examination MEDECINE
8th edition
A guide to physician training

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ELSEVIER
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Foreword by Professor Geoffrey Metz

The Royal Australasian College of Physicians (RACP) was established in 1938 with a core responsibility to train medical specialists including adult physicians and paediatricians.

In the initial decades, the expectation was that trainees should accumulate a vast bank of knowledge through extensive reading of prescribed textbooks and should concurrently acquire clinical skills through a process of observation and learning via the time-honoured apprenticeship model, promoted by Hippocrates over 2000 years ago.

This process was seen as ‘the best system available’, and indeed those passing the Membership (MRACP) or more recently the Fellowship (FRACP) were then and still are now seen internationally as highly trained and skilled practitioners.

However, assessment of the level of knowledge and skills was criticised at that time as lacking objectivity, and we all observed colleagues who had been seen as good clinicians but repeatedly failed the assessment at the end of their training. This uncertainty of outcome despite many years of study and clinical work produced enormous levels of stress for trainees.

The medical apprenticeship model remains the keystone for acquisition of the knowledge, skills and experience needed to become an expert specialist physician, but in recent decades there have been progressive and major advances in medical education that have included better definition of curricula, improved teaching and learning techniques to facilitate more rapid acquisition of the necessary knowledge and skills, and greater objectivity of assessment – all leading to improved predictability and fairness for trainees.

Just on thirty years ago, the first edition of Examination Medicine was published as a guide for trainees preparing for Fellowship of the RACP. It briefly described the requirements for basic physician training and discussed the written examination, but its main thrust was a comprehensive approach to the clinical examination.

Since that time, medical education has continued to evolve internationally, the RACP has concurrently introduced changes to its training and assessment requirements, and there have been seven further editions of Examination Medicine, each offering insightful advice as to how trainees should prepare for and then conduct themselves through the assessment process.

This edition also introduces the rationale behind and details of assessment for basic physician training in the first four chapters and outlines helpful tips on appropriate preparation for examination, as well as outlining the approach that examiners like to see from the well-prepared and professionally skilled candidate.

Subsequent chapters outline the important conditions within each subspecialty of medicine with which examination candidates should be fully familiar, with expert description of typical clinical presentations, examination findings, investigation abnormalities expected and up-to-date management commentary. There are also detailed lists of the conditions that are most commonly seen in the physician examination setting.

There have been a number of important changes since the last edition and this book will continue to be a hugely valuable resource for trainees preparing for the RACP examinations.
I note from discussions with current registrars that many in specialties other than physician training, such as anaesthetic trainees and FRACGP trainees, are increasingly turning to Examination Medicine as a valuable resource as they approach their college examinations.

Whilst trainees still do need to put in long hours of preparation to pass the FRACP examinations, this book gives important additional advice and factual detail that will often give the candidate the knowledge and the confidence to complete the examinations successfully.

Professor Geoffrey Metz
AM, MBBS, FRACP, MD, FRCP, FACP, FRCPI (Hon)
Professor of Medicine, The University of Melbourne
Dean of Medicine, Epworth Healthcare
Past President, Royal Australasian College of Physicians
Foreword by Professor John Kolbe

Despite changes to assessment that have taken place in the last decade, the clinical examination of the Royal Australasian College of Physicians (RACP) still represents a significant barrier for trainees in their journey to becoming a physician, a barrier that must be overcome before advanced training (PREP-AT) can commence. The examination was a memorable experience – one way or another! Most physicians, even those nearing the end of their professional careers, can clearly, and sometimes vividly, remember the cases that they were asked to assess in their clinical examination. The first edition of Examination Medicine appeared in 1986 and thus it is not an exaggeration to say that a generation of physician trainees have benefitted from the sound advice contained therein.

Despite this edition being published at a time when medical technology is celebrated for its influence on patient diagnosis and management, and when the advent of telemedicine has challenged the role of and requirement for ‘hands-on’ clinical examination, the need for a text such as this is undiminished. Most diagnoses in day-to-day care of patients are still made on the basis of accurate history-taking and physical examination, and simple investigations. Indeed, competent clinical assessment and good clinical reasoning remain paramount and are likely to remain so in a health system that is increasingly focused on ‘value for money’ and the avoidance of harm to patients. The importance of excellent clinical skills, clinical reasoning and synthesis of clinical material is even more important today in delivering healthcare, not only in internal medicine, but also in a variety of other specialties, in a variety of clinical settings and for a very wide variety of clinical problems. The use of modern technology is neither an appropriate nor cost effective alternative.

The format of Examination Medicine is similar to previous editions, with an outline of the clinical assessment process and then examination approaches to likely long and short cases, organised by systems. Not only does the book provide very useful guides to the approaches to the various clinical conditions that the examination candidate might encounter, it also provides advice to candidates on how to conduct themselves in the examination, how to make best use of their time, and how to avoid pitfalls. As Osler so eloquently said:

Observe, record, tabulate, communicate. Use your five senses. Learn to see, learn to hear, learn to feel, learn to smell, and know that by practice alone you can become expert.

The result should be the carrying out of a rational and thoughtful history, the performance of an organised and disciplined physical examination and the development, through a hypothetico-deductive approach, of a parsimonious differential diagnosis list and a discussion that both satisfies the examiners and will serve the interest of future patients.
This edition continues the tradition of previous editions, with a systematic and thorough approach, a clear and logical style, the avoidance of esotericism, and well-chosen illustrations in an appropriate-sized volume. An interesting aspect is that the authors encourage the trainee to view the examination process through the eyes of an examiner.

Despite the examination emphasis, there is a great deal of clinical medicine to be learned from this volume. The knowledge and skills obtained through this book will stand the reader in good stead for the whole of his or her professional career. The book recognises that the full range of clinical and humanistic skills required of a physician are acquired, in a large part, through the assessment of, and reflection upon, the presentation and clinical course of many, many patients. To quote Ossler again:

He who studies medicine without books sails an unchartered sea, but he who studies medicine without patients does not go to sea at all.

By obtaining this book, you will receive information and instruction, but only if you absorb its contents, reflect upon its advice and put it all into clinical practice. In that way you will derive the same benefits as earlier cohorts of aspiring physicians have obtained from earlier editions of this fine book.

Professor John Kolbe
MBBS, FRACP
Head of Department of Medicine, Faculty of Medical and Health Sciences, University of Auckland
Respiratory physician, Auckland City Hospital
Past President, Royal Australasian College of Physicians
Preface to the 8th edition

Practising medicine of the highest standard is both art and science; physicians are meant to think and think deeply. A mature clinical approach requires you to understand each patient’s unique personal and social environment, and complex medical problem solving must be considered in this context. Use this book as a help for your preparation, not something to be learned by heart.

Talley & O’Connor, 2014

Welcome to the new edition of Examination Medicine. We deeply appreciate the continued high interest in this book by basic trainees (candidate specialists) sitting for the Royal Australasian College of Physicians (RACP) written and clinical barrier examinations. The RACP examinations are among the most rigorous in the world (especially the clinical), and those who pass them are justifiably proud of the achievement all of their lives. We are also aware our book is very popular with candidates from many other specialist colleges who are required to successfully master long and short cases, from emergency medicine to psychiatry. Finally, we are delighted that senior medical students have found the book very useful as they polish their clinical skills for long cases and objective structured clinical examinations (OSCEs).

A new edition always gives us the chance to update and freshen the book, and we have worked to incorporate new knowledge, add more useful hints and tips, and eliminate less useful material. We are very grateful to the many peer reviewers who over the years have provided invaluable feedback including for this edition, and we also thank readers who have gone to the trouble to write to us with suggestions and recommendations. In the 8th edition we have updated and added new long cases, included a new guide on possible lines of questioning by the examiners for each case, inserted further points to consider as you prepare case presentations, revised the short cases as needed, in particular parts of the neurology examination that candidates still often find difficult, and expanded our chapter on how examiners think (so you the prepared candidate can, we hope, ace the exam). We have also added additional long- and short-case videos to help guide your learning.

First published in 1986 not long after we had both entered advanced RACP training, we have seen remarkable changes in medicine over the last 30 years since the first edition of Examination Medicine. However, what has not changed is the need for all physicians to have excellent communication skills, an empathic and person-centred approach to care, and the ability to masterfully apply the clinical skills of history taking and physical examination, which are still required for accurate diagnosis and optimal management in most cases. Competence in all of these skills is what is required to be a specialist physician and is what the RACP clinical examination aims to test. As we said in the last edition: ‘Preparation is the key to success and, to quote Winston Churchill, “Never give in. Never. Never. Never. Never.”’
We wish everyone using this book the very best of luck as they progress in their training and sit the required examinations. We have found being a part of the College community very rewarding; once you have passed, we encourage you to become an active College member and give back. Volunteer for a committee, become an excellent educator, work with your friends and colleagues and contribute to the future – in this way you can make a difference not just to medicine but also to our community.

Nicholas J Talley
Simon O’Connor
Newcastle and Canberra, 2017

Authors’ statement

Professor Nick Talley is a Past President of the Royal Australasian College of Physicians (RACP) (2014–2016), a local RACP examiner, and Chair of the College of Presidents of Medical Colleges (CPMC). Dr Simon O’Connor is a member of the RACP Senior Examination Panel (SEP). This book, first published in 1986, is not a College publication, nor is it endorsed by the RACP. Trainees should directly consult the College website to obtain up-to-date information about policy and procedures as these are subject to regular change.
Acknowledgements

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## Abbreviations

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<th>Abbreviation</th>
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<tr>
<td>AALF</td>
<td>agraphia, left–right disorientation, finger agnosia</td>
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<td>ABP</td>
<td>ambulatory blood pressure</td>
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<td>ABVD</td>
<td>adriamycin, bleomycin, vinblastine and dacarbazine</td>
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<tr>
<td>ACE</td>
<td>angiotensin-converting enzyme</td>
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<td>ACEI</td>
<td>angiotensin-converting enzyme inhibitor</td>
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<td>AChR</td>
<td>acetylcholine receptor</td>
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<td>ACPA</td>
<td>anti-citrullinated protein antibody</td>
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<td>ACTH</td>
<td>adrenocorticotropic hormone</td>
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<td>ADL</td>
<td>activities of daily living</td>
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<td>ADP</td>
<td>adenosine diphosphate</td>
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<td>ADPKD</td>
<td>autosomal dominant polycystic kidney disease</td>
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<td>atrial fibrillation</td>
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<td>acid-fast bacilli</td>
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<td>AHI</td>
<td>apnoea hypopnoea index</td>
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<td>AICD</td>
<td>automatic implantable cardioverter-defibrillator</td>
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<td>acquired immunodeficiency syndrome</td>
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<td>autoimmune haemolytic anaemia</td>
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<td>ALK1</td>
<td>activin receptor-like kinase type 1</td>
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<td>AMSAP</td>
<td>Adult Medicine Self-Assessment Programme</td>
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<td>AST to platelet ratio index</td>
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<td>aPTT</td>
<td>activated partial thromboplastin time</td>
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<td>angiotensin II receptor blocker</td>
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<td>aortic stenosis</td>
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<tr>
<td>ASAP</td>
<td>Australian Self-Assessment Programme</td>
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<tr>
<td>ASCA</td>
<td>anti-	extit{Saccharomyces cerevisiae} antibodies</td>
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<td>β2-GP-1</td>
<td>beta2-glycoprotein-1</td>
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BCG  bacille Calmette–Guérin
BD    twice a day
BGL   blood glucose level
BiPAP bilevel positive airways pressure
BMD   bone mineral density
BMI   body mass index
BMPR  bone morphogenetic protein receptor type 2
BMS   bare metal stent
BNP   B-type natriuretic peptide
BPPV  benign paroxysmal positional vertigo
BSL   blood sugar level
CABG  coronary artery bypass graft
CAD   coronary artery disease
CAIHA cold autoimmune haemolytic anaemia
CAPD  continuous ambulatory peritoneal dialysis
CCF   congestive cardiac failure
CCP   citrullinated cyclic peptide
CEA   carcinoembryonic antigen
CFE   Committee for Examinations
CIDP  chronic inflammatory demyelinating polyradiculoneuropathy
CKD   chronic kidney disease
CLD   chronic liver disease
CMC   carpometacarpal
CML   chronic myeloid leukaemia
CMT   Charcot–Marie–Tooth
CMV   cytomegalovirus
CNI   calcineurin inhibitor
CNS   central nervous system
COP   cryptogenic organisng pneumonia
COPD  chronic obstructive pulmonary disease
COX-2 cyclo-oxygenase 2
CPAP  continuous positive airways pressure
CPT   Committee for Physician Training
Cr    creatinine
CRAB  hypercalcaemia, renal disease, anaemia and bone lytic lesions
CREST calcinosis cutis; Raynaud’s phenomenon; (o)esophageal involvement;
sclerodactyly; telangiectasia
CRH   corticotrophin-releasing hormone
CRP   C-reactive protein
CRT   cardiac resynchronisation therapy
CS    coronary sinus
CSF   cerebrospinal fluid
CSII  continuous subcutaneous infusion
CT    computed tomography
CTEPH chronic thromboembolic pulmonary hypertension
CTPA  computed tomography pulmonary angiogram
CVA   cerebrovascular accident
CVP   cyclophosphamide, vincristine and prednisone
CXR   chest X-ray
DAF   decay-accelerating factors
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<td>DAPT</td>
<td>dual anti-platelet treatment</td>
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<td>DC</td>
<td>direct current</td>
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<td>DCD</td>
<td>donation after cardiac death</td>
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<tr>
<td>dcSSc</td>
<td>diffuse cutaneous systemic sclerosis</td>
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<tr>
<td>DES</td>
<td>drug-eluting stent</td>
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<tr>
<td>DEXA</td>
<td>dual-energy X-ray absorptiometry</td>
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<td>DIC</td>
<td>disseminated intravascular coagulation</td>
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<td>DIP</td>
<td>distal interphalangeal</td>
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<td>DLCO</td>
<td>diffusion capacity for carbon monoxide</td>
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<td>DLE</td>
<td>discoid lupus erythematosus</td>
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<td>DMARD</td>
<td>disease-modifying, antirheumatic drug</td>
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<tr>
<td>DMOAD</td>
<td>diabetes insipidus, diabetes mellitus, optic atrophy and deafness</td>
</tr>
<tr>
<td>DOAC</td>
<td>direct oral anticoagulant</td>
</tr>
<tr>
<td>DOT</td>
<td>direct observed treatment</td>
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<tr>
<td>DPE</td>
<td>Director of Physician Education</td>
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<tr>
<td>DPPIV</td>
<td>dipeptidyl peptidase IV</td>
</tr>
<tr>
<td>DPT</td>
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<td>dsDNA</td>
<td>double-stranded DNA</td>
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<tr>
<td>DVT</td>
<td>deep venous thrombosis</td>
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<td>DWI</td>
<td>diffusion-weighted image</td>
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<td>EBV</td>
<td>Epstein–Barr virus</td>
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<tr>
<td>ECG</td>
<td>electrocardiogram</td>
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<td>ECOG</td>
<td>Eastern Cooperative Oncology Group</td>
</tr>
<tr>
<td>EF</td>
<td>ejection fraction</td>
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<td>EGPA</td>
<td>eosinophilic granulomatosis with polyangiitis</td>
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<td>EIA</td>
<td>enzyme immunoassay</td>
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<td>ELISA</td>
<td>enzyme-linked immunosorbent assay</td>
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<td>EMG</td>
<td>electromyogram</td>
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<tr>
<td>EMQ</td>
<td>extended matching question</td>
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<tr>
<td>ENA</td>
<td>extractable nuclear antigen</td>
</tr>
<tr>
<td>EPG</td>
<td>electrophoretogram</td>
</tr>
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<td>EPS</td>
<td>electrophysiological studies</td>
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<td>ERCP</td>
<td>endoscopic retrograde cholangiopancreatography</td>
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<td>ES</td>
<td>educational supervisor</td>
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<tr>
<td>ESA</td>
<td>erythropoietin-stimulating agent</td>
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<td>ESR</td>
<td>erythrocyte sedimentation rate</td>
</tr>
<tr>
<td>FAP</td>
<td>familial adenomatous polyposis</td>
</tr>
<tr>
<td>FBC</td>
<td>full blood count</td>
</tr>
<tr>
<td>FET</td>
<td>forced expiratory time</td>
</tr>
<tr>
<td>FEV1</td>
<td>forced expiratory volume in 1 second</td>
</tr>
<tr>
<td>FFP</td>
<td>fresh frozen plasma</td>
</tr>
<tr>
<td>FHH</td>
<td>familial hypocalciuric hypercalcaemia</td>
</tr>
<tr>
<td>FODMAP</td>
<td>fermentable oligosaccharides, disaccharides, monosaccharides and polyols</td>
</tr>
<tr>
<td>FRACP</td>
<td>Fellow of the Royal Australasian College of Physicians</td>
</tr>
<tr>
<td>FS</td>
<td>fractional shortening</td>
</tr>
<tr>
<td>FSGS</td>
<td>focal segmental glomerulosclerosis</td>
</tr>
<tr>
<td>FSH</td>
<td>follicle-stimulating hormone / facioscapulohumeral</td>
</tr>
<tr>
<td>5FU</td>
<td>5-fluorouracil</td>
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</table>
FVC  forced vital capacity
G6PD  glucose-6-phosphate dehydrogenase
GADA  glutamic acid decarboxylase antibody
GALS  gait, arms, legs and spine
GBM  glomerular basement membrane
GFR  glomerular filtration rate
GGT  gamma-glutamyl transferase
GH  growth hormone
GI  glycaemic index/gastrointestinal
GIT  gastrointestinal tract
GLP-1  glycogen-like peptide
GM-CSF  granulocyte-macrophage colony stimulating factor
GN  glomerulonephritis
GOLD  Global Initiative for Chronic Obstructive Lung Disease
GORD  gastro-oesophageal reflux disease
GPI  glycosylphosphatidylinositol
GTH  general teaching hospital
GUG  get up and go
GVHD  graft versus host disease
HAART  highly active antiretroviral therapy
Hb  haemoglobin
HBc  hepatitis B core
HBs  hepatitis B surface
HBV  hepatitis B virus
HCC  hepatocellular carcinoma
HCM  hypertrophic cardiomyopathy
HCV  hepatitis C virus
HD  haemodialysis
HDL  high-density lipoprotein
HELLP  haemolysis, elevated liver enzymes, low platelets
Hib  *Haemophilus influenzae* type b
HIV  human immunodeficiency virus
HLA  human leukocyte antigen
HMG-CoA  hydroxymethylglutaryl coenzyme A
HMSN  hereditary motor and sensory neuropathy
HNPPCC  hereditary non-polyposis colon cancer
HPL  human placental lactogen
HPO  hypertrophic pulmonary osteoarthropathy
HRCT  high-resolution computed tomography
HRS  hepatorenal syndrome
HSV  herpes simplex virus
HT  hypertension
HUS  haemolytic uraemic syndrome
HZV  herpes zoster virus
IA-2  insulinoma-associated protein 2 antibody
IAA  insulin autoantibody
IBD  inflammatory bowel disease
IBS  irritable bowel syndrome
ICD  implantable cardioverter-defibrillator
ICU  intensive care unit
Chapter 5

The cardiovascular long case

A rule of thumb in the matter of medical advice is to take everything any doctor says with a grain of aspirin.

Goodman Ace (1899–1982)

Ischaemic heart disease

Patients with recent acute coronary syndromes (ASCs) including myocardial infarction are always available for long cases if required. Many with more exotic medical problems will also have ischaemic heart disease. The whims of the long-case examiners may lead to concentrated questioning about the ischaemic heart disease of a patient in hospital for the management of, say, renal transplant rejection. These patients are more likely to present management rather than diagnostic problems once they reach the status of long-case patients.

The classification of patients with episodes of acute coronary ischaemia is based on electrocardiogram (ECG) changes and on the detection of markers of myocardial damage (troponins), which have prognostic as well as diagnostic usefulness for patients with chest pain. Those who present with chest pain and ECG changes of ST elevation have an ST elevation myocardial infarction (STEMI). Those without ST elevation are said to have a non-ST elevation acute coronary syndrome (NSTEACS), but once abnormal cardiac markers have been detected the diagnosis can be revised to a non-ST elevation myocardial infarction (non-STEMI). The diagnosis unstable angina is no longer part of this classification, but is still often used to describe patients with increasing exertional angina.

Patients with ST elevation benefit from urgent action to re-open the blocked coronary artery (angioplasty or thrombolytic treatment). Those with non-STEMI are usually treated medically in the first instance. The presence of abnormal cardiac markers indicates an adverse prognosis (increased risk of further infarction or death) and these patients benefit from early but not immediate intervention (angioplasty or coronary surgery) and from immediate aggressive anti-platelet treatment and anticoagulation with fractionated or unfractionated heparin. Non-STEMI patients who have ST depression on the ECG have a worse prognosis than do those with T wave inversion or flattening. The concept of risk stratification is based on these factors and determines the urgency and type of treatment.
**The history**

1. Find out whether the patient has been or is in hospital because of a recent myocardial infarction or an acute coronary syndrome, or for some other cardiac or non-cardiac reason.

2. The patients with the worst prognosis are those with chest pain and ECG changes at rest (Table 5.1). Clearly, these may represent different pathophysiological states, varying from occlusion of a coronary artery and inadequate collateral flow to rupture of a lipid-rich plaque with thrombus formation. Ask about obvious precipitating factors, such as a gastrointestinal bleed or the onset of an arrhythmia. Also ask about the character of the chest pain and what precipitated the admission.

| Table 5.1 Risk stratification in patients with ischaemic chest pain at rest |
|-----------------------------|-----------------------------|-----------------------------|-----------------------------|-----------------------------|
| HIGHEST TO LOWEST RISK      | 1. ST elevation myocardial infarction |
|                             | 2. ST depression             |
|                             | 3. T wave inversion          |
|                             | 4. Non-specific ST–T wave changes |
|                             | 5. Normal ECG                |
| • The risk is higher in each group if cardiac biomarkers (troponins) are elevated. |
| • The risk is higher in each group for patients with previous ischaemic heart disease or diabetes. |
| • The higher the risk, the more the benefit of aggressive treatment. |

Remember that the diagnosis of angina can be suspected from the history, but needs to be established by investigations – an abnormal ECG or exercise test at least. You should be suspicious of the diagnosis unless it has been confirmed by investigations. The most common differential diagnosis is gastro-oesophageal reflux disease (GORD). This can be difficult to prove without endoscopy (and, if normal, oesophageal pH testing), but an excellent response to a trial of a proton pump inhibitor (PPI) is very suggestive. Oesophageal spasm is another cause of central chest pain.

3. Detail the patient’s current treatment and management history. Oral medications will probably include:
   - aspirin with or without an ADP inhibitor (clopidogrel, prasugrel, ticagrelor)
   - a beta-blocker or occasionally a calcium antagonist
   - nitrates (intravenous, oral or topical)
   - statin
   - an angiotensin-converting enzyme (ACE) inhibitor (ACEI) or angiotensin II receptor (AR) blocker (ARB).

   a. Acute coronary syndromes are managed with heparin and aspirin and clopidogrel, prasugrel or ticagrelor. Remember that prasugrel should not be used in patients older than 75 or in patients who have had a haemorrhagic stroke.

      Thrombolytic treatment is not effective for NSTEACS. This is possibly because acute coronary syndromes are not a single pathological entity and also because a state of increased thrombogenesis may follow initial thrombolysis with these drugs.

      Most patients have early angiography (within 48 hours) with the intention of angioplasty to the culprit lesion if this is practical. Ask whether the patient knows details of what investigations or treatment were performed.

   b. If the patient has had an infarct during this or previous admissions find out about the management, which may have included primary angioplasty or thrombolysis,
and treatment of complications such as arrhythmias, cardiac failure, further angina and embolic events.

c. In many hospitals a comprehensive cardiac rehabilitation program will have been offered to the patient. Ask whether this has been helpful and ask about the hospital staff’s explanations to the patient about his or her condition and prognosis. Also ask questions about the effect of this illness on the patient’s life and work.

4. Next ask standard questions about risk factors in addition to age and male sex. Remember that risk factors are of vital importance to long-term prognosis, but add little to the likelihood that undiagnosed chest pain is ischaemic. Risk factors include:

- previous ischaemic heart disease
- hyperlipidaemia
- diabetes mellitus (the increased risk in these patients is as high as that in non-diabetics who have already had an ischaemic event)
- hypertension
- family history (in particular, first-degree relatives with ischaemic heart disease before the age of 60; 92-year-old great-uncles with heart trouble do not count)
- smoking (how many; if stopped, how long ago – risk of infarction is no longer increased after 1 year and that of angina after 10 years)
- use of oral contraceptives or premature onset of menopause
- obesity and physical inactivity
- high serum homocysteine levels, which may have been measured if the patient has premature coronary disease and few other risk factors – levels in the top population quintile increase coronary risk twofold; trials of treatment (mostly with folate), however, have been negative and routine treatment is not recommended
- long-term use, in high doses, of cyclo-oxygenase 2 (COX-2) inhibitors or other non-steroidal anti-inflammatory drugs (NSAIDs) (which should be stopped)
- erectile dysfunction (which often precedes symptomatic ischaemic heart disease and is a marker of endothelial dysfunction). Remember that the presence of multiple risk factors is more than additive.

5. Then find out whether risk factor control has been successful. Remember the important results of recent secondary prevention trials.

a. Aggressive cholesterol lowering to below a level of 4 mmol/L of total cholesterol (low-density lipoprotein (LDL) < 1.8) is now considered appropriate for patients with established coronary disease.

b. There is some evidence that statins have beneficial effects beyond their effect on cholesterol levels (pleiotrophic effects).

6. Find out what investigations the patient can remember.

a. An echocardiogram may have been performed to assess ventricular function and possible complications of infarction, such as a pericardial collection, a left ventricular thrombus, mitral regurgitation or a ventricular septal defect (VSD).

b. An exercise test, a sestamibi or a stress echocardiogram may have been performed to assess ischaemia or myocardial viability (MRI scan).

c. Cardiac catheterisation is perhaps the most memorable of the investigations for ischaemic heart disease.

The patient may know how many coronaries are abnormal and whether angioplasty was performed. Ask whether a drug-eluting stent (DES) was used and for how long dual anti-platelet treatment was recommended. Remember clopidogrel, and most PPIs, use the same metabolic pathway in the liver and if used together may result in a theoretical loss of anti-platelet benefit. The clinical relevance of this is disputed.
7. Complications such as acute mitral regurgitation or an infarct-related VSD are usually treated surgically but have a relatively poor prognosis. All complications are less common if early coronary patency and normal flow have been achieved.

**The examination**
Examine the cardiovascular system (Ch 16).
1. Record the blood pressure.
2. Look for signs of valvular heart disease, cardiac failure, rhythm disturbances (e.g. atrial fibrillation (AF), frequent ectopic beats) and murmurs suggesting mitral regurgitation or a VSD caused by an infarct.
3. There may be spectacular bruises at venepuncture or femoral or radial puncture sites if the patient has had thrombolytic treatment. Abdominal wall bruising suggests subcutaneous low-molecular-weight heparin therapy. Occasionally the radial pulse may be absent after radial angioplasty. More general complications include a stroke owing to embolism from the heart.

**Management**
It is best to concentrate on discussing the management of the presenting problem. If the patient has only recently been admitted with an infarct, this means a discussion of thrombolysis and primary angioplasty.

1. Candidates should have some knowledge of the major thrombolysis and angioplasty trials.
   a. These have shown that early treatment has improved mortality. Treatment up to 12 hours after the onset of an infarct is worthwhile.
   b. Alteplase and reteplase have been shown to produce a small survival advantage compared with streptokinase, probably because they are more effective in opening occluded vessels, but have a slightly increased risk of causing cerebral haemorrhage.
   c. Alteplase is given as a bolus followed by an infusion, and reteplase is given as a double bolus injection with a 30-minute interval. Even when thrombolysis seems successful (resolution of symptoms and ST depression) patients are now routinely transferred so that angiography can be performed as soon as practical.

2. Urgent coronary (primary) angioplasty, if available, is of proven benefit and has been shown to reduce mortality compared with treatment with thrombolytic drugs.
   a. The advantages, theoretical and real, include definite re-opening of the infarct-related artery in more than 90% of patients (compared with <60% of patients given thrombolitics), normal flow in the infarct-related artery in most cases, dilation and stenting of the offending (culprit) lesion and often removal of clot, very low risk of stroke and shortening of hospital stay, often to just 3 days.
   b. Patients are treated with potent anti-platelet drugs: aspirin, clopidogrel (or prasugrel or ticagrelor) and sometimes with one of the platelet aggregation inhibitors, abciximab or tirofiban. Prasugrel is more rapidly effective than clopidogrel and in many protocols is now preferred for primary angioplasty patients. Ticagrelor may improve prognosis compared with the other drugs. Its most common side-effect is dyspnoea, which may develop after 5–10 days.
   c. There is now trial evidence that transport of patients to a hospital where this procedure can be performed is preferable to treatment with thrombolytic drugs, if transport time is less than 2–3 hours.
   d. Rapid transport to the catheter laboratory is important and the ‘door to balloon’ time should be less than 90 minutes when angioplasty is available in the hospital to which the patient presented.
Recent trials have not shown a benefit for routine thrombus aspiration for primary angioplasty procedures.

If the history has suggested complications resulting from the infarct, these will have to be discussed. Common complications include:

- ventricular arrhythmias
- bradyarrhythmias (especially following an inferior infarct)
- cardiac failure
- further ischaemia or reinfarction.

It is important to have planned an approach to the management of these problems.

Investigations

These are aimed at assessment of the infarct size, complications and presence of further ischaemia:

1. left ventricular function – echocardiogram, left ventriculogram
2. complications – echocardiogram for valvular regurgitation, left ventricular thrombus, infarct-related VSD
3. further ischaemia – exercise test, sestamibi stress test, cardiac catheterisation
4. viability – MRI scan, sestamibi scan.

Long-term treatment

1. Early revascularisation is of proven benefit for high-risk patients with acute coronary syndromes (ST elevation, troponin elevation).
2. Prognosis is improved with aspirin, beta-blockers and, for large infarcts (ejection fraction <40%), ACE inhibitors and beta-blockers (e.g. carvedilol, bisoprolol, nebivolol and extended-release metoprolol).
3. Patients with three-vessel disease and significant left ventricular damage or with left main coronary artery stenosis benefit prognostically from coronary artery bypass surgery even if their symptoms have settled on medical treatment. Those with tight proximal (before the first diagonal branch) left anterior descending lesions probably also benefit from surgery or angioplasty.
4. Epleronone, an aldosterone antagonist, is indicated for patients with cardiac failure following an infarct.

Secondary prevention

1. Control of cardiac risk factors is even more important once the presence of coronary artery disease has been established. It should be a routine part of the management.
2. Dietary advice for weight and lipid reduction may be indicated. Lipid-lowering drug treatment with a statin should be introduced for all patients who can tolerate it.
3. Patients should be encouraged to take part in a cardiac rehabilitation program, if this is available, where advice about safe exercise, weight reduction and changes to dietary and smoking habits can be encouraged.

Possible lines of questioning

1. How would you quantify and manage this patient’s future cardiac risk?
2. What would you advise a surgeon or anaesthetist about the risks of surgery for this patient?
3. How would you manage his or her anti-platelet treatment in the perioperative period?
Revascularisation

For some long-case patients with ischaemic heart disease the emphasis will be on revascularisation (coronary surgery or angioplasty). These procedures are so common that many patients with other presenting problems will have had them.

The history

Similar information to that outlined in the ischaemic heart disease long case is required.

1. Careful questioning about risk factor control, both before and after surgery or angioplasty, is very important. The patient should know whether he or she has ever had an infarct and may know whether there was significant left ventricular damage.

2. Find out what procedure (or procedures) the patient has had and whether there has been complete relief of symptoms.

3. If coronary artery surgery was performed, ask how many grafts were inserted and whether internal mammary or other arterial (e.g. radial artery) conduits were used. It may be possible to work out from the history whether surgery was performed to improve symptoms or prognosis (e.g. three-vessel or left main disease), or both.

4. The patient may know how many vessels were dilated if angioplasty was performed and whether stents were inserted. The patient should know whether bare metal stents (BMS) or drug-eluting stents were used. Ask whether the angioplasty was performed in the setting of a myocardial infarction or acute coronary syndrome. Find out for how long dual anti-platelet treatment was prescribed.

The examination

Examine the patient as for the ischaemic heart disease long case.

1. Note the presence of a median sternotomy scar. Patients who have had a left internal mammary artery (LIMA) graft often have a numb patch to the left of the sternum. This may be permanent.

2. Look at the sternal wound for signs of infection; osteomyelitis of the sternum is a rare but disastrous complication of surgery. Look and feel for sternal instability. Sternal wires are often palpable.

3. Examine the arms for the very large scar that results from radial artery harvesting.

4. Examine the legs for saphenous-vein-harvesting wounds. Infection and breakdown of these wounds are more common than for the sternal wound.

Management

Surgery

Use of the left internal mammary artery (LIMA) to graft the left anterior descending (LAD) coronary artery has been routine for more than 20 years. Other arterial conduits are used less often, but ‘all arterial revascularisation’ is performed routinely in some centres where saphenous vein grafts (SVGs) are not possible (e.g. previous coronary artery bypass graft (CABG) or varicose veins in both legs and thighs). In these cases the right internal mammary artery (RIMA) may be used, usually to graft the right coronary, or the radial artery is used as a free arterial graft. The RIMA may also be used as a free graft attached to the aorta, if that is necessary to make it reach. There is excellent evidence that LIMA grafts have a higher long-term patency rate (>90% at 10 years) than SVGs (50% at 10 years). There is less information about other arterial conduits.

In general CABG is better than angioplasty for patients with three-vessel disease and diabetes (improved mortality, fewer re-interventions, fewer infarcts).
In response to the increasing numbers of angioplasty procedures, surgeons have begun to perform fewer invasive bypass procedures. The most widely used alternative is the ‘off-pump’ LIMA graft to the LAD coronary artery. A median sternotomy incision is still used, but the LIMA is attached to the LAD coronary artery on the beating heart. A ‘Y’ graft from the LIMA to the circumflex and right coronaries can be performed using the RIMA attached to the LIMA. These operations avoid the need for cardiopulmonary bypass, speed recovery and possibly reduce the risk of intraoperative cerebral events. Minimally invasive bypasses are carried out in some centres. A series of lateral chest incisions are used as ports for surgery using thoracoscopic equipment. The technique is not easy and the chest wound, although small, is not necessarily less painful than a median sternotomy.

Angina may recur at any time after CABG. Very early angina suggests a technical problem, such as mammary artery spasm, thrombosis of an SVG, grafting of the wrong vessel or grafting of the correct vessel, but proximal to the area of stenosis. Sometimes revascularisation may be ‘incomplete’ because one or more vessels were unsuitable for grafting – usually because of distal disease in the target vessel.

Recurrence of angina is more common if risk factors have not been aggressively controlled. Low-dose aspirin has also been shown to prolong graft survival and patients with severe diffuse disease are often given dual anti-platelet treatment by their surgeons. When angina recurs the patient usually describes symptoms similar in character to the old ones. Recurrent chest pain that is different from the old angina is less likely to be ischaemic.

**ANGIOPLASTY**

Angioplasty is now performed more often than surgery in many centres. It has not been shown to improve the prognosis for patients with stable angina receiving optimal medical treatment (OMT) (Table 5.2).

### Table 5.2 Optimal medical treatment for stable angina

| 1. Aspirin +/− clopidogrel (for 1 year post ACS) |
| 2. Statin – target LDL 1.8 mmol/L |
| 3. ACEI or ARB at maximum tolerated dose |
| 4. Beta-blocker – heart rate down to <70 |
| 5. Hypertension treated to <130/80 |
| 6. Exercise 3 times a week for at least >150 minutes/week |

ACS = acute coronary syndrome; ACEI = angiotensin-converting enzyme inhibitor; ARB = angiotensin II receptor blocker.

Many angioplasties are performed to provide symptom relief for patients with one- or two-vessel disease. Increasingly, however, patients with acute coronary syndromes, and especially those with raised troponin levels, are treated with early angioplasty. There is now good evidence that this group of patients has an improved prognosis (fewer deaths and fewer large infarcts) and a shortened hospital stay when treated aggressively with angioplasty.

Dual anti-platelet treatment (DAPT) has made subacute stent thrombosis a rare event (<1%). DAPT is ideally given for 48 hours before angioplasty and for at least 4 weeks afterwards; 6 months to a year is often recommended for patients who have had an acute coronary syndrome or a drug-eluting stent.

For patients treated for an infarct or acute coronary syndrome, a loading dose of 300–600 mg of clopidogrel is given (60 mg of prasugrel, 180 mg of ticagrelor).
Figure 5.1 Splinter haemorrhages.

Figure 5.2 (a) Right fundus and (b) left fundus, showing multiple flame-shaped and blot hemorrhages in both eyes. Several hemorrhages are white centred, consistent with Roth spots. A retinal hemorrhage is centred on the fovea of each eye, accounting for the decreased visual acuity.
4. *Staphylococcus aureus* – particularly in drug addicts; usually presents acutely. Note, though, that only a small minority of *S. aureus* bacteraemias are associated with endocarditis.
5. *Staphylococcus epidermidis* – more common in patients with recent valve replacement but can be a contaminant in blood cultures.
6. Gram-negative coccobacilli – rarely a cause; more common with prosthetic valves. The responsible organisms are called the HACEK group:
   - *Haemophilus*
   - *Actinobacillus*
   - *Cardiobacterium hominis*
   - *Eikenella* spp.
   - *Kingella kingae*
7. Fungi (e.g. *Candida*, *Aspergillus*) – particularly in drug addicts and immunosuppressed patients.

**CAUSES OF CULTURE-NEGATIVE ENDOCARDITIS**
*Note:* This diagnosis should be made with caution. It condemns a patient to prolonged treatment with intravenous antibiotics.
1. Previous use of antibiotics.
2. Exotic organisms (e.g. *Haemophilus parainfluenzae*, histoplasmosis, *Brucella*, *Candida*, Q fever).
3. Right-sided endocarditis (rarely).

**POST-VALVE SURGERY ENDOCARDITIS**
Early infection is acquired at operation; late infection occurs from another source. This condition has a worse prognosis than native valve endocarditis.

**Diagnosis**
The diagnosis is usually a clinical one. The modified Duke criteria are often used to assist. Two major criteria, one major and three minor, or five minor criteria secure the diagnosis.

**MAJOR CRITERIA**
1. Typical organisms in two separate blood cultures.
2. Evidence of endocardial involvement: echocardiogram showing a mobile intracardiac mass on a valve or in the path of a regurgitant jet, or an abscess or new valvular regurgitation.
3. Transesophageal echocardiogram recommended for patients with a prosthetic valve and at least possible IE by clinical criteria. Transthoracic echocardiogram recommended for other patients initially.
4. Single positive blood culture for *Coxiella burnetii* or anti-phase IgG antibody >1:800.

**MINOR CRITERIA**
1. Predisposing cardiac condition or intravenous drug use.
2. Fever.
3. Embolic vascular phenomena or stigmata.
4. Serological or acute phase abnormalities.
Treatment
Early involvement in the management by a cardiac surgeon in a cardiac surgical unit is usually indicated, particularly for staphylococcal infection.

1. Intravenous administration of a bactericidal antibiotic. If the organism is a sensitive *S. viridans*, give benzylpenicillin, 6–12 g daily for 4–6 weeks. If it is an enterococcus, at least 4 weeks of intravenous treatment are necessary and the choice of antibiotic depends on the organism’s sensitivity. For prosthetic valves, 6–8 weeks of intravenous treatment are necessary.

2. Follow the progress by looking at the temperature chart, serological results and haemoglobin values.

3. The decision to go on to valve replacement is a difficult one; it is best made with the assistance of a cardiac surgeon who has been involved from the start. Indications for surgery include:
   a. resistant organisms (e.g. fungi)
   b. valvular dysfunction causing moderate-to-severe cardiac failure (e.g. acute severe aortic regurgitation)
   c. persistently positive blood cultures in spite of treatment
   d. invasive paravalvular infection causing conduction disturbances, or a paravalvular abscess or fistula (detected by TOE)
   e. recurrent major embolic phenomena, although this is controversial (an isolated vegetation is not in itself an indication for surgery).

Factors suggesting a poorer prognosis
1. Shock.
2. Congestive cardiac failure.
3. Extreme age.
4. Aortic valve or multiple valve involvement.
5. Multiple organisms.
6. Culture-negative endocarditis.
8. Prosthetic valve involvement.

Differential diagnosis
1. Atrial myxoma.
2. Occult malignant neoplasm.
4. Polyarteritis nodosa.
5. Post-streptococcal glomerulonephritis.
6. Pyrexia of unknown origin.
7. Cardiac thrombus.

Prognosis
Prior to antibiotic use, this was an invariably fatal disease. Currently, more than 70% of patients with endogenous infection survive, as do 50% of those with a prosthetic valve infection. Intravenous drug users have a good prognosis.

Prophylaxis
Confusion between rheumatic fever and endocarditis prophylaxis is common. Rheumatic fever prophylaxis consists of long-term, low-dose antibiotic administration.
Prophylaxis against endocarditis requires high-dose, short-term treatment only in patients with a very high risk, namely:

- a previous episode of endocarditis
- a prosthetic heart valve or prosthetic material used for valve repair
- a congenital heart malformation (unrepaired cyanotic heart disease, repaired cyanotic heart disease with residual defects or recent surgery using artificial material (within 6 months) or
- a cardiac transplant with valve disease.

According to the latest (2007) American Heart Association guidelines, all other lesions no longer require prophylaxis. Prophylaxis is also recommended according to the Australian (Heart Foundation) guidelines for:

- complex congenital heart disease, including patients who have had repair operations using shunts or artificial material and have persisting shunts (e.g. VSD repaired with Gortex, but with residual shunt)
- Aboriginal patients with any intermediate- or high-risk lesion.

Prophylaxis regimens are as follows (recommendations from Therapeutic Guidelines – Antibiotic):

1. **Dental procedures (e.g. periodontal procedures) or oral surgery**: amoxycillin 2 g, 1 hour before the procedure. For patients unable to take oral antibiotics, use ampicillin IV 15–30 minutes before the procedure. For those allergic to penicillin, cephalaxin 2 g orally 1 hour before the procedure is adequate.

2. **Gastrointestinal or genitourinary procedures**: no prophylaxis is recommended unless infection is already present.

Remember that the effectiveness of antibiotic prophylaxis has not been proven. Patients need to be reminded of the need for good dental hygiene and regular dental review.

**Possible lines of questioning**

1. What would persuade you that this patient now needs surgery for his or her infective endocarditis?
2. What would make you decide to treat this culture-negative patient for endocarditis?

**Congestive cardiac failure**

This is a common therapeutic problem, but it may be a diagnostic problem. It is uncommon the only major problem in a long case.

**The history**

1. It is important first to find out what may have precipitated episodes of cardiac failure. Precipitating problems include:
   a. arrhythmias (especially atrial fibrillation – these can be the cause or the result of heart failure)
   b. discontinuation of medications – usually the diuretic (particularly important)
   c. myocardial infarction
   d. anemia
   e. infection and fever
   f. thyrotoxicosis
g. anaesthesia and surgery
h. pulmonary embolism
i. high salt intake, drugs that cause salt and water retention (e.g. traditional NSAIDs, COX-2 inhibitors) or excessive physical exertion
j. pregnancy.

Note: Chronic lung disease can be a cause of, or a precipitating factor for, right and left ventricular failure.

2. Then ask about the symptoms of left ventricular failure, e.g.:
   a. dyspnoea
   b. orthopnoea
   c. paroxysmal nocturnal dyspnoea
   and right ventricular failure, e.g.:
   d. oedema
   e. ascites
   f. anorexia
   g. nausea.

   Ask about symptoms of ischaemic heart disease (e.g. angina). These may help distinguish dyspnoea caused by lung disease from that caused by cardiac failure.

3. Enquire about the history of previous heart disease:
   a. hypertension
   b. ischaemic heart disease – infarcts, angina
   c. rheumatic or other valve disease
   d. congenital heart disease
   e. cardiomyopathy
   f. previous cardiac surgery (e.g. coronary artery bypass grafting, valve replacement or resection of an aneurysm)
   g. cardiac transplantation.

4. Find out about coronary risk factors, in addition to previous ischaemic heart disease, age and male sex, including:
   a. hyperlipidaemia
   b. hypertension
   c. smoking
   d. diabetes mellitus
   e. family history of early coronary heart disease
   f. use of oral contraceptives or premature onset of menopause
   g. obesity
   h. physical inactivity
   i. erectile dysfunction.

5. Ascertain the risk factors for dilated cardiomyopathy:
   a. excessive alcohol intake
   b. family history of cardiomyopathy
   c. haemochromatosis.

6. Ask what medications are currently being taken.

7. Ask what investigations have been undertaken – particularly:
   a. echocardiography
   b. stress ECG testing
   c. nuclear studies
   d. cardiac catheterisation.

8. Find out how the disease affects the patient’s life and ability to cope at home (e.g. climbing stairs, sexual difficulties, etc.). Remember to classify the patient according to the New York Heart Association (NYHA) guidelines.
NYHA classification

I  No limitation of physical activity. Ordinary physical activity does not cause angina / dyspnoea.
II  Angina / dyspnoea on moderate activity.
III  Angina / dyspnoea on mild activity.
IV  Angina / dyspnoea at rest.

The examination

1. Perform a detailed cardiovascular system examination.
2. Look particularly for signs of cardiac failure, the underlying causes of the problem and any precipitating factors.
3. Look for a pacemaker or defibrillator box.
4. Note wasting as a result of cardiac cachexia.
5. Take the blood pressure lying and standing. Treatment with ACE inhibitors and beta-blockers often results in mild hypotension.

Investigations

1. Chest X-ray film (Fig 5.4): look for cardiomegaly and chamber size (e.g. left atrium), cardiac aneurysm, valve calcification, sternal wires suggesting previous cardiac surgery, signs of lung disease and pulmonary congestion.

Figure 5.4  Alveolar pulmonary oedema. When the pulmonary venous pressure reaches 30 mmHg, oedema fluid will pass into the alveoli. This causes shadowing (patchy to confluent depending on the extent) in the lung fields. This usually occurs first around the hila and gives a bat's wing appearance. These changes are usually superimposed on interstitial oedema. A lamellar pleural effusion (arrow) is seen at the right costophrenic angle where Kerley ‘B’ lines are also evident.

The Canberra Hospital X-Ray Library, reproduced with permission.
2. **ECG**: look for arrhythmias, signs of ischaemia or recent or old infarction (Fig 5.5), left ventricular hypertrophy and persisting ST elevation (aneurysm). Left bundle branch block (LBBB) is a common ECG finding in these patients (Fig 5.6). The ECG is rarely entirely normal in a patient with heart failure.

3. **Electrolytes and creatinine levels**: to exclude hypokalaemia (as a cause of arrhythmia), hyponatraemia (which may indicate severe longstanding cardiac failure, a poor prognostic sign) and renal failure.

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**Figure 5.5** Sinus rhythm. There are Q waves from V1 to V5. This is diagnostic of an extensive old anterior infarct, which is likely to be the cause of this patient’s heart failure.

**Figure 5.6** Sinus rhythm. Left bundle branch block. The QRS complexes may widen further as heart failure progresses. LBBB is a common finding in heart failure but is not diagnostic.
4. **B-type natriuretic peptide level** (BNP; previously called brain natriuretic peptide): although there is doubt about the reference range, a definitely elevated level may help distinguish cardiac from non-cardiac dyspnoea. Since BNP falls when heart failure is treated, trials of monitoring BNP are under way as a means of assessing the adequacy of cardiac treatment.

5. **Haemoglobin value**: to exclude anaemia as a precipitating cause.

   If the diagnosis is not already obvious, consider *dilated cardiomyopathy*. Investigations for this include those outlined below.

6. **Echocardiography** (Fig 5.7): this will show generalised or segmental wall motion abnormalities and reduced fractional shortening. An estimate of the left ventricular volume has proved difficult due to chest deformities.

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**Echocardiography Report**

**Reason for study:** Assess left ventricular function, cardiac failure

**Study quality:** Good *Satisfactory* Poor

<table>
<thead>
<tr>
<th></th>
<th>Value</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>RV</td>
<td>13 (mm)</td>
<td>(N 10–26)</td>
</tr>
<tr>
<td>Sept.</td>
<td>8 (mm)</td>
<td>(N 7–11)</td>
</tr>
<tr>
<td>LVEDD</td>
<td>66 (mm)</td>
<td>(N 36–56)</td>
</tr>
<tr>
<td>LVESD</td>
<td>49 (mm)</td>
<td>(N 20–40)</td>
</tr>
<tr>
<td>LVPW</td>
<td>10 (mm)</td>
<td>(N 7–11)</td>
</tr>
<tr>
<td>Aorta</td>
<td>22 (mm)</td>
<td>(N 20–35)</td>
</tr>
<tr>
<td>LA</td>
<td>36 (mm)</td>
<td>(N 24–40)</td>
</tr>
<tr>
<td>FS</td>
<td>26 %</td>
<td>(N 27–40)</td>
</tr>
<tr>
<td>EF</td>
<td>50 %</td>
<td>(N 55–70)</td>
</tr>
</tbody>
</table>

**Valves**

- **Mitral** Mild-to-moderate MR
- **Tricus.** Mild TR
- **Aortic** Thickened, not stenosed
- **Pulm.** Appears normal

**Doppler – 2-D**

The left ventricle is dilated. There is extensive antero-apical hypokinesis. The aortic valve is slightly thickened and there is mitral annular calcification. The mitral valve is not stenosed and there is no mitral valve prolapse.

**Doppler – colour flow mapping**

There is no aortic gradient; mild-to-moderate MR is present. MR jet to two-thirds of LA. Mild TR. RV pressure = 38 mmHg.

**Conclusions**

- Severe segmental LV dysfunction; moderate MR.

**Comments**

This echocardiography report demonstrates the typical findings when a patient has cardiac failure caused by previous anterior myocardial infarction. The left ventricular dysfunction is not global (typical of cardiomyopathy) but involves the infarcted area. There is overall LV dilatation with an increase of the LVEDD. The FS is the percentage change in LV size from diastole to systole measured at the base of the heart. It can be in the normal range despite the presence of LV dysfunction, if the base of the heart is not involved.
The ejection fraction can be estimated from the LVEDD and LVESD measurements. There are a number of formulas, which are applied automatically by the calculation software of the echocardiograph machine. It is difficult to obtain an accurate ejection fraction, which is a volume change measurement on the basis of two 2D-image measurements. These calculated ejection fractions tend to have a higher reference range than those obtained by nuclear heart pool scanning. MR is almost always detected when moderate LV dysfunction is present.

Mitral annular calcification is a common finding in elderly patients; it can be associated with MR, but not with mitral stenosis. The presence of left atrial enlargement suggests that the mitral regurgitation is not acute, but can also be associated with hypertension.

TR is commonly found in patients with heart failure, but may also be present in normal people. Interrogation of the regurgitant jet with continuous wave (CW) Doppler allows measurement of its velocity. This can be used to calculate the pressure difference across the valve. Since the pressure in the right atrium is usually close to 5 mmHg, the pressure in the RV can be calculated by adding 5 to the pressure difference. In this case the pressure difference across the valve is about 33 mmHg.

Key
EF = ejection fraction; FS = fractional shortening; LA = left atrium; LVEDD = left ventricular end-diastolic dimension; LVESD = left ventricular end-systolic dimension; LVPW = left ventricular posterior wall; MR = mitral regurgitation; Pulm. = pulmonary; RV = right ventricle; Sept. = septal thickness; TR = tricuspid regurgitation; Tricus. = tricuspid.

Figure 5.7 Echocardiography report in a patient with cardiac failure caused by anterior myocardial infarction.

ejection fraction can be made. Segmental hypokinesia suggests that ischaemia is the cause of the cardiac failure. Doppler echocardiography will usually show at least some mitral and tricuspid regurgitation in these patients. The presence of more severe valvular disease suggests a different aetiology for the cardiac failure. Serial echocardiograph measurements of left and right ventricular dimensions can be useful for following the patient’s progress.

7. Cardiac MRI or CT can help assess LV and RV function and MRI, myocardial viability.
8. Coronary angiography: this is often necessary to exclude coronary artery disease.
9. Right ventricular biopsy: this may help determine the aetiology in selected patients.

Treatment
1. Remove precipitating causes. Atrial fibrillation and other incessant tachycardias can be a cause of cardiac failure – tachycardia-induced cardiomyopathy. The prognosis is good if normal heart rate can be restored.
2. Correct underlying causes if possible (e.g. angioplasty for an acute infarct or coronary artery bypass grafting or angioplasty for ischaemia) (Table 5.3).
3. Control the failure.
   a. Decrease physical activity (e.g. bed rest for the acutely ill patient).
   b. Control fluid retention (e.g. by diuretics, low-salt diet, fluid restriction (1000–1500 mL for severe failure)).
      • Patients should be advised to weigh themselves daily. An increase in weight of 2 kg or more over a few days is usually an indication of significant fluid retention. A temporary increase in the diuretic dose will often prevent deterioration in symptoms.
Table 5.3 Causes of ventricular failure

**LEFT VENTRICULAR FAILURE**
1. Volume overload
   a. Aortic regurgitation
   b. Mitral regurgitation
   c. Patent ductus arteriosus
2. Pressure overload
   a. Systemic hypertension
   b. Aortic stenosis
3. Myocardial disease
   a. Ischaemic heart disease
   b. Dilated cardiomyopathy – causes include:
      i. idiopathic (most common)
      ii. alcohol
      iii. myocarditis
      iv. familial (autosomal dominant)
      v. tachycardia induced (usually AF)
      vi. peripartum
      vii. neuromuscular disease (e.g. dystrophia myotonica)
      viii. connective tissue disease (e.g. scleroderma)
      ix. haemochromatosis
      x. sarcoidosis
      xi. drugs (e.g. doxorubicin)
      xii. radiation

**RIGHT VENTRICULAR FAILURE**
1. Volume overload
   a. Atrial septal defect
   b. Tricuspid regurgitation
2. Pressure overload
   a. Pulmonary stenosis
   b. Pulmonary hypertension
3. Myocardial disease
   a. Cardiomyopathy secondary to left ventricular failure
   b. Right ventricular infarction (rare)

**Note:** Restrictive cardiomyopathy and hypertrophic cardiomyopathy can be causes of heart failure.

- Oppose inappropriate activation of the renin–angiotensin system.
  - ACE inhibitors are considered to be the drug class of choice for cardiac failure as they prolong life; symptomatic hypotension is the major side-effect in cardiac failure. ACEIs are indicated for all classes of heart failure, even for asymptomatic patients with left ventricular dysfunction. Every effort should be made to titrate the dose up to the maximum tolerated. The usual limitation is symptomatic hypotension.
  - AR blockers are indicated for patients intolerant (usually because of cough) of ACEIs. The most common reason for the cessation of ACEIs or ARBs is deterioration in renal function (usually in patients with renovascular disease).
  - The combination of an ARB and a neprilysin inhibitor (sacubitril / valsartan is the first one approved) has been shown more effective than an ACEI or ARB and may be used as first-line treatment for heart failure patients or as a replacement for an ACEI or ARB.
via the coronary sinus into one of the left ventricular veins. This complicated procedure enables both ventricles to be paced and dyssynchronous contraction of the ventricles associated with a very wide QRS to be corrected. About 70% of patients improve with the treatment. Although echocardiographic measurements of dyssynchrony are available, they have not yet been able to predict a response to resynchronisation treatment and the current guidelines allow their use for symptomatic patients with LBBB who are in sinus rhythm.

j. Ventricular assist devices are sometimes used as a bridge to transplant in very ill patients. Survival for weeks or months is possible with these devices. Trials of entirely artificial hearts continue in small numbers of patients.

4. Correct iron deficiency. A number of studies have shown improvement in symptoms when patients who are iron deficient but not anaemic have their iron stores replenished with intravenous iron.

Possible lines of questioning

1. Would you recommend that this patient have an implanted defibrillator or resynchronisation pacemaker?
2. How would you help this patient manage his or her symptoms of cardiac failure from day to day?
3. How would you investigate this patient with a recent worsening of symptoms of heart failure?

Diastolic heart failure (heart failure with preserved ejection fraction)

Most breathless patients with heart failure have abnormal left ventricular systolic function, which is characterised by dilatation and hypokinesis. Some cases of cardiac failure, however, may be caused by diastolic dysfunction. In such cases, the myocardium is stiff, often because it is hypertrophied and does not relax normally. The condition seems to be more common in elderly patients. Hypertension is a common cause. The diagnosis is difficult, but an echocardiogram will show preserved or increased systolic contraction without dilatation and there may be left ventricular hypertrophy and left atrial dilatation. Doppler echocardiography may show abnormalities of left ventricular filling caused by the stiffness of the ventricle. However, this is not easy to quantify and is dependent on variations in preload and afterload.

The condition may have a prognosis as bad as that of systolic heart failure. Treatment is similar, but beta-blockers are used early on and only small doses of diuretics should be required. At least in theory, digoxin should be avoided if the patient is in sinus rhythm. Every effort should be made to control hypertension. Treatment has not been shown to alter prognosis.

Hyperlipidaemia

Hyperlipidaemia may be present in patients under investigation for vascular disease, pancreatitis, hypothyroidism or diabetes mellitus. It often presents both diagnostic
and management problems. It is not likely to be the major problem for a long-case patient.

The history

1. The patient should be able to indicate whether or not the main problem is vascular. If the problem is one of premature coronary artery disease, hypercholesterolaemia is the likely lipid problem. The most important inherited cause is familial hypercholesterolaemia, which is caused by a defective or absent low-density lipoprotein (LDL) receptor. The heterozygous form occurs in about 1 person in 500. As the transmission is autosomal dominant, the patient may know of first-degree relatives who have been affected. There may even be family members with the homozygous form. These people usually present with a tenfold elevation in serum cholesterol levels as a result of an increase in plasma LDL levels and have a myocardial infarction before the age of 20 years. People with the heterozygous form typically have myocardial infarctions in their 30s and 40s and have a two- to threefold elevation in cholesterol level. More than 80% of affected men and nearly 60% of affected women have had myocardial infarcts by the age of 60 years. Find out whether the patient has already had a myocardial infarct and which relatives have been affected.

Familial combined hyperlipidaemia is associated with obesity or glucose intolerance and may be expressed as type IIa, IIb or IV hyperlipidaemia (Table 5.4). This is also an autosomal dominant trait. Patients develop hypercholesterolaemia and often hypertriglyceridaemia in puberty. Once again, there usually is a strong family history of premature coronary artery disease. There is no doubt that an elevated triglyceride level adds to the risk of hypercholesterolaemia.

Familial dysbetalipoproteinaemia is also associated with coronary artery disease. These patients have elevated cholesterol and triglyceride levels and are usually found to have obesity, hypothyroidism or diabetes mellitus. Find out whether there is any history of these and whether there has been atheromatous disease or vascular disease involving the internal carotid arteries and the abdominal aorta or its branches. Ask about claudication, which occurs in about one-third of patients.

2. The patient may be able to tell you his or her cholesterol and triglyceride levels and what they have been in the past. Some patients even know their LDL and high-density lipoprotein (HDL) levels.

3. If there is no history of coronary artery disease and the patient either knows the triglyceride level to have been very high or has a history of pancreatitis, the likely diagnosis is familial hypertriglyceridaemia. This is also a common autosomal dominant disorder and is associated with obesity, hyperglycaemia, hyperinsulinaemia, hypertension and hyperuricaemia. Although there is a slightly increased incidence of atherosclerosis, this is probably related to diabetes mellitus, obesity and hypertension than to the hypertriglyceridaemia itself.

Ask about the patient's alcohol consumption, any history of hypothyroidism or the ingestion of oestrogen-containing oral contraceptives. Any of these can precipitate a rapid rise in the triglyceride level, which may precipitate pancreatitis or the characteristic eruptive xanthomas. Between attacks, patients have moderate elevations of the plasma triglyceride level.

4. Next, find out about treatment. In familial hypercholesterolaemia, this will have been aimed at the cholesterol level itself and at any cardiovascular complications that have occurred. The patient should be well informed about a low-saturated-fat diet and may be aware of side-effects from medication usage.
Table 5.4 Hyperlipoproteinaemias

<table>
<thead>
<tr>
<th>TYPE</th>
<th>LIPOPROTEIN ELEVATED</th>
<th>ELECTRO-PHORETIC MOBILITY</th>
<th>MECHANISM</th>
<th>SECONDARY CAUSES</th>
<th>CLINICAL FEATURES</th>
<th>ASSOCIATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Chylomicrons</td>
<td>Origin</td>
<td>Deficiency; extrahepatic lipoprotein lipase or apo C-II deficiency</td>
<td>Rarely SLE</td>
<td>Eruptive xanthomata; lipaemia retinalis</td>
<td>Pancreatitis</td>
</tr>
<tr>
<td>IIa</td>
<td>LDL</td>
<td>β</td>
<td>Receptor defect</td>
<td>Cushing’s; hypothyroidism</td>
<td>Xanthelasma; corneal arcus</td>
<td>CAD, PVD</td>
</tr>
<tr>
<td>IIb</td>
<td>LDL and VLDL</td>
<td>β and pre-β</td>
<td>Cholestasis; nephrotic syndrome</td>
<td>Tendon xanthomata (Fig 5.8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>IDL</td>
<td>Broad β</td>
<td>Oversynthesis and/or abnormal apo E</td>
<td>Renal and liver disease</td>
<td>Palmar crease and tuboeruptive xanthomata; xanthelasma</td>
<td>CAD, PVD</td>
</tr>
<tr>
<td>IV</td>
<td>VLDL</td>
<td>Pre-β</td>
<td>Oversynthesis and/or under catabolism of VLDL</td>
<td>Diabetes mellitus; alcoholism; chronic renal failure</td>
<td>Usually no xanthomata</td>
<td></td>
</tr>
<tr>
<td>V</td>
<td>VLDL and chylomicrons</td>
<td>Origin and pre-β</td>
<td>Saturation lipoprotein lipase by VLDL</td>
<td>As for IV</td>
<td>As for I</td>
<td>As for I</td>
</tr>
</tbody>
</table>

**NOTES**

- Apo A-I deficiency is associated with the absence of plasma HDL and severe premature CAD.
- Apo B deficiency is the defect in abetalipoproteinaemia (autosomal recessive), which is characterised by haemolytic anaemia (acanthocytosis), fat malabsorption and neurological defects (proprioceptive loss, retinitis pigmentosa).
- LCAT deficiency results in decreased HDL, cloudy corneas and progressive renal disease.

Apox=apolipoprotein; CAD=coronary artery disease; HDL=high-density lipoprotein; IDL=intermediate-density lipoprotein; LCAT=lecinthin cholesterol acyltransferase; LDL=low-density lipoprotein; PVD=peripheral vascular disease; SLE=systemic lupus erythematosus; VLDL=very-low-density lipoprotein.

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5. The need for drug treatment of hyperlipidaemia depends on the lipid levels and on the patient’s other vascular risk factors. Ask about a family history of premature coronary disease (first-degree relatives under the age of 60), previous vascular disease (coronary, cerebral or peripheral), smoking and diabetes mellitus.

6. Ask about any history of cutaneous xanthoma. These may have resolved with treatment or have been surgically removed.

**The examination**

1. Examine the cardiovascular system. There may be evidence of cardiac failure from previous myocardial infarcts or a sternotomy scar from previous coronary surgery. Occasionally one sees the scandalous situation of a patient with untreated hyperlipidaemia presenting with more angina after initially successful coronary surgery.
2. Look specifically for the interesting skin manifestations of these conditions.

a. Patients with the heterozygous or homozygous form of familial hypercholesterolaemia may have tendon xanthomas. These are nodular swellings that tend to involve the tendons of the knee, elbow and dorsum of the hand and the Achilles tendon. They consist of massive deposits of cholesterol, probably derived from the deposition of LDL particles. They contain both amorphous extracellular deposits and vacuoles within macrophages, and sometimes become inflamed and cause tendonitis.

b. Cholesterol deposits in the soft tissue of the eyelid cause xanthelasma and those in the cornea produce arcus cornea (previously insensitively called arcus senilis). Xanthelasma occur in about 1% of the population and arcus cornea in 30% of people over 50. When corneal arcus is seen in younger people it is more often associated with hyperlipidaemia. Surveys of people with xanthelasma indicate a slightly higher than average cholesterol level. Tendon xanthomas are diagnostic of familial hypercholesterolaemia, but the other signs are not as specific – only 50% of people affected have hyperlipidaemia.

The majority of patients with the homozygous form have even more interesting signs.

• Yellow xanthomas may occur at points of trauma and in the webs of the fingers.
• Cholesterol deposits in the aortic valve may be sufficient to cause aortic stenosis; occasionally mitral stenosis and mitral regurgitation can occur for the same reason.
• Painful swollen joints may also be present. Obesity is uncommon in these patients.
underlying pathology that is responsible for the AF (e.g. mitral stenosis). It is important in helping to quantify embolic risk. Look at the echo for:

a. valve abnormalities
b. cardiomyopathy
c. diastolic dysfunction of the left ventricle (especially important in hypertensive patients)
d. left ventricular hypertrophy
e. atrial size (consider atrial septal defect)
f. mitral valve disease
g. segmental wall abnormalities consistent with ischaemic heart disease.

Tests may be needed because of actual or potential drug side-effects (e.g. liver function or thyroid function tests for patients on amiodarone).

5. **Cardiac catheterisation** is often indicated for patients with ventricular arrhythmias, as they may have an ischaemic substrate. Patients whose VT has been stable but becomes unstable (often leading to more activity on the part of their cardioverter defibrillator) may have developed new ischaemia and should have this possibility investigated and treated.

**Management**

Much depends on the rhythm abnormality.

1. Arrhythmias that are not life-threatening are usually managed, at least at first, with drugs. Candidates will be expected to have a thorough working knowledge of the common antiarrhythmic drugs, their methods of action, indications and side-effects. Remember that many antiarrhythmic drugs have a potentially dangerous proarrhythmic effect.

2. The indications for permanent pacing (Table 5.10) and different uses for VVI, VVIR (rate-responsive) and DDD (dual-chamber) pacers (Figs 5.16 and 5.17) should be well understood. A basic understanding of antitachycardia devices and indications for their use (Table 5.11) is also important.

### Table 5.10 Indications for permanent pacemaker insertion in adults

**GENERALLY AGREED INDICATIONS**

1. Intermittent or permanent complete heart block, with:
   a. symptomatic bradycardia
   b. cardiac failure
   c. arrhythmias that require treatment with drugs that slow conduction
   d. documented asystole of more than 3 seconds or escape rhythm with a rate < 40 beats/minute
   e. confusional states that improve with temporary pacing
2. Intermittent permanent second-degree AV block with symptomatic bradycardia
3. Sinus node dysfunction with symptomatic bradycardia

**LESS CERTAIN INDICATIONS**

1. Asymptomatic complete heart block; heart rate ≥ 40 beats/minute
2. Symptomatic type 2 second-degree heart block
3. Bifascicular or trifascicular block with syncope of unknown aetiology

**NOT INDICATED**

1. First-degree heart block
2. Asymptomatic type 1 second-degree heart block
### Table 5.11 Indications for implanted cardioverter-defibrillators (ICDs)

<table>
<thead>
<tr>
<th>Generally Agreed Indications</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Confirmed VF or hypotensive VT not related to acute infarct or severe electrolyte abnormality, but VF VT not inducible at EPS – this means drug treatment cannot be tested by EPS</td>
<td></td>
</tr>
<tr>
<td>2. VF VT with contraindications to drug treatment (intolerance)</td>
<td></td>
</tr>
<tr>
<td>3. Persistently inducible VT VF despite drug treatment, ablation or surgery</td>
<td></td>
</tr>
<tr>
<td>4. Persistent spontaneous VT VF despite drug treatment</td>
<td></td>
</tr>
<tr>
<td>5. Symptomatic long QT syndrome despite drug treatment</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Less Certain Indications</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Inducible but not spontaneous VT despite other treatment in high-risk patients</td>
<td></td>
</tr>
<tr>
<td>2. VT VF apparently controlled, but in a high-risk patient</td>
<td></td>
</tr>
<tr>
<td>3. Serial drug testing possible, but defibrillator preferred</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Not Generally Indicated</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Very frequent or incessant VT</td>
<td></td>
</tr>
<tr>
<td>2. Reversible cause</td>
<td></td>
</tr>
<tr>
<td>3. Recurrent syncope, VT VF not inducible</td>
<td></td>
</tr>
<tr>
<td>4. Poor life expectancy (e.g. class IV cardiac failure, but not a transplant candidate)</td>
<td></td>
</tr>
</tbody>
</table>

Note: Increasingly proven VT or VF in a patient with poor LV function is considered an indication regardless of EPS findings.

VF = ventricular fibrillation; VT = ventricular tachycardia; EPS = electrophysiological study.

3. Automatic implanted cardioverter-defibrillators (AICDs) are increasingly used to manage recurrent VT.
   - They are often used in combination with antiarrhythmic drugs of some sort. They are becoming smaller, cheaper (between $25,000 and $40,000) and more complicated. It is now established that they improve mortality rates in selected patients.
   - They are usually the treatment of choice for hypotensive VT or missed sudden death from VT. Drug treatment of these conditions is not very effective.
   - The current models have leads that can be placed intravenously into the vena cava and for pacing purposes into the right ventricle.
   - They are small enough to be implanted like pacemakers in the chest wall, but are still noticeably larger than pacemakers (Fig 5.18). Implantation takes place under local anaesthetic in the electrophysiology laboratory.
   - The periprocedural mortality rate is less than 1%, compared with over 5% when surgical implantation was required.

   The programming of these machines is complicated, but candidates should know that they are usually set to attempt reversion of VT by overdrive pacing (antitachycardia pacing, ATP) before administering a DC shock. Patients are usually, but not always, aware of the onset of ATP and almost always aware of DC shock administration. Ask how the device has affected the patient’s life and confidence, including how often it goes off and whether the box itself causes problems because of its size. Although AICDs can prevent sudden death, their presence is often associated with a feeling of insecurity. Patients may have clear memories of events leading up to activation of the device. They may avoid places where arrhythmias and activations have occurred. They often require repeated explanation and reassurance.

   Patients who have begun to experience frequent episodes requiring ATP or DC shocks need to be assessed (Table 5.12).
**Figure 5.18** (a) Posteroanterior chest X-ray showing ICD and biventricular pacemaker. (b) Lateral view: the large defibrillation electrode (which also serves as an RV pacing electrode) (arrow) and the right atrial and left ventricular pacing leads are visible.

Figures reproduced courtesy of The Canberra Hospital.
The particular management problems of atrial fibrillation

Examiners require candidates to have a sensible approach to the management of AF and the opportunity for examiners to ask about this common condition will often arise. The principles of management are to:

- maintain sinus rhythm
- control the heart rate (if maintaining sinus rhythm proves difficult)
- protect from embolic events.

1. There is good evidence from recent trials that control of heart rate is at least as satisfactory an approach as that of trying aggressively to maintain sinus rhythm. Nevertheless, patients with paroxysmal AF often find the arrhythmia very disturbing. They should be told at the outset that it may not be possible to keep them in sinus rhythm, but that rate control and freedom from embolic episodes can be achieved. The prophylactic drug treatment of paroxysmal AF involves the use of a class III drug (sotalol or amiodarone) in most cases, but occasionally the class 1C drug flecainide can be used if the heart is known to be structurally normal.

2. Rate control of persistent or paroxysmal AF can be achieved with less-toxic drugs. Digoxin is a common first-line treatment and is usually well tolerated. Some recent trials have suggested an increased mortality for patients treated with digoxin. It is not very effective on its own at controlling the heart rate during exercise. Many patients with chronic AF have persistent dyspnoea during exercise because of inadequate rate control. They benefit from the use of a beta-blocker or one of the non-dihydropyridine calcium channel blockers (diltiazem or verapamil). These can be used with or without digoxin. Control of the heart rate can prevent or reverse the impairment of left ventricular function that is associated with tachycardias (tachycardia-induced cardiomyopathy).

3. When patients remain unhappy with their symptoms despite rate control, further intervention should be considered.

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Table 5.12 Assessment of patients experiencing frequent activations of a defibrillator

1. Check programming, e.g. false activations for AF or sinus tachycardia.
2. Exclude new ischaemia.
3. Introduce or increase antiarrhythmic treatment – usually amiodarone or beta-blocker.
4. Consider VT ablation.

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Possible lines of questioning

1. How would you explain to *this* patient the risks and benefits of having an implanted defibrillator?
2. What would be your approach to a patient who has begun to receive frequent shocks from his or her device and has asked for it to be removed?