucose checks (a concern if >15 mmol/L).

Seek support and help.

Sport

Encourage sporting activities. Careful planning (use expert help) and monitoring of blood sugar is required. Insulin doses may need to be adjusted before activities.

Additional carbohydrate may be needed.

Glycaemic targets for adults with type 1 diabetes

- HbA1c: 7% (53 mmol/mol)
- Blood glucose: fasting preprandial 4–7 mmol/L; postprandial 5–10 mmol/L

Type 2 diabetes[8,19]

First-line treatment (especially if obese):

- diet therapy
- exercise program
- weight loss

Most symptoms improve within 1–4 weeks on diet and exercise.[3] Prescribe and ask about exercise at every visit. Aim for an average of 20–30 minutes a day. Suggest variations such as social-type exercises. The secret to success is patient adherence through good education and supervision. The role of a diabetic education service, especially with a dietitian, can be invaluable. If unsatisfactory control persists after 3–6 months, consider adding an oral hypoglycaemic agent (see TABLE 11.6). The usual first-line agent is metformin, which reduces insulin resistance. If glycaemic targets are not achieved on monotherapy, usual practice is to add in a secretagogue, such as a sulfonylurea, which increases insulin production. However, the newer agents SGLT2 inhibitors (the gliflozins) and GLP-1 receptor agonists (injected) should be considered for their cardioprotective and renoprotective effects. DPP-4 inhibitors (oral gliptins) and thiazolidinediones (glitazones) are other options.

Table 11.6 Commonly prescribed non-insulin hypoglycaemic agents[8,20] (with examples)

Drug	Duration of action (hours)	Daily dose range	Notes including main generic adverse effects
Metformin (a biguanide)	12 (also slow-release daily dosage)	0.5–3 g	Side effects: • GIT disturbances (e.g. diarrhoea, a/n/v) • avoid in cardiac, hepatic and kidney disease (eGFR <30) • lactic acidosis, a rare but serious complication
Sulfonylureas			
Gliclazide	18–24	40–320 mg	Hypoglycaemia most common side effect

			Others: weight gain (common), rash and GIT (rare) Shorter acting sulfonylurea is preferred in elderly
Glipizide	16–24	2.5–40 mg	Longer acting potent ones cause troublesome hypoglycaemia in elderly
Glibenclamide	18–24	2.5–20 mg	
Glimepiride	>24	1–4 mg	
α-glucosidase inhibitors			
Acarbose	3	150–600 mg	Flatulence, skin rashes, diarrhoea, liver effects
Thiazolidinediones (glitazones)			Dubious mortality benefit. Caution with heart failure.
Pioglitazone	24	15–45 mg	Oedema, weight gain, heart failure
Rosiglitazone	24	4–8 mg	Hepatic effects, fracture risks
DPP-4 inhibitors (gliptins)			Slight pancreatitis risk
Sitagliptin	>24	25–100 mg	Rhinorrhoea, headache hypersensitivity, e.g. urticaria
Linagliptin	>24	5 mg	
Saxagliptin	24	5 mg	
Vildagliptin	>24	50–100 mg	Dizziness, fatigue
Alogliptin	>24	25 mg	
$SGLT_2$ inhibitors			Modest short-term efficacy
Empagliflozin	24	10–25 mg	

Dapagliflozin	24	5–10 mg	• genitourinary infections • dehydration, dizziness, hypoglycaemia
Ertugliflozin	24	5–15 mg	
GLP-1 agonists			Nausea, pancreatitis
Dulaglutide	1 week	1.5 mg	
Exenatide	12 hours 1 week (MR)	5 mcg bd 2 mg weekly	
Liraglutide	24 hours	0.6–1.8 mg daily	

Consider metformin as the first-line agent for all patients with type 2 diabetes, irrespective of their weight, unless contraindicated. The usual starting dose is 500 mg once or twice daily. It has proven benefits over the sulphonylureas, especially in those that are overweight. Other benefits include no significant weight gain, no hypoglycaemia and an improved lipid profile. If monotherapy does not provide adequate glycaemic control, a combination of metformin with another agent (see FIG. 11.6) is recommended.[20]

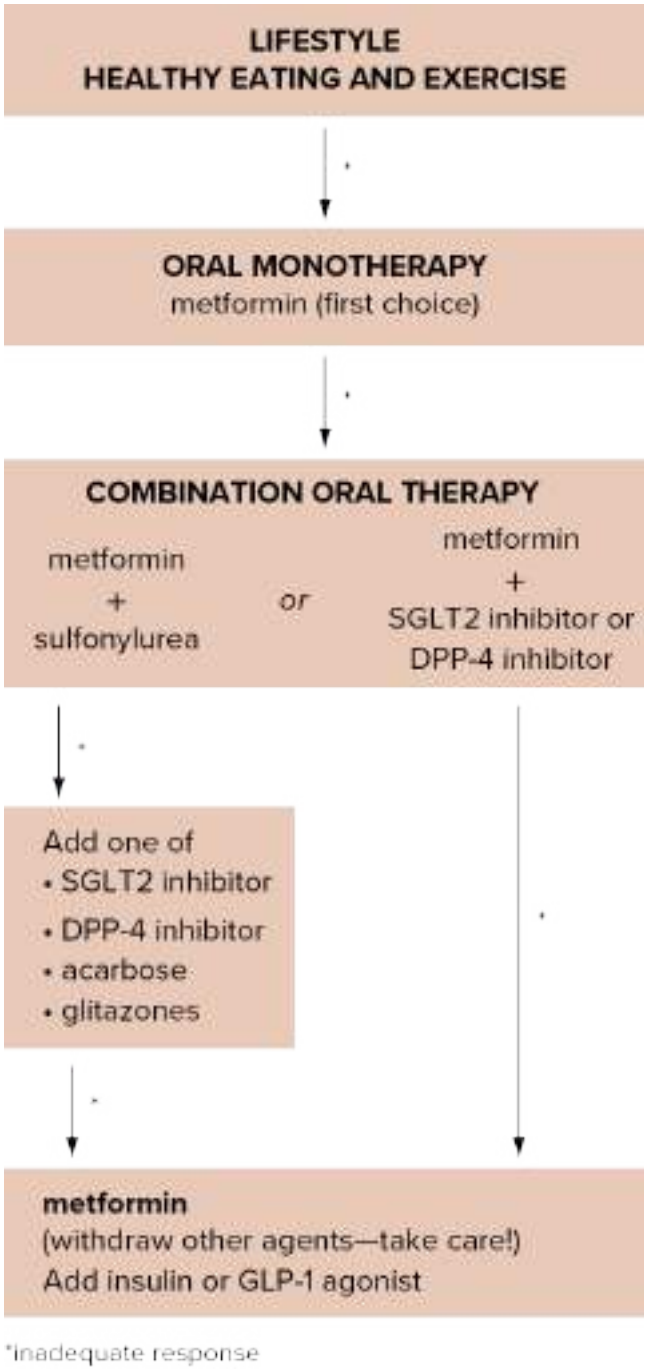

FIGURE 11.6 Step-up approach to management of type 2 diabetes[20]

When the first oral hypoglycaemics fail (secondary failure), a second agent can be added (usually sulfonylurea, DPP-4 inhibitor, SGLT2 inhibitor). Alternatives include GLP-1 receptor antagonists or insulin, and less commonly acarbose or a glitazone. The newer treatment options in type 2 diabetes include:[20]

- dipeptidyl peptidase-IV (DDP-4) inhibitors (gliptins, e.g. sitagliptin)
- sodium glucose cotransporter 2 (SGLT2) inhibitors taken orally, e.g. dapagliflozin, empagliflozin
- glucagon-like peptide-1 receptor (GLP-1) agonists (e.g. exenatide modified release—weekly dosing, liraglutide—daily dosing) given by SC injection. These improve satiety and are associated with weight loss. Nausea is fairly common, but tends to settle. Pancreatitis is a rare but important side effect.

Approximately 30% of those with type 2 diabetes eventually require insulin, although that figure may decrease with the availability of newer agents. An algorithm for the management of type 2 diabetes is presented in FIGURE 11.6 .

Remember that insulin is not a substitute for healthy eating and activity.

Starting insulin in type 2 diabetes[14,21]

Before commencing insulin one should be assured that the patient's lifestyle activities are being adequately addressed and that oral medication (at recommended maximum dose) is appropriate. There is no clear-cut rule about when to start insulin for those with HbA1c >7%, but this can be as early as when drug therapy does not provide adequate control. Two golden rules are 'don't delay initiating basal insulin' and then 'start low and go slow'.[21]

When commencing insulin, reassure the person that the injections are not as uncomfortable as finger pricks and that they will feel much improved with more energy.

It is appropriate to refer to your diabetic team for shared care at this point—when starting insulin.

Suggested stepwise approach[14,19]

Step 1

- Continue oral agents: metformin + sulfonylurea ± glitazone or acarbose or DPP-4 inhibitor (limited to 3).
- Add 10 units isophane insulin at bedtime.

Step 2

- Titrate insulin therapy according to fasting blood glucose (6 mmol/L).
- Increase insulin in about 4–5 U increments every 3–4 days (or more gradually).

Step 3

If larger or multiple daily doses of insulin are required (NPH or mixed regimen),

continue metformin but usually withdraw sulfonylureas, DPP-4s and thiazolidinediones.

Note: The combination of a glitazone and insulin has been shown to improve control of diabetes sometimes to the extent of being able to reduce insulin dosage. Consider using a glitazone, GLP1 agonist or SGLT2 inhibitor to reduce the insulin dose requirement. However, check PBS requirements for triple therapies.

The importance of diet and nutrition

Nutrition management is based on controlling weight, having a healthy eating plan and supplementing it with exercise. It is recommended to eat a wide variety from all food groups:

- protein 10–20%, fat 20–40%, carbohydrate 35–60%
- reduce fat, especially saturated fats, sugar and alcohol

People with type 1 diabetes often require three meals and sometimes regular snacks each day. People with type 2 usually require less food intake and restriction of total food intake.

Principles of dietary management

- Keep to a regular nutritious diet.
- Achieve ideal body weight.
- Reduce calories (kilojoules), particularly:
    - added sugar
    - dietary fat
- Follow the glycaemic index values (see: www.glycaemicindex.com).
- Increase proportions of vegetables, fresh fruit and cereal foods.
- Special diabetic foods are not necessary.
- ‘Whole foods’ are preferred to supplements.
- Qualitative diets are often more sustainable than quantitative diets (such as ‘exchanges’ or ‘portions’).

The importance of exercise[4,19]

Exercise is fundamental to good management. Exercise is any physical activity that keeps you fit. Good examples are brisk walking (e.g. 2 km per day), jogging, tennis, skiing and aerobics. Aim for at least 30 minutes three times a week, but daily exercise is ideal. Go slow when you

start and increase your pace gradually.

Psychosocial considerations

The psychological and social factors involving the patient are very influential on outcome. Considerable support and counselling may be necessary to help both patient and family cope with the ‘distress’ of the diagnosis and the discipline required for optimal control of their blood glucose. Reasons for poor dietary compliance and insulin administration must be determined and mobilisation of a supportive multidisciplinary network (where practical) is most helpful. The GP should be the pivot of the team. Encourage joining a self-support group where available.

Foot care

Foot problems are one of the commonest complications that need special attention; prevention is the appropriate approach. By international standards, Australia has an unenviably high rate of amputations in people with diabetes. Pressure sores can develop on the soles of the feet from corns, calluses, ill-fitting footwear, and stones and nails. Minor injuries such as cuts can become a major problem through poor healing. Infection of wounds is a major problem. Check the footwear.

Control of hypertension

Studies have highlighted the importance of blood pressure control to reduce macrovascular and microvascular complications in diabetes patients.[22] In fact, blood pressure control has more mortality benefits than blood glucose control. Try non-pharmacological measures first.

Preferred pharmacological agents are ACE inhibitors or ARBs and calcium-channel blockers.[5,19]

Getting to target blood pressure (<140/90 mmHg, lower if tolerated)*

Step 1: Diet, exercise, weight control

Step 2: ACEI or ARB

Step 3: ACEI/ARB and diuretic or calcium channel blocker

Step 4: Beta blocker

ARB = angiotensin II receptor blocker

*<125/80 mmHg if proteinuria >1 g/day (ACR >70) present

Current target recommendations vary according to guideline, but aim for blood pressure below 140/90 mmHg, and lower if tolerated, particularly in those with proteinuria or at high risk of stroke. Monitor for treatment-related adverse effects such as hypotension, syncope, electrolyte abnormalities and acute kidney injury, and review medication if any adverse events occur.[18]

The target may need to be relaxed for those at risk of postural hypotension, particularly the elderly.

Control of dyslipidaemia[4,19]

Mixed hyperlipidaemia is a common finding in patients with diabetes. Dyslipidaemia (especially hypercholesterolaemia) is an independent risk factor for the macrovascular complications of diabetes, and proper control is important. Non-pharmacological measures should be tried first. The preferred agents are HMG-CoA reductase inhibitors and resins for hypercholesterolaemia, and fibrates and resins for mixed hyperlipidaemia.

Targets should be:

- total cholesterol: <4 mmol/L
- triglycerides: <1.5 mmol/L
- HDL cholesterol: ≥1 mmol/L
- LDL cholesterol: <2.0 mmol/L

Management in summary[22]

The ABC of diabetic care is summarised in TABLE 11.7 . A key to ongoing control of diabetes is to maintain the HbA1c at or below 7%, and recognising that it is cardiovascular disease that causes most of the complications and excess mortality in type 2 diabetes. In patient review, the National Health and Medical Research Council (NHMRC) guidelines emphasise lifestyle review as step one.[19] A useful lifestyle evaluation mnemonic is NEAT:

- **N**utrition—eat less, reach ideal weight, healthy low fat/complex carbohydrate diet
- **E**xercise—including ‘walk more’, interesting physical activities
- **A**voidance of toxins—alcohol, tobacco, salt, sugar, illicit drugs
- **T**ranquillity—rest, recreation and stress reduction

Table 11.7 The ABC of diabetes care[3]

Risk factor	Target
Hb**A**1c	<7%
BP	<140/90*
Cholesterol	<4 mmol/L**
Smoking	Quit

*lower if tolerated
**corresponding to LDL cholesterol <2.0 mmol/L

Antihypertensives and statins have an important role in management. A meta-analysis of the use of low-dose aspirin (acetylsalicylic acid 75–150 mg/day) showed secondary risk reduction in people with diabetes and a history of a cardiovascular event (AMI or CVA).[23] However, aspirin is not indicated in people with diabetes who have not had a cardiovascular event.

Metabolic complications of diabetes

Hypoglycaemia

Hypoglycaemia is theoretically defined as blood glucose falling below 4.0 mmol/L, although symptoms usually start at <3.5 and become serious at <3.0.[24] It is most common with insulin use (especially type 1 diabetes but also type 2) and can occur on oral hypoglycaemic drugs, notably sulfonylureas (metformin hardly ever causes hypoglycaemia). It is appropriate to ask often about symptoms of hypoglycaemia: ‘recurrent hypoglycaemia begets hypoglycaemic unawareness’.

Clinical variations

1. Classic warning symptoms: sweating, tremor, palpitations, hunger, peri-oral paraesthesia
2. Rapid loss of consciousness, usually without warning
3. Coma: stuporose, comatose or ‘strange’ behaviour

- In alert patients able to swallow, give refined carbohydrate orally (15 grams, e.g. 7 jelly beans, 3 teaspoons sugar or honey, half glass soft drink or juice)
- Repeat BGL every 15 minutes. If <4, repeat above. If >4, give complex carbohydrate snack or meal (minimum 15 g, e.g. tub of yoghurt, slice of bread, piece of fruit)

Treatment (reduced conscious state or unconscious)[24]

Treatment of choice (after DRABC—call ambulance if unconscious)

30 mL 50% glucose slow IV push (instil rectally using the nozzle of the syringe if IV access

difficult). Usually 10 mL in children.

or

1 mL (= 1 ampoule) glucagon IM or SC (0.5 mL in child <25 kg)

When fully conscious, follow up with snack or meal. Admit to hospital if concerned. Ascertain cause of the hypoglycaemia and instruct the person how to avoid a similar situation in the future.

Diabetic ketoacidosis[24]

This life-threatening emergency requires intensive management. It usually occurs during an illness (e.g. gastroenteritis) when insulin is omitted. It can also occur in type 2 diabetes.

Clinical features

- Develops over a few days, but may occur in a few hours in ‘brittle’ diabetics
- Hyperglycaemia (often >20 mmol/L, lower or normal if on SGLT2 inhibitor)
- Preceded by polyuria, polydipsia, drowsiness
- Vomiting and abdominal pain, dehydration
- Hyperventilation—severe acidosis (acidotic breathing): ↓BP, ↑pulse, ↑resp. rate
- Ketosis (blood and urine)

Management

- Arrange urgent hospital admission
- Early IV fluids—normal saline fast first litre, then caution
- IV insulin—slow, e.g. 10 U in first hour
- ECG—arrhythmia in electrolyte disturbances

Tip: Diabetic ketoacidosis with coma requires fluid, sodium (eventually 3 L N saline), potassium (KCl) and insulin.

Hyperosmolar hyperglycaemia[4]

People with this problem may present with an altered conscious state varying from stupor to coma and with marked dehydration. The onset may be insidious over a period of weeks, with fatigue, polyuria and polydipsia. The key features are marked hyperglycaemia and dehydration

without ketoacidosis. It occurs typically in uncontrolled type 2 diabetes, especially in elderly patients. Sometimes they have previously undiagnosed diabetes. There may be evidence of an underlying disorder such as pneumonia or a urinary infection. The essential findings are extreme hyperglycaemia and high plasma osmolarity. The condition has a high mortality—even higher than ketoacidosis.

Treatment

- IV fluids, e.g. normal to ½ normal saline, given slowly
- Insulin—relatively lower doses than acidosis

Lactic acidosis[4,8]

Patients with lactic acidosis present with marked hyperventilation ‘air hunger’ and confusion. It has a high mortality rate and must be considered in the very ill person taking metformin, especially if kidney function is impaired. The risk of lactic acidosis is low if the therapeutic dose of metformin is not exceeded. Investigations reveal blood acidosis (low pH), low bicarbonate, high serum lactate, absent serum ketones and a large anion gap. Treatment is based on removal of the cause, rehydration and alkalinisation with IV sodium bicarbonate.

Other issues in diabetes

Erectile dysfunction[20]

The prevalence of erectile dysfunction in men with type 2 diabetes over 40 years may be as high as 50%. It may be caused by macrovascular disease, pelvic autonomic neuropathy or psychological causes. Those with organic-based ED may benefit from appropriate counselling and (if not taking nitrates) one of the phosphodiesterase inhibitors, starting with a low dose. The risk of cardiovascular disease needs to be evaluated.

Female sexual dysfunction

Autonomic dysfunction may result in reduced vaginal lubrication with arousal in women, but not the degree of sexual dysfunction that affects men. Appropriate education, reassurance and the use of lubricants should be helpful.

Postural hypotension[20]

Autonomic neuropathy-related postural hypotension may be compounded by medication, including antihypertensives and anti-angina agents. The usual strict blood pressure targets may need to be relaxed, particularly in the elderly. Persistent problems may be helped by graduated compression stockings to decrease venous pooling. If it continues to be a severe problem, the use of oral fludrocortisone may be helpful.

Gastroparesis

Symptoms of gastroparesis (due to autonomic neuropathy) with decreased gastric emptying include a sensation of fullness, dysphagia, reflux or recurrent nausea and vomiting, especially after meals. Treatment options include medication with domperidone, cisapride or erythromycin. Injections of botulinum toxin type A into the pylorus via gastroscopy may facilitate gastric emptying.

Diabetes and driving[4]

Diabetes may impair driving via hypoglycaemia (due to medication) or complications (particularly visual impairment). *Assessing Fitness to Drive 2016* (amended 2017) outlines the specific legal obligations of medical practitioners for assessing drivers of private and commercial vehicles. Drivers are obligated to provide details to the driver licensing authority and to their vehicle insurance company. In general terms, people controlled by diet alone have no restrictions for driving whereas those on insulin may obtain a conditional licence subject to annual or 2-yearly review. The main specific risk is hypoglycaemic episodes. Further details can be found at: www.austroads.com.au/drivers-and-vehicles/assessing-fitness-to-drive.

Contraception

Long-acting reversible contraceptives (e.g. Implanon, Mirena) or the combined oral contraceptive pill are appropriate options for birth control in women not interested in permanent sterilisation. Bear in mind the possibility of polycystic ovarian syndrome.

The future[4,20]

- Use of immunosuppressants and immunomodulators for type 1 diabetes
- Increased availability of glucagon-like and amylin-like peptides for type 2 diabetes
- Continuous implantable venous glucose monitoring
- Closed-loop sensor-and-insulin-delivery devices (‘the artificial pancreas’)
- Combination ‘type 2 polypill’
- Inhaled insulin
- Transplantation:

    combined kidney/pancreas

    islet cells

Treatment errors and pitfalls[19]

- Avoid prescribing oral hypoglycaemic agents prematurely. Allow a reasonable trial of diet and exercise for type 2 patients, especially if they are overweight.
- Review the need for continued oral therapy after 3 months of treatment.
- Glucose tolerance tests should be avoided if the diagnosis can be made on the basis of symptoms and fasting, or random blood sugar or HbA1c (a glucose load carries a small risk of hyperosmolar coma).
- Keep an eye on the development of ketones in type 1 diabetes by checking urinary ketones and, if present, watch carefully because diabetic ketoacidosis is a life-threatening emergency.

When to refer[20]

- Type 1 diabetes requires specialist evaluation and then 1- to 2-yearly review
- Type 2 diabetes: depends on the GP's comfort level and experience. Particularly consider referral for:

  young people

  those requiring insulin

  those with complications
- For ophthalmological screening: every 2 years to inspect retina (or use retinal photography)
- Those with treatable complications, including:

  retinopathy

  nephropathy

  neuropathy: test annually

Shared care

The management of the person with diabetes provides an ideal opportunity for shared care between a cooperative team comprising the patient, the GP and the specialist diabetic team. The objective is to encourage patients to attend their own doctor for primary care and be less reliant on hospital outpatient services or the diabetic clinics. A well-coordinated arrangement with good communication strategies provides optimal opportunities for the ongoing education of the patient, the GP and the specialist diabetic team.

Practice tips

- Many cases of type 2 diabetes remain undiagnosed, so vigilance is important.
- Follow-up programs should keep to a prepared format. Example formats are presented in TABLES 11.8 and 11.9 .
- Hyperglycaemia is a common cause of tiredness. If elderly people with type 2 diabetes are very tired, think of hyperglycaemia and consider giving insulin to improve their symptoms.
- The management of the person with diabetes is a team effort involving family members, a nurse education centre, podiatrists, domiciliary nursing service, GP and consultant.
- If a person with diabetes (particularly type 1) is very drowsy and looks sick, consider first the diagnosis of ketoacidosis.
- Foot care is vital: always examine the feet when the person comes in for review.
- Treat associated hypertension with ACE inhibitors or a calcium-channel blocker (also good in combination).
- Use a team approach and encourage joining special support groups (e.g. Diabetes Australia).
- 'Never let the sun go down on pus in a diabetic foot'—admit to hospital.[20]
- If a foot ulcer hasn't healed in 6 weeks, exclude osteomyelitis. Arrange for an MRI and investigate the vasculature.
- Prevention/detection of coronary heart disease should be an integral part of all consultations.

Table 11.8 Diabetes control: 3-monthly review

Discourage smoking and alcohol
Review symptoms
Review nutrition
Check weight (BMI), BP, urine
Review self-monitoring

Review exercise and physical activity
Review HbA1c (test at least every 6 months)
Review lipid levels (test every 12 months)

Table 11.9 Diabetes control: an annual review program[4]

1 History
Smoking and alcohol use
Symptoms of hypoglycaemia, hyperglycaemia
Check symptoms relating to eyes, circulation, feet*
Immunisation
2 Examinations
Weight, height, BMI
Blood pressure—standing and lying
Examine heart*
Carotid and peripheral pulses*
Eyes: • visual acuity (Snellen chart) • ?cataracts • optic fundi (or ophthalmologist referral)* • ?diabetic retinal photography
Tendon reflexes and sensation for peripheral neuropathy*
Skin (general)
Foot examination including footwear*
Check injection sites
Urine examination: albumin, ketones, glucose
3 Review biochemical levels

*These items comprise a program for detection of long-term complications. They should be conducted annually, commencing 5 years after diagnosis.

Patient education resources

Hand-out sheets from *Murtagh's Patient Education* 8th edition:

- Diabetes

- Diabetes: blood glucose monitoring at home
- Diabetes: foot care for diabetics
- Diabetes: healthy diet for diabetes
- Diabetes: insulin injections
- Diabetes: type 1
- Diabetes: type 2

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12 Drug and alcohol problems

A custome loathsome to the eye, hateful to the nose, harmeful to the braine, dangerous to the lungs and the blacke stinking fume thereof, neerest resembling the horrible Stigian smoke of the bottomless pit.

JAMES I (1566–1625), *ON SMOKING*

Ecstasy: a drug so strong it makes white people think that they can dance.

LENNY HENRY (1958–)

If you want to keep a dead man, put him in whisky; if you want to kill a live man put whisky in him.

THOMAS GUTHRIE (1803–1873)

Drug-related problems are true masquerades in family practice. This includes prescribed drugs, over-the-counter drugs and social or illegal street drugs. It is important therefore that all prescribing doctors maintain a high index of suspicion that any clinical problem may be associated with their treatment of the patient.

Adverse drug reactions

An adverse drug effect is defined as ‘any unwanted effect of treatment from the medical use of drugs that occurs at a usual therapeutic dose’. Almost every drug can cause an adverse reaction, which must be elicited in the history. Any substance that produces beneficial therapeutic effects may also produce unwanted, adverse or toxic effects. The severity of the reaction may range from a mild skin rash or nausea to sudden death from anaphylaxis. A study has shown that the incidence of adverse reactions increases from about 3% in patients 10–20 years of age to about 20% in patients 80–89 years of age.[1]

Reactions can be classified in several ways—side effects, overdosage, intolerance, hypersensitivity and idiosyncrasy. However, a useful classification of unwanted effects is divided into type A and type B.

Type A reactions are the most common and involve *augmented pharmacology;* that is, they are caused by unwanted, albeit predictable, effects of the drug. Examples include:

- constipation due to verapamil
- blurred vision and urinary outflow problems due to tricyclic antidepressants
- hyperuricaemia due to thiazide diuretics

Type A reactions are dose-dependent.

Type B reactions are idiosyncratic. The reactions are unpredictable from known properties of the drug. Examples include hepatotoxicity and blood dyscrasias.

Golden rules for prevention of adverse effects

Before prescribing any drug the prescriber should consider the following rules:

1. Is the drug really necessary?
2. What will happen if it is not used?
3. What good do I hope to achieve?
4. What harm may result from this treatment?

Common adverse effects

There is an extensive list of clinical problems caused by drugs as side effects or interactions that are highlighted throughout this book. Common side effects include:

- CNS—malaise, drowsiness, fatigue/tiredness, headache, dizziness
- CVS—palpitations, peripheral oedema, hypotension
- GIT—nausea, vomiting, dyspepsia, change in bowel habit (diarrhoea, constipation)
- skin—rash, pruritus, flushing
- psychiatric/emotional—insomnia, irritability, anxiety, depression, agitation

Drugs that commonly produce adverse effects

- Antidepressants (number 1 cause): tricyclics, MAOIs, SSRIs
- Antimicrobials: penicillin/cephalosporins, sulfonamides, tetracyclines, streptomycin, ketoconazole

- Anticonvulsants: carbamazepine, phenobarbitone, phenytoin, sodium valproate
- Anti-inflammatories and analgesics: aspirin/salicylates, opioids (e.g. codeine, morphine), NSAIDs, gold salts, DMARDs, bDMARDs

- Antihypertensive agents: several
- Cardiac agents: digoxin, quinidine, amiodarone, other antiarrhythmics
- Diuretics: thiazides, frusemide
- Tranquillisers: phenothiazines, benzodiazepines, barbiturates, chlordiazepoxide
- Other drugs: cytotoxics, hormones, allopurinol, warfarin

Tobacco use

'Smoking is good for you', according to an old Arab proverb. 'The dogs will not bite you because you smell so bad; thieves will not rob you at night because you cough in your sleep and you will not suffer the indignities of old age because you will die when you are relatively young.'

Tobacco smoking is the largest single, preventable cause of death and disease in Australia. It has been estimated to have caused approximately 15 000 deaths in 2004–2005, over six times the number of deaths from road accidents.[2] Diseases attributed to smoking are summarised in FIGURE 12.1 . Signs of major dependence are smoking within 30 minutes of waking and ≥20 cigarettes a day.

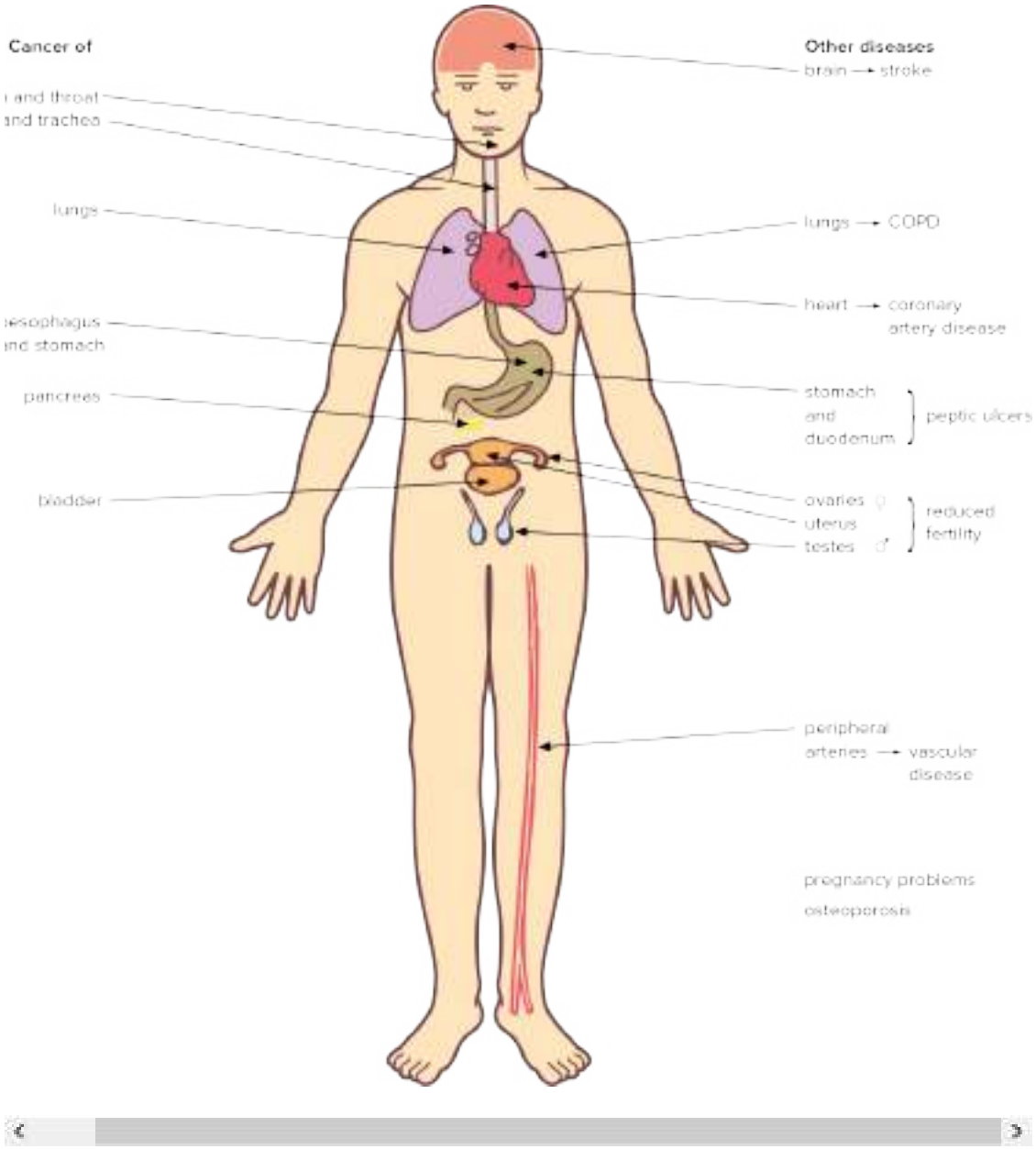

FIGURE 12.1 Possible serious adverse effects of nicotine smoking

Advice to patients (quitting)

Several studies have highlighted the value of opportunistic intervention by the family doctor. It is important not only to encourage people to quit but also to organise a quitting program and follow-up. In Australia, 80% of smokers (representing about 30% of the adult population) have indicated that they wish to stop smoking. Point out that it is not easy and requires strong will power. As Mark Twain said, ‘Quitting is easy—I’ve done it a thousand times.’

- Educate patients about the risks to their health and the many advantages of quitting smoking,

and emphasise the improvement in *health*, *longevity*, *money savings*, *looks* and *sexuality*.

- Point out the following advantages to quitting:

  food tastes better

  sense of smell improves

  exercise tolerance is better

  sexual pleasure is improved

  bad breath improves

  risk of lung cancer drops: after 10–15 years of quitting it is as low as someone who has never smoked

  early COPD can be reversed

  decreases URTIs and bronchitis

  chance of premature skin wrinkling and stained teeth is less

  removes effects of passive smoking on family and friends

  removes problem of effects on pregnancy

- The extent of nicotine dependence can be assessed using a questionnaire (based on the Fagerström test) and scoring system.[3] As a baseline, ask about the number of cigarettes smoked per day, how soon after waking to smoking the first cigarette of the day and any difficulties with coping with antismoking venues (e.g. cinemas, plane travel).

Intervention: the 5A framework[3]

- *Ask* about and document tobacco use at every opportunity.
- *Assess* motivation and confidence to quit: 'Are you interested in quitting?'
- *Advise* all smokers to quit (in a diplomatic way).
- *Assist* the smoker to stop with counselling and pharmacotherapy.
- *Arrange* follow-up to maintain quit advice or non-smoking.

- Ask them to keep a smoker's diary.

- If they say no to quitting, give them motivational literature and ask them to reconsider.
- If they say yes, make a contract (example below).

A contract to quit

'I ..... agree to stop smoking on ..... I understand that stopping smoking is the single best thing I can do for my health and that my doctor has strongly encouraged me to quit.'

..... (Patient's signature)

..... (Doctor's signature)

- These motivated patients will require educational and behavioural strategies to help them cope with quitting. Ongoing support by their GP is very important.
- Organise joining a support group.
- Contact your local Quitline (or similar service) for information about and support for quitting, especially if smoking ≥20 cigarettes a day.
- Arrange follow-up (very important), at least monthly, especially during first 3 months.

- Going 'cold turkey' (stopping completely) is preferable but before making the final break it can be made easier by changing to a lighter brand, inhaling less, stubbing out earlier and reducing the number. Changing to cigars or pipes is best avoided.

Withdrawal effects[3]

The initial symptoms are restlessness, cravings, hunger, irritability, poor concentration, headache and frustration (refer to TABLE 12.1). After about 10 days most of these effects subside but it takes about three months for a smoker to feel relatively comfortable with not smoking any more. Nicotine replacement therapy certainly helps patients cope.

Table 12.1 Nicotine withdrawal symptoms (DSM-5)

1. Irritability, frustration or anger
2. Anxiety
3. Difficulty concentrating
4. Increased appetite

5. Restlessness
6. Depressed mood
7. Insomnia

Treatment

Pharmacological[3]

Nicotine replacement therapy (NRT), which should be used in conjunction with an educational support program, has been proved to be effective and is available as chewing gum, inhaler, oral spray, lozenges, sublingual tablets or transdermal patches (the preferred method). Ideally the nicotine should not be used longer than 3 months. Eight weeks of patch therapy is as effective as longer courses.

NRT should be directed at smokers with a moderate to high nicotine dependency who are motivated to quit. There is little evidence that drug treatment will benefit individuals with low levels of nicotine dependence who smoke fewer than 10 cigarettes a day.[4]

All forms of NRT are effective: a pooled analysis of all NRT trials showed an absolute increase in cessation at 1 year of 7% compared to placebo.[4]

NRT should start at the quit date, not while still smoking.

The RACGP Expert Advisory Group for Supporting Smoking Cessation strongly recommend the use of NRT varenicline and bupropion with high certainty, and nortriptyline with moderate certainty.[5]

Nicotine gum[3]

This is available as 2 mg and 4 mg.

- Low dependence (less than 10 cigarettes per day): use non-pharmacological methods rather than replacement
- Moderate dependence (10–20 cigarettes per day): 2 mg every 1–2 hours, chew max. 8–12 pieces daily
- High dependence (>20 per day, waking at night to smoke or first thing after waking): 4 mg initially, 6–10 pieces chewed daily changing to 2 mg after 4–8 weeks (max. 12 pieces/24 hours)

  Useful points:

- Chew each piece slowly for about 30 minutes.

- Ensure all the nicotine is utilised.
- Chew at least 6 pieces per day, replacing at regular intervals (not more than 1 piece per hour).
- Use for 3 months, weaning off before the end of this period.

Transdermal nicotine[3]

This is available as 16-hour or 24-hour nicotine patches in three different strengths. The patients should stop smoking immediately on use.

Recommendations:

- low to moderate dependence (10–20 cigarettes/day): 15 mg/24 hour or 10 mg/16 hour patch, daily; aim to cease within 12 weeks
- high dependence (>20/day): 21 mg/24 hour or 15 mg/16 hour patch; change to 15 mg or 10 mg patch after 4–6 weeks; aim to cease within 12 weeks; use lower dose if patient is <45 kg or has CVS disease

Apply to non-hairy, clean, dry section of skin on upper outer arm or upper chest and leave in place for 24 hours. Rotate sites with a 7-day gap for reuse of a specific site.

Nicotine inhaler

Uses cartridges (15 mg) in a mouthpiece resembling smoking.

- 6/day for 12 weeks then taper

Nicotine oral spray

- 1 mg/spray: 1–2 sprays into mouth orally (max. 64 sprays/24 hours)

Nicotine lozenges and sublingual tablets

These are available in 2 mg and 4 mg strengths, the strength used according to the level of dependence, e.g. high dependence: suck 4 mg lozenges (max. 15/24 hours), or 4 mg SL every 1–2 hours (max. 80 mg/24 hours).

Combination therapy

Controlled trials have shown enhanced outcomes when nicotine patches are combined with gum or inhaler. Consider it for highly addicted smokers.

Other agents for smoking cessation[6]

Bupropion (Zyban)

This oral agent has a similar effectiveness to NRT.

Adverse effects include insomnia and dry mouth (both common), with serious effects, such as allergic reactions and increased seizure risk.[7] It is contraindicated in persons with a history of epilepsy.

Recommended dose: 150 mg (o) daily for 3 days then bd for 12 weeks.

Varenicline tartrate (Champix)[3,6]

- Commence with 0.5 mg (o) daily with food for 3 days titrating slowing to 1 mg bd by day 7 until the end of the 12-week course

It is an effective agent but there are several adverse side effects, especially nausea with a concern about neuropsychiatric effects.[7] Avoid in end-stage kidney disease and take care with diabetics.

Nortriptyline

- Start with 25 mg (o), increasing gradually to 75 mg (o) daily, starting 14 days before quit date then continue for 12 weeks

Note: Regular follow-up for all methods is essential for outcome.

Vaping

This involves inhaling and exhaling the vapour from 'electronic' e-cigarettes. Its use is controversial, especially since the addition of nicotine e-liquids ± other substances have led to serious adverse effects including death. Currently, nicotine e-cigarettes are allowed to be imported and purchased under a doctor prescription.[8] The RACGP Expert Group concludes 'conditional recommendation for intervention—low certainty'.[5]

Excessive and harmful drinking

Excessive drinking of alcohol is one of the most common and socially destructive problems in the world. One survey found that 5% of Australian men and 1% of women were alcohol-dependent. It also showed that 86% of men and 79% of women drink alcohol, with 8.3% of the population drinking alcohol every day.[7]

- Alcohol is estimated to have a harmful effect on about 1 in 10 people.
- At least 20–40% of acute general and psychiatric hospital admissions have an alcohol-related illness.
- About 20%-plus of fatal traffic accidents involve alcohol.

- The author's study[9] identified alcohol dependence in 9.7% of the population studied and a further group of problem drinkers that included the 'explosive' or binge drinker (see FIG. 12.2). Problem drinkers represent about 15–20% of the population.

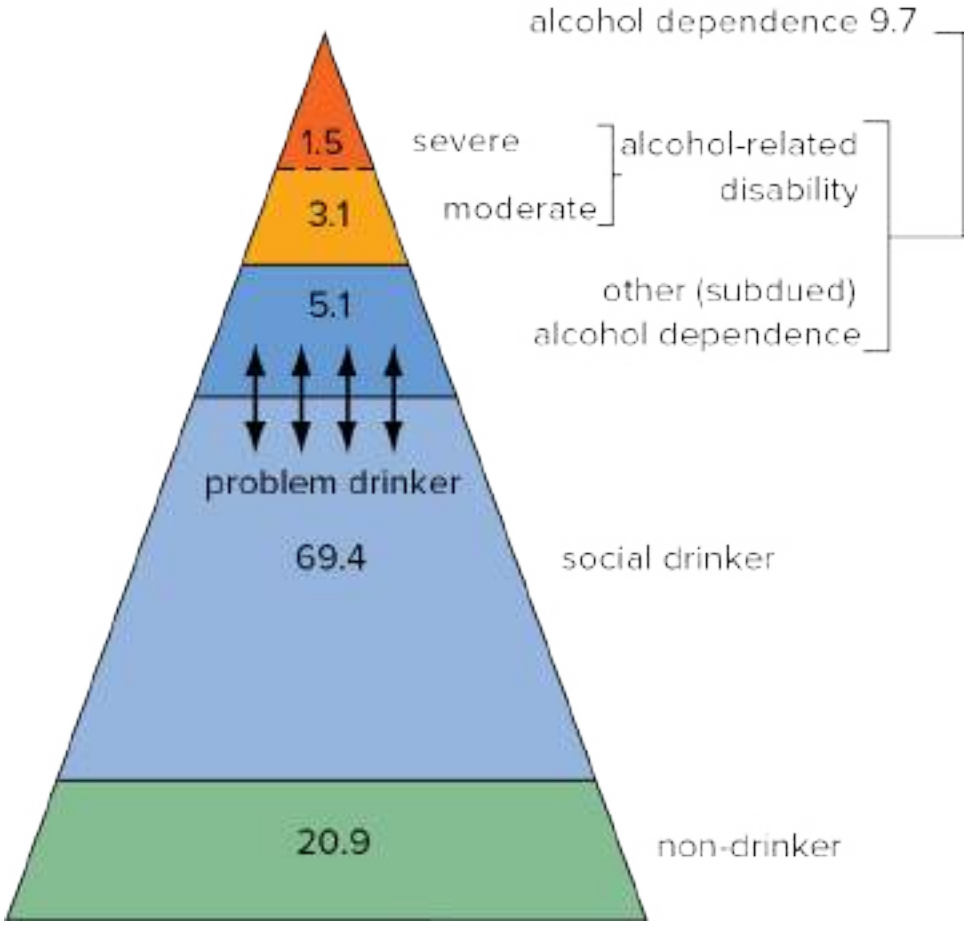

FIGURE 12.2 Prevalence of alcohol drinking patterns in the adult population (figures expressed as a percentage)

National Health and Medical Research Council (NHMRC) revised guidelines addressing harmful drinking are presented in TABLE 12.2 .[10,11]

Table 12.2 Recommended guidelines to reduce health risks from drinking alcohol, NHMRC 2020[10]

Guideline 1 Reducing the risk of alcohol-related harm over a lifetime For healthy men and women, drinking no more than 10 standard drinks a week reduces the lifetime risk of harm from alcohol-related disease or injury.
Guideline 2 Reducing the risk of injury on a single occasion of drinking

Healthy men and women should drink no more than 4 standard drinks on a single occasion, and then should not drink at all for 2–3 days.

Guideline 3 Children and young people under 18 years of age should not drink alcohol

Guideline 4 Maternal alcohol consumption can harm the developing fetus or breastfeeding baby

a. Women who are pregnant or planning a pregnancy should not drink alcohol.
b. For women who are breastfeeding not drinking is safest for their baby.

High-risk and harmful drinking occurs at >6 standard drinks (SDs) a day (average) for men and >4 SDs for women.

The main causes of alcohol-related deaths are road trauma, cancer and alcoholic liver disease.[10]

Clinical pointers to alcohol abuse

Facial features of the patient can be a helpful pointer, albeit of the more advanced drinker. These include:

- plethoric facies
- puffy ‘greasy’ facies
- telangiectasia
- rosacea + rhinophyma
- suffused (‘bloodshot’) conjunctivae
- prominent lower lip with cheilitis of corners of mouth
- smell of stale alcohol or very ‘minty’ sweet breath (masking effect)

Taking a drinking history

This requires tact and skill and it must be noted that many problem drinkers considerably understate the level of their intake.

Useful strategies[9]

- Ask questions as part of a matter-of-fact enquiry into health risk factors, such as smoking and diet.
- Place the onus of denial on the patient by asking questions such as ‘When did you

last drink alcohol?’ rather than ‘Do you ever drink alcohol?’

- Record your patient’s intake quantitatively in terms of standard drinks or grams of alcohol.
- Confirm the history by enquiring about the time spent drinking per day and expenditure on alcohol.

Useful questionnaires

There are several questionnaires that can be most helpful, assuming the patient is fully cooperative. Two or more positive replies for the CAGE questionnaire[12] are suggestive of a problem drinker.

1. Have you ever felt you should CUT down on your drinking?
2. Have people ANNOYED you by criticising your drinking?
3. Have you ever felt bad or GUILTY about your drinking?
4. Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover? (an EYE-OPENER)

Laboratory investigations

The following blood tests may be helpful in the identification of excessive chronic alcohol intake:

- blood alcohol
- serum GGT: elevated in chronic drinkers (returns to normal with cessation of intake)
- MCV: >96 fL

Other changes:

- abnormal liver function tests (other than GGT)
- carbohydrate-deficient transferrin (quite specific—dependent on an enzyme induced by alcohol)
- HDLs elevated
- LDLs lowered
- serum uric acid elevated

Measuring alcohol intake

One standard drink contains 10 g of alcohol, which is the amount in one middy (or pot) of standard beer (285 mL), two middies of low-alcohol beer or five middies of super-light beer. These are equal in alcohol content to one small glass of table wine (122 mL), one glass of sherry or port (60 mL), or one nip of spirits (30 mL) (see FIG. 12.3).

FIGURE 12.3 Standard drinks

- 1 stubbie or can of full-strength beer = 1.4 standard drinks
- 1 light beer = 0.9 standard drinks
- 1 × 750 mL bottle of beer = 2.6 standard drinks
- 1 × 750 mL bottle of wine = 7 standard drinks

Alcohol dependence

Alcohol dependence is a syndrome in which an individual demonstrates clinically significant impairment or distress as manifested by three or more of the following, occurring at any time in the same 12-month period:

1. tolerance
2. withdrawal
3. drinking in larger amounts or for a longer period than intended
4. unsuccessful attempts to cut down or control drinking
5. a great deal of time spent in activities necessary to obtain, use or recover from the effects of alcohol

6. important social, occupational or recreational activities reduced or given up because of drinking

7. continued drinking despite knowledge of having persistent or recurrent problems caused by or exacerbated by drinking

Approach to management

The challenge to the family doctor is early recognition of the problem. There are specific target areas which should be considered carefully by the GP. Several studies have shown that early intervention and brief counselling by the doctor are effective in leading to rehabilitation.[13] Some of the results are very revealing.

- Patients expect their family doctor to advise on safe drinking levels.
- They will listen and act on our advice.[14]
- Treatment is more effective if offered before dependence or chronic disease has developed.[14]

Of prime concern to the GP is the assessment of whether the patient is interested in changing his or her excessive drinking behaviour. The proposed model of change by Prochaska and DiClemente helps identify the stage reached by the patient (see FIG. 12.4).[15]

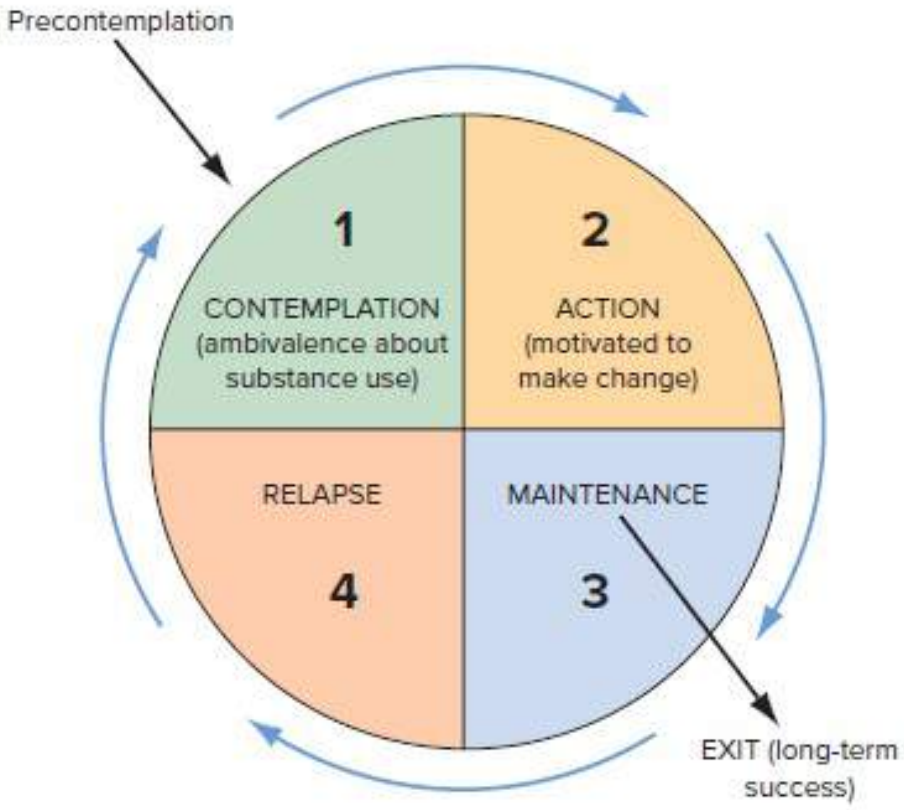

FIGURE 12.4 Prochaska and DiClemente's[15] proposed model of change to facilitate the identification of behavioural stages and the provision of counselling for treating dependence on alcohol, tobacco and other drugs

Precontemplators are satisfied users who are either unconcerned about their drinking or have no desire to change. However, if there is any evidence of ambivalence or concern about drinking, then the opportunity exists for motivational interviewing techniques.

Patients are not likely to offer concern about their drinking problem spontaneously but are often receptive to the initiative coming from their doctor.

The family doctor is ideally placed to identify and treat the problem of alcohol because the individual who abuses alcohol will tend to surface at some point in the provision of primary health care.

A brief practical management plan[16]

Giving patients feedback about their level of alcohol consumption, presenting objective evidence of harm and setting realistic goals for reducing alcohol intake induces many to change their drinking behaviour.

A six-step management plan, which has been employed in a general-practice early intervention program, is as follows:

1. Feed back the results of your assessment and specifically the degree of risk associated with their daily alcohol intake and bout drinking. Emphasise any damage that has already occurred.
2. Listen carefully to their reaction. They will need to vocalise their feelings and may respond defensively.
3. Outline the benefits of reducing drinking (e.g. save money, better health, weight loss).
4. Set goals for alcohol consumption that you both agree are feasible. In most cases this will involve reduction to below certain 'safe limits'.
    - For men: aim for fewer than 12 SDs per week.
    - For women: aim for fewer than 8 SDs per week. It is best for pregnant women not to drink.
    - For patients with illness who are physically dependent on alcohol, long-term abstinence is advisable.
5. Set strategies to keep below the upper safe limits, e.g.:
    - quench thirst with non-alcoholic drinks before having an alcoholic one
    - switch to low-alcohol beer
    - take care which parties you go to: avoid constant parties and other high-risk situations
    - explore new interests—fishing, cinema, social club, sporting activity
6. Evaluate progress by having patients monitor their drinking by using a diary; check that any abnormal blood test results are returning to normal. Make a definite appointment for follow-up and give appropriate literature such as *Alcohol: Harmful Use of Alcohol.* Obtain consent for a telephone follow-up. A useful minimum intervention plan is presented in TABLE 12.3 .

Table 12.3 Minimum intervention technique plan (5–10 minutes)

1. Advise reduction to safe levels
2. Outline the benefits
3. Provide a self-help pamphlet
4. Organise a diary or other feedback system
5. Obtain consent for a telephone follow-up
6. Offer additional help (e.g. referral to an alcohol and drug unit or to a support group)

Follow-up (long consultation 1 week later)

Review the patient's drinking diary. Explore any problems, summarise, listen and provide support and encouragement. If appointment is not kept, contact the patient.

Specialist services

According to progress and the patient's wishes and consent, specialist treatment units, group therapy and attendance at meetings of Al-Anon or AA are potential sources of help to keep the alcohol-dependent person abstinent and coping.

'Anti-craving' drugs

The following show a modest effect on assisting abstinence:

- acamprosate 666 mg (o) tds (if ≤60 kg)
- naltrexone 50 mg (o) daily (under close supervision)
- consider a combination of the above 2 drugs

Note: Disulfiram can be helpful in highly motivated people but its use, as for the above agents, is recommended under specialist advice.

Withdrawal symptoms

Symptoms of a 'hangover' include headache, nausea, irritability, malaise and a mild tremor. Withdrawal from alcohol in a chronic problem drinker includes:

- agitation/anxiety
- prominent tremor
- sweating
- insomnia
- seizures (occasionally)
- delirium tremens (DTs)

The aim of treatment for acute withdrawal symptoms is to prevent development of DTs. Maintain fluid, electrolytes and nutrition. Add vitamin B complex, including thiamine, because the patient is invariably thiamine deficient.

If medication is required (specialised advice):

- diazepam 20 mg (o) every 2 hours (up to 100 mg (o) daily, although 60 mg is usually adequate) titrated against clinical response (taper off after 2 days) in the hospitalised or well-supervised patient
- thiamine 100 mg IM or IV daily for three days, then 300 mg (o) daily for several weeks
- vitamin B group supplement IM daily
- for psychotic features add haloperidol 1.5–5 mg (o) bd or 5 mg IM as single dose if necessary

Alcohol withdrawal delirium (delirium tremens)

DTs is a serious life-threatening withdrawal state. It has a high mortality rate if inadequately treated and hospitalisation is always necessary.

Clinical features

- May be precipitated by intercurrent infection or trauma
- 1–5 days after withdrawal (usually 3–4 days)
- Disorientation, agitation
- Clouding of consciousness
- Marked tremor
- Visual hallucinations (e.g. spiders, pink elephants)
- Sweating, tachycardia, pyrexia
- Signs of dehydration

Treatment

- Hospitalisation with alcohol specialist advisory service
- Correct fluid and electrolyte imbalance with IV therapy
- Treat any systemic infection
- Thiamine (vitamin B1) 300 mg IM or IV daily for 3–5 days, then thiamine 300 mg (o) daily
- Diazepam 20 mg (o) every 2 hours (up to max. 100 mg daily) until symptoms subside. This dose is usually required for 2–3 days, then should be gradually reduced until finished. If psychotic features (e.g. hallucinations and delusions), add haloperidol 0.5–2 mg (o) bd every 2 hours, titrated to clinical response (max. 10 mg/24 hours).

Note: Chlorpromazine is not recommended because of its potential to lower seizure threshold. Diazepam and haloperidol may worsen the symptoms of hepatic toxicity.

Alcohol overdose

Overdose is potentially fatal. The average lethal blood alcohol concentration is about 0.45–0.5%. Death from a lower concentration may occur with other sedative drugs. Alcohol withdrawal may begin at 0.1%. Treatment of overdose is supportive and symptomatic. No stimulants should be given. Overdose may cause hypoglycaemia and metabolic acidosis.

Hangover

A type of acute drug toxicity causing headache, nausea and fatigue.

Prevention

- Drink alcohol on a full stomach.
- Select alcoholic drinks that suit you: avoid champagne.
- Avoid fast drinking—keep it slow.
- Restrict the quantity of alcohol.
- Dilute your drinks.
- Avoid or restrict smoking while drinking.
- Drink three large glasses of water before retiring.

Treatment

- Drink ample fluids especially water because of relative dehydration effect of alcohol.
- Take two paracetamol tablets for headache.

- Drink orange juice or tomato juice, with added sugar.
- A drink of honey in lemon juice helps.
- Tea is a suitable beverage.
- Have a substantial meal but avoid fatty food.

Illicit drugs

Several psychotropic substances are used for their effects on mood and other mental functions. Many of the severe problems are due to withdrawal of the drug. Symptomatic behaviour common to illicit drugs includes:

- rapid disappearance of clothing and personal belongings from home
- signs of unusual activity around hang-outs and other buildings
- loitering in hallways or in areas frequented by addicts
- spending unusual amounts of time in locked bathrooms
- inability to hold a job or stay in school
- rejection of old friends
- using the jargon of addicts

Illicit substance abuse

The drugs described below and in TABLES 12.4 and 12.5 are all commonly abused. Cannabis was the most widely used illicit drug in Australia in 2019, and was more frequently used than other illicit drugs.

Cocaine use increased from 2.5% in 2016 to 4.2% in 2019.[17] This includes crack, which is a cocaine base where the hydrochloride has mostly been removed, usually in a microwave oven. Crack can be inhaled or smoked (see FIGS 12.5 and 12.6). However, use of ice, which is the crude form of methamphetamine, a derivative of amphetamine (FIG. 12.6), was the main form of meth/amphetamine drug use during 2019. Speed is dexamphetamine.

FIGURE 12.5 ‘Meth mouth’ in a young man actively smoking methamphetamine

FIGURE 12.6 Methamphetamine ice with pipe

Party drugs

Ecstasy is another ‘designer’ drug which is an amphetamine derivative—methylenedioxy-methamphetamine (MDMA). It has high abuse potential, some hallucinogenic properties and a tendency to neurotoxicity, as proved on PET brain scans. It is popular in rave parties. Deaths have occurred, reportedly in association with relative dehydration or excessive hydration. Treatment for overdosage involves correction of fluid and electrolyte disturbances. An increasingly popular drug is fantasy (gamma-hydroxybutyrate), which has sedative and anaesthetic effects similar to alcohol. A popular party drug, it is implicated as a ‘date rape’ drug. There is no specific antidote.[16] Another party drug is ketamine, which is a short-acting anaesthetic with hallucinogenic properties. It can produce nausea and vomiting if used with alcohol. Like fantasy, treatment of overdosage is symptomatic. Local anaesthetics can be dangerous in amphetamine users because of cardiotoxicity. The most common party drugs reportedly used for drink spiking are alcohol, flunitrazepam (Rohypnol), GHB (fantasy), ecstasy, LSD and ketamine.

The drug list

In 2019, 16.4% of Australians had used an illicit drug in the past 12 months. The most commonly used drugs were:[18]

- cannabis 11.6%
- cocaine 4.2%
- non-medical use of pharmaceuticals (including opiods, benzodiazepines) 4.2%
- ecstasy 3%

- hallucinogens 1.6%
- inhalants 1.4%
- meth/amphetamine 1.3%
- ketamine 0.9%
- injected drugs 0.3%

Source: National Drug Strategy Household Survey 2019

A summary of the effects of illicit or hard street drugs is presented in TABLE 12.4 .

Table 12.4 Illicit substance abuse: a summary of hallmarks

Drug	Physical symptoms	Look for	Dangers
Amphetamines including methamphetamines (3 forms) • speed—powder • base—oily paste • ice—crystalline	Aggressive or agitated behaviour; giggling; silliness; euphoria; rapid speech; fever; confused thinking; anorexia; insomnia; extreme fatigue; dry mouth; shakiness; anxiety	Jars of pills of varying colours; chain smoking; white powder and crystals can also be snorted or injected	Hypertension; death from overdose; hallucinations; paranoia; may cause temporary psychosis; stroke; cardiac arrest
Ecstasy (methylene-dioxymethamphetamine)	Anxiety; panic; sweating; 'loving' feelings; jaw clenching, teeth grinding; bizarre overactive behaviour; hallucinations; increased heart rate, BP and body temperature; confidence; feelings of happiness and love	Small tablets of various colours, shapes, sizes and designs; also comes in powder and capsules	Convulsions; risk of death from heart attack, cerebral haemorrhage, hyperthermia, fluid imbalance with hyponatraemia, acute kidney failure, DIC, liver toxicity; hangover; depression
Fantasy (gamma-hydroxybutyrate)	Relaxation and drowsiness; dizziness; relaxed inhibition/euphoria; increased sexual arousal; impaired mobility and speech	Colourless, odourless liquid; also powder and capsules	Tremors and shaking; amnesia; coma; convulsions; death from high doses
Barbiturates	Drowsiness; stupor; dullness; slurred speech; drunk appearance; vomiting	Pills of various colours	Death from overdose or as a result of withdrawal; addiction; convulsions
Cannabis/marijuana	Initial euphoria; floating feeling; sleepiness; lethargy; wandering mind;	Strong odour of burnt leaves; small seeds in pocket lining;	Inducement to take stronger narcotics; recent medical findings reveal

	enlarged pupils; lack of coordination; craving for sweets; changes of appetite; memory impairment; tacchycardia	cigarette paper; discoloured fingers	that prolonged usage causes cognitive defects, precipitates or exacerbates schizophrenia; hyperemesis
Volatile substances including glue, solvents or petrol sniffing	Aggression and violence; drunk appearance; slurred speech; dreamy or blank expression; hallucinations; ataxia	Tubes of glue; glue smears; large paper or plastic bags or handkerchiefs	Lung/brain/liver damage; death through suffocation or choking
LSD (lysergic acid diethylamide)	Severe hallucinations; feelings of detachment; incoherent speech; cold hands and feet; vomiting; laughing and crying	Cube sugar with discolouration in centre; strong body odour; small tube of liquid	Suicidal tendencies; unpredictable behaviour; chronic exposure causes brain damage; LSD causes chromosomal breakdown
Narcotics (a) opioids (e.g. heroin)	Stupor/drowsiness; marks on body; watery eyes; loss of appetite; running nose; constricted pupils; loss of sex drive; agitation; hypoventilation	Needle or hypodermic syringe; cotton; tourniquet—string, rope, belt; burnt bottle, caps or spoons; bloodstain on shirt sleeve; glass in envelopes	Death from overdose; respiratory depression; mental deterioration; destruction of brain and liver; hepatitis; embolisms
(b) cocaine	Similar effects to amphetamines—muscle pains, irritability, paranoia	Powder: in microwave ovens; inhaled, snorted or	Hallucinations; death from overdose—sudden death from

	paranoia, hyperactivity, jerky movements, euphoria, dilated pupils	snorted or injected	from arrhythmias; seizures; mental disorders; severe respiratory problems

A list of street drugs and their slang names is presented in TABLE 12.5 .

Table 12.5 A street drug dictionary

Amphetamines or uppers	
Benzedrine	Roses, beanies, peaches
Dexedrine	Dexies, speed, hearts, pep pills, fast, go-ee, uppers, sulphate
Methamphetamines	Meth, crystals, white light, ice, whiz
Drinamyl	Purple hearts, goof balls
Amphetamine derivatives	
Ecstasy	E, eggs, eckies, XTC, ‘the love drug’, Mitsubishis, MDMA, vitamin E, X, Adam, death
Crank	Crystal M, crank
Hallucinogens	
LSD	Acid, blue cheer, strawberry fields, barrels, sunshine, pentagons, purple haze, peace pills, blue light, trips
Cannabis (Indian hemp)	Hash, resin
1 Hashish (the resin)	Pot, tea, grass, hay, weed, locoweed, Mary Jane, rope, bong, jive, Acapulco gold
2 Marijuana (from leaves)	Reefers, sticks, muggles, joints, spliffies, head, smoko, ganga
Cigarettes	Blow a stick, blast a joint, blow, get high, get stoned
Smoking pot	

Narcotics	
Morphine	Morph, Miss Emma
Heroin	H, Big H, Big Harry, GOM (God's own medicine), crap, junk, horse dynamite (high-grade heroin), lemonade (low-grade heroin). Injection of dissolved powder: mainlining, blast, smack. Inhalation of powder: sniffing
Cocaine	Coke, snow, lady of the streets, nose candy, ICE, snort, C, flake, rock, blow, vitamin C, crack, shabu, baby
H & C	Speed balls
Oxycontin	Hillbilly heroin
Miscellaneous	
Fantasy	GBH (grievous bodily harm), liquid G, liquid E, liquid ecstasy, liquid X, fantasy
Barbiturates	Devils, barbies, goof balls
Benzodiazepines	Rowies, moggies
Ketamine	'K', vitamin K, special K, K hole
Solvents	Chroming

Opioid (narcotic) dependence

This section will focus on heroin dependence, although opioids such as codeine and controlled dose agents such as oxycodone and morphine are problematic.

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Typical profile of a heroin-dependent person[19]

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- Male or female: 16–30 years
- Family history: often severely disrupted, such as parental problems, early death, separation, divorce, alcohol or drug abuse, sexual abuse, mental illness, lack of affection
- Personal history: low threshold for toleration, unpleasant emotions, poor academic record, failure to fulfil aims, poor self-esteem
- First experiments with drugs are out of curiosity, and then regular use follows with loss of job, alienation from family, finally moving into a 'drug scene' type of lifestyle

Methods of intake

1. Oral ingestion
2. Inhalation (see FIG. 12.5)
   - intranasal
   - smoking
3. Parenteral
   - subcutaneous
   - intramuscular
   - intravenous (see FIG. 12.7)

FIGURE 12.7 Intravenous heroin injection signs: linear tracks and scarring from repeated venepunctures along the course of a vein. Less common sites are the lower leg, dorsum of foot, neck and dorsal vein of penis.

Photo courtesy John Jagoda

Opioid withdrawal effects[19,20]

These develop within 12 hours of ceasing regular usage. Maximum withdrawal symptoms usually occur between 36 and 72 hours and tend to subside after 10 days.

- Anxiety and panic
- Irritability
- Chills and shivering
- Excessive sweating
- ‘Gooseflesh’ (cold turkey)
- Loss of appetite, nausea (possibly vomiting)
- Lacrimation/rhinorrhoea
- Tiredness/insomnia
- Muscle aches and cramps
- Abdominal colic
- Diarrhoea

A secondary abstinence syndrome is identified[19] at 2–3 months and includes irritability, depression and insomnia.

Complications of opioid dependence

Medical

- Overdose reaction: agitation, respiratory depression—may include fatal cardiopulmonary collapse. Since the early 2000s opioid deaths have fallen from peak levels of the 1990s, when there was a glut of heroin.
- Injection site: scarring, pigmentation, thrombosis, abscesses, ulceration (especially with barbiturates)
- Distal septic complications: septicaemia, infective endocarditis, lung abscess, osteomyelitis, ophthalmitis
- Viral infections: hepatitis B, hepatitis C (refer to CHAPTER 47), HIV infection (refer to CHAPTER 18)
- Neurological complications: transverse myelitis, nerve trauma
- Physical disability: malnutrition

Social

- Alienation from family, loss of employment, loss of assets, criminal activity (theft, burglary, prostitution, drug trafficking)

Management

Management is complex because it includes the medical management not only of physical dependence and withdrawal but also of the individual complex social and emotional factors. The issues of impaired liver function, hepatitis B and C and HIV prevention also have to be addressed. Sociological tests for these illnesses should be considered.

Patients should be referred to a treatment clinic and then a shared-care approach can be used. The treatments include cold turkey (abrupt cessation) with pharmacological support, acupuncture, high doses of vitamin C, methadone substitution and drug-free community education programs.

Maintenance programs that include counselling techniques are widely used for heroin dependence. Acute toxicity requires injections of naloxone.

Opioid withdrawal[20]

Buprenorphine controlled withdrawal (short term) is used to prevent the emergence of a withdrawal syndrome in contradistinction from buprenorphine maintenance, where there is an extended treatment period.

Initial dose

- buprenorphine 4–8 mg (sublingual) as a single daily dose, increasing to 12 mg (max) on day 3, then reduce gradually over the next 3–5 days

Note: If autonomic signs, use clonidine 5–15 mg/kg/day (o) in 3 divided doses for 7–10 days then taper off. If anxiety and agitation, use diazepam 5–20 mg (o) qid (with care). Clonidine can be used as first-line treatment because of relative safety but buprenorphine is preferred to clonidine and methadone for the management of opioid withdrawal. Avoid benzodiazepines unless supervision is available.

Maintenance programs for long-term opioid dependence[20]

There are currently three alternative programs—methadone, buprenorphine and naltrexone—which are substitutes for heroin and other opioids. Seek specialist advice for the management of these drugs.

Methadone

Seek specialist advice before starting treatment. The dose needs to be determined individually according to past use and initial response to methadone.

- methadone 20 mg (o) daily initially. Stabilise dose over 3 weeks. Beware of doses >40 mg, especially in unwell patients. Maintenance 50–80 mg (o) daily. Usual maximum dose 120 mg.

Buprenorphine

- buprenorphine 2–8 mg sublingual, once daily initially, increase to 8–24 mg daily or alternative days once stabilised. It is less dependent and prone to overdose than methadone but can precipitate withdrawal if used too soon.

Naltrexone

Care is required in giving naltrexone to a person physically dependent on opioids. A naloxone challenge test is used.[20] If no evidence of withdrawal give:

- naltrexone 25 mg (o) initially, increasing to 50 mg daily on day 2 if tolerated. Careful supervision with appropriate counselling is required.

The natural history of opioid dependence indicates that many patients do grow through their period of dependence and, irrespective of treatments provided, a high percentage become rehabilitated by their mid-30s.

Stimulant substance abuse

The stimulants include amphetamines and their analogues, ephedrine, designer drugs such as MDMA and ‘fantasy’, cocaine and certain appetite suppressants. The amphetamines include the common methamphetamine, dexamphetamine and the original amphetamines. Another disturbing drug is the stimulant ‘monkey dust’, a synthetic cathinone, also known as ‘bath salts’ or MDPV. It is similar to the amphetamine designer drug ‘meow-meow’ (mephedrone). These agents can induce a psychosis with dangerous behaviour including irrational risk taking and violent behaviour.

Stimulant-induced syndrome[20]

- Aggressive behaviour
- Paranoid behaviour
- Irritability
- Transient toxic psychosis
- Delirium
- Schizophrenic-like syndrome
- Increased sexual behaviour

Treatment

- Withdrawal of drugs

- Cognitive behaviour therapy
- Handle person carefully and respectfully
- No firm evidence on effectiveness of drugs

Stimulant-withdrawal syndrome[20]

This syndrome should be suspected in people whose occupation involves shift work, interstate transport driving or multiple jobs presenting with the following symptoms:

- drowsiness
- hypersomnia, then insomnia
- irritability
- hyperphagia
- aggressive behaviour
- depression/dysphoria; may last months
- urge to resume drugs

Treatment

- Psychological support and encouragement, e.g. CBT
- Desipramine (or similar tricyclic antidepressant) 75 mg (o) nocte (increasing as necessary)
- Bromocriptine 1.25 mg (o) bd has also been used for cocaine withdrawal

Hallucinogen abuse

Hallucinogens in use include lysergic acid diethylamide (LSD), phencyclidine (angel dust), the tropical plant products (Kava and Betel nuts) and many synthetics. Symptoms include psychotic behaviour, including severe hallucinations. Withdrawal from these drugs is not usually a problem but ‘flashbacks’ can occur. Treatment, especially where there is fear or anxiety, is diazepam 10–20 mg (o) statim.

Treatment (medication to counter symptoms)[14]

- haloperidol 2.5–10 mg (o) daily

  or

- diazepam 10–20 mg (o) repeated every 2 hours prn (to max. 120 mg daily)

Cannabis (marijuana) use

Cannabis is a drug that comes from the plant *Cannabis sativa*, the Indian hemp plant. It is a stimulant and a hallucinogen. It contains the chemical tetrahydrocannabinol, which makes people get 'high'. It is commonly called marijuana, grass, pot, dope, hash or hashish. Other slang terms are Acapulco gold, ganga, herb, J, jay, hay, joint, reefer, weed, locoweed, smoke, tea, stick, Mary Jane, Panama red and spliffy (see TABLE 12.5). Marijuana comes from the leaves, while hashish is the concentrated form of the resinous substances from the head of the female plant and can be very strong (it comes as a resin or oil). The drug is usually smoked as a leaf (marijuana) or a powder (hashish), or hashish oil is added to a cigarette and then smoked. The effects of taking cannabis depend on how much is taken, how it is taken, how often, whether it is used with other drugs and on the particular person.[21] The effects vary from person to person. The effects of a small-to-moderate amount include:

- feeling of well-being and relaxation
- decreased inhibitions
- woozy, floating feeling
- lethargy and sleepiness
- talkativeness and laughing a lot
- red nose, gritty eyes and dry mouth
- unusual perception of sounds and colour
- nausea and dizziness
- loss of concentration, slight cognitive impairment
- looking 'spaced out' or drunk
- lack of coordination
- delusions and hallucinations (more likely with larger doses)
- a form called skunk or mad weed causes paranoia

The effects of smoking marijuana take up to 20 minutes to appear and usually last 2 to 3 hours and then drowsiness follows.[21] The effect on psychomotor function is similar to alcohol and this can impair driving skills. The main problem is habitual use with the development of dependence; dependence (addiction) is worse than originally believed.

Long-term use and addiction

The influence of pot has a severe effect on the personality and drive of the users. They lose their energy, initiative and enterprise. They become bored, inert, apathetic and careless. A serious effect of smoking pot is the inability to concentrate and loss of memory. Some serious problems include:

- deterioration of academic or job performance
- anxiety and paranoia
- respiratory disease (more potent than tobacco for lung disease): causes COPD, laryngitis and rhinitis
- often prelude to taking illicit drugs
- becoming psychotic (resembling schizophrenia): the drug appears to unmask an underlying psychosis[21]
- impaired ability to drive a car and operate machinery

Withdrawal

Sudden withdrawal produces insomnia, night sweats, nausea, depression, myalgia, irritability and maybe anger and aggression. However, the effects are often mild with recovery within a few days in many, but heavy users have a severe withdrawal.

Management

No specific pharmacological treatment is available. CBT is advisable.

The best treatment is prevention. People should either not use it or limit it to experimentation. If it is used, people should be prepared to sleep it off and not drive.

Anabolic steroid misuse

The apparent positive effects of anabolic steroids include gains in muscular strength (in conjunction with diet and exercise) and quicker healing of muscle injuries. However, the adverse effects, which are dependent on the dose and duration, are numerous.

Adverse effects in women are:

- masculinisation—male-pattern beard growth
- suppression of ovarian function
- changes in mood and libido

- hair loss

In adult men, adverse effects are:

- feminisation: enlarged breasts, high-pitched voice
- acne
- testicular atrophy and azoospermia
- libido changes
- hair loss

Severe effects with prolonged use include:

- liver function abnormalities, including hepatoma
- tumours of kidneys, prostate
- heart disease

In prepubescent children there can be premature epiphyseal closure with short stature.

Drugs in sport[22]

It is important for GPs to have a basic understanding of drugs that are banned and those that are permissible for elite sporting use. The guidelines formulated by the International Olympic Committee (IOC) Medical Commission and the World Anti-Doping Agency (WADA) are generally adopted by most major sporting organisations.[13] TABLES 12.6 and 12.7 provide useful guidelines. The IOC's list of prohibited drugs is regularly revised. Banned drug groups include stimulants, narcotics, cannabinoids (e.g. marijuana), anti-oestrogen agents (e.g. tamoxifen), glucocorticosteroids (e.g. prednisolone), anabolic agents, diuretics and various hormones. Banned methods include blood doping (the administration of blood, red blood cells and related blood products), enhancement of oxygen transfer (e.g. erythropoietin, efaproxiral), gene doping and pharmaceutical, chemical and physical manipulation (substances or methods that alter the integrity and validity of the urine testing).

Table 12.6 Prohibited list: World Anti-Doping Guide (valid 1 January 2021)[22]

Classes	Examples
Prohibited substances at all times	
S1 Anabolic agents	Androstenediol, clenbuterol,

	DHEA, methandienone, methyl testosterone, nandrolone, oxandrolone, stanozolol, testosterone, tetrahydrogestrinone, tibolone, zeronol
S2 Peptide hormones, growth factors and related substances	Growth hormone, corticotrophin, chorionic gonadotrophin and LH (in males), erythropoietin (EPO), darbepoetin (dEPO), SERMS, insulin and insulin-like growth factor, ACTH *Note:* Masking agents such as probenecid, epitestosterone, diuretics and plasma expanders are banned.
S3 Beta-2 agonists	All oral beta-2 agonists, including both optical isomers (except inhaled salbutamol, eformoterol and salmeterol according to recommended guidelines)
S4 Hormone antagonists and metabolic modulators	Aromatase inhibitors, e.g. anastrozole, letrozole; SERMS, e.g. raloxifene, tamoxifen; other anti-oestrogenic substances, e.g. clomiphene; myostatin inhibitors; metabolic modulators —insulins
S5 Diuretics and other masking agents	Acetazolamide, frusemide, hydrochlorothiazide, triamterene, indapamide, spironolactone (and related substances)
Prohibited substances in competition	
S6 Stimulants	Amiphenazole, amphetamines, cocaine, ephedrine, ephedra, meldonium, mesocarb, terbutaline,* adrenaline, salmeterol,* salbutamol,* selegiline, pseudoephedrine, phenylpropanolamine,

	modafinil, phentermine
S7 Narcotics**	Diamorphine (heroin), methadone, morphine, pethidine, pentazocine, buprenorphine, hydromorphone, oxycodone, oxymorphone, fentanyl
S8 Cannabinoids	Natural (e.g. cannabis, hashish, marijuana) or synthetic THC and cannabimimetics (e.g. 'spice')
S9 Glucocorticosteroids	All glucocorticosteroids are prohibited when administered by oral, IV, IM or rectal routes
Substances prohibited in particular sports	
P1 Beta blockers	Prohibited in competition only, e.g. atenolol, carvedilol, metoprolol, propranolol, timolol (Check list and sports, e.g. archery, shooting, golf, skiing)
Prohibited methods	
Ml Manipulation of blood and blood components (blood doping, haemoglobin oxygen carriers)	
M2 Chemical and physical manipulation	
M3 Gene and cell doping	

*Permitted by inhaler but only with therapeutic use exemption (TUE).
**Caffeine, codeine, dextromethorphan, dextropropoxyphene, dihydrocodeine, tramadol, diphenoxylate and pholcodeine are permitted.
These lists are constantly being updated, so see www.wada-ama.org (World Anti-Doping Agency) or https://sportintegrity.gov.au for current information.

Table 12.7 Guidelines for treatment of specific conditions: International Olympic Committee Medical Code 2008

Asthma	
Allowed	Salbutamol inhaler, salmeterol inhaler, terbutaline inhaler, formoterol inhaler
Banned	Sympathomimetic products (e.g. ephedrine, pseudoephedrine, isoprenaline, systemic beta-2 agonists), oral corticosteroids
Cough	

Allowed	All antibiotics, steam and menthol inhalations, cough mixtures containing antihistamines, pholcodine, dextromethorphan, dihydrocodeine
Banned	Sympathomimetic products (e.g. ephedrine, phenylpropanolamine)
Diarrhoea	
Allowed	Diphenoxylate, loperamide, products containing electrolytes (e.g. Gastrolyte)
Banned	Products containing opioids (e.g. morphine)
Hayfever	
Allowed	Antihistamines, nasal sprays containing a corticosteroid or antihistamine, sodium cromoglycate preparations
Banned	Products containing ephedrine, pseudoephedrine
Pain	
Allowed	Aspirin, codeine, dihydrocodeine, ibuprofen, paracetamol, tramadol, all NSAIDs, dextropropoxyphene
Banned	Products containing opioids (e.g. morphine) or caffeine
Vomiting	
Allowed	Domperidone, metoclopramide

Restricted drugs include alcohol, marijuana, local anaesthetics, corticosteroids and beta blockers. Practitioners can check the guidelines and provide written notification to the relevant authority. Be cautious of anorectics and weight-reducing agents.

Other drug groups permitted by WADA:

- antidepressants
- antihypertensives (excluding beta blockers)
- caffeine
- eye medications
- oral contraceptives
- skin creams and ointments
- sleeping tablets

Check websites including: www.olympic.org.

Patient education resources

Hand-out sheets from *Murtagh's Patient Education* 8th edition:

- Alcohol: harmful use of alcohol
- Amphetamines
- Cocaine
- Ecstasy
- Heroin
- Smoking: quitting
- Cannabis (marijuana)

Resources

- Global DRO, Athletes, check your medications! (country-specific): www.globaldro.com; also see Sport Integrity Australia (superseded ASADA): https://www.sportintegrity.gov.au/
- Department of Health, National drug campaign: www.campaigns.health.gov.au/drughelp
- www.usada.org/substances/prohibited-list

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13 Anaemia

There's never none of these demure boys come to any proof; for thin drink doth so over cool their blood, and making many fish-meals, that they fall into a kind of male green-sickness.

WILLIAM SHAKESPEARE (1564–1616), *KING HENRY IV*

Anaemia is a label, not a specific diagnosis. Anaemia is defined as a reduction in red blood cell numbers or a haemoglobin (Hb) level below the normal reference level for the age and sex of that individual.

The WHO defines anaemia as haemoglobin <130 g/L for men, <120 g/L for women and <110 g/L in pregnant women and school-aged children.

Anaemia: a masquerade

Anaemia is regarded as a masquerade because the problem can develop surreptitiously and the patient may present with many seemingly undifferentiated symptoms before the anaemia is detected. Once identified, a cause must be found.

Key facts and checkpoints

- In Australia, most people with anaemia will have iron deficiency ranging from up to 5% for children to 20% for menstruating females.[1]
- The remainder will mainly have anaemia of chronic disorders.
- The incidence of haemoglobinopathy traits, especially thalassaemia, is increasing in multicultural Western societies.
- If a patient presents with precipitation or aggravation of myocardial ischaemia, heart failure or intermittent claudication, consider the possibility of anaemia.
- The serum ferritin level, which is low in cases of iron-deficiency anaemia, is probably the best test to monitor iron-deficiency anaemia as its level reflects the

amount of stored iron.

- Normal reference values for peripheral blood are presented in TABLE 13.1 .

Table 13.1 Normal reference values for peripheral blood: adults

	Male	Female
Haemoglobin (g/L)	130–180	115–165
Red cells (× 10^{12}/L)	4.5–6	4–5.5
PCV (haematocrit)	40–53	35–47
MCV (fL)	80–100	
Platelets (× 10^9/L)	150–400	
White cell count (× 10^9/L)	4–11	
Neutrophils	2.5–7.5	
Lymphocytes	1.5–4	
Monocytes	0.2–1	
Eosinophils	<0.5	
Reticulocytes (%)	0.5–2	
ESR (mm/hour)	<20 mm	
	<35 mm if >70 years	

Source: Reproduced with permission from Dr M Gribble[2]

DxT fatigue + palpitations + exertional dyspnoea → anaemia

Clinical features

Patients with anaemia may be asymptomatic. When symptoms develop they are usually non-specific. Symptoms can include:

- tiredness/fatigue
- muscle weakness

- headache and tinnitus
- lack of concentration
- faintness/dizziness
- dyspnoea on exertion
- palpitations
- angina on effort
- intermittent claudication
- pica—usually brittle and crunchy food, e.g. ice (iron-deficiency anaemia)

Signs

Non-specific signs include pallor, tachycardia, systolic flow murmur and angular cheilosis.

If severe, signs can include ankle oedema and cardiac failure.

Specific signs include jaundice—haemolytic anaemia, and koilonychias (spoon-shaped nails)—iron-deficiency anaemia.

History

The history may indicate the nature of the problem:

- iron deficiency: inadequate diet, pregnancy, GIT loss, menorrhagia, NSAID and anticoagulant ingestion
- folate deficiency: inadequate diet especially with pregnancy and alcoholism, small bowel disease
- vitamin B12 deficiency: previous gastric surgery, ileal disease or surgery, pernicious anaemia, selective diets (e.g. vegetarian, fad)
- haemolysis: abrupt onset anaemia with mild jaundice
- possibly lead toxicity, especially in children

Classification of anaemia

The various types of anaemia are classified in terms of the red cell size—the mean corpuscular volume (MCV):

- microcytic—MCV ≤ 80 fL

- macrocytic—MCV >100 fL
- normocytic—MCV 80–100 fL

Note: Upper limit of MCV varies from 95–100 fL depending on age and laboratory.

TABLE 13.2 outlines a classification of some of the more common causes of anaemia encountered in general practice. There can be an interchange of disorders between the above groups; for example, the anaemia of chronic disorders (chronic infection, inflammation and malignancy) can occasionally be microcytic as well as normocytic; the anaemia of hypothyroidism can be macrocytic in addition to the more likely normocytic; the anaemia of bone marrow disorder or infiltration can also be occasionally macrocytic.

Table 13.2 Classification of anaemia by mean RBC volume (MCV) with selected causes

Microcytic (MCV < 80 fL) • Iron deficiency • Thalassaemia • Anaemia of chronic disease • Sideroblastic anaemia
Microcytic (MCV > 100 fL) • Vitamin B12 deficiency • Folate deficiency • Myelodysplastic disorders • Cytotoxic drugs • Liver disease/alcoholism
Normocytic (MCV 80–100 fL) • Kidney disease • Anaemia of chronic disease • Endocrine failure/hypothyroidism • Haemolysis • Aplastic anaemia

Microcytic anaemia—MCV ≤80 FL

The main causes of microcytic anaemia are iron deficiency and haemoglobulinopathy,

particularly thalassaemia. Consider lead poisoning.

Iron-deficiency anaemia[3]

Iron deficiency is the most common cause of anaemia worldwide. It is the biggest cause of microcytic anaemia, with the main differential diagnosis of microcytic anaemia being a haemoglobinopathy such as thalassaemia. However, it is caused by bleeding until proved otherwise.

An understanding of the interpretation of iron studies is important in management.

Clinical and laboratory features

- Microcytic anaemia
- Serum ferritin level low (NR: F 15–200 mcg/L: M 30–300 mcg/L)
- Serum iron level low
- Increased transferrin level
- Microcytic hypochromic red cells
- MCV ↓, MCH ↓, MCHC ↓
- Reduced transferrin saturation
- Response to iron therapy

Non-haematological effects of chronic iron deficiency

- Angular cheilosis/stomatitis
- Glossitis
- Oesophageal webs
- Atrophic gastritis
- Brittle nails and koilonychias

Causes[1]

Blood loss

- Menorrhagia
- Gastrointestinal bleeding (e.g. carcinoma, haemorrhoids, peptic ulcer, hiatus hernia, GORD,

NSAID therapy)

- Frequent blood donations
- Malignancy
- Hookworm (common in tropics)

Increased physiological requirements

- Prematurity, infant growth
- Adolescent growth
- Pregnancy

Malabsorption

- Coeliac disease
- Postgastrectomy

Dietary

- Inadequate intake
- Special diets (e.g. fad, vegetarianism)
- Pica—eating unnatural food, e.g. dirt, ashes

Investigations

Investigations are based on the history and physical examination, including the rectal examination. If GIT bleeding is suspected, the faecal occult blood test is not considered very valuable but appropriate investigations include gastroscopy and colonoscopy, small bowel biopsy and small bowel enema.

Haematological investigations: typical findings

- Microcytic, hypochromic red cells
- Anisocytosis (variation in size), poikilocytosis (shape)—pencil-shaped rods
- Low serum iron level
- Raised iron-binding capacity
- Serum ferritin level low (the most useful index)

- Soluble transferrin receptor factor—this factor is increased in iron deficiency, but not in chronic disease. Therefore, it is very helpful in differentiating iron deficiency from other forms. It is an indirect marker of what is happening in the bone marrow.[4]

The state of the iron stores is assessed by considering the serum iron, the serum ferritin and the serum transferrin levels in combination. Typically, in iron deficiency, the serum iron and ferritin levels are low and the transferrin high, but the serum iron level is also low in all infections—severe, mild and even subclinical—as well as in inflammatory states, malignancy and other chronic conditions. Serum ferritin estimations are spuriously raised in liver disease of all types, chronic inflammatory conditions and malignancy; transferrin is normally raised in pregnancy. Since each of these estimations can be altered in conditions other than iron deficiency, all three quantities have to be considered together to establish the iron status (see TABLE 13.3).[2]

Table 13.3 The interpretation of iron studies[2]

Condition	Serum Fe	TIBC	% Transferrin saturation	Ferritin
Iron deficiency	↓	N or ↑	↓	↓ ↓
β-thalassaemia	N or ↑	N	N or ↑	N or ↑
Anaemia of chronic disease	↓	N or ↓	↓	N or ↑
Sideroblastic anaemia	N or ↑	N	N or ↑	↑
Haemochromatosis	↑	↓	↑ ↑	↑ ↑

N = normal

Treatment[4,5]

- Correct the identified cause.
- Diet—iron-rich foods, vitamin C-rich foods (see TABLE 13.4). Iron is present in meat and legumes as Fe^{+++} and therefore requires gastric acid for conversion to Fe^{++}.
- Elemental iron supplements 100–200 mg daily (adults).
- Iron preparations:

  oral iron (ferrous sulphate 1–2 tablets daily between meals for 6 months), e.g. Ferro-Gradumet or Ferro-grad C (avoid taking with milk) with orange juice or ascorbic acid until Hb is normal

  parenteral iron preferably by IV infusion is probably best reserved for special

circumstances such as a failed trial of oral iron for symptomatic iron-deficiency anaemia (there is a risk of an allergic reaction, a serum sickness-like illness for 48 hours and post-infusion skin staining around the cannula site). Cover with an antihistamine or IV hydrocortisone 30 minutes beforehand. Infusion is best with ferric carboxymaltose in 0.9% (N) saline.[6] Avoid blood transfusions if possible. IM iron is not recommended.

Table 13.4 Optimal adult diet for iron deficiency

Adults should limit milk intake to 500 mL a day while on iron tablets
Avoid excess caffeine, fad diets and excess processed bread
Eat ample iron-rich foods (especially protein)
Protein foods
Meats—beef (especially), veal, pork, liver, poultry
Fish and shellfish (e.g. oysters, sardines, tuna)
Seeds (e.g. sesame, pumpkin)
Eggs, especially egg yolk
Fruits
Dried fruit (e.g. prunes, figs, raisins, currants, peaches)
Juices (e.g. prune, blackberry)
Most fresh fruit
Vegetables
Greens (e.g. spinach, silver beet, lettuce)
Dried peas and beans (e.g. kidney beans)
Pumpkin, sweet potatoes
Grains
Iron-fortified breads and dry cereals
Oatmeal cereal
For better iron absorption, add foods rich in vitamin C (e.g. citrus fruits, cantaloupe, Brussels sprouts, broccoli, cauliflower)

Response

- Anaemia responds after about 2 weeks and is usually corrected after 2 months (if underlying cause addressed).[1]
- Oral iron is continued for 3 to 6 months to replenish stores.

- Monitor progress with regular serum ferritin levels.
- A serum ferritin level >50 mcg/L generally indicates adequate stores.

Failure of iron therapy

Consider:

- poor compliance
- continuing blood loss
- malabsorption (e.g. severe coeliac disease)
- incorrect diagnosis (e.g. thalassaemia minor, chronic disease)
- bone marrow infiltration

Thalassaemia

This inherited condition is seen mainly (although not exclusively) in people from the Mediterranean basin, the Middle East, north and central India and South-East Asia, including south China. The heterozygous form is usually asymptomatic; patients show little if any anaemia and require no treatment. The condition is relatively common in people from these areas. The homozygous form is a very severe congenital anaemia needing lifelong transfusional support but is comparatively rare, even among the populations prone to thalassaemia (refer to CHAPTER 23).[2]

The key to the diagnosis of heterozygous thalassaemia minor is significant microcytosis quite out of proportion to the normal Hb or slight anaemia, and confirmed by finding a raised HbA_2 on Hb electrophoresis. DNA screening analysis is now available. The importance of recognising the condition lies in distinguishing it from iron-deficiency anaemia, for iron does not help people with thalassaemia and is theoretically contraindicated. Even more importantly, it lies in recognising the risk that, if both parents have thalassaemia minor, they run a one in four chance of having a baby with thalassaemia major in every pregnancy, with devastating consequences for both the affected child and the whole family.

Treatment of thalassaemia major is transfusion to a high normal Hb with packed cells plus desferrioxamine.

Haemoglobin E

This Hb variant is common throughout South-East Asia.[4] It has virtually no clinical effects in either the homozygous or heterozygous forms, but these people have microcytosis, which must be distinguished from iron deficiency; moreover, if the *HbE* gene is combined with the thalassaemia gene, the child may have a lifelong anaemia almost as severe as thalassaemia

major. Both genes are well established in the South-East Asian populations in Australia as well as in their own countries.

Macrocytic anaemia—MCV >100 FL

Alcohol and liver disease

Each individually, or in combination, leads to macrocytosis with or without anaemia. The importance of this finding lies in its often being the first indication of alcohol abuse, which can so frequently go unnoticed unless there is a firm index of suspicion. Chronic liver disease due to other causes may also be late in producing specific clinical symptoms.

Drug toxicity

Cytotoxic drugs, anticonvulsants in particular, and various others (see TABLE 13.5) may cause macrocytosis. This is of little clinical significance and does not need correction unless associated with anaemia or other cytopenia.

Table 13.5 Drugs causing macrocytosis[2,5]

Alcohol	
Cytotoxics/immunosuppressants	Azathioprine Methotrexate, 5-fluorouracil
Antibiotics	Cotrimoxazole, Pyrimethamine (incl. Fansidar and Maloprim) Zidovudine
Anticonvulsants	Phenytoin Primidone Phenobarbitone

Myelodysplastic syndromes

These conditions have been recognised under a variety of names, such as ‘refractory anaemia’ and ‘preleukaemia’, for a long time, but only relatively recently have they been grouped together. They are quite common in the elderly but may be seen in any age group (refer to TABLE 13.2).

These conditions frequently present as a macrocytic anaemia with normal serum vitamin B12 and red cell folate, and are unresponsive to these or any other haematinics. They are usually

associated with progressive intractable neutropenia or thrombocytopenia or both, and progress slowly but relentlessly to be eventually fatal, terminating with infection, haemorrhage or, less often, acute leukaemia.

Vitamin B12 deficiency (pernicious anaemia)

Although well recognised, this is a much less common cause of macrocytosis than the foregoing conditions. It is usually caused by lack of intrinsic factor due to autoimmune atrophic changes and by gastrectomy. Anaemia does not develop for about 3 years after total gastrectomy. Vitamin B12 deficiency may also be seen together with other deficiencies in some cases of malabsorption and Crohn disease.

Vitamin B12 (cobalamin) is found in the normal diet but only in foods of animal origin and consequently very strict vegetarians may eventually develop deficiency. Causes of food vitamin B12 deficiency are:[4]

- atrophic gastritis
- *H. pylori* infection
- H_2 receptor blockers
- PPI drugs
- other drugs, e.g. OCP, metformin
- chronic alcoholism
- HIV
- strict vegan diet

The clinical features are anaemia (macrocytic), weight loss and neurological symptoms, especially a polyneuropathy. It can precipitate subacute combined degeneration of the cord. The serum vitamin B12 is below the normal level (normal range 150–700 pmol/L).

$$\begin{aligned} \mathbf{B12} > \mathbf{220\ pmol/L} &= \mathbf{deficiency\ unlikey} \\ < \mathbf{148\ pmol/L} &= \mathbf{deficiency} \end{aligned}$$

Intrinsic factor antibody level is diagnostic.

Treatment (replacement therapy)[1]

- Vitamin B12 (1000 mcg, i.e. 1 mg) IM injection; body stores (3–5 mg) are replenished after 10–15 injections given every 2 to 3 days
- Maintenance with 1000 mcg injections every third month

- Can use crystalline oral B12
- Co-therapy with oral folate 5 mg/day (initially) is indicated.[9,10]
- Transfusion is best avoided. May need additional iron.

Folic acid deficiency

Diagnostic test: serum folate (normal range 7–45 nmol/L) and red cell folate—best test (normal >630 nmol/L).[7]

The main cause is poor intake associated with old age, poverty and malnutrition, usually associated with alcoholism. It may be seen in malabsorption and regular medication with anti-epileptic drugs such as phenytoin.[9] It is rarely, but very importantly, associated with pregnancy, when the demands of the developing fetus together with the needs of the mother outstrip the dietary intake—the so-called 'pernicious anaemia of pregnancy' which, if not recognised and treated immediately, can still be a fatal condition. Unlike vitamin B12, folic acid is not stored in the body to any significant degree and requirements have to be satisfied by the daily dietary intake, which invariably meets the requirement of 5–10 mcg/day. Folic acid is present in most fruit and vegetables, especially citrus fruits, nuts and green leafy vegetables (see CHAPTER 5).

Treatment (replacement therapy)

Oral folate 5 mg/day to replenish body stores (5–10 mg). This takes about 4 weeks but continue for 4 months. Vitamin B12 is usually given unless levels normal.

Normocytic anaemia[2] (anaemias without change in the MCV)

Acute haemorrhage

This is the most common cause of normocytic anaemia and is usually due to haematemesis and/or melaena.

Chronic disease

Chronic inflammation

Intercellular iron transport within the marrow is suppressed in inflammation so that, despite normal iron stores, the developing red cells are deprived of iron and erythropoiesis is depressed. If the inflammation is short-lived, the fall in Hb is not noticeable but, if it continues, an anaemia may develop that responds only when the inflammation subsides.

Malignancy

Anaemia may develop for the same reasons that apply to chronic inflammation.

Kidney failure

This is often associated with anaemia due to failure of erythropoietin secretion and is unresponsive to treatment, other than by alleviating the insufficiency or until erythropoietin is administered.

Haemolysis

Suspect haemolytic anaemia if there is a reticulocytosis, mild macrocytosis, reduced haptoglobin, increased bilirubin and urobilinogen. Haemolytic anaemias are relatively infrequent. The more common of the congenital ones are hereditary spherocytosis, sickle-cell anaemia and deficiencies of the red cell enzymes, pyruvate kinase and G-6-PD, although most cases of G-6-PD deficiency haemolyse only when the patient takes oxidant drugs such as sulphonamides or eats broad beans —'favism'.

Acquired haemolytic anaemias include those of the newborn due to maternal haemolytic blood group antibodies passing back through the placenta to the fetus, and adult anaemias due to drug toxicity or to acquired autoantibodies. About half of the latter are idiopathic and half associated with non-Hodgkin lymphomas, and the anaemia may be the presenting sign of lymphoma. Some of these antibodies are active only at cool temperatures—cold agglutinin disease; others act at body temperature and are the more potent cause of autoimmune haemolytic anaemia.

Keep in mind the rare acquired genetic disorder of paroxysmal nocturnal haemoglobinuria if dark morning urine is observed in the presence of anaemia. Flow cytometry is required for diagnosis.

Aplastic anaemia

This presents with clinical features of anaemia (Hb ↓), infection (WCC ↓) or bleeding (platelets ↓). Hypoplasia of bone marrow causes pancytopenia and normoctyic normochromic anaemia. Most cases are due to an autoimmune disorder; others are due to drugs and radiotherapy. Diagnosis is by bone marrow examination. Treatment includes supportive care and options such as immunotherapy, allogeneic bone marrow transplantation, stem cell transplantation and haemopoietic grow factors such as erythropoietin.[11]

Bone marrow replacement

This may be due to foreign tissue, such as carcinomatous metastases, or fibrous tissue, as in myelofibrosis; it may also be due to overgrowth by one or other normal elements of the bone marrow, as in chronic myeloid leukaemia, chronic lymphocytic leukaemia and lymphoma, as well as by acute leukaemic tissue. A leuco-erythroblastic picture, in which immature red and

white cells appear in the peripheral blood, is often seen when the marrow is replaced by foreign tissue.

Anaemia in children

Haemoglobin reference range

Infant	Term (cord blood)	135–195 g/L
	3–6 months	95–135 g/L
Child	1 year	105–135 g/L
	3–6 years	105–140 g/L
	10–12 years	115–145 g/L

Important causes of anaemia in childhood include iron-deficiency anaemia (quite common), thalassaemia major, sickle-cell anaemia and drug-induced haemolysis. Consider one of the haemoglobinopathies in children of Mediterranean, South-East Asian, Arabic or African–American descent, especially with a family history, normal ferritin level or anaemia resistant to iron therapy. Investigate with Hb electrophoresis.

Drugs that can cause haemolysis (the film will have reticulocytosis, spherocytosis and fragmented red cells) include some antibiotics (e.g. sulfamethoxazole), antimalarials and some anti-inflammatories.

Think of anaemia in adolescents, especially females with a rapid growth spurt at menarche and a relatively poor diet.

Iron deficiency in children[10]

- Iron deficiency is present in up to 10–30% of children in high-risk groups.
- It is often subclinical and anaemia develops in relatively few.
- It can lead to reduced cognitive and psychomotor performance (even without anaemia).
- High-risk groups include those infants <6 months who are premature and/or with low birthweight; toddlers 6–36 months with a diet high in cow's milk and low in iron-containing foods; those exclusively breastfed after 6 months; those with delayed introduction of solids; those with general poor food intake; and those with lack of vitamin C in their diet. Bottle-feeding encourages a high milk intake and reduces the appetite for solid food.
- Possible clinical features include irritability, lethargy, minor behavioural changes, poor growth, dyspnoea and pallor.

Prevention

- Give iron and multivitamin supplements to very premature and low birthweight (<1000 g) infants.
- Introduce iron-containing solids early—at 4 to 5 months, e.g. cereals, vegetables, egg and meat.
- Encourage breastfeeding and avoid cow's milk in the first 12 months.[9]
- Avoid excessive cow's milk up to 24 months.
- Use iron-fortified formulas and cereals.

Important sources of iron

Infant milk formulas, meat (especially red meat, and also fish and chicken), green vegetables and legumes, dried fruit, juices, fortified cereals, egg yolk.

Treatment

Treatment is mainly with ferrous gluconate (1 mL/kg of 300 mg/5 mL mixture). Continue for 3 months after Hb has normalised.

Practice tips

- Iron-deficiency anaemia is blood-loss anaemia until proved otherwise.
- It is possible to be tired from iron deficiency without anaemia.
- Blood-loss anaemia is usually due to menorrhagia or gastrointestinal loss until proved otherwise.
- Investigations for suspected anaemia should include FBE, ESR and iron studies. Others to consider are Hb electrophoresis, vitamin B12 and folate levels, and kidney function tests.
- Hypothyroidism can cause a normocytic or a macrocytic anaemia.
- A therapeutic trial of iron (without investigations) is indefensible.
- Intramuscular injections of iron can tattoo so use with care: an IM iron dose is not 'stronger' than an oral iron dose.
- If microcytic anaemia is not responding to treatment, consider sideroblastic anaemia.

Patient education resource

Hand-out sheet from *Murtagh's Patient Education* 8th edition: Page 133

- Iron deficiency anaemia

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14 Endocrine and metabolic disorders

It would indeed be rash for a mere pathologist to venture forth on the uncharted sea of the endocrines, strewn as it is with the wrecks of shattered hypotheses, where even the most wary mariner may easily lose his way as he seeks to steer his bark amid the glandular temptations whose siren voices have proved the downfall of many who have gone before.

WILLIAM BOYD (1885–1979)

Endocrine, particularly thyroid, disorders can be a diagnostic trap in family practice and early diagnosis is a real challenge. A family practice of 2500 patients can expect one new case of thyroid disorder each year and 10 ‘cases’ in the practice.[1] Thyroid disease can be classified as thyroid dysfunction or structural, e.g. goitre. The diagnosis of an overactive or underactive thyroid can be difficult as the early clinical deviations from normality can be subtle.

The clinical diagnosis of classical Graves disease is usually obvious with the features of exophthalmos, hyperkinesis and a large goitre, but if the eye and neck signs are absent it can be misdiagnosed as an anxiety state. Elderly patients may present with only cardiovascular signs, such as atrial fibrillation and tachycardia, or with unexplained weight loss.

The hypothyroid patient can be very difficult to diagnose in the early stages, especially if the patient is being seen frequently. Hypothyroidism often has a gradual onset with general symptoms such as constipation and lethargy.

If suspected, only serum thyroid stimulating hormone (TSH) or thyrotropin should be requested initially.[2]

Other common endocrine disorders include diabetes mellitus, hyperprolactinaemia, calcium metabolic disorder, PCOS, sexual dysfunction and subclinical hypogonadism. They may be difficult to diagnose in the early stages of development. The pituitary is the master gland and its regulating hormones are depicted in FIGURE 14.1 .[3,4]

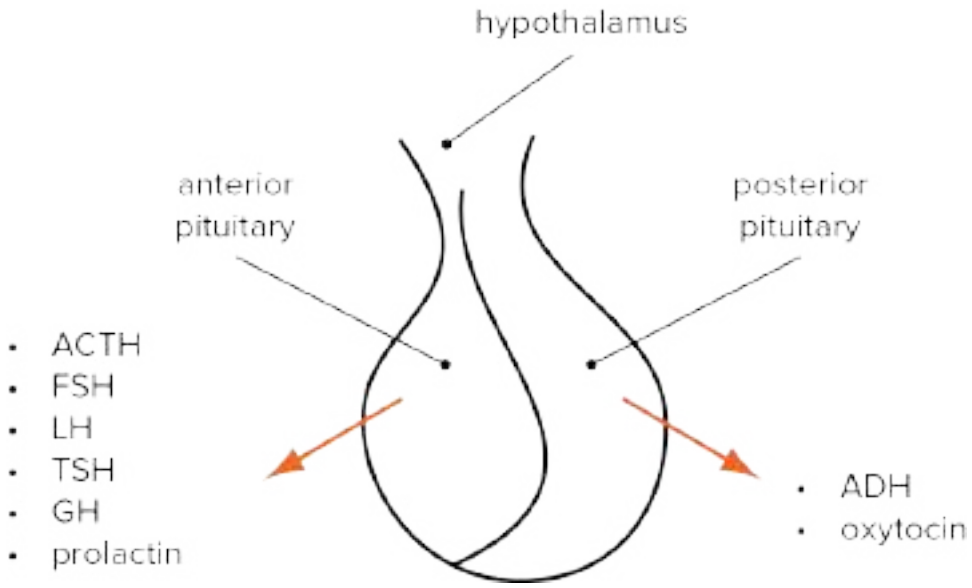

FIGURE 14.1 Pituitary hormones

Thyroid disorders

Tests for thyroid disorders[3,4]

Thyroid function tests

Advances in technology have allowed the biochemical assessment of thyroid function to change dramatically in recent years with the introduction of the serum free thyroxine (T_4) and the monoclonal TSH assays. With the highly sensitive TSH assays it is now possible to distinguish suppressed TSH levels (as in hyperthyroidism) from low and normal levels of TSH. However, the new assays are not foolproof and require interpretation in the context of the clinical picture. The serum TSH level is the most sensitive index of thyroid function and is the preferred test for suspected thyroid dysfunction. If necessary, repeat TSH in 3–6 months.

Serum free tri-iodothyronine (T_3) measurement and serum free thyroxine (T_4) can be useful in suspected T_3 toxicosis where serum T_4 level may be normal, and for monitoring patients with treated thyroid dysfunction.

The relative values are summarised in TABLE 14.1 .

Table 14.1 Summary of thyroid function tests[3]

	TSH	free T_4	free T_3	Antithyroid antibodies
Normal range	0.4–4 mU/L	10–25 pmol/L	2.6–6.0 pmol/L	
Hypothyroidism				
Primary (overt)	↑*	↓*	N or ↓ (not useful)	N or ↑
Subclinical	↑	N	N	N
Secondary (pituitary dysfunction)	N or ↓	↓	N or ↓ (not useful)	N
Hyperthyroidism (overt)	↓*	↑*	↑*	N or ↑
Subclinical	↓	N	N	N
Sick euthyroid	N or ↓	N or ↓	N or ↓	N

Notes: Results similar to hyperthyroidism can occur with acute psychiatric illness.
Normal ranges vary between laboratories.
*Main tests

Thyroid autoantibodies

Positive autoantibodies are specific for the following:[2]

- TSH receptor antibodies (TR Ab): Graves disease
- Thyroid peroxidase antibodies (TPO Ab): Hashimoto disease
- Thyroglobulin antibody (Tg Ab): Hashimoto disease

Fine-needle aspiration

This is the single most cost-effective investigation in the diagnosis of thyroid nodules. It is the best way to assess a nodule for malignancy. Care needs to be taken in the interpretation of the cytology results in conjunction with an experienced cytologist/pathologist.

Thyroid nuclear scan and imaging

The scan may help in the differential diagnosis of thyroid nodules and in causes of hyperthyroidism. A functioning nodule is said to be less likely to be malignant than a non-functioning nodule (cyst, colloid nodule, haemorrhage are non-functioning; carcinoma is usually non-functioning).

Thyroid ultrasound

A thyroid ultrasound is usually more sensitive in the detection of thyroid nodules. A

multinodular goitre may be diagnosed on ultrasound while the clinical impression may be that of a solitary nodule (the other nodules not being palpable clinically). A multinodular goitre is said to be less likely to be malignant than a solitary thyroid nodule. An ultrasound allows for follow-up of thyroid nodule(s) to note if there are any changes in size over a period of time and to then discuss appropriate intervention with the patient. It can also differentiate a solid from a cystic mass.

CT scan

CT scan of the thyroid may be used particularly to determine if there is significant compression in the neck from a large multinodular goitre with retrosternal extension. Again, follow-up CT scans may allow one to determine the progression or otherwise of such a goitre.

Hypothyroidism (myxoedema)

Primary hypothyroidism, which is relatively common, is more prevalent in elderly women (up to 5%).[5] The term *myxoedema* refers to the accumulation of mucopolysaccharide in subcutaneous tissues. The early changes are subtle and can be misdiagnosed, especially if only a single symptom is dominant.

Transient causes include subacute thyroiditis, postpartum thyroiditis and silent thyroiditis.

Common causes of primary hypothyroidism include radioactive iodine treatment, thyroid surgery and Hashimoto thyroiditis.

Patients at risk include those with:

- previous Graves disease
- autoimmune disorders (e.g. autoimmune lymphocytic thyroiditis, rheumatoid arthritis, type 1 diabetes)
- Down syndrome
- Turner syndrome
- drug treatment: lithium, amiodarone, interferon, iodine
- previous thyroid or neck surgery
- previous radioactive iodine treatment of the thyroid

Clinical features

The main features are:

- constipation

- cold intolerance
- tiredness/lethargy/somnolence
- physical slowing
- mental slowing
- depression
- huskiness of voice
- puffiness of face and eyes
- pallor
- loss of hair
- weight gain

DxT tiredness + husky voice + cold intolerance → myxoedema

Physical examination

See FIGURE 14.2 . The main signs are:

- sinus bradycardia
- delayed reflexes (normal muscular contraction, slow relaxation)
- coarse, dry and brittle hair
- thinning of outer third of eyebrows
- dry, cool skin
- skin pallor or yellowing
- obesity
- goitre

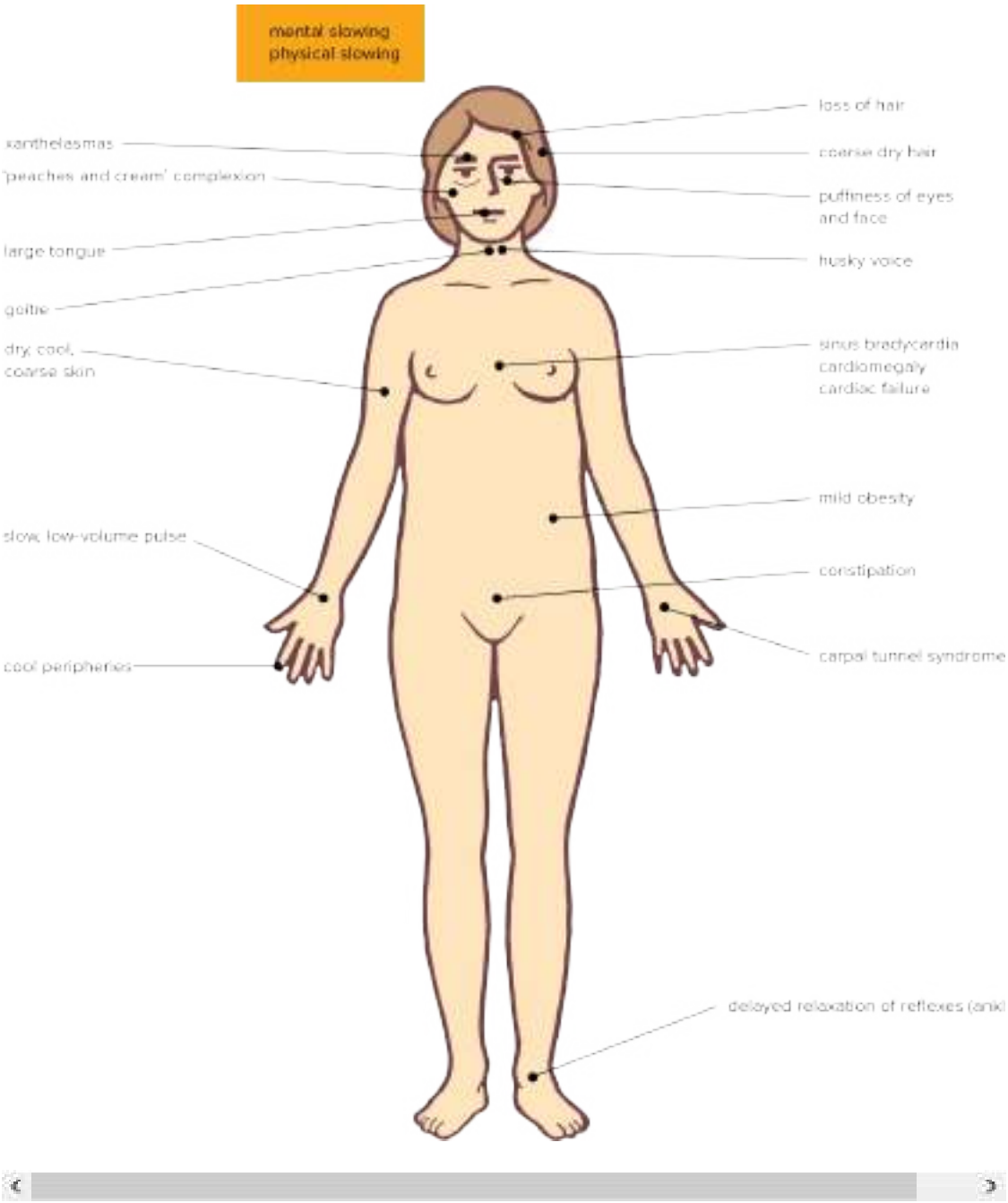

FIGURE 14.2 Clinical features of hypothyroidism

Other diverse presentations of thyroid disorders are given in TABLE 14.2 .

Table 14.2 Various diverse presentations of thyroid disorders[3,5]

	Hypothyroidism	Hyperthyroidism

General	Lethargy, tiredness Dry skin Husky voice	Weakness Sweaty skin, especially hands
Psychiatric	Depression Dementia Psychosis (myxoedema madness)	Anxiety/irritability Hyperkinesis Psychosis
Musculoskeletal	Myofibrositis Myalgia Joint effusions	Muscle weakness Proximal myopathy
Skin	Dry, cool skin Vitiligo	Warm, thin, soft, moist skin Vitiligo Pretibial myxoedema
Cardiovascular	Ischaemia Cardiomegaly Pericardial effusion Bradycardia Hyperlipidaemia	Tachycardia Atrial fibrillation Heart failure/breathlessness Systolic hypertension
Endocrine	Galactorrhoea Goitre Infertility	Goitre Gynaecomastia
Gynaecological	Menstrual irregularity Menorrhagia (mainly) Oligomenorrhoea	Other menstrual disturbances Oligomenorrhoea
Neurological	Neuropathy Nerve entrapment (e.g. carpal tunnel) Ataxia	Periodic paralysis Tremor
Haematological	Anaemia	–
Emergency	Myxoedema coma Postanaesthetic hypoventilation	Thyroid crisis
Other	Reduced libido Weight gain	Reduced libido Eye signs

Cold intolerance	Fever (uncommon)
Constipation	Onycholysis
	Premature grey hair
	Weight loss

Hashimoto thyroiditis (autoimmune thyroiditis)

Hashimoto thyroiditis, or lymphocytic thyroiditis, which is an autoimmune thyroiditis, is the commonest cause of bilateral non-thyrotoxic goitre in Australia. Features are:

- bilateral goitre
- classically described as firm and rubbery
- patients may be hypothyroid or euthyroid with a possible early period of thyrotoxicosis

Diagnosis is confirmed by a strongly positive antithyroid microsomal antibody (TPO Ab) titre and/or fine-needle aspiration cytology.[4]

Hashimoto thyroiditis may present as postpartum hypothyroidism. The hypothyroidism may resolve in 6–12 months or may be permanent.[4]

Laboratory diagnosis of hypothyroidism

Thyroid function tests (see TABLE 14.1):

- T_4—subnormal
- TSH—elevated (>10 is clear gland failure)

If T_4 is low and TSH is low or normal, consider pituitary dysfunction (secondary hypothyroidism) or sick euthyroid syndrome. A raised TSH and T_4 in normal range denotes ‘subclinical’ hypothyroidism and treatment is appropriate albeit controversial.[2,3]

Interpretation of TFTs can be difficult but requires matching to the clinical ‘picture’ and consultant advice.

Other tests

- Serum cholesterol level elevated
- Anaemia: usually normocytic; may be macrocytic
- ECG: sinus bradycardia, low voltage, flat T waves

Management[6,7,8]

Confirm the diagnosis, provide appropriate patient education and refer the patient where appropriate.

Exclude coexisting hypoadrenalism and ischaemic heart disease before T_4 replacement.

Note: Treatment as primary hypothyroidism when hypopituitarism is the cause may precipitate adrenal crisis.

Thyroid medication

- Levothyroxine (thyroxine) 50–100 mcg daily, increasing by 25 mcg up to 100–200 mcg if required

Note: Start with low doses (25–50 mcg daily) in >60 years and those with ischaemic heart disease and 50–100 mcg in others. Avoid overdosage.

- Aim to achieve TSH levels of 0.5–2 mU/L.
- Monitor TSH levels 6–8 weeks at first. As euthyroidism is achieved, monitoring may be less frequent (e.g. 2–3 months). When stable on optimum dose of T_4, monitor every 2–3 years. Treatment is usually lifelong.

Special treatment considerations

- *Ischaemic heart disease.* Rapid thyroxine replacement can precipitate myocardial infarction, especially in the elderly.
- *Pregnancy and postpartum.* Continue thyroxine during pregnancy; watch for hypothyroidism (an increased dose of T_4 is often required).
- *Elective surgery.* If euthyroid, can stop thyroxine for one week. If subthyroid, defer surgery until euthyroid.
- *Myxoedema coma.* Urgent hospitalisation under specialist care is required. Intensive treatment is required, which may involve parenteral T_4 or T_3 as liothyronine or thyroxine by slow IV injection.

Myxoedema coma

This is a life-threatening emergency with coma, extreme hyperthermia, areflexia and respiratory depression. Precipitating factors include illness, infection, trauma and cold. Treatment is supportive care, IV thyroxine or liothyronine and corticosteroids. Convert to oral T_4 when stable.

Neonatal hypothyroidism

Misdiagnosing this serious condition leads to failure to thrive, retarded growth and poor school performance. If untreated it leads to permanent intellectual damage (cretinism). The clinical features of the newborn include coarse features, dry skin, supra-orbital oedema, jaundice, harsh cry, slow feeding and umbilical hernia. It is detected by routine neonatal heel-prick blood testing. Thyroxine replacement should be started as soon as possible, at least before 2 weeks of age, to avert intellectual retardation.

When to refer—hypothyroidism[5,7]

- Doubt about diagnosis, diagnostic tests or optimum replacement dosage
- Apparent secondary hypothyroidism, severe illness and associated ischaemic heart disease
- Concurrent autoimmune disease
- Hypothyroidism with goitre, postpartum thyroid dysfunction and in the neonate
- Myxoedema coma

Hyperthyroidism (thyrotoxicosis)

Hyperthyroidism is also relatively common and may affect up to 2% of women, who are affected four to five times more often than men (see FIG. 14.3). Graves disease is the most common cause, followed closely by nodular thyroid disease.

FIGURE 14.3 Thyrotoxicosis patient with exophthalmos and goitre

Photo courtesy Duncan Topliss

Causes[4,9]

- Graves disease (typical symptoms with a diffuse goitre and eye signs)
- Autonomous functioning nodules/toxic adenoma
- Subacute thyroiditis (de Quervain thyroiditis)—viral origin (suspect if painful thyroid and malaise)
- Excessive intake of thyroid hormones—thyrotoxicosis factitia
- Exogenous iodine excess, e.g. food contamination
- Amiodarone (beware of this drug)

Key facts and checkpoints

- The classic symptoms may be lacking in elderly patients who may have only cardiovascular manifestations (e.g. unexplained heart failure or cardiac arrhythmias).
- Care has to be taken not to dismiss hyperthyroidism as severe anxiety.

Clinical features

- Heat intolerance
- Sweating of hands
- Muscle weakness
- Weight loss despite normal or increased appetite
- Emotional lability, especially anxiety, irritability
- Palpitations
- Frequent loose bowel motions

DxT anxiety + weight loss + weakness → thyrotoxicosis

Physical examination

See FIGURE 14.4 . Signs are (usually):

- agitated, restless patient
- warm and sweaty hands
- fine tremor (place paper on hands)
- goitre
- proximal myopathy
- hyperactive reflexes
- bounding peripheral pulse
- ± atrial fibrillation

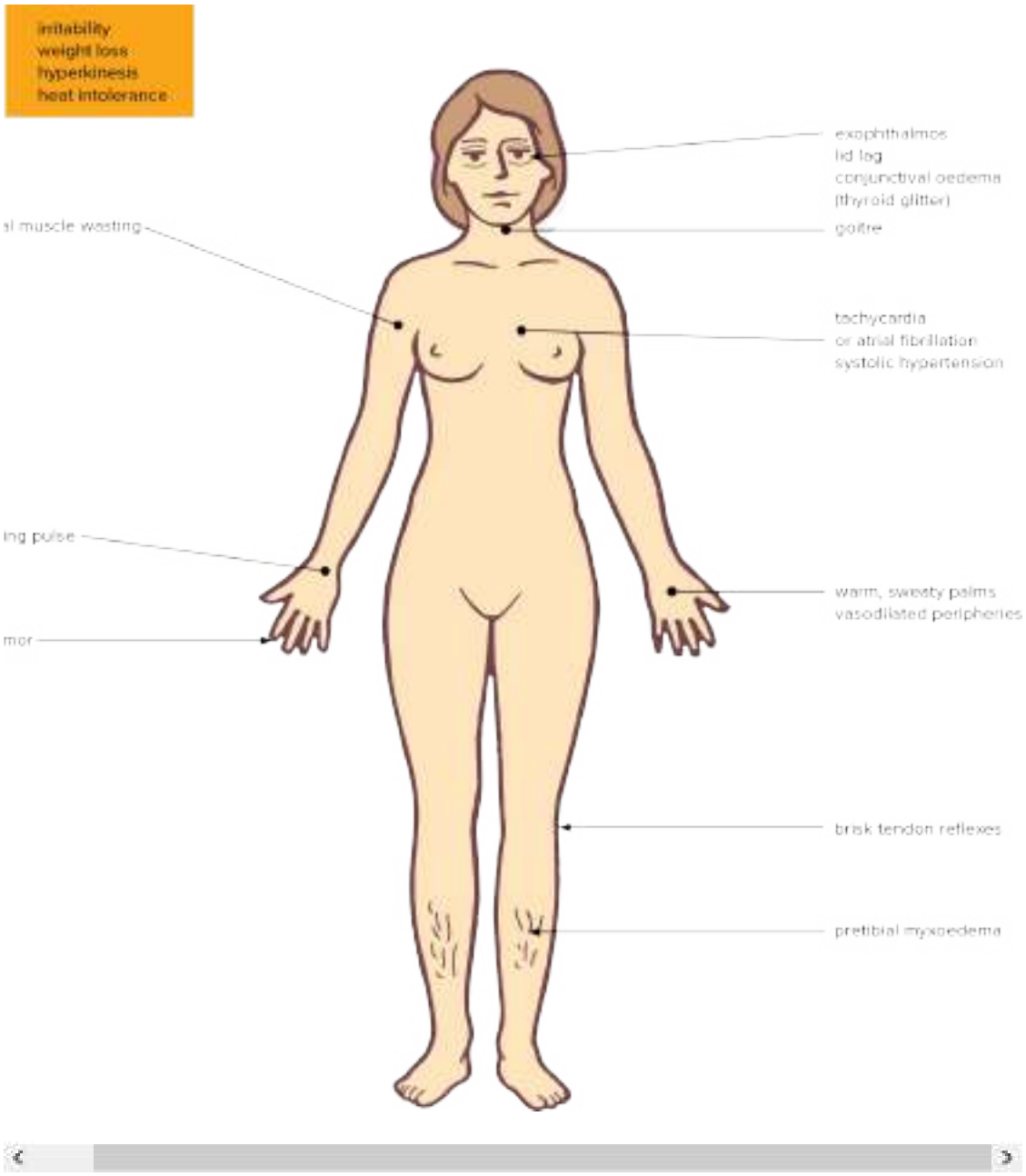

FIGURE 14.4 Clinical features of hyperthyroidism

Eye signs

- Lid retraction (small area of sclera seen above iris)
- Lid lag
- Exophthalmos
- Ophthalmoplegia in severe cases

Investigations

- T_4 (and T_3) elevated
- TSH level suppressed
- Radioisotope scan
- Antithyroid peroxidase (TPO Ab)—often positive

The isotope scan enables a diagnosis of Graves disease to be made when the scan shows uniform increased uptake. Increased irregular uptake would suggest a toxic multinodular goitre, while there is poor or no uptake with de Quervain thyroiditis and thyrotoxicosis factitia.

Management

- Establish the precise cause before initiating treatment.
- Refer to an endocrinologist to guide treatment.
- Educate patients and emphasise the possibility of development of recurrent hyperthyroidism or hypothyroidism and the need for lifelong monitoring.
- Monitor for cardiovascular complications, osteoporosis and eye problems.

Treatment[10,7,8]

- Radioactive iodine therapy (^{131}I)
- Thionamide antithyroid drugs (initial doses)

  carbimazole 10–45 mg (o) daily starting with 10–20 mg in divided doses depending on disease activity

  or

  propylthiouracil 200–600 mg (o) daily in divided doses or methimazole

- Adjunctive drugs

  beta blockers (for symptoms in acute florid phase, e.g. propranolol 10–40 mg, 6 to 8 hourly); diltiazem or atenolol are alternatives

  lithium carbonate (rarely used when there is intolerance to thionamides)

  Lugol's iodine: mainly used prior to surgery

- Surgery

subtotal thyroidectomy

or

total thyroidectomy

Treatment (Graves disease)

There is no ideal treatment and selection of antithyroid drugs, radioiodine or surgery depends on many factors, including age, size of goitre, social and economic factors and complications of treatment. Encourage cessation of smoking.

Guidelines[10,8]

- Younger patients with small goitres and mild case—18-month course antithyroid drugs
- Older patients with small goitres—as above or radioiodine (preferably when euthyroid)
- Large goitres or moderate-to-severe cases—antithyroid drugs until euthyroid, then surgery or ^{131}I
- In Australia (as in the US) ^{131}I is being increasingly used

Treatment (autonomous functioning nodules and toxic adenoma)

Control hyperthyroidism with antithyroid drugs, then surgery or ^{131}I. Long-term remissions on antithyroid drugs in a toxic nodular goitre are rare.

Subacute thyroiditis (de Quervain thyroiditis)[8]

Hyperthyroidism is usually transient for 1–2 months, and follows a surge of thyroxine after a viral-type illness, then followed by hypothyroidism for 4–6 months. Symptoms include pain and/or tenderness over the goitre (especially on swallowing), fever, ESR elevated, TPO Ab low or absent and radionuclide scan near absent uptake. In the acute phase treatment is based on rest, analgesics (aspirin 600 mg (o) 4–6 hourly) or ibuprofen 200–400 mg (o) and soft foods. Rarely, when pain is severe, corticosteroids (e.g. prednisolone) may be used. Antithyroid drugs are not indicated but beta blockers can be used to control symptoms.

Painless postpartum thyroiditis

Release of thyroid hormone from autoimmune destruction of thyroid. Typically 1–6 months post delivery. Hyperthyroidism initially, followed by hypothyroidism. Diffuse small goitre, poor radionuclide uptake, high TPO Ab. Treat with beta blockers for symptoms and thyroxine for hypothyroid phase.

Note: Autoimmune destruction of the thyroid with thyroiditis—painless or painful—can lead to

agranulocytosis, so monitor for signs of fever or mouth ulcers.

Thyroid crisis (thyroid storm)[8]

Clinical features are marked anxiety, weight loss, weakness, proximal muscle weakness, hyperpyrexia, tachycardia (>150 per minute), heart failure and arrhythmias. It is usually precipitated by surgery or an infection in an undiagnosed patient.

It requires urgent intensive hospital management with antithyroid drugs; IV saline infusion, IV corticosteroids, anti-heart failure and antiarrhythmia therapy, especially beta blockers.

When to refer—hyperthyroidism[9]

- Doubt about the diagnosis
- Severe hyperthyroidism, especially if there is coexisting thyrocardiac disease
- Pregnant patients with hyperthyroidism
- Progression of exophthalmos; eye disease
- Ideally all cases

Goitre

Thyroid enlargement may be diffuse or multinodular. Diffuse causes include physiological, Graves disease, thyroiditis (Hashimoto or de Quervain), iodine deficiency or it can be hereditary.

Investigations include TFTs, needle biopsy, ultrasound and CXR. Management is supportive; thyroxine if TSH elevated (may lead to marked regression) and subtotal or total thyroidectomy.

Thyroid nodules

A thyroid nodule is defined as a discrete lesion on palpation and/or ultrasonography that is distinct from the rest of the thyroid gland.

Causes

- Dominant nodule in a multinodular goitre (most likely)
- Colloid cyst
- True solitary nodule: adenoma, carcinoma (papillary or follicular)

Investigations

- Ultrasound imaging
- Fine-needle aspiration cytology
- Thyroid function tests

Thyroid carcinoma[8]

The main presentations are a painless nodule, a hard nodule in an enlarged gland or lymphadenopathy. Papillary carcinoma is the most common malignancy. Although rare compared with non-malignant lesions (such as colloid nodules, cysts, haemorrhage and benign adenomas), it is important not to miss carcinoma because of the very high cure rate with expert-directed treatment. This often involves total thyroidectomy, ablative ^{131}I treatment, thyroxine replacement and follow-up with serum thyroglobulin measurements, ^{131}I/thallium scanning and neck ultrasound. Fine-needle aspiration is the investigation of choice.

Pituitary disorders

Pituitary tumours[9]

These account for 10% of intracranial tumours and are invariably benign adenomas. They can present with hormone deficiencies, features of hypersecretory syndromes (e.g. prolactin, GH, ACTH) or by local tumour mass symptoms (e.g. headache, visual field loss, seizures, cranial nerve 3, 4, 6 palsy).

Hyperprolactinaemia[11]

The main causes (of many) are a pituitary adenoma (prolactinoma; micro- or macro), pituitary stalk damage, drugs—such as antipsychotics, various antidepressants, metoclopramide, cimetidine, oestrogens, opiates, marijuana—and physiological causes such as pregnancy and breastfeeding.

Clinical features

- Symptoms common to males and females: reduced libido, subfertility, galactorrhoea (mainly females)
- Females: amenorrhoea/oligomenorrhoea
- Males: erectile dysfunction, reduced facial hair

Diagnosis

- Serum prolactin and macroprolactin assays

- MRI: consider if headache, etc

Refer for management, which may include a dopamine agonist such as cabergoline or bromocriptine, surgical resection (rarely necessary) or radiotherapy.

Acromegaly

Symptoms suggestive of acromegaly include:

- excessive growth of hands (increased glove size)
- excessive growth of tissues (e.g. nose, lips, face)
- excessive growth of feet (increased shoe size)
- increased size of jaw and tongue; kyphosis
- general: weakness, sweating, headaches
- sexual changes, including amenorrhoea and loss of libido
- disruptive snoring (sleep apnoea)
- deepening voice

> **DxT** nasal problems + fitting problems (e.g. rings, shoes) + sweating → acromegaly

Diagnosis[9,12]

- Plasma growth hormone excess
- Elevated insulin-like growth factor 1 (IGF-1) (somatomedin)—the key test
- X-ray skull and hands
- MRI scanning pituitary
- Consider associated impaired glucose tolerance/diabetes

Obtain old photographs (if possible).

Treatment options: transsphenoidal pituitary microsurgery, drugs and radiotherapy.

Diabetes insipidus and SIADH

Impaired secretion of vasopressin (antidiuretic hormone) from the posterior pituitary leads to

polyuria, nocturia and compensatory polydipsia, resulting in the passage of 3–20 L of dilute urine per day. There are several causes of diabetes insipidus (DI), the commonest being postoperative (hypothalamic-pituitary), which is usually transient only. Other causes of cranial DI include tumours, infections and infiltrations. In nephrogenic DI the kidney tubules are insensitive to vasopressin. Differential diagnosis includes compulsive (psychogenic) water drinking. The syndrome of secretion of inappropriate antidiuretic hormone (SIADH) is caused by cancer (e.g. lung, lymphomas, kidney, pancreas), pulmonary disorders, various intracranial lesions and drugs such as carbamazepine and many antipsychotic agents. Management of SIADH is essentially fluid restriction.

The treatment of DI is desmopressin, usually given twice daily intranasally.

DxT weakness + polyuria + polydipsia → diabetes insipidus

Hypopituitarism[8]

This rare disorder (acute or chronic) should be considered with:

- a history of postpartum haemorrhage or head injury
- symptoms of hypothyroidism
- symptoms of adrenal insufficiency
- symptoms suggestive of a pituitary tumour
- thin, wrinkled skin: ‘monkey face’
- pale ‘alabaster’ skin/hairlessness

Causes: pituitary adenoma, other parasellar tumours and inflammatory/infiltrative lesions.

DxT (female): amenorrhoea + loss of axillary and pubic hair + breast atrophy → hypopituitarism

DxT (male): ↓ libido + impotence + loss of body hair → hypopituitarism

Investigate with serum pituitary hormones, imaging (MRI) and triple stimulation test.

Treatment includes HRT, surgery or radiotherapy.

Adrenal disorders

The primary zones of the adrenal gland and their secretions

Cortex

- Zona glomerulosa—mineral corticoids, especially aldosterone
- Zona fasciculata—glucocorticoids
- Zona reticularis—androgens, especially DHEA

Medulla

- Catecholamines—epinepherine, norepinephrine

It is worth keeping in mind these uncommon disorders of the adrenal gland which can be difficult to diagnose in the early stages, namely:

- chronic adrenal insufficiency (Addison disease)—deficiency of cortisol and aldosterone
- Cushing syndrome—cortisol excess
- primary hyperaldosteronism (refer to CHAPTER 77)

Addison disease[8,13]

Autoimmune destruction of the adrenals is the most common cause; others are infection, e.g. TB or fungal.

Clinical features

- Lethargy/excessive fatigue/weakness
- Anorexia and nausea
- Diarrhoea/abdominal pain
- Weight loss
- Dizziness/funny turns, syncope: hypoglycaemia (rare); postural hypotension (common)
- Hyperpigmentation, especially mucous membranes of mouth and hard palate, skin creases of hands

If Addison disease remains undiagnosed, wasting leading to death may occur. Severe dehydration can be a feature. Delayed diagnosis is a huge problem. Hypertension and heart failure requires careful monitoring.

DxT fatigue + a/n/v + abdominal pain (± skin discolouration) → Addison disease

Diagnosis

- Elevated serum potassium, low serum sodium
- Low plasma cortisol level (fails to respond to synthetic adrenocorticotropic hormone [ACTH])
- The short synacthen stimulation test is the definitive test
- Consider adrenal autoantibodies and imaging? calcification of adrenals

Treatment: corticosteroid replacement—hydrocortisone/fludrocortisone acetate, other options.

Addisonian crisis[8,13]

An Addisonian crisis develops because of an inability to increase cortisol in response to stress, which may include intercurrent infection, surgery or trauma.

Clinical features

- Nausea and vomiting
- Acute abdominal pain
- Severe hypotension progressing to shock
- Weakness, drowsiness progressing to coma

Urgent management[13]

- Establish IV line with IV fluids
- Hydrocortisone sodium succinate 100 mg IV initially and 50–100 mg 4–6 hourly until stable
- Arrange urgent hospital admission

Cushing syndrome[8]

The five main causes are:

- iatrogenic—chronic corticosteroid administration
- pituitary ACTH excess (Cushing disease)
- bilateral adrenal hyperplasia
- adrenal tumour (adenoma, adenocarcinoma)
- ectopic ACTH or (rarely) corticotrophin-releasing hormone (CRH) from non-endocrine tumours (e.g. oat cell carcinoma of lung)

The clinical features are caused by the effects of excess cortisol and/or adrenal androgens.

Clinical features

- Proximal muscle wasting and weakness
- Central obesity, buffalo hump on neck
- Cushing facies: plethora, moon face, acne
- Weakness
- Hirsutism
- Abdominal striae
- Thin skin, easy bruising
- Hypertension
- Hyperglycaemia (30%)
- Menstrual changes (e.g. amenorrhoea)
- Osteoporosis
- Psychiatric changes, especially depression
- Backache

DxT plethoric moon face + thin extremities + muscle weakness → Cushing syndrome

Diagnosis (apart from iatrogenic cause)

- Cortisol excess (plasma or 24-hour urinary cortisol)

- Dexamethasone suppression test
- Late night salivary cortisol (2 measurements)
- Inferior petrosal sinus sampling
- Serum ACTH
- Radiological localisation: MRI for ACTH-producing pituitary tumours; CT scanning for adrenal tumours

Management

Ideally transsphenoidal excision of pituitary tumour. Pharmacological blockade of corticosteroid production may be necessary, ketoconazole (o) is first line.

Primary hyperaldosteronism[8]

Most commonly due to an adrenal adenoma.

Conn syndrome

Usually asymptomatic and hypertensive but any symptoms are features of hypokalaemia:

- weakness, headaches
- palpitations
- cramps
- paraesthesia
- polyuria and polydipsia

Investigations

- Aldosterone (serum and urine) ↑
- Plasma renin ↓
- Plasma aldosterone to renin activity ratio
- Na ↑, K ↓, alkalosis
- Imaging (MRI or CT scan) of adrenals

Refer for treatment including possible surgery to excise adenoma. Spironolactone to prepare for surgery.

Phaeochromocytoma[8,12]

A dangerous tumour of the adrenal medulla. Clinical features are paroxysms or spells of:

- anxiety
- hypertension
- headache (throbbing); tremor
- sweating
- palpitations
- pallor/skin blanching
- rising sensation of tightness in upper chest and throat (angina can occur)

DxT episodic headache + sweating + tachycardia → phaeochromocytoma

Investigations

- Series of three 24-hour free catecholamines ↑ VMA
- Abdominal CT or MRI scan (both highly sensitive)

Treatment

- Excise tumour, cover with alpha and beta blockers

Congenital adrenal hyperplasia (adrenogenital syndrome)[6,8]

An AR condition with 21-hydroxylase deficiency being the most common of several forms. There is inadequate synthesis of cortisol and aldosterone with increased androgenisation. Major problem is adrenal failure ± salt-losing state (SLS). In females, ambiguity of external genitalia and hirsutism before puberty usually occurs. Males may have normal urogenital development but SLS is a concern. Infants of either sex may present with failure to thrive or vomiting and dehydration (SLS). Lifelong glucocorticoid treatment (e.g. prednisolone) is required. Wearing an alert bracelet or necklace is strongly recommended for these patients (www.medicalert.org.au).[6]

Adrenal tumours[9]

Most of those detected by abdominal imaging are benign and termed ‘incidentalomas’ but

serious tumours include adrenal carcinoma, phaeochromocytoma, neuroblastoma, glucocorticoid or a mineralocorticoid secreting tumour.

Rule: tumours >4 cm require thorough assessment as malignant tumours are large. Excision is usually advisable.

Incidentalomas

These are adrenal tumours ≥1 cm. Most are benign and non-functioning. An important issue is malignancy, and if this is the case, whether it is primary, secondary or functional (hormone secreting).

Investigations to consider include electrolytes, aldosterone/renin ratio, catecholamines, testosterone, DHEAs, dexamethasone suppression test, CT scan. Surgical excision should be considered under specialist guidance.

Calcium disorders

Hypercalcaemia[12,14]

Suspect hypercalcaemia if there is weakness, tiredness, malaise, anorexia, nausea or vomiting, abdominal pain, loin pain, constipation, thirst, fever, polyuria, drowsiness, dizziness, personality changes, muscle aches and pains, visual disturbances. Measure urea and electrolytes (especially calcium), creatinine, albumin.

Primary hyperparathyroidism, familial hypercalciuric hypercalcaemia and neoplasia, especially carcinoma of lung and breast (with metastases to bone), account for over 90% of cases. Other causes include Paget disease, Williams syndrome, prolonged immobilisation, dehydration, sarcoidosis and milk-alkali syndrome. Investigations include ESR, serum parathyroid hormone (N: 1.0–7 pmol/L), serum ACE levels, serum alkaline phosphatase, chest X-ray, Sestamibi scan and bone scan. Requires specialist referral.

DxT weakness + constipation + polyuria → hypercalcaemia

DxT cramps + confusion + tetany → hypocalcaemia

Primary hyperparathyroidism[12]

Hyperparathyroidism is caused by an excessive secretion of parathyroid hormone and is usually due to a parathyroid adenoma. The classic clinical features of hyperparathyroidism are due to the

effects of hypercalcaemia. Rarely, a parathyroid crisis in a misdiagnosed patient may result in death from severe hypercalcaemia.

> Classic mnemonic: bones, moans, stones, abdominal groans

Diagnosis

- Exclusion of other causes of hypercalcaemia
- Serum parathyroid hormone (elevated)
- TC-99m Sestamibi scan to detect tumour

Treatment

Refer for possible surgical management.

Hypocalcaemia[8,14]

Causes include parathyroid injury, autoimmune hyperparathyroidism, severe vitamin D deficiency and neonates of mothers with hypercalcaemia. This usually presents with tetany or more generalised neuromuscular hyperexcitability and neuropsychiatric manifestations. The sensory equivalents are paraesthesia in the hands, feet and around the mouth (distinguish from tetany seen in the respiratory alkalosis of hyperventilation). There may be seizures and cramps. The diagnosis is by measurement of serum total calcium concentration in relation to serum albumin (s. calcium <2.10 mmol/L).

Two important signs are:

- Trousseau sign: occlusion of the brachial artery with BP cuff precipitates carpopedal spasm (wrist flexion and fingers drawn together)
- Chvostek sign: tapping over parotid (facial nerve) causes twitching in facial muscles

Treatment involves careful adjustments in dosage of calcitriol and calcium to correct hypocalcaemia and avoid hypercalcaemia and hypercalciuria (the latter may lead to kidney impairment).

Hypoparathyroidism

Hypoparathyroidism is the most common cause of hypocalcaemia. Causes include postoperative thyroidectomy and parathyroidectomy, congenital deficiency (DiGeorge syndrome) and idiopathic (autoimmune) hypoparathyroidism. The main features are neuromuscular hyperexcitability, tetany and neuropsychiatric manifestations.

Other electrolyte disturbances

Hypernatraemia Na^+ >145 mmol/L

Causes

- Water depletion (e.g. diabetes insipidus)
- Water and sodium depletion (e.g. diarrhoea)
- Corticosteroid excess (e.g. Cushing syndrome, Conn syndrome)
- Iatrogenic: excess IV hypertonic Na solutions

Clinical features

- Thirst, confusion, lethargy, weakness, irritability, oliguria
- Orthostatic hypotension
- Muscle twitching or cramps
- Signs of dehydration
- Severe: seizures, delirium, hyperthermia, coma

Hyponatraemia Na^+ <135 mmol/L

Causes

- Water retention (e.g. CCF, hypoalbuminaemia)
- Kidney failure to conserve salt (e.g. nephritis, diabetes mellitus, Addison disease)
- Gastrointestinal loss of Na^+ (e.g. vomiting, diarrhoea)
- Drugs (e.g. diuretic excess, ACE inhibitors)

Clinical features

- Asymptomatic when mild
- Anorexia, nausea, lethargy, confusion, headache, ataxia, mental changes (e.g. in personality)
- Severe: convulsions, coma, death