CLINICAL MEDICINE

Lecture Notes

John R. Bradley
Mark Gurnell
Diana F. Wood

8th Edition

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Clinical Medicine
Lecture Notes

Lecture Notes

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Clinical Medicine
Lecture Notes

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Preface to the Eighth Edition

History-taking and examination remain the essential tools of clinical examination. However, the environment in which medicine is practised continues to change, with advances in technology and an increasing evidence base to guide decision-making. The eighth edition follows the format of previous editions of this book with two sections: Clinical Examination and Clinical Medicine. Each section has been updated to reflect the more objective methods of assessment that are now used and the increased evidence upon which clinical practice is based.

It is rewarding to discover how many readers have found the text useful for study, for revision, and for clinical practice. Please continue to let us have your views.

John R. Bradley
Mark Gurnell
Diana F. Wood

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We would like to thank Dr Francesca Crawley, Dr Ellie Gurnell, Dr Jane Sterling and Dr Mark Lillicrap for their contributions, help and advice during the preparation of the manuscript.

Preface to the First Edition

This book is intended primarily for the junior hospital doctor in the period between qualification and the examination for Membership of the Royal Colleges of Physicians. We think that it will also be helpful to final-year medical students and to clinicians reading for higher specialist qualifications in surgery and anaesthetics.

The hospital doctor must not only acquire a large amount of factual information but also use it effectively in the clinical situation. The experienced physician has acquired some clinical perspective through practice: we hope that this book imparts some of this to the relatively inexperienced. The format and contents are designed for the examination candidate but the same approach to problems should help the hospital doctor in his everyday work.

The book as a whole is not suitable as a first reader for the undergraduate because it assumes much basic knowledge and considerable detailed information has had to be omitted. It is not intended to be a complete textbook of medicine and the information it contains must be supplemented by further reading. The contents are intended only as lecture notes and the margins of the pages are intentionally large so that the reader may easily add additional material of his own.

The book is divided into two parts: the *clinical approach* and *essential background information*. In the first part we have considered the situation which a candidate meets in the clinical part of an examination or a physician in the clinic. This part of the book thus resembles a manual on techniques of physical examination, though it is more specifically intended to help the candidate carry out an examiner's request to perform a specific examination. It has been our experience in listening to candidates' performances in examinations and hearing the examiner's subsequent assessment that it is the failure of a candidate to examine cases systematically and his failure to behave as if he were used to doing this every day of his clinical life that leads to adverse comments.

In the second part of the book a summary of basic clinical facts is given in the conventional way. We have included most common diseases but not all, and we have tried to emphasise points which are under stressed in many textbooks. Accounts are given of many conditions which are relatively rare. It is necessary for the clinician to know about these and to be on the lookout for them both in the clinic and in examinations. Supplementary reading is essential to understand their basic pathology, but the information we give is probably all that need be remembered by the non-specialist reader and will provide adequate working knowledge in a clinical situation. It should not be forgotten that some rare diseases are of great importance in practice because they are treatable or preventable, e.g. infective endocarditis, hepatolenticular degeneration, attacks of acute porphyria. Some conditions are important to examination candidates because patients are ambulant and appear commonly in examinations, e.g. neurosyphilis, syringomyelia, atrial and ventricular septal defects.

We have not attempted to cover the whole of medicine, but by cross-referencing between the two sections of the book and giving information in summary form we have completely omitted few subjects. Some highly specialised fields such as the treatment of leukaemia were thought unsuitable for inclusion.

A short account of psychiatry is given in the section on neurology since many patients with mental illness attendgeneralclinicsandit ishopedthat readersmaybe warned of gaps in their knowledge of this important field. The section on dermatology is incomplete but should serve for quick revision of common skin disorders.

Wherever possible we have tried to indicate the relative frequency with which various conditions are likely to be seen in hospital practice in this country and have selected those clinical features which in our view are most commonly seen and where possible have listed them in order of importance. The frequency with which a disease is encountered by any individual physician will depend upon its prevalence in the district from which his cases are drawn and also on his known special interests. Nevertheless, rare conditions are rarely seen; at least in the clinic. Examinations, however, are a 'special case'.

We have used many generally accepted abbreviations, e.g. ECG, ESR, and have included them in the index instead of supplying a glossary.

Despite our best efforts, some errors of fact may have been included. As with every book and authority, question and check everything – and please write to us if you wish.

We should like to thank all those who helped us with producing this book and, in particular, Sir Edward Wayne and Sir Graham Bull who have kindly allowed us to benefit from their extensive experience both in medicine and in examining for the Colleges of Physicians.

David Rubenstein
David Wayne
November 1975

Part 1

Clinical Examination

1

The medical interview

Good communication between doctor and patient forms the basis for excellent patient care and the clinical consultation lies at the heart of medical practice. Good communication skills encompass more than the personality traits of individual doctors – they form an essential core competence for medical practitioners. In essence, good communication skills produce more effective consultations and, together with medical knowledge and physical examination skills, lead to better diagnostic reasoning and therapeutic intervention. The term 'communication skills', when applied to medical practice, describes a set of specific skills that can be taught, learned and assessed. A large evidence base shows that health outcomes for patients and both patient and doctor satisfaction within the therapeutic relationship are enhanced by good communication skills.

In this chapter the medical interview as a whole will be considered and then the way in which communication skills should be approached in different types of assessment encountered by students and trainees is reviewed.

There are a number of different models for learning communication skills in use throughout the world. They are generally similar and all emphasise the importance of patient-centred interview methods. This chapter is based on the Calgary–Cambridge model (Fig. 1.1) which has been widely adopted in Europe and the USA and with which the authors are familiar as a means of teaching and learning and as a framework for assessment (Silverman et al. 2005). Like all clinical skills, communication skills can only be acquired by experiential learning. This may take the form of small group learning with role play, the use of actors in simulated learning environments or, for more experienced learners, in recorded real consultations with subsequent feedback.

Effective consultation

Effective consultations are patient-centred and efficient, taking place within the time and other practical constraints that exist in everyday medical practice. The use of specific communication skills together with a structured approach to the medical interview can enhance this process. Important communication skills can be considered in three categories: content, process and perceptual skills (see Table 1.1); these mirror the essential knowledge, skills and attitudes required for good medical practice. These skills are closely interrelated so that, for example, effective use of process skills can improve the accuracy of information gathered from the patient, thus enhancing the content skills used subsequently in the consultation.

Structure

Providing structure to the consultation is one of the most important features of effective consultation. Process skills should be used to develop a structure that is responsive to the patient and flexible for different consultations. Six groups of skills can be identified and each will be considered in the sections that follow.
Sequential in the consultation:

- initiating the session
- gathering information (including from physical examination)
- explanation and planning
- closing the session

Throughout the consultation:

- organisation
- relationship building

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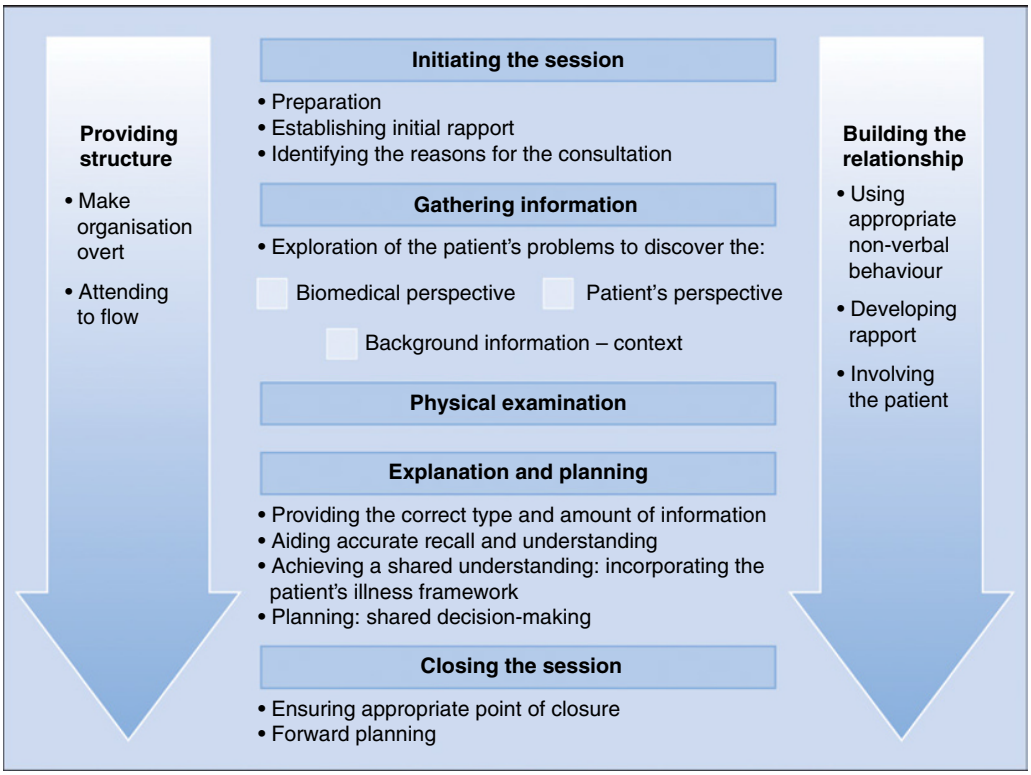

Figure 1.1 The Calgary–Cambridge Guide. Source: Kurtz, S. et al. (2005) *Teaching and Learning Communication Skills in Medicine,* 2nd edn. Oxford: Radcliffe Publishing.

Table 1.1 Categories of communication skills

Skill	Examples
Content skills	
What the doctor communicates	Knowledge-based: appropriate questions and responses; accurate information gathering and explanation to patient; clear discussion of investigation and treatments based on knowledge
Process skills	
How the doctor communicates	Skills-based: verbal and non-verbal communication skills; relationship building; organising and structuring the interview
Perceptual skills	
What the doctor is thinking	Attitude-based: clinical reasoning and problem-solving skills; attitudes towards the patient; feelings and thoughts about the patient; awareness of internal biases

Initiating the session

The initial part of a consultation is essential to form the basis for relationship building and to set objectives for the rest of the interview. Before meeting a patient, the doctor should prepare by focusing him- or herself, trying to avoid distractions and reviewing any available information such as previous notes or referral letters.

Initiating the session

Establish rapport: greet the patient, confirm their name, introduce yourself and explain your role, attend to the patient's comfort.

Identify the reason for the consultation: use an appropriate opening question, listen to the patient, confirm the problem and screen for any other issues that the patient may wish to discuss.

Confirm an agenda for the consultation.

Gathering information

An accurate clinical history provides about 80% of the information required to make a diagnosis. Traditionally, history-taking focused on questions related to the biomedical aspects of the patient's problems. Recent evidence suggests that better outcomes are obtained by including the patient's perspective of their illness and by taking this into account in subsequent parts of the consultation. The objectives for gathering information should therefore include exploring the history from both the biomedical and patient perspectives, checking that the information gathered is complete and ensuring that the patient feels that the doctor is listening to them.

Further information is gathered from the physical examination. Establishment of a good rapport during the first part of the consultation will facilitate communication during the examination. An appropriate chaperone should be present during the physical examination.

Gathering information

Ask the patient to tell their own story.

Listen attentively: do not interrupt; leave the patient time and space to think about what they are saying.

Use open and closed questions: clarify issues in the history; use clear, concise and easily understood questions; move from open to closed questions then back again.

Use verbal and non-verbal facilitation: silences, repetition, paraphrasing.

Pick up on patient's verbal and non-verbal clues: acknowledge them by checking.

Summarise at intervals: verify your understanding; allow the patient to correct or add to the history.

Encourage the patient to express their feelings: actively seek their ideas, concerns and expectations.

Physical examination

Ask permission: gain the patient's consent for examination.

Ensure that the patient is comfortable: position them adequately for the examination; if doing a full examination, cover parts of the body not being examined actively.

Be clear and precise: explain what you are going to do in advance.

Be aware: the patient may be embarrassed or in pain.

Explanation and planning

Explanation and planning is crucially important to the effective consultation. Establishment of a management plan jointly between the doctor and the patient has important positive effects on patient recall, understanding of their condition, adherence to treatment and overall satisfaction. Patient expectations have changed and many wish to be more

Explanation and planning

Avoid jargon: use clear concise language; explain any medical terminology.

Find out what the patient knows: establish prior knowledge; find out how much they wish to know at this stage.

'*Chunk and check*': provide information in small amounts and check understanding; use this to assess how to proceed.

Organise explanation: develop a logical sequence; categorise information; repeat and summarise; signpost what is coming next; use diagrams or charts, written information or instructions.

Relate the information to the patient's perspective.

Respond to patient's cues: verbal and non-verbal; allow patient to ask questions or clarify information.

Involve the patient: share thoughts; reveal rationale for opinions; offer your opinion of what is going on and name it where possible; explore management options; take the patient's lifestyle and cultural background into account in the discussion.

Negotiate a mutually agreeable action plan: check that this meets the patient's expectations and addresses their concerns.

involved in decision-making about investigation and treatment options. The goals of this part of the consultation are thus to gauge the amount and type of information required by each individual patient, to provide information in a way that the patient can remember and understand and which takes their perspectives into account, to arrive at a shared understanding of the problem and to engage the patient in planning the next moves.

Closing the session

Closing the interview allows the doctor to summarise and clarify the plans that have been made and what the next steps will be. It is also important to ensure that contingency plans are in place in case of unexpected events and that the patient is clear about follow-up arrangements. Continuing to foster the doctor–patient relationship in this way has positive effects on adherence to treatment and health outcomes.

Closing the session

Summarise: review the consultation and clarify the plan of action; make a contract with the patient about the next steps.

Describe contingency plans: explain any possible unexpected outcomes; how and when to seek help.

Final check: ensure that the patient agrees and has no further questions.

Two essential parts of effective consultation skills run throughout the interview – organisation and relationship building. The way in which these two are used is shown in Table 1.2.

Organisation allows a flexible but ordered and logical process to occur within an appropriate time-frame. It encourages patient participation and collaboration and facilitates accurate information gathering.

Building a relationship with the patient involves a number of communication skills that enable the doctor to establish rapport and trust between themselves and the patient. It maximises the chances of accurate information gathering, explanation and planning and can form part of the development of a continuing relationship over time. It is vital to patient and doctor satisfaction with the consultation process.

Special circumstances

Certain circumstances demand a special approach to communication skills, such as breaking bad news, dealing with cultural diversity, using an interpreter,

Table 1.2 Skills for organising the consultation and building the relationship

Organising the consultation		Building the relationship	
Summarising	Summarise the end of a specific line of enquiry; confirm your understanding; allow patient to correct; order information; reflect on what to do next.	Non-verbal communication	Includes eye contact, facial expression, posture, proximity, body movement, touch; use of time, your appearance, manner; the environment.
Signposting	Structure the interview overtly; draw attention to what you are about to say; introduce summaries; help patient to understand where the interview is going; ask permission to move on through the interview.	Rapport	Accept patient's views; empathise to show understanding of patient's views and feelings; support by expressing concern, willingness to help, acknowledge efforts to cope; be sensitive towards embarrassing or difficult issues.
Sequencing	Maintain a logical sequence to the interview; use flexible but ordered organisation by signposting and summarising.	Involve the patient	Share your thoughts to encourage patient interaction; explain your rationale for doing things; explain your actions during the physical examination.
Timing	Pace the interview; use other skills to achieve good timing.		

Breaking bad news

Prepare: ensure you have all the clinical details and know the facts; set aside enough time; encourage the patient to bring a relative or friend.

Start the session: review what has happened so far; assess the patient's state of mind; find out what they know and what they are thinking.

Share the information: warn the patient that the news is not good; give the information clearly and in small amounts; relate to the patient's perspective; do not overwhelm with information in the first instance; check repeatedly that the patient understands.

Be sensitive: respond to the patient's emotions – tears, anger, denial; allow time for silences and questions and respond to them honestly; gauge the patient's wishes for information and respond accordingly; be empathic and concerned; check the patient's understanding of what you have said and elicit their concerns and understanding of the situation; do not be afraid to show your own emotions.

Make a plan: explain what will happen next; give hope but be realistic; confirm your role as a partner in care.

Closure: summarise and check the patient's understanding; respond to additional questions; check the patient's support systems and offer to speak to family if requested; make an early follow-up appointment.

and consultation with the elderly, with mentally ill patients or the parents of a sick child. In essence, the core communication skills described here form the basis for any of the more difficult communication skills scenarios. Complex situations require the doctor to use basic skills to a higher level. Preparation and planning, listening to the patient, delivering information in small amounts with regular checking and allowing time for information to be assimilated and for questioning are paramount. Closure is also important, ensuring the patient knows what is happening and is clear about the next steps.

Assessment of communication skills

Clinical competence is assessed at all levels of medical education. Communication skills are usually assessed in undergraduate examinations by stations in Objective Structured Clinical Examinations (OSCEs). More varied assessments take place for postgraduates, including stations in the Royal College of Physicians MRCP Part 3 examination (Practical Assessment of Clinical Examination Skills; PACES) and mini-CEX assessments as part of ongoing workplace-based assessments for trainees. Students and trainees attempting these assessments should have been through appropriate communication skills

Approach to communication skills assessment

Past papers: the format of the examination should be available for review; look at the communication skills stations; familiarise yourself with the format; are the process and content components weighted and, if so, how?

Be prepared: obtain as much information as possible in advance of the assessment; how long are the stations? Is the station simply an observed communication scenario or is a structured viva involved? In some examinations the clinical scenario is available in advance of the examination to allow preparation of content – if so, read it carefully and be certain of the medical facts.

Read the instructions: in most summative assessments the scenario is presented at the station with a few minutes' reading time. Read the scenario carefully. Think about the content as well as the process skills.

Be clear about the task: are you required to take a history, to give information, gain consent for a procedure, talk to a relative or colleague?

Make a plan: before you enter the station, have a clear plan as to how you will approach the consultation and what you wish to achieve.

Think about what you might encounter: communication skills assessments usually use simulated patients who may respond in a number of ways. For example, how will you deal with an emotional patient, a non-communicative patient or an angry response to breaking bad news?

Listen to the examiner: if you are asked to present and discuss the case, listen carefully to the examiner and present the salient features in a clear and logical manner.

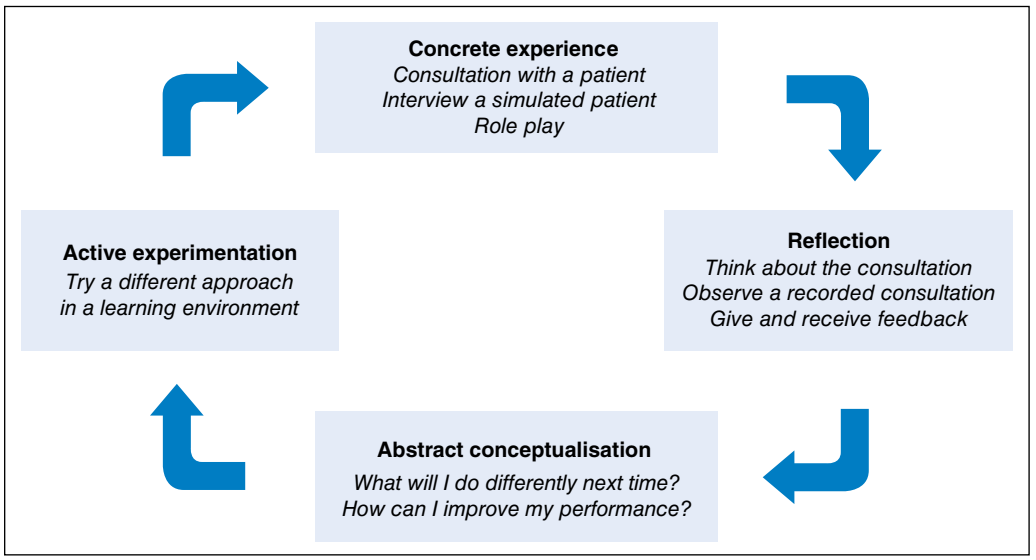

Figure 1.2 Application of the Experiential Learning Cycle to learning communication skills. The learning process may begin with any of the four types of experience. The cycle enables the learner to build on existing knowledge and skills, to take responsibility for their own progress and to use real-life clinical and simulated encounters to promote further learning. Source: Kolb, D.A. (1984) *Experiential Learning: Experience as the Source of Learning and Development*. Englewood Cliffs, NJ: Prentice Hall.

experiential learning programmes allowing them to develop skills in simulated environments and practise them in clinical settings (Fig. 1.2). Whatever the assessment format, a number of factors should be addressed.

REFERENCE

Silverman, J., Kurtz, S. and Draper, J. (2005) *Skills for Communicating with Patients*, 2nd edn. Oxford: Radcliffe Publishing.

2

General examination

Introduction

General examination can reveal abnormalities in a number of systems, which may assist in making an accurate diagnosis.

Disorders of gait, speech and mood should be apparent on first meeting the patient and during the consultation process. Dyspnoea may be observed and abnormal movements, including tremor or paucity of facial expression, should be noted.

During the general examination, obvious features of systemic disease in one site should be correlated with signs elsewhere.

Hands

Note

- joint disorders – arthritis, gout, deformity
- neuromuscular changes – muscle wasting, loss of function
- skin temperature
  - warm, cyanosed hands with bounding pulse in CO_2 retention
  - cold pale hands with arterial disease
- fingers
  - Raynaud's phenomenon, other signs of systemic sclerosis
  - nicotine staining
  - Osler's nodes (endocarditis, vasculitis)
- nails
  - anaemia (pallor, koilonychia)
  - peripheral cyanosis
  - splinter haemorrhages
- clubbing – swelling of the ends of the fingers with increased curvature of the nails and loss of the angle at the nail beds
- palmar erythema
- Dupuytren's contracture
- tremor
  - fine tremor may be exaggerated by placing a piece of paper over the patient's outstretched hands
  - outstretched hands exaggerate the coarse tremor of CO_2 retention
  - flapping tremor in hepatic failure, uraemia – ask the patient to cock their wrists with the hands outstretched
  - resting tremor of Parkinson's disease – hands flexed and showing coarse 'pill-rolling' tremor relieved by intention
  - intention tremor – benign or cerebellar

Face

Check for

- anaemia: examine the insides of the eyelids, look for glossitis (pernicious anaemia) and angular cheilitis
- dental hygiene, tonsillar enlargement, buccal pigmentation inside the mouth

Cardiorespiratory system

- cyanosis: examine the underside of the tongue
- observe pursing of the lips on expiration in obstructive airways disease
- lupus pernio indicates sarcoidosis

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Gastrointestinal system

- jaundice – examine the sclera
- spider naevi
- Peutz–Jeghers syndrome – intestinal polyps with skin and oral pigmentation
- hereditary haemorrhagic telangiectasia

Neurological system

- upper motor neuron (UMN) or lower motor neuron (LMN) facial palsy: differentiate stroke from Bell's palsy; examine the external auditory meatus for evidence of herpes zoster
- ptosis and oculomotor palsies
- Parkinsonism
- myopathy: cataract and frontal baldness suggest dystrophia myotonica
- ophthalmic herpes zoster including the conjunctiva

Endocrine system
Observe typical features of

- thyrotoxicosis: including thyroid eye disease in Grave's disease
- hypothyroidism
- Cushing syndrome
- acromegaly
- Paget's disease involving the skull

Autoimmune diseases

- systemic lupus erythematosus – photosensitive 'butterfly rash'
- scleroderma – microstomia, tightening of the skin
- dermatomyositis – 'heliotrope' periorbital rash

Skin diseases

- acne vulgaris
- acne rosacea
- psoriasis: check behind the ears
- port wine stain – capilary malformation

Rheumatological system

- gouty tophi – check the pinnae

Neck

Examine

- the jugular venous pressure (JVP)
- the thyroid gland
- cervical lymph nodes including the occipital group

Axillae

With shoulders relaxed examine

- anterior, posterior and lateral walls and apices of axillae for lymphadenopathy

Breasts (Fig. 2.1)

Examine systematically in women and men where indicated

- nipples
- four quadrants of each breast
- axillary tails of each breast
- axillae

Legs

Cardiorespiratory system

- pitting oedema – note the extent of peripheral oedema
- evidence of peripheral vascular disease – absent foot pulses, delayed capillary filling, ulceration, gangrene
- varicose veins – note and examine formally if present
- ulceration – note whether venous or arterial
- assess the temperature of the dorsum of each foot, noting asymmetry, interdigital infection and loss of hair
- palpate the dorsalis pedis and posterior tibial pulses
- palpate the popliteal and femoral pulses
- auscultate for bruits over femoral pulses

Rheumatological system

- obvious joint deformity consistent with specific arthritides including gout
- bone deformity – Paget's disease in the tibiae

Neurological system

- Charcot joints
- peripheral neuropathy

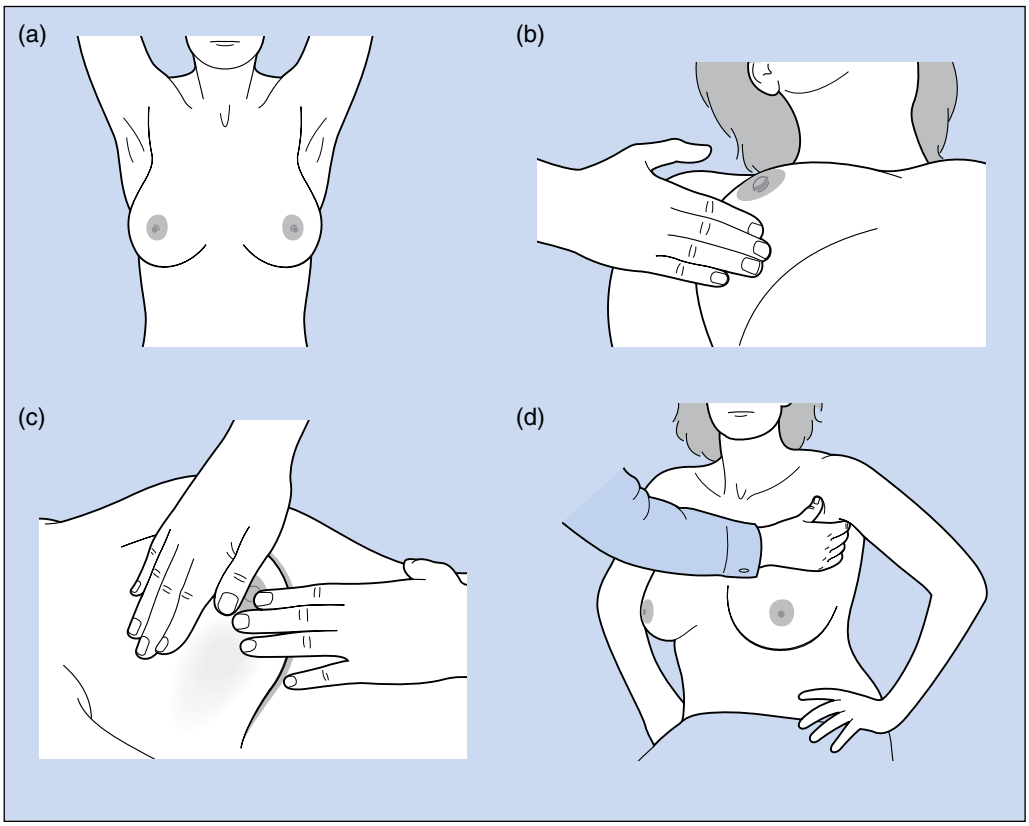

Figure 2.1 Breast examination. (a) Inspection. (b) Palpation. (c) Non-dominant hand stretching the skin. (d) Examination of the axilla.

- effects of chronic neurological lesions – UMN (stroke), multiple sclerosis, polio
- subacute combined degeneration of the cord; hereditary ataxias

Skin

- cellulitis
- varicose eczema with haemosiderosis
- psoriasis
- specific skin lesions – erythema nodosum

Endocrine system

- pretibial myxoedema
- pyoderma gangrenosum

Notes

Lymphadenopathy

A systematic approach to the detection of lymphadenopathy is required. Examine all the major lymph nodes in a logical order:

- cervical
- supraclavicular
- axillary
- inguinal

If lymphadenopathy is detected, examine the abdomen for hepatosplenomegaly.

3

Cardiovascular system

Introduction

Diagnostic accuracy when assessing patients with cardiovascular disease relies heavily on the medical history. A characteristic history of cerebrovascular or peripheral vascular disease may be elicited. Key features in the cardiovascular history are shown in Box 3.1.

Systematic and thorough examination of the cardiovascular system is a core skill for physicians. Accurate assessment of peripheral cardiovascular signs aids the interpretation of auscultatory findings. Patients with ischaemic heart disease may have few physical signs and physicians should be aware of the likely sites and significance of scars from previous surgical or radiological intervention. Cardiac valvular disease and septal defects usually give rise to murmurs, which may be diagnostic. In clinical practice arrival at the final cardiac diagnosis is aided by an electrocardiogram (ECG), chest X-ray (CXR) and echocardiogram (ECHO) and by more complex radiological intervention as appropriate including magnetic resonance imaging (MRI), computerised tomography (CT) and angiography.

General inspection

Note

- peripheral or central cyanosis: central cyanosis is accompanied by peripheral cyanosis by definition
- dyspnoea and orthopnoea
- malar flush
- xanthelasmata

Blood pressure

- Measure the blood pressure lying and standing

Hands

Inspect for

- clubbing
- splinter haemorrhages
- palmar erythema
- nicotine staining

Arterial pulses

Palpate

- radial pulse to assess the rate and rhythm
- radial and brachial pulses in both arms, comparing right and left
- carotid pulses
- radial and femoral pulses simultaneously to assess radiofemoral delay

Rate

- count radial for at least 15 s if rhythm regular, at least 30–60 s if irregular
- check the jugular venous pressure (JVP) whilst counting

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Box 3.1 Important features in the cardiovascular history

Chest pain:	onset duration nature precipitating factors relieving factors distribution and radiation previous episodes associated symptoms (nausea, vomiting, sweating)
Breathlessness:	on exertion orthopnoea paroxysmal nocturnal dyspnoea
Cough:	with or without sputum production
Oedema:	ankle swelling swelling of lower limbs and sacral area
Syncope:	on exertion postural sudden
Calf pain:	intermittent claudication
Peripheral vascular disease (PVD):	cold peripheries with colour/sensory changes
Others:	transient hemiparesis
Visual disturbance:	transient (e.g. amaurosis fugax)
Risk factors:	smoking obesity hypertension diabetes hyperlipidaemia oral contraception
Past history:	hypertension cerebrovascular disease peripheral vascular disease congenital heart disease
Medication:	antihypertensives anti-anginal therapy statins oral contraceptives and oestrogen replacement therapy (HRT)
Family history:	ischaemic heart disease diabetes hypertension hyperlipidaemia

Rhythm

- regular (sinus rhythm)
- regularly irregular (extra or dropped beats)
- irregularly irregular (atrial fibrillation; the pulse rate is different from the heart rate; listen and count at the apex whilst palpating the pulse)

Volume and character (Table 3.1)

- collapsing pulse: raise the patient's arm above the level of the heart whilst holding four fingers over the anterior forearm
- slow – rising, low volume, alternans, bisferiens and paradoxus by palpating the carotid pulse

Jugular venous pressure and pulse (Table 3.2; Fig. 3.1)

The patient should be lying at 45° with the neck relaxed. The JVP is seen welling up between the two heads of sternomastoid in the front of the neck on expiration.

Measure

- the vertical height of the top of the column of blood above the sternal angle.

The sternal angle is about 5 cm above the left atrium when the patient is lying at 45°. The normal central venous pressure (CVP) is 7 cm and therefore the jugular vein is normally just visible.

In the neck, the venous pulse differs from the arterial pulse:

- its height varies with posture, it is impalpable and low pressure means that it is easily abolished by light finger pressure
- it fills from above when light finger pressure is applied to the root of the neck
- the height varies with respiration (fall with inspiration and rise with expiration)
- there are two peaks with each pulsation, 'a' and 'v'.

The JVP is a better guide to right atrial pressure than the superficial external venous pulse which may be tortuous or obstructed by soft tissues in the neck.

If neither is obvious:

- Suspect a low level: unless the liver is tender, press on the abdomen gently but firmly. The

Table 3.1 Abnormalities of the arterial pulse

Type	Character	Seen in
Slow-rising	Low amplitude, slow rise, slow fall	Aortic stenosis
Collapsing	Large amplitude, rapid rise, rapid fall	Aortic incompetence; severe anaemia, hyperthyroidism; arteriovenous shunt, heart block, patent ductus arteriosus
Low volume	Thready	Low cardiac output states; hypovolaemic shock; valvular stenosis; pulmonary hypertension
Alternans	Alternate large- and small-amplitude beats (rarely noted in pulse; usually on taking blood pressure)	Left ventricular failure
Bisferiens	Double-topped ('notched')	Aortic stenosis with aortic incompetence
Paradoxus	Pulse volume decreases excessively with inspiration	Cardiac tamponade, constrictive pericarditis, severe inspiratory airways obstruction
Absent radial		Congenital anomaly (check brachials and blood pressure) Tied off at surgery or catheterisation. Arterial embolism

Table 3.2 Raised jugular venous pressure (JVP)

Character	Compression of neck and abdomen	Conclusion
Non-pulsatile	No change in JVP	Superior mediastinal obstruction • carcinoma of bronchus • large goitre • platysmal compression
Pulsatile	Jugular vein fills and empties	Right heart failure Expiratory airways obstruction Fluid overload Cardiac tamponade

'hepato-jugular reflux' (not 'reflex') has no pathophysiological significance; the sole purpose of this manoeuvre is to demonstrate the vein and to show that it can be filled (i.e. that the pressure is not high).

- Suspect a high level: the top of the column may be above the mastoid. Check if the ear lobes move with the cardiac cycle and sit the patient vertically to get a greater length of visible jugular vein above the right atrium.

If the jugular venous pressure is raised (especially if >10 cm):

- A large 'a' wave (corresponding with atrial systole) occurs when the right atrial pressure is raised, e.g. tricuspid stenosis, pulmonary hypertension, pulmonary stenosis and mitral stenosis.
- A cannon wave is a massive 'a' wave occurring in complete heart block when the right atrium contracts against a closed tricuspid valve.

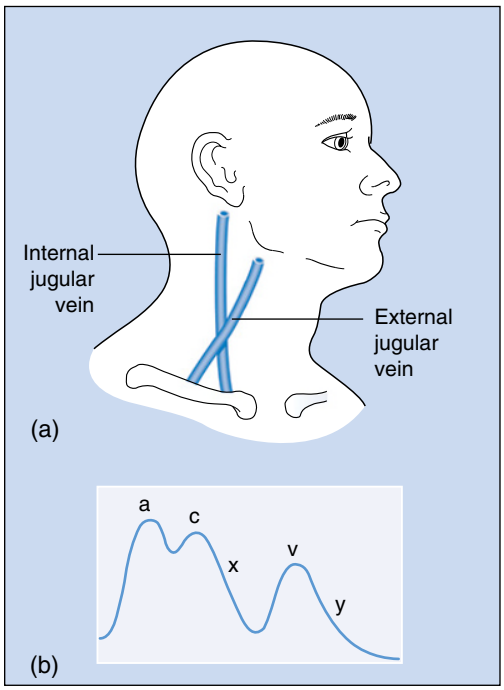


Figure 3.1 (a) Surface markings of the internal and external jugular veins. (b) Jugular venous pulse waveform.

- There is no 'a' wave in atrial fibrillation because there is no atrial systole.
- A large 'v' wave (corresponding with ventricular systole) indicates tricuspid incompetence (usually secondary to marked heart failure).

Heart

Observe

- scars of previous surgery
- visible apex beat
- visible parasternal heave

Palpate

- apex beat
- left parasternal area
- apex, base and aortic areas for thrills

The apex beat may be thrusting in left ventricular hypertrophy, displaced by cardiomegaly or left ventricular dilatation or tapping in nature, suggesting the accentuated first sound of mitral stenosis.

A parasternal heave is present when there is right ventricular hypertrophy.

Thrills are palpable murmurs felt over the relevant area in systole or diastole.

Auscultate the precordium (Fig. 3.2)

- Use the bell of the stethoscope to examine low-pitched noises, especially diastolic murmurs at the apex, and the diaphragm to examine high-pitched noises and the precordium generally.
- Palpate the right carotid artery when auscultating to identify the stages of the cardiac cycle.
- Ask the patient to roll onto their left side and listen over the apex to accentuate mitral murmurs and check their radiation.
- Ask the patient to sit up, lean forward and hold their breath in expiration to listen for aortic diastolic murmurs.
- Check for radiation of aortic stenotic murmurs to the carotid area.

Identify

- first and second heart sounds
- additional heart sounds
- murmurs and their radiation
- pericardial friction rub

First heart sound (S1): occurs at the onset of systole when mitral and tricuspid valves close; loud in hyperdynamic circulation and mitral stenosis, soft in heart failure and mitral regurgitation.

Second heart sound (S2): occurs at the end of systole when aortic and pulmonary valves close; split on inspiration (A2 then P2); fixed splitting in atrial septal defect; variable splitting with bundle branch blocks.

Third heart sound (S3): occurs immediately after S2 in early diastole; normal in young people and pregnancy; presents as 'gallop rhythm' in left ventricular failure.

Fourth heart sound (S4): occurs at the end of diastole before S1; present in severe left ventricular hypertrophy and aortic stenosis.

Systolic clicks: occur in early or mid-systole; indicate aortic or pulmonary stenosis, mitral valve prolapse and prosthetic heart valves.

Opening snap (OS): occurs in early diastole; indicates mitral stenosis with mobile valve leaflets and prosthetic valves; absent in calcific mitral stenosis.

Cardiac murmurs: auscultatory features are shown in Table 3.3.

Pericardial friction rub: low-pitched and scratchy; heard over the lower sternum; varies with posture and breathing.

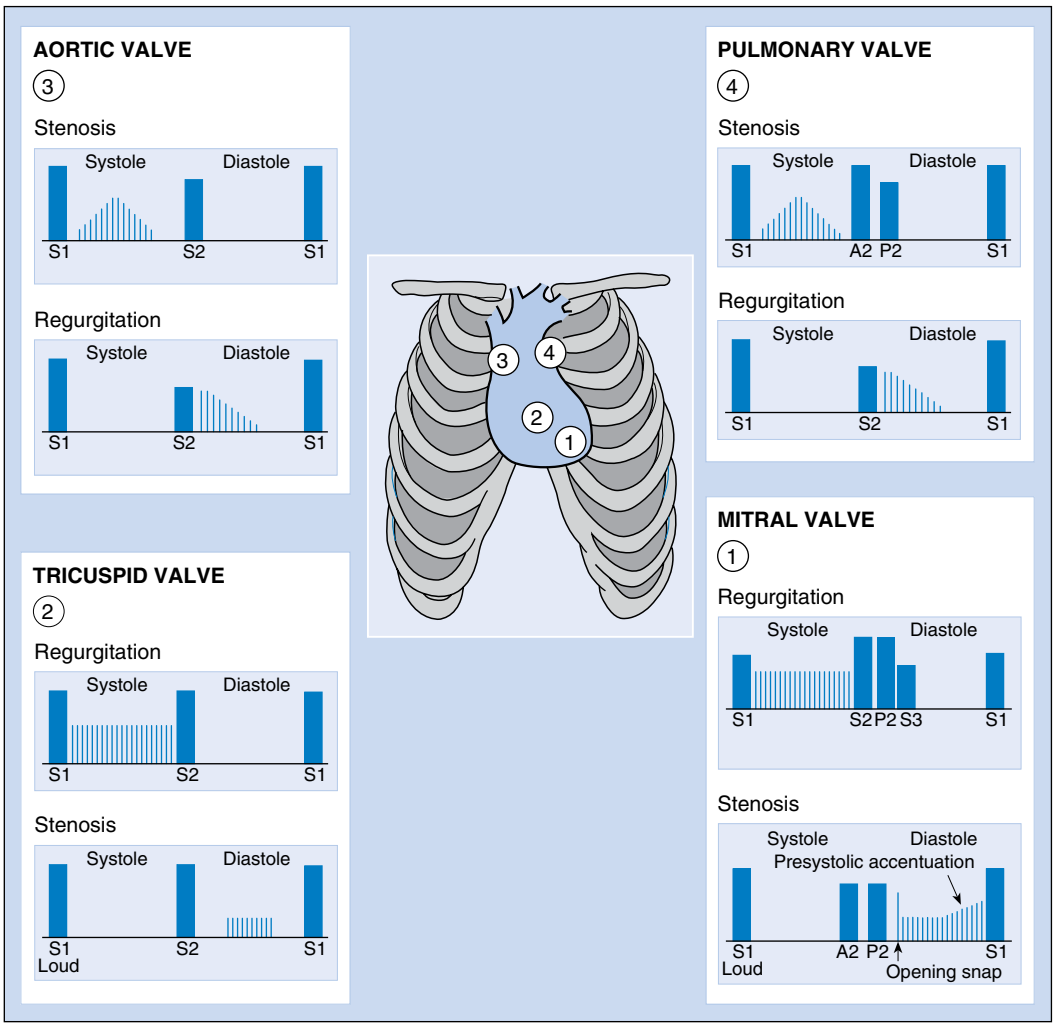


Figure 3.2 Suggested stethoscopic route (1–4) for listening to heart valves. 1, apex; 2, lower left sternal edge; 3, left second intercostal space; 4, right second intercostal space.

Complete the examination

- auscultate the carotids for bruits
- examine for ankle oedema
- sit the patient up and examine the lung bases for crackles and pleural effusions and check for sacral oedema
- examine the abdomen for hepatomegaly (which may be pulsatile) and abdominal aortic aneurysm
- dipstick the urine

Notes

Heart failure

A full cardiovascular history should be taken, focusing on evidence of chronic ischaemia or hypertension.

Left ventricular failure (LVF)

Signs

- dyspnoea on exertion or at rest
- tachycardia

Table 3.3 Characteristics of cardiac murmurs

Lesion	Murmur and position	Radiation and notes
Aortic stenosis	Harsh ejection systolic, maximal in second RICS; often loud with thrill. Possible ejection click	Radiates into neck
Aortic regurgitation	Blowing early diastolic decrescendo murmur maximal in third LICS, occasionally in second RICS. Best heard with patient sitting forwards in expiration	Radiates between right carotid and cardiac apex. Look for signs of coexistent connective tissue or other disorders
Mitral stenosis	Mid or late rumbling diastolic murmur at apex; presystolic accentuation if sinus rhythm. Loud mitral first sound. Opening snap if valve pliable	Turn patient on left side (and exercise) to accentuate murmur. Often atrial fibrillation
Mitral incompetence	Pansystolic at apex	Radiation to axilla; often heard parasternally
Mitral prolapse	Midsystolic at apex. High-pitched with click	Usually benign. Rarely associated connective tissue disease
Tricuspid incompetence Pulmonary stenosis	Pansystolic; maximal lower sternum Midsystolic; maximal in second LICS. Click if stenosis is valvular	'V' wave in neck and pulsatile liver increase on inspiration. Pulmonary component of second sound quiet and delayed
Pulmonary incompetence	Blowing early diastolic murmur, maximal in second and third LICS	Very rare
Ventricular septal defect	Loud, rough, pansystolic; maximal at third to fourth LICS parasternally	Small VSDs common
Atrial septal defect	Pulmonary systolic murmur with fixed split second sound	Possible tricuspid diastolic murmur if atrial septal defect flow is large
Patent ductus arteriosus	Machinery murmur, maximal in late systole, extending into diastole. Maximal in second to third LICS in mid-clavicular line	Also audible posteriorly
Coarctation of the aorta	Loud rough systolic, maximum over apex of left lung both posteriorly and anteriorly	Murmurs of scapular and internal mammary shunt collaterals. Radial femoral delay. Hypertension in arms

RICS, right intercostal space; LICS, left intercostal space; VSD, ventricular septal defect.

- gallop rhythm
- fine bi-basal crackles of pulmonary oedema
- pleural effusions

Right ventricular failure (RVF)

Signs

Signs of LVF plus

- raised JVP
- ankle oedema
- sacral oedema
- hepatomegaly
- ascites

If RVF is secondary to chronic lung disease (cor pulmonale) there is clinical evidence to suggest chronic obstructive pulmonary disease, pulmonary embolism or other forms of chronic lung disease.

Hypertension

A full cardiovascular history should be taken. Specific features in the history of a patient with hypertension are shown in Box 3.2.

Mild or moderate hypertension usually produces no abnormalities on physical examination other than raised blood pressure. Physical signs suggest long-standing or severe hypertension.

Box 3.2 History in a patient with hypertension

Symptoms:	angina breathlessness intermittent claudication headaches episodic palpitations visual disturbances
Risk factors:	smoking obesity alcohol excessive salt intake
Past history:	diabetes renal disease endocrine disease
Family history:	hypertension polycystic kidney disease
Medication:	combined oral contraceptive, non steroidal anti-inflammatory drugs

Examine for

- left ventricular hypertrophy
- loud A2 second heart sound
- heart failure
- cerebrovascular disease
- hypertensive retinopathy
- renal failure

Consider secondary causes of hypertension.

Look for evidence of

- renal artery stenosis: renal artery bruit in the epigastrium
- polycystic kidney disease
- other forms of chronic kidney disease
- coarctation of the aorta: radial-femoral arterial pulse delay, weak femoral pulses, bruits of the coarctation and of the scapular anastomoses, visible pulsation of the anastomoses
- Cushing syndrome
- acromegaly

Phaeochromocytoma and primary hyperaldosteronism (Conn syndrome) have no specific features on physical examination.

The ECG

A normal ECG is shown in Fig. 3.3.

Aide mémoire

- horizontally one little square is 0.04 s; one big square is 0.2 s

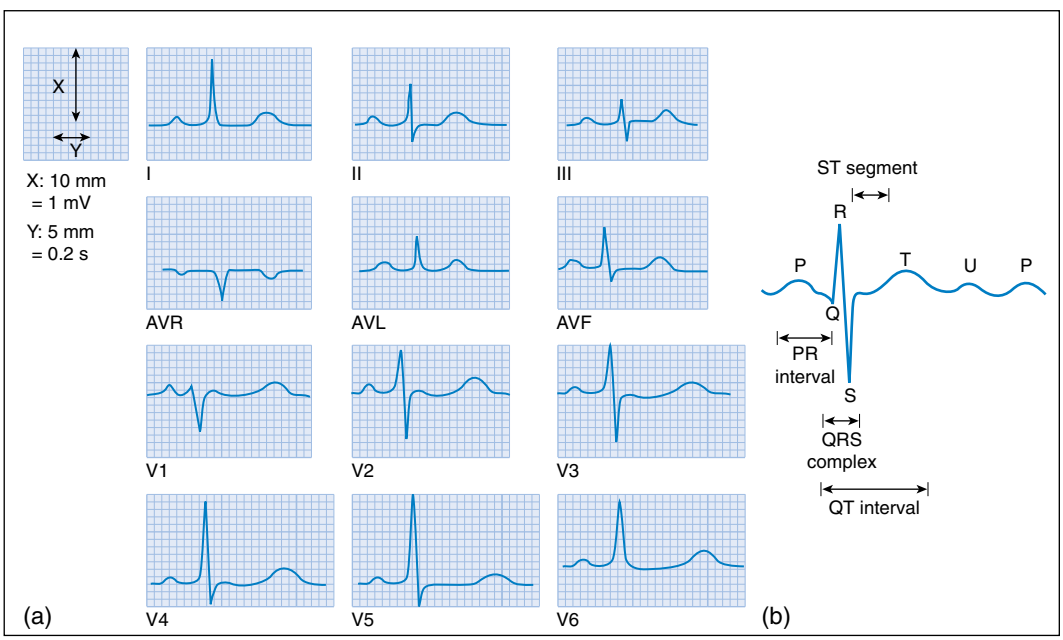


Figure 3.3 (a) Normal 12-lead electrocardiogram (ECG). (b) Waves of the normal ECG.

- vertically one little square is 0.1 mV
- normal PR interval is 0.12–0.2 s
- normal QRS duration is up to 0.12 s

The QT interval varies with rate. Upper limits of normal are approximately:

- rate 60/min QT 0.43 s
- rate 75/min QT 0.39 s
- rate 100/min QT 0.34 s.

Rate

- Count the large squares between two QRS complexes and divide into 300 (i.e. if two squares, the rate is 150/min).
- If the rate is less than 60/min the patient has a bradycardia; if greater than 100/min, a tachycardia.

Regularity

- Use the edge of a piece of paper to mark off a series of R waves, and then shift the paper along one or more complexes.
- The marks on the paper will still correspond with the R waves if the rhythm is regular. Total irregularity usually indicates atrial fibrillation.

Estimate the mean frontal QRS axis (Fig. 3.4)

- To gain a rough idea of the axis, find the limb lead with the maximum net positive deflection (sum of the positive R wave and negative Q and S waves); the axis lies close to this.
- Calculate the total deflection (R wave minus Q and S waves) in leads I and AVF which are perpendicular to each other (at 0 and 90°, respectively).
- Add these together as vectors (use the squares on the ECG paper); the net vector is the axis (see Fig. 3.4).
- The normal range is 0–90°.

Check individual waves and intervals

- for their presence, shape and duration.

P wave (atrial depolarisation)

- is most easily seen in V1 and V2
- is peaked in right atrial hypertrophy and bifid in left atrial hypertrophy (left atrial depolarisation occurs slightly later than right, giving a second peak)
- may be 'lost' (in the QRS complex) in nodal rhythm.

PR interval

- PR interval measured from the beginning of the P wave to the beginning of the QRS complex is usually 0.12–0.2 s.
- If the PR interval is prolonged or a normal 1 : 1 ratio of PQRS complexes is lost heart block is present (Fig. 10.14, Chapter 10).
- A short PR interval occurs in atrioventricular re-entrant tachycardias.

QRS complex

- The QRS complex is caused by the rapid depolarisation of the right and left ventricles.
- If the QRS complex is longer than 0.12 s bundle branch block exists.
- Pathological (broad, deep) Q waves are greater than 0.04 s (one small square wide) and greater than 0.2 mV. They may be normal in AVR or V1.

ST segment

- The ventricles are depolarised during the ST segment, which is normally isoelectric.

T wave

- The T wave is caused by repolarisation of the ventricles.

QT interval

- The QT interval is measured from the beginning of the QRS complex to the end of the T wave. It varies with heart rate, and the corrected QT (QTc) is calculated by dividing the QT interval by the square root of the preceding R–R interval. QTc values between 0.35 and 0.45 s are considered normal. Prolongation is associated with ventricular arrhythmias.

U wave

- occurs after the T wave and can be a normal finding.

Peripheral vascular system

Patients with peripheral vascular disease may complain of:

- transient motor or sensory loss in transient ischaemic attacks (TIAs)
- intermittent claudication
- intermittent visual loss.

General examination

Compare and assess

- temperature of the dorsum of each foot
- haemosiderosis, lipodermatosclerosis
- loss of hair over lower limbs and feet
- arterial ulceration
- venous ulceration

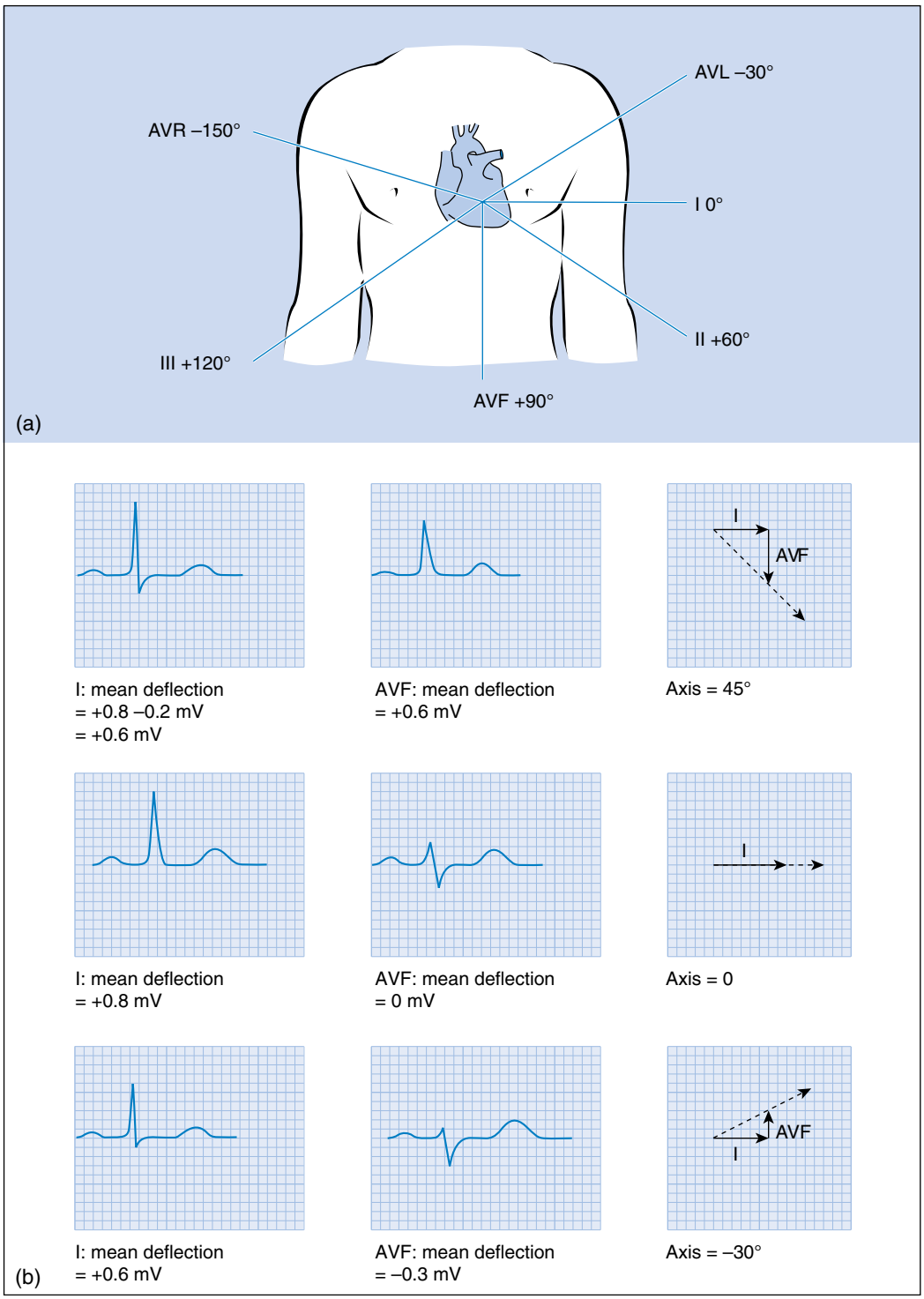


Figure 3.4 (a) Position of the limb leads. (b) Calculation of the cardiac axis.

Check

- capillary return in the great toe

Examine the arterial pulses

- radial
- brachial
- carotid
- femoral
- popliteal
- posterior tibial
- dorsalis pedis

Palpate

- the abdomen for evidence of abdominal aortic aneurysm

Auscultate for bruits

- carotid arteries
- epigastrium (renal artery stenosis)
- femoral arteries

Examine

- varicose veins where present

4

Respiratory system

Clinical assessment of the respiratory system is essential for accurate diagnosis, particularly in the context of acute respiratory disease where speedy clinical decision-making is of the essence. Whilst simple radiography, measurement of oxygen saturation and blood gas analysis are available in the majority of emergency clinical settings, the mainstay of diagnosis remains the clinical assessment. In chronic lung disease, the availability of sophisticated radiology and respiratory physiology can be used to confirm the diagnosis and monitor disease progress.

History

Key features of the history in a patient with respiratory disease are shown in Table 4.1.

Examination

Key abnormalities detected on examination of the chest are shown in Table 4.2.

General observation: note

- dyspnoea
- cyanosis
- evidence of loss of weight

Examine the hands for

- clubbing
- tobacco staining
- coarse tremor of outstretched hands
- bounding radial pulse

Check

- the pulse rate
- the height of the jugular venous pressure
- the tongue for cyanosis

Observe

- the shape of the chest and spine
- scars
- chest movements for symmetry and expansion
- the use of accessory muscles in the neck and shoulders
- visibly enlarged cervical lymph nodes

Count

- the respiratory rate

Examine the front and back of the chest in a logical manner, usually by palpating, percussing and auscultating the front of the chest first, followed by the rear. When examining the back of the chest, ask the patient to put their hands on their hips to facilitate examination of the lung bases laterally.

Palpation

The anterior surface markings of the lungs are shown in Fig. 4.1.

Palpate for

- chest expansion, comparing the movements of the two sides
- the trachea in the suprasternal notch to assess mediastinal shift with the patient's neck partially extended. (Local anatomical and pathological variants may produce tracheal deviation in the absence of lung disease, e.g. a goitre or spinal asymmetry.

Clinical Medicine Lecture Notes, Eighth Edition. John R. Bradley, Mark Gurnell, and Diana F. Wood.

Table 4.1 Key features of the history in respiratory disease

Feature	Details	Rationale
Basic details	Age, sex, current and previous occupations	Identify risk of occupational lung disease
Symptoms	Dyspnoea, chest pain, wheeze, cough, sputum and haemoptysis	Establish pattern of symptoms and their likely causes
Past medical history	Previous episodes or other lung disease	Suggestive of chronic or relapsing symptoms, i.e. asthma, COPD
Allergies	Identify known allergies in patient or family	Potential for allergic lung diseases
Smoking	Establish accurate smoking history	Increased risk of chronic lung disease and cancer

Table 4.2 Physical findings in common chest diseases

Pathology	Reduced chest wall movement	Mediastinal shift	Percussion note	Breath sounds	Vocal resonance	Added sounds
Pleural effusion	Affected side	Away from lesion (if large effusion)	Stony dull	Reduced or absent	Reduced or absent	Possibility of crackles above the effusion
Pneumothorax	Affected side	Away from lesion (if tension)	Normal or hyper-resonant	Reduced or absent	Reduced or absent	None
Consolidation	Affected side	None	Dull	Bronchial breathing	Increased	Crackles
Generalised fibrosis	Both sides	None	Normal	Vesicular	Increased	Fine, end-inspiratory crackles
Localised fibrosis	Affected side	Towards lesion	Dull	Bronchial breathing	Increased	Coarse crackles
Chronic obstructive pulmonary disease	None or both sides	None	Normal	Vesicular with prolonged expiration	Normal	Coarse crackles, expiratory wheezes
Asthma	Both sides	None	Normal	Vesicular with prolonged expiration	Normal	Expiratory wheezes

The position of the heart apex beat is of no help in assessing lung disease except if there is marked mediastinal shift.)

- cervical lymphadenopathy

Percussion

- examine the apices by percussing the clavicles
- move down the chest alternating right and left to compare both sides

Auscultation
Listen for

- bronchial breathing
- diminished breath sounds
- added sounds
  - wheezes
  - crackles
  - pleural rubs
- vocal resonance

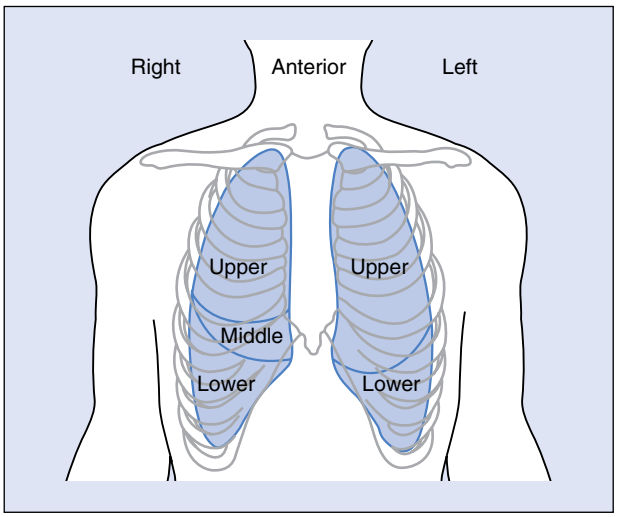


Figure 4.1 Surface markings of the lungs. Oblique fissures run along the line of the fifth/sixth rib; a horizontal fissure runs from the fourth costal cartilage to the sixth rib in the mid-axillary line. Note: On auscultation posteriorly you are listening mainly to lower lobes. Anteriorly you are listening mainly to upper lobes and on the right the middle lobe.

Notes

Haemoptysis

Aetiology: common

- lung cancer
- tuberculosis
- pulmonary embolism with infarction
- infection (e.g. pneumococcal pneumonia, lung abscess and *Klebsiella pneumoniae*)

Aetiology: uncommon

- foreign body – history of general anaesthetic, visit to dentist or inhalation of food
- coagulation disorders
- bronchiectatic cavities
- mitral stenosis
- Wegener's granulomatosis
- Goodpasture syndrome
- intrapulmonary vascular tumours

Investigation of haemoptysis

The usual clinical problem is to exclude carcinoma and tuberculosis. A full history and clinical examination will usually identify pulmonary infarction, foreign body, bronchiectasis, mitral stenosis and pulmonary oedema.

Perform

- sputum microscopy and culture, including for acid-fast bacilli
- sputum cytology for malignant cells
- chest X-ray
- CT or MRI scan to define the site and nature of the lesions seen on chest X-ray
- bronchoscopy with biopsy for cytology and culture
- CT-guided biopsy of mass lesions
- isotope (V/Q) lung scan +/– CT pulmonary angiogram if embolism is suspected

About 40% of patients with haemoptysis have no demonstrable cause. In patients who have had a single small haemoptysis, no other symptoms and a normal chest X-ray a follow-up chest X-ray after 1–2 months may be sufficient. Patients who have more than one small haemoptysis should be referred for further investigation.

Clubbing

Finger clubbing is associated with a range of respiratory diseases, but also with disease in the cardiovascular and gastrointestinal systems. Rarely, clubbing may be familial and innocent.

Respiratory causes

- carcinoma of bronchus
- chronic suppurative lung disease: empyema, lung abscess, bronchiectasis, cystic fibrosis
- fibrosing alveolitis
- asbestosis
- mesothelioma

Cardiac causes

- cyanotic congenital heart disease
- subacute bacterial endocarditis

Gastrointestinal causes

- Crohn's disease
- ulcerative colitis
- hepatic cirrhosis

Cyanosis

Cyanosis is a clinical description that refers to the blue-ish colour of a patient's lips and tongue (central) or fingers (peripheral). Central cyanosis is always accompanied by peripheral cyanosis.

Cyanosis is an unreliable guide to the degree of hypoxaemia. Central cyanosis is usually caused by the presence of an excess of reduced haemoglobin in the capillaries. Thus, in anaemia, severe hypoxaemia may be present without cyanosis.

Examine

- the underside of the patient's tongue and their finger nail beds (compare nail beds with your own)

If the tongue is cyanosed, the cyanosis is central in origin and secondary to:

- chronic bronchitis and emphysema, often with cor pulmonale
- congenital heart disease (cyanosis may be present only after exercise)
- polycythaemia
- massive pulmonary embolism.

If the tongue is not cyanosed but the finger nail beds are, the cyanosis is peripheral and secondary to:

- physiological causes (cold)
- pathology in peripheral vascular disease (the cyanosed parts feel cold).

Left ventricular failure may produce cyanosis that is partly central (pulmonary) and partly peripheral (poor peripheral circulation).

A rare cause of cyanosis, not caused by increased circulating reduced haemoglobin, is the presence of methaemoglobin (and/or sulphaemoglobin). The patient is relatively well and not necessarily dyspnoeic. Methaemoglobinaemia is usually drug-induced, e.g. sulphonamides, primaquine or nitrites.

Investigation of the respiratory system

Chest radiology

Normal chest X-rays are shown in Fig. 4.2 (postero-anterior) and Fig. 4.3 (lateral). Fig. 4.4 is a radiological chest diagram of lobar collapse. CT chest scans are shown in Fig. 4.5.

CT scanning is more sensitive than plain CXR and may be useful in detecting interstitial lung disease, cavitation and empyema.

Blood gases

The normal arterial values are:

- *Pa*O_2 10–13 kPa (values fall with age)
- *Pa*CO_2 4.7–6.0 kPa
- pH 7.35–7.45
- Standard HCO_3^- 23–27 mmol/l

The pH indicates acidosis or alkalosis.

PaCO_2 reflects alveolar ventilation.

PaO_2 reflects ventilation/perfusion imbalance, gas transfer or venous-to-arterial shunts.

$PaCO_2$

- raised may account for an acidosis of respiratory origin, e.g. respiratory failure
- reduced may account for an alkalosis as a result of hyperventilation

PaO_2

- raised suggests the patient is on added oxygen
- reduced indicates lung disease (the *Pa*CO_2 is usually high) or a right-to-left shunt

HCO_3^-

- raised standard HCO_3^- accounts for a metabolic alkalosis
- reduced accounts for a metabolic acidosis (usually renal or diabetic ketoacidosis)

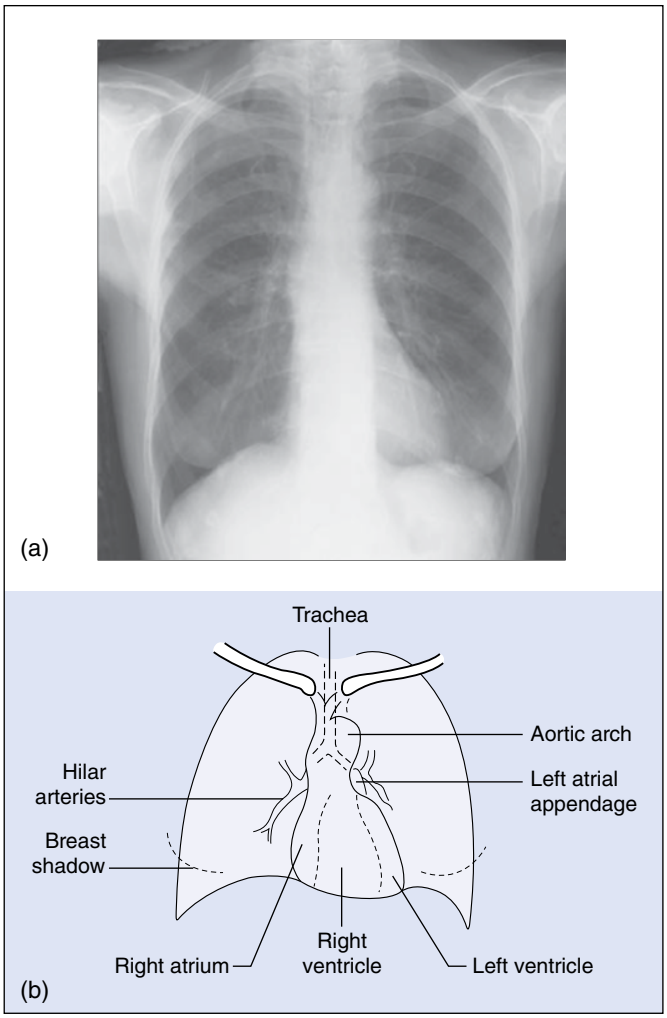


Figure 4.2 (a) Postero-anterior chest X-ray. (b) Diagrammatic representation.

Interpretation of blood gases

Arterial gas patterns

- High $PaCO_2$, low PaO_2: respiratory failure resulting from chronic obstructive pulmonary disease, asthma or chest wall disease (e.g. ankylosing spondylitis, neuromuscular disorders).
- Normal or low $PaCO_2$, low PaO_2: hypoxia as a result of parenchymal lung disease with normal airways. Hyperventilation due to hypoxia lowers the $PaCO_2$ (e.g. pulmonary embolism, fibrosing alveolitis. Also seen with venous admixture from right-to-left shunts
- Low $PaCO_2$, normal PaO_2: usually hyperventilation.

Causes of hypoxaemia

- *Hypoventilation*: sedative drugs, central nervous system disease, neuromuscular disease, chest trauma, obstructive sleep apnoea. The arterial $PaCO_2$ is characteristically high.
- *Ventilation/perfusion imbalance*: hyperventilation of some alveoli cannot compensate for the hypoxaemia resulting from the hypoventilation of other alveoli. Transfer factor is reduced.
- *Physiological shunt (venous admixture)*: deoxygenated blood passes straight to the left heart without

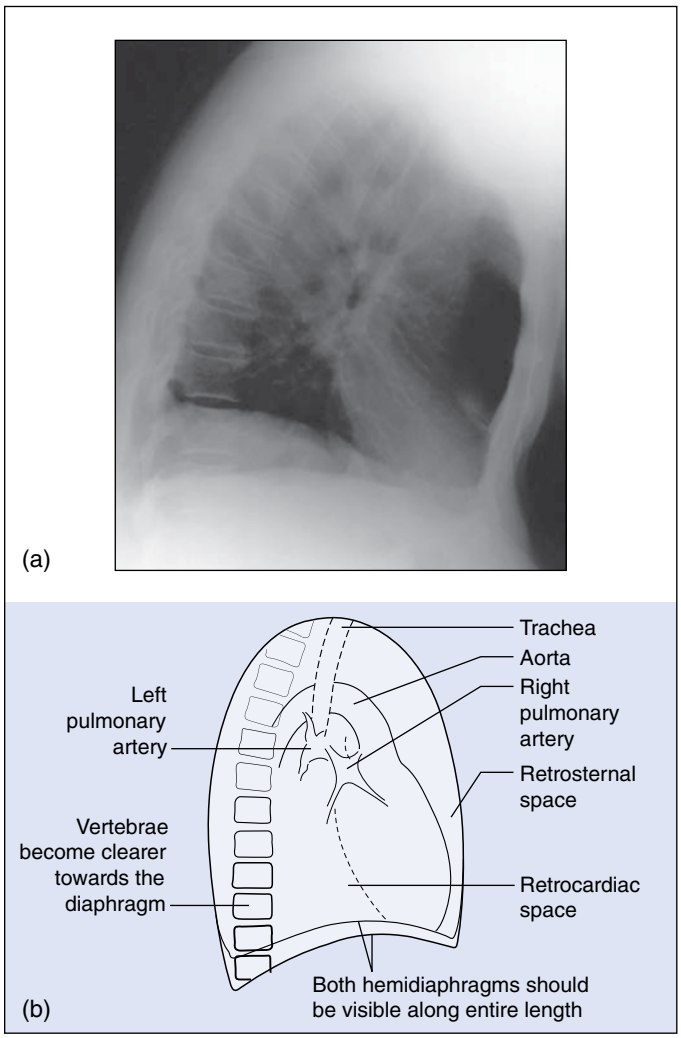


Figure 4.3 (a) Lateral chest X-ray. (b) Diagrammatic representation.

perfusing ventilated alveoli. This occurs in cyanotic congenital heart disease. The arterial PaO_2 is not significantly improved by the administration of oxygen.

- Low inspired oxygen concentration because of altitude or faulty apparatus.

Type 1 respiratory failure (low *Pa*O_2, normal/low *Pa*CO_2)

Patients with lung disease causing hypoxaemia with hyperventilation, e.g. pulmonary oedema, pneumonia, asthma, pulmonary fibrosis and pulmonary thromboembolism.

Type 2 respiratory failure (low *Pa*O_2, high *Pa*CO_2)

Patients with hypoxaemia and a high $PaCO_2$ due to defective ventilation caused by airways obstruction, reduced chest wall compliance or central nervous system disease.

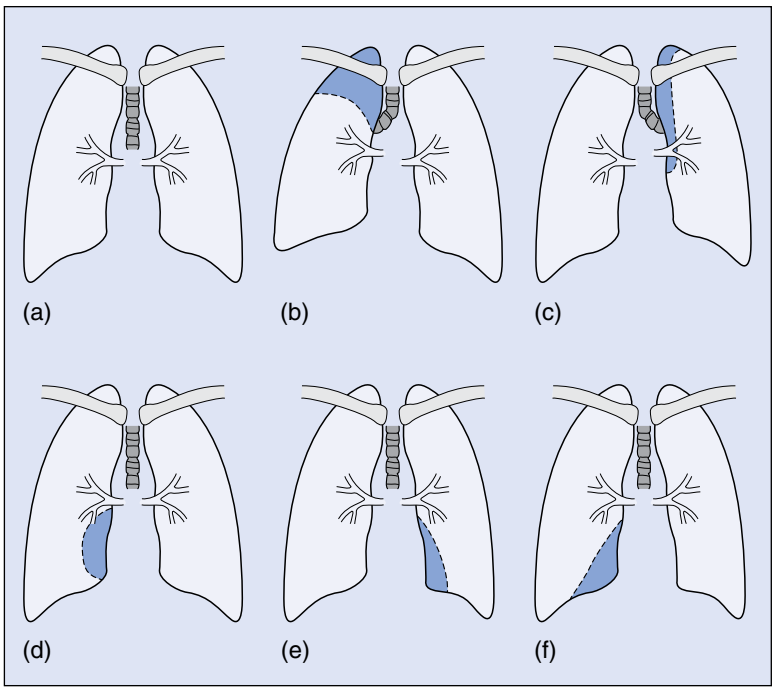


Figure 4.4 Diagrammatic representation of radiological appearance of lobar collapse. (a) Normal. (b) Right upper lobe: trachea deviated to right, right diaphragm and hilium elevated. (c) Left upper lobe: trachea deviated to left, left hilium and diaphragm elevated. (d) Right middle lobe: right heart border lost. (e) Left lower lobe: trachea may deviate to left, shadow behind heart. (f) Right lower lobe: trachea may deviate to right, outline of right diaphragm lost.

Pulmonary function tests

Spirometry (Fig. 4.6)

Subject exhales as fast and as long as possible from full inspiration into a spirometer, before and after bronchodilatation.

Interpretation

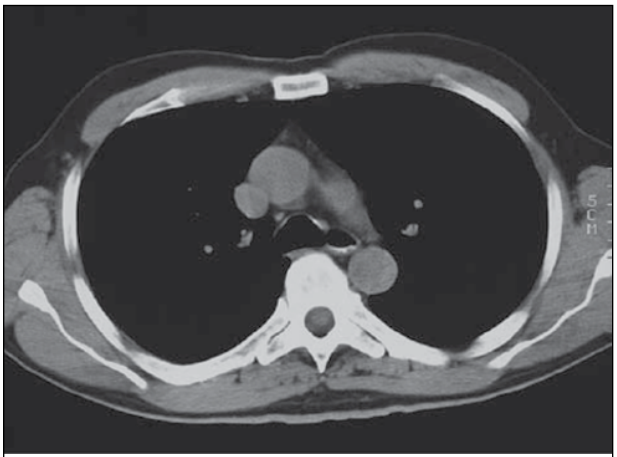
- Volume expired in the first second is the forced expiratory volume in 1 s (FEV1).
- Total expired is the forced vital capacity (FVC). Relaxed (slow) vital capacity may provide a better measure of trapped gas volume in chronic airways obstruction.
- Constriction of the major airways reduces the FEV1 more than the FVC.
- Restriction of the lungs reduces the FVC and, to a lesser degree, the FEV1.
- FEV1 : FVC (FEV%) ratio is low in obstructive airways disease (e.g. chronic bronchitis and asthma) and normal or high in fibrosing alveolitis and other interstitial lung diseases.
- Peak expiratory flow rate (PEFR) measures the rate of flow of exhaled air at the start of a forced expiration.

Normal values for all these tests vary with age, sex and size and appropriate nomograms should be consulted.

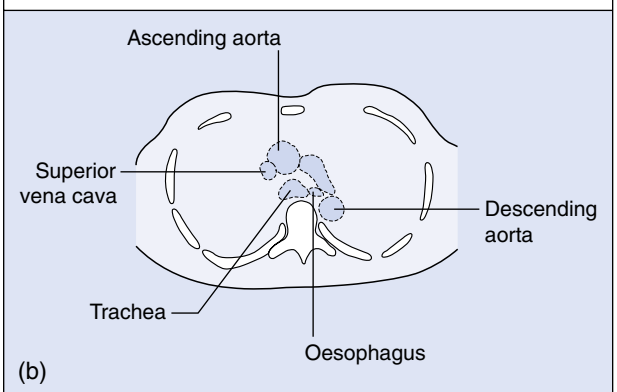
Transfer factor

- measures the transfer of a small concentration of carbon monoxide in the inspired air on to haemoglobin
- vital capacity must be over 1 litre and subject able to hold the breath for 15 s
- reduced in diseases that reduce ventilation or perfusion or alter the balance between them
- increased in pulmonary haemorrhage.

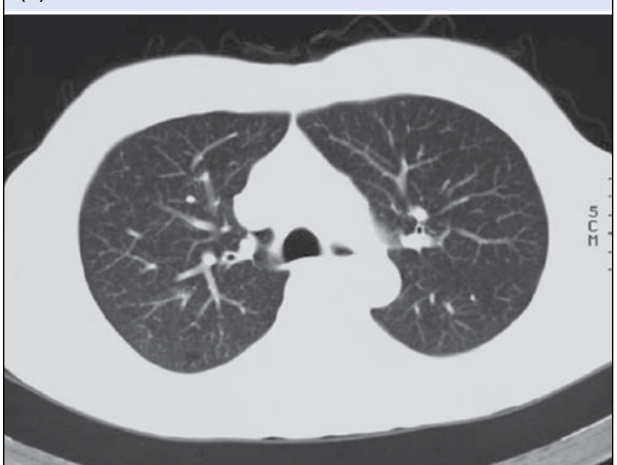
Correction must be made for haemoglobin concentration, because transfer factor varies directly with haemoglobin. Its chief value is for monitoring progression in interstitial disease and in confirming a diagnosis of pulmonary haemorrhage.



(a)



(b)



(c)

Figure 4.5 (a) CT of chest at the level of T4 vertebra. (b) Diagrammatic representation. (c) The same CT in which a different window setting has been used to visualise the lung markings (the window settings determine the range of densities displayed).

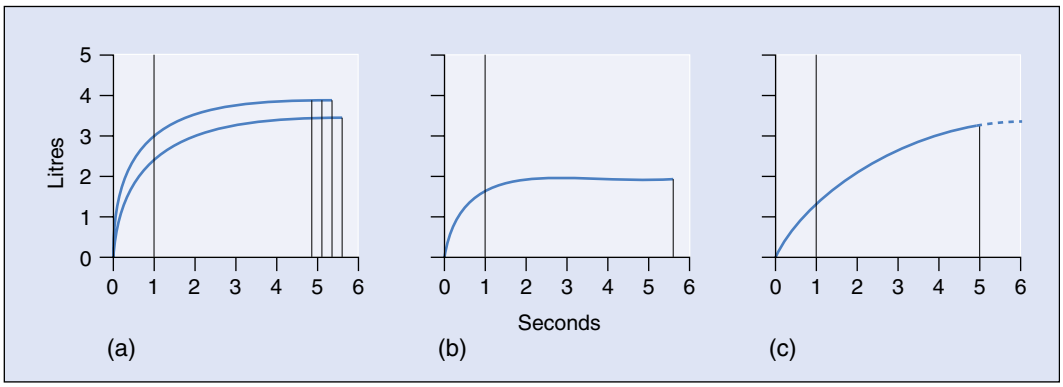


Figure 4.6 Spirometric patterns. (a) Normal (elderly man) forced expiratory volume/forced vital capacity (FEV/FVC) = 3.0/4.0 = 75%. (b) Restrictive, FEV/FVC = 1.8/2.0 = 90%. (c) Obstructive, FEV/FVC = 1.4/3.5 = 40%.

5

The abdomen

Examination of the abdomen may reveal abnormalities in a number of different systems including gastrointestinal, renal, haematological and cardiovascular disorders. Metabolic abnormalities including acute diabetic ketoacidosis and chronic hypercalcaemia may present with abdominal pain. In a patient with an acute abdomen, careful history-taking and examination forms a vital part of the initial management. In individuals with chronic disease, the history should dictate appropriate further investigations.

Key features in the history relating to gastrointestinal disease are shown in Table 5.1.

Examination of the abdomen

General observation: note

- is the patient in pain?
- evidence of weight loss

Inspect

- tongue, mouth, teeth and throat
- limbs for evidence of IV drug use

Examine the hands for

- clubbing
- leukonychia, koilonychia
- palmar erythema
- spider naevi
- Dupuytren's contracture

Inspect the eyes and conjunctivae for

- anaemia
- jaundice
- xanthelasmata

Palpate for lymphadenopathy

- neck
- supraclavicular fossae
- axillae
- groins

A scheme for examination of the abdomen is shown in Fig. 5.1. Lie the patient flat (one pillow) with arms by the sides. Look before palpation, have warm hands and palpate gently so as to gain the patient's confidence and to avoid hurting them. Ask the patient to let you know if you are hurting them. Check this by looking at the patient's face periodically during palpation, especially if you elicit guarding or rebound tenderness.

Observe the abdomen for

- general swelling with eversion of the umbilicus in ascites
- visible enlargement of internal organs: liver, spleen, kidneys, gall bladder, stomach, urinary bladder and pelvic organs
- abnormally distended veins: usually in cirrhosis with the direction of flow away from the umbilicus (portal hypertension). The flow is upwards from the groin in inferior vena cava obstruction (Fig. 5.2)
- scars of previous operations, striae, skin rashes and purpura
- pigmentation
- visible peristalsis

Clinical Medicine Lecture Notes, Eighth Edition. John R. Bradley, Mark Gurnell, and Diana F. Wood.

Table 5.1 Key features of the history in gastrointestinal disease

Feature	Details	Rationale
Basic details	Age, sex, occupation	Identify age-related and occupational risks
Symptoms	Dysphagia, dyspepsia, vomiting, haematemesis, abdominal pain, weight loss, diarrhoea, constipation, lower GI bleeding, symptoms of malabsorption including steatorrhoea, jaundice, pale stools, dark urine	Establish pattern of symptoms and their likely causes. Length of history important in aetiology of diarrhoea and jaundice
Past medical history	Previous episodes or other GI disease; blood transfusions; recent anaesthesia	May suggest recurrent or chronic GI disorder; hepatitis
Social history	Contacts with jaundiced patients; recent travel; residence abroad	Risk of infectious hepatitis; infectious diarrhoea
Family history	Family history of jaundice	Consider Gilbert's syndrome
Alcohol, smoking, drug abuse	Establish alcohol and smoking history; enquire after IV drug abuse	Important in aetiology of a number of GI and hepatic diseases
Medication	Careful history, in particular aspirin and NSAIDs, steroids; phenothiazines, oral contraception, antibiotics	Many drugs with GI/hepatic side effects

NSAIDs, non-steroidal anti-inflammatory drugs.

Palpate and percuss

- For internal organs and masses: start palpation in the right iliac fossa and work upwards towards the hepatic and splenic areas, first superficially and then deeper.
- Percuss the liver and spleen areas to avoid missing the lower border of a very large liver or spleen.

Liver

- The upper border is in the fourth to fifth intercostal space on percussion.
- The liver moves down on inspiration.
- Percussion over the liver is dull.
- If enlarged, the liver edge may be tender, regular or irregular, hard, firm or soft.
- Pulsatility suggests tricuspid incompetence.
- The liver may be of normal size but low because of hyperinflated lungs in chronic obstructive airway disease.

Spleen

- Smooth rounded swelling in left subcostal region, usually with a distinct lower edge.
- The spleen enlarges diagonally downward and across the abdomen in line with the ninth rib.
- The examining hand cannot get above the swelling.
- Percussion over the spleen is dull.
- There is a notch on the lower border of the spleen
- The spleen may be more easily palpated with the patient lying on the right side with the left leg flexed and abducted.

Kidneys

- Palpated in the loins bimanually, i.e. most easily felt by pushing the kidney forwards from behind on to the anterior palpating hand.
- They move slightly downwards on inspiration.
- The examining hand can easily get between the swelling and the costal margin.
- Percussion is resonant over the kidneys.
- The lower pole of the right kidney can often be felt in thin normal persons.

Abnormal masses

- Palpate for abnormal masses particularly in the epigastrium (gastric carcinoma) and suprapubic region (bladder distension, ovarian and uterine masses). Describe in terms of their size and margins.
- Note colonic masses. The descending colon is commonly palpable in the left iliac fossa.