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MASTER MEDICINE

Medicine
内科学

A clinical core text with self-assessment

EDITED BY
PAUL O'NEILL
TIM DORNAN
DAVID DENNING

北京大学医学出版社

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Medicine

A core text with self-assessment

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Contents

Using this book

Philosophy of the book

This chapter aims to help you:

- understand how the emphasis on self-assessment in this book can make learning easier and more enjoyable
- use this book to increase your understanding as well as knowledge
- plan your learning

How much do you know about diabetes? Are they the right things? Can you answer exam problems on diabetes? This book aims to help you with these questions. You probably have some knowledge of medicine, perhaps a bit patchy, and some clinical experience. We want to help you to be better at integrating knowledge and solving either real problems or simulated ones in examinations. We have tried to present essential information, for doctors practising in the UK, in a concise and ordered fashion. Principles are illustrated and mechanisms explained rather than simply giving you lots of facts to memorise.

Do not think though that this book offers a 'syllabus'. It is impossible to draw boundaries around medical knowledge and learning is a continuous process carried out throughout your career. As we see it in 2001, this book includes all that you *must* know, most of what you *should* know about, and some of what you *might* be aware of.

We assume that you are working towards one or more examinations, probably in order to qualify. Our purpose is to show you how to overcome this barrier. As we feel strongly that learning is not simply for the purpose of passing exams, the book aims both to help you to pass and to develop *useful* knowledge and understanding.

Layout and content

The first part of each chapter sets out the key learning objectives; those things which anyone starting a medical career needs to know and an examiner expects them to know. More detailed learning objectives are to be found at the start of each major section. One starting point might be to look at these objectives and then test yourself in the self-assessment section at the end of each chapter. This will help to steer you towards areas that you need to work on. Alternatively, you can go straight into the main body of the chapter and check that you have achieved the objectives at the end; if not then you will need to do further work and perhaps read about the topics elsewhere.

The main part of the text describes important topics in major subject areas. Within these sections, we have put down the essential information in a logical order with explanations and links. In order to help you, we have used lists to set out frameworks and to make it easier for you to put facts in a rational sequence. Tables are used to link quite complex information.

There are some situations in medicine that require you to act immediately and senior help may not be available. For some of these emergencies, we have put the steps that you must take in an 'emergency' summary box. These are based on current guidelines, but guidelines do change.

You have to be sure that you are reaching the required standards, so the final section of each chapter is there to help you to check out your knowledge and understanding. The self-assessment is in the form of multiple choice questions, case histories, extended matching questions (EMQs), short notes, data interpretation, possible viva questions, picture questions and sample stations that might be included in objective structured clinical examinations (OSCEs). Questions are designed to integrate knowledge across different chapters and to focus on the decisions you will have to take in a given clinical situation. Detailed answers are given with reference to relevant sections of the text; the answers also contain information and explanations that you will not find elsewhere, so you have to do the assessments to get the most out of this book.

Using the book

Your first task before using the book is to map out on a sheet of paper a series of three lists dividing the major subjects (corresponding to our chapter headings) into an assessment of your strong, reasonable and weak areas. This gives you a rough outline of your learning schedule, which you must then fit in with the time available. Clearly, if your examinations are looming large, you will have to be ruthless in reducing the time allocated to your strong areas. The major subjects should be further classified into individual topics. Encouragement to store information and to test your ongoing improvement is by the use of the self-assessment sections. You must keep checking your current level of knowledge.

Overall the aim is to help you to learn through the use of interlinked steps:

- What do you already know about the subject?
- Why do you want to learn more about the subject? You will acquire knowledge much more easily if you can put it in a framework.
- What things needs explaining? What do you not understand?
- Can you expand on these things? You should explain as much as you can from different aspects.
- Set yourself goals for where your knowledge is lacking.
- Check that you have achieved these.

If you can, discuss problems with colleagues/friends. The areas which you understand least well will become apparent when you try to explain them to someone else. You will also benefit from hearing a different perspective on a problem.

Approach to examinations

The discipline of learning is closely linked to preparation for examinations. Many of us simply opt for remembering facts because full understanding is often not required, such as in multiple choice questions. We would prefer it if you acquired a deeper knowledge and understanding but, recognising constraints on your time, advocate a pragmatic approach that combines the necessity of passing examinations with longer-term needs.

The hardest step is to determine what will be in the exam; medicine does not draw boundaries around knowledge either in breadth or depth. The best approach is to combine your lecture notes, textbooks (not reference) and past examination papers. From the last, for example, you may find out that not only are the pulmonary manifestations of HIV infection a possible examination topic but you will also get an indication of the depth of knowledge required.

You then have to choose what sources you are going to use for your learning and revision. Textbooks come in different forms. At one extreme, there is the large reference book, which includes extensive literature citation. At the other end of the spectrum is the condensed 'lecture note' format, which often relies heavily on lists. In the middle of the range are the medium-sized textbooks. You should choose the book(s) that suits your needs (and that of the examination!) and that you find readable.

You should now have a rough syllabus, your own lecture notes and some books that you feel comfortable in using. The next stage is to map out the time available for preparation. You must be realistic in this, allowing time for breaks and working steadily, not cramming. If you do attempt to cram, you have to realise that only a certain amount of information can be retained in short term memory, so as the classification of the lipid disorders moves in, then the terminology of the renal tubular acidoses moves out! Cramming is simply retention of facts. If the examination requires understanding you will be in trouble.

You might be tempted to do general reading of a large textbook. Even if this was feasible in the time available (the number of pages to be read tends to increase as the examination gets closer), it is not very effective in that often you cannot remember anything of the topic you have just covered. An analogy would be driving a car along a familiar route, arriving, but being unable to recall anything of the journey.

We advise an approach, as outlined above, based on the use of key steps, learning objectives and self-assessment. For a subject such as endocrinology, we would recommend setting out the topics to be covered and then attempting to summarise your knowledge about each in note form. By this means, gaps in your knowledge/understanding become apparent. Use of 'mind-maps' may be appropriate in helping to make connections, for example between the physiological control of thyroxine secretion and thyrotoxicosis. It is much more efficient to go to textbooks having thought about what you know, as you are then 'looking' for information and explanation.

Self-assessment will help in determining the time to be allocated to each system. If you are consistently scoring excellent marks in a particular subject it is not cost effective to spend a lot of time trying to achieve the 'perfect' mark. In an essay, it is many times easier to obtain the first mark (try writing your name) than the last. You should also try to decide on the amount of weight to be assigned to each subject; this should be heavily biased to the likelihood of it appearing in the examination! It is not sensible to devote large amounts of time to the ocular manifestations of systemic disease if ophthalmology is not included in the 'syllabus' you have devised.

As the examination draws near you should attempt practice questions and complete papers. It is not sufficient to have the necessary knowledge and understanding; you need to demonstrate these to the examiners. Many people pay insufficient attention to the type of question they are going to encounter. Moving the focus away from books and lecture notes to actual questions helps in identifying where knowledge is still lacking and what work is still to be done.

Methods of examination

Multiple choice questions

Unless very sophisticated, multiple choice questions test recall of information. The aim is to gain the maximum

marks from the knowledge that you can remember. You should read the stem with great care, highlighting the 'little' words such as *only, rarely, usually, never* and *always*. You will often lose marks because of 'negatives', such as *not, unusual and unsuccessful*. If the stem is phrased *may occur*, this has entirely different connotations to *characteristic*. The latter may mean a feature which should be there and the absence of which (for example central chest pain in myocardial ischaemia) would make you question the correctness of the diagnosis. Alternatively, it can also be used to describe rare features that would suggest the diagnosis, for example yellow vision (xanthopsia) in digoxin toxicity. If the stem is long with several lines of text or data then you should try and summarise it by extracting the essential elements.

You must check the marking method before starting. Most employ a negative system in which marks are lost for incorrect answers. The temptation is to adopt a cautious approach answering a relatively small number of questions. However, this can lead to problems as we all make simple mistakes or even disagree vehemently with the answer in the computer! Caution may lead you to answer too few questions to pass after the marks have been deducted for incorrect answers.

Distracters are the technical term for parts of questions which sound as though they are correct but are definitely incorrect. A good example would be symptoms and signs of *hyper*natraemia being included in a question on *hypo*natraemia. This is the most common cause of losing marks even though you know the answer.

Extended matched questions

In the second edition, we have included examples of extended matching questions (EMQs) as these are becoming increasingly common in undergraduate and postgraduate examinations. They consist of a theme (e.g. Weight loss), a series of options and then a question (e.g. for each patient, select from the list of options, the most likely diagnosis). You then have to read each patient vignette and decide the most likely cause. EMQs test your ability to recall knowledge and **apply** it to a clinical problem. They are not negatively marked. EMQs seem to separate out those students who can use their knowledge from those who have simply learned facts by rote.

Short notes

Short notes are not negatively marked. The system is for a 'marking template' to be devised which gives a mark(s) for each important fact. You will gain nothing for style or superfluous information. Your aim is to set out your knowledge in an ordered *concise* manner. The common faults are, first, devoting too much time to a single question thereby neglecting the rest and, second, not limiting the answer to the question asked. For example, in a question about the management of diabetes mellitus, you should not list all facts about diabetes, only those relevant to management.

Essays

Similar comments apply to essays, but you may get marks for logical development of an argument or theme. Conversely, you will not obtain good marks for an essay that is a set of unconnected statements. Length matters little if there is no cohesion. Most people are aware of the need to 'plan' their answer yet few do this. It is important in an examination based on essays that you manage your time and all questions are given equal weight, unless guided otherwise in the instructions. A brilliant answer in one essay will not compensate you for not attempting another because of time. Nobody can get more than 100% (usually 75%) on a single answer!

Data interpretation

Data interpretation involves the application of knowledge to solve a problem. In your revision, you should aim for an understanding of principles; it is impossible to memorise all the different data combinations. In the exam, a helpful approach is to translate numbers into a description, for example a serum potassium of 2.8 mmol/l is *low* and the ECG tracing of a heart rate of 120/min shows a *tachy*cardia. Pattern recognition can then be attempted.

Data questions are not usually negatively marked so put down an answer even if you are far from sure that it is right. Conversely, there is no point in listing four possibilities if the question asks for one response. The examiner will not choose from your answers, the first response will be taken!

Slide/picture questions

Pattern recognition is the first step in a picture question. You should couple this with a systematic approach looking for abnormalities. For example you should check the breast shadows, bony skeleton, soft tissues, retrocardiac space, etc. in a chest radiograph. Describe in your mind what you see and try to match it with common problems. Again, even if doubtful, put an answer down. Slides often come with an accompanying statement or data. You should use this alongside the visual image as it may give a clue as to the answer required; it may be essential in distinguishing between two conditions which give a similar slide appearance.

Case history questions

A more sophisticated form of examination question is an evolving case history with information being presented sequentially; you are asked to give a response at each stage. They are constructed so that a wrong response in the first part of the question still means that you can obtain marks from the subsequent parts. Patient management problems are designed to test the recall and application of knowledge through an understanding of the principles involved. As with the data interpretation, you should always give answers unless the exam instructions indicate the presence of negative marking.

Viva

The viva examination can be a nerve-wracking experience. You are normally faced with two examiners who may react with irritation, boredom or indifference to what you say. You may feel that the viva has gone well and yet you failed, or, more commonly, you think that it has gone badly simply because of the apparent attitude of the examiners.

Your main aim during the viva should be to steer the questioning of the examiners so that they are constantly asking you about things you know about. Despite what is often said, you can prepare for a viva. Questions are liable to take one of a small number of types centred around subjects that cannot be examined in the traditional clinical exam:

- emergency medicine (e.g. diabetic coma)
- management of common conditions (e.g. hypertension)
- clinical sciences (e.g. control of blood pressure).

For each heading, you can prepare a list of the *common* problems.

Another approach used by examiners is to invite you to start the viva by asking what you have read recently or what do you think is an important recent medical advance: prepare something along these lines beforehand.

During the viva there are certain techniques that will help you to make a favourable impression. When discussing the management of something, it is better to say 'I would do this' rather than 'the book says this'. You should try and strike a balance between saying too little and too much. It is hard on examiners when you will not expand on any of your answers and it is equally difficult if you refuse to shut up! Remember, the longer you talk without stopping the more likely it is that you have either gone off the topic or are showing the examiners the inadequacy of your knowledge.

In a viva, the examiners are likely to want to explore the *limits* of your knowledge; do not be upset if they push you hard. It is alright to say you do not know something, most examiners will want to change tack to see what you do know about.

Objective structural clinical examinations

Clinical examinations are not the main focus of this book. However, we have included some examples of objective structured clinical examination (OSCE) stations in the self-assessment to help *you* to develop the right balance between academic work and gaining clinical experience. For more help, you should look at Dornan and O'Neill *Core Clinical Skills for OSCEs in Medicine* (Churchill Livingstone, 2000), which we consider to be a companion book to this edition.

Normal values

In both examinations and clinical practice, most test results are given together with the normal reference range for that laboratory. However, you are expected to know certain normal ranges as they are essential for making decisions in an emergency. Furthermore, familiarity with the ranges for common indices will help you to obtain a 'feel' for data interpretation and abnormal patterns. Thus, a serum potassium of 1.9 mmol/l means much more than one of 3.1 mmol/l even though both are low.

In the list of normal values in Tables 1 and 2, we have indicated with an * the indices that you would be expected to be able to give an approximate normal range. Remember that laboratories do vary and that a normal range is simply that which 95% of the normal population served by that laboratory would fit into.

Conclusions

We have set out a framework for using this book, but you should amend this according to your own needs and the examinations you are facing. Whatever approach you adopt, the aim should be for an understanding of the principles involved rather than rote learning.

Table 1 Normal values for haematology

Index	Range	Unit
White blood cell*	4.0–11.0	$\times 10^9$/l
Neutrophils	2.0–7.5	$\times 10^9$/l
Lymphocytes	1.5–4.0	$\times 10^9$/l
Eosinophils	0.04–0.4	$\times 10^9$/l
Monocytes	0.2–0.8	$\times 10^9$/l
Basophils	< 0.1	$\times 10^9$/l
Red blood cells		
Male	4.5–6.5	$\times 10^{12}$/l
Female	3.8–5.8	$\times 10^{12}$/l
Haemoglobin: male*	130–170	g/l
Haemoglobin: female*	116–165	g/l
Packed cell volume (PCV)		
Male	0.40–0.54	l/l
Female	0.37–0.49	l/l
Mean cell volume (MCV)*	80.0–97.0	fl
Mean cell haemoglobin (MCH)	27.0–32.0	pg
Mean cell haemoglobin concentration	31.0–35.0	
(MCHC)		g/dl
Red cell distribution width (RDW)	11.5–15.0	–
Platelets*	150–400	$\times 10^9$/l
Erythrocyte sedimentation rate (ESR)		
Male*	< 5	mm/h
Female*	< 7	mm/h
Plasma viscosity	1.50–1.72	cp
Reticulocytes	0.2–2.0	%
Serum vitamin B_{12}	160–600	ng/l
Serum folate	2.0–10.0	µg/l
Red cell folate	125–600	µg/l
Ferritin		
Males	20–300	µg/l
Female premenopausal	12–250	µg/l
Female postmenopausal	20–300	µg/l
Haemoglobin (Hb) Hb_{A2}	1.8–3.5	%
HbF	0.2–1.0	%
Glucose 6-phosphate dehydrogenase	4.6–13.5	IU/g haemoglobin
Prothrombin time (PT)*	12.0–16.0	s
Activated partial thromboplastin time (APTT)	21.0–27.5	s
Fibrinogen	2.0–4.0	g/l
Fibrin degradation products (FDP) (D-dimer)	< 0.5	mg/l
Bleeding time (adults)	1.6–8.0	min

*Values that you would be expected to know.

Table 2 Normal reference ranges for biochemistry

Index	Range	Value
Sodium*	132–144	mmol/l
Potassium*	3.5–5.0	mmol/l
Chloride	95–108	mmol/l
Bicarbonate*	24–30	mmol/l
Urea*	2.7–7.5	mmol/l
Creatinine*	50–120	µmol/l
Glucose		
Fasting	3.2–6.0	mmol/l
Random	3.3–9.2	mmol/l
Bilirubin	1–20	µmol/l
Calcium*	2.10–2.65	mmol/l
Phosphate*	0.70–1.40	mmol/l
Total protein	60–80	g/l
Albumin	33–49	g/l
Globulin	21–38	g/l
Urate		
Female	< 0.38	mmol/l
Male	< 0.42	mmol/l
Blood gases		
pH*	7.38–7.42	
Pco_2*	34–45 (4.5–6.0)	mmHg (kPa)
Po_2*	90–110 (12–14.7)	mmHg (kPa)
Base excess*	−2 to +2	
Enzymes		
Alkaline phosphatase (ALP) (adult)	25–110	IU/l
Amylase	10–87	IU/l
Aspartate aminotransferase (AST)	5–45	IU/l
Alanine aminotransferase (ALT)	5–45	IU/l
Female/Male	5–45	IU/l
Gamma-glutamyl transferase (GGT)	< 65	IU/l
Creatine kinase (CK)	< 150	IU/l
Lactate dehydrogenase (LDH)	200–500	IU/l
Cerebrospinal fluid		
Protein*	0.25–0.75	g/l
Glucose* (depends on blood sugar)	2.5–5.5	mmol/l
Hormones		
Thyroxine (T_4)	50–150	nmol/l
Triiodothyronine (T_3)	1.1–2.8	nmol/l
Thyroid stimulating hormone (TSH)	0.5–5.0	mU/l
Cortisol	200–650	nmol/l (07:00–09:00 h)
	60–250	nmol/l (22:00–24:00 h)
Urine free cortisol	< 300	nmol/24 h
Lipids:		
Total cholesterol		
Satisfactory	< 5.2	mmol/l
Borderline	5.2–6.5	mmol/l
Unsatisfactory	> 6.5	mmol/l
Fasting triglycerides	0.3–2.0	mmol/l
Iron	12–30	µmol/l
Total iron-binding capacity (TIBC)	45–70	µmol/l

*Values that you would be expected to know.

1 Cardiovascular disease

1.1 Background

Learning objectives

You should:

- be able to take with absolute confidence a history from a patient with chest pain or other major symptom of cardiovascular disease and construct a differential diagnosis
- be able to interpret the chest radiograph and electrocardiograph (ECG)
- know the place of echocardiography, exercise testing, coronary angiography and the investigations used in particular cardiovascular diseases (described below under diagnoses) and when to request them
- be competent at performing cardio-pulmonary resuscitation.

Cardiovascular disease is the most common cause of death in the western world and a major preventable cause of chronic ill-health. It will impinge on whichever branch of medicine you choose and may dominate your practice. As a general practitioner, cardiovascular disease will present some of the commonest emergencies you have to treat. As an anaesthetist, you will have to decide whether patients with it can safely be anaesthetised. As a surgeon, you will have to exclude it as a cause of abdominal pain. Cardiovascular diseases are ubiquitous, so they figure large in the 'core' knowledge and skills of the medical graduate.

Anatomy

You need a knowledge of the surface anatomy of the heart, as seen from the front, to interpret physical signs, chest radiographs and electrocardiographs (Fig. 1). The heart has a triangular projection and lies mostly behind the sternum. The base of the triangle is parallel and slightly to the right of the right sternal border. The apex is in the interspace between the left fifth and sixth ribs in the mid-clavicular line. The right heart border is formed by the right atrium. The left border is composed of the left atrial appendage superiorly and left ventricle inferiorly. The anterior surface is composed, from right to left, of the right atrium, right ventricle, interventricular septum and left ventricle. The left atrium is at the back of the heart and, seen laterally, forms the upper posterior heart border. The

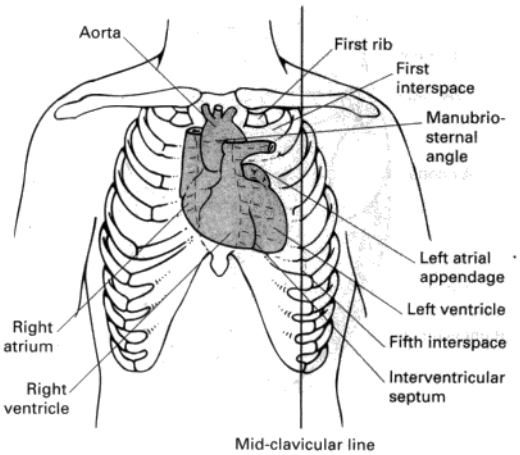

Fig. 1 Surface anatomy of the heart as seen from the front.

lower border is formed by the left ventricle. The anterior heart border in a lateral projection is formed by the right ventricle (as illustrated under chest radiography in Fig. 4 below). The anatomy of the coronary circulation is described on page 13.

Physiology

Cardiac output is maintained by:

- impulse generation and propagation
- cyclical myocardial contraction
- an intact valvular system.

Impulse generation

The cardiac impulse is generated by cyclical depolarisation of the sino-atrial (SA) node, a specialised area close to the junction of the superior vena cava and right atrium. The impulse spreads rapidly through the right and left atria to reach the atrio-ventricular (AV) node. The ventricles are isolated electrically from the atria by the annulus fibrosus. The impulse is conducted by the His bundle, which leads into the right and left bundle branches, activating the right and left ventricles, respectively. The anatomy of impulse propagation is summarised in Figure 2. Spontaneous, rhythmic depolarisation is not unique to the SA node. It can arise lower in the conducting system. The lower in the conducting system the impulse arises, the slower the rate (e.g. atrium 60 beats/min, AV node 50 beats/min, ventricle 30 beats/min). The rate of ventricular contraction is governed by the most rapidly depolarising focus in the heart. Lower and slower pacemakers are overridden by impulses from above. Impulses can arise in diseased myocardium, often at a very fast rate (see dysrhythmias, p. 27). Impulses may pass through myocardial tissue when the conducting tissues are blocked (bundle branch block, p. 11) or through abnormal accessory pathways (p. 11).

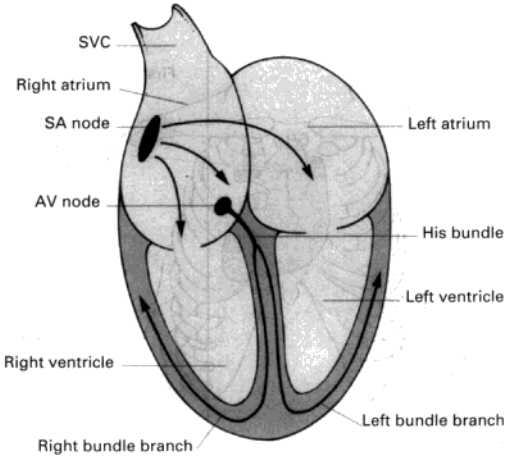

Fig. 2 The cardiac conducting system. SVC, superior vena cava; SA, sino-atrial; AV, atrio-ventricular.

The cardiac cycle

Atrial depolarisation causes contraction of the right then the left atrium, corresponding to (though lagging behind) the P wave on the electrocardiograph. There is an electrical pause (the PR interval) as the wave of depolarisation passes through the AV node then contraction of the left and right ventricles (corresponding electrically to the QRS complex). Rising ventricular pressures close the mitral and tricuspid valves then open the pulmonary and aortic valves. At the end of systole, repolarisation occurs (T wave) and the ventricles relax. When aortic and pulmonary artery pressures exceed left and right ventricular pressures, respectively, the valves close. Closure of the mitral and tricuspid valves is heard as the first heart sound at the start of ventricular systole and aortic and pulmonary valve closure as the second sound at the end of it. Atrial systole is responsible for 10% of ventricular filling in the normal heart, the rest occurring passively; atrial systole may account for up to 30% of filling in the abnormal fibrotic heart. Each ventricle ejects about 80 ml blood with each cardiac cycle. The proportion of end-diastolic volume ejected in each cardiac cycle (the ejection fraction) ranges from 55 to 75%.

Cardiac output

Cardiac performance is determined by cardiac rate and stroke volume. Rate is controlled by the balance between sympathetic stimulation, which increases it, and parasympathetic (vagal) stimulation, which reduces it. Stroke volume depends on contractility and the 'afterload' or resistance against which the heart is pumping; this principally comprises the peripheral vascular resistance or, in the case of aortic or pulmonary stenosis, the degree of outflow obstruction.

Stroke volume is also influenced by 'preload', the combined effect of venous filling pressure and the atrial 'kick'. The way in which cardiac muscle responds to changes in preload is an important physiological concept, known as **Starling's law of the heart**, which states that the energy of contraction is proportional to the initial length of the muscle fibres. The heart responds to increased preload by an increased stroke volume up to a level of preload at which it is overwhelmed and decompensation occurs (Fig. 3). Sympathetic stimulation, acting on cardiac β_1-adrenoceptors, increases cardiac performance for a given level of preload. How this relates to the pathophysiology and management of heart failure is considered on page 22.

Coronary artery perfusion occurs during diastole. A very fast heart rate both increases myocardial work and reduces oxygen delivery, so it can precipitate ischaemia. Tachycardias also reduce cardiac output by shortening the time for ventricular filling.

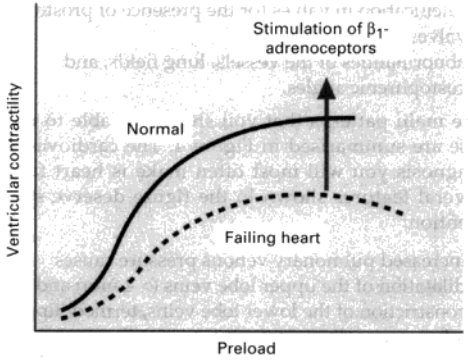

Fig. 3 The relationship between preload and cardiac contractility (Starling's relationship) and the effect of adrenergic stimulation.

Maintenance of blood volume
This is discussed in Chapter 4, which emphasises the close interrelationship between cardiovascular, renal and fluid/electrolyte physiology. That relationship is central to clinical management.

Clinical assessment

History

The main symptoms of cardiovascular disease are:

- chest pain
- breathlessness
- ankle swelling
- fatigue
- palpitations
- syncope.

They result from impaired oxygen delivery to the myocardium (chest pain), brain (syncope) and all other tissues (fatigue), increased pulmonary and systemic venous pressure (breathlessness and ankle swelling) and abnormal cardiac rate and rhythm (palpitations). You should remember that chest pain may be caused by other cardiovascular problems: disease of the aorta (p. 51) and disease of adjacent structures including the chest wall, oesophagus, pleura, disease of the aorta (e.g. dissection, p. 52), pulmonary circulation (pulmonary embolism/infarction, p. 50) and pericardium (pericarditis, p. 41).

Examination

Your examination should systematically test out the anatomy and physiology of the heart and circulation as described above and in Chapter 4. As with all other aspects of clinical examination, use all your senses in the order *look, feel, listen*.

Pulse rate and rhythm
Feel both radial pulses together and, if necessary, other pulses to measure the pulse rate and decide if the rhythm is fundamentally regular, perhaps with superimposed ectopic or dropped beats, or chaotic (atrial fibrillation).

Arterial circulation
The arterial circulation is assessed from:

- the blood pressure, measured first sitting or lying, then standing to detect postural hypotension caused by volume depletion, vasodilatation or autonomic neuropathy
- peripheral cyanosis and/or impaired capillary refill after blanching the nail beds, measures of impaired capillary perfusion
- the volume of the pulse, a crude way of assessing stroke volume
- the character of the carotid pulse, which may be abnormal in, for example, aortic valvular disease (p. 35).

Venous circulation
Venous circulation is assessed by examining the jugular venous pulse, auscultating the lung bases and testing for ankle and sacral oedema. There are several components to jugular venous examination:

- assessment of venous pressure as a sign of preload (p. 24)
- assessment of its response to respiration, discussed under pericardial effusions (p. 41)
- observation of the waveform, particularly important in detecting tricuspid incompetence.

Crackles at the lung bases may be a sign of pulmonary venous hypertension. Peripheral oedema indicates raised systemic venous pressure, fluid retention, a decreased serum albumin level or increased vascular permeability.

Heart and valves
The stethoscope allows you to detect:

- abnormalities of the first and second heart sounds
- added sounds as in, for example, valvular heart disease (p. 32), heart failure (p. 22), or pericarditis (p. 41).

Peripheral arterial system
You should remember to examine the abdominal aorta and femoral, popliteal, dorsalis pedis and posterior tibial pulses. Absence of pulses signifies arterial obstruction. Bruits are another sign of arterial obstruction but may also be caused by increased flow.

Stigmata of cardiovascular disease
Infective endocarditis (p. 38) is the classical cardiovascular disease in which non-cardiac signs are as important as cardiovascular examination in making the diagnosis.

Some other diseases, discussed under valvular heart disease (p. 32), have important stigmata which should be picked up by observation or general examination.

Investigation

The chest radiograph

Echocardiography is a more sensitive and specific way of detecting structural cardiovascular abnormalities than the chest radiograph but you are unlikely to be able to request one in the middle of the night and many patients can be managed without ever having one. Most relevant information can be obtained from a postero-anterior (PA) radiograph, but a lateral view can give extra information about individual chambers, particularly the left atrium. **Portable** films are taken antero-posterior (AP) and make the heart look larger than it is. Examine the radiograph systematically for:

- overall heart size
- changes in shape indicating disease of individual chambers
- calcification in valves (or the presence of prosthetic valves)
- abnormalities of the vessels, lung fields, and costophrenic angles.

The main patterns that you should be able to recognise are summarised in Figure 4. The cardiovascular diagnosis you will most often make is heart failure. Several features shown in the figure deserve special mention:

- increased pulmonary venous pressure causes dilatation of the upper lobe veins (> 4 mm) and constriction of the lower lobe veins, termed 'upper lobe venous diversion'
- you can assess heart size by measuring the ratio of the width of the heart to the width of the thorax, expressed as the cardiothoracic ratio: a ratio > 50% signifies cardiomegaly (except on an AP radiograph)
- septal ('Kerley') lines are caused by interstitial fluid; that are straight, often short (< 1 cm), horizontal, peripheral and present first at the bases

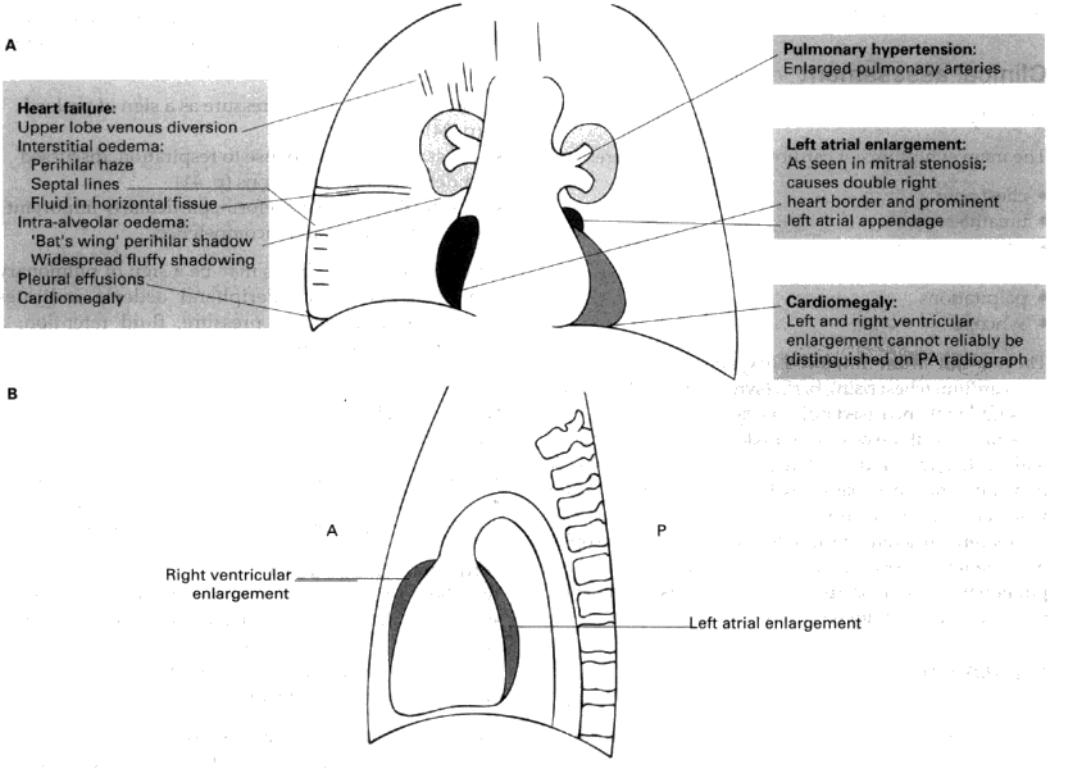

Fig. 4 Principal abnormalities that can be seen on a chest radiograph in cardiac disease. **A** Postero-anterior (PA) radiograph; **B** lateral radiograph.

- fluffy perihilar and more generalised shadowing, signifies fluid in the alveolar spaces, i.e., *severe* pulmonary venous hypertension (or capillary leakage/exudation).

The ECG

If you are unclear about ECG interpretation, you should read one of the excellent concise texts devoted to the subject; this description is very much revision. No matter how experienced you are, you should read an ECG systematically looking for:

- the cardiac rate
- the rhythm
- the electrical axis
- ventricular hypertrophy
- abnormal PQRST configurations.

You should also check the calibration, normally printed at the head of the paper. A normal paper speed is 25 mm/s. One large square (5 mm) represents 200 µs and 1 small square (1 mm) represents 40 µs. 1 mV causes a vertical deflection of 1 cm.

Rate, conduction and rhythm

Rate. First check the QRS complexes to see if they are regular or irregular. If regular, divide the number of large squares between two consecutive R waves into 300 to give the rate. If irregular, divide the number of large squares between four R waves into 900. A normal rate is 60–100 beats/min or 5–3 large squares between two consecutive R waves.

Conduction. Check for P waves as a sign that the impulses are arising within the atria. Next check the PR interval, which represents AV conduction. The PR interval (normal range 3–5 small squares) is short if the impulse is arising unusually close to the AV node or there is an electrical 'short-circuit' between the atria and ventricles ('accessory pathway'). The PR interval is lengthened by disease of the AV node or His bundle. Finally check the width of the QRS complexes; if they are 3 small squares in width or more, they are being propagated by slow electrical spread through muscle rather than the conducting tissues (bundle branch block or 'intraventricular conduction delay').

Rhythm. Abnormalities are discussed under dysrhythmias (p. 27).

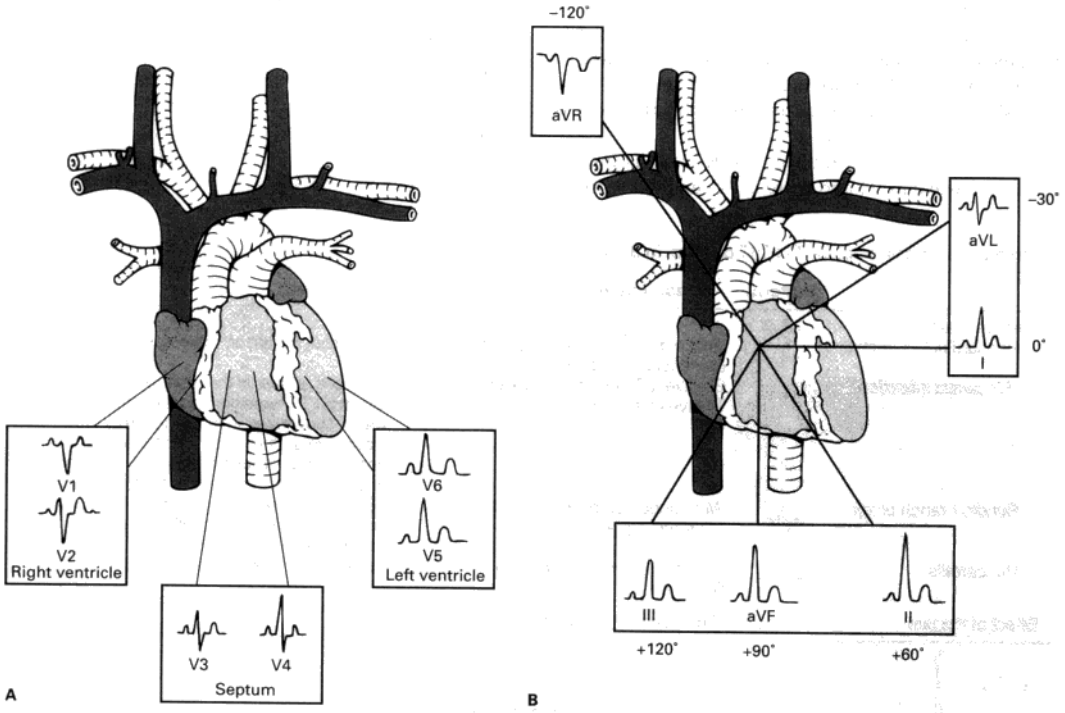

Fig. 5 The vectors of the chest leads for an ECG. **A** The chest leads are arranged radially across the pericardium. **B** The other six leads are calculated from values given by leads on the arms and legs. The electrical axis is the result of electrical activity during systole measured in the limb leads.