Editors: Thomas, James; Monaghan, Tanya
Title: *Oxford Handbook of Clinical Examination and Practical Skills, 1st Edition*

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Preface

There are very few people who, in the course of their daily work, can approach a stranger, ask them to remove their clothes, and touch their bodies without fear of admonition. This unique position of doctors, medical students, and other health care professionals comes with many strings attached. We are expected to act 'professionally' and be competent and confident in all our dealings. This is hard to teach and hard to learn and many students are rightly daunted by the new position in which they find themselves.

We felt a little let down by many books during our time in medical school, and often found ourselves having to dip into several texts to appreciate a topic. This book, then, is the one text that we would have wanted as students covering all the main medical and surgical subspecialties. We anticipate it will be useful to students as they make the transition to being a doctor and also to junior doctors. We hope that it will be carried in coat pockets for quick glances as well as being suitable for study at home or in the library.

The first three chapters cover the basics of communication skills, history taking and general physical examination. Chapters 4,5,6,7,8,9,10,11,12,13 and 14 are divided by systems. In each of these there is a section on the common symptoms seen in that system, with the appropriate questions to ask the patient, details of how to examine parts of that system, and important patterns of disease presentation. Each of these system chapters is finished off with an 'elderly patient' page provided by Dr Richard Fuller. Following the systems, there are chapters on paediatric and psychiatric patients—something not found in many other books of this kind. The penultimate chapter—practical procedures—details all those tasks that junior doctors might be expected to perform. Finally, there is an extensive 'data interpretation' chapter which, whilst not exhaustive, tries to cover those topics such as ECG, ABG, and X-ray interpretation that may appear in a final 'OSCE' examination.

Although we have consulted experts on the contents of each chapter, any mistakes or omissions remain ours alone. We welcome any comments and suggestions for improvement from our reader—this book, after all, is for you.

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Tanya Monaghan

2006

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An extra special word of thanks is reserved for our models Adam Swallow, Geoffrey McConnell, and our female model who would like to remain anonymous. Their bravery and good humour made a potentially difficult few days very easy. They were a joy to work with. We thank the staff at the St James's University Hospital Medical Illustration Studio, in particular Tim Vernon, for taking the photographs.

We would also like to thank the staff at Oxford University Press, especially Catherine Barnes for having faith in us to take this project on and Elizabeth Reeve for her seemingly endless patience, support, and guidance.

A special word of thanks is reserved for our official ‘friend of the book’, Dr Richard Fuller, who provided all the ‘elderly patient’ pages. Aside from this, his steadfast and overtly biased support helped carry us through.

Finally, we would like to thank our good friend Dr Paul Johns. He read through much of the text and provided invaluable advice and support from the very beginning. We wish Paul the very best with his own writing projects and hope to work with him in the future.

Our panel of readers were responsible for confirming the medical accuracy of the text. Most have performed far beyond our expectations, we are eternally grateful to them all.

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How to use this book

The systems chapters

In each chapter, there are suggestions as to what questions to ask and how to proceed depending on the nature of the presenting complaint. These are not exhaustive and are intended as guidance.

The history parts of the systems chapters should be used in conjunction with Chapter 2 to build a full and thorough history.

Practical procedures

This chapter describes those practical procedures that the junior doctor or senior nurse may be expected to perform. Some should only be performed once you have been trained specifically in the correct technique be a more senior colleague.

Each procedure has a difficulty icon as follows:

 Requires no specific training and all medical graduates should be competent to perform.

 Requires some skill. Doctors in their second year after graduating should be able to perform with ease.

 More complex procedures which you may only come across in specialty jobs and will not be required to perform without specific guidance from seniors.

Reality versus theory

In describing the practical procedures, we have tried to be ‘realistic’. The methods described are the most commonly used across the profession and are aimed at helping the reader perform the procedure correctly and safely within a clinical environment.

There may be slight differences, therefore, between the way that a small number of the procedures are described here and the way that they are taught in a clinical skills laboratory. In addition, local trusts may use different equipment for some procedures. The good practitioner should be flexible and make changes to their routine accordingly.

Data interpretation

A minority of the reference ranges described for some of the biochemical tests in the data interpretation chapter may differ very slightly from those used by your local laboratory—this is dependant on the equipment and techniques used for measurement. Any differences are likely to be very small indeed. If in doubt, check with your local trust.

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Symbols and abbreviations

Symbols and abbreviations

↑
increased

↓
decreased

~
approximately


cross-reference


warning

ACL
anterior cruciate ligament

ACSM
American College of Sports Medicine

ACTH
adrenocorticotrophic hormone

ADH
antidiuretic hormone

ADP
adenosine diphosphate

AED
automated external defibrillator

AITFL
antero-inferior tibio-fibular ligament

ANCOVA
analysis of covariance

ANOVA
analysis of variance

AP
antero-posterior

APL
antiphopholipid

ASIS
anterior superior iliac crest

ATFL
anterior talofibular ligament

ATLS
advanced trauma life support

ATP
adenosine triphosphate

AV
atrioventricular

AVN

avascular necrosis

a-vO_2 diff

arterio venous difference in oxygen concentration

BLa

blood lactate

BMD

bone mineral density

BMI

body mass index

BMR

basal metabolic rate

CABG

coronary artery bypass graft

CDC

Center for Disease Control

CFL

calcaneofibular ligament

CHF

cardiac failure

CHO

carbohydrate

CISS

Comité International Sports des Sourds

CNS

central nervous system

CON

concentric

COPD

chronic obstructive pulmonary disease

CP

cerebral palsy

CP

creatine phosphate

CP-IRSA

Cerebral Palsy International Sport and Recreation Association

CPK

creatine phosphokinase

CPR

cardio-pulmonary resuscitation

CRP

C-reactive protein

CSF

cerebro-spinal fluid

CT

computer tomography

CTD

connective tissue disease

CVD

cardiovascular disease

DCO

doping control officer

DCS

diffuse cerebral swelling

DEXA

dual energy X-ray absorptiometry

DIP

distal interphalangeal

DM

diabetes mellitus

ECC

eccentric

ECG

electrocardiogram

ECRB

extensor carpi radialis brevis

ECRL

extensor carpi radialis longus

ECU

extensor carpi ulnaris

EDC

estimated date of confinement

EEA

energy expenditure for activity

EIA

exercise-induced asthma

EIB

exercise-induced bronchospasm

EMG

electromyography

ENMG

electoneuromyography

EPB

extensor polaris brevis

EPO

erythropoetin

ESR

erythrocyte sedimentation rate

ET

endurance trained

EVH

eucapnic voluntary hyperpnoea

FABER

flexion abduction external rotation

FBC

full blood count

$FeCO_2$

expired air carbon dioxide concentration

FeO_2

expired air oxygen concentration

FCU

flexor carpi ulnaris

FDS

flexor digitorum superficialis

FHx

family history

FPL

flexor policis longus

FSH

follicle stimulating hormone

GCS

Glasgow Coma Scale

GFR

glomerular filtration rate

GH

growth hormone

GnRH

gonadotrophin releasing hormone

Hb

haemaglobin

HDL

high density lipoprotein

HMB

beta-hydroxy-beta-methylbutyrate

HR

heart rate

Hct

haematocrit

HT

highly trained

IA

intra-arterial

IBD

inflammatory bowel disease

ICP

intracranial pressure

IGF-1

insulin-like growth factor 1

IHCD

Institute of Health and Care Development

IHD

ischaemic heart disease

IIH

idiopathic intracranial hypertension

HIS

International Headache Society

IOC

International Olympic Committee

ITB

ilio-tibial band

ITBFS

inio-tibial band friction syndrome

IVP

intravenous pyelogram

IZ

injury zone

JVP

jugular venous pressure

LDL

low density lipoprotein

LH

luetinizing horming

LMA

laryngeal mask airway

LOC

loss of consciousness

LV

left ventricle

LVH

left ventricular hypertrophy

MANOVA

multivariate analysis of the variance

MCL

medial collateral ligament

MCS

microscopy, culture, and sensitivity

MDI

metered dose inhaler

MI

myocardial infarction

MPHR

maximum predicted heart rate

MRI

magnetic resonance imaging

MRSA

methicillin-resistant *Staphylococcus* aureus

MSU

mid-stream urine sample

MTPJ

metatarsophalangeal Joint

NGB

National Governing Body

NSAIDs

Non-steroidal anti-inflammatory drugs

OA

osteoarthritis

OCD

osteochondritis dissicans

OCP

oral contraceptive pill

OHCM6

Oxford Handbook of Clinical Medicine 6th edn

OHCS6

Oxford Handbook of Clinical Specialties 6th edn

ORIF

open reduction and internal fixation

OTC

over-the-counter

PCL

posterior cruciate ligament

PCR

phospho-creatine (energy system)

PCS

post-concussion syndrome

PFJ

patello-femoral joint

Pi

inrganic phosphate

PIN

posterior interosseous nerve

PIP

proximal interphalangeal joint

PMH

past medical history

PNF

proprioneurofacilitation

POMS

profile of mood states

PRICE

protection, rest, ice, compression, elevation

PSIS

posterior superior iliac crest

PSYM

parasympathetic

PTFL

posterior talofibular ligament

Q

cardiac output

QID

4 times a day (quarter in die)

QSART

quantitative sudomotor axon reflex tests

RCC

red cell count

ROM

range of movement

RR

respiratory rate

RSO

resting sweat output

RTA

road traffic accident

RV

residual volume

SA

sinoatrial

SAH

subarachnoid haemorrhage

SAID

specific adaptations to imposed demand

SARA

sexually acquired reactive arthritis

SCAT

Standardised Concussion Assessment Tool

SEM

sports and exercise medicine

SIJ

sacro-iliac joint

SLAP

superior labrum anterior to posterior

SLE

systemic lupus erythematosus

SLR

straight leg raise

SOB

shortness of breath

SPECT

single photon emission computer tomography

SYM

sympathetic

SV

stroke volume

TBI

traumatic brain injury

TFCC

triangular fibrocartilage complex

TGA

transposition of the great arteries

TLac

lactate threshold (aerobic/anaerobic threshold)

TUE

therapeutic use exemption

UCL

ulnar collateral ligament

URTI

upper respiratory tract infection

US

ultrasound

UT

untrained

ULTT

upper limb tension test

UV

ultraviolet

VA

alveolar ventilation

VEGF

vascular derived growth factor

VMO

vastus medialis obliquus

VE

minute ventilation

VI

visually impaired

VO_2

oxygen uptake

WADA

World Anti-Doping Agency

WCC

white cell count

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Chapter 1

Communication Skills

Introduction

Communication skills are notoriously hard to teach and describe. There are too many possible situations that one might encounter to be able to draw rules or guidelines. In addition, your actions will depend greatly on the personalities present—not least of all your own!

Using this chapter

Over the following pages, there is some general advice about communicating in different situations and to different people. We have not provided rules to stick to, but rather tried to give the reader an appreciation of the great many ways the same situation may be tackled.

Ultimately, skill at communication comes from practice and a large amount of common sense.

A huge amount has been written about communication skills in medicine. Most is a mix of accepted protocols and personal opinion—this chapter is no different.

The rule is: there are no rules.

Communication models

There are many models of the doctor-patient encounter which have been argued over at great length for years. These are for the hardened students of communication only. We mention them only so that the reader is aware of their existence.

Patient-centred communication

In recent years, there has been a significant change in the way healthcare workers interact with patients. The biomedical model has fallen out of favour. Instead, there is an appreciation that the patient has a unique experience of the illness involving the social, psychological, and behavioural effects of the disease.

The 'biomedical' model

- Doctor is in charge of the consultation.
- Focus is on disease management.

The patient-centred model

- Power and decision-making is shared.
- Address and treat the whole patient.

Box 1.1 Key points in the patient-centred model

- Explore the disease and the patient's experience of it:
  - Understand the patient's ideas and feelings about the illness.
  - Appreciate the impact on the patient's quality of life and psychosocial well-being.
  - Understand the patient's expectations of the consultation.
- Understand the whole person:
  - Family.
  - Social environment.
  - Beliefs.
- Find common ground on management.

- Establish the doctor-patient relationship.
- Be realistic:
  - Priorities for treatment.
  - Resources.

Box 1.2 Confidentiality

As a doctor, health care worker or student, you are party to personal and confidential information. There are certain rules that you should abide by and times when confidentiality must or should be broken (p.32). The essence for day-to-day practice is:

Never tell anyone about a patient unless it is directly related to their care.

This includes relatives and can be very difficult at times, particularly if a relative asks you directly about something confidential.

You can reinforce the importance of confidentiality to relatives and visitors. If asked by a relative to speak to them about a patient, it is a good idea to approach the patient and ask their permission, within full view of the relative.

This rule also applies to friends outside of medicine. As doctors and others, we come across many amazing, bizarre, amusing, or uplifting stories on a day-to-day basis but, like any other kind of information, these should not be shared with anyone.

If you do intend to use an anecdote for some after-dinner entertainment, at the very least, you should ensure that there is nothing in your story that could possibly lead to the identification of the person involved.

Essential considerations

Attitudes

Patients are entrusting their health and personal information to you—they want someone who is confident, friendly, competent, and above all, is trustworthy.

Personal appearance

First impressions count—and studies have consistently shown that your appearance (clothes, hair, make-up) has a great impact on the patients' opinion of you and their willingness to interact with you. Part of that intangible 'professionalism' comes from your image.

The white-coat is still part of medical culture although sadly appears to be dying out in the UK at this time. Fashions in clothing change rapidly but some basic rules still apply.

- Neutralize any extreme tastes in fashion that you may have.
- Men should usually wear a shirt and tie.
- Women may wear skirts or trousers but the length of the skirts should not raise any eyebrows.
- The belly should be covered—even during the summer!
- The shoulders, likewise, should usually be covered.
- Shoes should be polished and clean.
- *Clean* surgical scrubs may be worn if appropriate.
- Hair should be relatively conservatively styled and no hair should be over the face. It is advised to wear long hair tied up.
- Your name badge should be clearly visible—worn at the belt or shirt pocket is acceptable.*
- Stethoscopes are best carried or held in a coat-pocket—worn at the neck is acceptable but a little pretentious, according to some views.
  - Try not to tuck items in your belt—use pockets or belt-holders for mobile-phones, keys, and wallets.**

▸ Psychiatry, paediatrics, and a handful of other specialties require a different dress code as they deal with patients requiring differing tech—niques to bond.

** One of the authors once saw a female medical student carrying the *Oxford Handbook of Clinical Medicine* down the back of her trousers where it was practically wedged between her buttocks—not a pretty sight!

Timing

If in a hospital setting, make sure that your discussion is not during an allocated quiet time—or immediately before one is to start! You should also avoid mealtimes or when the patient's long-lost relative has just come to visit.

▸ If taking the patient from the bedside, ask the supervising doctor (if not you) and the nursing staff—and let all concerned know where you have gone in case the patient is needed.

Setting

Students, doctors, and others tend to see patients on busy wards which provide distractions that can break the interaction. Often this is necessary during the course of a busy day. However, if you are intending to discuss a matter of delicacy requiring concentration on both your parts, consider the following conditions:

- The room should be quiet, private, and free from disturbances.
- There should be enough seating for everyone.
- Chairs should be comfortable enough for an extended conversation.
- Arrange the seats close to yours with no intervening tables or other furniture.

Box 1.3 Becoming a good communicator

Learning

Like all aspects of medicine, learning is a lifelong process. One part of this, particularly true of communication skills and at the beginning of your career, is **watching others.**

The student should take every opportunity to observe doctor-patient interactions.

▸ You should ask to be present during difficult conversations.

Instead of glazing over during consultations in clinic or on the ward round, you should watch the interaction and consider if the behaviours you see are worth emulating or avoiding. Consider how you might adjust your future behaviour.

'Cherry-pick' the things you like and use them as your own—building up your own repertoire of communication techniques.

Spontaneity versus learnt behaviours

If you watch a good communicator (in any field) you will see them making friendly conversation, spontaneous jokes, and using words and phrases that put people at ease. It seems natural, relaxed, and spontaneous.

Watching that same person interact with someone else can shatter the illusion as you see them using the very same 'spontaneous' jokes and other gambits from their repertoire.

This is one of the keys to good communication—an ability to judge the situation and pull the appropriate phrase, word, or action from your internal catalogue. If done well, it leads to a smooth interaction with no hesitations or misunderstandings. The additional advantage is that your mental processes are free to consider the next move, mull over what has been said or consider the findings, whilst externally you are partially on 'auto-pilot'.

During physical examination, this is particularly relevant. You should be able to coax the wanted actions from the patient and put them at ease whilst considering the findings and your next step.

It must be stressed, that this is *not* the same as lacking concentration—quite the opposite, in fact.

Essential rules

Avoid medical jargon

The problem is that medics are so immersed in jargon that it becomes part of their daily speech. The patient may not understand the words or may have a different idea as to the meaning.

Technical words such as 'myocardial infarction' are in obvious need of avoidance or explanation. Consider terms such as 'exacerbate', 'chronic', 'numb', and 'sputum'—these may seem obvious in meaning to you but not to the patient.

You may also think that some terms such as 'angina' and 'migraine' are so well known that they don't need adjustment—but these are very often misinterpreted. Some examples are in the box opposite.

Remember the name

Forgetting someone's name is what we all fear but is relatively easy to disguise by simple avoidance. However, the use of a name will make you seem to be taking a greater interest. It is particularly important that you remember the patient's name when talking to family. Getting the name wrong is embarrassing and seriously undermines their confidence in you.

Aside from actually remembering the name, it is a good idea to have it written down and within sight—either on a piece of paper in your hand or on the desk, or at the head of the patient's bed. To be seen visibly glancing at the name is forgivable but does undermine you to a certain extent.

A few examples of words that may be misinterpreted by the patient.

Table 1.1 Some examples of differing interpretations.

Word	Your meaning	The patient's understanding
Acute	Rapid onset	Very bad, severe
Chronic	Long duration	Very bad, severe
Sick	Nauseated, vomiting	Unwell
Angina	Chest pain associated with ischaemic heart disease	Heart attack, shortness of breath, palpitations
Migraine	Specific headache disorder	Any severe headache
Numb	Without sensation	Weak

Getting started

The start of an encounter is important, but is fraught with potential difficulties. Like everything else in this chapter, there are no hard-and-fast rules but some issues that you should take into consideration.

- Are you using a language the patient can understand?
- Can they hear you?

Greeting

Beware of 'good afternoon' and 'good morning'. These can be inappropriate if you are about to break some bad news or if there is another reason for distress. Consider instead a simple 'hello'.

Shaking hands

A difficult issue which, again, needs to be judged at the time.

Physical contact always seems friendly and warms a person to you—but a hand-shake may be seen as overly formal by some. Perhaps consider using some other form of touch—such as a slight guiding hand on their arm as they enter the room or a brief touch to the forearm. (See also p.28.)

Introductions

This is a minefield! You may wish to alter your greeting depending on circumstances—choose terms that suit *you*.

Title—them

Older patients may prefer to be called Mr or Mrs; younger patients would find it odd. Difficulty arises with females when you don't know their marital status. Some younger or married patients may find the term ‘Ms’ offensive.

Using the patient's first name may be considered too informal by some—whilst a change to using the family name mid-way through the encounter will seem very abrasive and unfriendly.

There are no rules here and common sense is required to judge the situation at the time. When unsure, the best option is always to ask.

‘Is it Mrs or Miss Butterworth?’ ‘May I call you Mary?’

Title—you

The title ‘doctor’ has always been a status symbol and a badge of authority—within the healthcare professions, at least. Young doctors may be reluctant to part with the title so soon after acquiring it but, in these days when consultations are becoming two-way conversations between equals, should you really introduce yourself as ‘Dr’?

Many patients will simply call you ‘doctor’ and the matter doesn't arise. The authors prefer using first names in most circumstances but some elderly patients prefer—and expect—a certain level of formality so the situation has to be judged at the time.

Standing

Although this might be considered old-fashioned by some younger people, standing is a universal mark of respect. You should always stand when a patient enters a room and take your seat at the same time as them. You should also stand as they leave but, if you have established a good rapport during the consultation, this isn't absolutely necessary.

General principles

Demeanour

Give the patient your full attention. Appear encouraging with a warm, friendly manner. Use appropriate facial expressions—don't look bored!

Define your role

Along with the standard introductions, you should always make it clear who you are and what your role is. You might also wish to say who your seniors are, if appropriate.

Style of questioning

Open questions versus closed questions

Open questions are those where any answer is possible.

‘What's the problem?’
‘How does it feel?’

These allow the patient to give you the true answer in their own words. Be careful not to lead them with closed questions.

Compare ‘How much does it hurt?’ to ‘Does it hurt a lot?’. The former allows the patient to tell you how the pain feels on a wide spectrum of severity, the latter leaves the patient only two options—and will not give a true reflection of the severity.

Multiple choice questions

Often, patients have difficulty with an open question if they are not quite sure what you mean. A question about the character of pain, for example, is rather hard to form and patients will often not know quite what you mean (‘What sort of pain is it’, ‘What does it feel like, exactly?’).

In these circumstances, you may wish to give them a few examples—but leave the list open-ended for them to add their own words. You must be very careful *not* to give the answer that you are expecting from them. For example, in a patient who you suspect has angina (‘crushing’ pain), you could ask:

‘What sort of a pain is it … burning, stabbing, aching, for example…?’

Clarifying questions

Use clarifying questions to get the full details:

‘When you say dizzy, what exactly do you mean?’

Reflective comments

Use reflective comments to encourage the patient to go on and reassure them that you are following the story:

‘Yes, I see that.’

Staying on topic

You should be forceful but friendly when keeping the patient on the topic you want or moving the patient onto a new topic. Don't be afraid to interrupt them—some patients will talk for hours if you let them!

‘Before we move on to that, I would just like to get all the details of this dizziness.’ ‘We'll come to that in a moment.’

Difficult questions

Apologize for potentially offensive or embarrassing questions:

‘I'm sorry to have to ask you this but…’

Eye-contact

▸ Make eye-contact and look at the patient when they are speaking.

Make a note of eye-contact next time you are in conversation with a friend or colleague.

In normal conversations, the speaker usually looks away whilst the listener looks directly at the speaker. The roles then changes when the other person starts talking … and so on.

In the medical situation, whilst the patient is speaking, you may be tempted to make notes, read the referral letter, look at a test result, or similar—you should resist and stick to the ‘normal’ rules of eye-contact.

Adjusting your manner

You would clearly not talk to another doctor as you would someone with no medical knowledge. This is a difficult area, you should try to adjust you manner and speech according to the patient's educational level.

This is can be extremely difficult—you should not make assumptions on intellect or understanding solely on educational history.

A safe approach is to start in a relatively neutral way and then adjust your manner and speech based on what you see and hear in the first minute or two of the interaction—but be alert to whether this is effective and make changes accordingly.

Interruptions

Apologize to the patient if you are interrupted.

Don't take offence or get annoyed

As well as being directly aggressive or offensive, people may be thoughtless in their speech or manner and cause offence when they don't mean to. As a professional, you should rise above this.

Communicating with deaf patients

People who are hard of hearing may cope with the problem by using a hearing-aid, lip-reading, or using sign language. Whichever

technique is used (if any), some simple rules should always apply:

- Speak clearly but not too slowly.
- Don't repeat a sentence if it is misunderstood—say the same thing in a different way.
- Write things down if necessary.
- Use plain English and avoid waffling.
- Be patient and take the time to communicate properly.
- Check understanding frequently.
- Consider finding an amplifier—many elderly medicine wards will have one available.

Lip-readers

Patients who are able to lip-read do so by looking at the normal movements of your lips and face during speech. Exaggerating movements or speaking loudly will distort these and make it harder for them to understand. In addition to the points above, when talking to lip-readers:

- Maintain eye-contact.
- Don't shout.
- Speak clearly but not too slowly.
- Do not exaggerate your oral or facial movements.

British Sign Language (BSL)

- It should be appreciated that BSL is not a signed version of English—it is a distinct language with its own grammar and syntax.
- For BSL users, English is a 2nd or 3rd language so using a pen and paper may not be effective or safe for discussing complex topics or gaining consent.
- Seek an official BSL interpreter, if possible, and follow the rules on working with interpreters on p.18.

Cross-cultural communication

Cultural background and tradition may have a large influence on disease management. Beliefs about the origin of disease and prejudices or stigma surrounding the diagnosis can make dealing with the problem challenging.

Be aware of all possible implications of a person's cultural background. For example, a Muslim may not take anything by mouth in the daylight hours during Ramadan. This may have serious implications for tablettaking, particularly for chronic diseases such as diabetes.

Above all, be aware of prejudice—yours and theirs.

Interpreters

Official communicators are bound by a code of ethics, impartiality, and confidentiality—friends and relatives are not.

It is often impossible to be sure that a relative is passing on all that is said in the correct way.

Sometimes, the patient's children are used to interpret—this is clearly not advisable for a number of reasons. This not only places too much responsibility on the child but they may not be able to explain difficult concepts. In addition, conversations about sex, death, or other difficult topics may be unsuitable for the child to be party to.

Using an official interpreter

Before you start

- Brief the interpreter on the situation, clarify your role and the work of the department, if necessary.
- Allow the interpreter to introduce themselves to the patient and explain their role.
- Arrange seating so that the patient can see the interpreter and doctor equally.

- Allow enough time (at least twice as long as normal).

During the exchange

- Speak to the patient, *not* the interpreter. This may be hard at first, but you should speak to and look at the patient at all times.
- Be patient, some concepts are hard to explain.
- Avoid complex terms and grammar.
- Avoid jargon.
- Avoid slang and colloquialisms which may be hard to interpret correctly.
- Check understanding frequently.

Finishing off

- Check understanding.
- Allow time for questions.
- ▸ If the conversation has been distressing, offer the interpreter support and let their manager know.

Written information

- If interpreting written information, read it out loud. The interpreter may not necessarily be able to translate written language as easily.
- Many departments and charities provide some written information in a variety of languages—some also provide tapes. You should be aware of what your department has to offer.

Imparting information

There are some guidelines that should apply when you are imparting any information—good or bad—to a patient.

- Identify the topic for discussion.
- Identify the people present and ask if there is anyone else they would like to be there.
- Establish previous experience/knowledge.
- Keep the sentences and explanations short and simple.
- Repeat important information.
- Allow time for feedback, questions and check understanding.
- Be honest!

The importance of silence

In conversations that you may have with friends or colleagues, your aim is to avoid silence using filler noises such as ‘um’ and ‘ah’ whilst pausing.

In medical situations, silences should be embraced and used to extract more information from the patient. Use silence to listen.

Practice is needed as the inexperienced may find this uncomfortable. It is often useful, however, to remain silent once the patient has answered your question. You will usually find that they start speaking again—and often impart useful and enlightening facts which you would not otherwise have gleaned.

Angry patients

Use body language to take charge of the situation without appearing aggressive (📖 p.28). Throughout the exchange, you should remain polite, avoiding confrontation and resist becoming angry yourself.

- Look to your own safety first.
- Calm the situation *then* establish the facts of the case. Anger is often secondary to some other emotion such as loss, fear or guilt.
- Acknowledge their emotions.
  - 'I can see this has made you angry.'
  - 'It's understandable that you should feel like this.'
- Steer the conversation away from the area of unhappiness towards the positive and plans to move the situation forward.
- Don't incriminate colleagues—the patients may remember your throw-away comments which could come back to haunt you. Avoid remarks like 'he shouldn't have done that'.
- Emphasize any grounds, for optimism, or plans for resolving the situation and putting things right.

Telephone communication

- The essential rule of confidentiality is that you must not impart personal information to anyone without the express permission of the patient concerned—except in a few specific circumstances (📖 p.32).
- You must not give out any confidential information over the telephone as you cannot be sure of the identity of the caller. All communication should be done face-to-face. This may cause difficulty if a relative calls to ask about the patient, but you should remain strict.
- If telephone communication is essential but you are in doubt as to the caller's identity, you may wish to take their number and call them back.

Talking about sex

This is a cause of considerable embarrassment for the patient and for the inexperienced professional. Sexual questions are usually inappropriate to be overheard by friends or relatives—so ask them to leave. Your aim is to put the patient at ease and make their responses more forthcoming.

- The key is to ask direct, clear questions and show no embarrassment yourself.
- You should maintain eye contact.
- You should also show *no surprise* whatsoever—even if the sexual practices described differ from your own or those that you would consider acceptable.
- Try to become au fait with sexual slang and sexual practices which you might not be familiar with previously.
  - A failure to understand slang may lead to an immediate barrier in the consultation.
- In general, you should not use slang terms first. You may wish to consider mirroring the patient's speech as you continue the conversation.
- See 📖 p.408 for details of the sexual history.

Breaking bad news

Breaking bad news is feared by students and, indeed, no-one likes doing it. However, knowing that you have broken difficult news in a sensitive way and that you have helped the patient through a terrible experience can be one of the most uplifting aspects of working in healthcare.

Before you start

- Confirm all the information *for yourself* and ensure that you have all the information to hand, if necessary.
- Speak to the nursing staff to get background information on what the patient knows, their fears, and details of the relationship with any family or friends who may be present.

Choose the right place

- Pick a quiet, private room where you won't be disturbed.
- Ensure there is no intervening desk or other piece of furniture.
- Arrange the chairs so that everyone can be seen equally.
- Hand your bleep/mobile phone to a colleague.

Ensure the right people are present

- Invite a member of the nursing staff to join you—particularly if they have already established a relationship with the patient.
  - Remember, it is usually the nursing staff that will be dealing with the patient and relatives when you have left so they need to know exactly what was said.
- Would the patient like anyone present?

Remember the general principles

See  p.6 and p.8. Avoid jargon and speak slowly and clearly.

Establish previous knowledge

It is essential to understand what the patient already knows. The situation is very different in the case of a patient who knows that you have been looking for cancer to one who thinks their cough is due to a cold.

'What do you know so far?'
'What have the other doctors told you?'

How much do they want to know?

This is key. Before you consider breaking bad news, you have to discover if the patient actually wants to hear it. Ask an open question such as:

'Have you thought about what might be the cause of these problems?'
'Do you know why we've been doing these tests?'

You can also ask directly if they want to hear what you might have to say:

'Are you the sort of person who likes to know all the available facts?'

Warning shots

If they do want to know, you should break the news in a step-wise fashion, delivering multiple 'warning-shots'. This gives the patient a chance to stop you if they've heard enough, or to ask for more information. Keep your sentences, short, clear, and simple. A conversation may go like this:

You: I'm afraid the test results show that things are more serious than first thought.
Them: What do you mean more serious?
You: Some of the cells look abnormal.
Them: Do you mean that I have cancer?
You: Yes.

At any point, the patient may stop you, signalling that they don't want to hear more about it. Inexperienced practitioners sometimes feel that they 'ought' to tell the patient the full story but they must understand that many people would much rather not hear the words said aloud—this is their coping strategy and must be respected.

You: I'm afraid the test results show that things are more serious than first thought.
Them: Just tell me what we can do next.
You: OK.

Allow time for information to sink in

You should allow time for each piece of information to sink in, ensure that the patient understands all that has been said and repeat any important information.

Remember also that patients will not be able to remember the exact details of what you have said—you may need to reschedule at a later time to talk about treatment options or prognosis.

Honesty, above all else

Above all, you should be honest at all times. Never guess or lie.

The patient may break your pre-prepared flow of information requiring you to think on your feet. Sometimes you simply can't abide by the rules above. If asked a direct question, you must be honest and straightforward.

For example:

You: I'm afraid the test results show…

Them: Just tell me, have I got cancer?

You: Yes, I'm afraid you have.

Don't rush to the positive

When told of bad news, the patient needs a few moments to let the information sink in. After the 'yes' in the above examples, you should preferably wait in silence for the patient to speak next.

The patient may break down in tears—in which case they should be offered tissues and the support of relatives, if nearby.

If emotionally distressed, the patient will not be receptive to what you say next—you may want to give them some time alone with a relative or nurse before you continue to talk about prognosis or treatment options.

Above all, *you should not give false hope.* The moment after the bad news has been broken is uncomfortable and you must fight the instinctive move to the positive with 'there are things we can do', 'on the plus side…', 'the good news is…' or similar.

Ending

Summarize the information given, check their understanding, repeat any information as necessary, allow time for questions, and make arrangements for a follow-up appointment or a further opportunity to ask questions again.

Obviously, you shouldn't make promises that you can't keep. Don't offer to come back that afternoon if you're going to be in clinic!

'Do you understand everything that we've discussed?'

'Is there anything that you would like to ask me?'

'I'll be along to see you tomorrow morning. I'll be happy to come back in the meantime if you think of anything that you'd like to ask or if you need to talk. Just ask one of the nursing staff to give me a bleep.'

Questions about time

'How long have I got?' is one of the most common questions to be asked—and the hardest to answer.

- As always, don't guess and don't lie.
- It's often impossible to estimate. Giving a figure will almost always lead to you being wrong. If you don't know, it is perfectly acceptable to say so.
- Explain that it is impossible to judge and ask if there is any date in particular that they don't want to miss—perhaps they want to experience Christmas or a relative's birthday.
- Don't assume that they are asking out of fear, some people are surprisingly practical and want to put their affairs in order before their death.

Box 1.4 Fear-words

There are certain words which immediately generate fear, such as 'cancer' and 'leukaemia'. You should only use these if you are sure that the patient wants to know the full story.

Beware, however, of avoiding these words and causing confusion by not giving the whole story.

You should also be aware of certain words that people will instinctively assume mean something more serious. For example, to most people a 'shadow' on the lung means cancer. Don't then use the word when you are talking about consolidation due to pneumonia!

Body language: an introduction

Body language is rarely given the place it deserves in the teaching of communication skills. There are over 600 muscles in the human body; 90 in the face of which 30 act purely to express emotion. Changes in your position or expression—some obvious, others subtle—can heavily influence the message that you are communicating.

We've all met someone and thought 'I didn't like him' or 'she seemed trustworthy'. Often these impressions of people are not built on what is said but the manner in which people handle themselves. You subconsciously pick up cues from the other person's body. Being good at using body language means having awareness of how the other person may be viewing you and getting your subconscious actions and expressions under conscious control.

If done well, you can influence the other person's opinion of you, make them more receptive to your message, or add particular emphasis to certain words and phrases.

Touching

One of the most powerful forms of non-verbal communication and needs to be managed with care.

- ***Greeting:*** touch is part of greeting rituals in most cultures. It demonstrates that you are not holding a weapon and establishes intimacy.
- ***Shaking hands:*** there are many variations. The length of the shake and the strength of the grip impart a huge amount of information. For added intimacy and warmth, a double-handed grip can be used. For extra intimacy, one may touch the other's forearm or elbow.*
- ***Dominance:*** touch is a powerful display of dominance. Touching someone on the back or shoulder demonstrates that you are in charge—this can be countered by mirroring the action back.
- ***Sympathy:*** the lightest of touches can be very comforting and is appropriate in the medical situation where other touch may be misread as dominance or intimacy (you shouldn't hug a patient that you've only just met!). Display sympathy by a brief touch to the arm or hand.

Open body language

A cluster of movements concerned with seeming *open*. The most significant part of this is the act of opening—signalling a change in the way you are feeling. Openness demonstrates that you have nothing to hide and are receptive to the other person. Openness encourages openness.

This can be used to calm an angry situation or when asking about personal information.

▸ The key is to not have your arms or legs crossed in any way.

- ***Arms open:*** either at your side or held wide. Even better, hold your hands open and face your palms to the other person.
- ***Legs open:*** this does not mean legs wide but rather not crossed. You may hold them parallel. The feet often point to something of subconscious interest to you—point them at the patient!

Emphasis

You can amplify your spoken words with your body—usually without noticing it. Actions include nodding your head, pointing, or other hand gestures. A gesture may even involve your entire body.

Watch newsreaders—often only their heads are in view so they emphasize with nods and turns of their heads much more than one would during normal conversation.

- ***Synchrony:*** this is key. Time points of the finger, taps of the hand on the desk, or other actions with the words you wish to emphasize.
- ***Precision:*** signal that the words currently being spoken are worth paying attention to with delicate, precise movements. You could make an 'O' with your thumb and index finger or hold your hands such that each finger is touching its opposite counterpart—like a splayed prayer position.*

Eye level

This is a very powerful tool. In general, the person with their eye level higher is in control of the situation.

You can use this to your advantage. When asking someone personal questions or when you want them to open up, position yourself such that your eyes are below theirs—meaning they have to look down at you slightly. This makes them feel more in control and comfortable.

Likewise, anger often comes from a feeling of lack of control—put the angry person in charge by lowering your eye level—even if that means squatting next to them or sitting when they are standing.

Conversely, you may raise your eye level to take charge of a difficult situation, looking down on someone is intimidating. Stand over a

seated person to demonstrate that you are in charge.

Watch and learn

There is much that could be said about body language. You should watch others and yourselves and consider what messages are being portrayed by non-verbal communication.

Stay aware of your own movements and consider purposefully changing what would normally be subconscious actions to add to, or alter, the meaning of your speech.

Written communication

The medical notes serve a number of uses. The most important are:

- They are record of the patient's illness, treatments, and medical encounters for use by other medical practitioners in the future.
- They are the only record of your action—and the means by which you may be judged in case of future disputes.
- They are a record of events for the purposes of clinical audit.

How to write in the notes

Your entries in the notes should be tidy and legible.

- All entries should include:
  - Date.
  - Time.
  - Identity of the inscriber.
  - Signature.
  - Contact number (bleep/mobile phone).
- Use black ink only (blue often doesn't photocopy easily and can fade).
  - Previous fashion was for surgeons to use red ink for the operation notes and pharmacists to write in green—this practice is now fading.

What to write in the notes

- Everything that occurs should be recorded. If it isn't written down, it didn't happen!
- Remember especially to record discussions with relatives and the details of what the patient has been told of diagnoses.
- There are no specific rules as to how things should be written—there are a number of conventions which we introduce you to through the book. In general, entries should be easily understood by another.

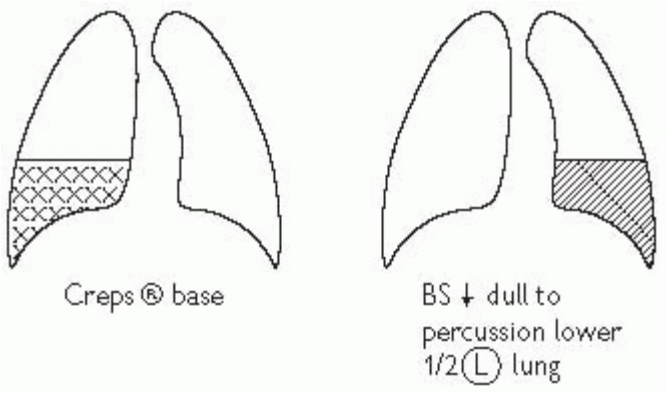
Box 1.5 ‘Standard’ examination drawings

The essential rule is that the record you make should be easily understood by another. If it is hard to describe where the cut on the patient's foot is, draw it!

There are a number of diagrams which, although not ‘official’, have become widely used and are accepted as ‘standard’.

Chest

This is usually represented as a stylized version of the lungs seen from the front. You can then add symbols indicating your clinical finding.



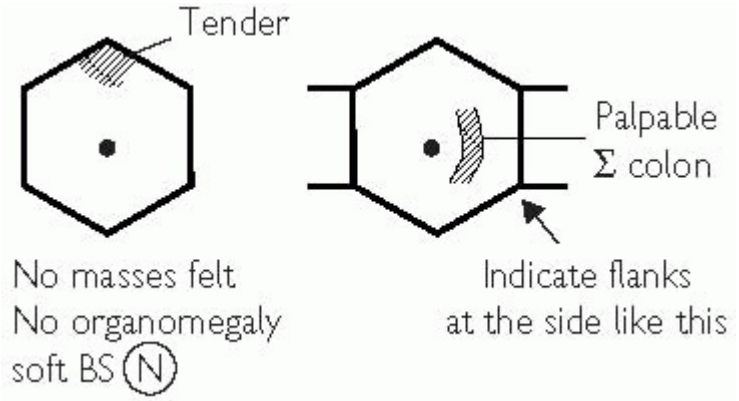
P

Heart sounds

These are often represented as a version of a phonogram—see p.185 for examples.

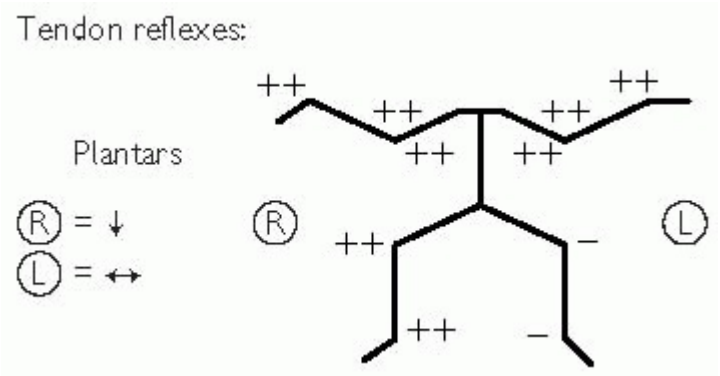
Abdomen

Usually drawn as a hexagon, although the anatomical stickler may add the xiphisternum and the genitalia.



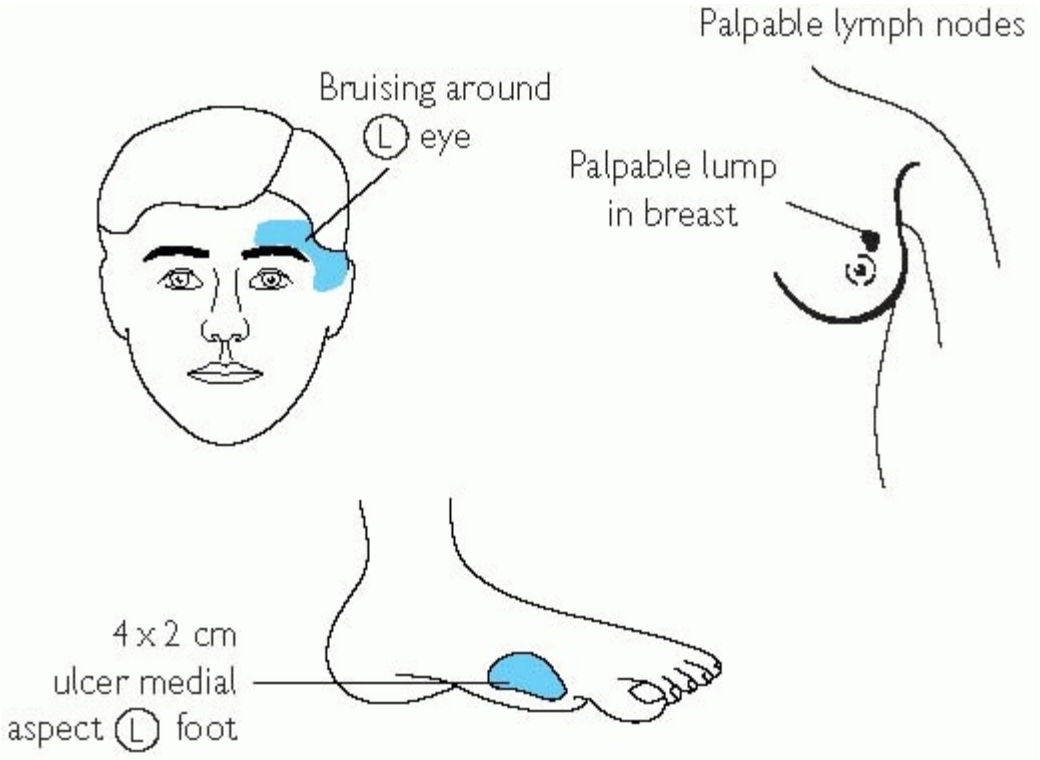
Peripheral pulses/tendon reflexes

These are often indicated on stick-men. Be sure to make clear which are the left and right sides of the person.



Other body parts

You should feel free to make drawings to illustrate your findings.



Law, ethics, and communication

No discussion of communication skills would be complete without mention of confidentiality, capacity, and consent. It is also worth knowing the four bioethical principles about which much has been written elsewhere.

Four bioethical principles

- ***Autonomy:*** a respect for the individual and their ability to make decisions regarding their own health.
- ***Beneficence:*** acting to the benefit of patients.
- ***Non-malificence:*** acting to prevent harm to the patient.
- ***Justice:*** 'fairness' to the patient and the wider community when considering the consequences of an action.

Confidentiality

Confidentiality is closely linked to the ethical principles described above. Maintaining a secret record of personal information shows respect for the individual's autonomy and their right to control their own information. There is also an element of beneficence where releasing the protected information may cause harm.

Breaking confidentiality

The rules surrounding the maintenance of confidentiality have been mentioned (see p.3). There are a number of circumstances where confidentiality can, or must, be broken. The exact advice varies slightly between different bodies. See the links opposite. In general, confidentiality may be broken in the following situations:

- With the consent of the individual concerned.
- If disclosure is in the patient's interest but consent cannot be gained.
- If required by law.
- When there is a statutory duty such as reporting of births, deaths, and abortions and in cases of certain communicable diseases.
- If it is overwhelmingly in the public interest.
- If it is necessary for national security or where prevention or detection of a crime may be prejudiced or delayed.
- In certain situations related to medical research.

Consent and capacity

There are 3 main components to valid consent. To be competent (or have capacity) to give consent, the patient:

- Must understand the information that has been given.
- Must believe that information.
- Must be able to retain and weigh-up the information to make a decision.

In addition, for consent to be valid, the patient must be free from any kind of duress.

▸ It should be noted that an assessment of capacity is valid for the specific decision in hand. It is not an all-or-nothing phenomenon—you cannot either have 'capacity' or not. The assessment regarding competence must be made for each new decision faced.

Young people and capacity

- All persons aged 18 and over are considered to be a competent adult unless there is evidence to the contrary.
- People aged between 16 and 18 are treated as adults (Family Law Reform Act 1969). However, the refusal of a treatment can be overridden by someone with parental responsibility or the courts.
- Children of 16 and younger are considered competent to give consent if they meet the three conditions mentioned previously. Their decisions can, however, be overridden by the courts or people with parental responsibility.

Gillick competence

In 1985, the well-known Gillick case was considered by the House of Lords and from this two principles (often known as the Fraser Guidelines) were established:

- A parent's right to consent to treatment on behalf of the child finishes when the child has sufficient understanding to give consent themselves (when they become 'Gillick competent').
- The decision as to whether the child is Gillick competent rests with the treating doctor.

Further reading

There are many other complex topics in this area and the law varies between countries and even between regions within the UK. There are very many sources of information on this topic. We suggest the following as a good start:

- The British Medical Association: www.bma.org.uk
- The General Medical Council: www.gmc-uk.org
- The Medical Defence Union: www.the-mdu.com
- The Medical Protection Society: www.medicalprotection.org
- The UK Department of Constitutional Affairs: www.dca.org.uk
- The UK Department of Health: www.dh.gov.uk

Editors: Thomas, James; Monaghan, Tanya
Title: *Oxford Handbook of Clinical Examination and Practical Skills, 1st Edition*

Chapter 2

The History

History-taking

The history is a patient's account of their illness together with other relevant information that you have gleaned from them. Like all things in medicine, there is a tried and tested standard sequence which you should stick to and is used by all practitioners.

It is good practice to make quick notes whilst talking to the patient that you can use to write a thorough history afterwards—don't document every word they say as this breaks your interaction!

By the end of the history taking, you should have a good idea as to a diagnosis or have several differential diagnoses in mind. The examination is your chance to confirm or refute these by gaining more information.

History-taking is not a passive process. You need to keep your wits about you and gently guide the patient into giving you relevant information using all the communication skills described in Chapter 1.

You should break the history down under the following headings and record it in the notes in this order—many people prefer to use the standard abbreviations (shown in Box 2.1) instead of writing out the heading in full...

Box 2.1 The standard history framework

- Presenting complaint (PC)
- History of presenting complaint (HPC)
- Systematic enquiry (SE)
- Past medical history (PMH)
- Allergies
- Drug history (DHx)
- Alcohol
- Smoking
- Family history (FHx)
- Social history (SHx)

The outline above is the authors' favoured method—slight variations exist.

▸ Many people will put ‘smoking’ and ‘alcohol’ as part of the ‘social history’. We feel that as these can have such an important impact on health they deserve their own spot and are more than simply ‘what the patient does in their spare time’.

It is good practice in medicine to watch what other practitioners do and adapt the parts that you feel are done well to your own style, making them part of your own routine.

Using this book

This book is divided into chapters by organ system. In each chapter, there are suggestions as to how to proceed depending on the nature of the presenting complaint and notes on what you should especially ask about under each of the above headings. These are not exhaustive and are intended as guidance to supplement a thorough history.

Box 2.2 Recording the history

Documentation is a vital part of all medical interactions.

The history should be recorded in the patient's notes according to the standard framework opposite using the rules and procedures described under ‘written communication’ in Chapter 1 (p.30). We direct readers there now.

Remember, if it isn't written down, it didn't happen!

Box 2.3 Collateral histories

There are many situations when the patient may be unable to give a history (e.g. they are unconscious, delirious, demented, dysphasic etc.). In these situations, you should make an effort to speak to all those who can help you fill in the gaps—not only

regarding what happened to bring the patient to your attention now, but also regarding their usual medication, functional state, living arrangements, and so on.

When taking a history from a source other than the patient, be sure to document clearly that this is the case and why the patient is unable to speak for themselves.

Useful sources of information include:

- Relatives/cohabitants.
- Close friends/room-mates.
- The GP or other members of the primary care team.
- The pharmacist.
- The warden (if in sheltered accommodation).
- The staff at the nursing or residential home.
- Anyone who witnessed the event.

Presenting complaint (PC)

- This is the patient's chief symptom(s) *in their own words* and should be no more than a single sentence.
- If the patient has several symptoms, present them as a list which you can expand on later in the history.
- Ask the patient an open question such as 'What's the problem?' or 'What made you come to the doctor?'. Each practitioner will have their own style. You should choose a phrase that suits you and your manner (one of the authors favours 'tell me the story' after a brief introduction).

The question 'what brought you here?' usually brings the response 'an ambulance' or 'the taxi'—each patient under the impression that they are the first to crack this show-stopper of a joke. This is, therefore, best avoided.

▸ Remember, this is the problem *in the patient's words.* 'Haemoptysis' is rarely a presenting complaint but 'coughing up blood' may well be!

History of the presenting complaint (HPC)

Here, you ask about and document the details of the presenting complaint. By the end of this, you should have a clear idea about the nature of the problem along with exactly how and when it started, how the problem has progressed over time, and what impact it has had on the patient in terms of their general physical health, psychology, social, and working lives.

This is best tackled in 2 phases:

First, ask an open question (as above) and allow the patient to talk through what has happened for about 2 minutes. Don't interrupt! Encourage the patient with non-verbal responses and make discreet notes. This also allows you to make an initial assessment of the patient in terms of education level, personality, and anxiety. Using this information, you can adjust your responses and interaction. It should also become clear to you exactly what symptom the patient is most concerned about.

In the ***second*** phase, you should revisit the whole story asking more detailed questions. It may be useful to say 'I'd just like to go through the story again, clarifying some details'. This is your chance to verify *time-lines* and the relationship of one symptom to another. You should also be careful to clarify pseudo-medical terms (exactly what does the patient mean by 'vertigo', 'flu' or 'rheumatism'?). Remember, this should feel like a conversation, not an interrogation!

▸ The standard features that should be determined for any symptom are shown opposite, along with the additional features regarding 'pain'.

▸ See the rest of this book for guidance on tackling other presenting complaints.

At the end of the history of presenting complaint, you should have established a *problem list* You should run through these with the patient, summarising what you have been told and ask them if you have the information *correct* and if there are is *anything further* that they would like to share with you.

Box 2.4 For each symptom, determine:

- The exact nature of the symptom.
- The onset:
  - The date it began.
  - How it began (e.g. suddenly, gradually—over how long?)
  - If longstanding, why is the patient seeking help now?
- Periodicity and frequency:
  - Is the symptom constant or intermittent?

    - How long does it last each time?
    - What is the exact manner in which it comes and goes?
- Change over time:
    - Is it improving or deteriorating?
- Exacerbating factors:
    - What makes the symptom worse?
- Relieving factors:
    - What makes the symptom better?
- Associated symptoms.

Box 2.5 For pain, determine:

- Site (where is the pain worst—ask the patient to point to the site with *one finger*).
- Radiation (does the pain move anywhere else?).
- Character (i.e. 'dull', 'aching', 'stabbing', 'burning' etc.).
- Severity (scored out of 10, with '10' as the worst pain imaginable).
- Mode and rate of onset (how did it come on—over how long?).
- Duration.
- Frequency.
- Exacerbating factors.
- Relieving factors.
- Associated symptoms (e.g. nausea, dyspepsia, shortness of breath).

Box 2.6 Long-standing problems

If the symptom is long-standing, ask why the patient is seeking help now. Has anything changed? It is often useful to ask when the patient was *last well*. This helps focus their minds on the start of the problem which may seem distant and less important to them.

Systematic enquiry (SE)

After talking about the presenting complaint, you should perform a brief screen of the other bodily systems.

This often proves to be more important than you expect, finding symptoms that the patient had forgotten about or identifying secondary, unrelated, problems that can be addressed.

The questions asked will depend on the discussion that has gone before. If you have discussed chest pain in the history of presenting complaint, there is no need to ask about it again!

Ask the patient if they have any of the following symptoms …

General symptoms

Weight change (loss or gain), change in appetite (loss or gain), fever, lethargy, malaise.

Respiratory symptoms

Cough, sputum, haemoptysis, shortness of breath, wheeze, chest pain.

Cardiovascular symptoms

Shortness of breath on exertion, paroxysmal nocturnal dyspnoea, chest pain, palpitations, ankle swelling, orthopnoea, claudication.

Gastrointestinal symptoms

Indigestion, abdominal pain, nausea, vomiting, a change in bowel habit, constipation, diarrhoea, PR blood-loss, dysphagia.

Genito-urinary symptoms

Urinary frequency, polyuria, dysuria, haematuria, nocturia, menstrual problems, impotence.

Neurological symptoms

Headaches, dizziness, tingling, weakness, tremor, fits, faints, ‘funny turns’, black-outs, sphincter disturbance.

Locomotor symptoms

Aches, pains, stiffness, swelling.

Skin symptoms

Lumps, bumps, ulcers, rashes, itch.

Past medical history (PMH)

Some aspects of the patient's past illnesses or diagnoses may have already been covered. Here, you should obtain detailed information about past illness and surgical procedures.

Ask if they're ‘under the doctor for anything else’ or have ever been to hospital before. Ensure you get dates and location for each event. There are some conditions which you should specifically ask patients about and these are shown below.

For each condition, ask:

- When was it diagnosed?
- How was it diagnosed?
- How has it been treated?

For operations, ask about any previous anaesthetic problems.

 Ask also about immunizations and company/insurance medicals.

Box 2.7 Past medical history—ask specifically about:

- Diabetes.
- Rheumatic fever.
- Jaundice.
- Hypercholesterolaemia.
- Hypertension.
- Angina.
- Myocardial infarction.
- Stroke or TIA.
- Asthma.
- TB.
- Epilepsy.
- Anaesthetic problems.
- Blood transfusions.

Box 2.8 Don't take anything for granted!

For each condition that the patient reports having, ask exactly how it was diagnosed (where? by whom?) and how it has been treated since.

For example, if the patient reports ‘asthma’, ask who made the diagnosis, when the diagnosis was made, if they have ever had lung function tests, if they have ever seen a chest physician at a hospital, if they are taking any inhalers? Occasionally, patients will give a long-standing symptom a medical name which can be very confusing. In this example, the patient's ‘asthma’ could be how they refer to their wheeze which is, in fact, due to congestive cardiac failure.

Allergies

This should be documented separately from the ‘drug history’ (below) due to its importance.

Ask if the patient has any allergies or ‘is allergic to anything’ if they are unfamiliar with the term. Be sure to probe carefully as people will often tell you about their hay-fever and forget about the rash they had when they took penicillin. Ask specifically if they have had any ‘reactions’ to drugs or medication.

▸ If an allergy is reported, you should obtain the exact nature of the event and decide if the patient is describing a true allergy, an intolerance, or simply an unpleasant side-effect.

Drug history (DHx)

Here, you should list all the medication the patient is taking, including the dose and frequency of each prescription. If the patient is unsure, you should confirm with the GP or pharmacy. You should make a special note of any drugs that have been started or stopped recently.

You should also ask about compliance—does the patient know what dose they take? Do they ever miss doses? If they are not taking the medication—what's the reason? Do they have any compliance aids such as a pre-packaged weekly supply?

The patient may not consider some medications to be ‘drugs’ so specific questioning is required. Don't forget to ask about:

- Eye-drops.
- Inhalers.
- Sleeping pills.
- Oral contraception.
- Over the counter drugs (bought at a pharmacy), vitamin supplements.
- Herbal remedies.
- ‘Illicit’ or ‘recreational’ drug-use.

Alcohol

You should attempt to quantify, as accurately as you can, the amount of alcohol consumed per week—and also establish if the consumption is spread evenly over the week or concentrated into a smaller period.

In the UK, alcohol is quantified in ‘units’. One unit is 10ml of pure alcohol. The unit-content of some common drinks is shown opposite.

In many European countries, and the US, alcohol is quantified as ‘standard drinks’. In the US, a ‘standard drink’ contains 0.54 ounces of alcohol which is about 1.5 UK ‘units’.

Box 2.9 Recommended weekly alcohol consumption

At the time of writing, the UK government recommends a maximum of 21 units/week for men and 14 units/week for women.

Box 2.10 Units of alcohol in common drinks

- 1 pint ordinary strength lager = 2 units
- 1 pint strong lager = 3 units
- 1 pint bitter = 2 units
- 1 pint ordinary strength cider = 2 units
- 1 175ml glass of red or white wine ≈ 2 units
- 1 pub measure of spirits = 1 unit
- 1 alcopop = 1.5 units

Source: Department of Health, UK (www.dh.gov.uk)

Smoking

- Attempt to quantify the habit in ‘pack-years’. 1 pack-year is 20 cigarettes per day for one year. (e.g. 40/day for 1 year = 2 pack-years; 10/day for 2 years = 1 pack-year).
- Ask about previous smoking as many will call themselves non-smokers if they gave up yesterday or even on their way to the hospital or clinic!
- Remember to ask about passive smoking.

Be aware of cultural issues—smoking is forbidden for Sikhs, for example, and they may take offence at the suggestion!

Beware of appearing judgemental.

Box 2.11 Haggling and the art of quantification

Smoking and alcohol histories are notoriously unreliable—alcohol especially so. The patient may be trying to please you or feel embarrassed about openly admitting their true consumption.

Gaining an accurate account of consumption can sometimes feel like haggling. There are two steps in this process.

Firstly, appear non-judgmental and resist acting surprised *in any way*, even in the face of liquor- or tobacco-consumption that you may consider excessive and unwise.

Secondly, if the patient remains reticent ('I smoke a few'), suggest a number—but start very high ('shall we say 60 a day?') and the patient will usually give you a number nearer the true amount ('oh no, more like 20'). If you were to start low, the same patient may only admit to half that.

Family history (FHx)

The FHx details:

- The make up of the current family, including the age and gender of parents, siblings, children, and extended family as relevant.
- The health of the family.

You should ask about any diagnosed conditions in other living family members. You should also document the age of death and cause of death for all deceased first degree relatives and other family members if you feel it is appropriate.

It may help to draw a family-tree as shown opposite. These are particularly useful in paediatric assessments.

Social history (SHx)

This is your chance to document the details of the patient's personal life which are relevant to the working diagnosis, the patient's general well-being and recovery/convalescence. It will help to understand the impact of the illness on the patient's functional status.

This is a vital part of the history but sadly, perhaps because it comes at the end, it is often given only brief attention. The disease, and indeed the patient, do not exist in a vacuum but are part of a community which they interact with and contribute to. Without these details, it is impossible to take an holistic approach to the patient's wellbeing.

Establish:

- Marital status.
- Sexual orientation.
- Occupation (or previous occupations if retired).
  - You should establish the *exact* nature of the job if it is unclear—does it involve sitting at a desk, carrying heavy loads, travelling?
- Other people who live at the same address.
- The type of accommodation (e.g. house, flat—and on what floor).
- Does the patient own their accommodation or rent it?
- Are there any stairs? How many?
- Does the patient have any aids or adaptations in their house? (e.g. rails near the bath, stairlift etc).
- Does the patient use any walking aids (e.g. stick, frame scooter)?
- Does the patient receive any help day-to-day?
  - Who from? (e.g. family, friends, social services.)
  - Who does the laundry, cleaning, cooking, and shopping?
- Does the patient have relatives living nearby?
- What hobbies does the patient have?
- Does the patient own any pets?
- Has the patient been abroad recently or spent any time abroad in the past?
- Does the patient drive?

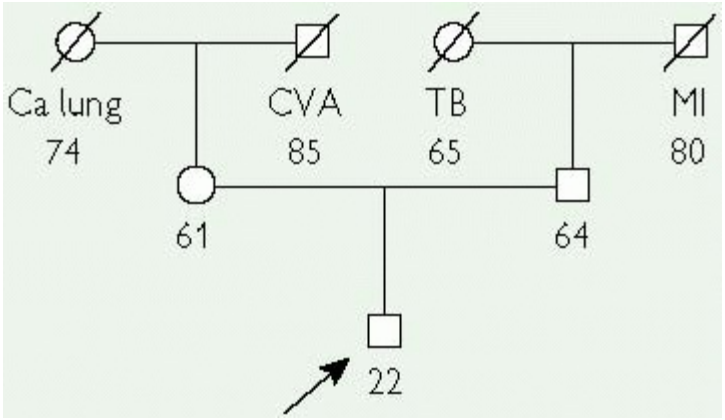
Box 2.12 Family trees

Conventionally, males are represented by a square (□) and females by a circle (○). The patient that you are talking to is called the *propositus* and is indicated by a small arrow (↗).

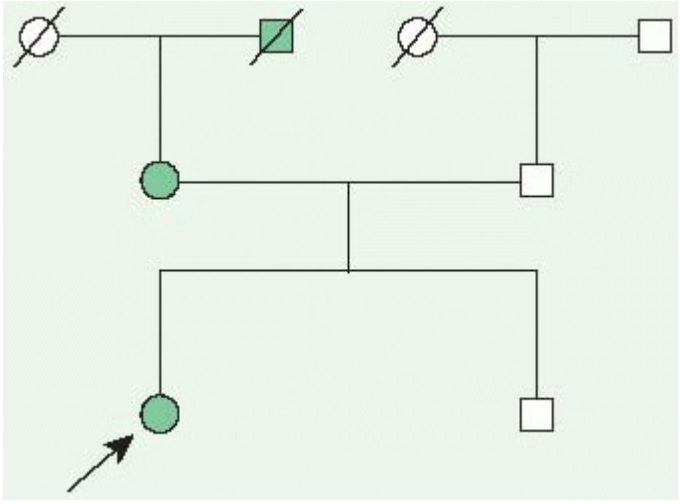
Horizontal lines represent marriages or relationships resulting in a child. Vertical lines descend from these, connecting to a horizontal line from which the children 'hang'. You can add ages and causes of death.

Family members who have died are represented by a diagonal line through their circle or square (⌀, ⧄) and those with the condition of interest are represented by shaded shapes (●, ■).

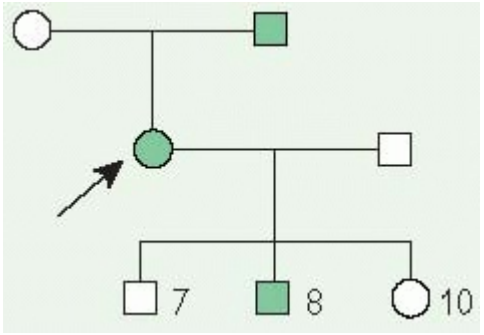
Example 1 Our patient is an only child and has no children, his parents are alive but all his grandparents have died of different causes.



Example 2 Our patient suffers from colon cancer and has no children. She has a brother who is well. Her parents are both alive and her mother also has colon cancer. Of her grandparents, only her paternal grandfather is alive. Her maternal grandfather died of colon cancer.



Example 3 Our patient has epilepsy, as does her father. She has 3 children, 2 boys and a girl. One of the boys also has epilepsy.



The elderly patient

Obtaining a history from older people might be regarded as no greater task than from any patient—however cognitive decline, deafness, and acute illness can make this difficult. Getting to grips with taking a good history from older people is a skill you will find useful in all other situations. Whilst the history is key for making diagnoses, it is an opportunity for so much more—your first interaction with an (older) patient sets important first impressions. A skilful history not only reaps diagnostic rewards, but marks you as a competent doctor who can gain trust, reassure, and communicate well with patients in any challenging situation.

Key points

- ***'Learn to listen':*** it can be tempting to ask lots of questions to obtain every fact in the history, particularly if you are rushed. Doing this will not only frustrate and offend your patient (because you clearly don't listen), but will also risk you missing important facts. Instead, learn to stay quiet—and listen in detail to the history of the presenting complaint which may only be 3-4 minutes, but gives your patient a chance to be heard; seemingly irrelevant detail is often useful when patients have the chance to put it in context. It often saves you time, as other key information may emerge straight away, and you can better focus the history.
- ***Problem lists:*** patients with chronic illness or multiple diagnoses may have more than one strand to their acute presentation. Consider breaking the history of the presenting complaint down into a problem list e.g. (1) worsening heart failure; (2) continence problems; (3) diarrhoea; (4) falls. This can often reveal key interactions between diagnoses you might not have thought about.
- ***Drug history:*** remember polypharmacy and that patients may not remember all the treatments they take. Be aware that more drugs mean more side effects and less concordance—so ask which are taken and why—(older) people are often quite honest about why they omit tablets. Eye drops, sleeping pills, and laxatives are often regarded as non-medicines by patients, so be thorough and ask separately—and avoid precipitating delirium due to acute withdrawal of benzodiazepines.
- ***Functional history:*** a comprehensive functional history is a cornerstone of your history taking in older people—we make no apologies for reminding you about this throughout this book. Diseases may not cured or modified, but their key component—the effects on patients and their lives might be easily transformed through manipulation of activities of daily living. Remember to ask about formal and informal support for the patient at home—have things resulted in a crisis for the patient because a caring neighbour or friend is unwell? Be polite—and ask tactfully about benefits, including Attendance Allowance—many patients do not realise they might be eligible, so couch your questions with an explanation that advice might be available too.

- ***Social history:*** is exactly that, and should complement the functional history. Occupation (other than 'retired') can be of value when faced with a new diagnosis of pulmonary fibrosis or bladder cancer and may give your patient a chance to sketch out more about their lives. Enquire about family—don't assume that a relative may be able to undertake more help, as they may live far away; the patient may still have a spouse—but be separated. Chat with patients about their daily lives—understanding interests and pursuits can help distract an unwell patient, give hope for the future, and act as a spur for recovery and meaningful rehabilitation.

Box 2.13 A note on narratives

- Akin to 'learning to listen' is a recognition that many patients might not deliver their histories in a style that fits the traditional pattern described in this chapter. Pushing (older) patients through histories is not to be recommended, as indicated above. Elders will often discuss events and preferences with a constituted story, and it is important to recognize the value of this. Narrative analysis at its most simple—i.e. your ability to listen and interpret—is a vital skill for all clinicians. Listening to stories allows you to understand patients' preferences, hopes and fears.
- Remember also that older patients often have different views about what they want from their doctors. Their 'agendas' may differ hugely from what you think treatment plans should be, but they may not make their views known through fear of offending you. If you are unsure, always ask—learning to involve your patients in key decisions about their care will make you a better clinician.

We thank Dr Richard Fuller for providing this page.

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Chapter 3

General Examination

Approaching the physical examination

General conduct

Doctors and medical professionals are in a position of trust. It is generally assumed that you will act with professionalism, integrity, honesty, and with a respect for the dignity and privacy of your patients.

In no part of the patient encounter is this more evident than at the physical examination. People who you may have only just met will take off their clothes and allow you to look at and touch their bodies—something that would be completely unacceptable to many people in any other situation. They will, of course, be more comfortable with this if you have established an appropriate rapport during history-taking. However, the communication does not stop at the end of the history. The manner in which you conduct yourself during the examination can make the difference between an effective examination and a formal complaint. You should also be alert to cultural and religious differences when it comes to disrobing in front of others.

This is not to say that you should shy away from examining for fear of acting inappropriately and causing offence. In particular, you should not avoid examining members of the opposite sex, especially their intimate body parts, as there should be no sexual undertones in the relationship whatsoever.

Projected confidence will be picked up by the patient, making them more at ease and constant verbal and non-verbal communication should ensure that no misunderstandings occur. You should, of course, ensure that you have a chaperone present—another student, doctor, nurse, or other healthcare professional—whenever you perform any intimate examination and that chaperone should ideally be the same gender as the patient.

The format of the examination

The examination techniques in medicine may seem forced and unnaturally formulaic at first, but these routines ensure that no part of the examination is missed.

The right approach

One important rule is that you should always stand at the patient's right hand side. This gives them a feeling of control over the situation (most people are right handed). All the standard examination techniques are formulated with this orientation in mind.

The systems examinations

The physical examination can be broken into body systems—and this is the format of this book.

You often need to examine several systems at a time and it is then that you must combine your learnt techniques. For example, you may wish to examine the patient's thorax with a view to the cardiovascular and respiratory systems, listening for both heart and breath sounds during your ‘auscultation’ stage rather than completing the heart exam and then returning to examine the chest.

The examination framework

Each system examination is divided into the following categories:

- Inspection (looking).
- Palpation (feeling).
- Percussion (tapping).
- Auscultation (listening).

In addition, there may be special tests and other added categories—but you will meet these as you go through the book.

Using this book

- Each chapter in this book is based around one body system and describes the standard examination format for that system only.
- At the beginning of each encounter, you should consider the topics covered in this chapter before moving to the more specific

examination.

- As mentioned above, you should not consider these examination routines to be entirely separate entities. If examining several systems at once, you should combine these frameworks to create a single fluid routine.

First impressions

Diagnosis at first sight

From the first moment you set eyes on the patient, you should be forming impressions of their general state of health. It takes experience and practice to pick up all the possible clues but much can be gained by combining common sense with medical knowledge. Ask yourself:

- Is the patient comfortable or distressed?
- Is the patient well or ill?
- Is there a recognizable syndrome or facies?
- Is the patient well nourished and hydrated?

Many of these features will be noted subconsciously—but you must make yourself consciously aware of them.

Bed-side clues

In a hospital setting, there may be additional clues as to the patient's state of health in the objects around them. In other circumstances, look at objects that they are carrying or are visible in their pockets.

Examples include oxygen tubing, inhalers, GTN spray, insulin injections, glucose meter, or cigarettes.

Vital signs

It may also be appropriate to assess vital signs at an early stage. These usually include:

- Temperature.
- Blood pressure.
- Pulse.
- Oxygen saturation.
- Respiratory rate.
- Blood glucose.

Conscious level

If necessary, a rapid and initial assessment of a patient's conscious level can be made using the AVPU scale (📖 p.361) or the GCS (📖 p.361).

Set-up

Before commencing a formal examination, introduce yourself, explain what you would like to do and obtain verbal consent.

- Ensure that the patient has adequate privacy to undress.
- Make sure that you will not be disturbed.
- Check that the examination couch or bed is draped/covered by a clean sheet or disposable towelling.
- If the patient is accompanied, ask them if they would like their companion/s to stay in the room.
- Check that any equipment you will require is available (torch, cotton wool, tendon hammer, stethoscope etc.).
- When ready, the patient should ideally be positioned supine with the head and shoulders raised to ~45°.

Colour

The colour of the patient, or parts of the patient, can give clues to their general state of health and to particular diagnoses. Look especially for evidence of pallor, central and peripheral cyanosis, jaundice, and abnormal skin pigmentation.

Pallor (paleness)

Facial pallor is often a sign of severe anaemia and is especially noticeable on inspecting the palpebral conjunctiva, nail beds and palmar skin creases.

Ask the patient to look upward and gently draw down their lower eyelid with your thumb—the conjunctiva should be red/pink.

It is, however, an unreliable sign in shocked patients and those with vascular disease since peripheral vasoconstriction or poor blood flow causes skin and conjunctival pallor, even in the absence of blood loss.

Cyanosis

See also  p.208. Cyanosis refers to a bluish discolouration of the skin and mucous membranes and is due to the presence of at least 2.5g/dL of deoxygenated haemoglobin in the blood.

In *central cyanosis*, the tongue appears blue due to an abnormal amount of deoxygenated blood in the arteries. This may develop in any lung disease in which there is a ventilation/perfusion mismatch such as chronic obstructive pulmonary disease ± cor pulmonale and massive pulmonary embolus. It will also occur in right to left cardiac shunts. Finally, polycythaemia and haemoglobinopathies (such as methaemoglobinaemia and sulphaemoglobinaemia) may give the appearance of cyanosis due to abnormal oxygen carriage.

Peripheral cyanosis is bluish discolouration at the extremities (fingers, toes) only. It is usually due to a ↓ in blood supply or a slowing of the peripheral circulation. The latter commonly arises through exposure to cold, reduced cardiac output or peripheral vascular disease.

Note that one cannot have central cyanosis without also demonstrating peripheral cyanosis. Peripheral cyanosis, however, can occur alone.

Jaundice

Jaundice (icterus) refers to a yellow pigmentation of those tissues in the body which contain elastin (skin, sclerae, and mucosa) and occurs due to an ↑ in plasma bilirubin (visible at >35μmol/L).

It is best appreciated in fair-skinned individuals in natural daylight. Expose the sclera by gently holding down the lower lid and asking the patient to look upwards.

▸ Jaundice should not be confused with carotenaemia, which also causes a yellow discolouration of the skin, but the sclerae remain white.

Other abnormalities of colouration

You will meet other distinctive colour patterns through this book, a list here would be lengthy and probably unnecessary.

These include the classic slate-grey appearance of haemachromatosis, the silver-grey colouration in argyra (silver poisoning), the ↑ skin-fold pigmentation seen in Addison's disease, and the non-pigmented patches of vitiligo ( p.93).

Temperature

- Record the patient's temperature using either a mercury or electronic thermometer. The recording will depend on the site of measurement.
- Normal oral temperature is usually considered to be 37°C, whereas rectal temperature is 0.5°C higher and axillary temperature is 0.5°C lower.
- There is also a diurnal variation in body temperature, with peak temperatures occurring between 6pm-10pm, lowest between 2am-4am.

High temperature

- The febrile pattern of most diseases also follows this diurnal variation. Sequential recording of temperature may show a variety of patterns which can be helpful in the diagnosis of disease.
- For example, *persistent pyrexia* may be a sign of malignant hyperthermia, a drug fever (e.g. halothane, suxathonium), typhus, or typhoid fever. An *intermittent pyrexia* can be suggestive of lymphomas and pyogenic infections such as milliary TB. A *relapsing high temperature* or *Pel-Ebstein* fever, occasionally occurs in patient with Hodgkin's disease and is characterized by 4-5 days of persistent fever which then returns to baseline before rising again.
- Also note any rigors (uncontrollable shaking) which may accompany high fever and are often characteristic of biliary sepsis or pyelonephritis.

Low temperature

- Hypothermia is a core (rectal) temperature of <35°C and occurs usually from cold exposure (e.g. near-drowning) or secondary to an impaired level of consciousness (e.g. following excess alcohol or drug overdose) or in the elderly (e.g. myxoedema).
- Patients may be pale with cold, waxy skin and stiff muscles, consciousness is often reduced.
- Patients typically lose consciousness at temperatures <27°C.

Hydration

When assessing hydration status, you may already have obtained clues from the history. For example, a patient may have been admitted with poor fluid intake and may feel thirsty. Sepsis, bleeding, or bowel obstruction and vomiting can also cause a person to become dehydrated.

Examination

- Begin with looking around the patient for any obvious clues including fluid restriction signs, catheter bag or nutritional supplements.
- Inspect the face for sunken orbits (a sign of moderate-severe dehydration).
- ***Mucous membranes:*** Inspect the tongue and mucous membranes for moisture.
  - Dehydration will cause these surfaces to appear dry.
- ***Skin turgor:*** assess by gently pinching a fold of skin on the forearm, holding for a few moments, and letting go.
  - With normal hydration, the skin will promptly return to its original position, whereas in dehydration, skin turgor is reduced and the skin takes longer to return to its original state.
  -  This sign is unreliable in elderly patients whose skin may have lost its normal elasticity.
- ***Capillary refill:*** test by raising the patient's thumb to the level of the heart, pressing hard on the pulp for 5 seconds and then releasing. Measure the time taken for the normal pink colour to return.
  - Normal capillary refill time should be <2 seconds; a prolongation is indicative of a poor blood supply to the peripheries.
- ***Pulse rate:*** a compensatory tachycardia may occur in dehydration or in fluid overload (📖 p.170).
- ***Blood pressure:*** check lying and standing blood pressure readings (📖 p.574) and look for a low blood pressure on standing which may suggest dehydration (along with many other diagnoses).
- **JVP***:* (see 📖 p.174). Assess the height of the JVP which is one of the most sensitive ways of judging intravascular volume.
  - The JVP is low in dehydration, but raised in fluid overload (e.g. pulmonary oedema). The latter commonly also causes fine basal inspiratory crepitations (📖 p.216).
- ***Oedema:*** another useful sign of fluid overload (think right heart failure, constrictive pericarditis, hypoalbuminaemia). Remember to test for both ankle and sacral oedema (📖 p.186).

Oedema

Oedema refers to fluid accumulation in the subcutaneous tissues and implies an imbalance of the Starling forces (↑ intravascular pressure or reduced intravascular oncotic pressure) causing fluid to seep into the interstitial space.

Oedema will occur in hypoproteinaemic states (especially nephrotic syndrome, malnutrition and malabsorption) and severe cardiac and renal failure.

Examination

In ambulant patients, palpate the distal shaft of the tibia for oedema by gently compressing the area for up to 10 seconds with the thumb. If the oedema is pitting, the skin will show an indention where pressure was applied which refills slowly.

▸ If oedema is present, note its upper level. Oedema may also involve the anterior abdominal wall and external genitalia.

When lying down, fluid moves to the new dependent area causing a sacral pad. This can be checked for by asking the patient to sit forwards, exposing the lower back and sacral region, and again applying gentle pressure with your finger-tips.

Box 3.1 Some causes of leg swelling

Local causes

- Cellulitis (usually unilateral).
- Ruptured baker's cyst (usually unilateral).
- Occlusion of a large vein—i.e. thrombophlebitis, DVT, extrinsic venous compression.
- Chronic venous insufficiency—pigmentation induration, inflammation, lipodermatosclerosis.
- Lipomatosis.
- Gastrocnemius rupture—swelling and bruising around the ankle joint and foot.

Systemic causes

- Congestive cardiac failure.
- Hypoproteinaemia (nephrotic syndrome, liver cirrhosis, protein-losing enteropathy, kwashiorkor).
- Hypothyroidism.
- Hyperthyroidism.
- Drugs (e.g. corticosteroids, NSAIDs, vasodilators).

Lymphoedema

This is non-pitting oedema associated with thickened and indurated skin. It can be idiopathic or secondary to proximal lymphatic obstruction such as post surgery, metastatic cancer, or chronic infection.

Nutritional status

The nutritional status of the patient may be an important marker of disease and is often overlooked in physical examination.

The following are simple clinical measures with can easily be undertaken to assess a patient's overall nutritional status.

General physical appearance

- Note the patient's overall body habitus; are they fat or thin? Do they appear to have recently lost or gained weight?
- Weight loss can lead to muscle wasting seen as skeletal prominence, especially cheek bones and heads of humerus and major joints, rib cage, and the bony landmarks of the pelvis.

Body weight and height

All patients should be weighed using accurate scales and have their height recorded (ideally using a stadiometer).

Body mass index

The body mass index (BMI) is a useful estimate of body fatness.

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

The world health organization has classified BMI as follows:

- 19-25 = normal.
- 25-30 = overweight.
- 30-40 = obese.
- >40 = extreme or ‘morbid’ obesity.

Regional fat distribution

A central distribution of body fat (waist:hip circumference ratio of >1.0 in men and >0.9 in women) is associated with a higher risk of

morbidity and mortality.

Skin fold thickness

Skin fold thickness is another useful method of assessing muscle and fat status and is usually measured at the triceps halfway between the olecranon and acromial processes. This is measured using specialist calipers.

The examiner should pinch a fold of skin and subcutaneous tissue between thumb and first finger and then apply the calipers to the skin fold. Three measurements are normally taken and the average calculated (normal values are 20mm in men and 30mm in women).

Mid-arm circumference

An additional method for estimating body fatness at the bedside is to measure mid-arm muscle circumference.

As with skin fold thickness, use the midpoint between the tip of the olecranon and acromial processes as your standard measurement point. With the arm in a flexed right-angle position, take 3 tape measurements at this point before calculating the average. Standard age/sex charts are available.

Box 3.2 Some conditions associated with malnutrition

- Any very ill patient.
- Malignancy.
- Metabolic disease (e.g. renal failure).
- Gastrointestinal disease (especially small bowel).
- Sepsis.
- Trauma.
- Post-surgery.
- Psychosocial problems (e.g. depression, anorexia nervosa, social isolation).
- Dementia.

Box 3.3 Some conditions associated with obesity

- Simple obesity ('biopsychosocial').
- Genetic e.g. Prader-Willi, Lawrence-Moon-Biedl syndrome.
- Endocrine (e.g. Cushing's syndrome, hypothyroidism).
- Drug-induced (e.g. corticosteroids).
- Hypothalamic damage due to tumour or trauma.

Lymph nodes

An examination of the lymph nodes forms part of the routine for most body systems. As there is no need to percuss or auscultate, examination involves inspection followed by palpation.

It should be remembered that there are a great many lymph nodes that are not accessible to the examining hand—for example, along the aorta, in the intestinal mesentery, and so on. There are several groups of lymph nodes that are accessible for the purposes of physical examination.

In the head and neck, these are located along the anterior and posterior aspects of the neck and on the underside of the jaw (see Fig. 3.1). In the upper limb and trunk, lymph nodes are located in the epitrochlear and axillary regions and in the lower limbs nodes can be examined in the inguinal and popliteal regions.

▸ Remember that the liver and spleen are often enlarged in the presence of generalized lymphadenopathy and these should be examined as on p.252 and p.254 respectively.

Inspection

Large nodes are often clearly visible on inspection, particularly if the enlargement is asymmetrical. If nodes are infected, the overlying skin may be red and inflamed.

Palpation

Lymph nodes should be palpated using the most sensitive part of your hands—the fingertips.

- ***Head and neck:*** the nodes should be palpated with the patient in an upright position and the examiner standing behind—similar to the examination of the thyroid gland (📖 p.116).
- ***Axillae:*** To examine the nodes at the *right* axilla:
  - The patient should be sitting comfortably and you should stand at their right-hand side.
  - Support their right arm abducted to 90° with your right hand.
  - Examine the axilla with your left hand.
  - To examine the nodes at the *left* axilla, perform the opposite manoeuvre to the above.
- ***Inguinal:*** with the patient lying supine, palpate their inguinal region along the inguinal ligament—the same position as feeling for a hernia (📖 p.266) or the femoral pulse (📖 p.170).
  - There are 2 chains of superficial inguinal lymph nodes—a horizontal chain which runs just below the inguinal ligament and a vertical chain which runs along the saphenous vein.
- ***Epitrochlear nodes:*** place the palm of the right hand under the patient's slightly flexed right elbow and feel with your fingers in the groove above and posterior to the medial epicondyle of the humerus.
- ***Popliteal:*** best examined by passively flexing the knee and exploring the fossa with the fingers of both hands—much like feeling for the popliteal pulse (📖 p.170).

Findings

Similar to the considerations to make when examining a lump (📖 p.98), during palpation of lymph nodes, the following features should be assessed:

- ***Site:*** important diseases such as both acute and chronic infections and metastatic carcinoma will cause localized lymphadenopathy depending on the site of primary pathology. It is often helpful to draw a diagram detailing exactly where the enlarged node is.
- ***Number:*** how many nodes are enlarged? Make a diagram as above and detail the palpable nodes clearly and carefully.
- ***Size:*** normal nodes are not palpable. Palpable nodes, therefore, are enlarged. You should measure their length and width.
- ***Consistency:*** malignant lymph nodes feel unusually firm or hard and irregular. Enlarged nodes secondary to infection may feel 'rubbery'.
- ***Tenderness:*** painful, tender nodes usually imply infection.
- ***Fixation:*** nodes that are fixed to surrounding tissue are highly suspicious of malignancy. Matted glands may occur in tuberculous lymphadenopathy.
- ***Overlying skin:*** inflamed nodes may cause redness and swelling in the overlying skin. Spread of a metastatic carcinoma into the surrounding tissue may cause oedema and surface texture changes.

Box 3.4 Some causes of generalized lymphadenopathy

- Haematological malignancies (e.g. lymphoma, acute, and chronic lymphatic leukaemia).
- Infections:
  - Viral (e.g. HIV, infectious mononucleosis, CMV).
  - Bacterial (e.g. tuberculosis, syphilis, brucellosis).
- Infiltrative diseases (e.g. sarcoidosis, amyloidosis).
- Autoimmune diseases (e.g. systemic lupus erythematosus, rheumatoid arthritis).
- Drugs (e.g. phenytoin causes a 'pseudolymphoma').

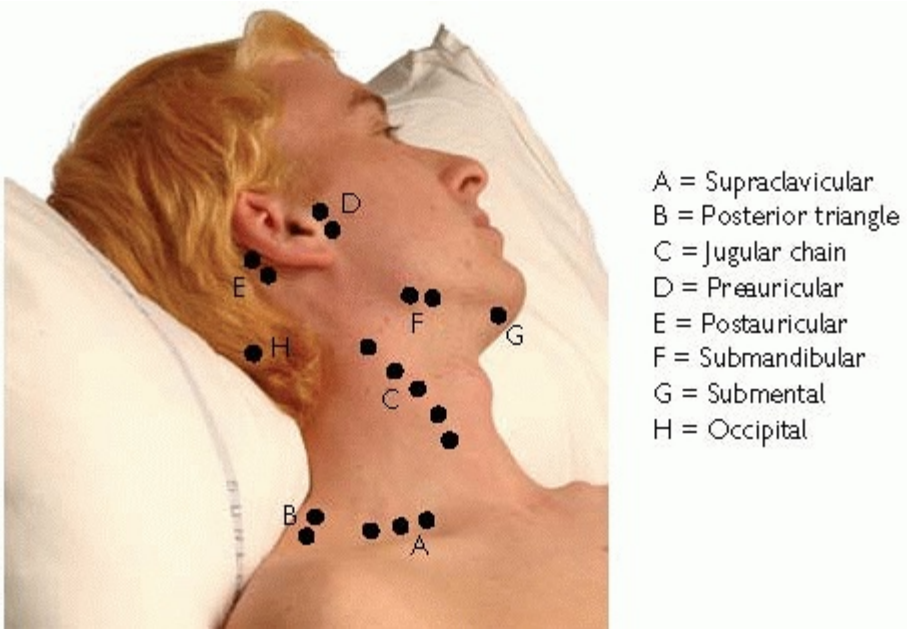

Fig. 3.1 Cervical and supraclavicular lymph nodes.

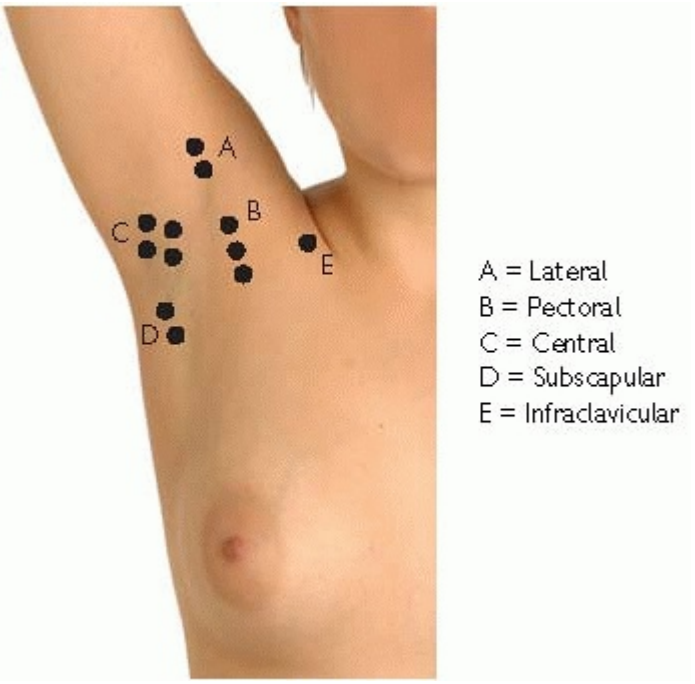

Fig. 3.2 Axillary lymph nodes.

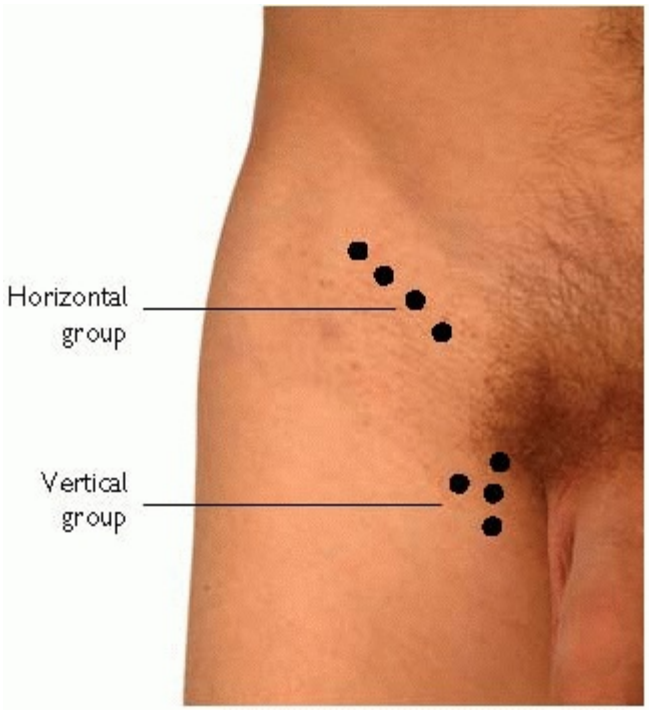

Fig. 3.3 Inguinal lymph nodes.

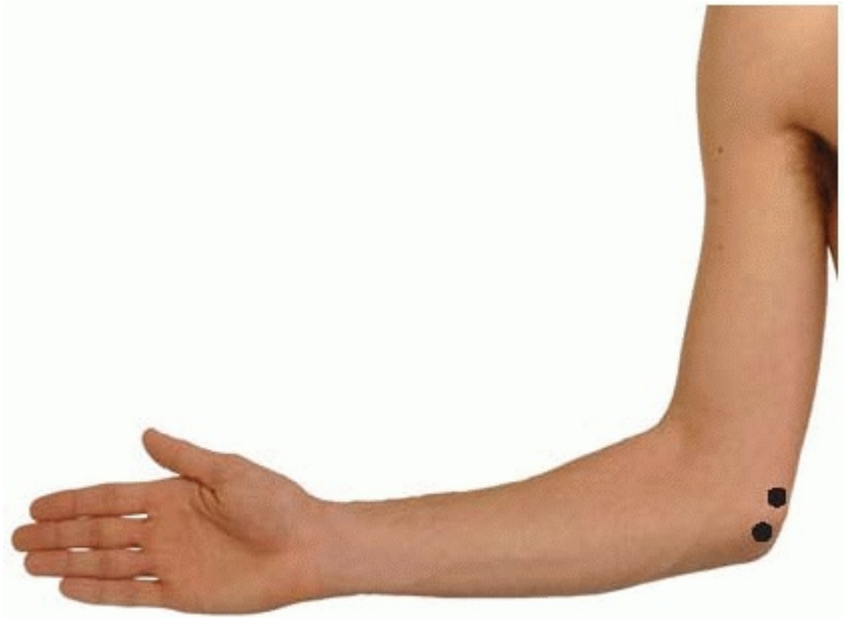

Fig. 3.4 Epitrochlear lymph nodes.

Hands

Examination of the hands is an important part of all examination routines and may provide a huge number of diagnostic clues. It is also something that the student may be asked to perform on a regular basis.

You will meet the various ‘hand signs’ relevant to the body systems throughout the book.

Set-up

Begin by exposing the forearms up to the elbows and asking the patient to place their hands on a pillow on their lap with you sitting opposite.

Bed-side clues

Make a point of looking around the room or the patient for any functional aids or adaptations.

Inspection

- ***Dorsum:*** ask the patient to hold their hands out flat, palms down.
- ***Palm:*** next, ask the patient to turn their hands over.
  - It is often possible to make a spot diagnosis with inspection of the palmar and dorsal surfaces as many diseases cause characteristic hand changes (e.g. rheumatoid arthritis, systemic sclerosis, psoriasis, ulnar nerve palsy).
- ***Skin colour:*** take note of the colour (e.g. palmar erythema, vasculitis, digital ischaemia, purpura) and consistency of the skin.
  - Note that pathological palmar erythema can also be found on the thenar and hypothenar eminences and also continues along the digits.
- ***Discrete lesions:*** are there any discrete lesions present? Examine as described as in Chapter 4.
- ***Muscles:*** look at the small muscles of the hand and the larger muscles of the forearm and make note of any wasting or fasciculation.
- ***Joints:*** make a point of looking at each joint in turn:
  - Distal interphalangeal (DIP).
  - Proximal interphalangeal (PIP).
  - Metacarpophalangeal (MCP).
  - Wrist.
- ***Bony deformities:*** look for evidence of swelling or deformities.
- ***Nails:*** the nails should be inspected carefully. See Box 3.7 for important nail signs not to be missed.

Box 3.5 Dupuytren's contracture

Dupuytren's fasciitis causes progressive thickening and contracture of fibrous bands on the palmar surface of the hand. This leads to a progressive fixed flexion of the fingers—usually the fourth and fifth digits.

Dupuytren's is more common in men than in women. The cause is unknown. It may be familial, sporadic, and has been associated with alcoholism, use of anticonvulsant drugs, and diabetes.

Box 3.6 Some finger joint deformities

- ***Swan neck:*** fixed flexion at the DIP and extension at the PIP joints—associated with rheumatoid arthritis (📖 p.394).
- ***Boutonniere:*** fixed extension at the DIP and flexion at the PIP joints—associated with rheumatoid arthritis (📖 p.394).
- ***Z-shaped thumb:*** flexion at the MCP joint of the thumb with hyperextension at the interphalangeal joint—associated with rheumatoid arthritis (📖 p.394).
- ***Ulnar deviation:*** a feature of rheumatoid arthritis and other conditions, the fingers are deviated medially (toward the ulnar aspect of the forearm) at the MCP joints.
- ***Wrist subluxation:*** deviation (either ulnar or radial) at the wrist.
- ***Heberden's nodes:*** swelling (due to osteophytes) at the DIP joints—a feature of osteoarthritis.
- ***Bouchard's nodes:*** similar to Heberden's nodes but at the PIP joints—a feature of osteoarthritis.

Box 3.7 Some important nail/finger-tip signs

- Important signs to look for are described elsewhere in this book:
- Leukonychia, koilonychia, Muehrcke's lines, blue lanulae = 📖 p.244.
- Xanthomata, Osler's nodes, Janeway lesions = 📖 p.168.
- Splinter haemorrhages, pitting, onycholysis, Beau's lines, paronychia, onychomycosis = 📖 p.91.

Clubbing

Also described on p.208. This is ↑ curvature of the nails. Early clubbing is seen as a softening of the nail bed but this is very difficult to detect. Progression leads to a loss of the angle at the base of the nail (the Lovibond angle) and eventually to gross curvature and deformity.

Objectively check for clubbing by putting the patients nails back-to-back as in Fig. 8.2. Clubbing leads to a loss of the diamond-shaped gap (Schamroth's sign).

Causes of clubbing

The full list of causes is huge. The diseases to be aware of are:

- *Pulmonary:* chronic interstitial lung diseases, chronic lung infections (e.g. bronchiectasis), cystic fibrosis, lung abscess, asbestosis, fibrosing alveolitis, lung cancer.
- *Cardiac:* cyanotic congenital heart disease, infective endocarditis.
- *Other:* liver cirrhosis, inflammatory bowel disease.

Palpation

- Palpate any abnormalities identified on inspection.
- Ask the patient if there is any tenderness and palpate those areas last.
- Pay attention to areas of temperature change.
- It is worth remembering to palpate the anatomical snuff box (Fig. 3.5).
  - At the base of the anatomical snuff box is the scaphoid and trapezium bones. Tenderness here may be the only sign of scaphoid damage. Pathology here is easily missed.

Movement

Before assessing movement, always ask the patient if they have pain anywhere in the hands. If allowed to continue, test passive and then active movements in all joints.

Passive movements

As in Chapter 11, move each joint and assess range of movement, any crepitus and whether there is any pain.

Active movements

▸ The examination here overlaps with that in Chapter 10.

- Ask the patient to open and close their hands quickly to test for signs of myotonic dystrophy (hand will be slow to relax— OHCM p.398).
- ***Wrist extension:*** test with the ‘prayer sign’ manoeuvre. Ask the patient to place their hands, palm to palm, in front of them with fingers extended as in Fig. 3.6.
- ***Wrist flexion:*** test with the ‘reverse prayer’ position. Ask the patient to place their hands back to back in front of them with fingers extended (Fig. 3.7).
- ***Finger flexion:*** ask the patient to make a fist.
- ***Finger extension:*** as the patient straighten their fingers out. Also tested with the prayer and reverse prayer positions.
- ***Dorsal interossei:*** (ulnar nerve). These can be assessed by asking the patient to spread the fingers apart (abduction) and resist your attempts to push them together.
- ***Palmar interossei:*** (ulnar nerve). These can be tested by asking the patient to hold a piece of paper between their fingers and resisting your attempts to pull it free.
- ***Abductor pollicis brevis:*** (median nerve). Ask the patient to put their hand out, palm upwards, and then point their thumb at the ceiling. You should then try to push the thumb back towards the hand whilst they resist you.
- ***Opponens pollicis:*** (median nerve). This can be assessed by asking the patient to put thumb and little finger together in an ‘O’ and again instructing the patient to try to stop you pulling them apart.

Sensation

Test modalities of light touch, pin prick (pain), vibration, and joint position sense in both peripheral nerve (ulnar, median, radial) and dermatomal distributions. Examining the hands neurologically is detailed on p.324.

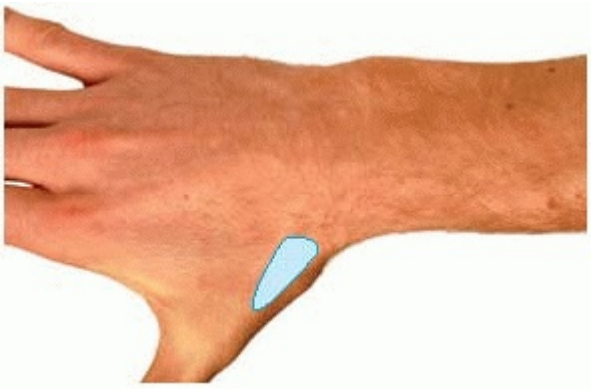

Fig. 3.5 The anatomical snuff box formed by the tendons of the extensor pollicis brevis and abductor pollicis longus laterally and the tendon of the extensor pollicis longus medially.

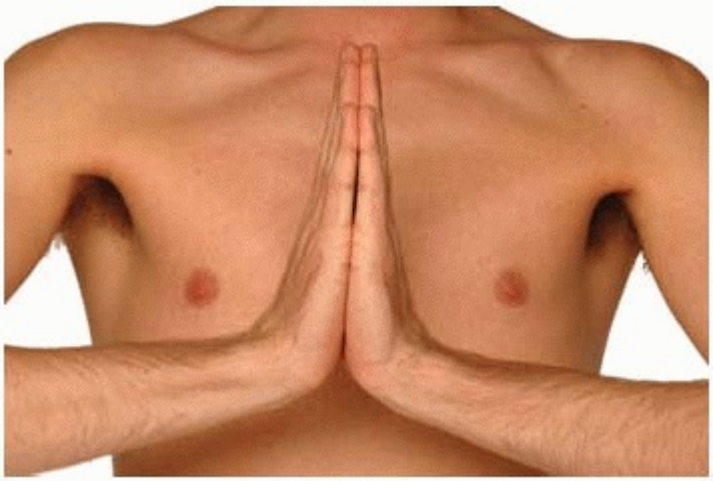

Fig. 3.6 The prayer position.

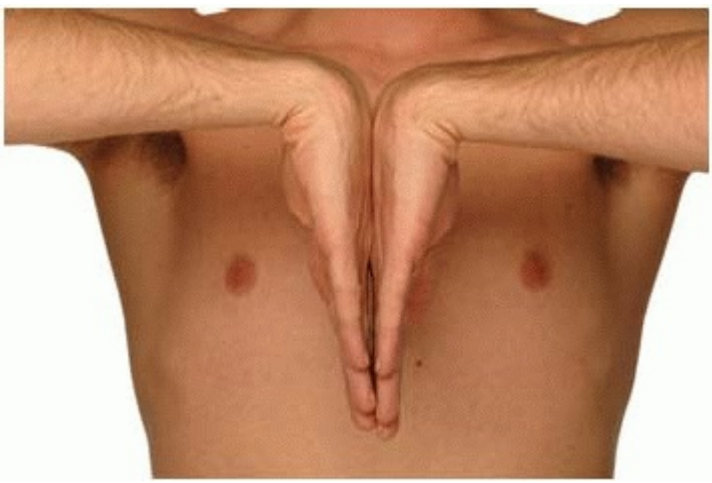

Fig. 3.7 The reverse prayer position.

Pulses

Palpate both radial and ulnar pulses (p.170).

Elbows

- Always examine the elbows to elicit any clues as to the cause of joint pathology.
- For example, there may be rheumatoid nodules, psoriatic plaques, xanthomata or scars.

Function

Testing function is a vital part of any hand examination and should not be overlooked. Ask the patient to:

- Write their name.
- Pour a glass of water.
- Fasten and unfasten a button.
- Pick a coin up from a flat surface.

Box 3.8 Allan's test

This is a test of hand perfusion.

- Ask the patient to make a fist.
- Next, occlude both radial and ulnar arteries by applying pressure over them for ~5 seconds.
- Ask the patient to open the palm—which should now be white.
- Release the pressure from the radial artery and look at the colour of the palm. If perfusion is adequate, it should change from pale to pink.
- Repeat for the ulnar artery.

Box 3.9 Some more eponymous signs at the hand

Tinel's sign

A test for nerve compression. Commonly used at the wrist to test for median nerve compression in carpal tunnel syndrome.

- Percuss the nerve over the site of possible compression (at the wrist, gently tap centrally near the flexor palmaris tendon).
- If the nerve is compressed, the patient will experience tingling in the distribution of the nerve on each tap.

Froment's sign

A test of ulnar nerve function. Ask the patient to grasp a piece of paper between their thumb and forefinger. Alternatively, ask them to make a fist. If there is ulnar nerve damage, the thumb will be unable to adduct so will flex instead (see Fig. 10.23, p.347).

Finkelstein's test

Ask the patient to flex the thumb then flex and ulnar deviate the wrist. Pain is indicative of De Quervain's tenosynovitis (tendons of abductor pollicis longus and extensor pollicis brevis).

Recognizable syndromes

Some physical (especially facial) characteristics are so typical of certain congenital, endocrine, and other disorders that they immediately suggest the diagnosis.

The following are the physical features of certain conditions that can be appreciated on first inspection—enabling a ‘spot diagnosis’. Most

of these conditions have many other features which are not detailed here.

Down's syndrome (trisomy 21)

Facies: oblique orbital fissures, epicanthic folds, hypertelorism (widely spaced eyes), conjunctivitis, lenticular opacities, small low set ears, flat nasal bridge, mouth hanging open, protruding tongue (large, heavily fissured).

Hands: single palmar crease (not pathognomonic), short broad hands, curved little finger, hyperflexible joints with generalized hypotonia.

Other: mental deficiency, wide gap between 1st and 2nd toes, short stature, dementia of Alzheimer type, hypothyroidism.

Turner's syndrome (45 XO)

Facies: micrognathia (small chin), epicanthic folds, low set ears, fish-like mouth, hypertelorism, ptosis, strabismus.

Neck: short, webbed neck, redundant skin folds at back of neck, low hairline.

Chest: shield-like chest, widely spaced nipples.

Upper limbs: short fourth metacarpal or metatarsal, hyperplastic nails, lymphoedema, ↑ carrying angle of the elbow.

Marfan's syndrome

Autosomal dominant condition caused by defects in fibrillin gene (ch15q).

Facies: long, narrow face, high-arched palate, lens dislocation, heterochromia of iris, blue sclera, myopia.

Limbs: tall stature, armspan > height, hyperextensibility of joints, recurrent dislocations.

Hands: elongated fingers and toes (arachnodactyly).

Chest: funnel or pigeon chest, pectus excavatum, kyphoscoliosis, aortic incompetence.

Other: cystic disease of the lungs (spontaneous pneumothorax, bullae, apical fibrosis, aspergilloma and bronchiectasis), inguinal or femoral hernias.

Tuberous sclerosis

Also known as Bourneville's disease of the skin. Autosomal dominant condition localized to chromosomes 16 and 9.

Skin: adenoma sebaceum (angiofibromata—papular, salmon-coloured eruption on centre of the face, especially at the nasolabial folds), Shagreen patches (flesh-coloured, lumpy plaques found mostly on the lower back), ungal fibromata (firm, pink, periungal papules growing out from nail beds of fingers and toes), hypopigmented 'ash-leaf' macules (trunk and buttocks), café-au-lait macules and patches.

Neurofibromatosis—type 1

Also known as von Recklinhausen's disease—autosomal dominant.

Skin: neurofribomatoma (single, lobulated or pedunculated, soft, firm, mobile, lumps or nodules along the course of nerves), Cafe-au-lait spots (especially in the axillae), axillary freckling.

Other: kyphoscoliosis, nerve root involvement or compression, muscle wasting, sensory loss (Charcot's joints), plexiform neuroma, lung cysts.

Peutz-Jegher's syndrome

Skin: sparse or profuse small brownish-black pigmented macules on lips, around mouth and on buccal mucosa, hands and fingers (see *OHCM* p.732).

Oculocutaneous albinism

Marked hypomelaninosis (pale skin), white hair or faintly yellow blonde. Nystagmus, photophobia, hypopigmented fundus, translucent iris (pink).

Myotonic dystrophy

Facies: myopathic facies (drooping mouth and long, lean, sad, sleepy expression), frontal balding in men, ptosis, wasting of facial muscles (especially temporalis and masseter), cataracts.

Other: wasting of sternomastoids, shoulder girdle and quadriceps, areflexia, myotonia (percussion in tongue and thenar eminence, delay before releasing grip), cardiomyopathy, slurred speech, testicular atrophy, diabetes, intellect and personality deterioration in later stages.

Parkinson's disease

Facies: expressionless, unblinking face, drooling, titubation, blepharoclonus (tremor of eyelids when eyes gently closed).

Gait: shuffling, festinant gait with reduced arm swing.

Tremor: pill-rolling tremor, lead-pipe rigidity, cog-wheel rigidity, glabellar tap positive (p.334) small, tremulous, untidy hand writing (micrographia).

Osler-Weber-Rendu syndrome

Also known as hereditary haemorrhagic telangiectasia (HHT).

Facies: telangiectasia (on face, around mouth, on lips, on tongue, buccal mucosa, nasal mucosa), telangiectasia may also be found on fingers. Associated with epistaxis, GI haemorrhage, iron deficiency anaemia, haemoptysis.

Systemic sclerosis/CREST syndrome

Face/hands: telangiectasia and pigmentation, pinched nose, perioral tethering, tight, shiny and adherent skin, vasculitis, atrophy of finger pulps, calcinosis (fingers), Raynaud's phenomenon.

Vitamin and trace element deficiencies

Fat soluble vitamins

Vitamin A (retinol)

- Found in dairy produce, eggs, fish oils, and liver.
- Deficiency causes night blindness, xerophthalmia, keratomalacia (corneal thickening) and follicular hyperkeratosis.

Vitamin D (cholecalciferol)

- Found in fish liver oils, dairy produce, and undergoes metabolism at the kidneys and the skin using UV light.
- Deficiency causes rickets (in children) and osteomalacia (in adults). Proximal muscle weakness may be evident.

Vitamin E (alpha-tocopherol)

- Widely distributed, green vegetables, and vegetable oils.
- Deficiency causes haemolytic anaemia (premature infants) and gross ataxia.

Vitamin K (K_1 = phylloquinine K_2 = menaquinone)

- Widely distributed but particularly in green vegetables. Synthesized by intestinal bacteria.
- Deficiency causes coagulation defects seen as easy bruising and haemorrhage.

Water soluble vitamins

Vitamin B_1 (thiamine)

- Found in cereals, peas, beans, yeast, and wholemeal flour. It is an essential factor in carbohydrate metabolism and transketolation reactions.
- Deficiency causes dry beri-beri (sensory and motor peripheral neuropathy), wet beri-beri (high output cardiac failure and oedema), Wernicke-Korsakoff syndrome.

Vitamin B_2 (riboflavin)

- Found in wholemeal flour, meat, fish, and dairy produce. It is a coenzyme in reversible electron carriage in oxidation-reduction reactions.

- Deficiency gives angular stomatitis (fissuring and inflammation at the corners of the mouth), inflamed oral mucous membranes, seborrhoeic dermatitis, and peripheral neuropathy.

Vitamin B_3 (niacin)

- Found in fish, liver, nuts, and wholemeal flour.
- Deficiency causes pellagra ( *OHCM*, p.250): dermatitis, diarrhoea, dementia.

Vitamin B_6 (pyridoxine)

- Widespread distribution, also synthesized from tryptophan.
- Deficiency causes peripheral neuropathy, convulsions, and sideroblastic anaemia. Deficiency may be provoked by a number of commonly used drugs (e.g. isoniazid, hydralazine, penicillamine) and is also seen in alcoholism and pregnancy.

Vitamin B_{12} (cyanocobalamin)

- Causes of a deficiency are numerous and include partial or total gastrectomy, Crohn's disease, ileal resection, jejunal diverticulae, blind loop syndrome, and tapeworm.
- Deficiency causes megaloblastic anaemia, peripheral neuropathy, subacute combined degeneration of the spinal cord ( *OHCM*, p,634), depression, psychosis, and optic atrophy.

Vitamin B_9 (folic acid)

- Deficiency can be caused by poor diet, malabsorption states, coeliac disease, Crohn's disease, gastrectomy, drugs (e.g. methotrexate, phenytoin), excessive utilization (e.g. leukaemia, malignancy, inflammatory disease).
- Consequences of deficiency include megaloblastic anaemia, and glossitis.

Vitamin C (ascorbic acid)

Deficiency causes scurvy (perifoillicular haemorrhage, bleeding swollen gums, spontaneous bruising, corkscrew hair, failure of wound healing), anaemia, and osteoporosis.

Trace elements

Copper

- Deficiency results in hypochromic and microcytic anaemia, Wilson's disease, impaired bone mineralization, Menks' kinky hair syndrome (growth failure, mental deficiency, bone lesions, brittle hair, anaemia).
- Usually caused by copper malabsorption.

Zinc

Deficiency causes achondromatosis enterpathica (infants develop growth retardation, hair loss, severe diarrhoea, candida and bacterial infections), impaired wound healing, skin ulcers, alopecia, night blindness, confusion, apathy, and depression.

Magnesium

Severe deficiency can cause cardiac arrhythmias, paraesthesia and tetany.

Iodine

Severe deficiency can cause cretinism (children), hypothyroidism, and goitre.

The elderly patient

For Nigel Hawthorne's on screen King George III, examination by his doctor during an attack of porphyria was ‘the very last resort’ and viewed as an ‘intolerable intrusion’. However, for older people, in whom the ‘typical’ presentations of illness may be subtle or unusual, a thorough physical examination is a cornerstone of assessment.

The value of a thorough physical examination can be underestimated by doctors, but be highly regarded as a therapeutic benefit by patients. This general overview complements the system-based chapters that follow, but the key message is repeated throughout—to reinforce the value of a comprehensive, holistic and unrushed examination.

General points

- ***Use your eyes:*** a key question in your mind should be ‘is the patient unwell?’. Learn not to overlook key indices such as hypothermia (see below) and delirium which point to an acutely unwell patient.
- ***Seek additional diagnoses:*** multiple illnesses are a typical feature of old age—seemingly incidental findings (to the presenting condition) are common, so look out for skin lesions (malignant?), new/isolated patches of ‘psoriasis’ (Bowen's disease?), asymptomatic peripheral arterial disease etc.
- ***Talk to your patient:*** during the examination. As indicated, it is often of huge therapeutic benefit, of reassurance, engendering trust, and potentially gaining additional history—especially if an incidental lesion is discovered.

Key points

- ***Observations:*** nurses spend time recording them—so do your colleagues the courtesy of recording them in the notes, *and act on them*.
  - Many patients may run low blood pressures, often as a consequence of medications—a small drop from this point is easily overlooked, but may be the only sign of a myocardial infarction.
  - Recognize the limits of temperature/fever—seriously unwell older people may actually be hypothermic.
- ***Hydration:*** may be difficult to assess—reduction in skin turgor through changes in elasticity with age, dry mucous membranes (e.g. through mouth breathing), or sunken eyes (muscle wasting, weight loss) are useful in younger patients, but less reliable in elders.
  - ▸ A useful alternative is axillary palpation—are they sweating?
- ***Skin and nail health:*** asteatosis and varicose eczema are common, but easily overlooked.
  - Look out for typical lesions in atypical places—squamous cell carcinomas are notorious in this respect.
  - Learn to look at footwear/toenails—is there onychogryphosis?
- ***Nutrition:*** signs of weight loss are often obvious—ill-fitting clothes and dentures are good examples.
- ***Joints:*** remember to look and examine—is the patient's mobility worse, or the reason for falling acute (pseudo) gout?
- ***MMSEIAMTS:*** should be mandatory for the majority of patients.

- ***Gait (where possible):*** Akin to mental state examination, should be undertaken whenever possible. See the locomotor chapter (p.403) for a brief description of the ‘get up and go’ test.

Box 3.8 Geriatric giants

So described by Bernard Isaacs, one of the key figures of contemporary geriatric medicine. Isaacs described five ‘giants’

- Immobility.
- Instability.
- Incontinence.
- Intellectual impairment.
- Iatrogenic illness.

These are not ‘diagnoses’, so avoid reaching them—but extremely common presentations of illness in older people, for which an underlying cause (or causes!) should be sought.

We thank Dr Richard Fuller for providing this page.

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Chapter 4

Skin, Hair, and Nails

Applied anatomy and physiology

The skin, nails, hairs, glands and associated nerve endings make up the 'integumentary system'.

Skin

The skin acts as a physical, biochemical, and immunological barrier between the outside world and the body. It also has a role in temperature regulation, synthesis of vitamin D, prevention of water loss, antigen presentation, and sensation.

It is important to remember that the skin also has an important psychosocial function. When we look at another person, we are in fact looking at their skin. As our skin represents our outward appearance to the world, unsightly blemishes or lesions can have a significant impact on a person's self esteem despite their small size.

The skin is made up of 3 layers—epidermis, dermis, and hypodermis.

Epidermis

This is the outermost layer and is formed of a modified stratified squamous epithelium. Almost 90% of epithelial cells are keratinocytes. These cells are produced in the basal layer and then rise to the surface as more are produced below and the outer cells are shed. The time taken from forming in the basal layer to shedding is usually about 3 months.

Melanocytes reside in the basal layer and secrete melanin into surrounding keratinocytes via long projections. This, along with the underlying fat and blood, gives the skin its colour. In this way, skin tone is determined by the size and number of melanin granules and not by the number of melanocytes.

Dermis

Below the epidermis, lies a layer of connective tissue consisting of collagen, elastic fibres, and ground substance. It is here where the 'skin appendages', muscles, nerves, and blood vessels lie.

Hypodermis

Also known as the subcutaneous layer or the superficial fascia, this consists of adipose tissue and serves both as a lipid store and provides insulation. It also contributes to the body contours and shape.

Glands

After infancy, sebaceous glands become active again at puberty and secrete sebum, a mixture of fatty acids and salts, directly onto the skin or into the necks of hair follicles. This water-proofs and lubricates the skin and hair. They are particularly numerous in the upper chest, back, face, and scalp.

Sweat glands secrete a mixture of water, electrolytes, urea, urate, ammonia, and mild acids. Eccrine sweat glands are found all over the body surface, besides the mucosa. Apocrine sweat glands are found in the axillae and pubic regions, secrete a more viscous sweat and are under clear autonomic control. These do not function until puberty.

Hair

Hairs are formed by 'follicles' of specialized epidermal cells buried deep in the dermis. Humans are covered with hair, apart from the palms, soles, inner surface of the labia minora, prepuce, and glans penis. Most is fine, unpigmented, *vellous* hair and not easily seen. *Terminal* hair is coarser, pigmented, and seen on the scalp, beard, and pubic regions. Growth is cyclical with each follicle shedding its hair and then regrowing. A cycle lasts ~4 years for scalp hair.

Nails

These are sheets of keratin which are continuously produced by the matrix at the proximal end of the nail plate. This can often be seen as the small, lighter, crescent-shaped area just before the cuticle. Nails grow at ~0.1 mm/day, toenails growing slower than fingernails.

The dermatological history

Patients with a skin condition may describe a variety of complaints. However, whether they talk of a rash, spots, ‘growth’, lump, ulcer, itch, or pain, the following guide should be used.

The history should help you establish the time course and behaviour of the complaint as well as any possible precipitating or exacerbating factors.

▸ Don't waste time listening to the patient describe what the rash looks like—you're about to examine it yourself!

History of the presenting complaint

- When was the problem first noticed?
- How have things changed since? Has it been a continuous or intermittent problem?
- Where did it start?
- Has it spread—is it still spreading?
- If spreading—is it spreading from the edge or appearing in crops?
- What is the distribution of the problem?
- Is there any discharge, bleeding, or scale?
- Is there pain, itch, or altered sensation?
- Has it started to resolve?
- Are there any obvious factors which either trigger or relieve the problem? Ask especially about:
  - UV light (sunlight).
  - Foods.
  - Temperature.
  - Contact with any other substances.
- Has it been treated with anything—was the treatment effective?
- Are there any systemic symptoms such as fever, headache, fatigue, anorexia, weight loss or sore throat?

PMH

- Previous skin problems?
- Diabetes, connective tissue disease, inflammatory bowel disease, asthma?
- What does the patient use on their skin—e.g. soaps, creams, cleansers?

Allergies

- Remember to ask about the *nature* of any allergic reaction claimed.

DHx

- Which drugs is the patient taking and for how long?
- Did the start of any therapy coincide with the start of the skin complaint? (Remember, there can be a delay of months before a rash becomes apparent.)
- Remember topical and over-the-counter drugs.

FHx

- Ask especially about atopy, eczema, psoriasis, and skin cancers.

SHx

- Occupation?
- Hobbies—include pets and any pets of close friends or relatives.
- Living conditions—how many share the house/living space?
- Recent travel? Were appropriate vaccinations taken before leaving?
- Insect bites?
- Has the patient been exposed to venereal disease or HIV? Consider a full sexual history—see Chapter 12.  Approach with caution.

Psychosocial impact

Be aware of the psychological and social function of the skin. Ask what effect the condition has had on the patient in this regard and consider whether this aspect of the condition needs to be formally addressed.

Hair and nail symptoms

Hair loss

'Alopecia' is the loss of hair and should be treated in much the same way as any other symptom, noting…

- Mode of onset (sudden/gradual).
- Associated symptoms.
- Pain.
- Rash.
- Family history of hair loss.

Note also:

- Regions of hair loss (scalp/body/face).
- A recognizable pattern of hair loss?
  - 'Male pattern' baldness is at the frontal and temporal areas of the scalp and at the crown.
  - Hair loss at the very front of the scalp (caused by pulling back of hair when styling—particularly in women).

Abnormal hair growth

Facial hair growth is common in post-pubertal women but many find this distressing. If the patient reports abnormal hair growth, treat as any other symptom but remember to ask about:

- FHx of a similar problem.
- Menstrual cycle (if female)—when was the last menstrual period, are they usually regular or erratic?
- Symptoms of virilization (if female)—e.g. voice change, clitoromegaly.
- DHx.

Nail symptoms

These should be treated as any other dermatological condition—but you should remember to direct your questions towards finding other conditions which have nail involvement (e.g. psoriasis, eczema, fungal infections).

Box 4.1 Important hair disorders/signs

- ***Male-pattern baldness:*** commonly occurs from the 2nd decade. Hair is lost first from the temporal regions, frontal and the crown.
- ***Alopecia areata:*** associated with autoimmune disorders and occurs in the 2nd or 3rd decade. Sharply defined, non-inflammatory bald patches on the scalp. There may be 'exclamation mark' hairs which are thinner at the base. Also affects the eyebrows and beard. Nails may be slow-growing and show pitting.

- ***Alopecia totalis:*** loss of hair from all of the scalp.
- ***Alopecia universalis:*** loss of all body hair.
- ***Telogen effluvium:*** normally, hairs are growing and shedding at different times and at different rates. A severe illness, high fever, or childbirth may synchronize all the hair follicles causing them to shed at the same time—about 3 months later. This gives a brief total hair loss which grows back.
- ***Scarring alopecia:*** inflammatory lesions causing hair loss include lichen planus, burns, and infection.

Box 4.2 Important nail disorders/signs

See also Chapter 3 (p.73).

- ***Splinter haemorrhages:*** tiny, longitudinal streak haemorrhages under the nails caused by micro-emboli or trauma. Can be a normal finding in manual workers.
- ***Pitting:*** tiny indentations in the surface of the nail. A feature of psoriasis and less commonly eczema, lichen planus and alopecia areata.
- ***Onycholysis:*** premature lifting of the nail.
- ***Leukonychia:*** white discolouration of the nail. A sign of low albumin or chronic ill-health.
- ***Beau's lines:*** transverse depressions in the nail. Coincide with arrested nail growth during a period of acute severe illness.
- ***Paronychia:*** infection of the skin adjacent to the nail—causing pain, swelling, redness, and tenderness.
- ***Koilonychia:*** spooning (concave indentation) of the nail. Associated with severe iron-deficiency.
- ***Clubbing:*** see p.73.
- ***Onychomycosis:*** fungal nail infection causing the nail to become thickened, opaque, crumbly and yellow. Often with onycholysis and may be indistinguishable from psoriatic nail changes.

Examining the skin

Be wary of only focusing on the area identified by the patient—the whole organ needs to be examined.

After explaining and asking permission, ask the patient to undress to their underwear, lie back comfortably on a couch or bed, and cover them with a sheet. Ensure that the room is warm and private and that you have adequate lighting—preferably in the form of an adjustable light source. You should have a chaperone—preferably one of the opposite sex to yourself.*

The examination in dermatology consists largely of a careful, thorough inspection along with an accurate description using recognized dermatological terms (some of which are overleaf).

General inspection of the skin

Begin by scanning the whole surface of the skin for any abnormal lesions. This can be done in any order but it will help you to build a pattern that you can consistently remember which does not miss any areas!

Remember to inspect those areas that are usually hidden:

- Inner thighs.
- Undersurfaces of female breasts.
- External genitalia.
- Axillae.
- Natal cleft (between the buttocks).

Remember also to inspect the mucosal surfaces of the mouth, nails, hair, and scalp.

Skin colour

Skin colour varies widely between individuals but should always be even in distribution with normal variation for sun-exposed surfaces.

Inspecting a lesion

Inspect each lesion carefully and note…

- Grouped or solitary? Pattern if grouped (see p.96).
- Distribution/location:
  - Symmetrical/asymmetrical?
  - Peripheral?
  - In only light exposed areas?
  - Dermatomal?
- Colour.
- Shape.
- Size.
- Surface.
- Edge.
- Nature of the surrounding skin.

For each of the previous points, describe as accurately as you can using dermatological terms. However, if a lesion is pear-shaped, it is perfectly acceptable to call it just that!

When noting the distribution, bear in mind what clothing (or lack of) is usually at that site and what other objects/substances that part of the body would come into contact with. (Consider especially belt buckles, watches, gloves and jewellery.)

Palpation

Each lesion should be felt (remember to ask for—and be granted—permission first). It is rare to catch an infection from touching a rash or lesion and it's even rarer to see a dermatologist wearing gloves. Each situation should be judged at the time—obviously, gloves should be worn if there is bleeding or exudate present or if you are examining the genitalia.

For each lesion, note:

- Tenderness (watch the patient's face).
- Consistency.
- Temperature:
  - Use the back of your hand (inflamed lesions are usually hot).
- Depth/height.
- Mobility:
  - What skin layer is the lesion in and is it attached to any underlying or nearby structures?
  - Can it be moved in all directions or only in one or two?
  - Does it move with movement of underlying muscle or tendons?

Beyond the lesion

The skin condition must be seen in the context of the whole patient and other organ systems should be examined as necessary.

Remember to palpate regional lymph nodes if appropriate (p.68).

Box 4.3 Köebner's phenomenon

This is the tendency for certain rashes or lesions to form at the site of skin trauma—including surgical scars.

* This is still controversial at the time of writing—attitudes vary between countries. In the UK, offii cial advice is that *all* doctors should have a chaperone when performing an intimate examination and the chaperone should be the same sex as the patient. In practice, male doctors performing an examination on a female and females performing an examination on a male should always have a chaperone present whilst the need for a chaperone in other situations is judged at the time.

Box 4.4 Some common skin colour abnormalities

- ***Jaundice:*** a yellow tinge to the skin. Best appreciated at the sclera.

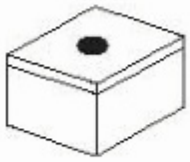
- ***Carotenaemia:*** a similar yellow/orange tinge to the skin as jaundice but the sclerae are spared.
- ***Haemochromatosis:*** 'slate grey' skin colouration
- ***Addison's disease:*** darkened scars and skin creases on the palms and soles—also darkening of mucosa.
- ***Albinism:*** a lack of pigmentation with very white skin and pink irises
- ***Vitiligo:*** autoimmune phenomenon resulting in patchy loss of skin colour.

Describing a lesion

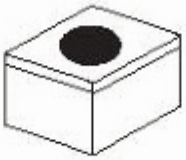
A careful description often clinches the diagnosis in dermatology. All lesions should be documented in accepted dermatological terms.

(a)

Flat, non-palpable changes in skin colour

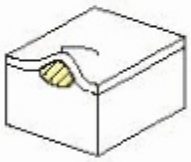


Macule
Flat, non-palpable change in skin colour <0.5cm diameter.
'Freckles' are pigmented macules

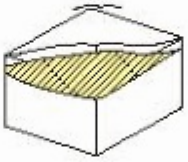


Patch
Flat, non-palpable change in skin colour >0.5cm diameter

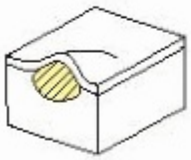
Elevation due to fluid in a cavity



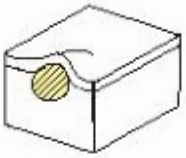
Vesicle
Fluid below the epidermis <0.5cm diameter



Bulla
Large, fluid-filled lesion below the epidermis >10cm diameter



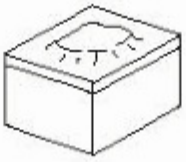
Blister
Fluid below the epidermis >0.5cm diameter



Pustule
Visible collection of pus in the subcutis

Elevation due to solid masses

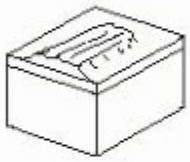
Papule
A raised area <0.5cm diameter



Plaque
A raised area >2cm diameter

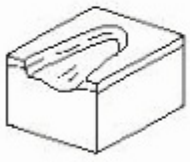
Nodule
A mass or lump >0.5cm diameter

Wheal
Dermal oedema

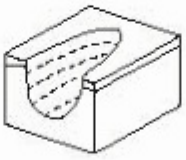


Callus
Hyperplastic epidermis, often found on the soles, palms or other areas of excessive friction/use

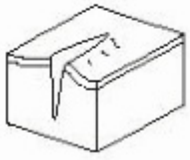
Loss of skin



Erosion
Partial epidermal loss
Heals without scarring



Ulcer
Full thickness skin loss
(see p.xxx)



Fissure
A linear crack

Atrophy
Thinning of the epidermis

Loss of tissue (epidermis/dermis + /or subcutis)

Fig. 4.1(a) Primary lesions.

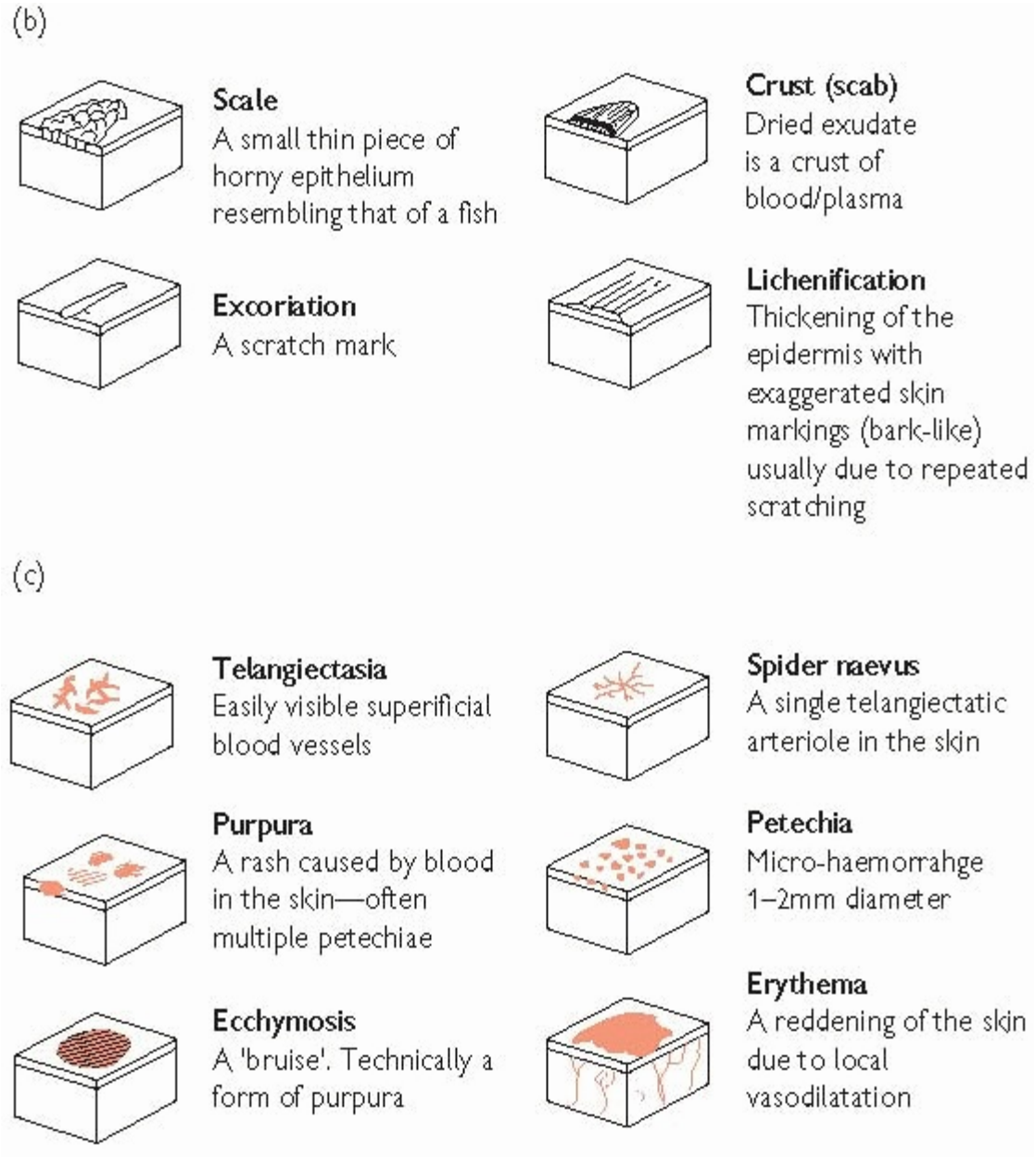


Fig. 4.1 (b) Secondary lesions, (c) Vascular lesions.

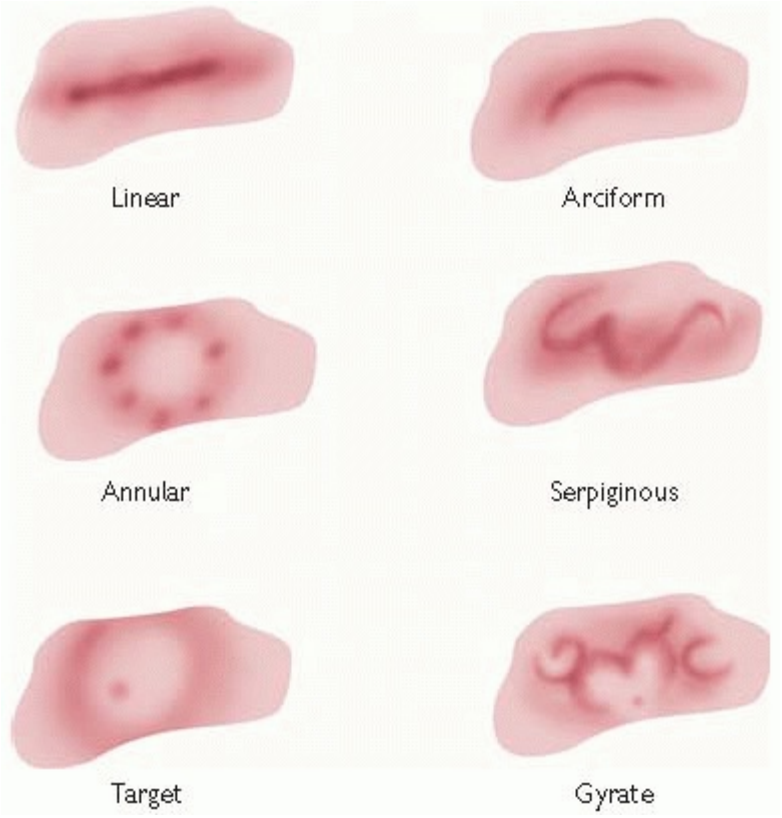


Fig. 4.2 Descriptive terms for lesion shapes and patterns of grouped lesions.

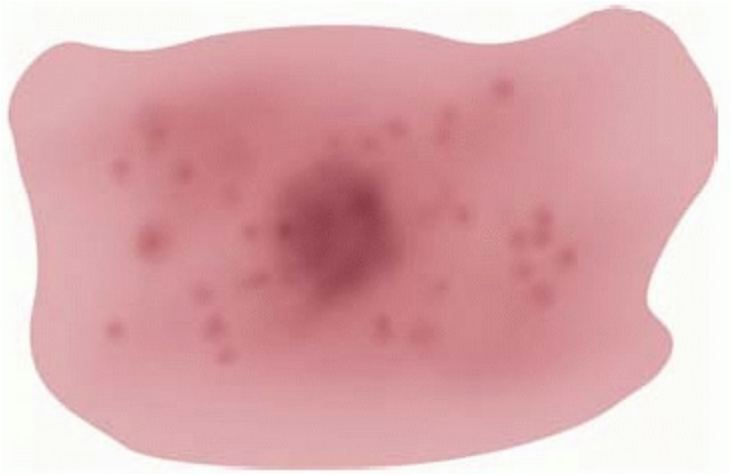


Fig. 4.3 Confluence of grouped lesions. Note how the smaller lesions coalesce to form a larger lesion.

Malignant melanoma

An invasive malignant tumour of melanocytes, mostly occurring in white adults, more common in women. See *OCHM*, p.430. You should be alerted to the possibility of a malignant mole if the patient describes a newly presenting pigmented lesion.

There are two systems to assist diagnosis of malignant melanoma—the American 'ABCD' and the British 'Glasgow 7-point checklist'.

ABCD

- A: asymmetry.
- B: irregular border.
- C: irregular colour.
- D: diameter >1cm.

Glasgow 7-point checklist

This is divided into major and minor criteria which depend upon the history and examination respectively. Any lesion with one major feature should be considered for removal. Additional minor features add to the clinical suspicion of melanoma.

Major features

- Change in size.
- Change in shape.
- Change in colour.

Minor features

- Diameter >5mm.
- Inflammation.
- Oozing or bleeding.
- Itch or altered sensation.

Examining a lump

Any raised lesion or lump should be inspected and palpated as described previously. Note: position, distribution, colour, shape, size, surface, edge, nature of the surrounding skin, tenderness, consistency, temperature, and mobility.

When examining a lump, there are some points to pay particular attention to:

Which layer is the lump in?

- Does it move with the skin? (Epidermal or dermal.)
- Does the skin move over the lump? (Subcutis.)
- Does it move with muscular contraction? (Muscle/tendon.)
- Does it move only in one direction? (Tendon or nerve.)
  - If the lesion belongs to a nerve, the patient may feel pins-and-needles in the distribution of the nerve when the lump is pressed.
- Is it immobile? (Bone.)

Additional characteristics to consider…

- ***Consistency:*** e.g. stony, rubbery, spongy, soft. (Remember the consistency does not always correlate with the composition—a fluid-filled lump will feel hard if it is tense.)
- ***Fluctuation:*** press one side of the lump—the other sides may protrude.
  - If the lump is solid, it will bulge at the opposite side only.
- ***Fluid thrill:*** this can only be elicited if the fluid-filled lesion is very large. Examine by tapping on one side and feeling the impulse on the other much as you would for ascites (📖 p.260).
- ***Translucency:*** darken the room and press a lit pen-torch to one side of the lump—it will 'glow' illuminating the whole lump in the presence of water, serum, fat, or lymph. Solid lumps will not transilluminate.
- ***Resonance:*** only possible to test on large lumps. Percuss as you would any other part of the body (📖 p.214) and listen (and feel) if the lump is hollow (gas-filled) or solid.
- ***Pulsatility:*** can you feel a pulse in the lump? Consider carefully if the pulse is transmitted from an underlying structure or if the lump itself is pulsating.
  - Use two fingers and place one on either side of the lump.
  - If the lump is pulsating, it will be 'expansile' and your fingers will move up and outwards, away from each other.
  - If the pulse is transmitted from a structure below, your fingers will move upwards but not outwards (see 📖 p.259).
- ***Compressibility:*** attempt to compress the lump until it disappears. If this is possible, release the pressure and watch for the lump reforming. Compressible lumps may be fluid-filled or vascular malformations. Note, this is not 'reducibility' (see below).

- ***Reducibility:*** a feature of hernias. Attempt to reduce the lump by manoeuvring its contents into another space (e.g. back into the abdominal cavity). Ask the patient to cough and watch for the lump reforming.

Auscultation

You should always listen with a stethoscope over any lump, you could gain important clues regarding its origin and contents. Listen especially for:

- Vascular bruits.
- Bowel sounds.

Examining an ulcer

Ulcers should be examined as any other skin lesion noting: position, distribution, colour, shape, size, surface, edge, nature of the surrounding skin, tenderness, consistency, and temperature.

If the shape of the ulcer, or position, is unusual or difficult to describe, make a drawing!

There are some characteristics particular to ulcers which should also be considered:

- ***Base:*** if the base of the ulcer can be seen (i.e. not covered with mucus, blood or crust), it should be carefully examined and described. Ulcers usually have either slough or granulation tissue at the base. Look especially for bone, tendons, and blood vessels.
- ***Edge:*** look carefully at the edge—it may help to make a quick drawing of the edge in cross-section. Some typical edges are described as follows (also see Fig. 4.4):
  - ***Sloping:*** these ulcers are usually shallow and a sloping edge implies that it is healing (e.g. venous ulcers).
  - ***'Punched out':*** this is full-thickness skin loss and typical of neuropathic ulceration and vasculitic lesions.
  - ***'Undermined':*** these extend below the visible edge creating a 'lip'. This is typical of pyoderma gangrenosum and infected ulceration such as TB.
  - ***'Rolled':*** here, the edge is mounded but neither everted or undermined and implies proliferation of the tissues at the edge of the ulcer. Basal cell carcinoma typically has a 'rolled' edge which is often described as 'pearly' in colour with thin overlying vessels.
  - ***Everted:*** here the tissues at the edge of the ulcer are proliferating too fast, creating an everted lip. This is typical of neoplastic ulceration.
- ***Depth:*** determine what layer (of skin or underlying tissues) the ulcer extends to. If possible, estimate the depth in mm.
- ***Discharge:*** if the ulcer has any discharge (e.g. serous fluid, pus, blood), this should be examined and noted. If there is overlying scab/crust (dried discharge/scale), this should be carefully removed so as to examine the base of the ulcer.

▸ Students should *not* remove a scab/crust unless asked to and supervised by someone more senior.

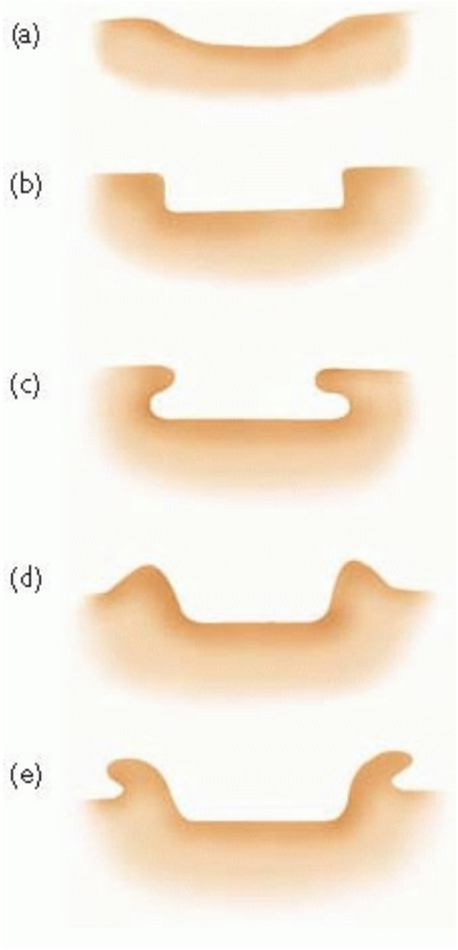


Fig. 4.4 Representation of some ulcer edges. A: sloping. B: punched out, C: undermined, D: rolled, E: everted.

Box 4.5 Leg ulcers

Leg ulcers are often a result of mixed venous and arterial disease, however, one pathology may predominate giving the findings below.

Venous ulceration

Venous hypertension causes fibrin to be laid down at the pericapillary cuff (lipodermatosclerosis), interfering with the delivery of nutrients to the surrounding tissues. There may be brown discolouration (haemosiderin deposition), eczema, telangiectasia and, eventually, ulcer formation with a base of granulation tissue and a serous exudate. Venous ulcers occur at the medial or lateral malleoli especially. These ulcers will often heal with time and care.

Arterial ulceration

Along with other symptoms and signs of leg ischaemia (📖 p.191), there may be loss of hair and toenail dystrophy. Chronic arterial insufficiency may lead to deep, sharply defined and painful ulcers which will not heal without intervention to restore blood supply. Arterial ulcers especially appear on the foot or mid-shin.

The elderly patient

Whilst the skin may be regarded as the largest organ of the body, it is sadly the one most often overlooked in any assessment of a patient. Many of the functional changes in aging skin make it increasingly susceptible to injury, with delayed resolution of wounds and consequent ↑ in infection risk. Systemic illnesses often manifest in skin and nail changes, and astute assessment can lead to resolve challenging diagnoses—e.g. erythema ab igne as a manifestation of hot water bottle use for abdominal pain and underlying pancreatic cancer or late onset icthyosis associated with lymphoma. For acutely unwell older people, being alert to the existence and development of pressure ulcers can significantly reduce pain, immobility and delays in their recovery.

History

- ***Symptoms:*** should be taken seriously. Whilst it is tempting to dismiss pruritus if there is no visible skin lesion, doing so risks missing a range of important diagnoses including iron deficiency anaemia and liver disease. Attributing symptoms due to age related changes in the skin should be a diagnosis of exclusion by generalists (and avoid the term 'senile' pruritus—older people find it offensive). Always remember that many systemic diseases may first manifest through skin changes.
- ***Pre-existing conditions:*** carefully documenting the presence (and treatment plan) for pressure ulcers is the obligation of both medical and nursing staff. Do not shirk this responsibility—it is important to plan pressure care as critically as any other intervention. You

should be particularly thorough in the presence of diabetes mellitus.

- ***DHx:*** important to ask about new changes in drugs and carefully document what an allergy or intolerance consists of—ring the patient's GP if needed. Consult the drug chart—watch for local reactions due to subcutaneous opiate infusions or skin necrosis due to low molecular weight heparins.
- ***Functional history:*** are overgrown toenails really a sign of self-neglect, or more likely poor vision, arthritis, poor hand grip, or neuropathy? Consider asking about diet—particularly in care home residents.

Examination

- ***General:*** an assessment of pressure areas is paramount—ask and look for sore heels too (and prescribe heel pads if needed). Is the skin frail, intact, marked, or broken. Asteatosis is extremely common, especially in states of dehydration. Prescribing emollients will earn the thanks of your patients (who may be uncomfortable and itching) and colleagues.
- ***Oedema:*** avoiding hurting your patient—palpate gently. Is it gravitational, are there signs of venous insufficiency or hypoalbuminaemia? Avoid rushing instantly to the diagnosis of heart failure.
- ***Gravitational eczema:*** often linked with oedematous change as above. Look out for pigmentatory change and ensure emollients are prescribed. For patients who may receive compression bandaging/hosiery—check peripheral pulses/ABPI carefully. Carefully describe any ulceration present.

- ECG ***stickers:*** if you perform an ECG—remove them immediately afterwards. Frail skin is easily torn and ulcerated when attempts at removal are made the next day, merely due to the thoughtlessness of the person recording the ECG.
- ***Subcutaneous fluids:*** are a key intervention in some unwell older people. Get into the habit of inspecting infusion sites, and be watchful for pooling and microabscesses.

Skin malignancies

- ***Common presentations:*** we all spend significant amounts of time examining and talking to our patients. Don't overlook the typical ulceration of a basal cell carcinoma around the eye/nasal region, or forget to refer to colleagues in dermatology. If you suspect a skin cancer, explore previous occupation or lifestyle.
- ***Atypical presentations:*** of common problems in atypical sites are legion—so be thoughtful, and carefully examine areas where patients might not look or be able to see (e.g. scalp, back, calves). Examine nails particularly carefully for signs of systemic disease or subungual melanoma. Be careful about rushing to a diagnosis of psoriasis in a new, isolated plaque. This is more likely to be Bowen's disease, so seek expert review.

We thank Dr Richard Fuller for providing this page.

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Title: *Oxford Handbook of Clinical Examination and Practical Skills, 1st Edition*

Chapter 5

The Endocrine System

Applied anatomy and physiology

The endocrine system is a complex, delicately balanced arrangement of hormonal feedback loops designed to co-ordinate organ functions. It maintains the internal environment (homeostasis), controls the storage and utilization of energy substrates, regulates growth and reproduction, and controls the organ responses to external stimuli.

The major glands that make up the human endocrine system are the hypothalamus, pituitary, thyroid, parathyroids, adrenals, pineal, and reproductive glands which include the ovaries and testes. The pancreas and the digestive system have endocrine components secreting insulin, glucagons, gastrin, and somatostatin.

The following is a very brief overview of those aspects of the endocrine system that may impact on the history and examination. Readers wanting more information on endocrine physiology are advised to seek an alternative source.

The hypothalamo-pituitary axis

The hypothalamus is a collection of specialized cells located in the lower central part of the brain and, along with the pituitary gland sitting just below the optic chiasm, forms the primary link between the endocrine and nervous systems. Neurons in the hypothalamus control the pituitary gland by producing chemical releasing factors that either ↑ or suppress hormone secretion.

This part of the brain is also important in the regulation of satiety, metabolism, and body temperature.

The releasing factors produced in the hypothalamus reach the pituitary via a short portal system running down the pituitary stalk (infundibulum). The pituitary gland is a pea-shaped structure lying in a bony walled cavity, the sella turcica, in the sphenoid bone at the base of the skull. It has an anterior lobe which develops from an outgrowth of ectoderm called the hypophyseal (Rathke's) pouch in the roof of the mouth and a posterior lobe that is directly linked to the hypothalamus.

The anterior pituitary hormones include

- *Growth hormone (*GH*):* stimulates general body growth and regulates aspects of metabolism.
- *Thyroid stimulating hormone (TSH):* controls the production of thyroid hormones by the thyroid gland.
- *Follicle stimulating hormone (*FSH*) and luteinizing hormone (*LH*):* together act on the secretion of oestrogen and progesterone from the ovaries, maturation of oocytes, and secretion of testosterone and production of spermatozoa in the testes.
- *Prolactin:* initiates milk production in mammary glands.
- *Adrenocorticotrophic hormone (*ACTH*):* stimulates the adrenal cortex to produce glucocorticoids.
- *Melanocyte stimulating hormone (MSH):* enhances skin pigmentation.

The posterior pituitary gland

Hormones released here are actually produced in the hypothalamus but travel down axons in the pituitary stalk to be stored ready for release.

They include anitdiuretic hormone (ADH) and oxytocin.

- *Oxytocin:* acts principally to stimulate contraction of smooth muscle cells in the uterus driving childbirth and around glandular cells of the mammary glands to cause milk ejection.
- ADH *(vasopressin):* is secreted by neurosecretory cells in the hypothalamus in response to ↑ blood osmotic pressure, dehydration, and loss of blood volume. It acts to conserve body water.

Thyroid

The thyroid gland is made up of the isthmus and 2 lateral lobes. The isthmus overlies the 2nd and 3rd rings of the trachea whilst the lobes extend from either side of the thyroid cartilage downward.

TSH stimulates the release of T_4 (thyroxine) and T_3 (triiodothyronine). T_3 is considered the 'active' hormone as it is about 2-4 times more potent than T_4 which can be considered as a 'pro-hormone'. Around 80% of circulating T_3 is derived from the deiodination (removal of

iodine) of T_4. This takes place in peripheral tissue—the remaining 20% is secreted directly by the thyroid gland. Most circulating T_3 and T_4 is bound to proteins including albumin and thyroid binding globulin (TBG).

The effects of thyroid hormones are multiple but include ↑ baseline metabolism, O_2 utilization, energy turnover, and thus heat production.

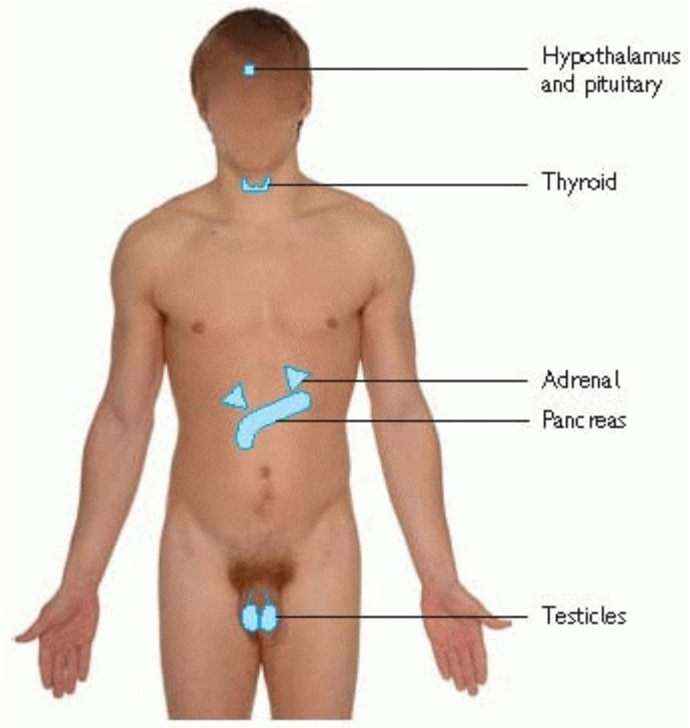

Fig. 5.1 The major endocrine glands of the body.

Presenting symptoms in endocrinology

As hormones have an impact on every body system, it is therefore necessary to cover all areas of general health in history-taking.

This section outlines some of the more important presenting symptoms in endocrine disease which should not be missed (if a high index of clinical suspicion is held regarding endocrine dysfunction), but it is by no means exhaustive.

Appetite and weight changes

Many people do not routinely weigh themselves but may have noticed the consequences of weight change—e.g. their clothes becoming looser or tighter.

Lethargy

Lethargy or fatigue is a difficult symptom to pin down. Ask the patient how the tiredness impacts on their daily life. What are they able to do before needing to rest—and has this changed?

Fatigue may be a feature of undiagnosed diabetes mellitus, Cushing's syndrome, hypoadrenalism, hypothyroidism, and hypercalcaemia.

Consider depression and chronic disease of any other kind (e.g. anaemia, chronic liver and renal problems, chronic infection, and malignancy).

Bowel habit

See p.230. Constipation is a common feature of hypercalcaemia and hypothyroidism. Hyperthyroidism and Addison's disease may give diarrhoea.

Urinary frequency and polyuria

See p.236. Common endocrine causes are diabetes mellitus and diabetes insipidus. Hyperglycaemia caused by Cushing's syndrome can also give polyuria. Polyuria may also be seen in the presence of hypercalcaemia.

Thirst and polydipsia

Consider diabetes mellitus, diabetes insipidus, and hypercalcaemia.

Sweating

↑ perspiration may be seen during episodes of hypoglycaemia as well as in hyperthyroidism and acromegaly, and is associated with the other adrenergic symptoms of a phaeochromocytoma.

Pigmentation

Localized loss of pigmentation may be due to vitiligo—an autoimmune disorder associated with other endocrine immune diseases such as hypoor hyperthyroidism, Addison's disease, and Hashimoto's thyroiditis.

- ↑ ***pigmentation:*** Addison's disease, Cushing's syndrome.
- ↓ ***pigmentation:*** generalized loss of pigmentation in hypopituitarism.

Hair distribution

Hirsuitism or excessive hair growth in a female may be due to endocrine dysfunction. Consider polycystic ovarian syndrome, Cushing's syndrome, congenital adrenal hyperplasia, acromegaly, and virilizing tumours.

Hypogonadism or adrenal insufficiency lead to ↓ adrenal androgen production and loss of axillary and pubic hair in both sexes.

Skin and soft tissue changes

Endocrine disorders cause many soft tissue changes including:

Hypothyroidism: dry, coarse, pale skin with xanthelasma formation and, classically, loss of the outer 1/3 of the eyebrows.

Hyperthyroidism: thyroid acropathy is seen only in hyperthyroidism due to Grave's disease. It is finger clubbing and new bone formation at the fingers. Also pretibial myxoedema—reddened oedematous lesions on the shins (often the lateral aspects).

Hypoparathyroidism: generally dry, scaly skin

Diabetes mellitus: xanthelasma, ulceration, repeated skin infections, necrobiosis lipoidica diabeticorum—shiny, yellowed lesions on the shins.

Acromegaly: soft tissue overgrowth with skin tags at the axillae and anus, 'doughy' hands and fingers, acanthosis nigricans—velvety black skin changes at the axilla. (Acanthosis nigricans can also be seen in Cushing's syndrome, polycystic ovarian syndrome and insulin resistance.)

Headache and visual disturbance

Visual field defects, cranial nerve palsies, and headache may be caused by space-occupying lesions within the skull. Pituitary tumours classically cause a bitemporal hemianopia by impinging on the optic chiasm (📖 p.294).

Blurred vision is rather non-specific, but consider osmotic changes in the lens due to hyperglycaemia.

Alteration in growth

Hypopituitarism, hypothyroidism, growth hormone deficiency, and steroid excess may present with short stature. Tall stature may be caused by growth hormone excess or gonadotrophin deficiency.

Growth hormone excess in adults (acromegaly) causes soft tissue overgrowth. Patients may notice an ↑ in shoe size, glove size, or facial appearance (do they have any old photographs for comparison?).

Changes in sexual function

Altered menstrual pattern in a female may be an early symptom suggestive of pituitary dysfunction. See 📖 Chapter 15 for more detail.

In men, hypogonadism may result in loss of libido and an inability to attain or sustain an erection (see 📖 Chapter 12). Remember to look for non-endocrine causes of sexual dysfunction such as alcoholism, spinal cord disease or psychological illness (see 📖 Chapters 12 and 15).

Flushing

Flushing may be a symptom of carcinoid or the menopause.

Ask about the nature of the flushing, any aggravating or relieving factors, and, importantly, any other symptoms at the time such as palpitations, diarrhoea, dizziness.

Remember to take a full menstrual history (see Chapters 15 and 16).

The rest of the history

A full history should be taken (Chapter 2). In a patient with endocrine symptoms, you should pay special attention to the following.

Drug history

As ever, a detailed medication history should be sought. Remember to ask especially about:

- Over-the-counter (OTC) medicines.
- Hormonal treatments—including the oral contraceptive pill, local, and systemic steroids.
- Amiodarone.
- Lithium.
- Herbal or other remedies.

Past medical history

- Any previous thyroid or parathyroid surgery.
- Any previous ^{131}I (radio-iodine) treatment or antithyroid drugs.
- Gestational diabetes.
- Hypertension.
- Any previous pituitary or adrenal surgery.

Family history

Ask especially about:

- Type II diabetes (Box 5.2).
- Related autoimmune disorders (pernicious anaemia, coeliac disease, vitiligo, Addison's disease, thyroid disease, type I diabetes).
  - Many patients will only have heard of these if they have a family member who suffers from them.
- Congential adrenal hyperplasia (CAH).
- Tumours of the MEN syndromes (Box 5.3).

Box 5.1 Weight, appetite, and endocrine disorders

↑ ***appetite***, ↓ ***weight:*** thyrotoxicosis, uncontrolled diabetes mellitus.
↑ ***appetite***, ↑ ***weight:*** Cushing's syndrome, hypoglycaemia, hypothalamic disease.
↓ ***appetite***, ↓ ***weight:*** gastrointestinal disease, malignancy, anorexia, Addison's disease, diabetes mellitus.
↓ ***appetite***, ↑ ***weight:*** hypothyroidism.

Box 5.2 The diabetic history

As with other diseases, you should establish when the diagnosis was made (and how) and the course and treatment of the disease. There are additional questions relating to disease monitoring and diabetic complications that you should ask patients with diabetes:

- When was it first diagnosed?
- How was it first diagnosed?
- How was it first managed?
- How is it managed now?
- If on insulin—when was that first started?

- Are they compliant with a diabetic diet?
- Are they compliant with their diabetic medication?
- How often do they check their blood sugar?
- What readings do they normally get (if possible, ask to see their monitoring booklet)?
- What is their latest Hb_A1_C (many will know this)?
- Have they ever been admitted to hospital with DKA?
- Do they go to a podiatrist or chiropodist?
- Have they experienced any problems with their feet? Do they use any moisturizers or cream on their feet?
- Do they attend a retinal screening program?
- Have they needed to be referred to an ophthalmologist?

In the newly diagnosed diabetic, ask about a history of weight loss (will differentiate type I and type II diabetes).

Box 5.3 The MEN syndromes

'Multiple endocrine neoplasias' which display autosomal dominant inheritance (see *OHCM6*, p.309).

- ***MEN 1:*** 'the 3 Ps.' Parathyroid hyperplasia (100%), pancreatic endocrine tumours (40-70%), pituitary adenomas (30-50%).
- ***MEN 2:*** medullary cell thyroid carcinoma (100%), phaeochromocytoma (50%) and …
  - ***MEN 2a:*** parathyroid hyperplasia (80%).
  - ***MEN 2b:*** mucosal and bowel neuromas, marfanoid habitus.

General examination

It is not possible to perform an examination of the endocrine system in the same way that you may examine other organ systems. Usually, an endocrine examination is focused—looking for signs to confirm or refute differential diagnoses that you have developed during history taking or examining the function of one or more specific glands (e.g. thyroid).

You may, however, perform a quick 'screening' general examination of a patient's endocrine status. Combine this with Chapter 3.

Hands/arms

Size, subcutaneous tissue, length of the metacarpals, nails, palmar erythema, sweating, tremor. Note also skin thickness (thin skin in Cushing's, thick skin in acromegaly) and look for signs of easy bruising.

Pulse and blood pressure—lying and standing. Test for proximal muscle weakness (p.326).

Axillae

Note any skin tags, loss of hair, abnormal pigmentation, or acanthosis nigricans.

Face and mouth

Look for hirsuitism, acne, plethora, or skin greasiness. Look at the soft tissues of the face for prominent glabellas (above the eyes) and enlargement of the chin (macrognathism). In the mouth, look at the spacing of the teeth and if any have fallen out. Note any buccal pigmentation and tongue enlargement (macroglossia). Normally, the upper teeth close in front of the lower set—reversal of this is termed 'prognathism'.

Eyes and fundi

See p.118 and p.122 respectively.

Neck

Note any swellings or lymphadenopathy (p.68). Examine the thyroid. Palpate the supraclavicular regions and note excessive soft tissue.

Chest

Inspect for any hair excess or loss, breast size in females and gynaecomastia in males. Note the nipple colour, pigmentation, or galactorrhoea.

Abdomen

Inspect for central adiposity/obesity, purple striae, hirsuitism. Palpate for organomegaly. Look at the external genitalia to exclude any testicular atrophy in males or virilization (e.g. clitoromegaly) in women.

Legs

Test for proximal muscle weakness (p.328) and make note of any diabetes-related changes (p.120).

Height and weight

Calculate the patient's BMI (p.66).

Box 5.4 Signs of tetany

Trousseau's sign

Inflate a blood pressure cuff just above the systolic pressure for 3 minutes. When hypocalcaemia has caused muscular irritability, the hand will develop flexor spasm.

Chvostek's sign

Gently tap over the facial nerve (in front of the tragus of the ear). The sign is positive if there is contraction of the lip and facial muscles on the same side of the face.

Examining the thyroid

The patient should be sitting upright on a chair or the edge of a bed.

Inspection

Look at the thyroid region. If the gland is quite enlarged (goitre), you may notice it protruding as a swelling just below the thyroid cartilage. The normal thyroid gland is usually neither visible nor palpable.

Thyroid gland

The gland lies ~2-3cm below the thyroid cartilage and has 2 equal lobes connected by a narrow isthmus.

If a localized or generalized swelling is visible, ask the patient to take a mouthful of water then swallow—watch the neck swelling carefully. Also ask the patient to protrude their tongue and watch the neck swelling.

- The thyroid is attached to the thyroid cartilage of the larynx and will move up with swallowing.
- Other neck masses such as an enlarged lymph node will hardly move.
- Thyroglossal cysts will not move with swallowing but will move upwards with protrusion of the tongue.

The rest of the neck

- Carefully inspect the neck for any obvious scars (thyroidectomy scars are often hidden below a necklace and are easily missed).
- Look for the JVP and make note of dilated veins which may indicate retrosternal extension of a goitre.
- Redness or erythema may indicate suppurative thyroiditis.

Palpation

Thyroid gland

Always begin palpation from behind. Stand behind the patient and place a hand either side of their neck. The patient's neck should be slightly flexed to relax the sternomastoids. Explain what you are doing.

- Ask if there is any tenderness.
- Place the middle 3 fingers of either hand along the midline of the neck, just below the chin.

- Gently ‘walk’ your fingers down until you reach the thyroid gland.
  - The central isthmus is almost never palpable.
- If the gland is enlarged, determine if it is symmetrical.
- Are there any discrete nodules?
- Assess the size, shape, and mobility of any swelling.
- Repeat the examination whilst the patient swallows.
  - Ask them to hold a small amount of water in their mouth—then ask them to swallow once your hands are in position.
- Consider the consistency of any palpable thyroid tissue.
  - Soft: normal.
  - Firm: simple goitre.
  - Rubbery hard: Hashimoto's thyroiditis.
  - Stony hard: cancer, cystic calcification, fibrosis, Riedel's thyroiditis.
- Feel for a palpable thrill which may be present in metabolically active thyrotoxicosis.

The rest of the neck

Palpate cervical lymph nodes, carotid arteries (to check for patency—can be compressed by a large thyroid) and the trachea for deviation.

Percussion

- Percuss downwards from the sternal notch.
- In retrosternal enlargement the percussion note over the manubrosternum is dull as opposed to the normal resonance.

Auscultation

Apply the diaphragm of the stethoscope over each lobe of the thyroid gland and auscultate for a bruit.

- A soft bruit is indicative of ↑ blood flow which is characteristic of the hyperthyroid goitre seen in Grave's disease.
  - You may need to occlude venous return within the IJV to rule out a venous hum.
  - Listen over the aortic area to ensure that the thyroid bruit is not, in fact, an outflow obstruction murmur conducted to the root of the neck.

Box 5.5 Pemberton's sign

A test for thoracic inlet obstruction (e.g. retrosternal goitre).

- Ask the patient to raise both arms above the head.
  - Patients with inlet obstruction may develop signs of venous compression (facial plethora, cyanosis, dizziness, syncope).
  - Look at the neck veins for congestion and listen for stridor.

Box 5.6 Assessing thyroid status: examination

- Observe the patient's composure (relaxed/agitated/fidgety?).
- Measure the heart rate and note if the patient is in atrial fibrillation.
- Inspect the hands—erythema, warmth, thyroid acropachy (phalangeal bone overgrowth similar to pulmonary osteopathy).
- Feel the palms—sweaty/dry?
- Look for peripheral tremor—ask the patient to stretch out their arms with fingers out straight and palms down. Resting a piece of paper on the back of the hand can make a tremor more obvious.
- Inspect the face.
  - Exophthalmos, proptosis (📖 p.118).

- Hypothyroid features (p.126).

- Examine the eyes (p.118).
- Examine the thyroid and neck (this topic).
- Test tendon reflexes at the biceps and ankle (p.330).
- Test for proximal myopathy by asking the patient to stand from a sitting position.
- Look for pretibial myxoedema.

Eye signs in thyroid disease

Examination

Inspection

- Look at the patient's eyes from the front, side, and from above.
- Note whether the sclera is visible above or below the iris and whether the eyeball appears to sit forward (proptosis—best seen from above).
- Note the health of the conjunctiva and sclera looking especially for any ulceration or conjunctivitis.
- Ensure both eyes can close (failure is a medical emergency).

Visual fields

It is wise to perform a quick screening test of the visual fields (p.292).

Eye movements

Test eye movements in all directions (p.304).

Lid lag (von Graefe's sign)

- Hold your finger high and ask the patient to look at it and follow it with their eyes as it moves (keeping their head still).
- Quickly move your hand downwards—in this way the patient is made to look upwards and then quickly downwards.
- Watch the eyes and eyelids—do they move smoothly and together?
  - If lid lag is present, the upper eyelid seems to lag behind the movement of the eye, allowing white sclera to be seen above the iris as the eye moves downward.

Findings

Proptosis

- Protrusion of the globes as a result of an ↑ in retro-orbital fat, oedema, and cellular infiltration.
- It can be formally assessed using ‘Hertel's exophthalmometer’.

Exophthalmos

This is more severe form of proptosis. Sclera becomes visible below the lower edge of the iris (the inferior limbus). In very severe cases, the patient may not be able to close their eyelids and can develop:

- Corneal ulceration.
- Chemosis (oedema of the conjunctiva and sclera caused by obstruction of the normal venous and lymphatic drainage).

- Conjunctivitis.

Lid retraction

The upper eyelid is retracted such that you are able to see white sclera above the iris when the patient looks forwards.

Caused by ↑ tone and spasm of levator palpebrae superioris as a result of thyroid hormone excess (Dalrymple's sign).

Lid lag

Described above. Caused by sympathetic overstimulation of the muscles supplying the upper eyelid—seen in thyroid hormone excess.

Box 5.7 Eye signs of thyrotoxicosis and Grave's disease

A common misconception is that proptosis and exophthalmos are caused by thyrotoxicosis. This is not the case. Proptosis and exophthalmos may be seen in 50% of patients with Grave's disease … and thyrotoxicosis may occur in Grave's disease. However, the proptosis may persist once thyroid hormone levels have been normalized.

The eye signs of thyrotoxicosis are:

- Lid retraction.
- Lid lag.

The eye signs of Grave's disease (Grave's ophthalmopathy) are:

- Periorbital oedema and chemosis.
- Proptosis/exophthalmos.
- Ophthalmoplegias (particularly of upward gaze).
- Lid retraction and lid lag *only* when thyrotoxicosis is present.

Visual blurring may indicate optic neuropathy, therefore, fundoscopy (📖 p.296) should be performed.

Examining the patient with diabetes

As diabetes has an impact on every body system, you can make the examination of a diabetic patient complex or simple depending on the circumstance.

In diabetes clinics, a quick screening examination is performed looking for major complications—particularly those involving the feet.

In general, you should be alert to: cardiovascular disease, renal disease, retinal disease, peripheral neuropathy—especially sensory, health of insulin injection sites, the diabetic foot, secondary causes of diabetes (e.g. acromegaly, Cushing's syndrome, haemochromatosis), and associated hyperlipidaemia.

Box 5.8 Important points for a thorough diabetic examination

Inspection

Hydration, weight, facies associated with a known endocrine disease, pigmentation (hyperpigmentation or patchy loss).

Legs

Muscle wasting, hair loss, skin atrophy, skin pigmentation, leg ulceration (especially around pressure points and toes), skin infections.

Injection sites

Inspect and palpate for fat atrophy, fat hypertrophy, or local infection.

Associated skin lesions

Necrobiosis lipoidica diabeticorum—look on the shins, arms and back. Sharply demarcated oval plaques with a shiny surface, yellow waxy atrophic centres and brownish-red margins with surrounding telangiectasia. Also look for granuloma annulare.

Hyperlipidaemia

Eruptive xanthoma, tendon xanthoma, xanthelasma.

Neurological examination

Visual acuity, fundoscopy, peripheral sensory neuropathy—evidence of injury, ulceration, and Charcot joint formation. Test muscle strength and examine feet.

Cardiovascular examination

Ideally a full cardiovascular examination including lying and standing blood pressure measurements.

The diabetic foot

The combination of peripheral vascular disease and peripheral neuropathy can lead to repeated minor trauma to the feet leading to ulceration and infection which are very slow to heal. Chronic infection and other ‘foot complications’ are a major cause of morbidity and mortality in the diabetic patient.

Box 5.9 Framework for the diabetic foot examination

Inspection

- Colour.
- Ulceration.
- Dryness.
- Callous formation.
- Infection.
- Evidence of injury—shoes rubbing?
- Charcot's joints (grossly abnormal and dysfunctional joints due to repeated minor trauma and poor healing due to a loss of pain sensation).

Neurology

- 10g monofilament test (see below).
- Light touch sensation, pain sensation, vibration sense, and joint position sense (proprioception).

Circulation

- Peripheral pulses (dorsalis pedis and posterior tibial).
- Temperature.
- Capillary filling time.

Using a 10g monofilament

Small, thin plastic filaments are used for testing peripheral sensation in the diabetic foot. They are designed such that it bends under approximately 10g of pressure.

- Apply the filament to the patient's skin at the spots shown in Fig. 5.1.
- Press firmly so that the filament bends (Fig. 5.2b).
- Hold the filament against the skin for ~1.5 seconds and ask the patient if they can feel it.
- The filament should not slide, stroke, or scratch the skin.
- Do not press on ulcers, callous, scars, or necrotic tissue.
  - The patient's feet are ‘at risk’ if they cannot feel the monofilament at any of the sites.

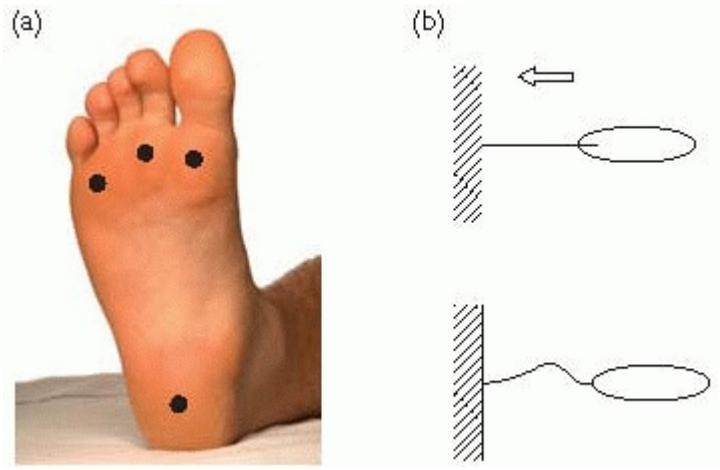

Fig. 5.2 (a) Sites to test with a 10g monofilament in the diabetic patient. (b) Apply the monofilament to the skin with enough force to make it bend.

The fundus in endocrine disease

Diabetes mellitus

This is the most common cause of blind registration between ages 30-65 in the UK. Early diagnosis and treatment of diabetic retinopathy can eliminate >95% of diabetic blindness. For this reason it is essential for all diabetics to undergo regular eye examination.

Mechanisms of damage

The precise metabolic mechanisms underlying the retinal changes seen in diabetes are still unclear. There may be a role for aldose reductase, the enzyme responsible for the conversion of glucose to sorbitol. High levels of sorbitol are found in the lens, pericytes, and Schwann cells of diabetic patients and are thought to lead to cell damage. A great deal of the damage may be caused by the release of vascular endothelial derived growth factor (VEGF) in response to retinal ischaemia.

The changes seen in the fundus of diabetic patients arise due to common microvascular lesions. These include:

- Microaneurysms.
- Haemorrhages—dot and blot.
- Hard exudates—lipid precipitated out of the plasma.
- Cotton wool spots—represent ischaemia and occur due to interruption of axoplasmic flow in the nerve fibre layer.
- Intraretinal microvascular abnormalities (IRMA).
- Venous beading.
- Neovascularization.

Classification of diabetic retinopathy

A simple classification system exists and is detailed below:

Background diabetic retinopathy

Microaneurysms, hard exudates, and haemorrhages. May be extensive and widespread in severe disease.

Pre-proliferative retinopathy

Ischaemia is evinced by cotton wool spots, venous beading may also be present.

Proliferative

New retinal vessel formation. This may progress to vitreous bleeding, traction, retinal detachment and blindness.

Maculopathy

Pathology affecting the macula causes catastrophic visual loss:

- Exudates and haemorrhages in the macular area.
- ▸ The patient may have reduced visual acuity with no abnormality seen on fundoscopy.

We thank Dr Tom Fearnley for contributing this page.

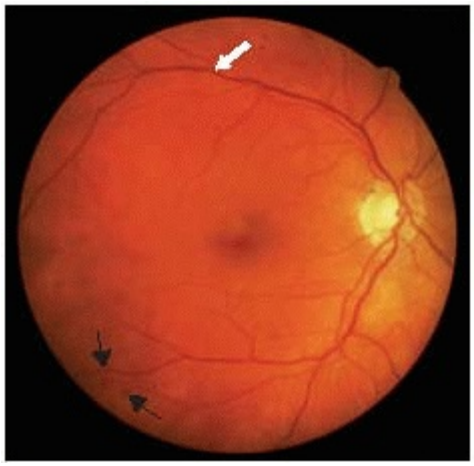

Fig. 5.3 Retinal photograph showing background diabetic retinopathy. White arrow shows a micro-aneurysms, black arrows show haemorrhages.

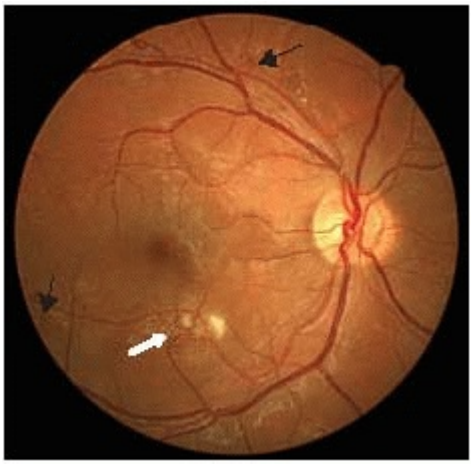

Fig. 5.4 Retinal photograph showing proliferative diabetic retinopathy. White arrow shows new vessels growing into an ischaemic area (cotton wool spot). You can also see some dot haemorrhages (black arrows).

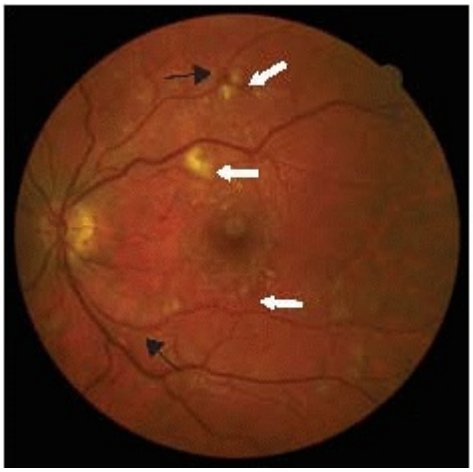

Fig. 5.5 Retinal photograph showing diabetic maculopathy. Thin white arrows show hard exudates, black arrows show haemorrhages —both within the macula. You can see new vessels growing into the macula (thick white arrow).

Further ocular manifestations of diabetes

Whilst a great deal of focus is placed on the retinal changes seen in diabetes it is also worth noting that diabetic patients are also predisposed to a number of other sight threatening conditions such as:

Glaucoma

- Open angle.
- Neovascular secondary to rubeosis iridis—new vessel formation on the iris and interruption of the drainage angle.

Cataract

Treatment of this can be very difficult and in general it is necessary to treat diabetic retinopathy first before contemplating cataract surgery.

Optic neuropathy

- Acute ischaemic optic neuritis.
- Diabetic optic neuropathy.

Cranial nerve palsy

See cranial nerve examination in Chapter 12 for the common palsies associated with diabetes.

Box 5.9 Avoiding visual loss in diabetes

Management of the diabetic eye requires a multidisciplinary approach involving the GP, diabetes physician, Diabetes Centre staff, optician, ophthalmologist, and not least of all the patient.

Ocular examination at the time of diagnosis and yearly screening thereafter coupled with tight control of weight, blood pressure, cholesterol, and blood glucose can help to avoid the devastating consequences of diabetic eye disease.

Hypertensive retinopathy

Classified into mild, moderate, and severe forms to better correlate with the duration of systemic hypertension and associated risk of coronary artery and cerebrovascular disease.

Appearance—mild

- Generalized or focal arteriolar narrowing of the retinal arterioles.
- Opacity of the retinal artery walls—so called silver/copper wiring.
- Arterio-venous (A-V) nipping—the retinal arteries cross the veins at a more perpendicular angle and impinge upon the surface of the vein.

Appearance—moderate

- Retinal haemorrhage.
- Cotton wool spots—small areas of ischaemia with resulting disruption of axoplasmic flow in the nerve fibre layer of the retina.
- Hard exudates—lipid exudates.
- Microaneurysms.

Appearance—severe

All the above plus optic disc swelling.

We thank Dr Tom Fearnley for contributing this page.

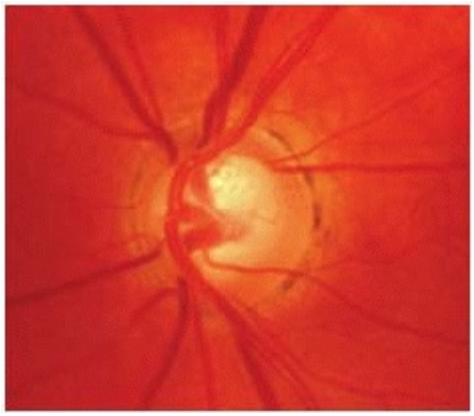

Fig. 5.6 Retinal photograph showing close-up of the optic disc in glaucoma. Note how the disc is sunken—the vessels appearing to disappear into it (left of picture).

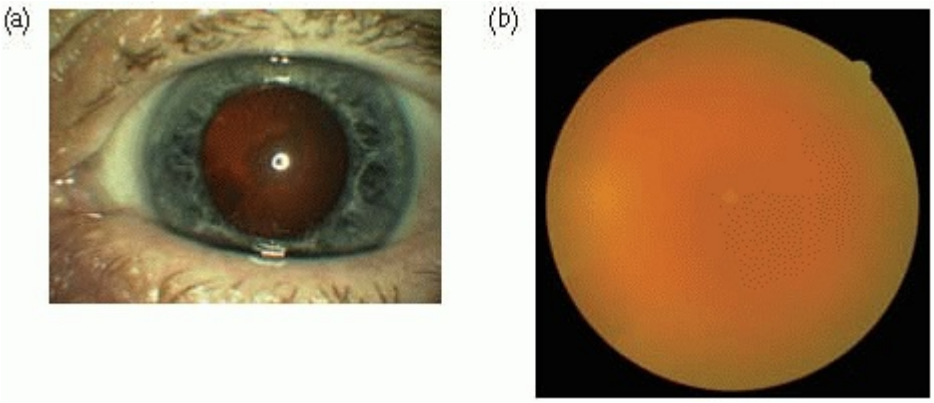

Fig. 5.7 Cataract. (a) External appearance. (b) Fundoscopy becomes very difficult or impossible.

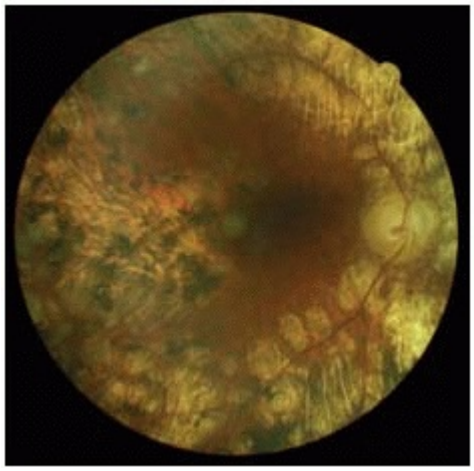

Fig. 5.8 Retinal photograph showing proliferative diabetic retinopathy treated with laser therapy. Note the multiple rounded scars on the retina.

Important presenting patterns

Hypothyroidism (📖 OHCM6, p.306)

Causes: dietary iodine deficiency, autoimmune thyroiditis (Hashimoto's thyroiditis), lymphocytic thyroiditis (10% of post-partum women), drugs (amiodarone, interferon alpha, thalidomide, dopamine, lithium), radioactive iodine treatment, surgical thyroid injury, external irradiation (e.g. for head and neck neoplasms or breast cancer), pituitary adenoma.

Symptoms: tiredness, weight gain, anorexia, cold intolerance, poor memory, depression, ↓ libido, goitre, puffy eyes, brittle hair, dry skin, arthralgia, myalgia, muscle weakness, constipation, menorrhagia.

Signs: croaking voice, mental and physical sluggishness, pseudodementia 'myxoedema madness'.

- *Inspection:* coarse cool dry skin (look for yellowish tint of carotennaemia 'peaches and cream' complexion), palmar crease pallor, peripheral cyanosis, puffy lower eyelids, loss of outer 1/3 of eyebrows, thinning of scalp hair, tongue swelling, xanthalasma,
- *Cardiovascular and chest:* mild hypertension, pericarditis, pleural effusion, low cardiac output cardiac failure, bradycardia, small volume pulse.
- *Neurological:* carpal tunnel syndrome, peripheral neuropathy, cerebellar syndrome, proximal muscle weakness, myotonia, muscular hypertrophy, delayed ankle jerks, bilateral neural deafness (seen in congenital hypothyroidism).

Hyperthyroidism (📖 OHCM6, p.304)

Causes: Graves disease, chronic thyroiditis (Hashimoto thyroiditis), subacute thyroiditis (de Quervain thyroiditis), postpartum thyroiditis, drugs (iodine-induced, amiodarone), bacterial thyroiditis, postviral thyroiditis, idiopathic, toxic multinodular goitre, malignancy (toxic adenoma, TSH-producing pituitary tumours).

Symptoms: weight loss, ↑ appetite, irritability, restlessness, muscle weakness, tremor, breathlessness, palpitations, sweating, heat intolerance, itching, thirst, vomiting, diarrhoea, eye complaints (Graves's ophthalmopathy), oligomenorrhoea, loss of libido, gynaecomastia.

Signs: irritability, weight loss.

- *Inspection:* onycholysis, palmar erythema, tremor, sweaty palms, thyroid acropachy, hyperkinesis, gynaecomastia, pretibial myxoedema, Grave's ophthalmopathy.
- *Cardiovascular and chest:* resting tachycardia, high cardiac output, systolic flow murmurs.
- *Neurological:* proximal myopathy, muscle wasting, hyper-reflexia in legs.

Polycystic ovarian syndrome (PCOS)

Abnormal metabolism of androgens and oestrogen with abnormal control of androgen production.

Symptoms: oligomenorrhoea with anovulation and erratic periods, infertility. Some patients present complaining of hirsuitism.

Signs: obesity (50%), male-pattern hair growth, male-pattern baldness, increased muscle mass, deep voice, clitoromegaly, acanthosis nigricans.

Glucocorticoid excess (Cushing's syndrome 📖 OHCM6, p.310)

Causes include: high ACTH production from a pituitary adenoma and ectopic ACTH (e.g. small cell lung cancer). Primary hypercortisolaemia caused by adrenal hyperplasia, adrenal tumour (adenoma or carcinoma), exogenous steroids; ectopic CRF production (very rare), depression, alcohol-induced.

Symptoms: weight gain (central/upper body), change in appearance, menstrual disturbance, thin skin with easy bruising, acne, excessive hair growth, muscle weakness, ↓ libido, depression, insomnia.

Signs: supraclavicular fat pads, 'moon face', thoracocervical fat pads ('buffalo hump'), centripetal obesity, hirsutism, thinning of skin, easy bruising, purple striae, poor wound healing, skin infections, proximal muscle weakness (shoulders and hips), ankle oedema, hypertension, fractures due to osteoporosis, hyperpigmentation (if raised ACTH), glycosuria.

Hypoadrenalism (Addison's 📖 OHCM6, p.312)

Causes include: autoimmune adrenalitis (>80% in UK), tuberculosis, metastatic malignancy, amyloidosis, haemorrhage, infarction, bilateral adrenalectomy, HIV.

Symptoms: anorexia, weight loss, tiredness, nausea, vomiting, diarrhoea, constipation, abdominal pain, confusion, erectile dysfunction, amenorrhoea, dizziness, syncope, myalgia, arthralgia.

Signs: skin pigmentation (especially on sun-exposed areas, mucosal surfaces, axillae, palmar creases and in recent scars), cachexia, loss of body hair, postural hypotension, low grade fever, dehydration.

Growth hormone excess (Acromegaly 📖 OHCM6, p.324)

Causes: pituitary tumour (>95%), hyperplasia due to GHRH excess (very rare), tumours in hypothalamus, adrenal or pancreas.

Symptoms: headache, diplopia, change in appearance, enlarged extremities, deepening of voice, sweating, tiredness, weight gain, erectile dysfunction, dysmennorrhoea, galactorrhoea, snoring, arthralgia, weakness, numbness, paraesthesia, polyuria, polydipsia.

Signs: prominent supraorbital ridges, large nose and lips, protrusion of lower jaw (prognathism), interdental separation, macroglossia, 'spade-like' hands, 'doughy' soft tissues, thick oily skin, carpal tunnel syndrome, hirsutism, bitemporal hemianopia (if pituitary tumour impinging on optic chiasm), cranial nerve palsies (particularly III, IV, and VI), hypertension.

Prolactinoma

A pituitary tumour (the most common hormone-secreting tumour).

Symptoms: depend on age, sex, and degree of prolactinaemia. In females: oligomenorrhagia, vaginal dryness, dyspareunia, galactorrhoea. In males: ↓ libido, erectile dysfunction, infertility, galactorrhoea. If before pubery in the male, may have female body habitus and small testicles.

Signs: visual field defects (?bitemporal hemianopia), cranial nerve palsies (III, IV, and VI), galactorrhoea. In males: small testicles and female pattern of hair growth.

Hypercalcaemia

Causes: common—hyperparathyroidism, malignancy (PTHrp production or metastases in bone). *Less common*—vitamin D intoxication, granulomatous disease, familial hypocalciuric hypercalaemia. Rare—drugs (e.g. bendrofluazide), hyperthyroidism, Addison's disease.

Symptoms: depend largely on the underlying cause. Mild hypercalcaemia is asymptommatic. Higher levels may cause nausea, vomiting, drowsiness, confusion, abdominal pain, constipation, depression, muscle weakness, myalgia, polyuria, headache, and coma.

Signs: often there are signs of the underlying cause. There are no specific signs of hypercalcaemia.

Hypocalcaemia

Causes: hypoalbuminaemia, hypomagnesaemia, hyperphosphataemia, surgery to the thyroid or parathyroid glands, PTH deficiency or resistance, and vitamin D deficiency.

Symptoms: depression, paraesthesia around the mouth, muscle spasms.

Signs: carpopedal spasm (flexion at the wrist and the fingers) when blood supply to the hand is reduced by inflating a sphygmomanometer cuff on the arm (*Trousseau's sign*). Nervous excitability—tapping a nerve cause the supplied muscles to twitch (*Chvostek's sign*—tapping facial nerve at the parotid gland about 2cm anterior to the tragus of the ear causes the facial muscles to contract).

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Chapter 7

The Cardiovascular System

Applied anatomy and physiology

Cardiologists consider this to be the most important system in the body.

This system is fundamentally rather straightforward and a good deal of information about its functioning can be gleaned from physical examination. The basic anatomy of the cardiovascular system should be familiar to readers. Below is a summary of some points which have particular implications for the clinical assessment.

The heart

The heart rotates anticlockwise during embryonic development, finally settling such that the left ventricle lies almost entirely posteriorly and the right anteriorly—the whole seeming to hang in the chest, held by the aorta ('aorta' comes from the Greek 'aorte' meaning 'to suspend').

The myocardium is arranged in a complex spiral such that a contraction causes the heart to elongate and rotate slightly, hitting the anterior chest wall as it does—this can be felt as the apex beat.

All this movement is lubricated by a double-lined cavity filled with a very small amount of fluid that the heart sits in—the pericardial sac.

Heart sounds

As the ventricles contract, the tricuspid and mitral valves close, heard as the 1st heart sound. As the ventricles relax, intraventricular pressure drops and blood expelled into the great vessels begins to fall back, the aortic and pulmonary valves slam closed—this is heard as the 2nd heart sound. The sounds are often described as sounding like *'lub dub'*.

As each heart sound is, in fact, *two* valves closing, any mistiming will cause a double or 'split' heart sound as one valve closes shortly after the other. A split 2nd heart sound is normal in young adults and children. During inspiration, the intrathoracic pressure drops, drawing blood into the chest, ↑ delivery to the right side of the heart, and ↓ delivery to the left as it pools in the pulmonary veins. Consequently, the stroke volume will be greater on the right than the left and the right ventricular contraction will take slightly longer. Thus, the pulmonary valve will close very slightly later than the aortic valve, producing the split 2nd sound. (*'lub da-dub'*). This is 'physiological splitting'. Split heart sounds are considered on p.181.

Jugular venous pulse (JVP)

There is no valve between the right heart and the large vessels supplying it. Thus, filling and contraction of the right atrium will cause a pressure wave to travel back through the feeding veins. This can actually be seen in the neck at the internal jugular vein. See p.174.

Arteries

As the ventricle expels blood into the arteries, it sends a pulse wave to the periphery which can be felt. This is *not* the actual flow of blood from the ventricle at that contraction but a pressure wave. The shape and feel of the wave can be altered by the force of expulsion, any obstacles (such as the aortic valve) and the state of the peripheral vasculature.

The arteries have their own intrinsic elasticity, allowing a base-line, or diastolic, pressure to be maintained between each pulse wave.

Veins

Blood flows at a much lower pressure in the veins.

Above the level of the heart, gravity does most of the work in returning the blood. Below, blood return is facilitated by contraction of muscles surrounding the deep veins, helped by numerous one-way valves to prevent backflow. Blood moves initially from the surface to the deep veins before moving upwards, again mediated by one-way valves (if these valves become damaged, blood flows outward to the surface veins causing them to swell and look unsightly—'varicose veins')

Blood return is also aided by a negative pressure created by blood being pumped out of the right ventricle—and therefore drawn in through the right atrium at each beat.

Chest pain

This is the most common—and most important—cardiovascular symptom. Patients who mention it may be surprised to find themselves whisked away for an ECG before they can say any more. It is, however, usually possible to determine the probable cause of the pain with a detailed history.

As for any other type of pain, the history must include the:

- Nature (crushing, burning, aching, stabbing etc.).
- Exact location.
- Any radiation.
- Severity (scored out of 10).
- Mode and rate of onset. What was the patient doing at the time?
- Change in the pain over time (and current score out of 10).
- Duration (if now resolved).
- Exacerbating factors (particularly, is it affected by respiration or movement?).
- Relieving factors (including the use of GTN).
- Associated symptoms (nausea, vomiting, sweating, belching etc).

Patients with a history of cardiac pain can also usually tell you whether the pain experienced is the same as, or different to, their 'usual' angina.

Angina

Full name 'angina pectoris', this is the pain caused by myocardial ischaemia and the build-up of toxic products of respiration in the muscle. This is usually due to coronary artery disease but can also be caused by other cardiac diseases such as aortic stenosis or hypertrophic cardiomyopathy.

'Angina' comes from the Latin for 'choking' and this is often what the patient describes. As the brain cannot interpret pain from the heart *per* se, it is felt over the central part of the anterior chest and can radiate up to the jaw, shoulder, or down the arms or even to the umbilicus. This pattern is due to the common embryological origins of the heart and these parts of the body. Indeed, some patients may experience angina pain *only* in the arm, for example.

The 'pain' of angina is usually an unfamiliar sensation, consequently, patients may be more comfortable with the term 'discomfort'.

In true angina, you can expect the following features:

- Retrosternal.
- 'Crushing', 'heaviness' or 'like a tight band'.
- Worse with physical or emotional exertion, cold weather and after eating.
- Relieved by rest and nitrate spray (within a couple of minutes).
- Not affected by respiration or movement.
- Sometimes associated with breathlessness.

In addition, patients classically clench their right fist and hold it to their chest when describing the pain.

In patients with known angina, a change in the nature of the symptom is important. Ask them how much exercise they can do before feeling the discomfort and whether this has changed.

Myocardial infarction

Patients will know this as a 'heart attack'. The pain is similar to that of angina but much more severe, persistent (despite GTN spray) and associated with nausea, sweating, and vomiting. Patients may also describe a feeling of impending doom or death—'angor animi'.

Pericarditis

The commonest causes are viral or bacterial infection, MI, or uraemia.

- Constant retrosternal 'soreness'.
- Worse on inspiration (pleuritic).
- Relieved slightly by sitting forwards.
- Not related to movement or exertion.

Oesophageal spasm

Often mistaken for MI or angina.

- A severe, retrosternal burning pain.
- Onset often after eating or drinking.
- May be associated with dysphagia.
- May have a history of dyspepsia.
- May be relieved by GTN as this is a smooth muscle relaxant (hence the confusion with angina) but GTN will take up to 20 minutes to relieve this pain whereas angina is relieved within a few minutes.

Gastro-oesophageal reflux disease ('heartburn')

- Retrosternal, burning pain.
- Relieved by antacids, onset after eating.

Dissecting aortic aneurysm

Must be differentiated from an MI as thrombolysis here may prove fatal.

- Severe 'tearing' pain.
- Felt posteriorly—classically between the shoulderblades.
- Persistent, most severe at onset.
- Patient is usually hypertensive and 'marfanoid' (see p.78).

Pleuritic (respiratory) pain

This is covered in more detail in Chapter 8. May be caused by a wide range of respiratory conditions, particularly pulmonary embolus and pneumothorax.

- Sharp pain, worse on inspiration and coughing.
- Not central—may be localized to one side of the chest.
- No radiation.
- No relief with GTN.
- Associated with breathlessness, cyanosis etc.

Musculoskeletal pain

May be caused by injury, fracture, chondritis, etc. Will be localized to a particular spot on the chest and worsened by movement and respiration. May be tender to palpation.

'Tietze's syndrome' is costochondritis (inflammation of the costal cartilages) at ribs 2, 3, and 4. Will be associated with tender swelling over the costo-sternal joints.

Breathlessness and oedema

Breathlessness and oedema are presented together here as, usually, they are linked pathophysiologically in the cardiovascular patient.

Excess tissue fluid caused by a failing heart will settle where gravity pulls it. In someone who is on their feet, it will settle in their ankles causing swelling. If the patient is bed bound, the swelling will occur about their sacrum and if the patient is lying down, fluid will collect on their lungs (pulmonary oedema) causing breathlessness.

Dyspnoea (breathlessness)

'Dyspnoea' is an abnormal awareness of one's breathing and is described in detail in Chapter 8. There are certain aspects of breathlessness that you should ask of the cardiovascular patient in particular.

As with everything, you must *quantify* the symptom if you are able, so as to gauge its severity and as a baseline so that the effects of treatment or disease progression can be monitored. The New York Heart Association (NYHA) has devised a classification of breathlessness which is shown in Box 7.1. In practice, this is only used in clinical trials and it makes more sense to measure the functional result of breathlessness. Ask especially:

- How far can the patient walk on the flat before they have to stop ('march tolerance')?
- What about stairs and hills—can they make it up a flight?
- Are they sure that they stop due to breathlessness or is it some other reason (arthritic knees for example)?
- Has the patient had to curtail their normal activities in any way?

Orthopnoea

This is breathlessness when lying flat. Patients will not usually volunteer this as a symptom so ask them:

- How many pillows does the patient sleep with and has this changed?
  - Some patients may describe having to sleep sitting upright in a chair.
- If the patient sleeps with a number of pillows, ask why. Are they breathless when they lie down or is it for some other reason?

Paroxysmal nocturnal dyspnoea

As the name suggests, this is episodes of breathlessness occurring at night—usually thought to be due to pulmonary oedema. Again, patients won't usually volunteer this information and will often react with surprised pleasure when you ask them about it. Sufferers will experience waking in the night spluttering and coughing—they find they have to sit up or stand and many go to the window for 'fresh air' in an attempt to regain their normal breathing.

- Do they wake up in the night coughing and trying to catch their breath?
- If so, glean as much detail as you can—including how often and how badly the symptom is disturbing the patient's sleep cycle.

Cough

Pulmonary oedema may cause a cough productive of frothy white sputum. This may be flecked with blood ('pink') due to ruptured bronchial vessels but this is not usually a worrying sign in itself.

Ankle oedema

As already mentioned, in ambulant patients, fluid will collect at the ankles and cause swelling. It is often surprising just how severe the swelling can get before people seek medical attention. Ask:

- How long has this been going on for?
- Is it worse at any particular time of day? (Typically cardiac oedema is worse toward the evening and resolved somewhat overnight as the oedema redistributes itself.)
- Exactly how extensive is the swelling? Is it confined to the feet and ankles or does it extend to the shin, knee, thigh, or even the buttocks, genitalia, and anterior abdominal wall?
- Is there any evidence of abdominal swelling and ascites? (p.260).

Box 7.1 NYHA classification of breathlessness

- I—nil at rest, some on vigorous exercise.
- II—nil at rest, breathless on moderate exertion.
- III—mild breathlessness at rest, worse on mild exertion.
- IV—significant breathlessness at rest and worse on even slight exertion (the patient is often bed-bound).

Palpitations

To have palpitations is to have an awareness of one's own heart beat. This is one of the many situations in which the patient may have a very different idea of the word's meaning than you. You should spend some time teasing out exactly what they mean. Patients may be unfamiliar with the term altogether and, instead, describe the heart ‘jumping’ or ‘missing a beat’. Attempt to determine:

- When did the sensation start and stop?
- How long did it last?
- Did it come on suddenly or gradually?
- Did the patient blackout? If so, for how long?
- Was the heart beat felt as fast, slow, or some other pattern?
- Was it regular or irregular?
    - It is useful at this stage to ask the patient to tap out what they felt on their knee or a nearby table.
- What was the patient doing when the palpitations started?
- Is there any relationship to eating or drinking (particularly tea, coffee, wine, chocolate)?
- Could it have been precipitated or terminated by any medication?
- Has this ever happened before? If so, what were the circumstances?
- Any associated symptoms? (chest pain, shortness of breath, syncope, nausea, dizziness)
- Did the patient have to stop their activities or lie down?
- Was the patient able to stop the palpitations somehow? (Often, people discover they can terminate their palpitations with a vagal manoeuvre such as a Valsalva manoeuvre, a cough, or swallow).

Syncope

This is a faint or a swoon. You must determine whether there truly was a loss of consciousness and not simply the feeling that the patient was about to faint (pre-syncope). In particular, can the patient remember hitting the floor? If there really was a loss of consciousness, attempt to gain a collateral history from witnesses. Determine also:

- Was the onset gradual or sudden?
- How long was the loss of consciousness?
- What was the patient doing at the time? (Standing, urinating, coughing.)
- Were there any preceding or associated symptoms such as chest pain, palpitations, nausea, sweating (see previously)?
- Was there any relationship to the use of medication? (Antihypertensives and use of GTN-spray are common culprits.)
- When the patient came round, were there any other symptoms remaining?
- Was there any tongue-biting or urinary or faecal incontinence?
- Was there any motor activity during the unconscious episode?
- How long did it take for the patient to feel ‘back to normal’?

Other cardiovascular symptoms

Claudication

This comes from the Latin ‘claudicatio’ meaning ‘to limp’. These days, however, it is used to describe muscle pain that occurs during exercise as a sign of peripheral ischaemia.

In true claudication, the patient describes the pain thus:

- Feels like a tight ‘cramp’ in the muscle.
- Usually occurs in the calf, thigh, buttock, and foot.
- Appears *only* on exercise.
- Disappears at rest.
- May also be associated with numbness or pins-and-needles on the skin of the foot (blood is diverted from the skin to the ischaemic muscle).

As always, you should attempt to quantify wherever possible. In this case, determine the 'claudication distance'—that is how far the patient is able to walk before the pain starts. This will be useful in judging the severity of the disability and in monitoring the condition.

Rest pain

A similar pain to claudication, but this comes on *at rest* and is usually continuous—a sign of severe ischaemia. The patient may describe:

- Continuous, severe pain in the calf, thigh, buttock, or foot.
- 'Aching' in nature.
- Lasts through the day and night.
- Exacerbations of the pain may wake the patient from sleep.
- The patient may find slight relief by hanging the affected leg off the side of the bed.

Fatigue

A difficult symptom to determine as you'll find that most people will claim to be more tired than normal if asked. However, this pathological fatigue is caused by ↓ cardiac output and ↓ blood supply to muscles and needs to be taken seriously. Again, quantify and determine:

- Is the patient able to do less than they were previously?
- Is any ↓ in activity due to fatigue or some other symptom (e.g. breathlessness)?
- What activities has the patient had to give up due to fatigue?
- What are they able to do before they become too tired?

The rest of the history

Cardiac risk factors

These are important aspects of the history that have an impact on the risk of cardiovascular disease. When documenting a history of a cardiovascular case, it is worth pulling these out of the usual order and documenting as a list with ticks/crosses and details where appropriate at the end of the presenting complaint (as below). They should *not* then be repeated again later in the clerking.

- ***Age:*** ↑ risk with age.
- ***Gender:*** risk in males > females.
- ***Obesity:*** how heavy is the patient? (Calculate their BMI—see p.66.)
- ***Smoking:*** see p.44 for further advice. Quantify in pack-years. Don't be caught out by the 'ex-smoker' that gave up yesterday!
- ***Hypertension:*** find when it was diagnosed? How was it treated? Is it being monitored?
- ***Hypercholesterolaemia:*** increasingly, patients will know about this, some will even know their last reading. When was it diagnosed? How is it treated and monitored?
- ***Diabetes:*** what type? When was it diagnosed? How is it treated and monitored? What are the usual glucose readings?
- **FHx***:* particularly 1st degree relatives who have had cardiovascular events/diagnoses before the age of 60.

Past medical history

Ask especially about:

- Angina—if they have a GTN spray, ask how often they need to use it and whether this has changed significantly recently.
- MI—when? How was it treated?
- Ischaemic heart disease—how was the diagnosis made? Any angiograms? What other investigations has the patient had?
- Cardiac surgery—bypass? How many arteries?
- AF or other rhythm disturbance—what treatment? On warfarin?
- Rheumatic fever.

- Endocarditis.
- Thyroid disease.

Drug history

Take particular note of cardiac medication and attempt to assess compliance and the patient's understanding of what the medication does.

Social history

As in any other case, take note of the patient's employment—both how the disease has affected their ability to work and bear in mind how any cardiac diagnosis may affect the patient's employability.

Also record the home arrangements—are there any carers present, aids or adaptations, stairs, and so on.

General inspection and hands

The full examination framework is shown in Box 7.2. The order is not to be strictly adhered to but the authors feel that this is the easiest routine, working from the hands and face to more intimate areas of the body.

Positioning

The patient should be seated, leaning back to 45°, supported by pillows with their chest, arms, and ankles (if appropriate) exposed. Their head should be well supported allowing relaxation of the muscles in the neck. Ensure the room is warm and there is enough privacy. In an 'exam' condition, the patient should be undressed to their underwear.

If you intend to measure that patient's blood pressure seated and standing (remember to make the patient stand for 3 minutes before measuring), it may be wise to do this at the beginning of the examination.

General inspection

As always, take a step back and take an objective look at the patient.

- Do they look ill? If so, in which way?
- Are they short of breath at rest?
- Is there any cyanosis (see p.208)?
- What is their nutritional state?
  - Are they overweight?
  - Are they cachectic (underweight with muscle wasting)?
- Do they have features of any genetic syndrome such as Turner's, Down's, or Marfan's?

Hands

Take the patient's right hand in yours as if to greet them, look at it carefully and briefly compare with the other side. Look especially for:

- Temperature (may be cold in congestive cardiac failure)?
- Sweat.
- The state of the nails.
  - Blue discolouration if peripheral blood flow is poor.
  - Splinter haemorrhages (small streak-like bleeds in the nail bed) seen especially in bacterial endocarditis but may also be a sign of rheumatoid arthritis, vasculitis, trauma, or sepsis from any source.
- Finger clubbing (see p.208). Cardiac causes include infective endocarditis, and cyanotic congenital heart disease.
- Xanthomata—raised yellow lesions caused by a build up of lipids beneath the skin. Often seen on tendons at the wrist.
- Osler's nodes—rare manifestation of infective endocarditis (a late sign and the disease is usually treated before this develops). Red, tender nodules on the finger pulps or thenar eminence.
- Janeway lesions—non-tender macular-papular erythematous lesions seen on the palm or finger pulps as a rare feature of bacterial

endocarditis.

Box 7.2 Framework for the cardiovascular examination

An example framework for a thorough examination of the cardiovascular system—the information in this chapter is presented in a slightly different order for the purpose of clarity.

This is the authors' recommendation. Other methods exist and none are right or wrong so long as nothing is missed.

- General inspection.
- Hands.
- Radial pulse.
- Brachial pulse.
- Blood pressure.
- Face.
- Eyes.
- Tongue.
- Carotid pulse.
- Jugular venous pressure and pulse waveform.
- Inspection of the precordium.
- Palpation of the precordium.
- Auscultation of the precordium.
- Auscultation of the neck.
- Dynamic manoeuvres (if appropriate).
- Lung bases.
- Abdomen.
- Peripheral pulses (lower limbs).
- Oedema.
- Peripheral veins.

Peripheral pulses

All the major pulses are described below but should be examined in the order described on the previous page. For each, you should attempt to detect the rate and rhythm of the pulsation. For the brachial and carotid pulsations in particular, you should also determine the volume and character (waveform) of the pulse.

Technique

Examination technique is described below and illustrated in Fig. 7.1.

It is good practice *not* to use your thumb to feel pulses as you may mistake your own pulse (which can be felt weakly in the thumb) for the weak pulse of the patient—especially in the peripheral arteries.

Radial artery

Feeling for the waveform is not useful here as it is too far from the heart.

Use your 1st and 2nd fingers to feel just lateral to the tendon of the flexor carpi radialis and medial to the radial styloid process at the wrist.

Brachial artery

Feel at the medial side of the antecubital fossa, just medial to the tendenous insertion of the biceps.

Carotid artery

This is the best place to assess the pulse volume and waveform.

Find the larynx, move a couple of centimetres laterally and press backwards medial to the sternomastoid muscle.

Be sure not to compress both carotids at once for fear of stemming blood flow to the brain—particularly in the frail and elderly.

Femoral artery

This is another useful place for assessing the waveform unless there is disease or abnormality in the abdominal aorta.

The patient is usually stripped to their underwear by this point in the examination and should be lying on a bed or couch with their legs outstretched. Ask them to lower their clothes a little more, exposing the groins. The femoral pulsation can be felt midway between the pubic tubercle and the anterior superior iliac spine.

Popliteal artery

This lies deep in the popliteal fossa and is surrounded by strong tendons. It can be difficult to feel and usually requires more pressure than you expect. There are several techniques but we recommend:

- With the patient lying flat and knees slightly flexed, press into the centre of the popliteal fossa with tips of the fingers of the left hand and use the fingers of the right hand to add extra pressure to these.

Posterior tibial artery

Palpate at the ankle just posterior and inferior to the medial malleolus.

Dorsalis pedis

This runs lateral to the exterior hallucis longus tendon on the superior surface of the foot between the bases of the 1st and 2nd metatarsals.

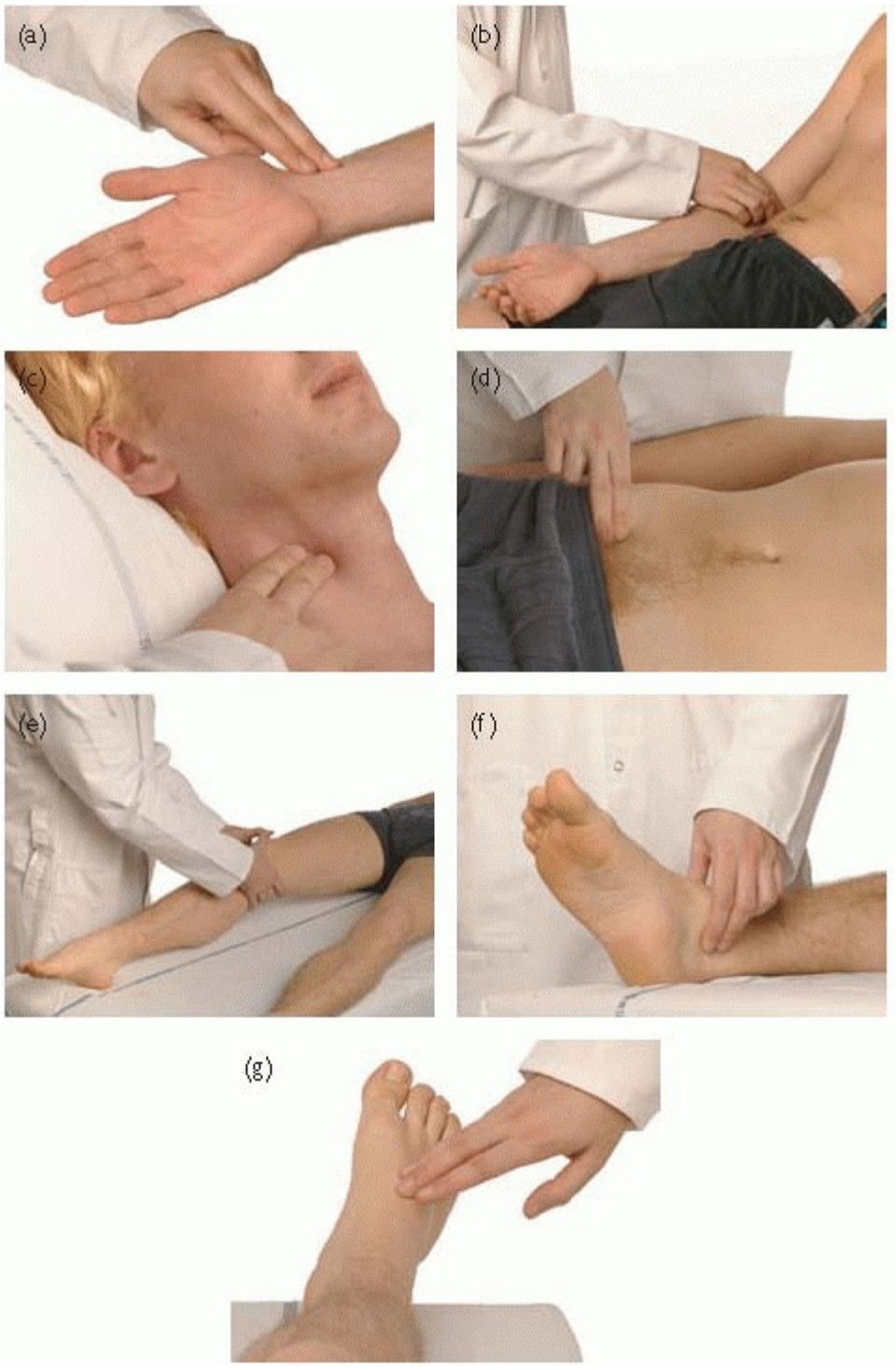


Fig. 7.1 Palpation of the peripheral pulses. (a)The radial pulse. (b) The brachial pulse. (c) The carotid pulse. (d)The femoral pulse. (e) The popliteal pulse. (f) The posterior tibial pulse. (g) The dorsalis pedis pulse.

Pulse rate

This should be expressed in 'beats per minute'. A rate <60bpm is called 'bradycardia' whilst 'tachycardia' is a pulse >100bpm. A normal healthy adult pulse rate should be ~60-100bpm.

The most accurate method is to count the pulse for a full minute. In practice, you count for a portion of this time and calculate the rate by multiplication. Commonly, people count for 15 seconds and multiply the number by 4.

Rhythm

You should feel the pulse as long as it takes to be sure of the rhythm. In general, the pulse can be either regular or irregular but variations exist.

- ***Regular:*** a self-explanatory definition. It must be remembered that the pulse rate may ↓ with inspiration and ↑ with expiration in the normal state.

- ***Irregularly irregular:*** this is a completely random pattern of pulsation and is synonymous with atrial fibrillation in which the atria twitch and contract in an irregular fashion sending electrical impulses to the ventricles (and therefore causing contraction and arterial pulsation) at random intervals.
- ***Regularly irregular:*** not quite the contradiction that it seems—you can have a non-regular pulse that occurs in some other regular pattern. For example, pulsus bigeminus will cause regular ectopic beats resulting in alternating brief gaps and long gaps between pulses. In Wenckebach's phenomenon, you may feel increasing time between each pulse until one is 'missed' and then the cycle repeats.
- ***Regular with ectopics:*** a very difficult thing to feel and be sure of without an ECG. A 'normal' regular heart rate may be intermittently interrupted by a beat that is out of step, making the pulse feel almost 'irregularly irregular'.

Character/waveform and volume

This is best assessed at the carotid artery. You are feeling for the speed at which the artery expands and collapses and force with which it does so. It takes some practise to master and it may be useful to imagine a graph such as those shown in Fig. 7.2. Some examples are below:

- ***Aortic stenosis:*** a 'slow rising' pulse, maybe with a palpable shudder. Sometimes called 'anacrotic' or a 'plateau' phase.
- ***Aortic regurgitation:*** a 'collapsing' pulse which feels as though it suddenly hits your fingers and falls away just as quickly. You could try feeling at the brachial artery and raising the arm above the patient's heart. Sometimes referred to as a 'waterhammer' pulse.
- ***Pulsus bisferiens:*** a waveform with 2 peaks, found where aortic stenosis and regurgitation co-exist.
- ***Hypertrophic cardiomyopathy:*** this pulse may feel normal at first but peter out quickly. Often described as 'jerky'.
- ***Pulsus alternans:*** an alternating strong and weak pulsation, synonymous with a severely impaired left ventricle in a failing heart.
- ***Pulsus paradoxus:*** pulse is weaker during inspiration (causes include cardiac tamponade, status asthmaticus, and constrictive pericarditis).

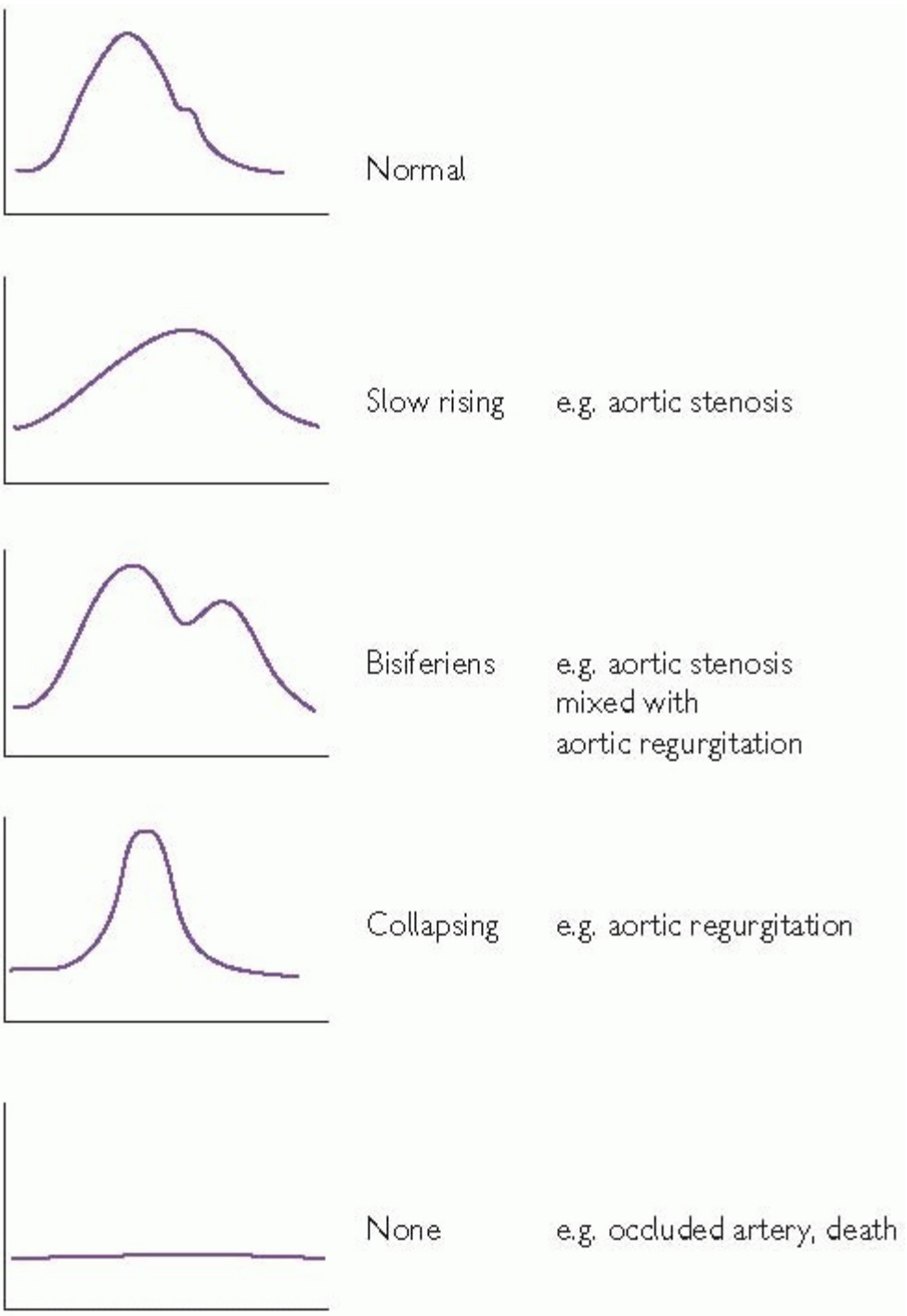


Fig. 7.2 Graphical representation of different arterial pulse waveforms and their causes.

Other tests of arterial pulsation

These are not routinely performed unless the history and rest of the examination has made the examiner suspicious of the specific pathologies that they represent.

Radio-radial delay

You should feel both radial pulses simultaneously. In the normal state, the pulses will occur together. Any delay in the pulsation reaching the radial artery on one side may point to pathology such as an aneurysm at the aortic arch or subclavian artery stenosis.

Radio-femoral delay

You should palpate the radial and femoral pulses on the same side simultaneously. They should occur together. Any delay in the pulsation reaching the femoral artery may point to aortic pathology such as coarctation.

The face and neck

Face

Examine the patient's face at rest. It's a good idea to develop your own pattern for this. The authors recommend starting with an overview, moving to the eyes, the mouth, then the neck. The order is not important as long as all aspects are examined. Be sure to ask them to:

- Look up whilst you gently pull down one lower eyelid, exposing the conjunctiva.
- 'Open wide' and look inside their mouth.
- Protrude their tongue.

In the cardiovascular examination, you should be looking especially for:

- ***Jaundice:*** seen as a yellow discolouration of the sclera.
- ***Anaemia:*** seen as an unusually pale conjunctiva (practise needed here).
- ***Xanthelasma:*** yellow, raised lesions found particularly around the eyes, indicative of a high serum cholesterol.
- ***Corneal arcus:*** a yellow ring seen overlying the iris. Significant in patients <40 years but not in older persons.
- ***Mitral facies:*** rosy cheeks suggestive of mitral stenosis.
- ***Cyanosis:*** seen as a bluish discolouration of the lips and tongue.
- ***High arched palate:*** suggestive of diseases such as Marfan's syndrome.
- ***Dental hygiene:*** a common source of organisms causing endocarditis.

Carotid pulse

At this point, the carotid pulse should be examined (p.170).

Jugular venous pressure

Theory

The jugular veins connect to the SVC and the right atrium without any intervening valves. Therefore, changes in pressure in the right atrium will transmit a pressure wave up these veins which can be seen in the neck. By measuring the height of the impulse, the pressure in the right side of the circulation can be expressed in centimetres.

Examination

It is often said that the JVP must only be measured in the internal jugular vein (IJV). This is not strictly the case. The external jugular vein (EJV) is easily seen as it makes a winding course down the neck (see Fig. 7.3). Its tortuous course means that impulses are not transmitted as readily or as reliably. It is for this reason that the IJV is used.

The centre of the right atrium lies ~5cm below the sternal angle which is used as the reference point.

The normal JVP is ~8cm of blood (therefore 3cm above the sternal angle). With the patient tilted back to 45°, the upper border of the pulse is just hidden at the base of the neck. This, therefore, is used as the standard position for JVP measurement.

▸ Remember, it is the vertical distance from the sternal angle to the upper border of the pulsation that must be measured.

▸ You must add 5cm to the figure to give the true JVP.

- With the patient lying back at 45°, expose the neck.
- Ask the patient to turn their head away from you (their left) and ensure that the neck muscles are relaxed.
- Look for the JVP and measure the *vertical* distance from the top of the pulsation to the sternal angle.
- The result is often expressed along the lines of '3cm raised'. It must be remembered that that is a total JVP of 8cm after adding the extra 5cm that are not measured.
- Try to look upwards, along the line of the sternomastoid. Don't get too close and use oblique lighting to make the pulsation more obvious.
- ▸ It can sometimes be difficult to distinguish the jugular venous pulse from the carotid pulse—see overleaf for some advice.

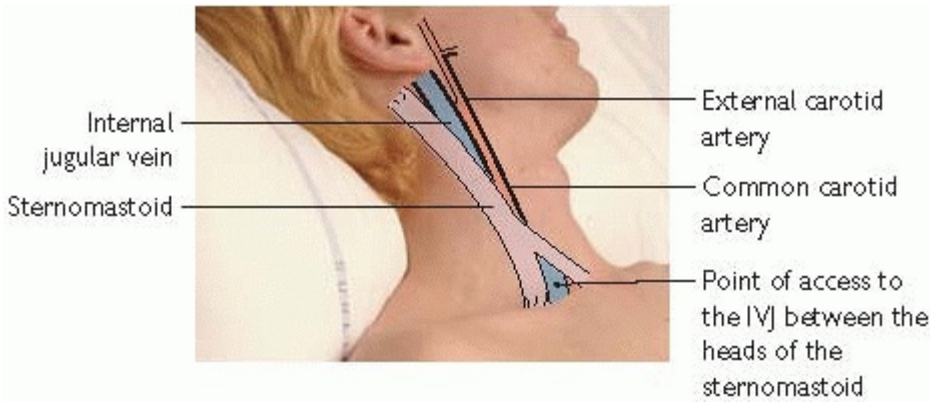


Fig. 7.3 The surface anatomy of the vasculature in the neck. Note that the IJV is partly hidden by the sternocleidomastoid at the base of the neck

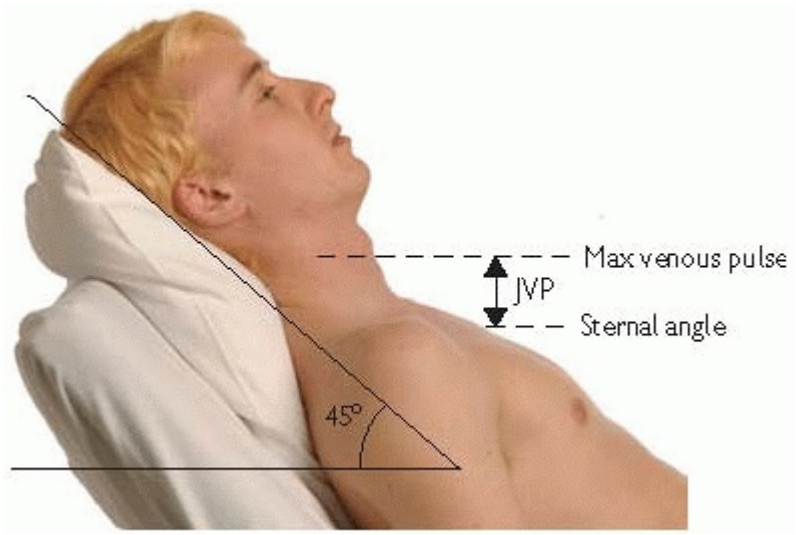


Fig. 7.4 Measuring the JVP. You must measure the vertical distance from the top of the pulsation to the sternal angle and then add 5cm to get the JVP.

Differentiating the jugular and carotid pulsations

The rules for differentiating the jugular and carotid pulsations below are guides only and not always true. For example, in severe tricuspid regurgitation, the jugular pulse is palpable and is not easily abolished by compression. If proving difficult, test the hepatojugular reflex.

Table 7.1

Jugular Pulsation	Carotid Pulsation
2 peaks (in sinus rhythm)	1 peak

Impalpable	Palpable
Obliterated by pressure	Hard to obliterate
Moves with respiration	Little movement with respiration

Hepatojugular reflux

- Watch the neck pulsation.
- Exert pressure over the liver with the flat of your right hand.

The JVP should rise by approximately 2cm, the carotid pulse will not.

Character of the jugular venous pulsation

This is rather difficult without experience. The jugular pulsation has 2 main peaks (see Fig. 7.5). You should establish the timing of the peaks in the cardiac cycle by palpating the carotid pulse at the same time. The key features are:

- *a wave:* caused by atrial contraction. Seen just *before* the carotid pulse.
- *c point:* slight AV-ring bulge during ventricular contraction.
- *x decent:* atrial relaxation.
- *v wave:* tricuspid closure and atrial filling.
- *y decent:* ventricular filling as tricuspid valve opens.

Findings

- *Raised* JVP*:* right ventricular failure, tricuspid stenosis, tricuspid regurgitation, superior vena cava obstruction, pulmonary embolus, fluid overload.
- *Large a waves:* caused usually be a hypertrophied right atrium (pulmonary hypertension, pulmonary stenosis, tricuspid stenosis).
- *Absent a wave:* atrial fibrillation.
- *'Cannon' a waves:* large, irregular waves caused by contraction of the atrium against a closed tricuspid valve. Seen in complete heart block.
- *Large v waves:* regurgitation of blood though an incompetent tricuspid valve.
- *Sharp y decent:* characteristic of constrictive pericarditis.
- *Sharp × decent:* characteristic of cardiac tamponade.

Kussmaul's sign

The JVP will ↓ during inspiration in the normal state. The JVP will rise during inspiration (Kussmaul's sign) in the presence of pericardial constriction, right ventricular infarction or, rarely, cardiac tamponade.

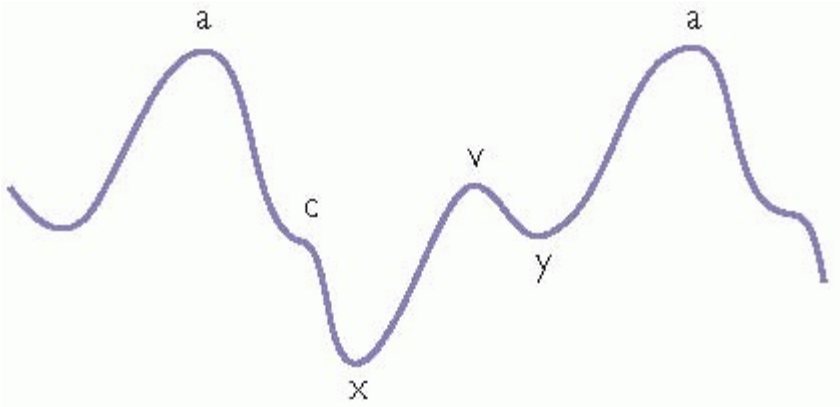


Fig. 7.5 Representation of the normal jugular venous pulsation.

Box 7.3 A word about honesty in learning

Students often find the JVP hard to see whilst they are learning the various examination techniques which reminds the authors of an important point.

There is, in medicine, an almost overwhelming pressure to say 'yes' when asked 'can you see that?' by the teacher. You may be motivated by a fear of appearing stupid, taking too much of the tutor's time, or delaying the ward round further. This, however, is useful to no-one. The student fails to learn the correct technique or the correct identification of the sign and the teacher fails to discover that their demonstration is inadequate. Misconceptions are born and are passed from person to person.

The authors, therefore, urge students of medicine of all ages and at all stages to say 'no, please show me again' and we will all be better for it.

Examining the precordium

The 'precordium' refers to that part of the chest overlying the heart. Inspection and palpation are below. Auscultation is discussed separately.

Inspection

The patient should be lying at 45° with the chest exposed. Look for:

- Scars—sternal split is used to access the median structures and to perform coronary artery bypass surgery. A left lateral thoracotomy may be evidence of previous closed mitral valvotomy, resection of coarctation, or ligation of a patent ductus arteriosus.
- Any abnormal chest shape or movements (p.210).
- Pacemaker or implantable defibrillator—usually implanted over the left pectoral region.
- Any visible pulsations.

Palpation

Explain what you are doing—particularly to female patients.

General palpation

Place the flat of your right hand on the chest wall—to the left, then to the right of the sternum. Can you fell any pulsations?

- *'Heave'*—this is a sustained, thrusting pulsation usually felt at the left sternal edge indicating right ventricular enlargement.
- *'Thrill'*—this is a palpable murmur felt as a shudder beneath your hand. Caused by severe valvular disease (If systolic: aortic stenosis, ventricular septal defect or mitral regurgitation; diastolic: mitral stenosis.)

Palpating the apex beat

This is the lowermost lateral point at which a definite pulsation can be felt. Usually at the 5^{th} intercostal space in the mid-clavicular line (Fig. 7.6).

Findings

- *Abnormal position of the apex beat:* usually more lateral than expected. Caused by an enlarged heart or disease of the chest wall.
- *No apex beat felt:* usually caused by heavy padding with fat or internal padding with an over-inflated emphysematous lung. Can sometimes be felt by asking the patient to lean forwards or laterally.

Character of the apex beat

This can only be learnt by experience, after having felt many 'normal' impulses. Some common abnormalities are:

- *Stronger, more forceful:* hyperdynamic circulation (e.g. sepsis, anaemia).
- *Sustained:* impulse 'longer' than expected (left ventricular hypertrophy, aortic stenosis, hypertrophic cardiomyopathy or hyperkinesia).
- *Double impulse:* (palpable atrial systole) characteristic of hyptertrophic cardiomyopathy
- *'Tapping':* this is the description given to a palpable 1st heart sound in severe mitral stenosis.
- *Diffuse:* a poorly localized beat caused by left ventricular aneurysm.
- *Impalpable:* emphysema, obesity, pericardial effusion, death.
- Beware of dextrocardia. If no beat is felt—check the right side.

Percussion

This is not useful and is not usually included in the cardiovascular examination.

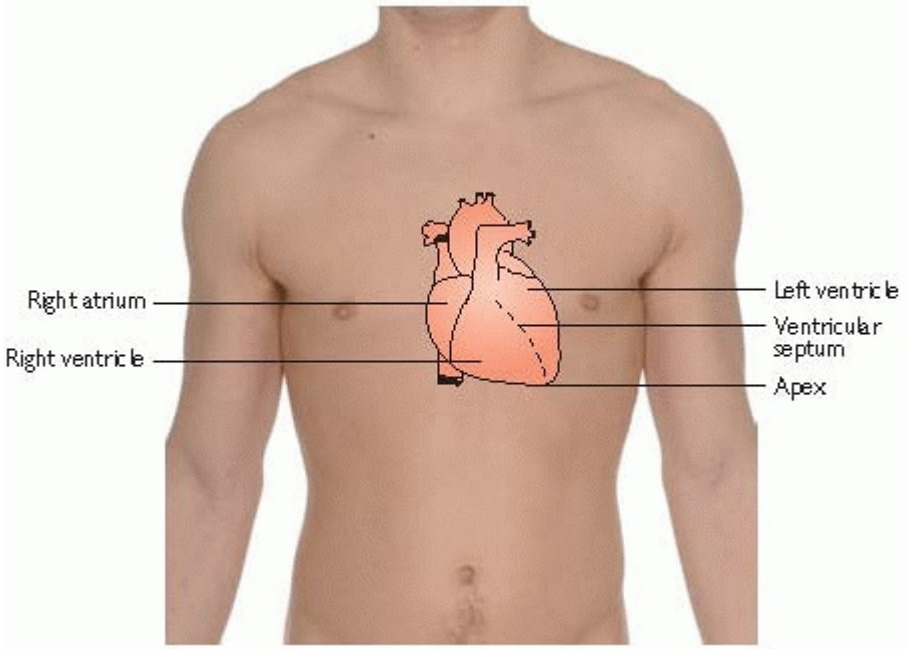


Fig. 7.6 Surface anatomy of the heart and most common location of the apex beat.

Auscultating the precordium

Technique

The 'bell' of the stethoscope is used to detect lower-pitched sounds, the diaphragm higher-pitched.

You should auscultate at each of the 4 standard areas (see Fig. 7.7).

Note that these areas do not relate exactly to the anatomical position of the valves but are the areas at which the sound of each valve can be best heard.

Different methods exist for this examination. A sensible approach would be to listen with the diaphragm at each area and then repeat using the bell. You can then 'go back' and concentrate on any abnormalities. You can then examine other areas looking for the features of certain murmurs and extra sounds as described on the following pages.

Box 7.4 The 4 areas

- ***Mitral:*** 5th intercostal space in the mid-axillary line (the apex).
- ***Tricuspid:*** 5th intercostal space at the left sternal edge.
- ***Pulmonary:*** 2nd intercostal space at the left sternal edge.
- ***Aortic:*** 2nd intercostal space at the right sternal edge.

Practice is needed here and many hearts should be listened to in order to be familiar with the normal sounds. The physiology behind the heart sounds and physiological splitting have been described on p.156 and may be worth revisiting at this point.

If you are unsure which is the 1st and 2nd heart sound—or where a murmur is occurring—you can palpate one carotid pulse whilst listening to the heart—enabling you to 'feel' systole. The carotid pulsation occurs with S_1. (Remember to only palpate one carotid pulse at a time.)

Findings—the heart sounds

1st heart sound (S_1)

Mitral valve closure is the main component of S_1 and the volume depends on the force with which it closes.

- *Loud:* forceful closing (mitral stenosis, tricuspid stenosis, tachycardia).
- *Soft:* prolonged ventricular filling or delayed systole (left bundle branch block, aortic stenosis, aortic regurgitation).
- *Variable:* variable ventricular filling (atrial fibrillation, complete heart block).

2nd heart sound (S_2)

- *Soft:* ↓ mobility of aortic valve (aortic stenosis) or if leaflets fail to close properly (aortic regurgitation).
- *Loud:* aortic component loud in hypertension or congenital aortic stenosis (here the valve is narrowed but mobile). Pulmonary component loud in pulmonary hypertension.

Splitting of S_2

See also the physiology section (p.156).

- *Exaggerated normal splitting:* caused by a delay in right ventricular emptying (right bundle branch block, pulmonary stenosis, ventricular septal defect, or mitral regurgitation).
- *Fixed splitting:* no difference in the extent of splitting between inspiration and expiration. Usually due to atrial septal defect.
- *Reversed splitting:* i.e. the pulmonary component of S_2 comes before the aortic component. Caused by a delay in left ventricular emptying (left bundle branch block, aortic stenosis, aortic coarctation).

3rd heart sound

This is a low frequency (can just be heard with the bell) sound occurring just after S_2. Described as a 'triple' or 'gallop' rhythm. 'Da-da-dum' or 'ken-tuck-y'. Occurs at the end of rapid ventricular filling, early in diastole and is caused by tautening of the papillary muscles or ventricular distension.

- *Physiological:* soft sound heard only at the apex, normal in children and fit adults up to the age of 30.
- *Pathological:* indicates some impairment of left ventricular function or rapid ventricular filling (dilated cardiomyopathy, aortic regurgitation, mitral regurgitation, or constrictive pericarditis). May be associated with a high-pitched pericardial knock.

4th heart sound

A late diastolic sound (just before S_1) caused by ↓ compliance—or ↑ stiffness—of the ventricular myocardium. 'Da-lub dub' or 'Ten-ne-ssee'. Coincides with abnormally forceful atrial contraction and raised end diastolic pressure in the left ventricle.

- Never physiological.
- Causes include hypertrophic cardiomyopathy and systemic hypertension.

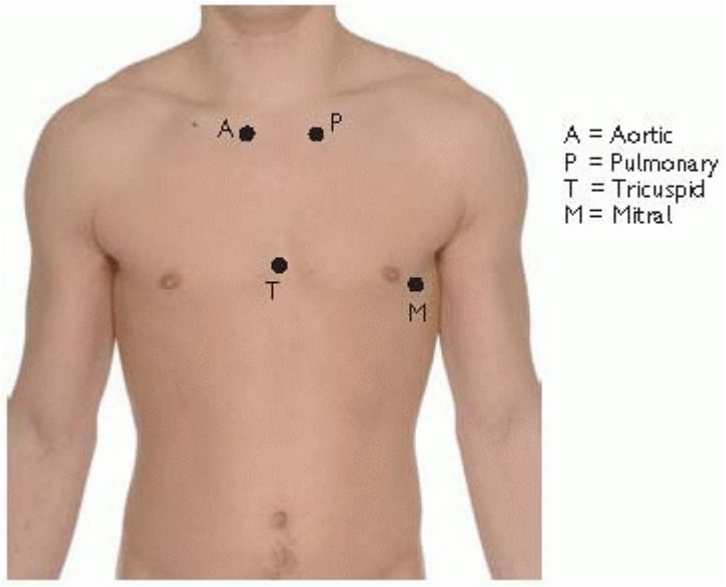


Fig. 7.7 The 4 standard areas for auscultation of the precordium and the valves that are best heard at each area.

Findings—murmurs

These are 'musical' humming sounds produced by the turbulent flow of blood. For each murmur heard, you should determine:

- The timing.
- The site and radiation (where is it heard the loudest?).
- The loudness and pitch.
- The relationship to posture and respiration.

The timing of the murmur is particularly essential in establishing the sound's origin. You must decide whether the noise occurs in systole or diastole (you should feel the patient's pulse at the carotid artery to be sure) and then when, within that period, it occurs.

Systolic murmurs

- ***Pansystolic:*** this is a murmur that lasts for the whole of systole and tends to be due to backflow of blood from a ventricle to an atrium (tricuspid regurgitation, mitral regurgitation). A ventricular septal defect will also cause a pansystolic murmur.
- ***Ejection systolic:*** these start quietly at the beginning of systole, quickly rise to a crescendo and decrescendo creating a 'whoosh' sound. Caused by turbulent flow of blood out of a ventricle (pulmonary stenosis, aortic stenosis, hypertrophic cardiomyopathy). Also found if flow is particularly fast (fever, fit young adults).
- ***Late systolic:*** audible gap between S_1 and the start of the murmur which then continues until S_2. Typically due to tricuspid or mitral regurgitation through a prolapsing valve.

Diastolic murmurs

- ***Early:*** usually due to backflow through incompetent aortic or pulmonary valves. Starts loudly at S_2 and decrescendos during diastole. (You can produce a similar sound by whispering the letter 'R' out loud. Try it).

- ***Mid-diastolic:*** these begin later in diastole and may be brief or continue up to S_1. Usually due to flow through a narrowed mitral or tricuspid valve. Lower pitched than early diastolic murmurs.
- ***Austin-Flint murmur:*** this is audible vibration of the mitral valve during diastole as it is hit by flow of blood due to severe aortic regurgitation.
- ***Graham-Steele murmur:*** pulmonary regurgitation secondary to pulmonary artery dilatation caused by ↑ pulmonary artery pressure in mitral stenosis.

Continuous murmurs

These are murmurs heard throughout both systole and diastole. Common causes include a patent ductus arteriosus or an arteriovenous fistula.

Radiation

The murmur can sometimes be heard in areas where heart sounds are not normally auscultated—the murmur will tend to radiate in the direction of the blood flow that is causing the sound (see p.185 for summary).

For example, the murmur of aortic stenosis will radiate up to the carotids, a mitral regurgitation murmur may be heard in the left axilla.

Box 7.5 Grading the volume of a murmur

The experienced examiner should be able to give the murmur a 'grade' according to its loudness as below.

- 1—very quiet (students will only hear it if they have already been told that it is there!).
- 2—quiet but can be heard with a stethoscope wielded by an examiner with some experience.
- 3—moderate. Easily heard.
- 4—loud, obvious murmur.
- 5—very loud heard over the whole of the precordium and may be accompanied by a palpable thrill.
- 6—heard without the aid of a stethoscope.

Position

Some murmurs will become louder if you position the patient so as to let gravity aid the flow of blood creating the sound.

- Aortic regurgitation is heard louder if you ask the patient to sit up, leaning forwards, and listen at the left sternal edge.
- Mitral stenosis is louder if you ask the patient to lie on their left-hand side (listen with the bell at the apex).

Dynamic manoeuvres

The following may help in identifying the origin of a murmur.

- ***Respiration:*** right-sided murmurs (e.g. pulmonary stenosis) tend to be louder during inspiration and quieter during expiration (because of ↑ venous return—see p.156). Ask the patient to breathe deeply whilst you listen. Left-sided murmurs are louder during expiration.
- ***Valsalva manoeuvre:*** this is forceful expiration against a closed glottis (consider straining over the toilet bowl!). Replicate by asking the patient to blow into the end of a syringe, attempting to expel the plunger. This will ↓ cardiac output and cause most murmurs to soften. Murmurs of hypertrophic obstructive cardiomyopathy, mitral regurgitation and mitral prolapse will get louder on release of Valsalva.

Findings—extra sounds

These are added sounds that are often associated with a specific murmur—see Table 7.2 opposite.

Opening snap

The mitral valve normally opens immediately after S_2. In mitral stenosis, sudden opening of the stiffened valve can cause an audible high-pitched snap. May be followed by the murmur of mitral stenosis. If there is no opening snap, the valve may be rigid.

- Best heard over the left sternal edge with the diaphragm of the stethoscope.

Ejection click

Similar to the opening snap of mitral stenosis, this is a high-pitched click heard early in systole caused by the opening of a stiffened semilunar valve (aortic stenosis). Associated with bicuspid aortic valves.

- Heard at the aortic or pulmonary areas and down the left sternal edge.

Mid-systolic click

Usually caused by mitral valve prolapse, this is the sound of the valve leaflet flicking backward (prolapsing) mid-way through ventricular systole. Will be followed by the murmur of mitral regurgitation.

- Best heard at the mitral area.

Tumour plop

A very rare finding due to atrial myxoma. If there is a pedunculated tumour in the atrium, it may move and block the atrial outflow during atrial systole causing an audible sound.

Pericardial rub

This is a scratching sound, comparable with creaking leather, heard with each heartbeat caused by inflamed pericardial membranes rubbing against each other in pericarditis. Louder as the patient is sitting up, leaning forward, and heard best in expiration.

Metallic valves

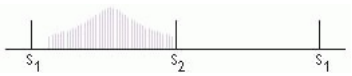
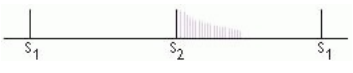
Patients who have had metallic valve replacement surgery will have an obviously audible mechanical 'click' corresponding to the closing of that valve. These can often be heard without the aid of a stethoscope and are reminiscent of the ticking crocodile in *Peter Pan*.

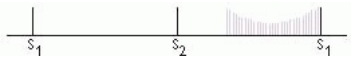
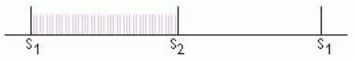
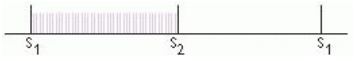
Some valves have both opening and closing clicks.

If a patient's valve click is unusually soft, this may indicate dysfunction e.g. thrombus or pannus.

All patients with prosthetic valves will have a flow murmur when the valve is open.

Table 7.2 A selection of cardiac abnormalities and the expected clinical findings. More information on p.188.

Abnormality	Primary site of murmur	Radiation	Timing	Added sounds*	Graphical Representation of the sounds
Aortic stenosis	'Aortic area' and apex	To carotid arteries	Ejection systolic	Ejection click (esp.bicuspid valve)	
Aortic regurgitation	Left sternal edge	Towards apex	Early diastolic	(Austin-Flint murmur p.182)	

Mitral stenosis	Apex	Nil	Mid-diastolic	Opening snap	
Mitral regurgitation	Apex	Toward left axilla or base of left lung	Pansystolic	Mid-systolic click (if prolapsing)	
Tricuspid regurgitation	Lower left sternal edge	Lower right sternal edge, liver	Pansystolic		
Pulmonary stenosis	Upper left sternal edge	Left clavicular region	Ejection systolic		
Ventricular septal defect	Left sternal edge	Whole of the precordium	Pansystolic		

* Note that added sounds such as clicks and snaps may only be present in certain patients and should not be ‘expected’ when examining someone with a certain abnormality.

The rest of the body

The lung bases

Examination of the lungs should form part of a thorough cardiovascular examination ( Chapter 8). Look especially for crackles or sign of effusion.

The abdomen

(See also  Chapter 9.) Look especially for:

- Hepatomegaly. Is the liver pulsatile (severe tricuspid regurgitation)?
- Splenomegaly.

- Ascites.
- Abdominal aortic aneurysm.
- Renal bruits (renal artery stenosis).
- Enlarged kidneys.

Peripheral oedema

An abnormal ↑ in tissue fluid resulting in swelling—its causes are multiple ( *OHCM6*, p.456) but often due to heart failure. Oedema is under gravitational control so will gather at the ankles if the patient is standing or walking, at the sacrum if sitting and in the lungs if lying (orthopnoea,  p.160).

- Make a note of any peripheral swelling, examining both the ankles and the sacrum.
- Note if the oedema is 'pitting' (are you able to make an impression in it with your finger?—Best tested over the anterior of the tibia).
- Note how high the oedema extends (ankles, leg, thighs, etc.).
- If the oedema extends beyond the thighs, it is important to examine the external genitalia—particularly in men—where the swelling may cause outflow obstruction.

Varicose veins

Inspection

Varicosities appear as visible, dilated, tortuous, subcutaneous veins caused by the backflow of blood from the deep veins (usually a branch of the long saphenous vein).

- The patient should be examined in a standing position with the legs fully exposed.
- Note any surrounding oedema, eczema, brown pigmentation, or ulcers.

Palpation

- Gently feel the varicose veins— hard veins may contain thrombus.
- Ask the patient to cough. If there is a palpable pulsation in the varicosity, there may be valvular incompetence at the long saphenous vein in the groin.

Percussion

- Apply the fingers of one hand to the upper part of the varicose vein.
- Gently flick the lower part of the vein with the other hand.
    - If there is a palpable wave sent up the vein, there are incompentent valves between those 2 points.

Trendelenburg's test

- Ask the patient to lie down and raise their leg so as to drain the veins.
- Apply a tourniquet over the saphenous vein (upper half of thigh).
- Ask the patient to stand.
    - You can then determine the site of the incompetent perforating vein … do the varicose veins fill above or below the tourniquet?
- Repeat the procedure until you are able to pin-point the exact location of the incompetence and, by applying localized pressure, prevent the varicose veins from filling at all.

Important presenting patterns

Valvular disease (see also 📖 p.185)

Mitral stenosis

- *Symptoms:* dyspnoea, cough productive of frothy (pink?) sputum, palpitations (often associated with atrial fibrillation and resultant emboli).
- *Signs:* palmar erythema, malar flush, 'tapping' apex beat, left parasternal heave, loud S_1, mid-diastolic murmur ±opening snap.

Mitral regurgitation

- *Symptoms:* acute dyspnoea and pulmonary congestion.
- *Signs:* collapsing pulse, sustained apex beat displaced to the left, left parasternal heave, soft S_1, loud S_2 (pulmonary component), pansystolic murmur heard at the apex radiating to left axilla ± mid-systolic click, 3^{rd} heart sound.

Aortic stenosis

- *Symptoms:* angina, syncope, dyspnoea, sudden death.
- *Signs:* Slow rising pulse, low blood pressure, narrow pulse pressure, sustained and powerful apex beat, ejection systolic murmur radiating to carotids, soft S_2, ± ejection click.

Aortic regurgitation

- *Symptoms:* similar to aortic stenosis.
- *Signs:* collapsing pulse, wide pulse pressure, sustained and displaced apex beat, soft S_2, early diastolic murmur at the left sternal edge (often described as 'blowing' or decrescendo), ± ejection systolic murmur (↑ volume), you may also hear a 'pistol shot' sound over the femoral artery with severe aortic regurgitation. See also Box 7.6.

Tricuspid stenosis

Usually occurs along with mitral or aortic valvular disease (e.g. in rheumatic fever) and is often the less serious of the patient's problems.

- *Signs include:* auscultation similar to that of mitral stenosis, hepatomegaly, pulsatile liver, and venous congestion.

Tricuspid regurgitation

Signs include: dilated neck veins, prominent v wave in JVP which may, rarely, cause the earlobe to oscillate, pansystolic murmur louder on inspiration with a loud pulmonary component of S_2, left parasternal heave, pulsatile liver, peripheral and sacral oedema, ascites. You may also hear a 3^{rd} heart sound and evidence of atrial fibrillation.

Pulmonary stenosis

Signs include: normal pulse with an ejection systolic murmur radiating to lung fields often with a palpable thrill over the pulmonary area. Other signs of right heart strain or failure.

Pulmonary regurgitation

Usually a coincidental finding.

- *Signs include:* loud S_2 which may be palpable, early diastolic murmur heard at the pulmonary area and high at the left sternal edge.

Box 7.6 Some eponymous signs of aortic regurgitation

- Prominent carotid pulsation (Corrigan's sign).
- Head-nodding in time with the heartbeat (De Musset's sign)*.
- Pulsation of the uvula in time with the heartbeat (Mueller's sign).
- Higher blood pressure in the legs than the arms (Hill's sign).
- Nailbed capillary pulsation (Quincke's sign).

Congenital heart disease

Ventricular septal defect (VSD)

- *Symptoms:* children with this are often asymptomatic. If a large defect, patient may suffer congestive cardiac failure with dyspnoea and fatigue.
- *Signs:* there may be cyanosis and clubbing of the fingers. Heart sounds usually appear normal but if pulmonary hypertension develops, may hear a loud pulmonary component of S_2 with right-ventricular heave. May also be a pansystolic murmur heard at the left sternal edge often accompanied by a palpable thrill. The signs may settle with time, the right heart pressure increases causing less shunting and a softer murmur.

Atrial septal defect (ASD)

The commonest congenital lesion and often an asymptomatic finding discovered on investigating a murmur.

- *Symptoms of secondum defect:* asmptommatic if small. Fatigue, dyspnoea, palpitations (atrial arrythmias), recurrent pulmonary infections, other symptoms of right heart failure. Also associated with migraine and paradoxical emboli.
- *Symptoms of primum defect:* symptoms of heart failure in childhood with failure to thrive, chest infections and poor development. In adults, there may be syncope (heart block) and symptoms suggestive of endocarditis.
- *Signs:* fixed splitting of S_2, ↑ flow over the normal pulmonary valve may give an ejection systolic murmur. Also left parasternal heave with a normal or diffuse apical impulse. Particularly in ostium primum defects (endocardial cushion defects), you may hear the pansystolic murmur of mitral regurgitation or co-existant ventricular septal defect (or both). Look also for signs of pulmonary hypertension.

Patent ductus arteriosus (PDA)

A persistent embryonic connection between the pulmonary artery and the aorta. Blood flows from the aorta into the pulmonary artery giving:

- *Symptoms:* often asymptomatic. Severe cases—dyspnoea on exertion.
- *Signs:* collapsing pulse, heaving apex beat, ‘machinery’ (continuous) murmur heard all over the precordium, S_2 not heard, systolic or diastolic thrill in the 2^{nd} intercostal space on the left.

Coarctation of the aorta

A congenital narrowing of the aorta at, or beyond, the arch.

- *Symptoms:* usually asymptomatic. May include headache, epistaxis, dizziness, and palpitations. Claudication, leg fatigue are also features. The coarctation may also cause the heart to strain and give symptoms of congestive cardiac failure.
- *Signs:* ↑ blood pressure in the upper limbs, radio-femoral delay, ejection systolic murmur at the left sternal edge, sometimes palpable collateral arteries over the scapulae with interscapular bruits. May also have underdeveloped lower limbs.
- Often associated with aortic stenosis, aortic aneurysms and bicuspid aortic valves. Seen also in Turner's syndrome.

Tetralogy of Fallot

Consists of pulmonary stenosis, ventricular septal defect (infundibular), right ventricular hypertrophy, and an overriding aorta. (If associated with an atrial septal defect also—‘Fallot's pentalogy’.)

- *Symptoms:* syncope, squatting relieves breathlessness, growth retardation.
- *Signs:* finger clubbing and central cyanosis with superadded paroxysms (‘spells’). Murmurs of pulmonary stenosis or the VSD may be

heard along with a systolic thrill and left parasternal heave.

Pericarditis

Causes include collagen diseases, TB, post-infarction, and idiopathic.

- *Symptoms of acute pericarditis:* constant retrosternal 'soreness', worse on inspiration (pleuritic), relieved slightly by sitting forwards, not related to movement or exertion.
- *If chronic, constrictive, may cause:* Kussmaul's sign, impalpable apex beat, S_3, hepatomegaly, splenomegaly, ascities ('pseudo-cirrhosis').

Pericardial effusion

- Pulsus paradoxus, ↑ JVP, impalpable apex beat, soft heart sounds, hepatomegaly, ascites, peripheral oedema.

Ischaemic heart disease

See 📖 p.158.

Congestive heart failure

In simple terms, this refers to the inability of the heart to maintain an adequate cardiac output for perfusion of vital organs with variable severity. (see 📖 *OHCM6*, p.136) It is usually described in terms of 'left' and 'right' heart failure but there is usually an element of both ('biventricular').

Left ventricular failure

- *Symptoms:* may include shortness of breath on exertion, orthopnoea, paroxysmal nocturnal dyspnoea, cough with pink frothy sputum, fatigue, weight loss, muscle wasting, and anorexia.
- *Signs:* may appear tired, pale, sweaty, clammy, tachycardic, thready pulse, low blood pressure, narrow pulse pressure, displaced apex beat, (murmur of an underlying valvular abnormality?), 3^{rd} and 4^{th} heart sounds, tachypnoea, crepitations at the lung bases.

Right ventricular failure

- *Symptoms:* as above with peripheral oedema and facial swelling.
- *Signs:* many of the above. Also raised JVP, hepatomegaly, ascites, peripheral (sacral?) oedema, pulsatile liver (if tricuspid regurgitation).

Hypertrophic cardiomyopathy

There may be a FHx although the vast majority of cases are 'sporadic' caused by new mutations.

- *Symptoms:* often none. If present, patient may suffer from shortness of breath, angina, syncope.
- *Signs:* sharp rising (jerky) pulse, prominent JVP a wave, double apex beat, late systolic murmur at left sternal edge which ↑ with Valsalva maneuvre.

Peripheral vascular disease

- *Symptoms:* claudication as 📖 p.164.
- *Signs:* (see also Box 7.7) shiny, pale, cold limb, hair loss, absent peripheral pulse(s). If severe, ischaemic ulceration and gangrene.

Deep vein thrombosis (DVT)

Often confused with cellulitis and ruptured popliteal cyst.

- *Symptoms:* calf pain, swelling and loss of use.
- *Signs:* warm, tense, swollen limb, erythema, dilated superficial veins, cyanosis. There may be palpable thrombus in the deep veins. Often pain on palpating the calf.

Box 7.7 The acutely ischaemic limb

The rule of Ps. The acutely ischaemic limb will be:

- Painful (at first becoming …)
- Painless (numb).
- Pale.
- Paralysed.
- Pulseless.

The elderly patient

Geriatricians are equally interested in cardiovascular disease—with an ageing population, the prevalence of cardiac, peripheral vascular, and stroke disease is due to rise. Whilst age is one of many risk factors for vascular disease, older people are one of the biggest groups to benefit from primary and secondary risk factor reduction—so be comprehensive in all assessments. A careful history is of far more use than an inaccurate one and list of physical findings.

History

- ***Angina:*** presents in a multitude of ways. Avoid labelling the symptoms as pain (which can irritate many patients) but listen to their complaints—'discomfort', 'twinges', and 'aches' are equally common presentations. Many elders have few symptoms, and may present with sweating or breathlessness. Be astute and ask if these relate to exertion.
- ***Orthopnoea:*** ask why patients sleep on extra pillows—often due to other symptoms such as arthritis. Do they sleep upright in a chair?
- ***Breathlessness:*** relates to a low-output state and not necessarily to pulmonary oedema. Fatigue is a common presenting symptom and should not be overlooked.
- ***DHx:*** Always a difficult balance of compliance, managing symptoms, achieving target doses and avoiding side effects. Avoid rushing to 'optimize' doses and upsetting a careful regimen. Ask about β-blocker eye drops—they can be absorbed systemically and exert significant effects.
- ***Lifestyle:*** don't forget to ask about smoking and seek opportunities to explore smoking cessation—it's never too late! Ask about alcohol; this may have a bearing on decisions around anticoagulation. Advice about healthy eating is often welcome, and more palatable than more tablets.
- ***Functional history:*** As ever, a key part of all histories. Targeted interventions—help with bathing to avoid over-exertion (and symptoms) can have significant impact.

Examination

- ***General:*** look out for clues—the breathless patient returning from the bathroom, the GTN spray close at hand etc.
- ***Ausculate and think:*** especially about valve lesions. It is more valuable to assess how much a valvular problem is contributing to a patient's symptoms, and arrange investigations. Aortic valve replacement and CABG is often hugely successful in older people.
- ***Oedema:*** be careful when palpating—contrary to popular teaching, pitting and non-pitting oedema are painful! Could it be gravitational?
- ***Peripheral pulses:*** often overlooked, but a vital part of the examination. Document carefully, and look for skin changes and ulceration that might be causing significant problems, but not necessarily raised in the history.

Additional points

- ***Alternative diagnoses:*** Respiratory illnesses often overlap, and may mimic—e.g. pulmonary fibrosis and left ventricular failure. If things 'don't add up', or there is little response to treatment, revisit your diagnosis.

We thank Dr Richard Fuller for providing this page.

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Chapter 8

The Respiratory System

Applied anatomy and physiology

Respiratory physicians consider this to be the most important system in the body.

Anatomy

The respiratory tract extends from the nostrils to the alveoli and includes the pulmonary blood supply. Clinically, it is divided into the upper respiratory tract (URT) which is the nose and pharynx and the lower respiratory tract (LRT) which consists of the larynx and all distal structures.

Trachea, bronchi, and bronchioles

The trachea lies in the midline deep to the sternal notch and divides into the left and right main bronchi at the 'carina', level with the sternal angle.

There are about 25 further divisions before reaching the alveoli. The last 16 orders are termed 'bronchioles' and differ from the bronchi by having no cartilage, fewer goblet cells, and progressively less muscular walls.

Lungs

The right lung has 3 lobes (upper, middle, and lower) whilst the left lung has 2 (upper and lower), see Fig. 8.1. Note the angle of the oblique fissure, dividing the upper and lower lobes is such that examination of the anterior chest is mostly that of the upper (and middle) lobes whereas to examine the back is to examine the lower lobe.

The right middle lobe sits anteriorly, separated from the upper lobe by the horizontal fissure. The chest wall corresponding to this is between the right 4^{th} and 6^{th} ribs anteriorly and is easily missed. The same area on the left is the 'lingula', part of the left upper lobe which reaches around the anterior side of the heart.

Note the slant of the diaphragm is such that the inferior border of the lungs is at the 6^{th} rib anteriorly but extends down to the 12^{th} posteriorly.

Physiology

Lung function

Ventilation is under the influence of the respiratory centre in the brain. This, in turn, can be influenced by higher voluntary centres and by chemoreceptors which respond to changes in blood gas partial pressures. In health, the most important are the brainstem chemoreceptors, stimulating breathing in response to rising blood CO_2.

The main muscle of breathing is the diaphragm, innervated by the phrenic nerve. Contraction causes a flattening of the central part and a slight elevation of the peripheral parts. This causes expansion of the chest cavity and, as the intrathoracic pressure drops, air is sucked into the lungs. The intercostal and scalene muscles maintain the chest wall stability. When a larger inspiration is needed, these 'accessory' muscles, including the sternocleidomastoids, act to further expand the chest.

Expiration is largely passive, the air being expelled as the lungs recoil under their innate elasticity.

Aside from inadequate ventilation, ineffective lung functioning can result from inadequate perfusion of the ventilated areas. Also, impeded gas

transfer across the alveoli may be a factor—either by a thickening of the wall (e.g. fibrosis), a loss of surface area (as in COPD), or fluid in the alveoli such as oedema, pus, or blood.

Defence

Mechanical defence against infection includes the narrow passages of the nose as well as the larynx which separates the respiratory and GI tracts. Cough receptors in the pharynx and lower airways initiate mechanisms resulting in a deep inspiration followed by expiration against a closed glottis and a sudden glottal opening. This causes a rapid, forceful expulsion of air which we know as a cough.

Most of the respiratory tract is lined with mucous secreted from goblet cells which catch small particles and microbes. This is continuously swept upwards towards the larynx by beating cilia on the epithelium.

In the smaller airways and alveoli, macrophages and a variety of secreted defensive proteins act against microbes at a microscopic level.

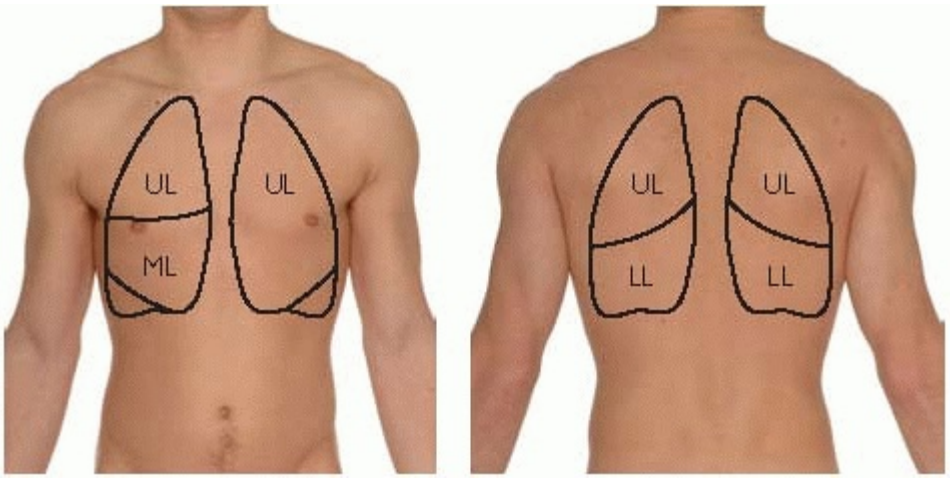

Fig. 8.1 Surface anatomy of the lungs. (UL = upper lobe, ML = middle lobe, LL = lower lobe.)

Dyspnoea

Defining dyspnoea

Shortness of breath (SOB), or 'dyspnoea', is the sensation that one has to use an abnormal amount of effort in breathing. Patients may describe 'breathlessness', an inability to 'get their breath', or being 'shortwinded'.

'Tightness' is often described and may actually be the sensation of airway narrowing as in asthma or may be chest pain, as in cardiac disease. Tease out exactly what the patient means.

Pleuritic chest pain is worse at the height of deep inspiration and patients may say they are 'not able to get my breath'. Thus, seemingly complaining of breathlessness, their actual problem is pain on inspiration. Ask if they feel the need to breathe faster than normal or are unable to breathe deeply. If the latter, ask if that is due to pain or to some other sensation. If in doubt, ask the patient to take a deep breath and watch the results.

Onset and duration

How quickly did the SOB come on? Pulmonary embolus, pneumothorax, and asthmatic attacks can come on suddenly. Pneumonia, heart failure, and anaemia cause SOB which worsens over days or weeks. Smoking-related lung disease, on the other hand, causes ↑ breathlessness over many years.

Slower onsets are poorly reported. The patient often reports the onset of a *worsening* of breathlessness. Ask when they were last able to run up the stairs and the real duration of breathlessness becomes apparent.

Severity

Several classifications exist but it is often more useful to quantify in terms of functional impairment. Ask: 'how far can you walk before you have to stop?'. 'How many flights of stairs can you manage?' Restriction of hobbies such as gardening and dancing are also useful guides.

Be sure that activities are restricted by SOB as opposed to arthritic hips, knees, chest pain, or some other ailment.

Exacerbating and relieving factors

What makes the breathlessness worse? Can it be reliably triggered by a particular activity or situation? Divide into shortness of breath at rest (SOBAR) and shortness of breath on exertion (SOBOE) and quantify as above. Remember to ask about position e.g. orthopnoea (p.160).

Have they used their inhalers? Did they help?

Associated symptoms

See the following pages for a full breakdown. Be aware of the hyperventilation syndrome in which the ↓ of blood CO_2 will cause paraesthesia in the lips and fingers along with light headedness and, in severe cases, tetany.

Cough and expectoration

Cough

A common but often overlooked symptom in respiratory disease, usually caused by URT infection (URTI) and/or smoking. Duration of cough is important, as well as character, exacerbating factors, and whether any sputum is produced.

Note that cough may be the only symptom of childhood asthma.

'Chronic cough' is that lasting >3 weeks and may be caused by asthma, carcinoma, interstitial disease and bronchiectasis, gastro-oesphageal reflux disease, and post-nasal drip.

Smokers will have a chronic cough, particularly in the mornings, so a history of a change is important.

The character of the cough often reveals the cause (Table 8.1).

Associated pain is common and the patient will be able to localize this to the URT (e.g. laryngitis; tracheitis is particularly painful) or the chest wall (i.e. pleurisy secondary to lobar pneumonia or collapse).

Remember non-pulmonary causes: post-nasal drip (dry, tickly), gastro-oesophageal reflux (dry), pharyngeal pouch or tracheo-oesophageal fistula (worse after eating or lying down), ACE-inhibitors (dry).

Sputum

Excess respiratory secretions that are coughed up. Patients will usually understand the term 'phlegm' better. Features to glean are: how often? How much? How difficult is it to cough up? Colour? Consistency? Smell?

Attempt to quantify sputum production in terms of well-known objects such as tea-spoons, egg-cups etc. 'Mucoid' sputum is white or clear in colour but can be turned grey in cigarette smokers. Yellow or green sputum is termed 'purulent' and usually indicates infection although eosinophils in the sputum of asthmatics can also produce a green colour. See Table 8.2.

Harder 'plugs' of sputum from the airways are seen in asthmatic sputum. Miniature tree-like bronchial casts are produced in bronchopulmonary aspergillosis.

Haemoptysis

The coughing up of blood can vary from streaks or a pink hue (e.g. CCF) to massive, life-threatening bleeds. Establish the amount, colour, frequency and nature of any associated sputum. ('Massive' haemoptysis = >500mL in 24 hours.)

Easily confused with blood originating in the nose, mouth, and GI tract (haematemesis). Ask about, and check for, bleeds in these areas also.

Causes of haemoptysis include bronchitis, carcinoma, pulmonary embolus, and infarction, TB, cystic fibrosis, and lung abscesses. 'Infective' causes will usually produced blood-stained sputum as opposed to pure haemoptysis.

Be alert to other potential sites of bleeding (skin, GI tract, mouth) which could point to a coagulation disorder.

Table 8.1 Some characteristic coughs

Cause	Character
Laryngitis	Cough with a hoarse voice
Tracheitis	Dry and very painful
Pleurisy	Sharp pain (chest wall)
Post-nasal drip	Tickly

Asthma	Chronic, paroxysmal, worse after exercise and at night
Oesophageal reflux	Dry and nauseating. Often first thing in the morning.
Tracheo-oesophageal fistula (rare)	Nauseating and worse after eating
Epiglottitis	‘Barking’
Laryngeal nerve palsy	‘Bovine’ hollow, brassy
Left heart failure	Productive and worse on lying flat

Table 8.2 Some characteristics of sputum

Nature of sputum	Causes
White/grey	Asthma Smoking
Green/yellow	Bronchitis Bronchiectasis
Green and offensive	Bronchiectasis Abscesses
Sticky, rusty	Lobar pneumonia

Frothy, pink	Congestive cardiac failure
Separates to 3 layers (mucoid, watery, rusty)	Severe bronchiectasis
Very sticky, often green	Asthma
Sticky, with ‘plugs’	Allergic aspergillosis (complication of asthma)

Other respiratory symptoms

Wheeze

This is a high-pitched whistling ‘musical’ sound produced by narrowing of airways, large or small. Occurs in inspiration and expiration, but usually louder and more prominent in the latter. If due to small airway narrowing, as in asthma, will be accompanied by a prolonged expiratory phase.

Causes include asthma, smoking related lung disease, mucosal oedema, airway obstruction, airway collapse, and pulmonary oedema (‘cardiac asthma’).

Stridor

A harsh ‘crowing’ inspiratory and expiratory sound with a constant pitch. Signals large airway narrowing, usually at the larynx or trachea. Can precede complete airway obstruction so is treated as a medical emergency.

Hoarseness/dysphonia

Described on p.141.

Pain

Chest pain is explored fully on p.158. Pain arising from respiratory disease is usually ‘pleuritic’ in nature and arises from the chest wall, parietal pleura, or mediastinum as the lungs have no pain fibres. It is felt as a severe, sharp pain at the height of inspiration or on coughing. Usually localized to a small area of chest wall. Note that patients will avoid deep breathing and may complain of breathlessness (see p.198).

Pain resulting from lung parenchymal lesions may be dull and constant and is usually a sinister sign of spread into the chest wall.

The pain of tracheitis is a poorly localized, central soreness. Diaphragmatic pain may be felt at the ipsilateral shoulder tip whilst pain from the costal parts of the diaphragm may be referred to the abdomen.

In general, muscular and costal lesions will be tender to touch over the corresponding chest wall and exacerbated by certain twisting movements while pleurisy is not—although this is not always the case. Costochondritis is a common cause of pleuritic pain of which Tietze's syndrome is a specific cause associated with pain and swelling of the superior costal cartilages.

Pain in a nerve-root distribution may be due to spinal lesions or herpes zoster.

The rest of the history

Other symptoms

Fever: particularly at night may be a sign of infection such as TB, malignancy, or a connective tissue disorder.

Weight loss: can be a symptom of carcinoma, chronic lung disease, or chronic infection. Attempt to quantify any loss (how much in how long?).

Peripheral oedema: manifesting as ankle swelling at the end of the day may be a sign of right heart failure secondary to chronic lung

disease (cor pulmonale). If severe enough, patients may exhibit signs and symptoms of left heart failure (p.190).

Obstructive sleep apnoea: is caused by upper airway obstruction by the flaccid palatal muscles during REM sleep. This causes snoring and progressive hypoxia which briefly wakes the patient from sleep, often accompanied by thrashing around disturbing those sharing the bed. The bed-time partner will graphically tell this tale. The patient will describe early morning headaches caused by CO_2 retention and day-time somnolence due to the sleep deprivation.

Past medical history

- Vaccination for respiratory illnesses, particularly BCG.
- Previous respiratory infections especially TB before 1950 when operations may have been performed resulting in lifelong deformity.
- X-ray abnormalities previously mentioned to the patient.
- Childhood (a 'chesty child' may have had undiagnosed asthma).
- Previous respiratory high dependency or ITU admissions and NIV.

Drug history

- What inhalers are used and how often? Check inhaler technique.
- Previous successful use of bronchodilators and steroids.
- Oral steroid therapy predisposes to infection and TB especially.
- ß-blockers may exacerbate obstructive lung diseases.
- ACE inhibitors cause a dry cough.
- If O_2 therapy—cylinders or concentrator? How many hours a day?
- Illicit drug use (cocaine is associated with respiratory disease).

Family history

- Asthma, eczema and allergies.
- Inherited conditions (e.g. CF, α_1-antitrypsin deficiency).
- Family contacts with TB.

Smoking

Attempt to quantify the habit in 'pack-years'. 1 pack-year is 20 cigarettes per day for one year. (e.g. 40/day for 1 year = 2 pack-years; 10/day for 2 years = 1 pack-year). Beware of appearing judgemental.

Ask about previous smoking as many will call themselves non-smokers if they gave up yesterday or even on their way to the hospital!

Remember to ask about passive smoking.

Alcohol

Alcoholics are at greater risk of chest infections and binging may result in aspiration pneumonia.

Social history

Pets: dogs and cats are a common source of allergens. Remember birds and caged animals. Ask about exposure beyond the home in the form of close friends and relations, and hobbies such as pigeon fancying or horse riding.

Travel: ask about travel (recent or previous) to areas where respiratory infections are endemic. Think particularly about TB. Remember also, that *Legionella* can be caught from water systems and air-conditioning even in developed 'safe' countries.

Occupation: is more important in chest medicine than any other field. Be alert to exposure to asbestos, coal, cotton, nitrogen dioxide, metals such as tin, silver, iron oxide, titanium, as well as hay, air conditioner systems, and so on. Trace the occupational history back as there may be a lag of 20 or 30 years between exposure and resultant disease. Remember that exposure may not be obvious and the patient may have been unaware of it at the time. Plumbers, builders, and electrical engineers may have been exposed to asbestos in the past.

Ask also about close personal contacts—partners may be significantly exposed by handling and washing clothing for example (although this

is relatively rare).

General appearance

Respiratory patients may be short of breath and it may be easiest to examine them sitting at the edge of the bed as opposed to the classic position of sitting back at 45°. Choose a position comfortable to you both. They should be undressed to the waist.

As ever, a surprising amount of information can be obtained by observing the patient before laying on a finger.

Bedside clues

Look for evidence of the disease and its severity around the patient:

- Inhalers? Which ones?
- Any additional inhaler devices (e.g. aerochambers)?
- Nebulizer?
- Is the patient receiving O_2 therapy? If so, how much and by what method (i.e. face mask, nasal cannulae etc.)?
- Sputum pot? Sputum-laden tissues?
- Remember to inspect the sputum carefully and record the findings.
- Any mobility aids nearby?
- Look for cigarettes, lighter, or matches at the bedside or in a pocket.

Respiration

Watch the patient from the foot of the bed. Or watch them approach your clinic room.

- Do they appear out of breath at rest?
- If so, do they appear in distress?
- Are they breathing through the mouth or the nose?
- Are they breathing through pursed lips? (↑ the expiratory pressure—an indication of smoking-related lung disease.)
- If mobile, did they have to stop on the way to the room? How quickly did they recover?
- Count the respiratory rate. At rest, this should be <12/minute.
- Are the breaths of normal volume?
- Are they using the accessory respiratory muscles (e.g. sternomastoids)?
- Are they using their arms to splint their chest? (The classic position is sitting forwards, hands on knees.)

Speech

- Is their speech limited by their breathlessness? If so, can they complete a full sentence? (An important indicator of severity in many conditions.)
- Listen for hoarseness as well as the gurgling of excess secretions.
- A nasal voice may indicate neuromuscular weakness.

Cough and abnormal sounds

Watch and listen for coughing (see previous pages) as well as stridor and wheeze.

Hands, face, and neck

Temperature

- Cold fingers indicate peripheral vasoconstriction or heart failure.

- Warm hands with dilated veins are seen in CO_2 retention.

Staining

Fingers stained with tar appear yellow/brown where the cigarette is held (nicotine is colourless and does *not* stain). This indicates smoking but is not an accurate indicator of the number of cigarettes smoked.

Cyanosis

This is a bluish tinge to the skin, mucous membranes and nails (📖 p.58), evident when >2.5g/dL of reduced haemoglobin is present (O_2 Sats about 85%). Easier to see in good, natural light.

Central cyanosis is seen in the tongue and oral membranes (severe lung disease e.g. pneumonia, PE, COPD). Peripheral cyanosis is seen *only* in the fingers and toes and caused by peripheral vascular disease and vasoconstriction.

Finger clubbing

↑ curvature of the nails. Early clubbing is seen as a softening of the nail bed (nail can be rocked from side to side) but this is very difficult to detect. Progressive clubbing leads to a loss of the nail angle at the base and eventually to a gross longitudinal curvature and deformity.

Objectively check for clubbing by putting the patient's nails back-to-back as in Fig. 8.2. Clubbing leads to a loss of the diamond-shaped gap.

Important respiratory causes are carcinoma, asbestosis, fibrosing alveolitis, and chronic sepsis (bronchiectasis, abscess, empyema, CF).

Pulse

Rate, rhythm, character. Tachycardic 'bounding' pulse = CO_2 retention.

Tremor

- ***Fine tremor:*** caused by use of ß-agonist drugs (e.g. salbutamol).
- ***Flapping tremor (asterixis):*** flapping when holding the hands dorsiflexed with the fingers abducted (Fig 8.3). Identical to the flap of hepatic failure (📖 p.244). Late sign of CO_2 retention.

Blood pressure

Pulsus paradoxus (📖 p.172). Causes: pericardial effusion, severe asthma.

JVP

See 📖 p.174. Raised in pulmonary vasoconstriction or pulmonary hypertension and right heart failure. Markedly raised, without a pulsation, in superior vena cava obstruction (SVCO, see 📖 *OHCM6*, p.436) with distended upper chest wall veins, facial and conjunctival oedema (chemosis).

Nose

Examine inside and out, looking for polyps (asthma), deviated septum and lupus pernio (red/purple nasal swelling of sarcoid granuloma).

Eyes

- ***Conjunctiva:*** evidence of anaemia?
- ***Horner's syndrome:*** (📖 p.303) caused by compression of the sympathetic chain in the chest cavity (tumour, sarcoidosis, fibrosis).
- ***Iritis:*** TB, sarcoidosis.
- ***Conjunctivitis:*** TB, sarcoidosis.
- ***Retina:*** Paplilloedema in CO_2 retention or cerebral metastases. Retinal tubercles in TB. Choroiditis in TB or syphilis.

Lymph nodes

See p.68 for full description of technique. Feel especially the anterior and posterior triangles, the supraclavicular areas. Don't forget the axillae—receive lymph drainage from the chest wall and breasts.

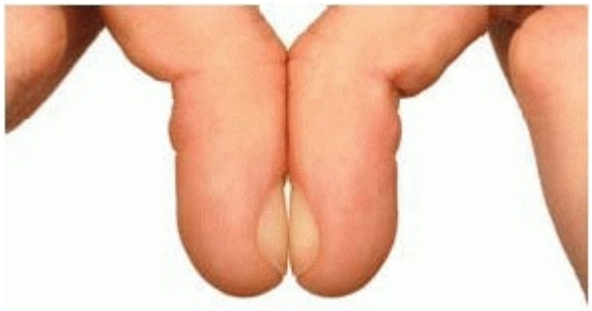

Fig. 8.2 Examining for clubbing. In the normal state, a diamond window will be seen between opposed nail beds which is lost in finger clubbing.

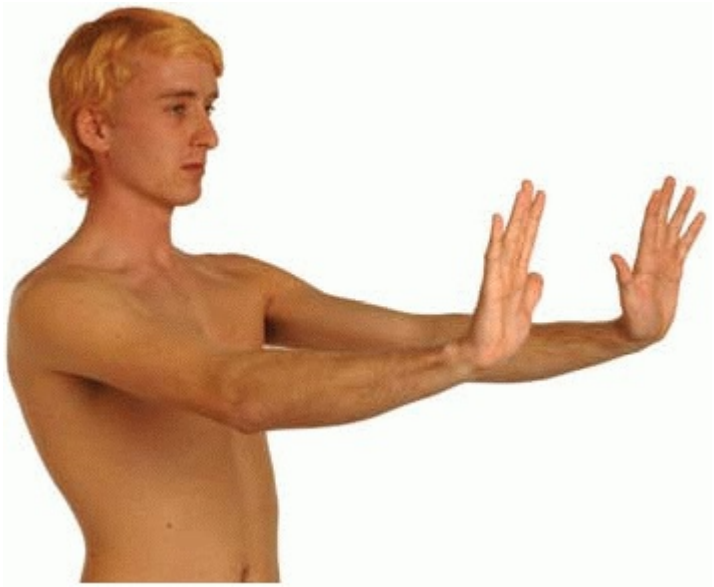

Fig. 8.3 Looking for a flapping tremor. Wrists are dorsiflexed and fingers abducted.

Inspection of the chest

Look at the shape and movement of the chest up-close.

Surface markings

Check the whole chest for scars and lesions.

Scars: may indicate previous surgery. Look especially in the mid-axillary lines for evidence of past chest-drains.

Radiotherapy: will often cause lasting local skin thickening and erythema.

Veins: look for unusually prominent surface vasculature.

Shape

Deformity: any asymmetry of shape? Remember to check the spine for scoliosis or kyphosis.

Surgery: TB patients from the 1940s and 1950s may have had operations resulting in lasting and gross deformity (thoracoplasty).

'Barrel chest': a rounded thorax with ↑ AP diameter. Hyperinflation, a marker of smoking related lung disease.

'Pectus carinatum': also called 'pigeon chest'. Sternum and costal cartilages are prominent and protrude from the chest. Caused by ↑ respiratory effort when the bones are still malleable in childhood—asthma, Rickets.

'Pectus excavatum': also called 'funnel chest'. Sternum and costal cartilages appear depressed into the chest. A developmental defect—usually a normal variant with no significance to pathology.

Surgical emphysema: air in the soft tissues will appear as a diffuse swelling. Especially in the neck; may feel 'crackly' to the touch.

Breathing pattern

Again, examine rate and depth of breathing.

- Fast, deep breaths are seen in anxiety states.
- Deep, sighing breaths 'Kussmaul's respiration' = systemic acidosis.
- Cheyne-Stokes breathing: alternating pattern deep, regular breathing with very slow, shallow breaths. Due to failure of the normal respiratory regulation in response to blood CO_2 levels.
- Prolonged expiratory phase = marker of outflow limitation, sign of smoking related lung disease if coupled with pursed-lip breathing.

Movement

Observe chest wall movement during breathing at rest. Also, ask the patient to take a couple of deep breaths in and out and watch closely.

- Look for asymmetry. ↓ movement indicates lung disease on that side.
- ↓ movement globally is seen in COPD, along with a 'pump handle' movement of the ribs (hinged posteriorly only) compared with the normal 'bucket handle' (hinged at the front and back).
- 'Harrison's sulcus' is a depression of the lower ribs just above the costal margins and indicates severe childhood asthma.

Palpation

Mediastinal position

Trachea: the trachea should lie in the midline just deep to the sternal notch. The trachea will shift as the mediastinum is pulled or pushed laterally. This is a late sign and not easy to assess, unless shift is marked.

Several methods for checking the position exist, all of which are somewhat uncomfortable for the patient. These are the two most popular…

- Use a single finger to feel for the trachea, the distance between it and the sternomastoids on each side should be the same.
- Use 2 fingers and palpate the sulci either side of the trachea at the same time. They should feel of identical size.

Apex beat: is normally at the 5^{th} intercostal space in the mid-clavicular line. It will shift with the mediastinum. However, is very difficult to palpate in the presence of hyperexpanded lungs and may be shifted to the left if the heart is enlarged.

Chest expansion

An objective measure of chest movement, using your hands as a guide.

- Put both hands on the patient's chest, just below the level of their nipples, anchoring your fingers laterally at the sides (Fig. 8.4).
- Extend your thumbs so that they touch in the midline—don't press them against the chest.
- Ask the patient to take a deep breath. As they do this, watch your thumbs, they should move apart equally. Any ↓ in movement on one side should be visible.
- It is easy to move your thumbs yourself in the expected direction.

  Beware of this and allow them to follow the movement of the chest.

It is important to explain what you are doing here, as suddenly reaching for a female chest may be misconstrued!

Female breasts come in different sizes and the placement of your hands for this part of the examination should vary accordingly. In particular, if faced with an older or particularly large-busted woman, it may be easier to place your hands above the breasts, at about the level of the 5th rib.

Tactile vocal fremitus

This is the vibration felt on the chest as the patient speaks. Each part of the chest is tested, as for percussion.

- Place the medial edge of your hand horizontally against the chest.
- Ask the patient to say 'ninety nine' or 'one, one, one'.
- You should feel the vibration against your hand.

This test is rather crude and often neglected by clinicians. The changes are identical to those for vocal resonance…

- ↑ vibration in consolidation.
- ↓ in pneumothorax, collapse, COPD and pleural effusion.

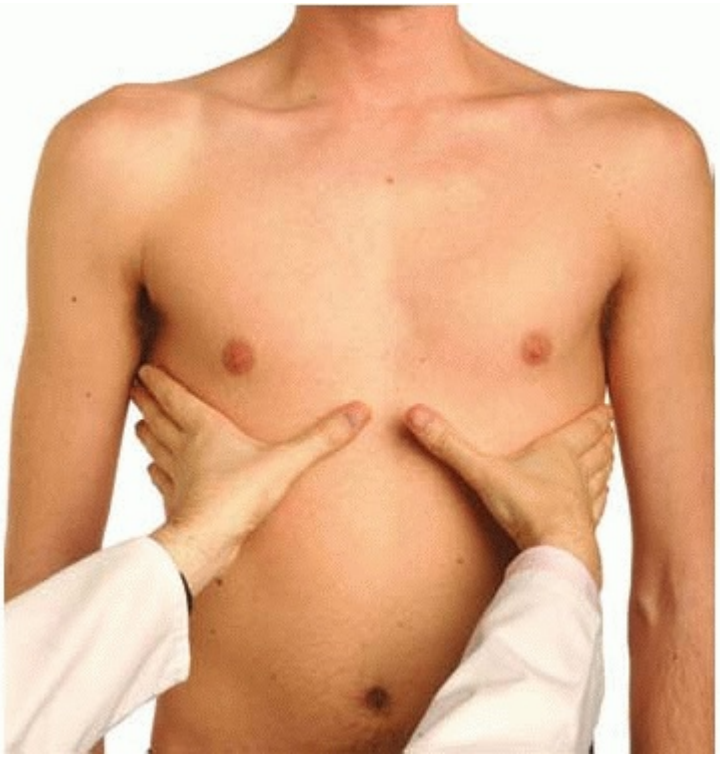

Fig. 8.4 Placement of the hands for testing chest expansion. Anchor with the fingers and leave the thumbs free-floating.

Percussion

Technique

This takes some practise to master fully so serves as an excellent indicator of how much time the medical student has spent on the wards. Students would do well to ensure they are proficient at this.

The aim is to tap the chest by the standard method and listen to and feel for the resultant noise (see Fig. 8.5 and 8.6). For a *right-handed* doctor:

- Place the left hand on the chest wall, fingers separated and lying between the ribs.
- Press the middle finger firmly against the chest.
- Using the middle finger of the right hand, strike the middle phalanx of the middle finger of the left hand (Fig. 8.5).

- The striking finger should be moved away again quickly as keeping it pressed on the left hand may muffle the noise.
- The right middle finger should be kept in the flexed position, the striking movement coming from the wrist (much like playing the piano).

Medical students quickly learn to keep the middle fingernail of their right hand well-trimmed!

- Students should practise on themselves and each other. Learn the different sounds produced percussing over the lung and the more dense liver just below.
- In clinical practice, one should percuss each area of the lung, each time comparing right then left, as shown opposite.
- Don't forget the apices which can be assessed by percussing directly onto the patient's clavicle (no left hand needed).
- If an area of dullness if heard (or felt) this should be percussed in more detail so as to map out the borders of the abnormality.

Findings

- Normal lung sounds *'resonant'*.
- *'Dullness'* = heard over areas of ↑ density (consolidation, collapse, alveolar fluid, pleural thickening, peripheral abscess, neoplasm).
- *'Stony dullness'* = the unique extreme dullness heard over a pleural effusion.
- *'Hyper-resonant'* = areas of ↓ density (emphysematous bullae or pneumothorax). NB COPD will create a globally hyper-resonant chest.

▸ There should be an area of dullness over the heart which may be diminished in hyperexpansion states (e.g. COPD or severe asthma).

▸The liver is manifested by an area of dullness below the level of the 6th rib anteriorly on the right. This may also be lost with hyperinflated lungs.

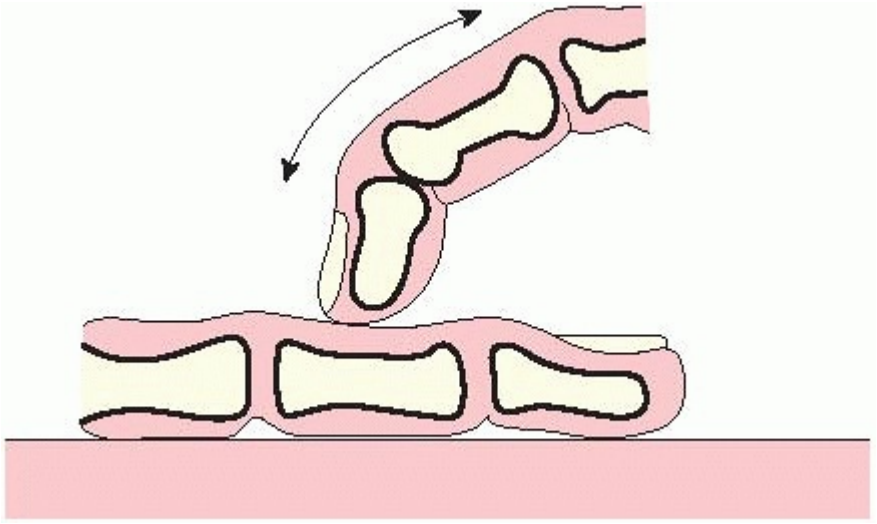

Fig. 8.5 Strike the middle phalanx of the middle finger of the left hand with the middle finger of the right hand.

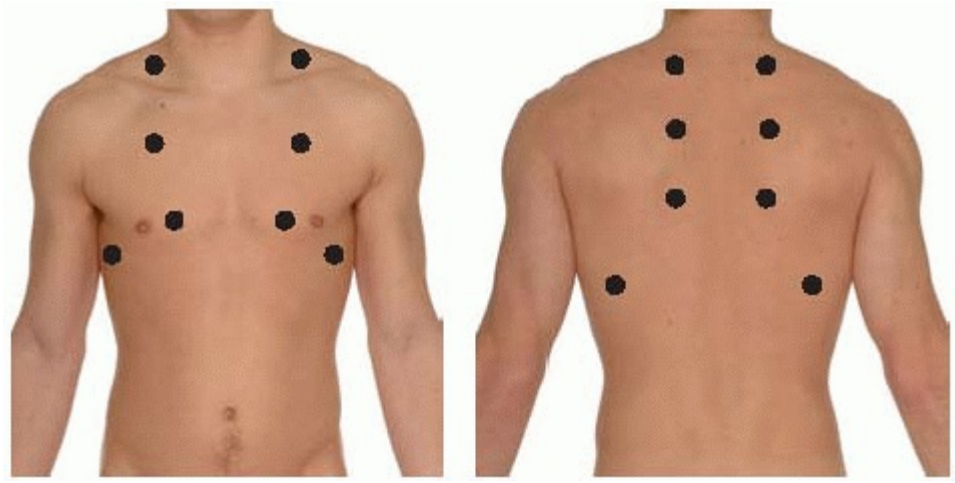

Fig. 8.6 Areas of the chest to percuss. Test right versus left for each area, front and back.

Auscultation

Technique

The diaphragm should be used except where better surface contact is needed in very thin or hairy patients.

- Ask the patient to 'take deep breaths in and out through the mouth'.
- Listen to both inspiration and expiration.
- Listen over the same areas percussed, comparing left to right.
- If an abnormality is found, examine more carefully and define borders.
- Listen for the breath sounds and any added sounds—and note at which point in the respiratory cycle they occur.

Many patients have difficulty performing correctly here. They may take one deep breath and hold it, may breath through the nose, or may take only one breath. Simple prompts ('keep going, in and out') will help. A brief demonstration will usually solve things if all else fails.

Findings

Breath sounds

- ***Normal:*** 'vesicular'. Produced by airflow in the large airways and larynx and altered by passage through the small airways before reaching the stethoscope. Often described as 'rustling'. Heard especially well in inspiration and early expiration.
- *Reduced sound:* if local = effusion, tumour, pneumothorax, pneumonia or lung collapse. If global = COPD or asthma (the 'silent chest' is a sign of a life threatening asthma-attack).
- *Bronchial breathing:* caused by ↑ density of matter in the peripheral lung allowing sound from the larynx to the stethoscope unchanged. Has a 'hollow, blowing' quality, heard equally in inspiration and expiration, often with a brief pause between. (Think of a certain black-helmeted villain in a popular space movie franchise.) A similar sound can be heard by listening over the trachea in the neck. Heard over consolidation, lung abscess at the chest wall and dense fibrosis. Also heard at the upper border of a pleural effusion.

Added sounds

- *Wheeze (rhonchi):* musical whistling sounds caused by narrowed airways. Heard easier in expiration.
  - Different calibre airways = different pitch note ∴ asthma and COPD can cause a chorus of notes termed 'polyphonic wheeze'.
  - 'Monophonic' wheeze indicates a single airway is narrowed, usually by a foreign body or carcinoma.
  - Not a good marker of disease severity as ↓ air entry → ↓ wheeze!
- *Crackles (crepitations, râles):* caused by air entering collapsed airways and alveoli producing an opening snap. Heard in inspiration.

- *'Coarse'* crackles made by larger airways opening and sound like the snap and pop of a certain breakfast cereal. Causes: fluid or infection.

- *'Fine'* crackles occur later in inspiration. The sounds like the tear of 'Velcro®' and can also be reproduced by rolling the hair at your temples between the thumb and forefinger. Causes: fluid, infection or fibrosis (particularly at lung bases).
- Crackles are often a normal finding at the lung bases, If so, they will clear after asking the patient to cough.

- *Rub:* creaking sound likened to the bending of new leather or the creak of a footstep in fresh snow. Heard at the height of inspiration. Caused by inflamed pleural surfaces rubbing against each other.
  - Causes: pneumonia, pulmonary embolism with infarction.
  - Movement of the stethoscope on the chest wall sounds similar.

Vocal resonance

- Auscultatory equivalent of vocal fremitus.
- Low pitched sounds transmit well so create a vocal 'booming' quality.
- Ask the patient to say 'ninety-nine' or 'one, one, one' and listen over the same areas as before.
- The changes are the same as those for vocal fremitus.
- Marked ↑ resonance, such that a whisper can be clearly heard is termed 'whispering pectoriloquy'.

Important presenting patterns

Consolidation

- ↓ air entry locally, 2° to infection.
- ↓ chest wall movement locally.
- Dullness to percussion.
- Bronchial breathing or ↑ breath sounds.
- Coarse or fine crackles, localized.
- ↑ vocal resonance.

Collapse

- Blockage of a major airway and collapse of the distal lung segment.
- Mediastinal shift towards the abnormality.
- ↓ chest wall movement locally.
- Dullness to percussion restricted to affected lobe.
- ↓ breath sounds.
- ↓ vocal resonance.

Pleural effusion

- Collection of fluid between the 2 pleural layers, creating a sound barrier between the examiner and the patient's lung.
- Mediastinal shift away from the lesion (with a large effusion).
- ↓ chest wall movement locally.
- 'Stony' dull to percussion.
- ↓ breath sounds with bronchial breathing at the upper border.

- ↓ vocal resonance.
- Sometimes a pleural rub just above.

Pneumothorax

- Air in the pleural space.
- Mediastinal shift away from the lesion (with a tension pneumothorax).
- ↓ chest wall movement locally.
- Hyper-resonant to percussion.
- ↓ breath sounds.
- ↓ vocal resonance.

Interstitial fibrosis

- No mediastinal shift. Trachea may move towards the fibrosis in upper lobe disease).
- ↔ or ↓ chest wall movement.
- ↔ percussion note.
- ↔ breath sounds.
- ↔ vocal resonance.
- Fine crackles present.

The elderly patient

Up to 60% of older people may suffer respiratory symptoms, but less readily see their doctors about them. Lung function declines with age and exertional breathlessness rises, often with concurrent (non-respiratory) illnesses. Careful, thoughtful assessment is therefore vital.

History

- ***Clarify diagnosis:*** not all disease in elders is COPD and many older people are lifelong non-smokers. Asthma and pulmonary fibrosis are often underdiagnosed.
- ***Fatigue:*** often associated with chronic respiratory illnesses and this may be more disabling to individuals than respiratory symptoms themselves.
- ***DHx:*** should be comprehensive and 'dovetail' other medical problems. Anticholinergic drugs (e.g. atrovent) may precipitate glaucoma or worsen bladder and bowel symptoms, so be thorough. Ask about vaccinations—many miss their annual 'flu vaccine through hospitalization. Consider vaccination in hospital.
- ***Nutrition and mood:*** undernutrition is common with chronic diseases and those in long-term care, impacting on illnesses with higher resting metabolic rates (e.g. COPD). Low mood is similarly common and should be sought.
- ***SHx:*** functional history is paramount and may reveal key interventions. A thorough occupational history is vital; many people do not know they have worked/lived with someone exposed to e.g. asbestos.

Examination

- ***General:*** poorly fitting clothes/dentures may point to weight loss (undernutrition, chronic disease, malignancy).
- ***Hands:*** arthritis/other deformities may make inhaler use difficult and point to related diagnoses (e.g. rheumatoid lung disease). Clubbing may not be present in later onset pulmonary fibrosis.
- ***Chest:*** beware 'basal crepitations' which are common in older age. Pick out discriminating signs—tachypnoea, *position* of crackles, added sounds etc.
- ***Inhaler technique:*** key examination; may reveal why prior treatments were unsuccessful.

Diagnoses not to be missed

- ***Asthma:*** up to 8% of over-60s, but under-recognized and undertreated. Spirometry is a key investigation.
- ***Tuberculosis:*** ↑ in the elderly—through reactivation, chronic illness, undernutrition. Presents non-specifically—cough, lethargy, weight loss.

We thank Dr Richard Fuller for contributing this page.

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Chapter 9

The Abdomen

Applied anatomy

The abdomen includes the perineum, external and internal genitalia and the inguinal regions. However, these components are discussed in  Chapters 12 and 13.

Boundaries

The abdomen is defined as the region lying between the thorax above and the pelvic cavity below. The anterior abdominal wall is bounded by the 7^{th} to 12^{th} costal cartilages and the xiphoid process of the sternum superiorly and the inguinal ligaments and pelvic bones inferiorly.

The abdominal cavity is separated from the thoracic cavity by the diaphragm. There is no such delineation, however, between the abdomen and the pelvis and, as a consequence, definitions vary.

Abdominal contents

The abdomen contains structures which form part of just about every body system.

The ***digestive*** organs of the oesophagus, stomach, small intestine, large intestine and the associated organs (liver, gall bladder and biliary system, exocrine pancreas) all lie within the abdomen. The ***endocrine*** portion of the pancreas, the adrenal glands and gonads represent the endocrine system. From the ***cardiovascular*** system the abdominal aorta with its important branches to the liver, spleen, intestine, kidneys, and lower limbs. The ***immunological*** system is represented by the spleen. The multiple lymph nodes surrounding the aorta and intestines and the MALT tissue within the intestine itself. The whole of the ***urinary*** system is present (kidneys, ureters, bladder, and urethra).

It is worth remembering that, much like the thorax, the abdomen is lined by a rather thin layer of membranous tissue: the *peritoneum*. This is a double lining—the 'parietal' peritoneum covers the internal surface of the abdominal walls whilst the 'visceral' peritoneum covers the organs. Between the two layers (the 'peritoneal cavity') is a small amount of fluid which acts a lubricant allowing the abdominal contents to move against each other as the body changes position or, for example, as the gut contorts with peristalsis.

A select few organs lie behind the peritoneum on the posterior abdominal wall. They are the pancreas, a portion of the duodenum, the ascending and descending colon and the kidneys.

Abdominal regions

The anterior abdominal wall is artificially divided into 9 portions for descriptive purposes. 4 imaginary lines can be drawn (see Fig. 9.1)…

- 1 horizontal line between the anterior superior iliac spines.
- 1 horizontal line between the lower border of the ribs.
- 2 vertical lines at the mid-clavicular point.

To make life easier, the abdomen can also be simply divided into 4 quadrants by imagining 1 horizontal and 1 vertical line crossing at the umbilicus (see Fig. 9.2).

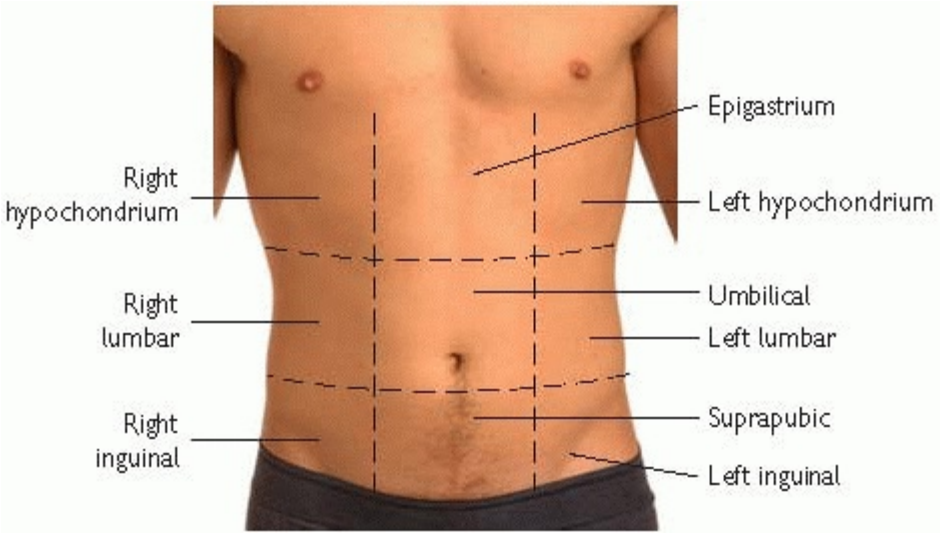

Fig. 9.1 The 9 segments of the anterior abdominal wall. Students should familiarize themselves with these along with the organs lying in each area.

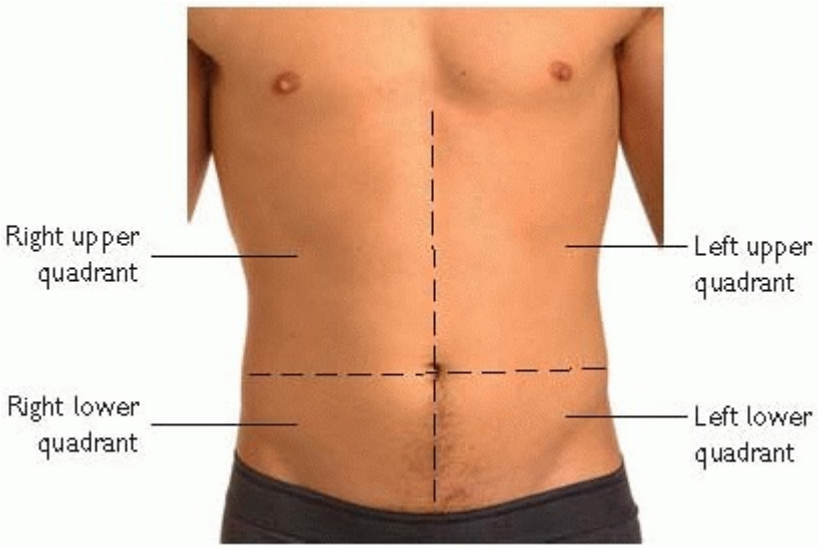

Fig. 9.2 The 4 quadrants of the anterior abdominal wall.

Oesophageal symptoms

Dysphagia

This is difficulty swallowing and is the principal symptom of oesophageal disease. (See Box 9.1 for important causes.) When a patient complains of dysphagia you should attempt to establish:

- ***Level of obstruction:*** where does the patient feel the food/liquid sticking? Patients can often point to a level on the chest although the sensation usually correlates poorly with the actual level of obstruction.
- ***Onset:*** How quickly did the symptoms emerge? Obstruction caused by cancer, for example, may progress rather rapidly over a few months whereas those with a benign peptic stricture may describe a very long history of GORD and progressive dysphagia.
- ***Course:*** intermittent? Present for only the first few swallows (lower oesophageal ring, spasm)? Progressive (cancer, stricture, achalasia)?
- ***Solids/liquids:*** both solids and liquids being affected equally suggests a motor cause (achalasia, spasm). However, if solids are affected more than liquids, some physical obstruction is more likely (e.g. cancer).
- ***Associated symptoms:*** heartburn (leads to oesophageal strictures), weight loss, wasting, fatigue (perhaps suggestive of cancer). Coughing and choking suggest 'pharyngeal dysphagia' due to motor dysfunction (e.g. motor neuron disease causing bulbar- or pseudobulbar palsy).

Odynophagia

This is pain on swallowing. Usually a rather unpleasant substernal sensation *during* the swallow and suggestive of oesophageal inflammation (infective oesophagitis—candidida, herpes, cytomegalovirus; peptic ulceration; caustic damage; oesophageal perforation).

Remember to ask of potential causes during the DHx.

Heartburn and acid reflux

Also known as gastro-oesophageal reflux disease (GORD). It is caused by the regurgitation of stomach contents into the oesophagus due to an incompetent anti-reflux mechanism at the gastro-oesophageal junction.

Typical features

- *Site:* mid-line, retrosternal
- *Radiation:* to the throat and occasionally the infra-scapular regions
- *Nature:* 'burning'
- *Aggravating factors:* worse after meals and when performing postures which raise the intra-abdominal pressure (bending, stooping, lying supine). Also worse during pregnancy.
- *Associated symptoms:* Often accompanied by acid or bitter taste (acid regurgitation) or sudden filling of the mouth with saliva* ('waterbrash')

Acid reflux may be worsened by certain foods (alcohol, caffeine, chocolate, fatty meals) and some drugs (calcium channel blockers, anticholinergics) which act to ↓ the GOJ sphincter pressure.

Hiatus hernia is another important cause of reflux symptoms—be sure to enquire about this in the history.

Dyspepsia

Commonly known as indigestion. Very common and presents as a variety of symptoms including:

- Upper abdominal discomfort
- Bloating
- Belching.

You should be on the alert for features suggestive of a serious pathology (anaemia, weight loss, dysphagia, PR blood loss, melaena, and abdominal masses).

Box 9.1 Some causes of dysphagia

- ***Oral:*** painful mouth ulceration, oral, or throat infections.
- ***Neurological:*** cerebrovascular event, bulbar and pseudobulbar palsies, myasthenia gravis.
- ***Dysmotility:*** achalasia, systemic sclerosis, presbyoesophagus.
- ***Mechanical:*** pharyngeal pouch, oesophageal cancer, peptic stricture, other benign strictures, extrinsic compression of the oesophagus (e.g. large lung or thyroid tumour).

Nausea, vomiting, and vomitus

Nausea and vomiting

- ***Nausea****:* a feeling of sickness—the inclination to vomit. It usually occurs in waves and may be associated with retching or heaving. It can last from seconds to days depending on the cause.
- ***Vomiting (emesis):*** usually follows nausea and autonomic symptoms such as salivation. It is the forceful expulsion of the gastric contents by reflex contractions of the thoracic and abdominal muscles.

The ‘vomiting centre’ is in the medulla and is composed of many efferent nuclei in serial communication with each other. When the entire circuit is activated by afferent stimuli, the complete set of actions required to cause vomiting are triggered.

Timing

You should be clear on exactly when the vomiting tends to occur—particularly its relation to meals e.g. vomiting delayed for >1 hour after meals is suggestive of gastro-oesophageal obstruction or gastroparesis. Early morning vomiting is typical of pregnancy or raised intracranial pressure.

Nature of the vomitus

Although unpleasant, you should enquire about the exact nature of any vomited material and attempt to see a sample, if possible.

Blood (haematemesis)

Presence of blood indicates bleeding in the upper gastrointestinal tract (oesophagus, stomach, duodenum). A history of bleeding must be explored in the context of other abdominal symptoms. Ask especially about:

- The amount of blood and exact nature of it (see Box 9.3).
- Previous bleeding episodes, treatment and outcome (e.g. previous surgery?)
- Cigarette smoking.
- Use of drugs such as aspirin, NSAIDs and warfarin.
-  Remember to ask about weight loss, dysphagia, abdominal pain and melaena (consider the possibility of neoplastic disease).

Bile

Assess the presence or absence of bile. Remember that bile comes largely in 2 colours—the green pigment (biliverdin) often seen to colour the vomitus in the absence of undigested food. The yellow pigment (bilirubin) appears as orange, often occurring in small lumps.**

Undigested food without bile suggests a lack of connection between the stomach and the small intestine (e.g. pyloric obstruction).

Box 9.2 Important causes of vomiting

- ***Acute:*** GI tract infections (viral gastroenteritis e.g. ‘food poisoning’ Norwalk, viral hepatitis), systemic bacterial infection, mechanical bowel obstruction, alcohol intoxication, acute upper GI bleed, urinary tract infection.
- ***Chronic:*** pregnancy, uraemia, drugs (narcotics, digitalis, aminophylline, cancer chemotherapy), gastroparesis (diabetes mellitus, scleroderma, drugs).
- ***Other:*** peptic ulcer disease, motor disorders (post-surgery or autonomic dysfunction), hepatobiliary disease, alcoholism, cancer.

▸ Don't forget about central nervous system and vestibular problems.

Box 9.3 Causes of upper GI bleeding

- Peptic ulceration
- Erosive or ulcerative oesophagitis
- Gastritis
- Varices (oesophageal/gastric)
- Gastric and oesophageal tumours
- Mallory-Weiss tear
- Dieulafoy's lesion
- Vascular anomalies (e.g. angiodysplasia, AV malformation)
- Herediatory haemorragic telangectasia
- Connective tissue disorders
- Vasculitis
- Bleeding disorders.

Box 9.4 Nature of haematemesis

- ***Large volume of fresh, red*** blood suggests active bleeding (co-incident liver disease and/or heavy alcohol intake may suggest bleeding oesophageal varices, abdominal pain and heartburn suggest a gastric or oesophageal source such as a peptic ulceration or GORD).
- ***Small streaks*** at the end of prolonged retching may indicate minor oesophageal trauma at the GOJ (Mallory-Weiss tear).
- ***Coffee-grounds:*** this is the term used for blood that has been 'altered' by exposure to stomach acid. It appears brown and in small lumps.

Abdominal pain

Like pain in any other region, abdominal pain may present in very different ways and has many different causes. You should establish the site, radiation, severity, character, frequency, duration, any exacerbating or relieving factors, and associated symptoms.

Site

Like most organs, those in the abdomen cannot be felt directly—the pain is referred to areas of the abdominal wall according to the organ's embryological origin (see Fig. opposite).

- Ask the patient to point to the area affected. They often find this challenging and may indicate a wide area. In this case, ask them to 'use one finger' and point to the area of maximum intensity.

'Use one finger and point to where the pain is worst.'

Box 9.5 Sites of abdominal pain and embryologic origins

- ***Epigastric:*** foregut (stomach, duodenum, liver, pancreas, gallbladder)
- ***Periumbilical:*** midgut (small and large intestines including appendix)
- ***Suprapubic:*** hindgut (rectum and urogenital organs)

A very localized pain may originate from the parietal peritoneum. E.g. appendicitis—may begin as an umbilical pain (referred from the appendix) then 'move' to the right iliac fossa as the inflammation spreads to the peritoneum overlying the appendix.

Radiation

Ask the patient if the pain is felt elsewhere or if they have any other pains (they may not associate the radiated pain with the abdominal pain). Some examples include:

- Right scapula: gallbladder
- Shoulder-tip: diaphragmatic irritation
- Mid-back: pancreas.

Character

Ask the patient what *sort* of pain it is. Give some examples if they have trouble but be careful not to lead the patient. A couple of examples include:

- ***Colicky:*** this is pain that comes and goes in waves and indicates obstruction of a hollow, muscular-walled organ (intestine, gall-bladder, bile duct, ureter).
- ***Burning:*** usually indicates an acid cause and is related to the stomach, duodenum or lower end of the oesophagus.

Aggravating/relieving factors

Ask the patient what appears to make the pain better or worse—or what they do to get rid of the pain if they suffer from it often.

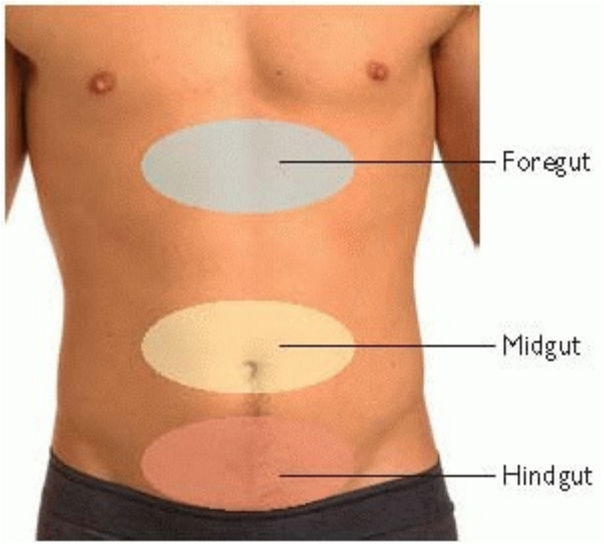

Fig. 9.3 Typical sites of pain according to origin.

Findings

Some characteristic pains:

- ***Renal colic:*** colicky pain at the renal angles ± loins, which are tender to touch, radiating to the groins/testicles/labia. Typically, the patient writhes around, unable to find a position that relieves the pain.
- ***Bladder pain:*** a diffuse severe pain in the suprapubic region.
- ***Prostatic pain:*** a dull ache which may be felt in the lower abdomen, rectum, perineum or anterior thighs.
- ***Urethral pain:*** variable in presentation ranging from a 'tickling' discomfort to a severe sharp pain felt at the end of the urethra (tip of the penis in males) and exacerbated by micturition. Can be so severe that patients attempt to 'hold on' to urine causing yet more problems!
- ***Small bowel obstruction:*** colicky central pain associated with vomiting, abdominal distension ± constipation.
- ***Colonic pain:*** as above under 'small bowel' but sometimes temporarily relieved by defaecation or passing flatus.
- ***Bowel ischaemia:*** dull, severe, constant, right upper quadrant/central abdominal pain exacerbated by eating.
- ***Biliary pain:*** severe, constant, right upper quadrant/epigastric pain that can last hours and is often worse after eating fatty foods.
- ***Pancreatic pain:*** epigastric, radiating to the back and partly relieved by sitting up and leaning forward.
- ***Peptic ulcer pain:*** dull, burning pain in the epigastrium. Typically episodic at night, waking the patient from sleep. Exacerbated by eating and sometimes relieved by consuming milk or antacids.

Bowel habit

Patients should be asked how often they open their bowels and if this has changed recently. Ask also about the other symptoms on these pages.

Constipation

A disorder that can mean different things to different people. Normal bowel habit ranges from 3 times/day to once every 3 days.

'Constipation' is the passage of stool <3 times/week, or stools that are hard or difficult to pass.

A thorough history should include:

- Duration of constipation.
- Stool size and consistency.
- Straining, particularly at the end of evacuation.
- Associated symptoms (nausea, vomiting, weight loss).
- Pain on defaecation.

- Rectal bleeding.
- Intercurrent diarrhoea?
- Fluid and fibre intake.
- Depression, lack of exercise.
- DHx (prescription and over-the-counter). Particularly codeine, antidepressants, aluminium and calcium antacids.
- Metabolic or endocrine diseases (thyroid disorders, hypercalcaemia, diabetes, phaechromocytoma, Hirshsprung's disease).
- Neurological problems (autonomic neuropathy, spinal cord injury, multiple sclerosis).

Diarrhoea

Defined as an increase in stool volume (>200ml daily) and frequency (3/day). Also a change in consistency to semi-formed or liquid stool.

You should establish the time course since acute diarrhoea is suggestive of infection. Ask especially about:

- Colour, consistency, offensive smell, ease of flushing.
- Duration.
- Does the diarrhoea disturb the patient's sleep?
- Is there any blood, mucus, or pus?
- Associated pain or colic?
- Is there urgency?
- Nausea, vomiting, weight loss?
- Any difference if the patient fasts?
  - No change in 'secretory' diarrhoea—e.g. *E. coli, Staph. aureus*.
  - Disappears on fasting: 'osmotic' diarrhoea.
- Foreign travel.
- Recent antibiotics.

Box 9.6 Some causes of constipation

- Low-fibre diet.
- Physical immobility.
- Functional bowel disease.
- Drugs (e.g. opiates, antidepressants, aluminium, antacids).
- Metabolic and endocrine diseases (e.g. hypothyroidism, hypercalcaemia, hypokalaemia, diabetes mellitus, porphyria, phaeochromocytoma).
- Neurological disorders (e.g. autonomic neuropathy, spinal cord injury, multiple sclerosis).
- Colonic stricture.
- Anorectal disease (e.g. anal fissure—causes pain to the extent that the patient may avoid defaecating altogether).
- Habitual neglect.
- Depression.
- Dementia.

Box 9.7 Some causes of diarrhoea

- ***Malabsorption:*** may cause steatorrhoea, a fatty, pale stool which is extremely odorous and difficult to flush. See box on p.233.
- ↑ ***intestinal motility:*** hyperthyroidism, irritable bowel syndrome (see below).
- ***Exudative:*** inflammation of the bowel causes small volume, frequent stools, often with blood or mucus. (e.g. colonic carcinoma, Crohn's disease, ulcerative colitis).
- ***Osmotic:*** large volume of stool which disappears with fasting. Causes include lactose intolerance, gastric surgery).
- ***Secretory:*** high volume of stool which persists with fasting. No pus, blood or excessive fat. Causes include: gastrointestinal

infections, carcinoid syndrome, villous adenoma of the colon, Zollinger-Ellison syndrome, VIP*-secreting tumour.

Rectal bleeding and melaena

There are many causes of PR blood-loss but, as always, a detailed history will help. Determine:

- The amount.
  - Small amounts can appear dramatic, colouring toilet water red.
- The nature of the blood (red, brown, black).
- Is it mixed with the stool or 'on' the stool?
- Is it spattered over the pan, with the stool or only seen on the paper?
- Any associated features (mucus may indicate inflammatory bowel disease or colonic cancer).

Melaena

This is jet-black, tar-like and pungent-smelling stools representing blood from the upper GI tract (or right side of the large bowel) that has been 'altered' by passage through the gut.

The presence of melaena is often queried in hospital in-patients but those who have smelt true melaena rarely forget the experience!

Ask about iron supplementation or bismuth-containing compounds—cause blackened stools but without the melaena smell or consistency.

Mucus

Clear, viscoid secretion of the mucus membranes.* Contains mucus, epithelial cells, leukocytes and various salts suspended in water.

The presence of mucus in, or on, stools may indicate:

- Inflammatory bowel disease.
- Solitary rectal ulcer.
- Small or large bowel fistula.
- Colonic villous adenoma.
- Irritable bowel syndrome.

Flatus

Small amounts of gas frequently escape from the bowel via the mouth (eructation) and anus and the notable excess of this is a common feature of both functional and organic disorders of the gastrointestinal tract.

Often associated with abdominal bloating and caused by the fermentation of certain foods by colonic flora.

Excessive flatus is a particular feature of:

- Hiatus hernia.
- Peptic ulceration.
- Chronic gall-bladder disease.
- Air-swallowing (aerophagy).
- High-fibre diet.

Box 9.8 Causes of lower GI bleeding

- Haemorrhoids.
- Anal fissure.
- Diverticular disease.

- Colonic carcinoma.
- Colonic polyp.
- Angiodysplasia.
- Inflammatory bowel disease.
- Ischaemic colitis.
- Meckel's diverticulum.
- Small bowel disease (e.g. tumour, diverticulae, intussusception, Crohn's).
- Solitary rectal ulcer.
- Haemobilia (bleeding into the biliary tree).

Box 9.9 Fat malabsorption (steatorrhoea)

A common feature of pancreatic insufficiency (e.g. due to chronic pancreatitis, cystic fibrosis). Also caused by diseases such as coeliac disease, inflammatory bowel disease, blind bowel loops, and short bowel syndrome.

You should be aware of these features and explore them all fully if one is mentioned by the patient:

- Pale stool.
- Offensive smelling.
- Poorly formed.
- Difficult to flush (floats).

Jaundice and pruritus

Jaundice

Jaundice ('icterus') is a yellow pigmentation of skin, sclera, and mucosa caused by excess bilirubin in the body fluids. It is usually considered a 'sign' as it is seen on examination. See also p.58 and Box 9.10.

Ask about:

- The colour of the urine (dark in cholestatic jaundice).
- The colour and consistency of the stools (pale in cholestatic jaundice).
- Abdominal pain (e.g. caused by gallstones).

The following should be included in any thorough history but you should make a special point of asking about:

- Previous blood transfusions.
- Past history of jaundice.
- Drugs (e.g. antibiotics, NSAIDs, oral contraceptives, phenothiazines).
- IV drug use.
- Tattoos and body piercing.
- Foreign travel.
- Sexual history.
- FHx of liver disease.
- Alcohol consumption.
- Any personal contacts who also have jaundice.

Pruritus

This is itching of the skin and may be either localized or generalized.

It has many causes—it is particularly associated with cholestatic liver disease (e.g. primary biliary cirrhosis, sclerosing cholangitis).

Abdominal swelling

The five classic causes of abdominal swelling ('the 5 Fs') are shown opposite in Box 9.11. To these, you should also add 'tumour'.

In decompensated cirrhosis, a combination of portal (sinusoidal) hypertension and Na and H_2O retention favours the transudation of fluid into the peritoneal cavity (ascites). The resultant swelling may be unsightly—it can also cause shortness of breath by putting pressure on the diaphragm from below, particularly when supine and may be associated with pleural effusions.

See p.737 for the causes and classification of ascites.

Box 9.10 Causes of jaundice

Prehepatic

- Haemolysis.
- Gilbert's disease.
- Dubin-Johnson syndrome.
- Rotor syndrome.
- Haemodialysis.

Hepatocellular

- Cirrhosis (and the causes thereof—see *OHCM6*, p.232).
- Acute hepatitis (viral, alcoholic, autoimmune, drug-induced).
- Liver tumours.
- Cholestasis from drugs (e.g. chlorpromazine).

Posthepatic

Obstruction of biliary outflow due to:

- *Luminal obstruction:* gallstones.
- *Wall pathology:* congenital bile duct abnormalities, primary biliary cirrhosis, trauma, tumour.
- *External compression:* pancreatitis, lymphadenopathy, pancreatic tumour, Ampulla of Vater tumour.

Box 9.11 Five causes of abdominal swelling—'the 5 Fs'

- Fat.
- Fluid.
- Flatus.
- Faeces.
- Fetus.

Urinary and prostate symptoms

Urinary frequency

This is the passing of urine more often than is normal for the patient. Quantify this—how many times in a day—and also ask about the volume of urine passed each time (you are attempting to decide whether the patient is producing more urine than normal or simply feeling the urge to urinate more than normal).

Urgency

This is the sudden need to urinate, a feeling that the patient may not be able to make it to the toilet in time. Ask about the volume expelled.

Nocturia

Urination during the night. Does the patient wake from sleep to urinate? How many times a night? How much urine is expelled each time?

Urinary incontinence

The loss of voluntary control of bladder emptying. Patients may be hesitant to talk about this so try to avoid the phrase 'wetting yourself. You could ask about it immediately after asking about urgency… 'Do you ever feel the desperate need to empty your bladder?… Have you ever not made it in time?' or by asking about a 'loss of control'.

There are 5 main types of urinary incontinence:

- ***'True':*** total lack of control of urinary excretion. Suggestive of a fistula between the urinary tract and the exterior or a neurological condition.
- ***Giggle:*** incontinence during bouts of laughter. Common in young girls.
- ***Stress:*** leakage associated with a sudden ↑ in intra-abdominal pressure of any cause (e.g. coughing, laughing, sneezing).
- ***Urge:*** intense urge to urinate such that the patient is unable to get to the toilet in time. Causes include over-activity of the detruser muscle, urinary infection, bladder stones and bladder cancer.
- ***Dribbling or overflow:*** continual loss of urine from a chronically distended bladder. Typically in elderly males with prostate disease.

Terminal dribbling

A male complaint and usually indicative of prostate disease. This is a dripping of urine from the urethra at the end of micturition, requiring an abnormally protracted shake of the penis and may cause embarrassing staining of clothing.

Hesitancy

Difficulty in starting to micturate. The patient describes standing and waiting for the urine to start flowing. Usually due to bladder outflow obstruction due to prostatic disease or strictures.

Dysuria

'Pain on micturition' usually described by the patient as 'burning' or 'stinging' and felt at the urethral meatus. Ask whether it is throughout the passage of urine or only at the end ('terminal dysuria').

Haematuria

The passage of blood in the urine. Always an abnormal finding.

 Remember that 'microscopic haematuria' will be undetectable to the patient, only showing on dip-testing.

Incomplete emptying

This is the sensation that there is more urine left to expel at the end of micturition. Suggests detruser dysfunction or prostatic disease.

Intermittency

The disruption of urine flow in a stop-start manner. Causes include prostatic hypertrophy, bladder stones, and ureterocoeles.

Oliguria and anuria

Oliguria is scanty or low-volume urination and is defined as the excretion of <300ml urine in 24 hours. Causes can be physiological (dehydration) or pathological (intrinsic renal disease, shock or obstruction).

Anuria is the absence of urine formation and you should attempt to rule out urinary tract obstruction as a matter or urgency. Other causes include severe intrinsic renal dysfunction and shock.

Polyuria

This is excessive excretion of large volumes of urine and must be carefully differentiated from urinary frequency (the frequent passage of small amounts of urine).

Causes vary widely but include the ingestion of large volumes of water (including hysterical polydipsia), diabetes mellitus (the osmotic effect of glucose in the tubules encourages more urine to be made), failure of the action of ADH at the renal tubule (as in diabetes insipidus) and defective renal concentrating ability (e.g. chronic renal failure).

 Remember also to ask the patient about the use of diuretic medication!

Appetite and weight

Loss of appetite and changes in weight are rather non-specific symptoms but should raise suspicion of a serious disease if either is severe,

prolonged, or unexpected.

▸ Remember that weight loss has many causes outside of the abdomen and a thorough systems enquiry should be conducted.

Weight loss may not be noticed by patients if they don't regularly weigh themselves—ask about clothes becoming loose.

Remember that the patient may have been intentionally losing weight—throwing you off the scent. Ask if the loss is 'expected'.

▸ Beware! Ascites weights 1kg/L and some patients with liver failure may have 10—20L of ascites, masking any 'dry weight' loss.

Ask the patient about their eating habit and average daily diet.

Try to determine:

- When the symptom was first noticed.
- Quantify the problem. In the case of weight loss, determine exactly how and over what time period.
- The cause of the anorexia—does eating make the patient feel sick?
- Does eating cause pain? (E.g. gastric ulcer, mesenteric angina, pancreatitis.)
- Any accompanying symptoms (abdominal pain, nausea, vomiting, fever).

Ask also about:

- The colour and consistency of stools (e.g. steatorrhoea?).
- Urinary symptoms (see p.236
- Recent change in temperature tolerance.

In every case, you should calculate the patient's BMI as on p.66.

The combination of weight loss with ↑ appetite may suggest malabsorption or a hypermetabolic state (e.g. thyrotoxicosis).

The rest of the history

Past medical history

Ask especially about:

- Previous surgical procedures including peri- and postoperative complications and anaesthetic complications.
- Chronic bowel diseases (e.g. IBD including recent flare-ups and treatment to date).
- Possible associated conditions (e.g. diabetes with haemachromatosis).

Drug history

Think about drugs that can precipitate abdominal diseases and remember to ask about over-the-counter drugs. For example:

- ***Hepatitis:*** halothane, phenytoin, chlorothiazides, pyrazinamide, isoniazid, methyl dopa, HMG CoA reductase inhibitors ('statins'), sodium valproate, amiodarone, antibiotics, NSAIDs.
- ***Cholestasis:*** chlorpromazine, sulphonamides, sulphonylureas, rifampicin, nitrofurantoin, anabolic steroids, oral contraceptive pill.
- ***Fatty liver:*** tetracycline, sodium valproate, amiodarone.
- ***Acute liver necrosis:*** paracetamol.
- Ask also about previous blood transfusions.

Smoking

Smokers are at ↑ risk of peptic ulceration, oesophageal cancer, colorectal cancer. Smoking may also have a detrimental outcome on the natural history of Crohn's disease. There is some evidence that smoking may protect against ulcerative colitis.

Alcohol

As always, a detailed history is required—see p.44. If dependence is suspected, run through the CAGE questionnaire—see Box 9.12.

Family history

Ask especially about a history of inflammatory bowel disease, coeliac disease, peptic ulcer disease, hereditary liver diseases (e.g. Wilson's, haemochromatosis) bowel cancer, jaundice, anaemia, splenectomy, and cholecystectomy.

Social history

- Risks of exposure to hepatotoxins and hepatitis through occupation.
- Tattoos.
- Illicit drug use (especially sharing needles).
- Social contacts with a similar disease (particularly relevant to jaundice).
- Recent foreign travel.

Dietary history

- Amount of fruit, vegetables and fibre in the diet.
- Evidence of lactose intolerance.
- Change in symptoms related to eating certain food groups.
- Sensitivities to wheat, fat, caffeine, gluten.

Box 9.12 The CAGE questionnaire

A positive response to any of the 4 questions may indicate someone at risk of alcohol abuse. A positive answer to 2 or more questions makes the presence of alcohol dependence likely.

C Have you ever felt that you should **C**ut down your drinking?

A Have you ever got **A**ngry when someone suggested that you should cut down?

G Do you ever feel **G**uilty about your drinking?

E Do you ever need an '**E**ye-opener' in the morning to steady your nerves or get rid of a hangover?

Outline examination

As always, ensure adequate privacy. Ideally the patient should be lying flat with the head propped on a single pillow, arms lying at the sides.

The abdomen should be exposed at least from the bottom of the sternum to the symphysis pubis—preferably the whole upper torso should be uncovered. Do not expose the genitalia unless needed later.

The examination should follow an orderly routine. The authors' suggestion is shown below. It is standard practice to start with the hands and work proximally—this establishes a 'physical rapport' before you examine more delicate or embarrassing areas.

Box 9.13 Framework for the abdominal examination

- General inspection.
- The hands.

- The arms.
- The axillae.
- The face.
- The chest.
- Inspection of the abdomen.
- Palpation of the abdomen.
  - Light.
  - Deep.
  - Specific organs.
  - Examination of the hernial orifices.
  - External genitalia.
- Percussion (± examination of ascites).
- Auscultation.
- Digital examination of the anus, rectum ± prostate.

General inspection

Looking at the patient from the end of the bed to assess their general health and look for any obvious abnormalities described in Chapter 3 before moving closer. Look especially for:

- High or low body mass.
- The state of hydration.
- Fever.
- Distress.
- Pain.
- Muscle wasting.
- Peripheral oedema.
- Jaundice.
- Anaemia.

Hand and upper limb

Take the patient's right hand in yours and examine carefully for the following signs.

Nails[*]

- ***Leukonychia:*** whitening of the nail bed due to hypoalbuminaemia (e.g. malnutrition, malabsorption, hepatic disease, nephritic syndrome).
- ***Koilonychia:*** 'spooning' of the nails making a concave shape instead of the normal convexity. Causes include congenital and chronic iron deficiency.
- ***Muehrcke's lines:*** these are transverse white lines. Seen in hypoalbuminaemic states including severe liver cirrhosis.
- ***Clubbing:*** described on p.208. Abdominal causes are cirrhosis, inflammatory bowel disease and coeliac disease.
- ***Blue lunulae:*** a bluish discolouration of the normal lanulae seen in Wilson's disease.

[*] See also Chapter 4.

Palms

- ***Palmar erythema:*** 'liver palms'. This is a blotchy reddening of the palms of the hands, especially affecting the thenar and hypothenar

eminences. It can also affect the soles of the feet. Associated with chronic liver disease, pregnancy, thyrotoxicosis, rheumatoid arthritis, polycythaemia and (rarely) chronic leukaemia. It can also be a normal finding.

- ***Dupuytren's contracture:*** this is thickening and fibrous contraction of the palmar fascia. In early stages, palpable irregular thickening of the fascia is seen, especially that overlying the 4^{th} and 5^{th} metacarpals. This can progress to a fixed flexion deformity of the fingers starting at the 5^{th} and working across to the 3^{rd} or 2^{nd}. Often bilateral, it may also affect the feet. Seen especially in alcoholic liver disease but may also be seen in manual workers (or may be familial).
- ***Anaemia:*** pallor in the palmar creases suggests significant anaemia.

Hepatic flap (asterixis)

This is identical to the flap seen in hypercapnic states (see p.208).

Ask the patient to stretch out their hands in front of them with the hands dorsiflexed at the wrists and fingers outstretched and separated (see fig.).

The patient should hold that position for at least 15 seconds. If 'flap' is present, the patient's hands will move in jerky, irregular flexion/extension at the wrist and MCP joints. The flap is nearly always bilateral. May be subtle and intermittent.

This is characteristic of encephalopathy due to liver failure.

If a sign of hepatic encephalopathy in a patient with previously compensated liver disease, it may have been precipitated by infection, diuretic medication, electrolyte imbalance, diarrhoea or constipation, vomiting, centrally acting drugs, upper GI bleeding, abdominal paracentesis, or surgery.

The upper limb

Examine the arms for any signs of:

- ***Bruising:*** may be a sign of:
  - Hepatocellular damage and the resulting coagulation disorder.
  - Thrombocytopenia due to hypersplenism.
  - Marrow suppression with alcohol.
- ***Petechiae:*** pin-prick bleeds which do not blanche with pressure. Possibly a sign of thrombocytopenia.
- ***Muscle wasting:*** seen as a ↓ in muscle mass, possibly with overlying skin hanging loosely. A late manifestation of malnutrition and often seen in patients with chronic alcoholic liver disease.
- ***Scratch marks (excoriations):*** suggests itch (pruritus) is present and may be the only visible feature of early cholestasis.

Be careful not to miss AV fistulae or haemodialysis catheters!

The axillae

Examine carefully for:

- Lymphadenopathy
- Acanthosis nigricans (a thickened, blackening of the skin. Velvety in appearance. May be associated with intra-abdominal malignancy).

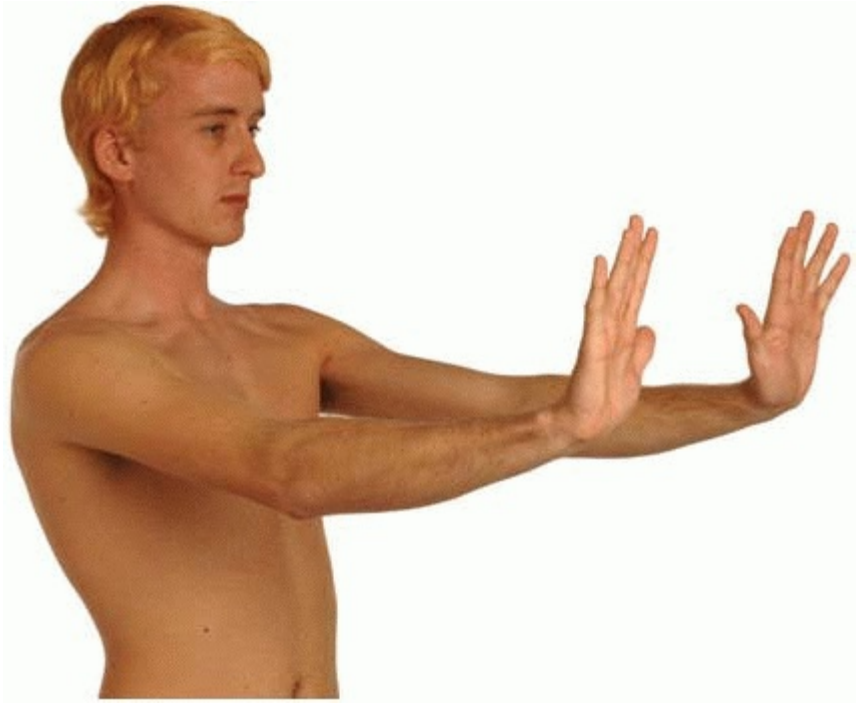

Fig. 9.4 Testing for hepatic flap. The patient should hold their arms outstretched with wrists dorsiflexed and fingers extended and abducted for at least 15 seconds.

Face and chest

Eyes

Ask the patient to look straight ahead whilst you look closely at their eyes, orbits and surrounding skin. Then ask the patient to look up whilst you gently retract the lower lid with a finger, looking at the underlying sclera and conjunctiva. Look especially for:

- ***Jaundice:*** a yellow discolouration of the sclera. This is usually the first place that jaundice can be seen. Particularly useful in patient with dark skin tones in whom jaundice would not be otherwise obvious.
- ***Anaemia:*** pallor of the conjunctivae. You'll need experience to spot this easily.
- ***Kayser-Fleisher rings:*** best seen with a slit-lamp in an ophthalmology clinic. A greenish-yellow pigmented ring just inside the cornea-scleral margin. Due to copper deposition. Seen in Wilson's disease.
- ***Xanthelasma:*** raised yellow lesions caused by a build up of lipids beneath the skin—often seen encircling the eyes, especially at the nasal side of the orbit.

Mouth

Ask the patient to show you their teeth then 'open wide' and look carefully at the state of the teeth, the tongue and the inner surface of the cheeks. You should also subtly attempt to smell the patient's breath.

- ***Angular stomatitis:*** a reddening and inflammation at the corners of the mouth. A sign of thiamine, vitamin B_{12}, and iron deficiencies.
- ***Circumoral pigmentation:*** Hyperpigmented areas surrounding the mouth. Seen in Peutz-Jegher's syndrome.
- ***Dentition:*** note false teeth or if there is evidence of tooth decay.
- ***Telangiectasia:*** dilatation of the small vessels on the gums and buccal mucosa. Seen in Osler-Weber-Rendu syndrome (📖 *OHCM6*, p.732).
- ***Gums:*** look especially for ulcers (causes include coeliac disease, inflammatory bowel disease, Behçet's disease and Reiter's syndrome) and hypertrophy (caused by pregnancy, phenytoin use, leukaemia, scurvy [vitamin C deficiency] or inflammation [gingivitis]).
- ***Breath:*** smell especially for:
  - *'Fetor hepaticus'* a sweet-smelling breath.
  - *Ketosis:* sickly sweet 'pear-drop' smelling breath

- *Uraemia:* a fishy smell

- ***Tongue:*** look especially for:
  - *Glossitis:* smooth, erythematous swelling of the tongue. Causes include deficiencies of iron, vitamin B_{12}, and folate deficiencies
  - *Macroglossia:* enlarged tongue. Causes include amyloidosis, hypothroidism, acromegaly, Down's syndrome, and neoplasia.
  - *Leukoplakia:* a white-coloured thickening of the tongue and oral mucus membranes. A premalignant condition caused by smoking, poor dental hygiene, alcohol, sepsis and syphilis.
  - *Geographical tongue*: painless red rings and lines on the surface of the tongue looking rather like a map. Can be caused by vitamin B_2 (riboflavin) deficiency or may be a normal variant.

- ***Candidiasis:*** 'thrush'. A fungal infection of the oral membranes seen as creamy white curd-like patches which can be scraped off revealing erythematous mucosa below. Causes include immunosuppression, antibiotic use, poor oral hygiene, iron deficiency and diabetes.

The neck

Examine the cervical and supraclavicular lymph nodes as on p.68.

Look especially for a supraclavicular node on the left-hand side which, when enlarged, is called Virchow's node (Troisier's sign-suggestive of gastric malignancy).

The chest

Look at the anterior chest and notice especially:

- ***Spider naevi:*** telangiectatic capillary lesions.
  - A central red area with engorged capillaries spreading out from it in a 'spidery' manner.
  - Caused by engorgement of capillaries from a central 'feeder' vessel.
  - If the lesion is truly a spider naevus, it will be completely eliminated by pressure at the centre using a pen-point or similar and will fill outwards when the pressure is released.
  - Can range in size from those that are only just visible up to 5 or 6mm in diameter.
  - Found in the distribution of the superior vena cava (see Fig. 9.5).
  - A normal adult is 'allowed' up to 5 spider naevi.
  - Causes include chronic liver disease and oestrogen excess
- ***Gynaecomastia:*** the excessive development of male mammary glands due to ductal proliferation such that they resemble post-pubertal female breasts.
  - This is often embarrassing for the patient so be sensitive.
  - Caused by alcoholic liver disease, congenital adrenal hyperplasia and several commonly used drugs including spironolactone, digoxin, and cimetidine.
  - Can also be seen during puberty in the normal male.

Fig. 9.5 Distribution of drainage to the superior vena cave and the area to look for spider naevi. The normal adult may have up to 5 such lesions.

Inspection of the abdomen

With the abdomen exposed, you should make a careful and methodical inspection. Note especially:

Scars

These may be the result of trauma or previous surgery. Recent scars will be pink and vascular. Old scars are white and may be indurated.

Abdominal distension

Does the abdomen look swollen? Consider the 5 Fs (📖 Box 9.11 p.34) and note the state of the umbilicus. (Everted? Deep?)

Focal swellings

Treat an abdominal swelling as you would do any other lump (📖 p.98) and bear in mind the underlying anatomy and possible organ involvement.

Divarication of the recti

Particularly in the elderly and in patients who have had abdominal surgery, the twin rectus abdominis muscles may separate laterally on contraction, causing the underlying organs to bulge through the resultant mid-line gap.

Ask the patient to lift their head off the bed or to sit up slightly and watch for the appearance of a longitudinal midline bulge.

Prominent vasculature

If veins are seen coursing over the abdomen, note their exact location.

- Attempt to map the direction of blood flow within them:
  - Place 2 fingers at one end of the vein and apply occlusive pressure
  - Move 1 finger along the vein, emptying that section of blood in a ‘milking’ action.
  - Release the pressure from one finger and watch for flow of blood back into the vein.
  - Repeat, emptying blood in the other direction.
  - Due to the venous valves, you should be able to determine the direction of blood flow in that vein.
- Inferior flow of blood suggests superior vena cava obstruction.
- Superior flow of blood suggests inferior vena cava obstruction.
- Flow radiating out from the umbilicus (‘caput medusae’) indicates portal vein hypertension (porto-systemic shunting occurs through the umbilical veins which become engorged).

Obvious pulsations

Look across the abdomen for any pulsations. A pulsatile, expanding mass in the epigastrium may be an abdominal aortic aneurysm.

Peristaltic waves

Usually only seen in thin, fit, young individuals. A very obvious bowel peristalsis is seen as rippling movements beneath the skin and may indicate intestinal obstruction.

Striae

'Stretch marks' are pink or white streaky lines caused by changes in the tension of the abdominal wall. These may be normal in rapidly growing

pubescent teens. Also seen in obesity, pregnancy ('striae gravidarum'), ascites and following rapid weight loss or abdominal paracentesis.

Bear in mind that these will turn pink/purple in Cushing's Syndrome like other scars (see p.127).

Skin discolouration

There are 2 classical patterns of bruising/discolouration indicating the presence of retroperitoneal blood (seen especially in pancreatitis):

- *Cullen's sign:* discolouration at the umbilicus and surrounding skin
- *Grey-Turner's sign:* discolouration at the flanks

Stomas

Look for surgical stomas or fistulae, noting their exact location, nature of the stoma and appearance of the exposed mucosa (if any). Remember that a stoma may be from the large bowel, small bowel, or renal tract. Look also at the contents of the stoma bag noting any abnormalities such as diarrhoea, pus, mucus or blood.

- ***Colostomy:*** usually seen in the left iliac fossa and will be flush to the skin. (Bag may contain semi-solid to formed stool.)
- ***Ileostomy:*** usually in the right iliac fossa and formed as a 'spout' of bowel mucosa extending from the abdominal wall to prevent the luminal contents harming the abdominal wall. (Bag will contain semi-formed and liquid stool.)
- ***Urostomy:*** often formed as an ileal conduit with ureters connected to a portion of small bowel and then to the abdominal wall. Usually in the right iliac fossa. (Bag will contain urine.)
- ***Nephrostomy:*** drainage of urine from the kidney pelvis to the exterior. Usually a temporary measure following operative procedures to the renal tract or to decompress an obstructed system. Usually at the flank. (Bag will contain urine.)

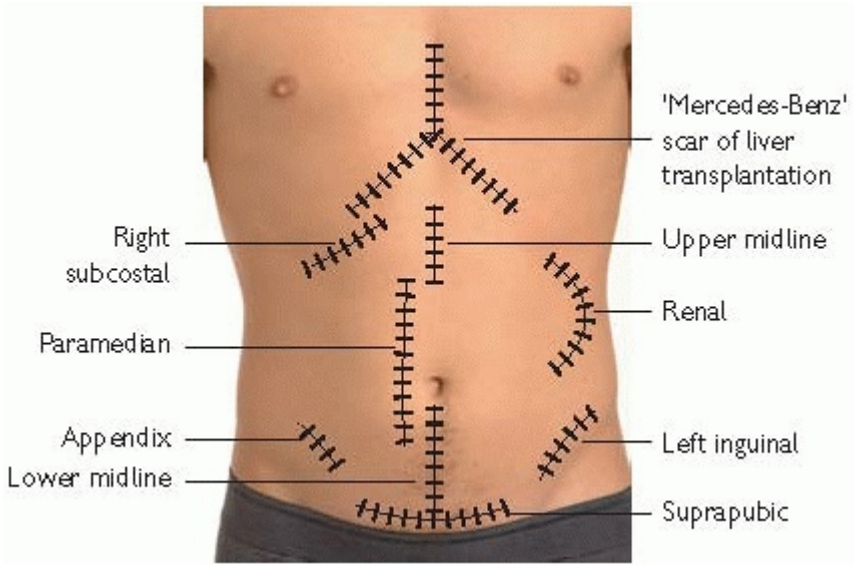

Fig. 9.6 Some common abdominal surgical scars.

Palpation

General approach

The patient should be positioned lying supine with the head supported by a single pillow and arms at their sides.

Squat by the side of the bed or couch so that the patient's abdomen is at your eye level.

Each of the 4 quadrants (see p.222) should be examined in turn with light, and then deep palpation before focusing on specific organs (p.252). The order they are examined in doesn't matter—find a routine that suits you. Ask the patient if there is any area of tenderness and remember to examine this part *last*.

Before you begin, ask the patient to let you know if you cause any discomfort. You should be able to examine the abdomen without looking at it closely. Instead, you should watch the patient's face for signs of pain.

Light palpation

For this, you use the finger-tips and palmar aspects of the fingers.

- Lay your right hand on the patient's abdomen and gently press in by flexing at the metacarpo-phalangeal joints.
- If there is pain on light palpation, attempt to determine whether the pain is worse when you press down or when you release the pressure (*'rebound tenderness'*).
- If the abdominal muscles seem tense, determine whether it is localized or generalized. Ensure the patient is relaxed-it may be helpful for the patient to bend their knees slightly, relaxing the abdominal muscles. An involuntary tension in the abdominal muscles—apparently protecting the underlying organs—is called *'guarding'*.

Deep palpation

Once all 4 quadrants are lightly palpated, re-examine using more pressure. This should enable you to feel for any masses or structural abnormalities.

- If a mass is felt, treat it as you would any other lump describing its exact location, size, shape, surface, consistency, mobility, movement with respiration, tenderness and whether or not it is pulsatile.
- It is often possible to detect the putty-like consistency of stool in the sigmoid colon. You should treat this as any other 'lump' to be sure of its nature.

Box 9.14 Signs of peritonitis

- Pain on light palpation.
- Rebound tenderness.
- Involountary guarding.
- Pain recurring with slight movement of the examining hand.
- Absent bowel sounds (p.262).

Palpating the abdominal organs

Liver

The normal liver extends from the 5th intercostal space on the right of the midline to the costal margin, hiding under the ribs so is often not normally palpable—don't worry if you can't feel one!

- Using the flat of the right hand, start palpation from the right iliac fossa.
- You should angle your hand such that the index finger is aligned with the costal margin (see Fig. 9.7).
- Exert gentle pressure and ask the patient to take a deep breath.
- With each inward breath, your fingers should drift slightly superiorly as the liver moves inferiorly with the diaphragm. Relax the

pressure on your hand slightly at the height of inspiration.

- If the liver is just above the position of your hand, the lateral surface of your index finger will strike the liver edge and glide over it with a palpable 'step'.
- If the liver is not felt, move your hand 1-2cm superiorly and feel again.
- Repeat the process, moving towards the ribs until the liver is felt.

If a liver edge is felt, you should note:

- How far below the costal margin it extends in finger-breadths or (preferably) centimetres and record the number carefully.
- The nature of the liver edge (is the surface smooth or irregular?).
- The presence of tenderness.
- Whether the liver is pulsatile.

Findings

- It is often possible to palpate the liver just below the costal margin at the height of inspiration in normal, healthy, thin people.
- An enlarged liver has many causes—see *OHCM6*, p.518.
- A normal liver may be palpable in patients with COPD or asthma in whom the chest is hyper-expanded or in patients with a subdiaphragmatic collection.
- The liver may also be palpable in the presence of 'Riedel's lobe'—a normal variant in which a projection of the liver arises from the inferior surface of the right lobe. More common in females. Commonly mistaken for a right kidney or enlarged gallbladder.

Gallbladder

Lies at the right costal margin at the tip of the 9th rib, at the lateral border of the rectus abdominis. Normally only palpable when enlarged due to biliary obstruction or acute cholecystitis.

- Felt as a bulbous, focal, rounded mass which moves with inspiration.
- Position the right hand perpendicular to the costal margin and palpate in a medial → lateral direction (see Fig. 9.8).

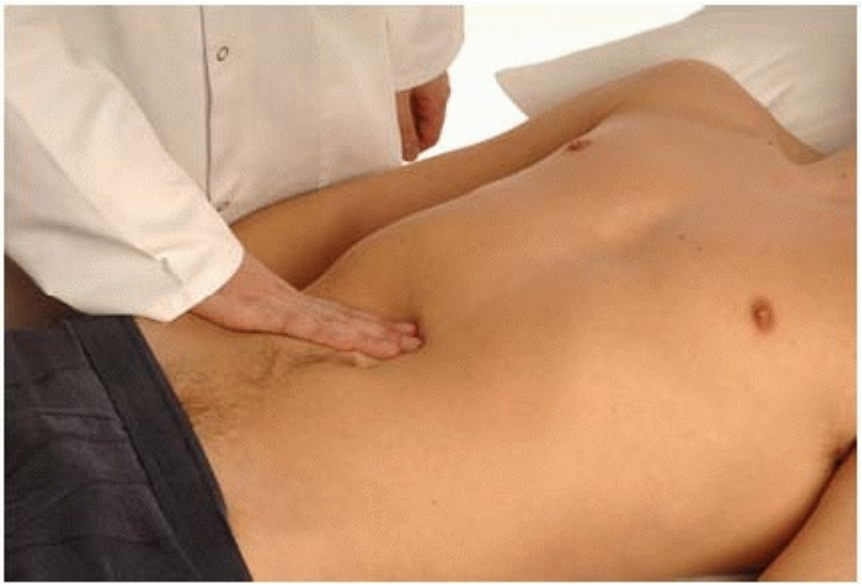

Fig. 9.7 Palpation of the liver—align the lateral surface of the index finger with the costal margin and palpate from the right iliac fossa to the ribs in a step-wise fashion.

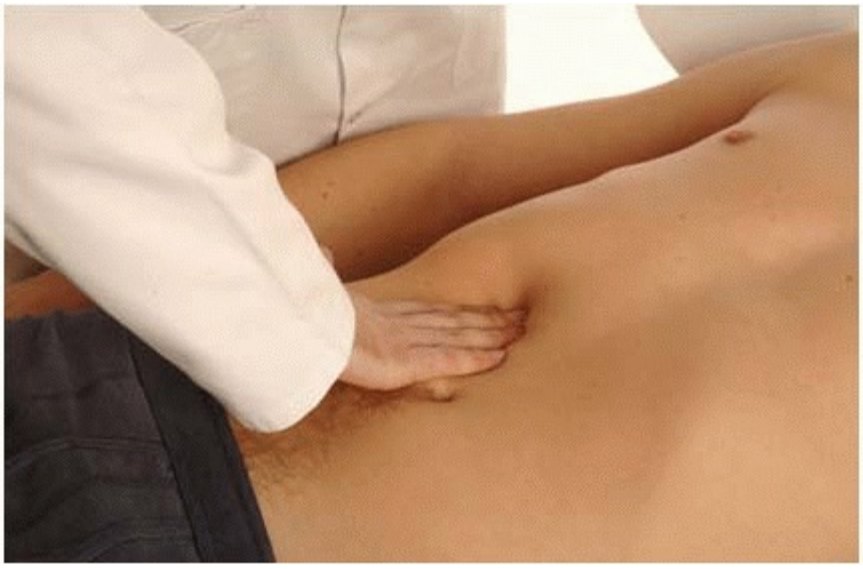

Fig. 9.8 Palpation of the gallbladder—the examining hand should be perpendicular to the costal margin at the tip of the 9th rib (where the lateral border of the rectus muscle meets the costal cartilages).

Box 9.15 Important gallbladder signs

Murphy's sign

A sign of cholecystitis—pain on palpation over the gallbladder during deep inspiration. Only positive if there is NO pain on the left at the same position.

Courvoisier's law

In the presence of jaundice, a palpable gallbladder is probably NOT caused by gallstones.

Spleen

The largest lymphatic organ which varies in size and shape between individuals—roughly the size of a clenched fist (12cm x 7cm).

Normally hidden beneath the left costal cartilages and impalpable.

Enlargement of the spleen occurs in a downward direction, extending into the left upper quadrant (and even the left lower quadrant) across towards the right iliac fossa.

- Palpated using a similar technique to that used to examine the liver (📖 p.252).
- Your left hand should be used to support the left of the ribcage posterolaterally. Your right hand should be aligned with the fingertips parallel to the left costal margin (see Fig. 9.9).
- Start palpation just below the umbilicus in the midline and work towards the left costal margin asking the patient to take a deep breath in and feeling for the movement of the spleen under your fingers—much like palpating the liver.
- The inferior edge of the spleen may have a palpable 'notch' centrally which will help you differentiate it from any other abdominal mass.
- If a spleen is felt, measure the distance to the costal border in finger-breadths or (preferably) centimetres.

▸ An impalpable spleen may sometimes become palpable by repositioning the patient. Ask them to roll onto their right hand side and repeat the examination as above.

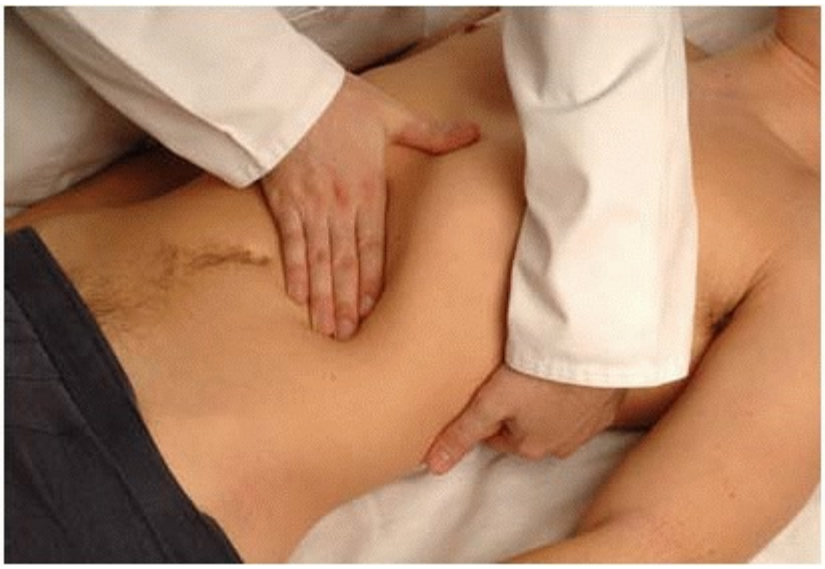

Fig. 9.9 Palpation of the spleen—align the fingertips of your right hand with the left costal border and start palpating just below the umbilicus working towards the left upper quadrant.

Box 9.18 Some causes of…

Hepatomegaly

- Alcohol.
- Right heart failure.
- Neoplasia (primary cancer, metastases, myeloproliferative disorders, leukaemia, lymphoma).
- Chronic liver disease (NB cirrhosis causes a small, shrunken liver).
- Infections (acute viral hepatitis, brucellosis, tuberculosis).
- Amyloidosis.
- Haemochromatosis.
- Biliary obstruction.

Splenomegaly

- *Massive (>8cm):* malaria, Kala-azar, Gaucher's disease.
- *Moderate (4-8cm):* portal hypertension secondary to cirrhosis, lymphoproliferative disorders and many others.
- *Mild:* lymphoproliferative disorders, portal hypertension secondary to cirrhosis, infectious hepatitis, glandular fever, subacute endocarditis, sarcoidosis, rheumatoid arthritis, connective tissue diseases, haematological disorders (idiopathic thrombocytopaenia, hereditary spherocytosis, polycythaemia rubra vera).

Hepatosplenomegaly

Myeloproliferative disorders, lymphoproliferative disorders, chronic liver disease with portal hypertension, infection (acute viral hepatitis, brucellosis, Weil's disease, toxoplasmosis, CMV), lupus, amyloidosis, sarcoidosis, thyrotoxicosis, acromegaly, pernicious anaemia, sickle-cell anaemia.

Kidneys

The kidneys are retroperitoneal, lying on the posterior abdominal wall either side of the vertebral column between T12 and L3 vertebrae. They move slightly inferiorly with inspiration. The right kidney lies a little lower than the left (displaced by the liver).

Palpation is ‘bimanual’ (both hands). You may be able to feel the lower pole of the right kidney in normal, thin people.

- Place your left hand behind the patient at the right loin.
- Place your right hand below the right costal margin at the lateral border of the rectus abdominis.
- Keeping the fingers of your right hand together, flex them at the metacarpo-phalangeal joints pushing deep into the abdomen.
- Ask the patient to take a deep breath—you may be able to feel the rounded lower pole of the kidney between your hands, slipping away when the patient exhales.
- This technique of using one hand to move the kidney toward the other is called ‘renal ballottement’.

- Repeat the procedure for the left kidney-leaning over and placing your left hand behind the patient's left loin.

Table 9.1 Differentiating an enlarged spleen and an enlarged left kidney

Enlarged spleen	Enlarged kidney
Impossible to feel above	Can feel above the organ
Has a central ‘notch’ on the leading edge	No notch—but you may feel the central hilar notch medially
Moves early on inspiration	Moves late on inspiration
Moves inferio-medially on inspiration	Moves inferiorly on inspiration
Not ballottable	Ballottable
Dullness to percussion	Resonant percussion note due to overlying bowel gas
May enlarge toward the umbilicus	Enlarges inferiorly lateral to the midline

Findings

- *Unilateral palpable kidney:* hydronephrosis, polycystic kidney disease, renal cell carcinoma, acute renal vein thrombosis, renal abscess, acute pyelonephritis.
- *Bilateral palpable kidneys:* bilateral hydronephrosis, bilateral renal cell carcinoma, polycystic kidney disease, nephrotic syndrome, amyloidosis, lymphoma, acromegaly.

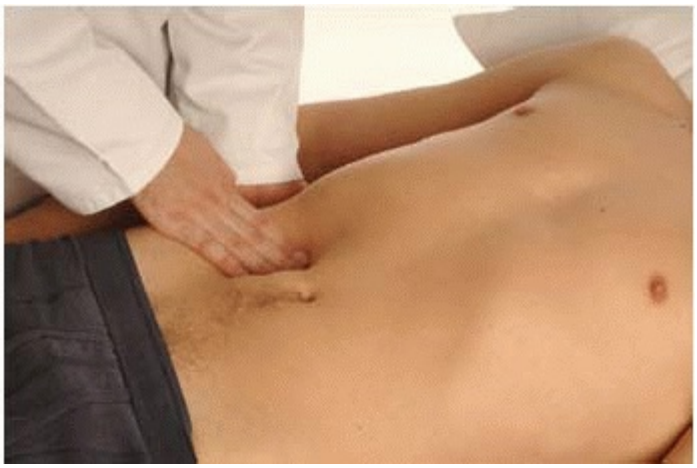

Fig. 9.10 Palpation of the right kidney.

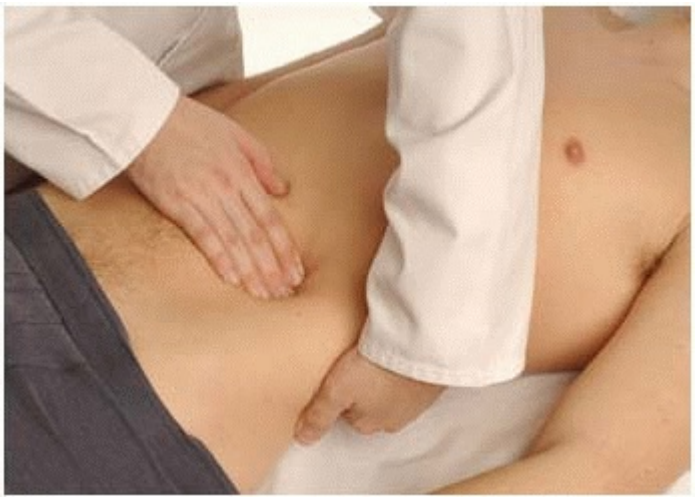

Fig. 9.11 Palpation of the left kidney.

Bladder

The urinary bladder is pyramid-shaped and lies within the pelvic cavity. It is not palpable when empty.

As it fills, it expands superiorly and may even reach as high as the umbilicus or just beyond if very full.

It may be difficult to differentiate it from an enlarged uterus or ovarian cyst. The full bladder will be:

- A palpable, rounded mass arising from behind the pubic symphysis.
- Dull to percussion.
- You will be unable to feel *below* it.
- Pressure on the full bladder will make the patient feel the need to urinate.

Aorta

The abdominal aorta may be palpated in the midline above the umbilicus, felt as a longitudinal pulsatile mass. It is particularly palpable in thin people. If felt:

- Position the fingers of each hand either side of the outermost palpable margins.
- Measure the distance between your fingers. Normal diameter ≈2-3cm.

- Decide whether the mass you feel is pulsatile/expansile in itself (in which case your fingers will move outwards) or whether the pulsation is transmitted through other tissue (in which case your fingers will move upwards). See Fig. 9.12.

Inguinal lymph nodes

The inguinal chain of lymph nodes lies along the inguinal ligament between the pubic tubercle and the anterior superior iliac spine and should not be missed.

- Feel along this line for any lumps treating each as you would any other (📖 p.98).

Small, firm mobile lymph nodes are common in healthy people and are often the result of minor sepsis or abrasions of the lower limbs.

▸ By this stage of the examination, you should have examined the nodes in the axillae, neck, supraclavicular areas, and the inguinal regions.

The hernial orifices

Described on 📖 p.266.

The external genitalia

No thorough abdominal examination is complete without examining the genitalia—although in clinical practice many leave this out, considering it inappropriate if you are not suspicious of any genito-urinary pathology.

See 📖 Chapter 12.

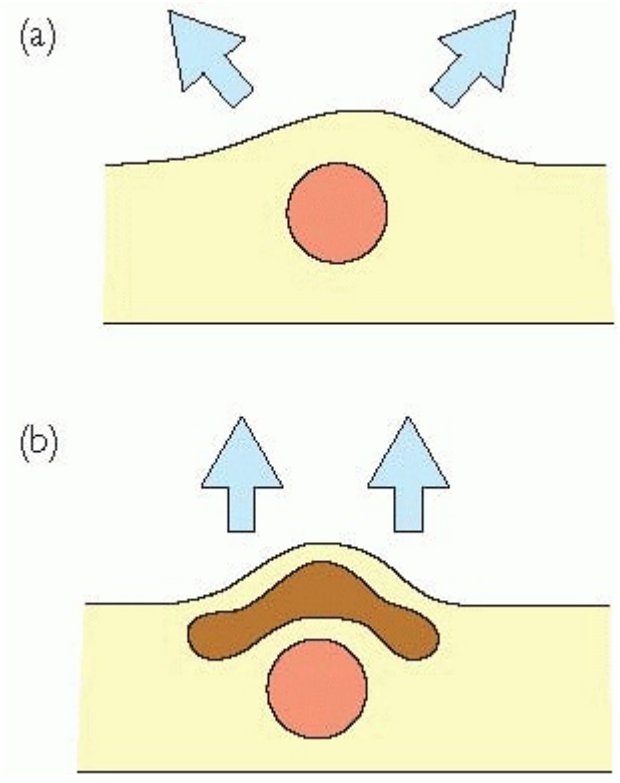

Fig. 9.12 Palpating a pulsatile mass. If the mass itself is expansile (a), yourfingers will move outwards. If the pulsatility is being transmitted through overlying tissues (b), your fingers will move upwards.

Percussion

In the examination of the abdomen, percussion is useful for…

- Determining the size and nature of enlarged organs or masses.

- Detecting shifting dullness (below).
- Eliciting rebound tenderness (📖 p.250).

Organs or masses will appear as dullness whereas a bowel full of gas will seem abnormally resonant. Good technique comes with experience. Practice percussing out your own liver. Percussion technique is described on 📖 p.214.

Examining for ascites

If fluid is present in the peritoneal cavity (ascites), gravity will cause it to collect in the flanks when the patient is lying flat—this will give dullness to percussion laterally with central resonance as the bowel floats.

Ascites will give a distended abdomen, often with an everted umbilicus. If you suspect the presence of ascites:

- Percuss centrally → laterally with the fingers spread and positioned longitudinally (see Fig. 9.13).
- Listen (and feel) for a definite change to a dull note.

There are then 2 specific tests to perform…

*Shifting dullness**

- Percuss centrally → laterally until dullness is detected. This marks the air-fluid level in the abdomen.
- Keep your finger pressed there as you…
- Ask the patient to roll onto the opposite side (i.e. if dullness is detected on the right, roll the patient to their left-hand side).
- Ask the patient to hold the new position for ~half a minute.
- Repeat percussion moving laterally to central over your mark.
- If the dullness truly was an air-fluid level, the fluid will now be moved by gravity away from the marked spot and the previously dull area will be resonant.

Fluid thrill

In this test, you are attempting to detect a wave transmitted across the peritoneal fluid. This is only really possible with massive ascites.

You need an assistant for this test (you can ask the patient to help).

- Ask your assistant to place the ulnar edge of one of their hands in the midline of the abdomen (see Fig. 9.13).
- Place your left hand on one side of the abdomen, about level with the midclavicular line.
- With your right hand, flick the opposite side of the patient's abdomen.
- If a ‘fluid thrill’ can be detected, you will feel the ripple from the flick transmitted as a tap to your left hand.

The assistant's hand is important-it prevents transmission of the impulse across the surface of the abdominal wall.)

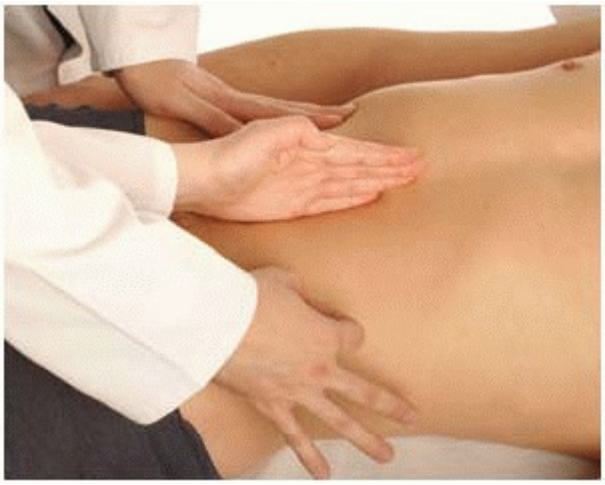

Fig. 9.13 Testing for a fluid thrill. Ask an assistant to place their hand centrally on the abdomen—this prevents transmission of the impulse through the abdominal wall.

Liver

Percuss to map the upper and lower borders of the liver-note the length, in centimetres, at the midclavicular line.

Spleen

Percussion from the left costal margin towards the midaxillary line and the lower left ribs may reveal dullness suggestive of splenic enlargement that could not normally be palpated.

Kidneys

Useful in differentiating an enlarged kidney from an enlarged spleen or liver. The kidneys lie deep in the abdomen and are surrounded by perinephric fat which makes them resonant to percussion. Splenomegaly or hepatomegaly will appear dull.

Bladder

Dullness to percussion in the suprapubic region may be helpful in determining whether an ill-defined mass is an enlarged bladder (dull) or distended bowel (resonant).

Auscultation

An important part of the abdominal examination which is easily missed.

Bowel sounds

These are low-pitched gurgling sounds produced by normal gut peristalsis. They are intermittent but will vary in timing depending on when the last meal was eaten. Practise listening to as many abdomens as possible to understand the normal range of sounds.

Listen with the diaphragm of the stethoscope just below the umbilicus.

- ***Normal:*** low-pitched gurgling, intermittent.
- ***High-pitched:*** often called 'tinkling'. These sounds are suggestive of partial or total bowel obstruction.
- ***Borborygmus:*** this is a loud low-pitched gurgling that can even be heard without a stethoscope. (The sounds are called 'borborygmi'.) Typical of diarrhoeal states or abnormal peristalsis.
- ***Absent sounds:*** if no sounds are heard for 2 minutes, there may be a complete lack of peristalsis—i.e. a paralytic ileus or peritonitis.

Bruits

These are sounds produced by the turbulent flow of blood through a vessel—similar in sound to heart murmurs. Listen with diaphragm of the stethoscope.

Bruits may occur in normal adults but raise the suspicion of pathological stenosis (narrowing) when heard throughout both systole and diastole. There are several areas you should listen at on the abdomen…

- Just above the umbilicus over the aorta (abdominal aortic aneurysm).
- Either side of the midline just above the umbilicus (renal artery stenosis).
- At the epigastrium (mesenteric stenosis).
- Over the liver (AV malformations, acute alcoholic hepatitis, hepatocellular carcinoma).

Friction rubs

These are creaking sounds like that of a pleural rub (📖 p.217) heard when inflamed peritoneal surfaces move against each other with respiration.

Listen over the liver and the spleen in the right and left upper quadrants respectively.

Causes include hepatocellular carcinoma, liver abscesses, recent percutaneous liver biopsy, liver or splenic infarction and STD-associated perihepatitis (Fitz-Hugh-Curtis Syndrome).

Venous hums

Rarely, it is possible to hear the hum of venous blood flow in the upper abdomen over a caput medusa (📖 p.248) 2° to porto-systemic shunting of blood.

'Per rectum' examination

This is an important part of the examination and should not be avoided simply because it is considered unpleasant. It is particularly important in patients with symptoms of PR bleeding, tenesmus, change in bowel habit and pruritus ani.

▸ Remember: 'If you don't put your finger in it, you may put your foot in it!'

Before you begin

Explain to the patient what is involved and obtain verbal consent. Choose your words carefully, adjusting your wording to suit the patient! Favourite phrases include 'tail-end', 'back-passage', and 'bottom'. Say that you need to examine their back passage 'with a finger'. Warn that it 'probably won't hurt' but may feel 'cold' and 'a little unusual'.

You should ask for another member of staff to chaperone-guarding yourself against future claims of inappropriate treatment and reassuring the patient.*

As you proceed, explain each stage to the patient.

* This is still controversial in the UK at the time of writing. Official advice is that *all* doctors should have a chaperone when performing an intimate examination. In practice, male doctors performing an examination on a female always have a chaperone present whilst the need for a chaperone in other situations is judged individually at the time.

Equipment

- Chaperone
- Non-sterile gloves
- Tissues
- Lubricating jelly (e.g. Aquagel®)

Technique

- With informed verbal consent obtained, ensure adequate privacy.
- Uncover the patient from waist to knees.
- Ask the patient to lie in the left lateral position with their legs bent such that their knees are drawn up to their chest and their buttocks facing towards you—preferably projecting slightly over the edge of the bed/couch.
- Ensure that there is a good light source—preferably a mobile lamp.
- Put on a pair of gloves.
- Separate the buttocks carefully by lifting the right buttock with your left hand.
- Inspect the perianal area and anus
    - Look for rashes, excoriations, skin tags, anal warts, fistulous openings, fissures, external haemorrhoids, abscesses, faecal soiling, blood, and mucus.
- Ask the patient to strain or 'bear down' and watch for the projection of pink mucusa of a rectal prolapse.
- Lubricate the tip of your right index finger with the jelly.
- Begin by placing the pulp of your right index finger against the anus in the midline and press in firmly but slowly.
    - Most anal sphincters will reflexly tighten when touched but will quickly relax with continued pressure.
- When the sphincter relaxes, gently advance the finger into the anal canal.
- Assess anal sphincter tone by asking the patient to clench your finger.

- Rotate the finger backwards and forwards covering the full 360°, feeling for any thickening or irregularities.

- Push the finger further—up to the hilt if possible—to the rectum.
- Examine all 360° by moving the finger in sweeping motions. Note:
  - The presence of thickening or irregularities of the rectal wall.
  - The presence of palpable faeces—and its consistency.
  - Any points of tenderness.
- Next, in the male, identify the prostate gland which can be felt through the anterior rectal wall.
  - The normal prostate is smooth-surfaced, firm with a slightly rubbery texture measuring 2-3cm diameter. It has 2 lobes with a palpable central sulcus.
- Gently withdraw your finger and inspect the glove for faeces, blood, or mucus and note the colour of the stool, if present.
- Tell the patient that the examination is over and wipe any faeces or jelly from the natal cleft with the tissues. Some patients may prefer to do this themselves.
- Thank the patient and ask them to redress. You may need to help.

Findings

If any mass or abnormality is identified on the exterior or interior of the areas examined, its exact location should be noted. It is conventional to record as the position on a clock face with 12 o'clock indicating the anterior side of the rectum at the perineum. Other features of the mass should be recorded as described on 📖 p.98.

- ***Benign prostatic hyperplasia:*** the prostate is enlarged but the central sulcus is preserved, often exaggerated.
- ***Prostate cancer:*** the gland loses its rubbery consistency and may become hard. The lateral lobes may be irregular and nodular. There is often distortion or loss of the central sulcus. If the tumour is large and has spread locally, there may be thickening of the rectal mucosa either side of the gland creating 'winging' of the prostate.
- ***Prostatitis:*** the gland will be enlarged, boggy, and very tender.

▸ *Hints*

- If the patient experiences severe pain, with gentle pressure on the anal opening, consider… anal fissure, ischiorectal abscess, anal ulcer, thrombosed haemorrhoid, or prostatitis.
- In this situation, you may have to apply local anaesthetic gel to the anal margin before proceeding. If in doubt, ask a senior.

The hernial orifices

A hernia is an abnormal protrusion of a structure, organ or part of an organ out of the cavity in which it belongs. A hernia can usually be 'reduced' i.e. its contents returned to the original cavity either spontaneously or by manipulation.

Abdominal hernias are usually caused by portions of bowel protruding through weakened areas of the abdominal wall. In the abdomen, hernias usually occur at natural openings of the abdominal wall (e.g. inguinal canals, femoral canals, umbilicus, oesophageal hiatus) or acquired weak spots such as surgical scars.

Most abdominal hernias have an expansile cough impulse-asking the patient to cough will ↑ the intra-abdominal pressure causing a visible or palpable impulse.

Strangulation: hernias that cannot be reduced (irreducible) may become fixed and swollen as their blood supply is occluded causing ischaemia and necrosis of the herniated organ. The hernias are painfully swollen with overlying erythema and may cause disruption of normal gut function (e.g. intestinal obstruction).

An approach to hernias

- Determine the characteristics as you would any lump (📖 p.98) including position, temperature, tenderness, shape, size, tension, and composition.
- Make note of the characteristics of the overlying skin.
- Palpate the hernia and feel for a cough impulse.
- Attempt reduction of the hernia.
- Percuss and auscultate the hernia (listening for bowel sounds or bruits).

- Always remember to examine the same site on the opposite side.

Inguinal hernias

Anatomy

The inguinal canal extends from the pubic tubercle to the anterior superior iliac spine. In the male, it carries the spermatic cord (vas deferens, blood vessels and nerves). In the female, it is much smaller and carries the round ligament of the uterus.

After testicular descent, the canal closes but the site is weakened.

The internal ring is an opening in the transversalis fascia lying at the midinguinal point, halfway between the anterior superior iliac spine and the pubic symphysis (about 1.5cm above the femoral pulse).

The external ring is an opening of the external oblique aponeurosis and is immediately above and medial to the pubic tubercle (see Fig. 9.14).

- *Direct inguinal hernia:* this is herniation at the site of the external ring.
- *Indirect inguinal hernia*: this is the most common site (85% of all hernias). Herniation is through the internal ring with bowel or omentum travelling down the inguinal canal and may protrude through the external ring into the scrotum. More likely to strangulate than direct inguinal hernias.

Examination

- The patient should be examined standing-up and undressed from the waist down (some hernias may spontaneously reduce when supine).
- Palpate especially for tenderness and consistency of the lump.
  - Herniated omentum will appear rubbery, non-fluctuant, and dull to percussion.
  - Herniated gut will be fluctuant, resonant. You may be able to hear bowel sounds within the hernia.
- With 2 fingers on the mass, ask the patient to cough and feel for an expansile cough impulse.
- Attempt to reduce the hernia by massaging it back towards it suspected site of origin.
  - For indirect hernias, you should use the flat of your hand, directing the hernia form below and guide it through the external ring, up the inguinal canal laterally towards the internal ring.
- Once reduced, the hernia should not reappear until you release the pressure.
- With the hernia reduced, try pressing over the site of the internal ring and asking the patient to cough. An indirect hernia will remain reduced whereas a direct hernia will protrude once more.

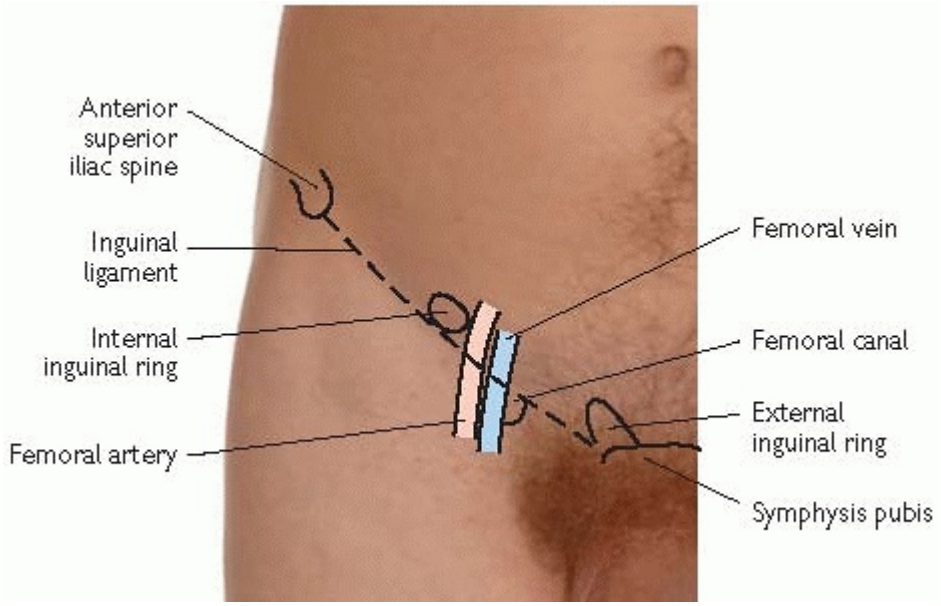

Fig. 9.14 Sites of the internal and external inguinal rings.

Table 9.2 Differentiation of inguinal hernias

Indirect inguinal hernia	Direct inguinal hernia
Can descend into the scrotum	Very rarely descends to the scrotum
Reduces upwards, laterally, backwards	Reduces upwards and backwards
Remains reduced with pressure at the internal ring	Not controlled by pressure over the internal ring
The causative defect is not palpable	Defect in the abdominal wall is palpable
Reappears at the internal ring and flows medially	Reappears in the same position as before reduction

Femoral hernias

Anatomy

The femoral canal is the small component of the femoral sheath medial to the femoral vessels and contains loose connective tissue, lymphatic vessels and lymph nodes. It is bordered anteriorly by the inguinal ligament, the pectineal ligament posteriorly, the femoral vein laterally, and the lacunar ligament medially.

Femoral hernias are protrusions of bowel or omentum through this space. They are more common in middle-aged and elderly women and can easily strangulate due to the small, rigid opening they pass through.

Examination

- Examine with the patient standing up and undressed from the waist down.
- Examine as you would any other hernia and attempt reduction.
- If present, a femoral hernia will appear as a lump just lateral and inferior to the pubic tubercle, about 2cm medial to the femoral pulse.

Box 9.19 Differential diagnosis of a femoral hernia

- Inguinal hernia.

- Very large lymph node.
- Ectopic testicle.
- Psoas bursa or abscess.
- Lipoma.

Other abdominal wall hernias

- ***Umbilical/paraumbilical:*** herniation through a defect near the umbilicus (considered congenital if identified in children).
- ***Epigastric:*** herniation through the linea alba above the umbilicus.
- ***Spigalean:*** herniation through the linea semilunaris (lateral to the rectus sheath), usually below and lateral to the umbilicus. Rare.
- ***Obturator:*** herniation through the obturator canal, associated with increasing age and multiparity.
- ***Perineal:*** herniation through the pelvic floor diaphragm. Rare.
- ***Incisional:*** Herniation through the site of previous surgery. The bulge is usually seen underlying a surface surgical scar. Increasing incidence with advanced age but can be caused by wound infection and associated fasciitis or muscle necrosis.

Important presenting patterns

Chronic liver disease

Any of the following features may be seen. With severe disease and 'decompensation', more will become apparent…

- Jaundice.
- Palmar erythema.
- Leuconychia.
- Clubbing.
- Spider naevi.
- Telangiectasia.
- Hepatomegaly.
- Ascites.
- Purpura.
- Easy bruising.
- Epistaxis.
- Menorrhagia.
- Loss of libido.
- Hair loss.
- Bilateral parotid swelling.
- Encephalopathy.
- Variceal bleeding-manifesting as haematemesis and/or melaena.

♂	♀
• Gynaecomastia.	• Breast atrophy.

- Testicular atrophy.
- Irregular menses.
- Impotence.
- Amenorrhoea.

Portal hypertension

Raised pressure in the hepatic portal vein often secondary to liver disease or non-cirrhotic causes such as portal vein thrombosis. Causes porto-systemic shunting and oesophageal varices. Signs:

- Fetor hepaticus
- Splenomegaly
- Risk of gastrointestinal blood loss from varices (anaemia, haematemesis, melaena)
- Ascites
- Caput medusae.

Alcoholic liver disease

May cause all the features of chronic liver disease as described above. In addition, alcohol dependency or addiction is associated with:

- Tolerance.
- Withdrawal symptoms.
- Alcohol taken in larger amounts and for longer than intended.
- Persistent desire to ‘cut down’.
- Excessive time spent in activities related to alcohol intake.
- Abandoning social, occupational or recreational activities.
- Continued use despite an awareness of the adverse physiological and psychological effects of continued use.

Fatty liver

‘Hepatic steatosis’ and has many other causes including drugs, pregnancy and diabetes mellitus.

Deposition of fat as a result of preferential alcohol oxidation. Reversible with abstinence but may proceed to cirrhosis with continued use. No specific clinical features.

Alcoholic hepatitis

Hepatocellular inflammation with lymphocyte infiltration, steatosis, cholestasis, fibrosis and necrosis. Clinical features include:

- Fever.
- Jaundice.
- Tender hepatomegaly.
- May hear a bruit over the liver.

Cirrhosis

Severe hepatic fibrosis with ‘micronodules’. Loss of hepatocytes, impaired synthetic function and portal hypertension. Other causes of cirrhosis include chronic viral hepatitis (B or C), sclerosing cholangitis, Wilson's disease, haemachromotosis, α_1-antitrypsin deficiency, primary biliary cirrhosis, Budd-chiari syndrome and several drugs (e.g. amiodarone, methyldopa and methotrexate). Clinical features can be any of those listed under ‘chronic liver disease’ above.

Extra-hepatic manifestations of alcoholic liver disease/alcoholism

- Obesity or malnutrition.
- Diarrhoea.
- Gastric erosions.
- Peptic ulcer disease.
- Pancreatitis.
- Varices.
- Ascites.
- Splenomegaly.
- Hypertension.
- Loss of 2° sexual characteristics.
- Osteomalacia.
- Osteoporosis.
- Falls.
- Seizures.
- Cognitive impairment (p.502).
- Metabolic encephalopathy.
- Peripheral neuropathy.
- Ataxic gait (p.350).
- Wernicke's encephalopathy.
- Korsakoff's syndrome.
- Cardiomyopathy.
- Arrythmias (esp. atrial fibrillation).

Hepatic encephalopathy

Shunting of blood away from the portal circulation, seen in chronic liver disease, allows potentially neurotoxic substances absorbed in the gut to bypass the liver where they would normally be removed. See *OHCM6*, p.230 for management.

Hepatic encephalopathy is graded as follows …

Grade 0	Normal mental state
Grade I	Altered mood or behaviour (↓ attention span, difficulty with numbers and lack or awareness)
Grade II	↑ drowsiness, slurred speech, mild/mod confusion
Grade III	Stupor but responsive to stimuli, significant confusion, restlessness
Grade IV	Coma

Malabsorption

Numerous disorders can cause malabsorption states. They can be grouped as pancreatic insufficiency, bile salt malabsorption, small bowel mucosa defects (coeliac disease, tropical sprue, giardiasis, disaccharidase deficiency, Whipple's disease, short bowel syndrome), bacterial overgrowth, and specific delivery defects.

General symptoms and signs of malabsorption include:

- Muscle wasting.
- Weight loss.
- Pallor.
- Diarrhoea (watery).
- Steatorrhoea: pale, fatty stools; offensive smelling and difficult to flush.
- Glossitis.
- Angular stomatitis (vitamin B_2, B_{12} and folic acid deficiencies).
- Intra-oral purpura and easy bruising (vitamin K deficiency).
- Follicular keratitis: hyperkeratotic white patches (vitamin A deficiency).

Acute pancreatitis (see also OCHM6, p.478)

Symptoms

- Pain—central abdominal or epigastric, radiating through to the back. Sometimes relieved slightly by sitting forwards.
- Vomiting.

Signs

- Tachycardia.
- Fever.
- Jaundice (rarely).
- Peritonitis (bowel ileus, very tender abdomen, guarding).
- Retroperitoneal bleed: Cullen's or Grey-Turner's signs (p.249).

Chronic pancreatitis

In developed countries, the commonest cause is chronic heavy alcohol intake. See *OHCM6*, p.252 for more information. A small group of patients can inherit chronic pancreatitis through an autosomal dominant gene with incomplete penetrance.

Clinical features are usually due to pancreatic enzyme deficiencies and malabsorption and chronic pain. There may be acute exacerbations, presenting as acute pancreatitis. Loss of pancreatic endocrine function may cause diabetes.

Cholangitis

Biliary sepsis. Suggested by ‘Charcot's triad’:

- Right upper quadrant pain.
- Fever.
- Jaundice.

You may also be able to elicit Murphy's sign (p.253).

Coeliac disease

A common cause of malabsorption. Affects 1 in 2000 in the UK (1 in 300 in Ireland). T-cell mediated autoimmune disease of the small bowel mucosa characterized by villous atrophy and ↑ intra-epithelial lymphocytosis in response to ingestion of gluten. For treatment and prognosis, see *OHCM6*, p.252.

Gluten is a high-molecular weight compound containing gliadins and peptides. Found in a huge number of founds containing wheat, barley

and rye. Controversy exists over eating oats.

Clinical features:

Symptoms

- Tiredness.
- Malaise.
- Diarrhoea or steatorrhoea.
- Abdominal discomfort and bloating.
- Weight loss.
- Anxiety.
- Depression.
- Peripheral paraesthesia.

Signs

- Muscle wasting.
- Mouth ulceration.
- Angular stomatitis.
- Ankle oedema (low serum albumin).
- Polyneuropathy.
- Muscle weakness.
- Tetany.

Associated with

Autoimmune thyroid disorders, chronic liver disease, fibrosing alveolitis, ulcerative colitis, insulin-dependent diabetes mellitus.

Possible complications to be aware of

- Small bowel lymphoma (rare).
- Small bowel adenocarcinoma (rarer).
- Ulcerative jejunitis.
- Splenic atrophy.
- Anaemia.
- Osteomalacia.
- Osteoporosis.
- Secondary lactose intolerance.

Inflammatory bowel disease: ulcerative colitis (UC)

A chronic relapsing disease of unknown aetiology involving superficial inflammation of the colonic mucosa, starting from the rectum and working proximally without any breaks. The terminal ileum may be affected by 'backwash ileitis'. See also *OHCM6*, p.244. Periods of remission may give no symptoms at all.

Symptoms

- Diarrhoea (often with blood or mucus).
- Weight loss.

- Fever.
- Abdominal pain.
- Procitis may cause rectal bleeding, mucus, tenesmus, and constipation.

Complications to be aware of:

- Toxic megacolon.
- Iron deficiency anaemia.
- ↑ risk of colorectal carcinoma.
- Fistula-formation (rare).

Inflammatory bowel disease: Crohn's disease

Like ulcerative colitis (above), this is a chronic inflammatory disease of the gastrointestinal tract but differs from UC in that lesions occur anywhere from mouth to anus but especially at the terminal ileum and ano-rectum. Pathology involves deep ulceration, 'cobblestoning' of the mucosa, fissuring and abscess formation with 'skip lesions' and non-caseating granulomas. See also 📖 *OHCM6*, p.246.

Symptoms

If disease is limited to the colon, symptoms may be identical to UC.

- Loose stools or diarrhoea (usually not bloody).
- Anorexia.
- Malaise.
- Weight loss.
- Abdominal pain (insidious, often in the right lower quadrant).
- Perianal pain.
- Joint pains.

Note on examination… (these can occur in UC also)

- Aphthous mouth ulcers.
- Uveitis.
- Anaemia.
- Arthropathy.

Active Crohn's disease

- Colicky pain often in the right iliac fossa.
- May have diarrhoea with blood and mucus.
- Weight loss.
- Borborygmus (📖 p.262).
- May be a palpable inflammatory mass in the right iliac fossa.
- Abdominal distension.
- ± Bowel obstruction.

Active Crohn's colitis

- Similar presentation to ulcerative colitis.
- Perianal disease more likely to produce fissuring and fistula formation.

Complications to be aware of

- Fistula formation (from the bowel to any other abdominal organ or the exterior).
- Small ↑ risk of colorectal carcinoma (especially in long-standing disease limited to the colon).
- Vitamin B_{12} deficiency.
- Iron deficiency.
- Abscess formation.
- Stricture formation.
- Systemic infection.

Extra-intestinal features of inflammatory bowel disease

- Sero-negative arthropathy of large or small joints (peripheral, non-deforming, particularly at the knees, ankles, and wrists).
- Sacroiliitis.
- Anterior uvetitis.
- Erythema nodosum.
- Pyoderma gangrenosum.
- Ureteric calculi.
- Gallstones.
- Sclerosing cholangitis.
- Cholangiocarcinoma.
- Nutritional deficiencies (Osteoporosis? Osteomalacia?)
- Bile salt malabsorption
- Osteoporosis secondary to long-term steroid use or malabsorption.
- Systemic amyloidosis.

Irritable bowel syndrome-Rome II diagnostic criteria

At least 12 weeks, which need not be consecutive, in the preceding 12 months of abdominal discomfort or pain that has 2 out of 3 features:

- Relief with defaecation.
- Onset associated with a change in frequency of stool.
- Onset associated with a change in form of stool.

Other symptoms which support the diagnosis of IBS:

- Abnormal stool frequency (>3/day or <3/week).
- Abnormal stool form (lumpy/hard, loose/watery).
- Abnormal stool passage (straining, urgency, feeling of incomplete evacuation).
- Passage of mucus.
- Bloating or feeling of abdominal distension.

The elderly patient

Gastrointestinal disease presents as a huge spectrum in elders, encompassing nutrition, oral care, and continence in addition to the range of presentations described in this chapter. Whilst many older people suffer gastrointestinal symptoms, often due to underlying illnesses or the effect of medication, they may be embarrassed about discussing them. Thoughtful and holistic assessment is paramount, and simple interventions can pay dividends.

History

- ***Oral care:*** is often overlooked, but a key part of any assessment. Dentures may be ill fitting or lost, and dietary intake can suffer as a consequence, and hospital inpatients are particularly prone to losing their dentures.
- ***Clarify symptoms and diagnoses:*** Does the patient really have an irritable bowel? (See below.) Many patients may describe themselves as having such diagnoses, but take the time to clarify what this means. Recent changes of bowel habit, even in later life must always be viewed with a degree of alarm and causes considered.
- ***Constipation:*** can often lead to serious decline in patients with constipation. This is often easily remediable.
- ***Weight and nutrition:*** ask yourself why has the patient lost weight. The range of diagnoses is broad, but contemplate mood, dietary habits, and functional abilities in your assessments-it may be a matter of dislike of delivered frozen meals!
- ***Drug history:*** always consider the side effects of medication-analgesics and constipation, recent antibiotics, and diarrhoea. Ask about over-the-counter drugs including NSAIDs (topical drugs too!) and aperients.
- ***Continence:*** another key part of the assessment; try to discuss sensitively and determine if there factors additional to any GI disturbance, including mobility, cognition and visual problems. This dovetails with the ever important functional history.

Examination

- ***General:*** look out for signs of weight loss—wasting, poorly fitting clothes etc. For inpatients, a completed weight chart and careful consideration may alleviate some of the problems of poor nutrition and acute illness.
- ***Look in the mouth:*** as a range of diagnoses is often apparent. Denture care should be assessed (poor cleaning associated with recurrent stomatitis), and other problems such as oral candida are obvious.
- ***Observe:*** for other signs of systemic disease that might point to the cause of the gastrointestinal symptoms (e.g. multiple telangiectasia, valvular heart disease in GI bleeding).
- ***Examine:*** thoroughly for lymphadenopathy. Remember to examine hernial orifices—the cause of abdominal pain may be instantly obvious—and correctable.
- ***Rectal examination:*** vital-changes in bowel habit, continence, iron deficiency anaemia, bladder symptomatology all indicate this.

Diagnoses not to be missed

- ***Functional bowel disorders:*** tend to be less common in older people, so always consider underlying organic problems. Endoscopic examinations are often well tolerated and have a good diagnostic yield.
- ***Biliary sepsis:*** is the 3rd most common source of infection in older people (after chest and urine sepsis), and may lack many of the salient presenting features described previously in this chapter. Be alert to this possibility when considering differential diagnoses and choosing antibiotics.

We thank Dr Richard Fuller for providing this page.

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Chapter 10

The Nervous System

Presenting symptoms in neurology

The history is key is many neurological cases. If the patient cannot give a complete story (e.g. when describing a loss of consciousness or seizure), collateral histories should be gained from any witnesses to the event(s)—relatives, friends, the GP, or even passers-by.

An approach to neurological symptoms

Symptoms can vary wildly in neurology and the intricacies of a few are discussed below. For *all* symptoms, you should try to understand:

- The exact nature of the symptom.
- The onset (sudden? Slow—hours? Days? Weeks? Months?).
- Change over time (progressive? Intermittent? Episodes of recovery?).
- Precipitating factors.
- Exacerbating and relieving factors.
- Previous episodes of the same symptom.
- Previous investigations and treatment.
- Associated symptoms.
- Any other neurological symptoms.

Dizziness

Narrow the exact meaning down without appearing aggressive or disbelieving. This term is used by different people to describe rather different things including…

- A sense of rotation = ‘vertigo’.
- ‘Swimminess’ or ‘lightheadedness’—a rather nonspecific symptom which can be related to pathology in many different systems.
- ‘Pre-syncope’—the rather unique feeling one gets just prior to fainting.
- Incoordination—many will say they are dizzy when, in fact, they can't walk straight due to either ataxia or weakness.

Headache

This should be treated as you would any other type of pain. Establish character, severity, site, duration, time course, frequency, radiation, aggravating and relieving factors, associated symptoms.

- Ask about facial and visual symptoms. (Some different types of headaches are described on p.359).

Numbness and weakness

These two words are often confused by patients-describing a leg as ‘numb’ when it is *weak* with normal sensation.

Also, patients may report ‘numbness’ when, in fact, they are experiencing pins-and-needles or pain.

Tremor

Here, you should establish if the tremor occurs only at rest, only when attempting an action or both. Is it worse at any particular time of the day? The severity can be established in terms of its functional consequence (can't hold a cup/put food to mouth?).

Again, establish exactly what is being described. A tremor is a shaking, regular or jerky involuntary movement.

Syncope

This is discussed in  Chapter 7.

Falls and loss of consciousness (LOC)

An eyewitness account is vital. Establish also whether the patient actually lost consciousness or not. People often describe ‘blacking out’ when in fact they simply fell to the ground (drop attacks have no LOC). An important question here is ‘can you remember hitting the ground?’.

Ask about preceding symptoms and warning signs-they may point towards a different organ system (sweating or weakness could be a marker of hypoglycaemia; palpitations may indicate a cardiac dysrhythmia).

Seizures

Very difficult even for experienced history-takers! Establish early on if there was any impairment of consciousness and seek collateral histories. Lay persons usually consider ‘seizure’ = ‘fit’ = tonic clonic seizure. Doctors' understanding of ‘seizure’ may be rather different. A surprising number of people also suffer ‘pseudoseizures’ which are non-organic and have a psychological cause.

A few points to consider:

- Syncopal attacks can often cause a few tonic-clonic jerks which may be mistaken for epilepsy.
- True tonic-clinic seizures may cause tongue-biting, urinary and faecal incontinence, or all of the above.
- People presenting with pseudoseizure can have true epilepsy *as well* and vice versa.

Visual symptoms

Commonly visual loss, double-vision, or photophobia (pain when looking at bright lights). Here, establish exactly what is being experienced—‘double vision’ (diplopia) is often complained of when, in fact, the vision is blurred or sight is generally poor (amblyopia) or clouded.

The rest of the history

▸ Remember to ask if the patient is right or left-handed (consider disability from loss of function and may also be useful when thinking about cerebral lesions).

Direct questioning

In every patient, enquire about neurological symptoms other than the presenting complaint (headaches, fits, faints, ‘funny turns’, blackouts, visual symptoms, pins-and-needles, tingling, numbness, weakness, incontinence, constipation, or urinary retention).

Past medical history

▸ A birth history is important here, particularly in patients with epilepsy. Brain injury at birth has neurological consequences.

A thorough history is required, as always, but enquire especially about:

- Hypertension—if so, what treatment?
- Diabetes mellitus—what type? What treatment?
- Thyroid disease.
- Mental illness (e.g. depression).
- Meningitis or encephalitis.
- Head or spinal-injuries.
- Epilepsy, convulsions, or seizures.
- Cancers.
- HIV/AIDS ( tread carefully, be tactful).

Drug history

Ask especially about:

- Anticonvulsant therapy (current or previous).
- Oral contraceptive pill.
- Steroids.
- Anticoagulants or anti-platelet agents.

Family history

A thorough history, as always, is important. Ask about neurological diagnoses and evidence of missed diagnoses (seizures, blackouts etc.).

Tobacco, alcohol

As important here as in any other system.

Social history

- Occupation—neurological disease can impact significantly on occupation so ask about this at an early stage—some suggest right at the beginning of the history. Also ask about exposure to heavy metals or other neurotoxins.
- Driving?—Many neurological conditions have implications here.
- Ask about the home environment thoroughly (will be very useful when considering handicaps and consequences of the diagnosis).
- Ask about support systems—family, friends, home-helps, day centre visits etc.

The outline examination

It is easy to get bogged down in some of the complexities of the neurological examination but it is not something to be afraid of. Students should embrace it—practise often, as a competent neurological examination is a sure sign of someone who has spent plenty of time on the wards. The following is a brief outline of how it should be approached.

- Inspection, mood, conscious level.
- Speech and higher mental functions.
- Cranial nerves II-XII.
- Motor system.
- Sensation.
- Co-ordination.
- Gait.
- Any extra tests.
- Other relevant examinations.
    - Skull, spine, neck stiffness, ear drums, blood pressure, anterior chest, carotid arteries, breasts, abdomen, lymph nodes.

General inspection and mental state

The neurological exam should start with any clues that can be gleaned from simply looking at, and engaging with, the patient.

- Are they accompanied by carers—and how do they interact with those people?
- Do they use any walking aids or other forms of support?
- Any abnormal movements? (📖 p.355).
- Observe the gait as they approach the clinic room, if able (📖 p.350).
- Any speech disturbance? (📖 p.286).
- What is their mood like?

- A detailed mood assessment (📖 p.498) is not necessary here…
- Ask the patient how they feel.
- What is the state or their clothing, hair, skin, and nails?
- Is there any restlessness, inappropriately high spirits, or pressure of speech?
- Are they obviously depressed with disinterest?
- Are they denying any disability?

Speech and language

Speech and language difficulties, especially expressive dysphasia, may be extremely distressing for the patient and their family. This topic must be approached with caution, reassurance, and a calm seriousness in the face of possible bizarre and amusing answers to questions.

Examination

- Speech and language problems may be evident from the start of the history and require no formal testing. You should briefly test their language function by asking them to read or obey a simple written command (e.g. close your eyes) and write a short sentence.
- If apparently problematic, speech *can* be tested formally by asking the patient to respond to progressively harder questions … yes/no questions, simple statements, more complicated sentences, and finally by asking them to repeat complex phrases or tongue-twisters (see below).
- Before jumping to conclusions, ensure that the patient is not deaf (or that their hearing-aid is working) and that they can usually understand English.

Dysarthria

A defect of articulation with language function intact (writing will be unaffected). May be a cerebellar lesion, a LMN lesion of the cranial nerves, an extrapyramidal lesion, or a problem with muscles in the mouth and jaws or their nerve supply.

- Listen for slurring and the rhythm of speech.
- Test function of different structures by asking the patient to repeat:
  - 'Yellow lorry' or words with 'D', 'L' and 'T' (*tongue* function).
  - 'Peter Piper picked a pickle' or words with 'P' and 'B' *(lip* function).
- *Cerebellar lesions:* slow, slurred, low volume with equal emphasis on all syllables ('scanning').
- *Facial weakness:* speech is slurred.
- *Extrapyramidal lesions:* monotonous, low volume and lacking in normal rhythm.

Dysphonia

Defective volume—huskiness. Usually from laryngeal disease, laryngeal nerve palsy or, rarely, muscular disease such as myaesthenia gravis.

May also be 'functional' (psychological).

Dysphasia

This is a defect of *language*, not just speech, so reading and writing may also be affected (some patients attempt to overcome speaking difficulties with a notepad and pen only to be bitterly disappointed).

In very simple terms, the main language areas of the brain are illustrated opposite. Deficits can be understood in terms of lesions in one or more of these areas. There are 4 main types of dysphasia…

Global dysphasia

(Both Broca's and Wernicke's areas affected) The patient is unable to speak or understand speech at all.

Expressive dysphasia

Also called 'anterior', 'motor', or 'Broca's' dysphasia.

- Lesion in Broca's area (frontal lobe), involved in language production.
- Understanding remains intact.
- Unable to answer questions appropriately.
- Speech is non-fluent, broken with abnormal word ordering.
- Unable to repeat sentences.
- ▸ Can be very distressing for patients. Ask ‘do you know what you want to say, but can't get it out?’ and you'll be met with a grateful smile, nod, and handshake.

Receptive dysphasia

Also called ‘posterior’, ‘sensory’, or ‘Wernicke's’ dysphasia.

- Lesion in Wernicke's area creates problems understanding spoken or written language (dyslexia) and problems with word-finding.
- Unable to understand commands or questions.
- Speech is fluent with lots of meaningless grammatical elements.
- May contain meaningless words.
- Unable to repeat sentences.
- Patients are often unaware of their speech difficulty and will talk nonsense contentedly—although may become frustrated with other people's lack of understanding!
    - *‘Jargon dysphasia’* describes a severe form of receptive dysphasia containing only meaningless words (‘neologisms’) and sounds.
    - Paraphasia is the supplementation of one word with another.

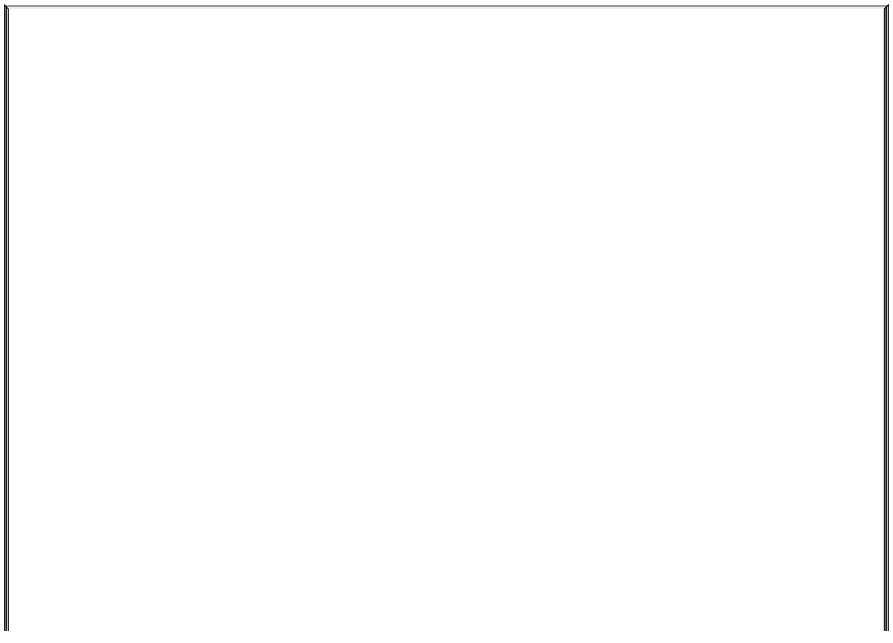
Conductive dysphasia

Lesion in the arcuate fasciculus and/or other connections between the 2 primary language areas.

- Patient can comprehend and respond appropriately.
- Unable to repeat a sentence.

Nominal dysphasia

- All language function is intact *except* for naming of objects.
- Caused by lesion in angular gyrus.
- Patient may function with ‘circumlocution’. (e.g. says ‘that thing that I write with’ if unable to say ‘pen’).



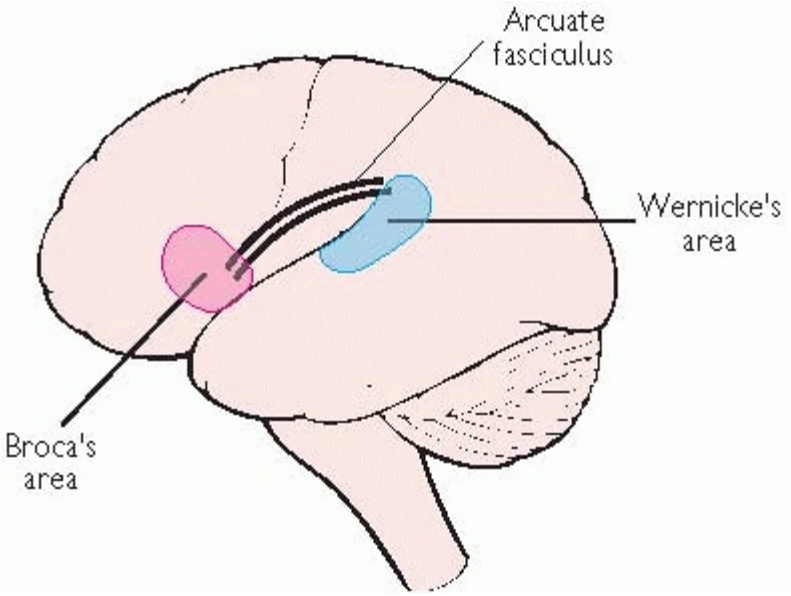


Fig. 10.1 Simple representation of the main language areas of the brain.

Cognitive function

Neurological diseases may affect function such that patients' *appearance* or communication skills are at odds with their social standing or educational level—formal assessment of a person's mental state is important.

This also allows for any future change to be noted and monitored.

The abbreviated mental test score (10 points)

This serves as a brief screening tool with a maximum score of 10 points. A more detailed, 30 point, score is shown in Box 15.11 p.503.

▸ Approach this gently—patients often dislike being tested without warning. 'Do you think I'm stupid?' Always explain the purpose of the questions—and ask their permission to proceed.

Table 10.1 The abbreviated mental test score

1.	Date of birth	•	'What is your date of birth?'
2.	Age	•	'How old are you?'
3.	Time	•	'What time is it?'
		•	Correct to the nearest hour.

4.	Year	•	‘What year is it now?’
		•	Note that hospital patients often lose track of the day or month not the year.
5.	Place	•	‘Where are we?’ or ‘What is this place?’
		•	The name of the hospital/clinic/surgery.
6.	Head of state	•	‘Who's the Prime Minister at the moment?’
		•	A name is required. Such descriptions as ‘That man in all the trouble’ won't do-even if it is potentially correct!
7.	World War II	•	‘What year did the second World War start?’
8.	5-minute recall	•	Tell the patient an address (often ‘42 West Street’ is used) and ask them to repeat it back to you to ensure they've heard it correctly. Ask them to remember it. Five minutes later, ask them to recall the address.
		•	They must remember the address *in full to* score the point.
9.	20-1	•	‘Count backward from 20 down to 1’
		•	Patients sometime need a prompt here ‘Like this: 20, 19, 18, and so on’.

‘What job do I do?’ (doctor) and ‘What job does this man/woman

10.	Recognition	•	do?' (nurse)
		•	*Both* must be correct to score a point.

▸ ***Hints***

- Obviously, if your country does not have a Prime Minister, substitute with 'President' or 'Monarch'. Again, a name is required-'the Queen' is not a correct answer.
- When testing 5-minute recall:
  - If thinking of an address for the patient to remember-be careful not to give out your own!
  - Beware of repeating the test too often. Patients may well remember '42 West Street' from the last time it was asked!

Cranial nerve I: olfactory

Applied anatomy

Sensory: smell.

Motor: none.

Fibres arise in the mucous membrane of the nose. Axons pass across the cribiform plate to the olfactory bulb. Olfactory tract runs backwards below the frontal lobe and projects, mainly, in the uncus of the ipsilateral temporal lobe.

Note: olfactory epithelium also contains free nerve endings of the 1st division of cranial nerve V.

Examination

Not routinely tested unless the patient complains of a loss of sense of smell (anosmia) and exhibits other signs suggestive of a frontal or temporal lobe cause (e.g. tumour).

Casual: take a nearby odorous object (e.g. coffee or chocolate) and ask the patient if it smells normal.

Formal: a series of identical bottles containing recognizable smells are used. The patient is asked to identify them. Commonly used agents: coffee, vanilla, camphor, vinegar.

Test each nostril separately and determine if any loss of smell is uni- or bilateral.

Findings

Bilateral anosmia: usually nasal, not neurological.

Causes include upper respiratory tract infection, trauma, smoking, old age, and Parkinson's disease. Less commonly, tumours of the ethmoid bones or congenital ciliary dysmotility syndromes.

Unilateral anosmia: mucous-blocked nostril, head trauma, subfrontal meningioma.

▸ ***Hints***

Peppermint, ammonia, and menthol stimulate the free trigeminal endings so are not a good test of cranial nerve I.

Cranial nerve II: optic

Applied anatomy

With an understanding of the anatomy of the optic nerves, defects in the visual field enable the localization of a lesion within the brain.

The optic nerve begins at the retina (and is the only part of the central nervous system that can be directly visualized). The nerve passes through the optic foramen and joins its fellow nerve from the other eye at the 'optic chiasm' just above the pituitary fossa. Here, the fibres from the nasal half of the retina cross over. They continue in the optic tract to the lateral geniculate body. From there, they splay

out such that those from the upper retina pass through the parietal lobe and the others through the temporal lobe.

Students easily get confused here and would do well to get to grips with this at an early stage! Because of the refraction at the lens, images are represented on the retina upside-down and back-to-front. Therefore, the *nasal* half of the retinal receives input from the *temporal* part of the visual field in each eye, whist the temporal half of the retinal receives input from the nasal half of the eye.

Further back in the optic system, fibres from the nasal halves of the retinas cross, so, for example, the left side of the brain receives input from the right side of vision (the left temporal retina and the right nasal retina) and vice versa (see Fig. 10.2 p.295).

Visual acuity

Sharpness, clarity of vision. Formally tested using a Snellen's Chart.

- In good light, the patient should stand 6m away from the chart.
- Each eye is tested in turn and the patient is asked to read the chart.
- The number above each line indicates the distance at which a person with normal sight should be able to read it.
- Record the line reached—allow a maximum of errors per line.
    - Indicate results as: distance from chart/distance it should be read e.g. '6/36'.

If the patient can't see any of the letters, record whether they can:

- Count fingers held in front of their face (CF).
- See hand movements (wave your hand).
- Perceive light.
    - Record as CF, HM, PL, or NPL (not perceive light).

Colour vision

- Not tested routinely and not considered in this book.
- Tested using Ishihara plates.

Visual fields

The area that each eye can see without moving can be mapped out. They are not circular—eyebrows and nose obstruct superiorly and nasally whereas there is no obstruction laterally.

Sitting opposite the patient, the examiner's left visual field (for example) should be an exact mirror image of the patient's right visual field. In this way, the patient's fields can be tested against the examiner's.

Gross defects and visual neglect (inattention)

- Sit opposite the patient, ~1m apart, eyes level.
- Test first for gross defects and visual neglect with both eyes open.
    - Raise your arms up and out to the sides so that one hand is in the upper right quadrant of your vision and one in the upper left.
    - Ask the patient to look directly at you ('look at my nose').
    - Move one index finger and ask the patient, whilst looking straight at you, to point to the hand which is moving.
    - Test with the right, left, and then both hands.
    - Test the lower quadrants in the same way.
    - If visual neglect is present, the patient will be able to see each hand moving individually but reports seeing only one hand when both are moving (compare with sensory inattention p.340).

Testing each eye

- In the same position as above, ask the patient to cover their right eye while you cover your left and look directly at one another.
  - (If you were now to trace the outer borders of your vision in the air half way between yourself and the patient, if should be almost identical to the area seen by the patient).
- Test each quadrant individually:
  - Stretch your arm out and up so that your hand is just outside your field of vision, an equal distance between you and the patient.
  - Slowly bring your hand into the centre (perhaps wiggling one finger) and ask the patient to say 'yes' as soon as they can see it.
  - You should both be able to see your hand at the same time.
  - Test upper right and left, lower right and left individually, bringing your hand in from each corner of vision at a time.
  - ▸ Ensure that the patient remains looking directly at you (many will attempt to turn and look at the hand if not prompted correctly).
  - Map out any areas of visual loss in detail, finding borders. Test if any visual loss extends across the midline horizontally or vertically.
  - Test each eye in turn (you both may require a short break between eyes as this requires considerable concentration).
- Repeat the above procedure with a red-headed pin or similar small red object to map out areas of visual loss in more detail.
  - Ask the patient to say 'yes' when they see the pin *as red*.
  - Start by mapping out the blind spot which should be ~15° lateral from the centre at the midline (this tests both your technique and the patient's reliability as a witness before proceeding).
- Decide if any defect is of a quadrant, half the visual field or another shape and in which eye, or both. Record by drawing the defect in 2 circles representing the patient's visual fields as shown in  Fig. 10.2 p.295.

If the patient is unable to cooperate

Like much of the neurological examination, gross defects can be seen without the patient's cooperation (confused or drowsy). Test for response to 'menace' by bringing your hand in sharply from the side, stopping just short of hitting the patient in the eye. If your hand can be seen, the patient will blink. Test vision on the left and the right.

Some common visual field defects

Compare the defects below with the corresponding number on Fig. 10.2 showing the position of the lesion and a representation of the fields as it should be recorded in the patient's notes.

- ***Tunnel vision:*** a confusing term. A constricted visual field, giving the impression of looking down a pipe or tunnel may be caused by glaucoma, retinal damage or papilloedema. 'Tubular' vision is often functional.
- ***Enlarged blind spot:*** caused by papilloedema.
- ***Unilateral field loss:*** (1) blindness in one eye caused by devastating damage to the eye, its blood supply, or optic nerve.
- ***Central scotoma:*** a hole in the visual field (macular degeneration, vascular lesion or, if bilateral, toxins). If bilateral, may indicate a very small defect in the corresponding area of the occipital cortex (multiple sclerosis).
- ***Bitemporal hemianopia:*** (2) the nasal half of both retinas and, therefore, the temporal half of each visual field is lost (damage to the centre of the optic chiasm such as pituitary tumour, craniopharyngioma, suprasellar meningioma).
- ***Binasal hemianopia:*** the nasal half of each visual field is lost (very rare).
- ***Homonymous hemianopia:*** (3) may be 'left' or 'right'. Commonly seen in stroke patients. The right or left side of vision in both eyes is lost (e.g. the nasal field in the right eye and the temporal field in the left eye). If the central part of vision (corresponding to the macula) is spared, the lesion is likely in the optic radiation, without macula sparring, the lesion is in the optic tract.
- ***Homonymous quadrantanopia:*** corresponding quarters of the vision is lost in each eye (e.g. the upper temporal field in the right and the upper nasal field in the left).
  - Upper quadrantanopias (4) suggest a lesion in the temporal lobe.
  - Lower quadrantanopias (5) suggest a lesion in the parietal lobe.

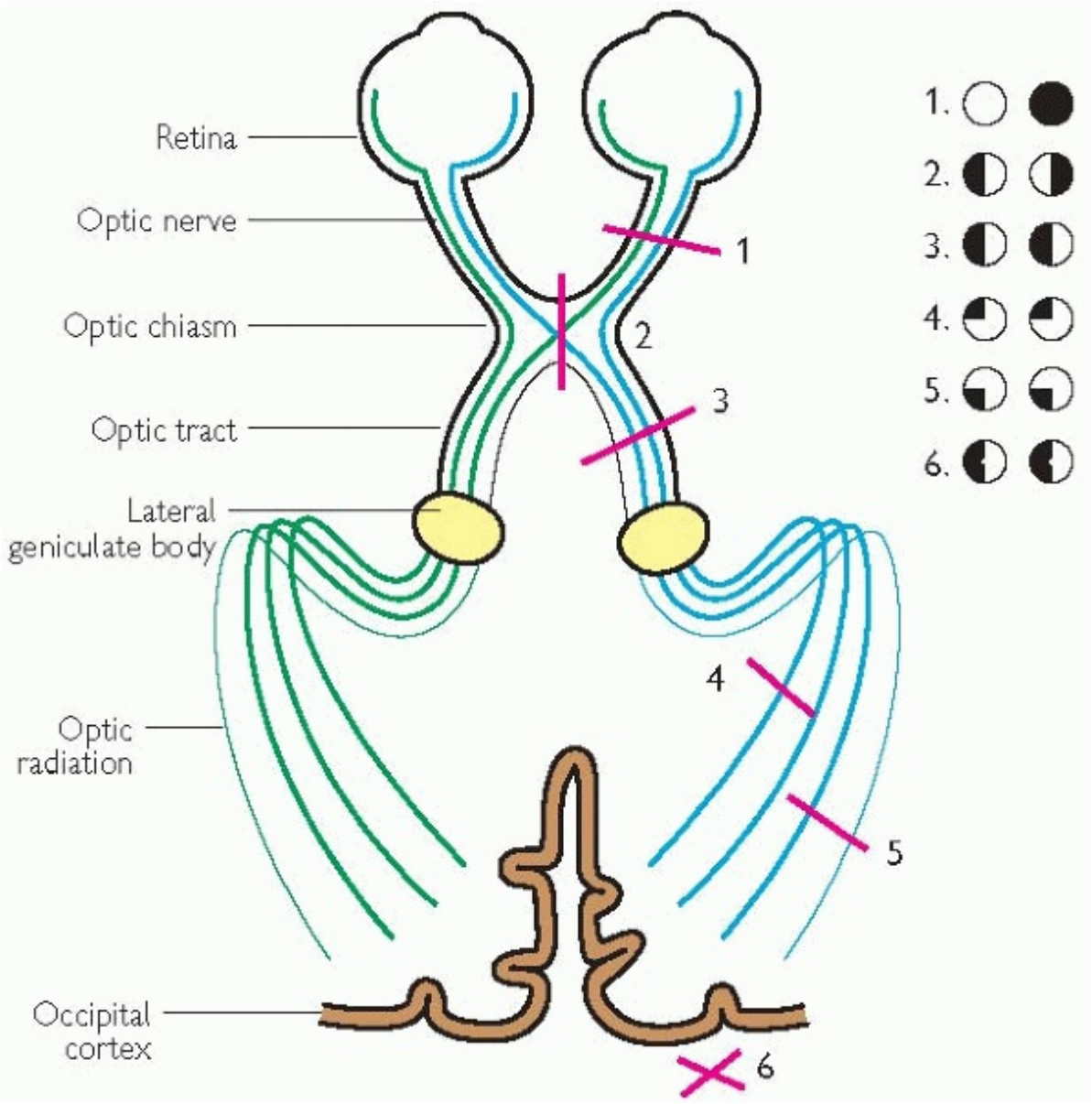


Fig. 10.2 Representation of the visual tracts from the retina to the occipital cortex showing main structures and expected visual field loss according to the site of the lesion.

Cranial nerve II: ophthalmoscopy

Direct ophthalmoscopic examination of the fundus is a vital part of any neurological examination but often avoided as it is considered difficult.

It can provide the observer with vital information about the condition of the optic nerve head. It takes practise but the experienced observer can gain views of the fundus, macular region, and the retinal vascular arcades. It is worth practising at every opportunity. The direct ophthalmoscope gives a greatly magnified view of the fundus but gaining a view of the peripheral retina beyond the equator requires examination with the slit lamp or the indirect ophthalmoscope—not covered in this book.

For a complete ophthalmoscopic examination it is often worth dilating the pupil by instilling a few drops of mydriatic (1% tropicamide or 1% cyclopentolate) into the inferior conjunctival sac. With a little practise one often finds that this is not necessary for a routine examination.

If you plan to dilate the pupil, ask the patient if they have any history of angle closure glaucoma or episodes of seeing haloes around lights at night-time. If you suspect this, or the anterior chamber of the eye appears shallow, it is best to err on the side of caution—dilating the pupil could occlude the drainage angle and precipitate an acute attack.

Examination

- Performed in a dimly lit room with the patient sitting or lying down.
- Ask the patient to focus on a distant object and keep their eyes still (relaxes accommodation as much as possible).
- Look through the ophthalmoscope ~30cm away from the patient and bring the light in nasally from the temporal field to land on the

pupil.

- The pupil will appear red and opacities in the visual axis will appear as black dots or lines.
- By cycling through the different lenses of the ophthalmoscope, you should be able to gain an impression of where these opacities lie. Possible locations are the cornea, aqueous, lens (and its anterior and posterior capsules) and vitreous.

- Dial up a hypermetropic (plus) lens on the ophthalmoscope to focus on the corneal surface and move in as close as possible to the patient's eye—by gradually ↓ the power of the lens you can examine the cornea, iris and lens in turn. (Formal examination of these structures is best done with the slit lamp but a great deal of information can be gained with the direct ophthalmoscope.)
- Continue to ↓ the power of the lens until you can sharply focus on the retinal vessels. It is often best to pick up one of the vascular arcades in the periphery and track them in towards the optic disc. This allows the peripheral quadrants to be examined in turn before viewing the optic disc. Take time to look at the vessels carefully, particularly where the arteries cross the veins.
- Ask the patient to look directly into the light of the ophthalmoscope to gain a view of the macular region.

We thank Dr Tom Fearnley for contributing this topic.

The normal fundus

The optic disc

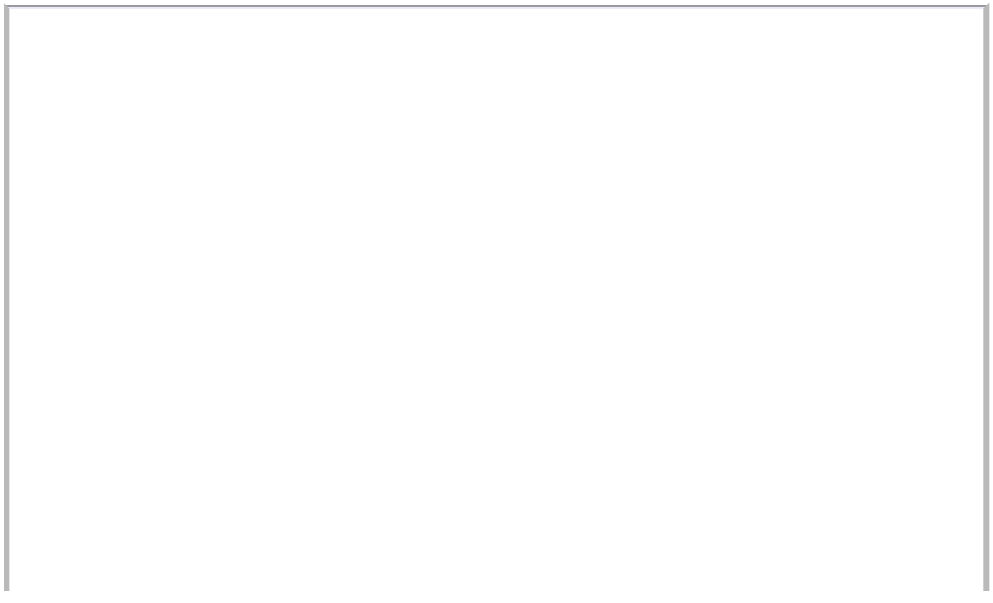
- The healthy disc is a pale pink/yellow colour and round or slightly oval in shape.
- The margins between the disc and the surrounding retina should be crisp and well defined. Occasionally a surrounding ring is present which may be slightly lighter or darker in colour.
- At the centre of the disc is the physiological cup. It appears paler in colour compared to the rest of the disc.

The macular region

- Located temporally from the optic disc.
- This is the region with the maximum concentration of cones.
- At the centre of the macula is the fovea-a tiny pit devoid of blood vessels and responsible for fine resolution.
- Disease involving the macula and fovea can cause devastating visual loss.

The retinal vessels

- The central retinal artery and vein enter and leave the globe in the centre of the optic disc.
- Veins appear larger and darker in colour in comparison to the arteries.
- Spontaneous venous pulsations are seen in many normal eyes.
- Arterial pulsations should not be visible in normal eyes.



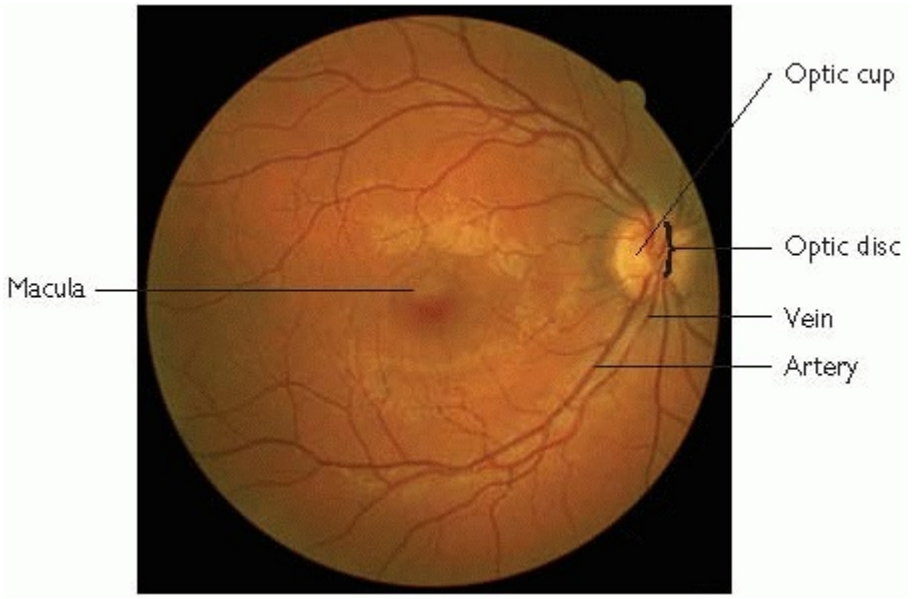


Fig. 10.3 The normal appearance of the fundus of the right eye.

▸ *Hint*

View the macula by directing the light on the most sensitive part of the eye. This can often be unpleasant for the patient and will lead to more marked miosis and a restricted view.

Abnormal findings on fundoscopy

Optic disc swelling

Appearance

- The optic disc is raised, swollen, and enlarged.
- The disc often appears darker in colour.
- The margins of the disc are blurred and become indistinct from the adjacent retina.
- Retinal vessels can be seen arching down from the raised disc towards the peripheral retina.
- In severe cases retinal haemorrhage may be seen around the disc.

▸ The term papilloedema is often, incorrectly, used to describe optic disc swelling. ‘Papilloedema’ is swelling of the optic disc due to raised intracranial pressure.

Causes

- Space occupying lesions including intracranial malignancy, subdural haematoma, and cerebral abscess.
- Subarachnoid haemorrhage (commonly associated with vitreous haemorrhage).
- Chronic meningitis.
- Idiopathic intracranial hypertension (IIH).
- Malignant hypertension.
- Ischaemic optic neuropathy.

Optic disc cupping

Appearance

- The physiological cup is ↑ in respect to the rest of the disc.
- Retinal vessels kink sharply as they emerge over the rim of the cup.
- Haemorrhages may be present.

Causes

Most commonly one of the various types of glaucoma.

Optic atrophy

Appearance

Pale optic disc due to loss of nerve fibres in the optic nerve head.

Cause

- Ischaemic optic neuropathy.
- Optic neuritis.
- Trauma.
- Optic nerve compression.

We thank Dr Tom Fearnley for contributing this topic.

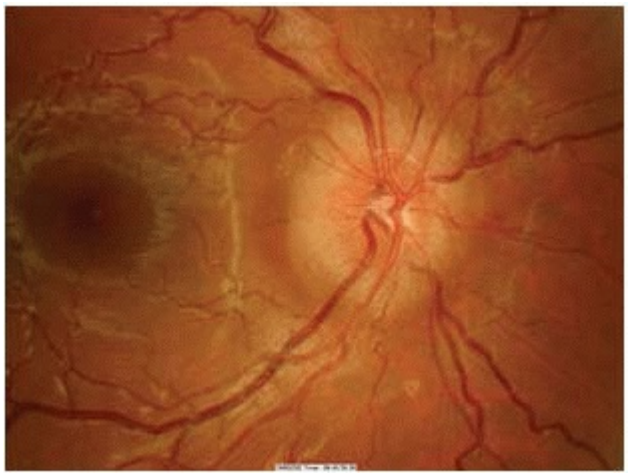


Fig. 10.4 Severe papilloedema. Note how the disc margins are blurred and there is a lack of the normal cupping at the disc.

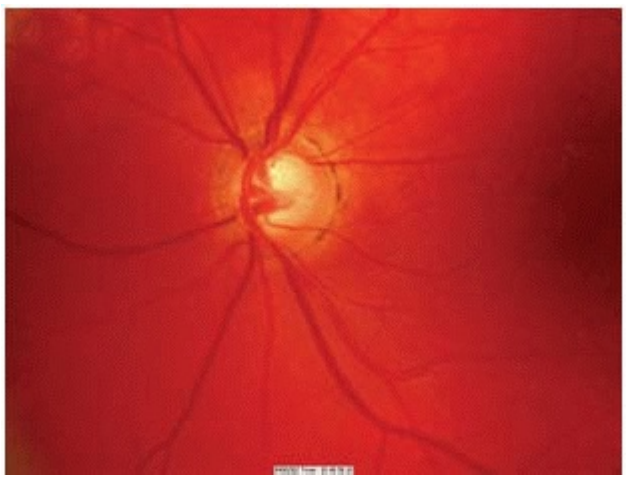


Fig. 10.5 Optic disc cupping, in this case secondary to glaucoma. Note how the vessels seem to disappear over the edge of the disc as if falling down a hole.

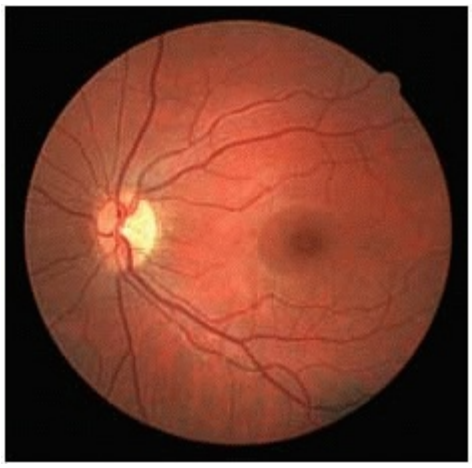


Fig. 10.6 Optic atrophy. The optic disc is pale and well demarcated.

Retinal haemorrhages

Appearance

The appearance of a haemorrhage depends on its location within the various layers of the retina. Deep haemorrhages appear as ‘dots’ due to the close packing of the cells in this region. More superficial haemorrhages in the nerve fibre layer appear as more widespread ‘blotches’.

Causes

Many pathological processes including:

- Diabetes mellitus.
- Hypertension.
- Subarachnoid haemorrhage.
- Blood dyscrasias.
- Systemic vasculitis.
- Valsalva related.
- Trauma.
- Bacterial endocarditis (known specifically as Roth spots).

Central/branch retinal artery occlusion

Appearance

- Large areas of ischaemic white retina associated with sudden catastrophic visual loss.
- Calcific, cholesterol or fibrin-platelet emboli can often be seen occluding the retinal artery/ branch.

Causes

Either embolic or thrombotic (remember giant cell arteritis also).

Central/branch retinal vein occlusion

Appearance

- Large, widespread flame shaped haemorrhages classically giving the fundus a ‘stormy sunset’ appearance. Associated with gradual onset painless blurred vision and visual loss.
- Optic disc swelling may be present.

Causes

- Blood dyscrasias.
- Diabetes mellitus.
- Glaucoma.

Foster-Kennedy Syndrome

Appearance

- Unilateral optic atrophy.
- Contralateral papilloedema.
- Central scotoma.
- Anosmia (variable).
- Systemic symptoms such as headache, dizziness, vertigo, and vomiting.

Causes

- Meningioma of optic nerve, olfactory groove or sphenoid wing.
- Frontal lobe tumour.

The pupils

Applied anatomy

The pupil is the aperture at the centre of the iris. Variation in pupil size is brought about by 2 muscles in the iris under the control of the autonomic nervous system.

- Sphincter pupillae muscle—found in the iris at the margin of the pupil. Innervated by parasympathetic fibres. Constricts the pupil (miosis).
- Dilator pupillae muscle—radially arranged smooth muscle. Innervated by sympathetic nervous system. Dilates the pupil (mydriasis).

The pupillary light response

The pupillary light response has afferent and efferent limbs that can be affected separately in a number of pathologies. The afferent fibres leave the eye in the optic nerve and separate in the midbrain to synapse with the 3rd nerve nuclei. Efferent pathway fibres then travel to synapse in the ciliary ganglion before innervating the sphincter pupillae.

Examination

- Inspect both pupils in good light—is there a discrepancy in size (anisocoria) or shape? (This is present in 25% of the ‘normal’ population and does not necessarily indicate pathology. It may be secondary to previous ocular inflammatory disease, trauma or surgery.)

- If anisocoria is present one must determine which of the pupils is the ‘correct size’.
  - A pathologically constricted pupil is more obvious in dim light as the normal pupil dilates.
  - A pathologically large pupil will be more apparent in bright illumination when the normal pupil will constrict.
- Test the pupil responses to direct and consensual light. This is best done in a dimly lit room. Ask the patient to look into the distance to ensure the eye is relaxed and ‘dis-accommodated’. Shine a light upwards from just inferior to the lower lid to avoid dazzling the patient.
  - Constriction of the pupils should be seen almost instantaneously in response to illumination in both the illuminated eye (direct) and the non-illuminated eye (consensual). Repeat for both eyes.
- The afferent limb of the pupillary light pathway is assessed using the Marcus-Gunn swinging light test to look for a relative afferent pupil defect (RAPD). If present:
  - Shine light in the normal eye and both pupils constrict. (The consensual response in the affected eye is intact.)
  - Swing the light to the affected eye and both pupils dilate. (Afferent drive to cause constriction of the pupils from the affected eye is reduced in comparison to that of the unaffected eye.)
  - Swing the light back to the normal eye and both pupils constrict.
- Finally, check the near reflex (the efferent limb of the pupil reflex). Ask the patient to focus on a distant object and then look immediately to your index finger held ~30cm in front of their face.
  - The normal response will be for the pupils to constrict in response to convergence and accommodation.

Findings: some pupil abnormalities

Argyll-Robertson pupil

Midbrain lesions caused by neurosyphilis target the more dorsally located fibres that subserve the light response. The ventrally located fibres responsible for accommodation are spared.

- *Appearance:* a small irregular pupil that accommodates but does not react to light.
- *Causes:* neurosyphilis and diabetes mellitus.

Holmes-Adie pupil

Denervation of iris and ciliary body due to ciliary ganglionitis (although some would dispute this!). Associated loss of tendon reflexes is seen in some patients and is termed Holmes-Adie syndrome.

- *Appearance:* unilateral dilated pupil—accommodates (and relaxes) very slowly and shows absent or depressed light reflex Supersensitive to 0.1% pilocarpine (muscarinic agonist causing constriction).
- *Causes:* usually idiopathic and predominates in young adult females (♂:♀ ≈ 2:1). May also follow iridoplegia or ocular trauma.

Horner's syndrome

Interruption of the sympathetic nerve supply to the iris.

- *Appearance:* unilateral miotic pupil with partial ptosis (due to paralysis of Muller's muscle—a small smooth muscle in the upper lid). Movement of the upper lid should be intact as the levator muscle is supplied by the oculomotor nerve. There is also a variable interruption of sudomotor innervation to the ipsilateral side of the face. Sweating is absent if the lesion occurs proximal to the carotid plexus, after which the sudomotor fibres separate.
- *Causes:* the protracted course of the sympathetic pathway makes it vulnerable to disruption at many different points. Causes include: congenital—often associated with an alteration in iris pigment (heterochromia); injury or surgery to the neck (avulsion of C8 and T1 nerve roots results in Klumpke's paralysis); multiple sclerosis; cavernous sinus disease; neoplasia involving the mediastinum, cervical cord or the apex of the lung; infarction—secondary to occlusion of the basilar or posterior inferior cerebellar artery; thoracic aortic aneurysm; syringomyelia or syringobulbia.

Box 10.1 More on the RAPD

At rest the patient's pupils will be equal and of normal size. It is ‘relative’ because the response seen when light is shone on the affected pupil is diminished relative to the response seen when light is shone in the normal pupil, and ‘afferent’ since it demonstrates a problem in the afferent limb of the light response in the affected eye. It indicates unilateral/asymmetrical optic nerve disease or extensive retinal pathology. An RAPD will not be seen in patients with corneal or lens opacities.

We thank Dr Tom Fearnley for contributing this topic.

Cranial nerves III, IV, and VI

The 3rd *(oculomotor)*, 4th *(trochlear)* and 6th *(abducens)* nerves are considered together as their primary function is to provide motor innervation to the extrinsic muscles of the eye. Connections exist with the horizontal gaze centre in the pons and the vertical gaze centre in the midbrain.

Applied anatomy: III

Motor: levator palpebrae superioris, superior rectus, medial rectus, inferior rectus, inferior oblique. (All the extrinsic muscles of the eye except the lateral rectus and superior oblique.)

Autonomic: parasympathetic supply to the constrictor (sphincter) pupillae of the iris and ciliary muscles.

The main oculomotor nucleus lies anterior to the aqueduct of the midbrain.

The Edinger-Westphal nucleus (accessory parasympathetic nucleus) lies posterior to the oculomotor nucleus. Fibres pass anteriorly, through the cavernous sinus and enter the orbit through the superior orbital fissure.

Applied anatomy: IV

Motor: superior oblique.

The nucleus lies just inferiorly to that of the oculomotor nerve and has connections with the cerebral hemispheres, visual cortex and nerves III, VI, and VIII. Its fibres pass posteriorly and immediately cross one another. They then travel through the cavernous sinus, entering the orbit through the superior orbital fissure.

Applied anatomy: VI

Motor: lateral rectus.

The nucleus lies beneath the 4th ventricle. It connects with the nuclei of the III and IV cranial nerves through the medial longitudinal fasciculus. It emerges from the pons and travels through the cavernous sinus to enter the orbit through the superior orbital fissure.

Examination

The patient should be sitting facing you with their eyes straight ahead. Ensure visual acuity has already been assessed and recorded.

- Inspect the position of the lids.
  - Is there ptosis (drooping of the lid)?
  - Are the epicanthic folds prominent? (This may cause pseudosquint).
- Look at the position of the eyes in neutral gaze.
  - An asymmetrical position suggests strabismus (squint) and this should be assessed with the cover test (see Box 10.2 p.306).
- Ask the patient to follow your index finger in vertical, horizontal and oblique planes avoiding extremes of gaze. Drawing a large imaginary 'H' directly in front of the patient.
  - Is nystagmus present (rapid 'to and fro' movements of the eyes)?
  - Ask the patient if they see double at any stage? (Diplopia.)

- The patient's eyes should be able to follow the moving target smoothly. This is termed ***pursuit***. (Often slowed or interrupted with saccades in Huntington's chorea and Parkinson's disease.)
- Now hold up your index finger on one side of their head and your thumb on the other—in their temporal visual fields. Ask the patient to look quickly between finger and thumb. This tests saccadic eye movements—they should be accurate, smooth and rapid.
- Ask the patient to look from a distant object to a near object—the eyes should converge smoothly and equally in association with accommodation and pupil constriction. This is called convergence.

▸ *Hints*

Patients will often attempt to turn their head to look at your moving finger. This can be overcome in 2 ways:

- Fully explain the examination before beginning. Often, an instruction such as 'please follow the tip of my finger with your eyes but

keep your head still' works wonders.

- If the patient continues to turn their head, you can stabilize it by gently placing your free hand on their forehead.

We thank Dr Tom Fearley for contributing this topic.

Abnormal findings

Ptosis (drooping of the lid)

Causes include:

- Weakness of the levator muscle in myasthenia gravis.
- 3rd nerve palsy.
- Disruption of the insertion of the levator muscle into the tarsal plate of the lid either through surgery or trauma.

Strabismus/squint

Abnormality of coordinated eye movements. Divergent squint: one eye is directed towards the target, the other is turned laterally. In convergent squint, the other eye is turned medially.

Squint is broadly categorized into 2 forms.

- *Non-paralytic* seen in childhood. Both eyes have a full range of movement but only one of the eyes is directed towards the target of fixation.
- *Paralytic squint:* movement of one or more of the extraocular muscles is ↓ due to disease of the muscle, a nerve palsy, or a physical obstruction to movement in a particular direction (e.g. tethering, trauma, or neoplasm).

Box 10.2 The cover-uncover test

Used for further analysis of non-paralytic squint.

- The patient should be sitting in front of you.
- Present a fixation target in front of them (the top of your pen for example).
- Ask them to cover their right eye.
- Closely observe the uncovered left eye-one of three responses is possible:
  - The eye doesn't move—normal
  - The eye moves nasally to fixate—divergent squint present
  - The eye moves temporally to fixate—convergent squint present.
- Now repeat the test covering the left eye.

▸ Pick up a subtle squint by holding a pen torch about 30cm away from the centre of the patients face. The reflection of light should be from the same position on the cornea in both eyes. If this is not the case, the fixating eye will have the central reflection.

A more sophisticated assessment of squint is made in eye clinics using a syntophore.

▸ Further assessment of a squint should always involve a detailed examination of the cornea, lens, vitreous and retina to exclude opacities and abnormalities.

Nystagmus

Oscillating movements of the eyes—several subclassifications exist based on clinical appearance and lesion location. Watch the movements carefully. Are the to and fro phases of the movements the same speed in both directions or is one more rapid than the other?

- *Vestibular:* a type of jerk nystagmus (to and fro movements are of different velocities). Caused by disease in the labyrinth or its central connections. The fast phase is away from the side of the lesion. There are often horizontal and rotary components. Usually only present in the acute phase of labyrinthine disease.
- *Pendular nystagmus:* the velocity of the movements is the same in both directions. Often a congenital condition associated with ↓ visual acuity. Also seen in cerebrovascular disease and multiple sclerosis.

- ▸Patients with acquired nystagmus will often complain of continual movement of their visual environment (oscillopsia), which is not the case with congenital nystagmus.

- *Optokinetic nystagmus:* this is a normal response of the eye when trying to follow a moving object (e.g. when looking from the window of a train). It is formally assessed using a rotating drum painted with vertical black and white lines. Movements are controlled by the cerebral hemisphere towards which the drum is rotating causing pursuit movement in the direction of rotation followed by saccadic movement back in the opposite direction. Defective optokinetic nystagmus is seen in lesions of the deep parietal lobe when drum rotation is towards the affected cerebral hemisphere.
- *Upbeat nystagmus:* the fast phase is upwards. Causes include brainstem disease or intoxication with alcohol and a number of other drugs including phenytoin.
- *Downbeat nystagmus:* the fast phase is downward. Seen in toxic states and demyelinating disease. Also herniation of cerebellar tissue through the foramen magnum as seen in Chiari malformation.
- *Gaze evoked nystagmus:* the fast phase is toward the direction of action of the affected muscle. Usually seen in dysfunction of extraocular muscles secondary to intrinsic weakness or nerve palsy.

▸ *Hint*

When assessing nystagmus try to avoid the extremes of lateral gaze (i.e. not >30°). This will elicit end-point nystagmus—a physiological response not to be confused with a pathological process.

We thank Dr Tom Fearnley for contributing this topic.

Palsies of cranial nerves III, IV, and VI

III: oculomotor

Appearance: the pupil is dilated and responds to neither light nor accommodation. All the extraocular muscles are paralysed except for the lateral rectus and the superior oblique. The unopposed action of these cause the eye to look down and out. Paralysis of the levator muscle causes complete ptosis.

Causes: diabetes mellitus (pupil sparing), lesions involving the superior orbital fissure, cavernous sinus disease, aneurysm of the posterior communicating artery, Weber's syndrome (associated contralateral hemiplegia).

IV: trochlear

Appearance: Paralysis of the superior oblique causes the eye to elevate when adducting. The patient complains of diplopia and will have difficulty looking downwards and inwards on the affected side. The patient may try to compensate for this by tilting their head away from the side of the lesion (ocular torticollis).

Causes: trauma, surgery, diabetes mellitus, atherosclerosis, neoplasia.

VI: abducens

Appearance: paralysis of the lateral rectus muscle means the eye cannot be abducted from the midline and the unopposed action of the medial rectus leaves the eye deviated nasally at rest. The patient complains of diplopia in horizontal gaze. Lesions in the 6^{th} nerve nucleus also involve the lateral gaze centre and lead to a gaze paresis.

Causes: diabetes mellitus, atherosclerosis, multiple sclerosis, neoplastic lesions, raised intracranial pressure leading to compression of the nerve on the edge of the petrous temporal bone (a false localizing sign), trauma, surgery.

Combined nerve palsies

Due to the close proximity of nerves III, IV, and VI at points along their courses, lesions at specific anatomical locations can lead to combined nerve palsies.

The cavernous sinus

All 3 nerves involved in oculomotor control along with sympathetic fibres to the iris and the ophthalmic and maxillary divisions of the trigeminal nerve pass through here. Common lesions include: carotico-cavernous fistula; expanding pituitary tumour; cavernous sinus thrombosis-associated with proptosis and injection of conjunctival vessels (chemosis); aneurysm.

The orbit

A complex range of ophthalmoplegias can result from any compressive lesion located within the orbit. Proptosis may be present with variable optic nerve involvement. Many lesions may directly impinge upon the extraocular muscles as well as the innervating nerves.

The superior orbital fissure

The superior orbital fissure transmits all the nerves supplying the extraocular muscles along with the ophthalmic division of the trigeminal

nerve. Inflammation or a lesion at the superior orbital fissure leads to *Tolosa-Hunt syndrome*—a complex unilateral ophthalmoplegia associated with anaesthesia over the forehead and ocular pain.

Some other eye movement disorders

Internuclear ophthalmoplegia

This is interruption of the medial longitudinal fasciculus, connecting the nuclei of cranial nerves III and VI on opposite sides.

Voluntary gaze towards one side is initiated by the opposite cerebral hemisphere. Descending fibres then decussate to the horizontal gaze centre in the pons and parapontine reticular formation where further impulses are transmitted directly to the 6^{th} nerve nucleus causing abduction of the ipsilateral eye. Conjugate adduction of the contralateral eye is brought about by impulses transmitted via the medial longitudinal fasciculus to the 3^{rd} nerve nucleus thus maintaining binocular single vision.

Appearance

- Impaired adduction in the ipsilateral eye in unilateral lesions-nystagmus is often seen in the abducting eye.
- Bilateral lesions often cause vertical nystagmus and impaired vertical pursuit.
- Convergence remains intact.
- The patient will complain of horizontal diplopia due to impaired adduction on the affected side—not due to nystagmus in the abducting eye.

Common causes: cerebrovascular disease, multiple sclerosis.

Lesions of the parapontine reticular formation (PPRF)

The PPRF is responsible for conjugate eye movements in horizontal gaze.

Appearance

- Failure of horizontal eye movements towards the side of the lesiona horizontal gaze paresis.
- An ipsilateral internuclear ophthalmoplegia if the lesion extends to involve the MLF.
- Preservation of vertical gaze.
- Contralateral deviation of the eyes in the acute phase.

Causes: vascular disease, demyelinating disease, neoplasia.

Parinaud's syndrome

Lesions occurring in the dorsal midbrain involve the vertical gaze centrehence also known as dorsal midbrain syndrome.

Appearance

- Impaired upward gaze in both eyes resulting in convergence, retraction of the globe into the orbit and nystagmus.
- Light-near dissociation of the pupils-the near reflex is intact but response to light is poor.

Causes: demyelinating disease, vascular disease affecting the dorsal midbrain, enlarged 3^{rd} ventricle.

Cranial nerve V: trigeminal

Applied anatomy

Sensory: facial sensation in 3 branches-ophthalmic (V_1), maxillary (V_2), mandibular (V_3). Distribution shown in Fig. 10.3.

Motor: muscles of mastication.

Nerve originates in the pons, travels to trigeminal ganglion at the petrous temporal bone and splits… V_1 passes through the cavernous sinus with III and exits via the superior orbital fissure; V_2 leaves via the infraorbital foramen (also supplies the palate and nasopharynx); V_3 exits via the foramen ovale with the motor portion.

Examination

Inspection

Inspect the patient's face—wasting of the temporalis will show as hollowing above the zygomatic arch.

Testing motor function

- Ask the patient to clench their teeth and feel both sides for the bulge of the masseter and temporalis.
- Ask the patient to open their mouth wide-the jaw will deviate towards the side of a V lesion.
- Again ask them to open their mouth but provide resistance by holding their jaw closed with one of your hands.

Testing sensory function

- Assess light-touch for each branch and ask the patient to say 'yes' if they can feel it.
  - Choose 3 spots to test on each side to make the examination easy to remember—forehead, cheek, and mid-way along jaw.
- For each branch, compare left to right. Ignore minor differences (it's rather difficult to press with exactly the same force each time!)
- Test pin-prick sensation at the same spots using a sterile pin.
- Temperature sensation is not routinely tested-consider only if abnormalities in light-touch or pin-prick are found. Use specimen tubes or other small containers full of hot or cold water.

Findings

- Wasting of muscles: long-term V palsy, MND, myotonic dystrophy.
- Loss of all sensory modalities: V ganglion lesion (?herpes zoster).
- Loss of light touch only—with loss of sensation on ipsilateral side of the body: contralateral parietal lobe (sensory cortex) lesion.
- Loss of light-touch in V only: lesion at sensory root pons.
- Loss of pin-prick only—along with contralateral side of body: ipsilateral brainstem lesion.
- Loss of sensation in a 'muzzle' distribution (nose, lips, anterior cheeks): damage to the lower part of the spinal sensory nucleus (syringomyelia, demyelination).

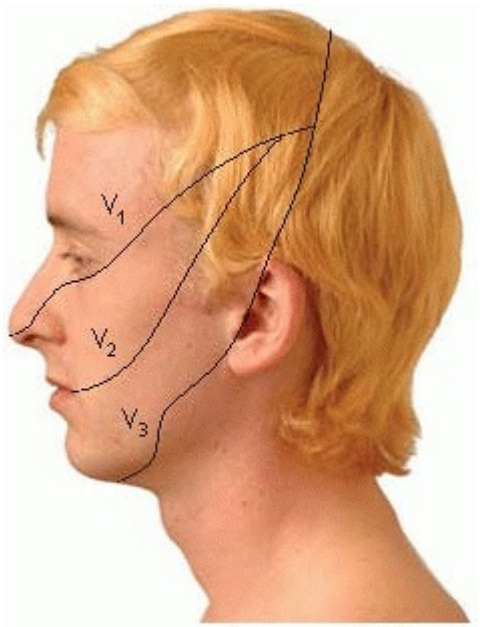


Fig. 10.7 Distribution of the sensory branches of the trigeminal nerve. V_1 = ophthalmic, V_2 = maxillary, V_3 = mandibular. Note that V_1 extends to the vertex and includes the cornea and V_3 does *not* include the angle of the jaw.

Reflexes

Jaw jerk

- Explain to the patient what is about to happen as this could appear rather threatening!
- Ask the patient to let their mouth hang loosely open.
- Place your finger horizontally across their chin and tap your finger with a patella hammer.
- Feel and watch jaw movement.
  - There should be a slight closure of the jaw but this varies widely in normal people. A brisk and definite closure may indicate an UMN lesion above the level of the pons (e.g. pseudobulbar palsy).

Corneal reflex

Afferent = V_1, efferent = VII.

- Ask the patient to look up and away from you.
- Gently touch the cornea with a wisp of cotton wool. Bring this in from the side so it cannot be seen approaching.
- Watch both eyes. A blink is a normal response.
  - No response = ipsilateral V_1 palsy.
  - Lack of blink on one side only = VII palsy.
- ▸ Watch out for contact lenses!-will give reduced sensation. Ask the patient to remove them first.

▸ *Hints*

- Note the sensory distribution! The angle of the jaw is **not** supplied by V_3 but by the great auricular nerve (C2, C3).
- When testing the corneal reflex, touch the **cornea** (overlies the iris), not the conjunctiva (overlies the sclera).

Cranial nerve VII: facial

Applied anatomy

Sensory: external auditory meatus, tympanic membrane, small portion of skin behind ear. Special sensation: taste anterior 2/3 of tongue.

Motor: muscles of facial expression, stapedius.

Autonomic: parasympathetic supply to lacrimal glands.

The nucleus lies in the pons, the nerve leaves at the cerebellopontine angle with VIII. The nerve gives off a branch to the stapedius at the geniculate ganglion whilst the majority of the nerve leaves the skull via the stylomastoid foramen and travels through the parotid gland.

Examination

Muscles of facial expression

Here, you test both left and right at the same time. Some patients have difficultly understanding the instructions—the authors recommend a quick demonstration following each command allowing the patient to mirror you (e.g. 'puff out your cheeks like this…'). This exam can be rather embarrassing—the examiner pulling equally strange faces lightens the mood and aids the patient's co-operation and enthusiasm.

- Look at the patient's face at rest. Look for asymmetry in the nasolabial folds, angles of the mouth and forehead wrinkles.
- Ask the patient to raise their eyebrows ('look up!') and watch the forehead wrinkle.
- Attempt to press their eyebrows down and note any weakness.

- Ask the patient to ‘close your eyes tightly’. Watch, then test against resistance with your finger and thumb. ‘Don't let me pull them apart.’
- Ask the patient to blow out their cheeks. Watch for air escaping on one side.
- Ask the patient to bare their teeth. ‘Show me your teeth!’ Look for asymmetry.
- Ask the patient to purse their lips. ‘Whistle for me!’ Look for asymmetry. The patient will always smile after whistling (see below).

The ‘whistle-smile’ sign

A failure to smile when asked to whistle (whistle-smile negative) is usually due to ‘emotional paresis’ of the facial muscles and is synonymous with Parkinsonism.

External auditory meatus

This should be examined briefly if only VII is examined-can be done as part of VIII if examining all the cranial nerves.

Taste

This is rarely tested outside specialist clinics.

- Each side is tested separately by using cotton buds dipped in the solution of choice applied to each side of the tongue in turn. Be sure to swill the mouth with distilled water between each taste sensation.
- Test: sweet, salty, bitter (quinine), and sour (vinegar).

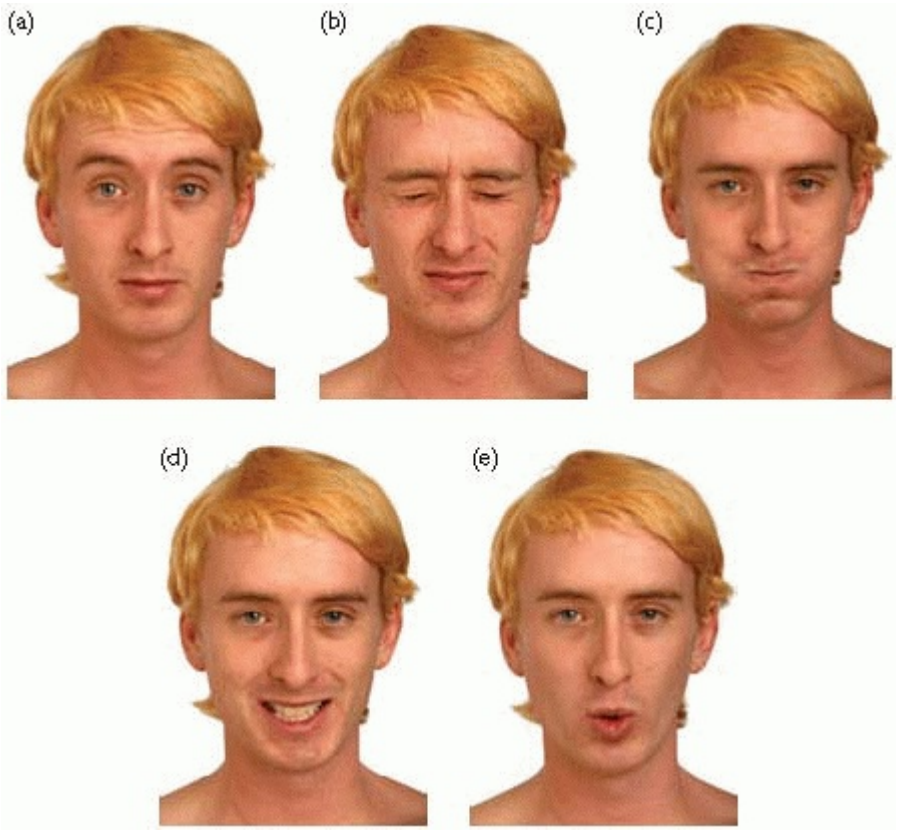


Fig. 10.8 Testing the muscles of facial expression as described opposite. (a) eyebrows; (b) eyelids; (c) puffing out the cheeks; (d) baring teeth; (e) whistle

Findings

- ***Upper motor nerve lesion:*** will cause loss of facial movement on the ipsilateral side but with preservation of forehead wrinkling-both sides of the forehead receive bilateral nervous supply. (Unilateral = CVA etc, Bilateral = pseudobulbar palsy, motor neuron disease.)
- ***Lower motor nerve lesion:*** will cause loss of all movement on the ipsilateral side of the face (unilateral = demyelination, tumours, Bell's

palsy, pontine lesions, cerebellopontine angle lesions; bilateral = sarcoid, GBS, myaesthenia gravis).

- ***Bell's palsy:*** idiopathic unilateral LMN VII paresis.
- ***Ramsay-Hunt syndrome:*** unilateral paresis caused by herpes at the geniculate ganglion (look for herpes rash on the external ear).

▸ *Hints*

- Bell's *phenomenon* is the upward movement of the eyeballs when the eye closes. This occurs in the normal state but can be clearly seen if the eyelids fail to close due to VII palsy.
- VII palsy does *not* cause eyelid ptosis.
- Longstanding VII palsy can cause fibrous contraction of the muscles on the affected side resulting in more pronounced nasolabial fold (the reverse of the expected findings).
- Bilateral VII palsy will cause a sagging, expressionless face and is often missed.

Cranial nerve VIII: vestibulocochlear

Applied anatomy

Sensory: hearing (cochlear), balance/equilibrium (vestibular).

Motor: none

The 8th nerve comprises 2 parts. The cochlear branch originates in the organ of Corti in the ear, passes through the internal auditory meatus to its nucleus in the pons. Fibres pass to the superior gyrus of the temporal lobes.

The vestibular branch arises in the utricle and semicircular canals, joins the auditory fibres in the facial canal, enters the brainstem at the cerebellopontine angle and ends in the pons and cerebellum.

Examination

Enquire first about symptoms—hearing loss/changes or balance problems. Peripheral vestibular lesions cause ataxia during paroxysms of vertigo but not at other times.

- Begin by inspecting each ear as described in  Chapter 6.

Hearing

Test each ear separately. Cover one by pressing on the tragus or create white-noise by rubbing your fingers together at the external auditory meatus.

Simple test of hearing

- Whisper a number into one ear and ask the patient to repeat it.
- Repeat with the other ear.
- Be careful to whisper at the same volume in each ear (the end of expiration is best) and at the same distance (about 60cm).

Rinne's test

- Tap a 512Hz* tuning fork and hold adjacent to the ear (air conduction, Fig. 10.9a).
- Then apply the base of the tuning fork to the mastoid process (bone conduction)—see Fig. 10.9b.
- Ask the patient which position sounds louder.
  - (Normal = air conduction > bone conduction = ‘Rinne's positive’)
  - In neural (or perceptive) deafness, Rinne's test will remain positive.
  - In conductive deafness, the findings are reversed (bone >air).

Weber's test

- Tap a 512Hz tuning fork and hold the base against the vertex or forehead at the midline (see Fig. 10.9c).
- Ask the patient if it sounds louder on one side.
  - In neural deafness, the tone is heard better in the intact ear.
  - In conductive deafness, the tone is heard better in the affected ear.

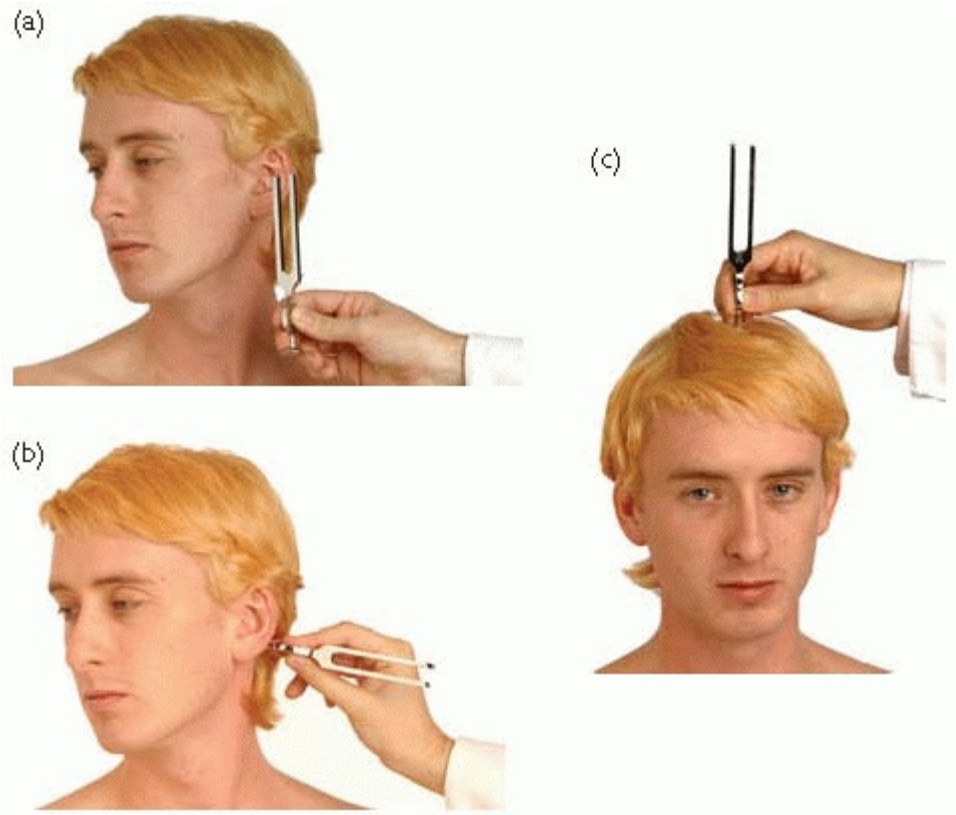


Fig. 10.9 (a) Testing air conduction. (b) Testing bone conduction. (c) Position of the tuning fork for Weber's Test.

Vestibular function

Turning test

- Ask the patient to stand facing you, arms outstretched.
- Ask them to march on the spot, then close their eyes (continue marching).
- Watch!
  - The patient will gradually turn toward the side of the lesionsometimes will turn right round 180°.

Hallpike's manoeuvre

A test for benign positional vertigo (BPV). Do **not** test those with known neck problems or possible posterior circulation impairment.

- Warn the patient about what is to happen.
- Sit the patient facing away from the edge of the bed such that when they lie back their head will not be supported (over the edge).
- Turn their head to one side and ask them to look in that direction.
- Lie them back quickly—supporting their head so that it lies about 30° below the horizontal.

- Watch for nystagmus (affected ear will be lowermost).
- Repeat with the head turned in the other direction
  - No nystagmus = normal.
  - Nystagmus, with a slight delay (~10 secs) and fatigable (can't be repeated successfully for ~10-15 minutes) = BPV.
  - Nystagmus, no delay and no fatiguing = central vestibular syndrome.

Cranial nerves IX and X

The 9th (*glossopharyngeal*) and 10th (*vagus*) nerves are considered together as they have similar functions and work together to control pharynx, larynx and swallow.

Applied anatomy: IX

Sensory: pharynx, middle ear. Special sensation: taste on posterior 1/3 of tongue.

Motor: stylopharyngeous.

Autonomic: parotid gland.

Originates in the medulla, passes through the jugular foramen.

Applied anatomy: X

Sensory: tympanic membrane, external auditory canal, and external ear.

Also proprioception from thorax and abdomen.

Motor: palate, pharynx, and larynx.

Autonomic: carotid baroreceptors.

Originates in medulla and pons, leaves the skull via jugular foramen.

Examination

Pharynx

- Ask the patient to open their mouth and inspect the uvula (use a tongue depressor if necessary). Is it central or deviated to one side? If so, which side?
- Ask the patient to say ‘aah’. Watch the uvula. It should move upwards centrally. Does it deviate to one side?

Gag reflex

This is unpleasant for the patient and should only be tested if a IX or X nerve lesion is suspected (afferent signal = IX, efferent = X).

- With the patient's mouth open wide, gently touch the posterior pharyngeal wall on one side with a tongue depressor or other sterile stick.
- Watch the uvula (it should lift up).
- Repeat on the opposite side.
- Ask the patient if they felt the 2 touches-and was there any difference in sensation?

Larynx

- Ask the patient to cough—normal character? Gradual onset/sudden?
- Listen to the patient's speech—note volume, quality and whether it appears to fatigue (quieter as time goes on).
- Test swallow:
  - At each stage, watch the swallow action—2 phases or one smooth movement? Delay between fluid leaving mouth (oral phase) and pharynx/larynx reacting (pharyngeal phase)? Any coughing/choking? Any ‘wet’ voice?
  - ▸ Terminate the test at the first sign of the patient aspirating.

- Offer the patient a teaspoon of water to swallow. Repeat x 3.
- Offer the patient a sip of water. Repeat x 3.
- Offer the patient the glass for a mouthful of water. Repeat x 3.

Findings

Uvula

- Moves to one side = X lesion on the *opposite* side.
- No movement = muscle paresis.
- Moves with 'aah' but not gag and ↓ pharyngeal sensation = IX palsy.

Cough

- Gradual onset of a deliberate cough = vocal cord palsy.
- 'Wet', bubbly voice and cough (before the swallow test) = pharyngeal and vocal cord palsy (X palsy).
- Poor swallow and aspiration = combined IX and X or lone X lesion.

Cranial nerve XI: accessory

Applied anatomy

Sensory: none.

Motor: sternocleidomastoids and upper part of trapezii.

The accessory nerve is composed of 'cranial' and 'spinal' parts.

The cranial accessory nerve arises from the nucleus ambiguus in the medulla. The spinal accessory nerve from the lateral part of the spinal cord down to C5 as a series of rootlets. These join together and ascend adjacent to the spinal cord, passing through the foramen magnum to join with the cranial portion of the accessory nerve. It leaves the skull via the jugular foramen.

The cranial portion joins with the vagus nerve (X).

The spinal portion innervates the sternocleidomastoids and the upper fibres of the trapezii.

▸ Note that each cerebral hemisphere controls the *ipsilateral* sternocleidomastoid and the contralateral trapezius.

Examination

The cranial portion of the accessory nerve cannot be tested separately.

- Inspect the sternocleidomastoids. Look for wasting, fasciculations, hypertrophy, and any abnormal head position.
- Ask the patient to shrug their shoulders and observe.
- Ask the patient to shrug again, using your hands on their shoulders to provide resistance.
- Ask the patient to turn their head to each side, first without and then with resistance (use your hand on their cheek).

Findings

Isolated accessory nerve lesions are very rare. XI lesions usually present as part of a wider weakness or neurological syndrome.

- *Bilateral weakness:* with wasting caused by muscular problems or motor neuron disease.
- *Unilateral weakness (trapezius and sternomastoid same side):* suggests a peripheral neurological lesion.
- *Unilateral weakness (trapezius and sternomastoid of opposite sides):* usually with hemiplegia suggests an UMN lesion ipsilateral to the weak sternomastoid.

▸ *Hints*

- Remember that the action of the sternocleidomastoid is to turn the head to the *opposite* side (e.g. poor head turning to the *left* indicates a weak *right* sternocleidomastoid).
- When providing resistance to head turning, be sure to press against the patient's cheek (see Fig. 10.10). Lateral pressure to the jaw can cause pain and injury, particularly in the elderly and frail.

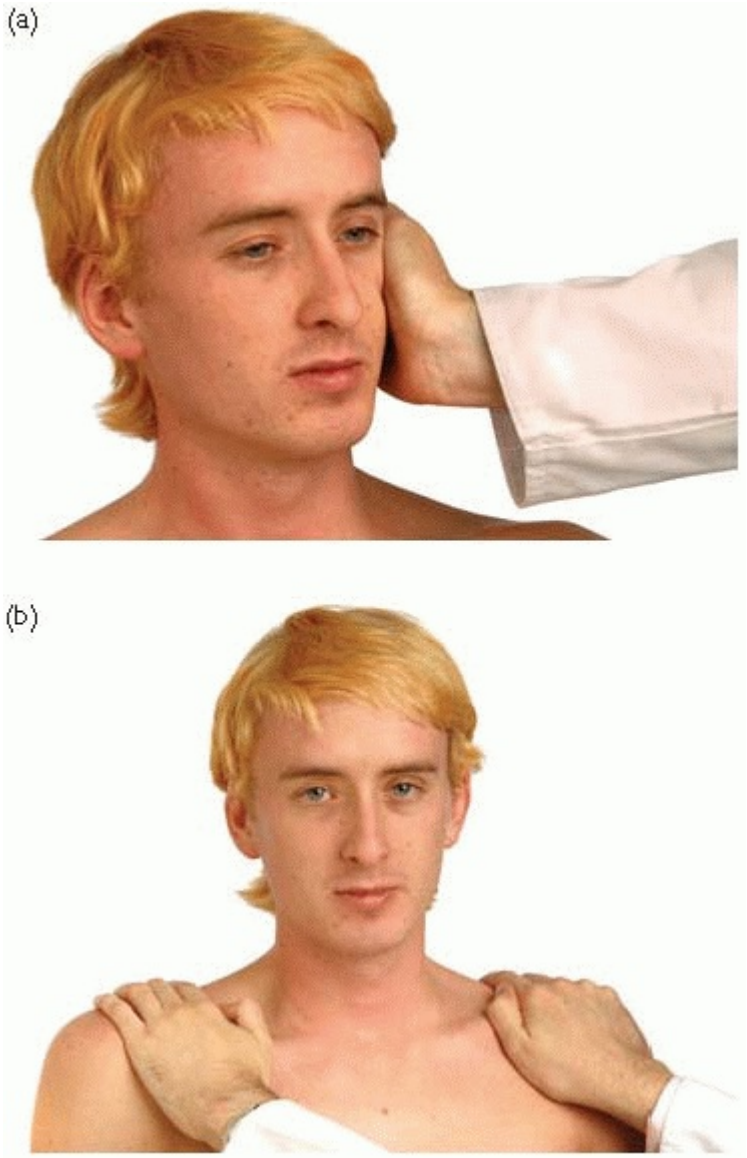


Fig. 10.10 (a) Using resistance against lateral head-turning. Be careful not to apply pressure to the patient's jaw. (b) Testing the trapezius against resistance.

Cranial nerve XII: hypoglossal

Applied anatomy

Sensory: none.

Motor: muscles of the tongue.

Nucleus lies on the floor of IV ventricle. Fibres pass ventrally, leaving the brainstem lateral to the pyramidal tracts. Leaves the skull via the hypoglossal foramen.

Examination

- Ask the patient to open wide and inspect the tongue on the floor of the mouth. Look for size and evidence of fasciculation.
- Ask the patient to protrude the tongue. Look for deviation or abnormal movements.
- Ask the patient to move the tongue in and out repeatedly, then side-to-side.
- To test for subtle weakness, place your finger on the patient's cheek and ask them to push against it from the inside using their tongue.

Findings

- A LMN neuron lesion will cause fasciculations on the affected side and a deviation towards the affected side on protrusion. There will also be a weakness on pressing the tongue away from the affected side.
- A unilateral upper motor neuron lesion will rarely cause any clinically obvious signs.
- A bilateral upper motor neuron lesion will give a small, globally weak tongue with reduced movements.
- A bilateral LMN lesion (e.g. motor neuron disease) will also produce a small, weak tongue.
- A rapid 'in and out' movement on protrusion (trombone tremor) can be caused by cerebellar disease, extra-pyramidal syndromes and essential tremor.

▸ *Hint*

Rippling movements may be seen if the tongue is held protruded for long periods. This is normal and should not be mistaken for fasciculations.

Motor: applied anatomy

The motor system is complex and a detailed description is beyond the scope of this book. What follows is a brief overview.

Cortex

The primary motor area is the precentral gyrus of the cerebrum and it is here, along with adjacent cerebral areas, that initiation of voluntary movement occurs. Muscle groups are represented by areas of the cortex from medial to lateral as shown opposite. The size of the area dedicated to muscle corresponds with the precision of movement (= the number of motor units) that are involved.

Pyramidal (direct) pathways

These are concerned with precise, voluntary movements of the face, vocal cords, hands and feet.

The simplest pathways consist of 2 neurons. The first 'upper motor neuron' (UMN) originates in the cerebral cortex, passes down through the internal capsule, brainstem and spinal cord where it synapses with a 'lower motor neuron' (LMN). This, in turn, leaves the cord to synapse with the skeletal muscle fibres.

There are 3 pyramidal tracts:

- ***Lateral corticospinal:*** control of precise movement in the hands and feet and represents 90% of the UMN axons. These crossover (decussate) in the medulla oblongata before continuing to descend so that nerves from the right side of the brain control muscles on the left of the body and vice versa.
- ***Anterior corticospinal:*** control of the neck and trunk and holds 10% of the UMN axons. These do not cross in the medulla but descend in the anterior white columns of the spinal cord. They decussate at several spinal levels and exit at the cervical and upper thoracic segments.
- ***Corticobulbar:*** voluntary muscles of the eyes, face, tongue, neck, and speech. Terminate at nuclei in the pons and medulla, some crossed, others not. Control of cranial nerves III, IV, V, VI, VII, IX, X, XI, and XII.

Extrapyramidal (indirect) pathways

All the other descending pathways. These are complex circuits involving the cortex, limbic system, basal ganglia, cerebellum, and cranial nerve nuclei. There are 5 major tracts controlling precise movements of the hands and feet, movement of the head and eyes in response to visual stimuli, muscle tone, and truncal stability and balance.

Basal ganglia/nuclei: complex circuits concerned with the production of automatic movement, planning movement sequences. Also appear to inhibit intrinsically excitable circuits.

Cerebellum

Involved in learning and performing skilled, automatic movements (e.g. running, playing the piano), posture, and balance. Monitors

intention, receives signals as to actual movements, compares the difference, and makes corrective adjustments.

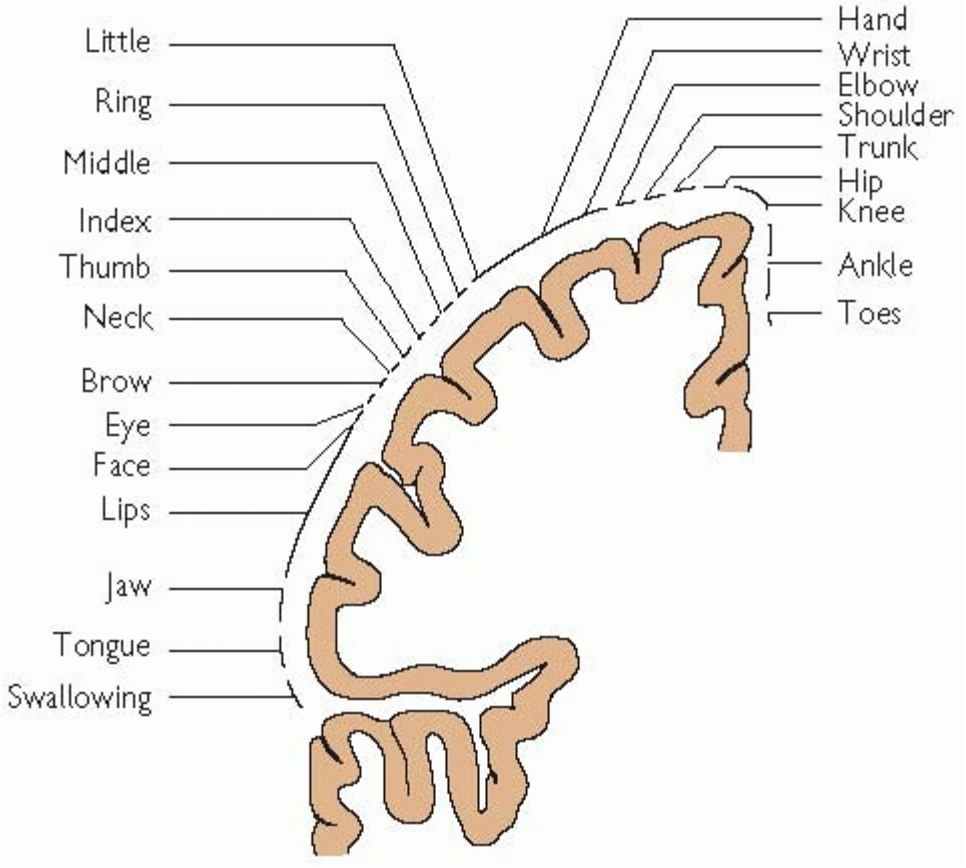


Fig. 10.11 Coronal section through the motor cortex showing the representation of different muscle groups. Note the larger areas given to those muscles performing precise movements—hands, face, lips.

Box 10.3 A word about functional weakness

Large parts of the neurological examination rely on the co-operation of the patient. Occasionally, patients give the appearance of neurological disability which does not exist-for any number of psychiatric or psychosocial reasons. The examination here is very difficult even for very experienced practitioners. Consider a 'functional' component to the problem if you see:

- Abnormal distribution of weakness.
- Normal reflexes and tone despite weakness.
- Movements are variable and power erratic.
- Variation is seen on repeat testing.

Careful here! Don't jump to conclusions. Do not assume symptoms are functional if they are unusual. All patients should be given the benefit of the doubt. 'Functional' weakness is a diagnosis largely of exclusion.

Motor: inspection and tone

Inspection

As for any other system, the examination begins when you first set eyes on the patient and continues through the history taking.

- Any walking aids or abnormal gait (see p.350)?
- Shake hands—abnormalities of movement? Strength? Relaxation?
- Any abnormal movements when sitting?
- Any obvious weaknesses (e.g. hemiplegia)?

- Does the patient have good sitting balance?

Inspection can then be formalized at the examination stage of the encounter. The patient should be seated or lying comfortably with as much of their body exposed as possible. Look at all muscle groups for:

- Abnormal positioning—due to weakness or contractures.
- Wasting.
- Fasciculation (irregular contractions of small areas of muscle).

Make a point of inspecting the shoulder girdle, small muscles of the hand, quadriceps, anterior compartment of lower leg and ankle.

Look at the foot for contractures or abnormalities of shape.

Tone

The aim is to test *resting* tone in the limbs. This takes practise and the feel of normal, ↓ or ↑ tone can only be taught through experience.

The assessment can be difficult as it relies on the patient being relaxed and telling the patient to relax usually has the opposite effect! They can be distracted by a counting task or told to relax the limb 'as if you're asleep'. However, distracting the patient with light conversation is a generally successful ploy. You should also repeat the following manoeuvres at different speeds and intervals to catch the patient at an unguarded moment.

Arms

- Take the patient's hand in yours (as if shaking it) and hold their elbow with your other hand (see Fig. 10.12a). From this position, you can:
  - Pronate and supinate the patient's forearm.
  - Roll the patient's wrist through 360°.
  - Flex and extend the patient's elbow.

Legs

- *Hip:* with the patient lying flat, legs straight, hold onto the patient's knee and roll it from side to side (see Fig. 10.12b).
- *Knee:* with the patient in the same position, put your hand behind the patient's knee and raise it quickly (Fig. 10.12c). Watch the heel- it should lift from the bed/couch slightly if tone is normal.
- *Ankle:* holding the foot and the lower leg, flex and dorsiflex the ankle (Fig. 10.12d).

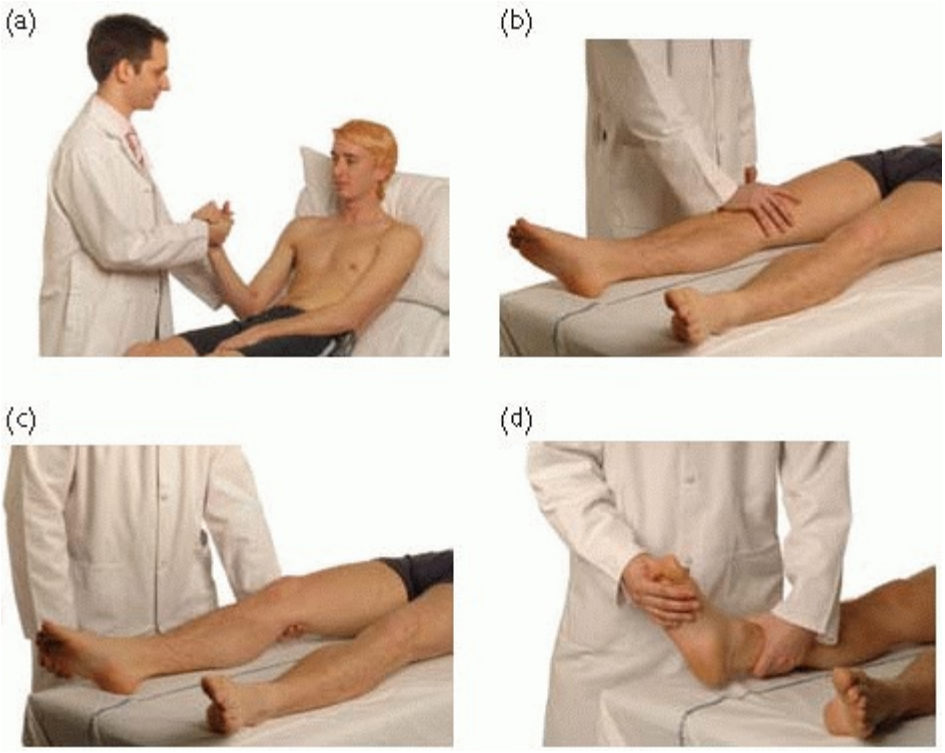


Fig. 10.12 Testing tone. (a) Testing the upper limb. (b) Testing tone at the hip. (c) Testing tone at the knee. (d) Testing tone at the ankle.

Findings

- ***Normal tone:*** slight resistance in movement (feel through experience!)
- ↓ ***tone:*** 'flaccid' due to LMN or cerebellar lesions or myopathies.
- ↑ ***tone:***
  - *Spasticity (clasp-knife rigidity):* the limb appears stiff. With ↑ pressure, there is a sudden 'give' and the limb moves. Seen in UMN lesions.
  - *Rigidity (lead-pipe):* the limb is equally stiff through all movements.
  - *Rigidity (cogwheel):* an extrapyramidal sign, caused by a tremor superimposed on a rigid limb. The limb moves in a stop-go halting fashion.
  - *Gegenhalten:* (paratonia) seen in bilateral frontal lobe damage and catatonic states. Tone ↑ with ↑ pressure from the examiner—the patient *appears* to be resisting movement.
  - *Myotonia:* a slow relaxation after action—when asked to make a fist, the patient is unable the release it quickly and will be slow to let go of a hand-shake (e.g. myotonic dystrophy).
  - *Dystonia:* the limb or head has an abnormal posture that looks rather uncomfortable.

Motor: upper limb power

▸ As for the muscles of the face, the examiner should demonstrate each movement, mirroring the patient. (see Fig. 10.13).

This also allows each action that the patient makes to be opposed by the same (or similar) muscle groups in the examiner—test their fingers against your fingers and so on. Each muscle group should be graded from 0 to 5 according to the MRC system shown opposite.

Examining the upper limbs also allows for both sides to be tested at once, allowing a direct comparison between left and right. (see Fig.10.13).

Be careful not to hurt frail and elderly patients or those with OA, RA, and other rheumatological disease!

Shoulder

- ***Abduction:*** (C5). Ask the patient to abduct their arms with elbows bent. 'Arms up like a chicken!' Ask them to hold still as you attempt

to push their arms down.

- ***Adduction:*** (C6, C7). The patient should hold their arms tightly to their sides with elbows bent. You attempt to push their arms out.

Elbow

- ***Flexion:*** (C5, C6). the patient should hold their elbows bent and supinated in front of them. Hold the patient at the elbow and wrist and attempt to extend their arm. 'Don't let me straighten your arm!'
- ***Extension:*** (C7). Patient holds position above as you resist extension at the elbow by pushing on their distal forearm/wrist. 'Push me away!'

Wrist

- ***Flexion:*** (C6, C7). With arms supinated, the patient should flex the wrist and hold as you attempt to extend it by pulling from your own wrists.
- ***Extension:*** (C6, C7). The opposite manoeuvre to that above. The patient holds their hand out straight and resists your attempts to bend it.

Fingers

- ***Flexion:*** (C8). Ask the patient to squeeze your fingers *or* (better) ask the patient to grip your fingers palm-to-palm (see Fig. 10.13c) and resist your attempts to pull their hand open.
- ***Extension:*** (C7, C8). Ask the patient to hold their fingers out straight—you support their wrist with one hand and attempt to push their fingers down with the side of your hand over their first interphalangeal joints.
- ***Abduction:*** (T1). Ask the patient to splay their fingers out and resist your attempts to push them together.
- ***Adduction:*** (T1). Holding the patient's middle, ring, and little finger with one hand and their index finger with the other, ask the patient to pull their fingers together *or* place a piece of paper between their outstretched fingers and ask them to resist your attempts to pull it away.

Pronator drift

A useful test of subtle weakness. The patient is asked to hold their arms outstretched in front, palms upwards and eyes closed. If one side is weak, the arm will pronate and slowly drift downwards.

Box 10.4 Medical Research Council (MRC) power classification

- 5 = Normal power.
- 4 = Movement against resistance but not normal.
- 3 = Movement against gravity, but not against resistance.
- 2 = Movement with gravity eliminated (e.g. can move leg side-to-side on bed but not lift it).
- 1 = Contractions but no movement seen.
- 0 = No movement.

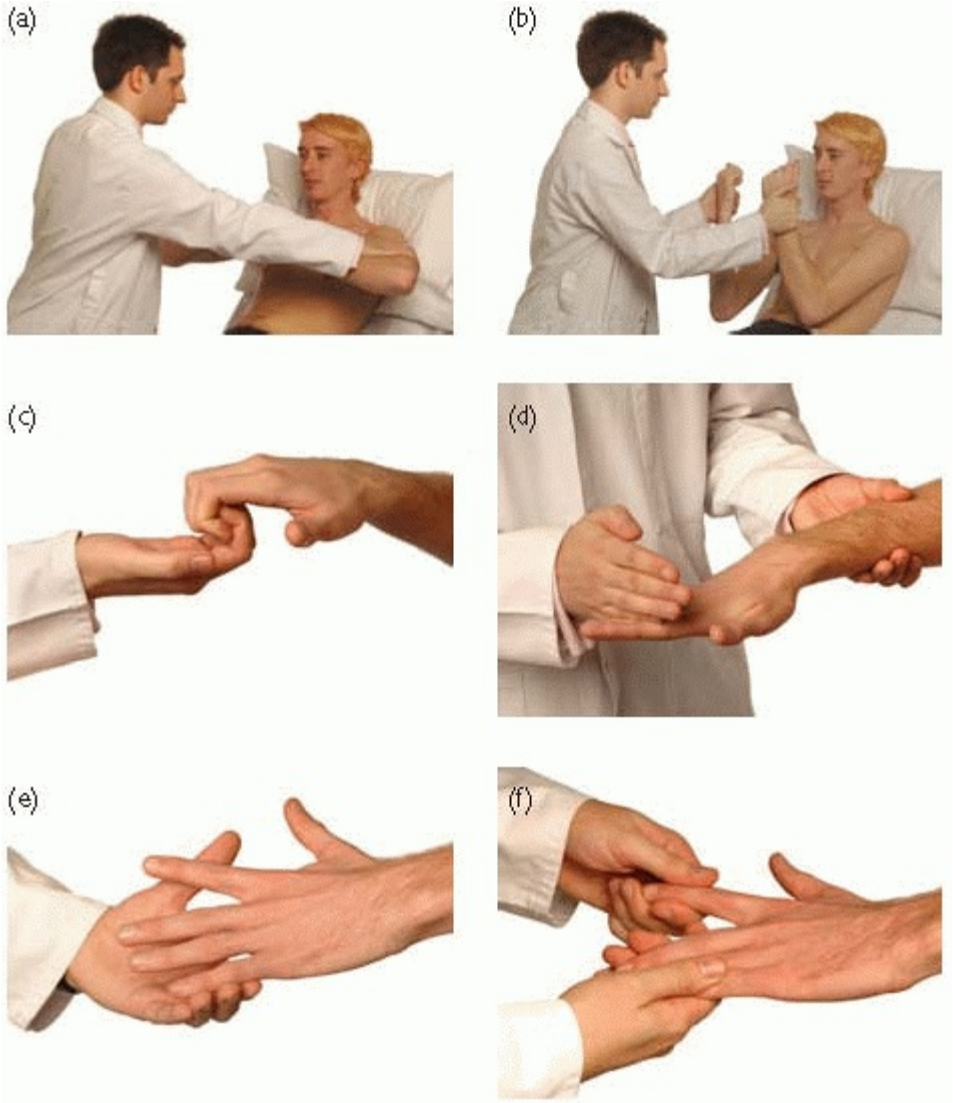


Fig. 10.13 Testing power in the upper limbs. (a) Shoulder movements. (b) Elbow movements. (c) Finger flexion. (d) Finger extension. (e) Finger abduction. (f) Finger adduction.

Motor: lower limb power

The patient should be seated on a couch or bed with their legs outstretched in front of them. The limbs should be exposed as much as possible so that contractions of the muscles can be *seen*.

Again, power is tested for each muscle group on one side then the other, comparing left with right and scored according to the MRC scale.

Hip

- ***Flexion:*** (L1, L2, L3). With the lower limbs lying on the bed/couch, the patient is asked to raise each leg, keeping the knee straight. The examiner can oppose the movement by pushing down on the thigh just above the knee. 'Stop me from pushing down!'
- ***Extension:*** (L5, S1). Ask the patient to keep their leg pressed against the bed as you attempt to lift it—either with a hand beneath the calf or the ankle. 'Stop me lifting your leg up!'
- ***Abduction:*** (L4, L5, S1). Ask the patient to move their leg out to the side as you oppose the movement with a hand on the lateral thigh. 'Stop me pushing your legs together!'
- ***Adduction:*** (L2, L3, L4). With the legs central, put your hand on the medial thigh and attempt to pull the leg out to the side against resistance. 'Don't let me pull your legs apart!'

Knee

- ***Flexion:*** (L5, S1). Take hold of the patient's knee with one hand and their ankle with the other and flex the leg to about 60°. (The patient may think you want them to resist this so often a quick instruction 'bend at the knee' is required.) Ask the patient to bend

their leg further (‘stop me straightening your leg out’) and oppose the movement at their ankle.

- ***Extension:*** (L3, L4). With the patient's leg in the position above, ask the patient to extend their leg (‘push me away’, ‘straighten your leg out’) as you oppose it. Alternatively, attempt to bend the patient's leg from a straightened starting position.

Ankle

- ***Plantar flexion:*** (S1, S2). With the patient's leg out straight and ankle relaxed, put your hand on the ball of the foot and ask the patient to push you away. ‘Push down and stop me pushing back!’
- ***Dorsiflexion:*** (L4, L5). From the starting position above, hold the patient's foot just above the toes and ask them to pull their foot backwards. Patients often attempt to move their entire leg here so ‘cock your foot back and stop me pushing your foot down’ with an accompanying hand gesture helps.

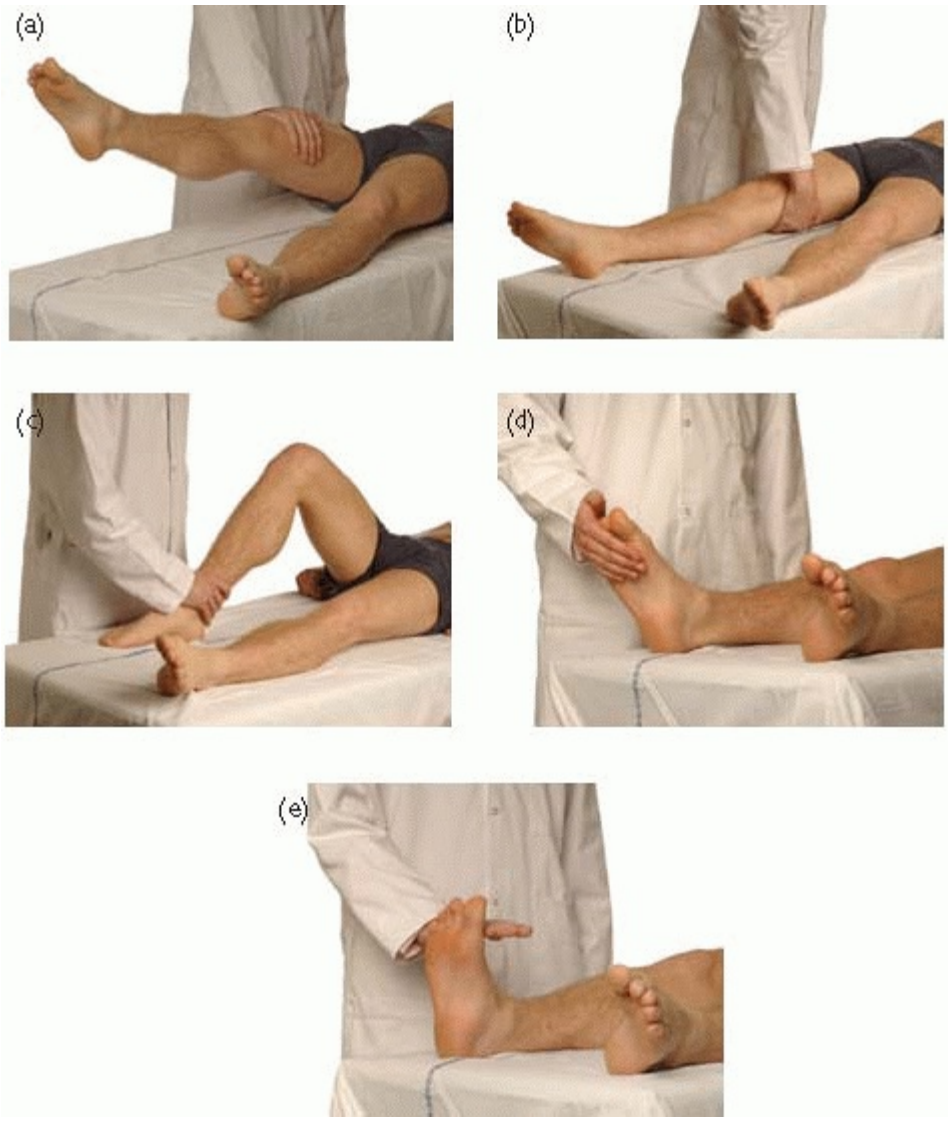


Fig. 10.14 Testing power in the lower limb against resistance. (a) Flexing the hip. (b) Extending the hip. (c) Flexing/extending the knee. (d) Plantar flexion at the ankle. (e) Dorsiflexion at the ankle.

Tendon reflexes

Theory

The sudden stretch of a muscle is detected by the muscle spindle which initiates a simple 2 neuron reflex arc, causing that muscle to contract.

Tendons are struck with a tendon hammer (causing a sudden stretch of the muscle) and the resultant contraction observed. In LMN

lesions or myopathies, the reflex is ↓ or absent, but ↑ or 'brisk' in UMN lesions.

Technique

The tendons are tapped with a tendon hammer. For each reflex, test the right, then left and compare. The hammer should be held at the far end of the handle and swung in a loose movement from the wrist. The patient should be relaxed (see 📖 p.324).

Examination

- ***Biceps:*** (C5, C6). With the patient seated, lie their arms across their abdomen. Place your thumb across the biceps tendon and strike it with the tendon hammer as above. Watch the biceps for contraction.
- ***Supinator:*** (C5, C6). The muscle tested is actually the brachioradialis. With the patient's arms lying loosely across their abdomen, put your fingers on the radial tuberosity and tap with the hammer. The arm will flex at the elbow. If brisk, the fingers may also flex.
- ***Triceps:*** (C7). Taking hold of the patient's wrist, flex their arm to ~90°. Tap the triceps tendon about 5cm superior to the olecranon process of the ulna. Watch the triceps.
- ***Fingers:*** (C8). This is only present if tone is pathologically t. With your palm up and the patient's arm pronated, lie their fingers on yours. Strike the back of your fingers. The patient's fingers will flex.
- ***Knee:*** (L3, L4). With the patient's leg extended, use one hand behind their knee to lift their leg to ~60°. Tap the patella tendon and watch the quadriceps. If brisk, proceed to testing for clonus here:
  - *Knee clonus:* with the patient's leg extended, place your thumb and index finger over the superior edge of the patella. Create a sudden downward (toward the feet) movement, and *hold*. Watch the quadriceps. *Any* beat of clonus here is abnormal.
- ***Ankle:*** *(S1, S2).* With the hip flexed and externally rotated and the knee flexed to ~90°, hold the foot and tap the Achilles tendon. Watch the calf muscles for contraction/ankle flexion.
  - Alternatively, with the leg extended and relaxed, place your hand on the ball of the foot and strike your hand with the hammer.

Reinforcement

If the reflex is absent, it can sometimes be elicited by asking the patient to perform a 'reinforcing' action which acts to increase the activity of neurons in the spinal cord. This effect is short-lived, however, so you should aim to test the reflex in the first 10 seconds of the reinforcement.

- For upper limb reflexes, ask the patient to clench their teeth.
- For lower limb reflexes, ask the patient to lock their fingers together, pulling in opposite directions.

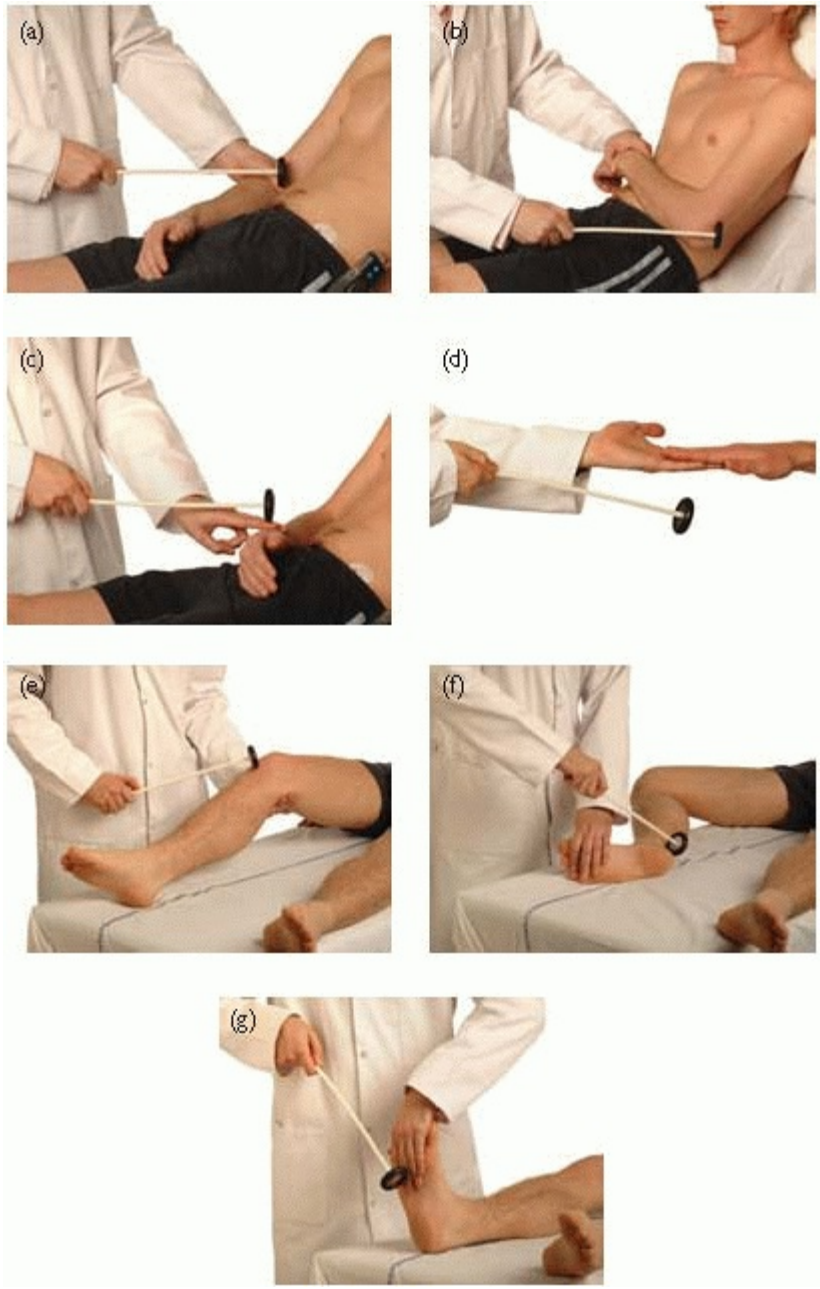


Fig. 10.15 Testing tendon reflexes. (a) Biceps. (b) Triceps. (c) Supinator. (d) Fingers. (e) Knee. (f) Ankle. (g) Alternative method for ankle.

Box 10.5 Recording tendon reflexes:
These are usually recorded as a list—or often by applying the numbers below to the appropriate area of a stick-man sketch.

- 0 = absent
- ± = present only with reinforcement
- 1+ = ↓/less than normal
- 2+ = normal
- 3+ = brisk/more than normal.

Other reflexes

In normal practice, the plantar response is the only one of the following routinely tested.

Abdominal reflex

This test relies on observing the abdominal muscles—is, therefore, less easy in those with a covering of fat. It is also less obvious in

children, the elderly, multiparous patients, or those who have had abdominal surgery.

- The patient should be lying on their back, relaxed, abdomen exposed.
- Using an orange stick or similar, stroke each of the 4 segments of the abdomen, in a brief movement towards the umbilicus.
- As each segment is stroked, the abdominal muscles will reflexly contract.
- Summarize the findings diagrammatically using a simple 2×2 grid and indicating the presence or absence of a response by marking marking '+' and '-' respectively. ('±' for an intermediate response).

The upper segments are supplied by T8-T9, the lower by T10-T11.

Cremasteric reflex

(L1, L2.) Due to its nature, this reflex is very rarely tested and requires a full explanation and consent from the patient first.

- With the male patient standing and naked from the waist down, you should lightly stroke the upper aspect of their inner thigh.
- The ipsilateral cremaster muscle contracts and the testicle will briefly rise.

Plantar response

(L5, S1, S2.) This is sometimes, inappropriately, called the Babinski reflex

- The patient should be lying comfortably, legs outstretched.
- Warn the patient that you are about to touch the sole of their foot (this may prevent a startled 'withdrawal' response).
- Stroke the patient's sole—with an orange stick or similar disposable item (many people use their finger nail but this has obvious implications for sterility!).
- You should stroke from the heel, up the lateral aspect of the sole to the base of the 5^{th} toe. If there is no response, the stroke can be continued along the ball of the foot to the base of the big toe.
- Watch the big toe for its *initial* movement.
  - Normal response is plantar flexion of the big toe.
  - Upper motor nerve lesions will cause the big toe to dorsiflex. This is 'the Babinski response'.
- Document your findings using arrows:
  - ↓ for plantar flexion.
  - ↑ for dorsiflexion.
  - - for an absent response.
- If the leg is withdrawn and the heel moves in a 'ticklish' reaction, this is called a 'withdrawal' response and the test should be repeated.

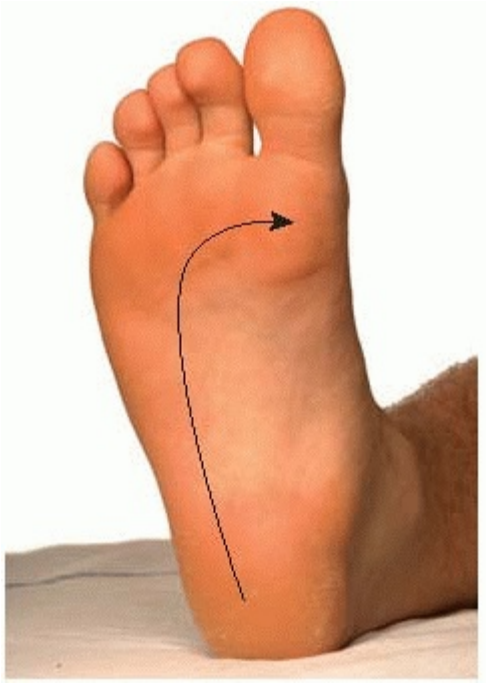


Fig. 10.16 Testing the plantar response. The arrow shows the direction that the stroke should take.

Ankle clonus

A rhythmical contraction of a muscle when suddenly stretched—a sign of hyperreflexia due to UMN lesion. With the patient lying on the bed, knee straight and thigh slightly externally rotated, suddenly dorsiflex the foot. More than 3 beats of clonus—as long as the foot is held dorsiflexed—is abnormal.

Primitive reflexes

These are reflexes seen in the newborn—but may still be present in a few normal adults. They return somewhat in the elderly but are seen mainly in frontal lobe disease and encephalopathy.

The primitive reflexes are not routinely tested unless the examiner is looking specifically for frontal lobe signs or Parkinson's disease.

Glabellar tap

- Using your index finger, repeatedly tap (gently) the patient's forehead between the eyebrows.
- If normal, the patient will blink only with the first 3 or 4 taps.

Palmo-mental reflex

- You should stroke the patient's palm, using sharp firm pressure from the radial side to the ulnar.
- Watch the patient's chin.
- If the reflex is present, there will be a contraction of the ipsilateral mentalis seen in the neck and chin.

Grasp reflex

- Gently stroke your fingers over the patient's palm in a radial-ulnar direction, telling the patient *not* to grip your hand.
- If present, the patient will involuntarily grasp your hand and seemingly refuse to let go.

Snout (or pout) reflex

- With the patient's eyes closed, gently tap their lips with your fingers or (very cautiously) with a patellar hammer.

- An involuntary puckering of the lips is a positive reflex.

Suckling reflex

With the patient's eyes closed, gentle stimulation at the corner of their mouth will result in a suckling action at the mouth. The patient's head may also turn towards the stimulus.

Sensory: applied anatomy

The sensory system, like the rest of the nervous system, is vastly complex. The following is a simplified explanation which should provide enough background to make sense of the examination technique and findings.

Spinal roots and dermatomes

A spinal nerve arises at each spinal level, containing sensory and motor neurons serving a specific segment of the body. The area of skin supplied by the sensory neurons corresponding to each spinal level can be mapped out—each segment is called a dermatome. See Figs. 10.18 and 10.19 on the following pages.

There is considerable overlap such that loss of sensation in just one dermatome is usually not testable (and textbooks show a marked variation in dermatome maps!). Medical students should strive to become familiar with the dermatomal distribution at an early stage.

Somatic sensory pathways

There are 2 main spinal pathways for sensory impulses. The clinical importance of these can be seen in spinal cord damage and is summarised on p.356.

Posterior columns

These convey light touch, proprioception, and vibration sense—as well as stereognosis (the ability to recognize an object by touch), weight discrimination, and kinaesthesia (the perception of movement).

Nerves from receptors extend up the *ipsilateral* side of the spinal cord to the medulla, their axons forming the 'posterior columns' (fasciculus gracilis and fasciculus cuneatus). The second order neurons decussate (cross over) at the medulla and travel in the medial lemniscus to the thalamus. From there, the impulse is conveyed to the sensory cortex.

Spinothalamic tract

This carries pain and temperature sensation. From a clinical point of view, the important difference here is that the first order neurons synapse in the posterior grey horn on joining the spinal cord. The second order neurons then cross over and ascend the *contralateral* side of the cord in the spinothalamic tract to the thalamus.

The sensory cortex

This is located at the postcentral gyrus, just posterior to the motor cortex. Much like the motor strip, the areas receiving stimuli from various parts of the body can be mapped out (see Fig. 10.17). A lesion affecting one area will cause sensory loss in the corresponding body area on the *contralateral* side (see sensory pathways above).

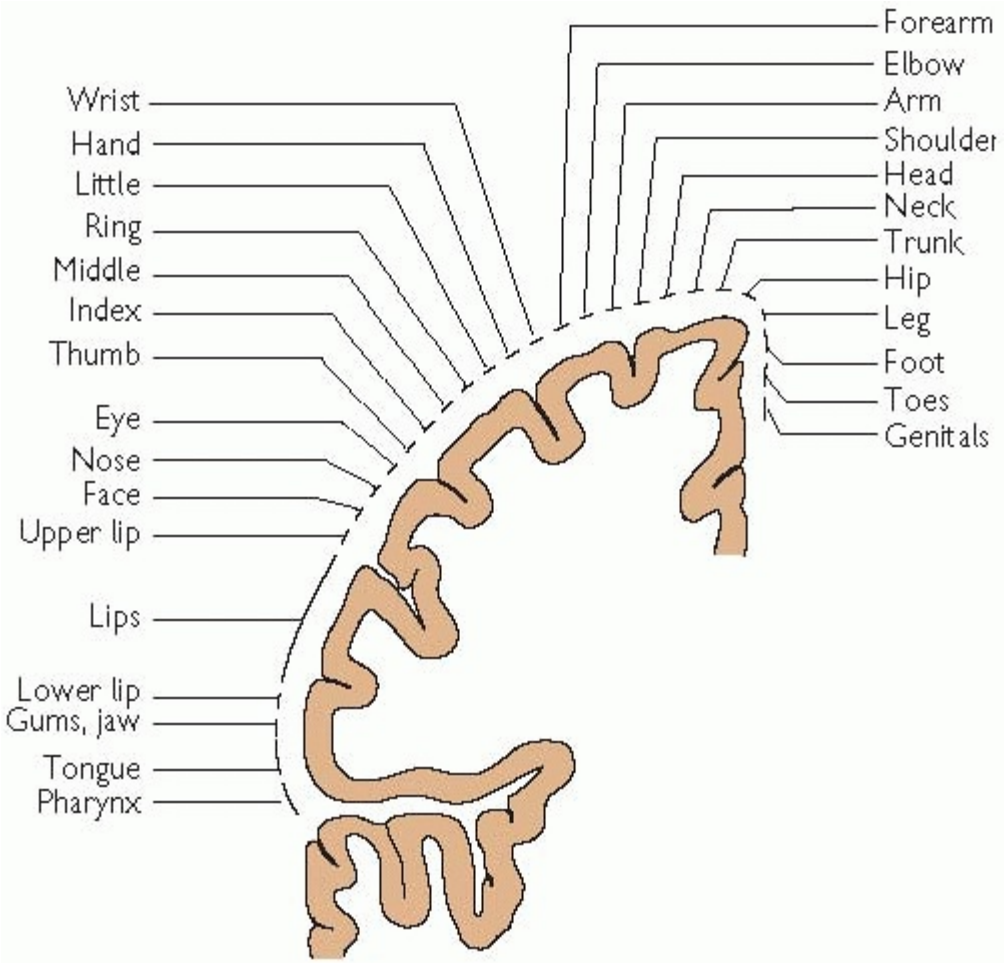


Fig. 10.17 The sensory cortex map showing areas corresponding to the different parts of the body. Note the large areas given over to the fingers and lips.

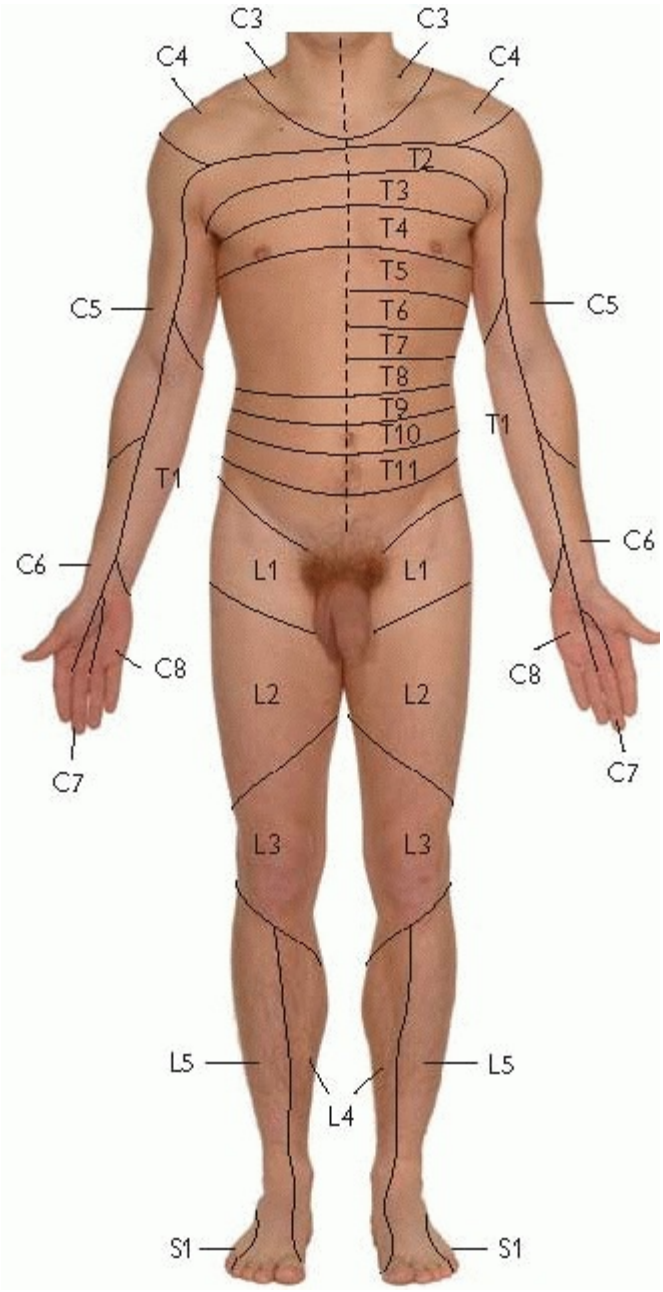


Fig. 10.18 The dermatomes (anterior view). Students would do well to be familiar with these diagrams, particularly the limbs.—note important landmarks to aid recall. (C7 covers the middle finger, T4 lies at the level of the nipples, T10 over the umbilicus).

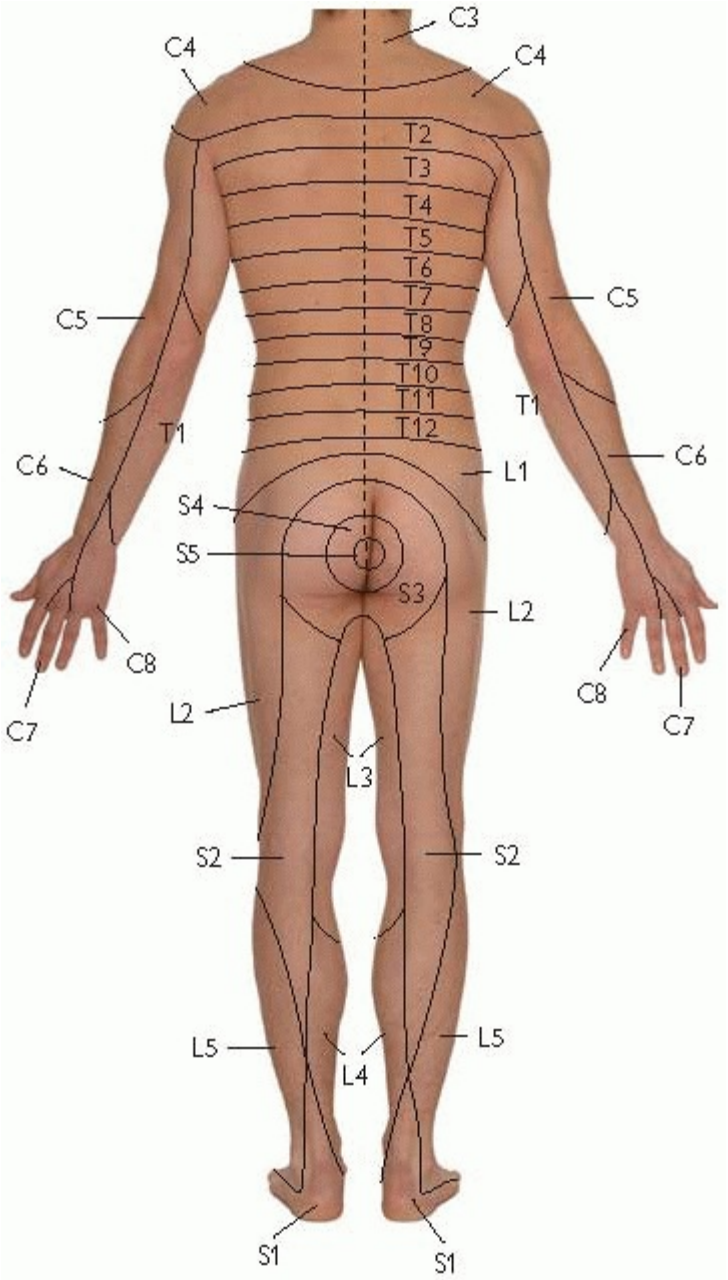


Fig. 10.19 The dermatomes (posterior view).

Sensory examination

This examination can be difficult—as it requires concentration and co-operation on the part of both the patient and the examiner. The results depend on the patient's response and are, therefore, partly subjective. Many patients prove unreliable witnesses due to a lack of understanding or attempts to please the examiner. Education, explanation, and reassurance are, therefore, important at all times.

Often, sensory loss (particularly vibration and temperature) are not noticed by the patient and revealing them during the course of an examination may be upsetting. This should be borne in mind as you proceed.

Technique

Your examination should be influenced by the history. In practice, only light touch is tested as a quick ‘screening’ exam if no deficit is expected.

If you are to test vibration sense and proprioception, it may be best to test these first as they require the least concentration and can be used by you to assess the patients' reliability as a witness before testing the other sensory modalities.

For each modality, you should begin at any area of supposed deficit and work outwards, mapping the affected area—then move to a systematic examination from head to toe. Always test one side, then the other for each limb/body area. You should aim, at least, to test one spot in each dermatome.

Light touch

With the patient's eyes closed, touch their skin with a wisp of cotton wool and ask them to say ‘yes’ when it is felt. The interval between

each touch should be irregular and unpredictable.

- In practice, a gentle touch with a finger is often used. However, this risks testing ‘pressure’ not ‘light touch’ sensation—it is also harder to ensure equal force is applied in all areas.
- Do not, as many do, make tiny stroking movements on the skin—this stimulates hair fibres and, again, is not a test of ‘light touch’.
- Be aware of areas where ↓ sensation is expected (foot calluses etc.).
- After testing each limb/body area, double check with the patient ‘did that feel the same all over?’ and explore any areas of abnormal sensation more thoroughly before moving on.

Sensory inattention

- This is a subtle but often clinically important sign of parietal lobe dysfunction. The patient feels a stimulus on the affected part—but *not* when there is competition from a stimulus on the opposite side.
- Ask the patient to close their eyes and to tell you if they feel a touch on their left or right—use any body part—commonly hands and feet as a quick ‘screen’.
- Touch the right hand, then the left hand, then both.
- The touches should be repeated randomly to confirm the result.
  - E.g. in a right sided parietal lesion, the patient will feel both left and right stimuli but when both sides are touched, they will only be able to feel the stimulus on the left.

Vibration sense

A 128Hz tuning fork (compare with CN-VIII) is used.

- Ask the patient to close their eyes, tap the tuning fork and place the base on a bony prominence—ask if the patient can feel the *vibration*.
- If ‘yes’, confirm by taking hold of the tuning fork with your other hand to stop the vibration after asking the patient to tell you when the vibration ceases.
- As always, compare left to right and work in a systematic fashion, testing bony prominences to include:
  - Finger tip, wrist, elbow, shoulder, anterior superior iliac spine, tibial tuberosity, matatarso-phalangeal joint and toes.

Proprioception

Testing proprioception in the way described below is rather crude and results must be interpreted with the rest of the history and examination.

Loss of position sense is usually distal. Start by testing the patient's big toe as below. This technique can be used at any joint.

- With the patient's eyes closed and leg relaxed, grasp the distal phalanyx of the big toe from the sides.
- Whilst stabilizing the rest of the foot, you should move the toe up and down at the joint.
- Ask the patient if they can feel any movement—and in which direction.
- Flex and extend the joint, stopping at intervals to ask the patient whether the toe is ‘up’ or ‘down’.
- If proprioception is absent, test other joints, working proximally.
- ▸ The toe is gripped from the *sides*—if held incorrectly, pressure on the nail may suggest the toe is pressed down and so on.
- ▸ Normal proprioception should allow the patient to identify very subtle movements which are barely visible.

Box 10.6 Romberg's sign

A further test of joint position sense. When proprioception is lost in the limbs, patients can often stand and move normally as long as they can see the limb in question.

Perform with care—only if you are able to safely catch the patient in the event of a fall!

- Ask the patient to stand—and you stand facing them.

- Ask the patient to close their eyes.
- If there is loss of proprioception, the patient will lose their balance and fall—if so, catch them with care, asking them to open their eyes again immediately if they haven't already done so.

Pain (pin-prick)

Use a disposable pin or safety pin—*not* a hypodermic needle as these break the skin (a line of tiny wounds up a patient's arm or weeping oedema up a shin is an infection risk and very embarrassing!).

- Test as you would for light touch, gently pressing the pin on the skin.
- Test each dermatome in a systematic way, mapping out abnormalities.
- On each touch, ask the patient to say whether it feels *sharp* or *dull*.
- Occasionally test the patient's reliability as a witness with a negative control by using the opposite (blunt) end of the pin.

Temperature

- This is not routinely tested outside of specialist clinics. Loss of temperature sensation may be evident from the history (accidental burns?).
- When tested, test tubes or similar vessels containing hot and ice-cold water are used—and each dermatome tested as above.
- Remember to ensure the exterior of the tube is dry.

Co-ordination

Co-ordination should be tested in conjunction with gait (📖 p.350). Cerebellar lesions cause incoordination on the *ipsilateral* side (📖 p.357). For each of the following, compare performance on the left and right.

Upper limbs

Finger-nose test

- Ask the patient to touch the end of their nose with their index finger.
- Hold your own finger out in front of them—at arm's reach from the patient—and ask them to then touch the tip of your finger with theirs.
- Ask them to move between their nose and your finger (Fig. 10.20).
- Look for intention tremor (worse as it approaches the target) and 'past-pointing' (missing the target entirely).
- The test can be made more difficult by moving the position of your finger each time the patient touches their nose.

Rapid alternating movements

- (This is hard to describe and should be demonstrated to the patient). Ask the patient to repeatedly supinate and pronate their forearm keeping the other arm still such that they clap their hands palm-to-palm, then back-to-palm and so on (see Fig. 10.21).
- Alternatively, ask them to mimic screwing in a light-bulb.
  - Slow and clumsy = dysdiadokokinesis.
  - This is the *inability* to perform rapidly alternating movements (diadoke = Greek for succession). Much confusion seems to surround this, with many students thinking that the actual test is called dysdiadokokinesis. This is not the case.

Rebound

- From a resting (arms at their side) position, the patient should be asked to quickly abduct their arms and stop suddenly at the horizontal.
    - In cerebellar disease, there will be delay in stopping and the arm will oscillate about the intended final position.
- Alternatively, pull on the patient's flexed arms (as if testing elbow flexion power) and suddenly let go. If lacking co-ordination, the patient will hit his/herself in the face. This test does little for doctor-patient trust and rapport and is rarely performed for obvious reasons(!)

Lower limbs

Heel-shin test

- With the patient sitting, legs outstretched, ask them to slide the heel of one foot up and down the shin of the other leg at a moderate pace.
    - A lack of coordination will manifest as the heel moving side to side about the intended path.
    - In sensory—as opposed to cerebellar—ataxia (lack of proprioception), patients will perform worse with their eyes closed.

Foot tapping

- The patient taps your hand with their foot as fast as possible.
    - NB The non-dominant side performs poorly in normal individuals.

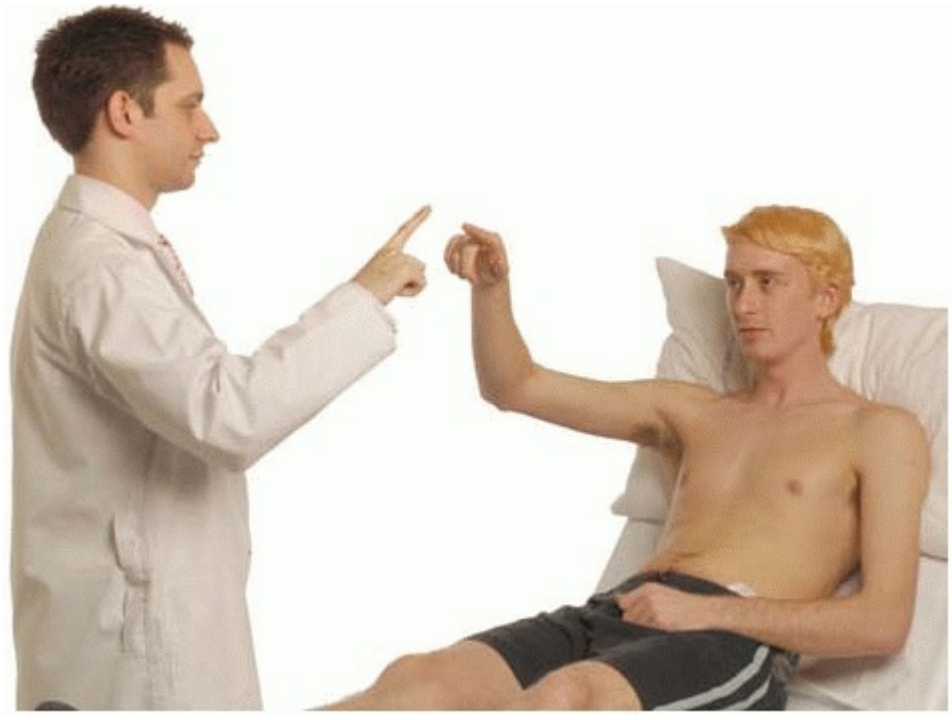


Fig. 10.20 Finger-nose testing.

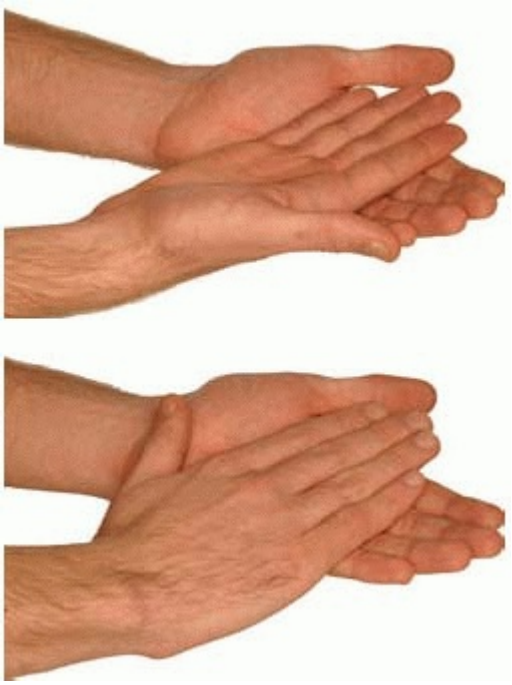


Fig. 10.21 Testing rapidly alternating movements. This can be rather hard to describe to a patient—a brief demonstration is usually required.

Some peripheral nerves

Peripheral nerve lesions may occur in isolation (e.g. trauma, compression, neoplasia) or as part of a wider pathology (e.g. mononeuritis multiplex). The following pages describe the signs following lesions of a selection of peripheral nerves.

Upper limb

Median nerve (C6-T1)

- *Motor:* muscles of the anterior forearm, except flexor carpi ulnaris, and 'LOAF' (lateral 2 lumbricals, opponens pollicis, abductor pollicis brevis and flexor pollicis brevis).
- *Sensory:* thumb, anterior index and middle fingers as well as some of the radial side of the palm (see Fig. 10.22).
- *Examining a lesion:*
  - Weakness and wasting of the thenar eminence.
  - With the hand lying flat, palm up, hold your pen above the thumb and ask the patient to move their thumb vertically to touch it ('pen-touching test')—they will not be able to.
  - NB—often power is good here despite symptoms and obvious carpel tunnel syndrome!
  - For lesions of the nerve at the cubital fossa, perform Ochsner's Clasping Test for weakness of flexor digitorum superficialis—ask the patient to clasp their hands together. If a lesion is present, the index finger will fail to flex.

Ulnar nerve (C8-T1)

- *Motor:* all the small muscles of the hand except LOAF (see above) and flexor carpi ulnaris.
- *Sensory:* ulnar side of hand, little finger and half of ring finger (see Fig. 10.22).
- *Examining a lesion:*
  - Hard to test. There may be visible wasting of the small muscles of the hand with clawing of the fingers (extension at the phalangeo-metacarpal joints and flexion at the interphalangeal joints).
  - Froment's sign: Ask the patient to grasp a piece of paper between their thumb and forefinger. Alternatively, ask them to make a fist. The thumb is unable to adduct so will flex instead (see Fig. 10.23).

Radial nerve (C5-C8)

- *Motor:* triceps, brachioradialis, and extensors of the hand.
- *Sensory:* a small area over the anatomical snuff box—hard to test.
- *Examining a lesion:*
  - Look for ‘wrist drop’. If not obvious, ask the patient to flex at the elbow, pronate the forearm and extend the wrist (you may need to demonstrate). Wrist weakness will become clear.

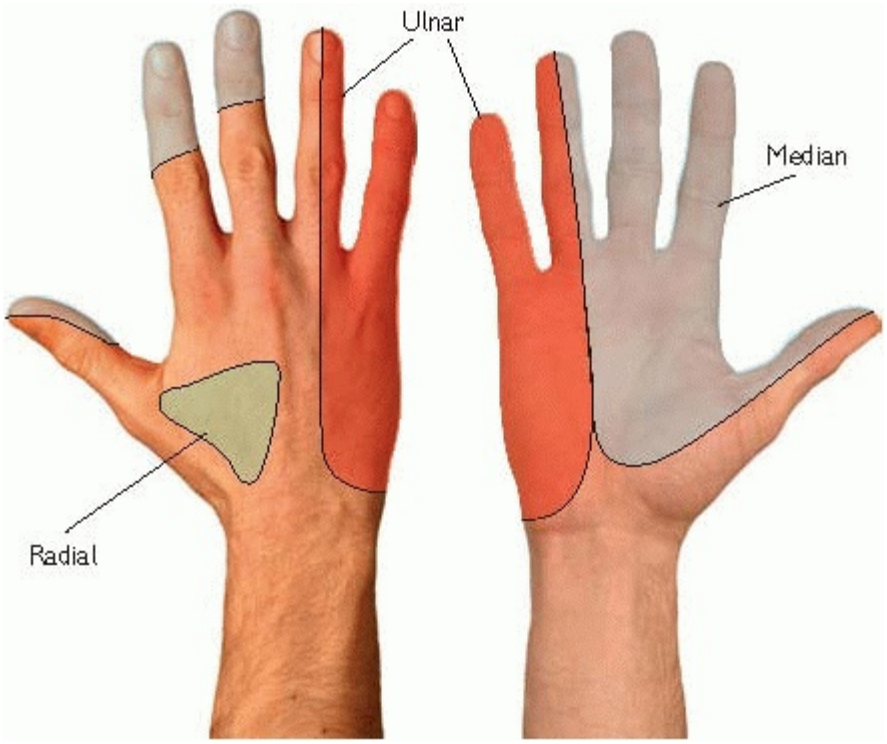


Fig. 10.22 Sensory distribution of the major peripheral nerves in the hand. There is considerable overlap and the small area supplied by the radial nerve may not be detectable clinically.

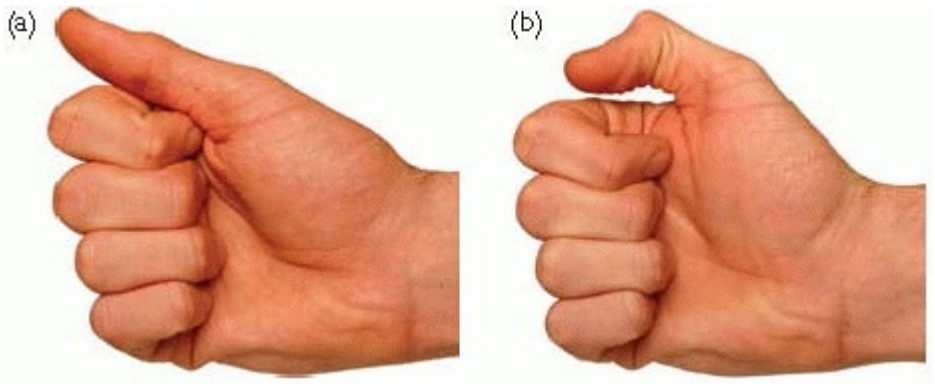


Fig. 10.23 Froment's sign. (a) Normal. (b) Froment's positive.

Lower limb

Lateral cutaneous nerve of the thigh (L2-L3)

- *Motor:* none.
- *Sensory:* the lateral aspect of the thigh (see Fig. 10.24).
- *Examining a lesion:*
  - There may be some sensory loss as indicated but, in practice, this is very hard to test.

Common peroneal nerve (L4-S2)

- *Motor:* anterior and lateral compartments of the leg.
- *Sensory:* the dorsum of the foot and anterior aspect of the leg.
- *Examining a lesion:*
  - 'Foot-drop' with corresponding gait (📖 p.350). Weakness of foot dorsiflexion (📖 p.328) and eversion. Preserved inversion. (Fig. 10.24b.)
  - NB. In an L5 lesion, there will be a similar deficit but will also display a weakness of inversion, hip abduction and knee flexion.

Femoral nerve (L2-L4)

- *Motor:* quadriceps.
- *Sensory:* medial aspect of thigh and leg (see Fig. 10.24d).
- *Examining a lesion:*
  - Weakness of knee extension is only slightly affected—hip adduction is preserved (📖 p.328).
  - Stretch: With the patient lying prone, abduct the hip, flex the knee and plantar-flex the foot. The stretch test is positive if pain is felt in the thigh/inguinal region.

Sciatic nerve (L4-S3)

- *Motor:* all the muscles below the knee and some hamstrings.
- *Sensory:* posterior thigh, ankle and foot (see Fig. 10.24c).
- *Examining a lesion:*
  - Foot drop and weak knee flexion (📖 p.328).
  - Knee jerk reflex (📖 p.330) is preserved but ankle jerk and plantar response (📖 p.332) are absent.
  - Stretch test: with the patient lying supine, hold the ankle and lift the leg, straight, to 90°. Once there, dorsiflex the foot. If positive, pain will be felt at the back of the thigh.

Box 10.7 Tinel's sign

A test for nerve compression. Commonly used at the wrist to test for median nerve compression in carpal tunnel syndrome.

- Percuss the nerve over the site of possible compression (at the wrist, gently tap centrally near the flexor palmaris tendon).
- If the nerve is compressed, the patient will experience tingling in the distribution of the nerve on each tap.

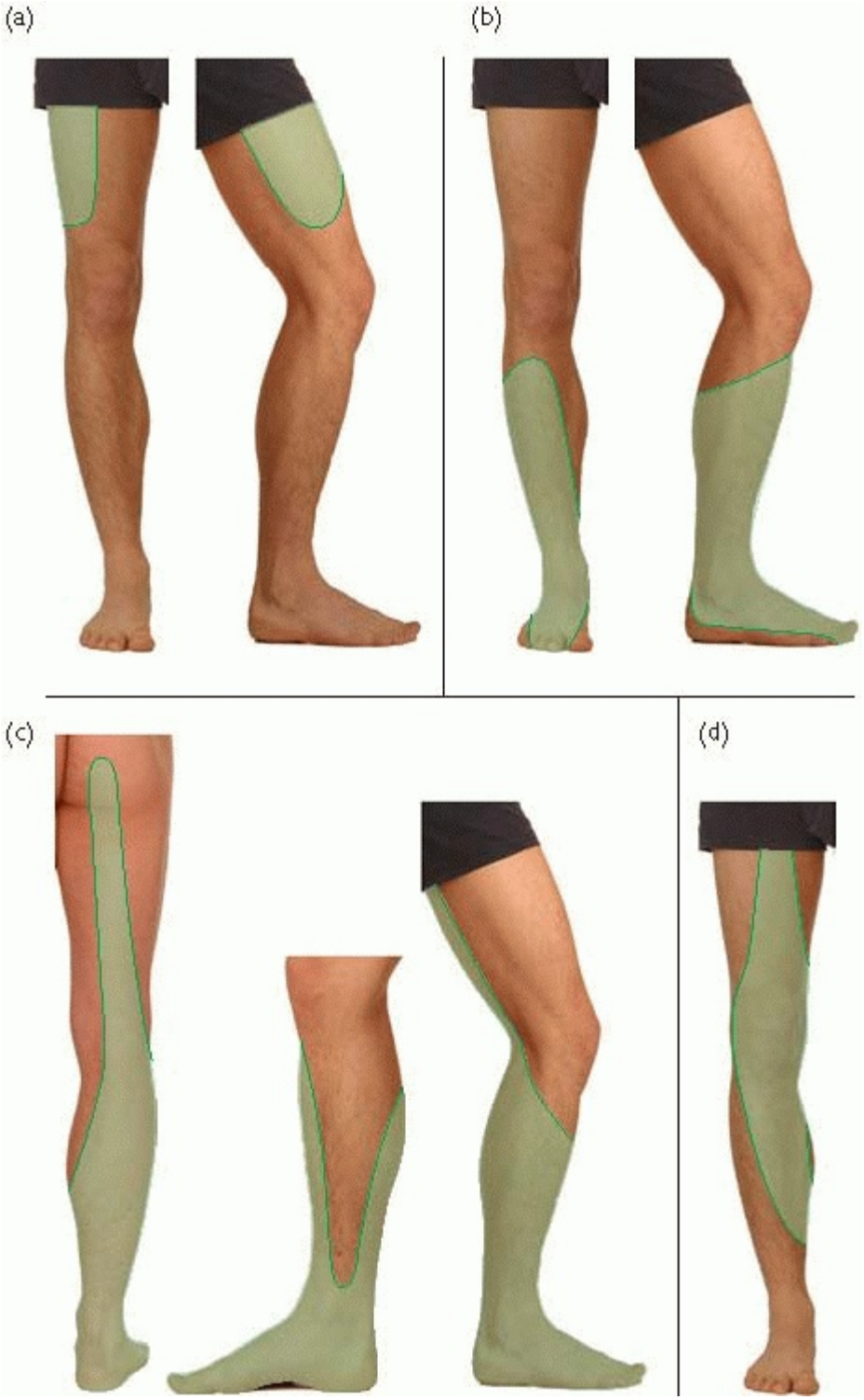


Fig. 10.24 Distribution of the sensory component of some lower limb nerves. (a) Lateral cutaneous nerve of the thigh. (b) Common peroneal nerve. (c) Sciatic nerve. (d) Femoral nerve.

Gait

This is easily missed from the neurological examination—it is often difficult to test in a crowded ward or cramped consulting room. However, you should try to incorporate it into your assessment.

Gait can be observed informally as the patient makes their way to the clinic room or returns to their chair on the ward. Watch the patient stand—and use the same opportunity for Romberg's test (Box 10.6 p.341).

Patient may be simply lacking in confidence and this will be evident later. Do not test if you suspect a severe problem with balance.

Examination

- Ask the patient to walk a few metres, turn and walk back to you.

- Note especially:
  - Use of walking aids.
  - Symmetry.
  - Size of paces.
  - Lateral distance between the feet.
  - How high the feet and knees are lifted.
  - Bony deformities.
  - Disturbance of normal gait by abnormal movements.
- You may want to consider asking the patient to:
  - Walk on tip-toes (inability = S1 or gastrocnemius lesion).
  - Walk on their heels (inability = L4/L5 lesion—foot drop).

Findings

- ***Hemiplegia:*** one side will be obviously weaker than the other with the patient tilting pelvis to lift the weak leg which may swing out to the side. Gait may be unsafe without the use of walking aid.
- ***'Scissoring':*** if both legs are spastic (cerebral palsy, MS), toes drag on floor, trunk sways from side-to-side, and legs cross over on each step.
- ***Parkinsonism:*** flexed posture with small, shuffling steps. No or little arm-swing. Difficulty starting, stopping and turning. Gait seems hurried ('festinant') as legs attempt to prevent body falling forwards.
- ***Cerebellar ataxia:*** broad based (legs wide) gait with lumbering body movements and variable distance between steps. Difficulty turning (be there to catch them!).
- ***Sensory ataxia:*** (loss of proprioception) patient requires more sensory input to be sure of leg position so lifts legs high ('high-stepping') and stamps feet down with a wide-based gait—may also watch legs as they walk. Romberg's positive (see  Box 10.6 p.341).
- ***Waddling:*** (weakness of proximal lower limb muscles) patient fails to tilt pelvis as normal so ↑ rotation to compensate—also at the shoulders. May also see ↑ lumbar lordosis.
- ***Foot drop:*** (L4/L5 lesion, sciatic, or common peroneal nerves) failure to dorsiflex the foot leads to a 'high-stepping' gait with ↑ flexion at the hip and knee. If bilateral, may indicate peripheral neuropathy.

- ***Apraxic:*** (usually frontal lobe pathology such as normal pressure hydrocephalus or cerebrovascular disease) problems with gait even if all other movements may be normal. Patient may appear frozen to the spot and unable to initiate waking. Movements are disjointed once walking.
- ***Marche à petits pas:*** (diffuse cortical dysfunction) Upright posture, small steps with a normal arm-swing.
- ***Painful gait:*** The cause will normally be obvious from the history. The patient limps with an asymmetrical gait due to painful movement.
- ***Functional:*** (also known as hysterical) Gait problems will be variable and inconsistent, often with bizarre and elaborate consequences. May fall without causing injury. Often worse when watched.

Important presenting patterns

Neck stiffness

Caused by a number of conditions provoking painful extensor muscle spasm including bacterial and viral meningitis, subarachnoid haemorrhage, parkinsonism, raised intracranial pressure, cervical spondylosis, cervical lymphadenopathy, and pharyngitis.

▸ None of the following tests should be conducted if there is suspicion of cervical injury or instability.

Testing stiffness

- Lie the patient flat.
- Taking their head in your hands, gently rotate it to the sides in a 'no' movement, feeling for stiffness.
- Lift the head off the bed and watch the hips and knees—the chin should easily touch the chest.

Brudzinski's sign

When the head is flexed by the examiner, the patient briefly flexes at the hips and knees—a test for meningeal irritation.

Kernig's sign

- A further test of meningeal irritation.
- With the patient lying flat, flex their hip and knee, holding the weight of the leg yourself.
- With the hip flexed to 90°, extend the knee joint so as to point the leg at the ceiling.
- If ‘positive’, there will be resistance to leg straightening (caused by hamstring spasm as a result of inflammation around the lumbar spinal roots) and pain felt at the back of the neck.

Lhermitte's phenomenon

- A test for an intrinsic lesion in the cervical cord (not meningeal irritation).
- When the neck is flexed as above, the patient feels an electric shock-like sensation down the centre of their back.

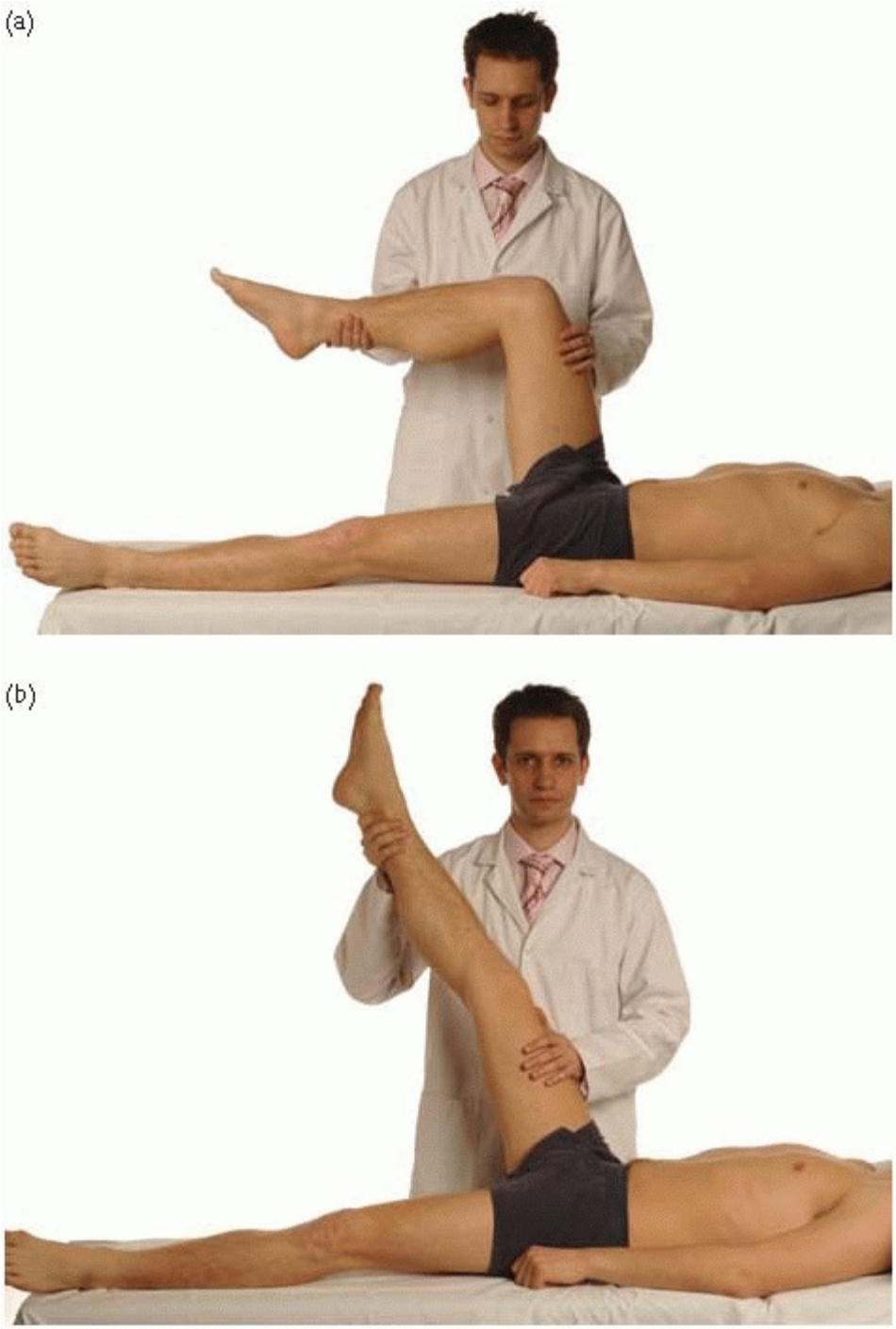


Fig. 10.25 Testing for Kernig's sign. The patient's leg is flexed at the hip and knee, then extended at the knee as above. If positive, there is resistance to knee extension in this position and pain is felt at the back of the neck.

Upper motor and lower motor nerve lesions

Upper motor neuron (UMN) lesions

Defined as damage *above* the level of the anterior horn cell—anywhere from the spinal cord to the primary motor cortex.

- No muscle wasting (although will have disuse atrophy in long-term weakness).
- ↑ tone. 'Spasticity' (clasp-knife) due to stretch reflex hypersensitivity.
- Typical pattern of weakness is termed 'pyramidal':
  - Upper limbs: weak abductors and extensors.
  - Lower limbs: weak adductors and flexors.
- ↑ tendon reflexes and clonus. Up-going plantar response.

Lower motor neuron (LMN) lesions

- Muscle wasting. Fasciculations.
- ↓ tone
- Flaccid weakness.
- ↓ tendon reflexes. Plantar response may be down-going or absent.

Box 10.8 Some definitions of weakness
Monoplegia: one limb affected.
Hemiplegia: one side of the body (left or right).
Paraplegia: both lower limbs affected.
Quadriplegia: all 4 limbs.

Motor neuron disease (MND)

See *OHCM6*, p.394. Damage to anterior horn cells, medulla, and spinal tracts.

- UMN and LMN pattern of weakness.
- Fasciculations almost always present.
- Reflexes normal or ↑ until later in the disease.
- Plantar response is up-going.
- External ocular muscles almost never involved.
- No sensory disturbance (distinguishing the presentation from a polyneuropathy).

Parkinsonism

A pattern of symptoms comprising an akinetic-rigid syndrome. Parkinsonism has a number of causes including drug-induced and other intracranial pathologies. The diagnosis of Parkinson's disease (loss of dopaminergic neurons in the subtantia nigra) is often inaccurate and there is no single test. See *OHCM6*, p.382.

- A triad of resting tremor, bradykinesia and rigidity.
- ***Face:*** mask-like and expressionless facies, little blinking, positive glabellar tap reflex (p.334).
- ***Gait:*** flexed posture with ↓ arm swing. Gait is 'festinant' meaning 'hurried' often in small, shuffling steps with feet barely lifted off ground. Slow to start and difficulty stopping.
- ***Tone:*** ↑ tone with 'cogwheel' or 'lead-pipe' rigidity (p.324).
- ***Tremor:*** 'pill-rolling' flexion at the thumb and forefinger at 4-8Hz.
- ***Speech:*** extrapyramidal dysarthria, soft, quiet, and hesitant speech. You may have to wait some time for the answer to a question!
- ***Writing:*** writing is small and neat 'micrographia'.

Abnormal movements defined

- ***Akathisia:*** motor restlessness with a feeling of muscle quivering and an inability to remain in a sitting position.
- ***Athetosis:*** slow, writhing involuntary movements often with flexion, extension, pronation, and supination of the fingers and wrists.
- ***Blepharospasm:*** intermittent spasm of muscles around the eyes.
- ***Chorea:*** non-rhythmical, dance-like, spasmodic movements of the limbs or face. Appear pseudo-purposeful (the patient often hides the condition by turning a spasm into a voluntary movement—e.g. the arm suddenly lifts up and the patient pretends they were adjusting their hair).
- ***Dyskinesia:*** repetitive, automatic movements that stop only during sleep.

- ***Tardive dyskinesia:*** dyskinetic movements often of the face (lip-smacking, twisting of the mouth). Often a side-effect of neuroleptic therapy.
- ***Dystonia:*** markedly ↑ tone often with spasms causing uncomfortable-looking postures.
- ***Hemiballismus:*** violent involuntary flinging movements of the limbs on one side—rather like severe chorea.
- ***Myoclonus:*** brief, shock-like movement of a muscle or muscle-group.
- ***Pseudoathetosis:*** writhing limb movements (often finger/arm) much like athetosis but caused by a loss of proprioception. The arm returns to the normal position when the patient notices it straying.
- ***Myokymia:*** continuous quivering and rippling movements of muscles at rest like a 'bag or worms'. Facial myokymia: especially near the eyes.
- ***Tic:*** repetitive, active, habitual, purposeful contractions causing stereotyped actions. Can be suppressed for brief periods with effort.
- ***Titubation:*** rhythmical contraction of the head. May be either 'yes-yes' or 'no-no' movements.
- ***Tremor:*** repetitive, alternating movements, usually involuntary.

Spinal cord lesions

As neurons in some spinal cord tracts relate to the contralateral side of the body, others the ipsilateral side, certain types of spinal cord damage will give predictable patterns of motor and sensory loss.

Complete section of the cord

Loss of all modalities below the level of the lesion.

Hemisection of the cord

Also known a 'Brown-Sequard syndrome'*.

- Motor: below the level of the lesion, UMN pattern of weakness on ipsilateral side.
- Sensory: below the level of the lesion:
  - Contralateral loss of pain and temperature sensation.
  - Ipsilateral loss of light-touch, vibration sense, and proprioception.
  - (Light touch may remain intact as some fibres travel in the spinothalamic tract.)

Posterior column loss

Loss of vibration sense and proprioception on both sides below the level of the lesion.

Subacute combined degeneration of the cord

'Posterolateral column syndrome' often due to vitamin B_{12} deficiency.

- Loss of vibration sense and proprioception on both sides below the level of the lesion.
- UMN weakness in lower limbs, *absent* ankle reflexes.
- (also peripheral sensory neuropathy, optic atrophy and dementia—see 📖 *OHCM6*, p.634).

Anterior spinal artery occlusion

- Loss of pin-prick and temperature sensation below the lesion.
- Intact light-touch, vibration sense, and proprioception

Central lesions

E.g. syringomyelia—longitudinal cavities in the central part of the spinal cord and brainstem.

- Loss of pain and temperature sensation over the neck, shoulders, and arms in a ‘cape’ distribution.
- Intact vibration sense, proprioception, and light touch.
- Atrophy and areflexia in the arms.
- UMN weakness in the lower limbs.
- Look also for scoliosis due to weakness of paravertebral muscles.

Cerebellar lesions

(See also 📖 *OHCM6*, p.387) Signs are *ipsilateral* to the lesion and may include:

- Nystagmus (📖 p.307).
- Speech is staccato, scanning (📖 p.286).
- ↓ tone, drift and tremor in limbs (upper especially).
- Finger-nose testing (📖 p.344) may reveal intention tremor and past-pointing.
- Dysdiadokokinesis (📖 p.344).
- Rebound (📖 p.344).
- Pendular jerks—best seen in the knee. Test the tendon reflex at the knee as normal. If ‘pendular’, the extensor response will continue to several beats.
- Poor sitting balance.
- Ataxic gait.

Disturbance of higher functions

A selection of testable consequences of cortical lesions…

Parietal lobe

- Sensory and visual inattention (📖 p.340 and p.293).
- Visual field defects (📖 p.294).
- Agnosias (lack of sensory perceptual abilities).
  - *Hemi-neglect*—patient ignores one side of their body.
  - *Asomatagnosia*—patient fails to recognize own body part.
  - *Anosagnosia*—patient is unaware of neurological deficits.
  - *Finger agnosia*—patient is unable to show you different fingers when requested (e.g. ‘show me your index finger’).
  - *Astereognosis*—inability to recognize an object by touch alone.
  - *Agraphaesthesia*—inability to recognize letters or numbers when traced on the back of the hand.
  - *Prosopagnosia*—inability to recognize faces (test with family members or famous faces from a nearby magazine).
- Apraxias (inability to perform movements or use objects correctly).
  - *Ideational apraxia*—unable to perform task but understands what is required.
  - *Ideomotor apraxia*—performs task but makes mistakes (e.g. puts tea into kettle and pours milk into cup).
  - *Dressing apraxia*—inability to dress correctly (test with a dressing gown). One of a number of apraxias named after the action tested.
- *Gerstmann's syndrome:* Right-left dissociation, finger agnosia, dysgraphia (writing defect), dyscalculia (test with serial 7s— 📖 p.502).

Temporal lobe

- Memory loss—confabulation (invented stories and details).

Frontal lobe

- Primitive reflexes.
- Concrete thinking (unable to explain proverbs—e.g. ask to explain what ‘a bird in the hand is worth two in the bush’ means).
- Loss of smell sensation.
- Gait apraxia (📖 p.350).

Myopathies

Muscle disease causes a weakness similar to that of LMN lesions with no sensory loss. Tendon reflexes are reduced or absent. The causes and types are numerous (📖 *OHCM6*, p.398).

Myotonias

Characterized by continued, involuntary muscle contraction after volountary effort has ceased.

- Myotonic dystrophy: (a defect of skeletal muscle Cl^- channels caused by a trinucleotide repeat, usually. Becomes evident age 20-50).
  - Distal limb weakness.
  - Weak sternomastoids (evident by weakness of neck flexion with normal power on neck extension).
  - Weak facial muscles (expressionless face).
  - Test by shaking the patient's hand—they may be unable to let go.
  - Also associated with frontal baldness, cataracts (look for thick glasses), mild intellectual impairment, cardiomyopathy, hypogonadism, and glucose intolerance.
- Percussion myotonia:
  - Tap the patient's thenar eminence, this will cause contraction and very slow relaxation of abductor pollicis brevis.

Myaesthenia gravis

An autoimmune disease with antibodies against acetylcholine receptors.

- Complex palsies—including extraocular muscles. Weakness is proximal and often includes the eyelids (ptosis) and muscles of mastication.
- Weakness increases with use. Patients report ↑ severity of symptoms at the end of the day.
- In the early stages, will feel tired as they expend extra energy in performing routine tasks. Friends may notice ptosis (see Fig. 10.25).
- Test for fatiguability:
  - Ask the patient to look to the ceiling and hold the position. Watch for ptosis.
  - Ask the patient to hold their arms above their head—or out to the sides—watch for ↑ weakness.

Some characteristic headaches summarized

Tension headache

- Bilateral—frontal, temporal.
- Sensation of tightness radiating to neck and shoulders.

- Can last for days.
- No associated symptoms.

Subarachnoid haemorrhage

- Sudden, dramatic onset ‘like being hit with a brick’.
- Occipital initially—may become generalized.
- Associated with neck stiffness and sometimes photophobia.

Sinusitis

- Frontal, felt behind the eyes or over the cheeks.
- Ethmoid sinusitis is felt deep behind the nose.
- Overlying skin may be tender.
- Worse on bending forwards.
- Lasts 1-2 weeks. Associated with coryza.

Temporal (giant cell) arteritis

- Diffuse, spreading from the temple—unilateral.
- Tender overlying temporal artery (painful brushing hair).
- ?jaw claudication whilst eating.
- ?blurred vision—can lead to loss of vision if severe and untreated.

Meningitis

- Generalized.
- Associated with neck stiffness and signs of meningism (📖 p.352).
- Nausea, vomiting, photophobia.
- (Purpuric rash is caused by septicaemia, not meningitis *per se*).

Cluster headache

- Rapid onset, usually felt over one eye.
- Associated with a blood-shot, watering eye, and facial flushing.
- May also have rhinorrhoea (runny nose).
- Last for a few weeks at a time.

Raised intracranial pressure

- Generalized headache, worse when lying down, straining, coughing, on exertion or in the morning.
- Headache may wake the patient in the early hours.
- May be associated with drowsiness, vomiting, and focal neurology.

Migraine

- Unilateral—rarely crosses the midline*.
- Throbbing/pounding headache.
- Associated with photophobia, nausea, vomiting, and neck stiffness.
- May have preceding aura.

* Hence the name. 'Migraine' is a shortening of 'hemicranium' (say the 2nd and 3rd syllables out loud if you don't believe us) meaning 'half the head'.

The unconscious patient

History

- Eye-witness account? State of clothing—loss of continence?
- Look for alert necklace/bracelet. Look in wallet, purse etc.

Examination

(see also *OHCM6*, p.777)

- ***ABC:*** (covered in detail in other Oxford Handbooks).
  - Is the airway patent? Should the patient be in the recovery position?
  - Measure respiratory rate, note pattern of breathing. Is O_2 needed?
  - Cyanosis? Feel pulse. Listen to chest. Measure heart rate, BP.
- ***Skin:*** look for injury, petechial haemorrhage, evidence of IV drug-use.
- ***Movements/posture:***
  - Watch! Is the patient still or moving? All 4 limbs moving equally?
  - Any abnormal movements—fitting, myoclonic jerks?
  - Test tone and compare both sides.
  - Squeeze the nail bed to test the response to pain (all 4 limbs).
  - Test tendon reflexes and plantar response.
  - *Decorticate posture:* (lesion above the brainstem) flexion and internal rotation of the arms, extension of the lower limbs.
  - *Decerebrate posture:* (lesion in the midbrain) extension at the elbow, pronation of the forearm and extension at the wrist. Lower limbs extended.
- ***Consciousness:*** attempt to wake the patient by sound. Ask their name. If responsive, are they able to articulate appropriately?—Note the best response. Be aware of possible dys- or aphasia which may cause an inappropriate response in an otherwise alert individual.
  - Score level of consciousness according to the GCS (Box 10.9).
- ***Neck:*** do not examine if there may have been trauma. Test for meningeal irritation (p.352)—these signs ↓ as coma deepens.
- ***Head:*** inspect for signs of trauma and facial weakness. Test pain sense.
  - Battle's sign: bruising behind the ear = a base of skull fracture.
- ***Ears/nose:*** look for CSF leakage or bleeding. Test any clear fluid for glucose (positive result = CSF). Inspect eardrums.
- ***Tongue/mouth:*** Look for cuts on the tongue (seizures), corrosive material around the mouth. Smell breath for alcohol or ketosis. Test the gag reflex—absent in brainstem disease or deep coma.
- ***Eyes:***
  - Pupils: measure size in mm. Are they equal (p.302)? Test direct and consensual light responses. Pupils ↑ with atropine, tricyclic antidepressants, and amphetamine; ↓ with morphine and metabolic coma.

- Test corneal reflex.
- Fundi: look especially for papilloedema and retinopathy.
- Doll's head manoeuvre: take the patient's head in your hands and turn it from side-to-side. The eyes should move to stay fixed on an object—indicates an intact brainstem.

- ***Rest of the body:*** a brief but thorough exam. Look especially for trauma, fractures, signs of liver disease (p.218) and added heart sounds.
- ***Other bedside tests:*** test urine, capillary glucose and temperature.

Box 10.9 Glasgow Coma Scale (GCS)

This is an objective score of consciousness. Repeated testing is useful for judging whether coma is deepening or lifting. There are 3 categories as below. Note that the lowest score in each is '1' meaning that the lowest possible GCS = 3 (even if the patient is dead!)

Eye opening (max 4 points)

Spontaneously open	4
Open to (any) verbal stimulus	3
Open in response to painful stimulus	2
No eye opening at all	1

Best verbal response (max 5 points)

Conversing and orientated (normal)	5
Conversing but disorientated and confused	4
Inappropriate words (random words, no conversation)	3
Incomprehensible sounds (moaning etc)	2
No speech at all	1

Best motor response (max 6 points)

Obeying commands (e.g. raise your hand)	6
Localising to pain (moves hand towards site of stimulus)	5
Withdraws to pain (pulls hand away from stimulus)	4
Abnormal flexion to pain (decorticate posturing)	3

Abnormal extension to pain (decerebrate posturing)	2
No response at all	1

Box 10.10 AVPU

A much more simplified score used in rapid assessment of consciousness and often by non-specialist nurses in monitoring conscious level.

A = **A**lert

V = responds to **V**oice

P = responds to **P**ain

U = **U**nresponsive

The elderly patient

Diagnosing and managing neurological illness can be complex, but the combination of cognitive failure and the effects of an ageing neurological system can present significant challenges for clinicians.

Presentations of neurological disease are varied, and the range of diagnoses diverse. Epilepsy, parkinsonism, and dementias are all common problems in older age—so resist the temptation to restrict your diagnoses to stroke or TIA.

History

- ***Witness histories:*** are vital. Many patients may attend with vague symptoms that may be underplayed. Partial complex seizures may be very difficult to diagnose—so pursue witness histories from families, neighbours, home care staff etc. Enquire not just about the present incident, but prior function and any decline.
- ***Drug history:*** falls are a common presentation and often multifactorial. Always remember to ask about any drugs that may lower blood pressure, even if the primary cause of the fall is due to neurological disease.
- ***Intercurrent illness:*** may precipitate further seizures or make pre-existing neurological signs seem worse. Don't rush to diagnose a worsening of the original problem—careful assessment pays dividends.
- ***Cognition and mood disorders:*** often complicate presentations. Look for clues in the history and ask witnesses.
- ***Functional history:*** a key part of the neurological history. The disease itself may be incurable—functional problems often are not.

Examination

- ***Observe:*** Non verbal clues may point to mood or cognitive disorders. Handshakes and facial expressions are an important part of the examination
- ***Think:*** about patterns of illness, and attempt to identify if there are single or multiple lesions. There may often be more than one diagnosis—e.g. cerebrovascular disease and peripheral neuropathy due to diabetes.
- ***Assess cognition:*** use a scale you are comfortable with such as the Abbreviated Mental Test Score (AMTS p.288; MMSE p.503)—but remember no half marks!
- ***Gait:*** even simple observation of a patient's walking can reap rewards. Always include it in your examination where practicable and note why if unable.
- ***Therapy colleagues:*** sharing observations is a useful practice. Therapists are a huge fount of knowledge and experience so seek to learn from them.

Additional points

- ***Communicating diagnoses:*** many diagnoses—e.g. dementia and motor neuron disease,—can be devastating, so be thoughtful in your approach. Clarify what the patient knows, and what has already been said—learn first from your seniors how to explain the diagnosis, and more importantly talk about its impact. It is also vital to reassure—many patients with benign essential tremors are terrified that they may have Parkinson's disease
- ***Managing uncertainty:*** many diagnoses are not clear, especially in the early stages of diseases. Try to resist labelling your patients

when a diagnosis is unclear; be open about uncertainty—patients often cope with it better than their doctors.

We thank Dr Richard Fuller for providing this page.

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Title: *Oxford Handbook of Clinical Examination and Practical Skills, 1st Edition*

Chapter 11

The Locomotor System

The **hand examination** is described in Chapter 3, p.72

Applied anatomy and physiology

The joint

A joint (articulation) is a connection or point of contact between bones or between bone and cartilage.

Joints are classified according to the type of material uniting the articulating bones as well as the degree of movement they allow. There are 3 types:

- ***Fibrous joints:*** held together by fibrous (collagenous) connective tissue and are 'fixed' or 'immoveable'. They do not have a joint cavity. Examples include the connections of the skull bones.
- ***Cartilagenous joints:*** held together by cartilage, are slightly moveable and again have no cavity. An example is the vertebral joints.
- ***Synovial joints:*** covered by cartilage with a synovial membrane enclosing a joint cavity. These joints are freely moveable and are the most common type of joint functionally, being typical of nearly all the joints of the limbs.

Synovial joints

Articular cartilage covers the surface of the bones and ↓ the friction at the joint and facilitates shock absorption.

A sleeve-like bag (a fibrous capsule lined with a synovial membrane) surrounds the synovial joint.

The inner synovial membrane secretes synovial fluid which has a number of functions including lubrication and the supply of nutrients to the cartilage. The fluid contains phagocytic cells that remove microbes and debris within the joint cavity.

Synovial joints are usually supported by accessory ligaments and muscle. There are different types of synovial joints and some of the more important types are:

- ***Hinge:*** movement occurs primarily in a single plane (e.g. elbow, knee, and interphalangeal joints).
- ***Ball and socket:*** allows movement around 3 axes (flexion/extension, abduction/adduction, and rotation). Examples are the shoulder and hip.
- ***Pivot:*** a ring of bone and ligament surrounds the surface of the other bone allowing rotation only (e.g. atlanto-axial joint at C1 and C2 vertebrae and the connection between the radius and ulna).
- ***Gliding:*** flat bone surfaces allow side-to-side and backwards and forwards movements (e.g. between carpals, tarsals, sternum and clavicle and the scapula and clavicle).
- ***Saddle:*** similar to a hinge joint but with a degree of movement in a second plane (e.g. base of thumb).

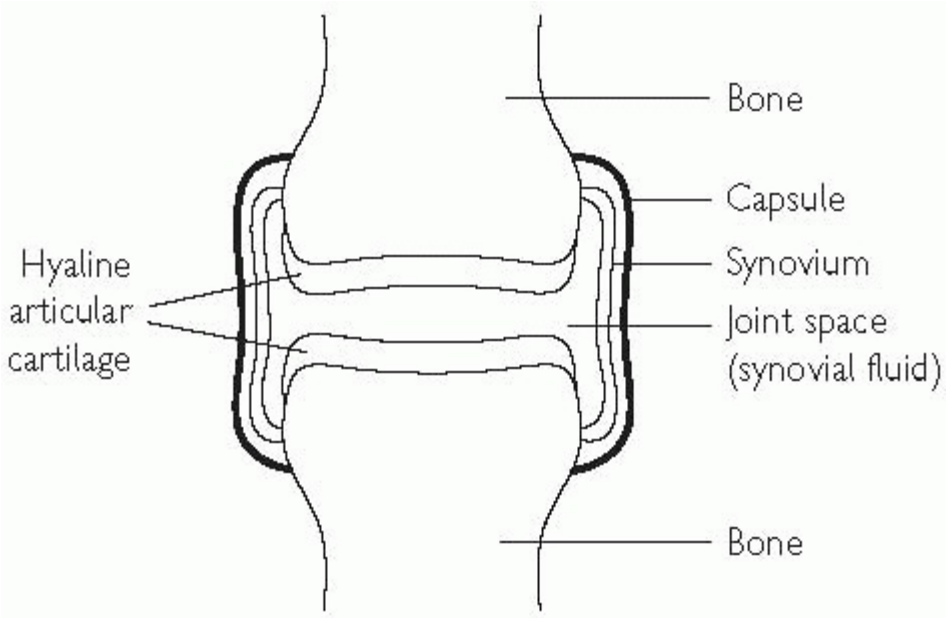

Fig. 11.1 Diagrammatic representation of a cross-section through atypical synovial joint).

Box 11.1 Some movements at synovial joints

Angular movements

- *Flexion:* a decrease in the angle between the articulating bones (e.g. bending the elbow = elbow flexion).
- *Extension:* an increase in the angle between the articulating bones (e.g. straightening the elbow = elbow extension).
- *Abduction:* movement of a bone away from the midline (e.g. moving the arm out to the side = shoulder abduction).
- *Adduction:* movement of a bone towards the midline (e.g. bring the arm in to the side of the body = shoulder adduction).

Rotation

Movement of a bone about its longitudinal axis.

- *Internal or medial rotation:* rotating a bone towards the midline (e.g. turning the lower limb such that the toes point inwards = internal rotation at the hip).
- *External or lateral rotation:* rotating a bone away from the midline (e.g. turning the lower limb such that the toes point outwards = external rotation at the hip).

Special movements

These occur at specific joints only.

- *Pronation:* moving the forearm as if turning a door-knob anticlockwise (internal rotation of the forearm in the anatomical position).
- *Supination:* moving the forearm as if turning a door-knob clockwise (external rotation of the forearm in the anatomical position).
- *Dorsiflexion:* moving the ankle to bring the dorsum of the foot towards the tibia.
- *Plantar flexion:* moving the ankle to bring the plantar surface in line with the tibia (e.g. pointing the toes or depressing a pedal).
- *Inversion:* tilting the soles of the feet inwards to face each other.
- *Eversion:* tilting the soles of the feet outwards away from each other.
- *Protraction:* moving the mandible forward.
- *Retraction:* moving the mandible backwards.

Important locomotor symptoms

As with any system, but especially the locomotor system, a carefully and accurately compiled history can be very informative and may point to a diagnosis even before examination or laboratory tests.

Pain

The most common symptom in problems of the locomotor system and should be approached in the same manner as any other type of pain (

 p.xxx). Determine the character, nature of onset, site, radiation, severity, periodicity, exacerbating and relieving factors (with particular reference to how it is influenced by rest and activity), and diurnal variation.

- Pain in a joint is called ‘arthralgia’.
- Pain in a muscle is called ‘myalgia’.

Character

- Bone pain is typically experienced as boring, penetrating and often worse at night. Causes include tumour, chronic infection, avascular necrosis, and osteoid osteoma.
- Pain associated with a fracture is usually sharp and stabbing in nature and often exacerbated by movement.
- Shooting pain is suggestive of nerve entrapment (e.g. disc protrusion).

Onset

- Acute onset of pain is often a manifestation of infection such as septic arthritis or crystal arthropathies (e.g. gout).
- Osteoarthritis and rheumatoid arthritis can cause chronic pain.

Site

Determine the exact site of maximal pain if possible and any associated lesser pains.

Remember that the site of pain is not necessarily the site of pathology; often pain is referred. Referred pain is due to the inability of the cerebral cortex to distinguish between sensory messages from embryologically related sites. See tables opposite.

Box 11.2 Some causes of knee arthralgia explained

Chondromalacia patellae

This arises due to softening of the patellar articular cartilage and is felt as a patellar ache after prolonged sitting. Usually seen in young people.

Osteochondritis dissecans

Usually associated with trauma resulting in an osteochondral fracture which forms a loose body in the joint with underlying necrosis.

Osgood-Schlatter's disease

Arises as a result of a traction injury of the tibial epiphysis which is classically associated with a lump over the tibia.

Box 11.3 Some causes of arthralgia in adults

Knee

- Osteoarthritis.
- Referred from the hip.
- Chondromalacia patellae.
- Trauma.
- Osteochondritis dissecans.
- Bursitis.
- Tendonitis.
- Osgood-Schlatter's disease.
- Rheumatoid arthritis.
- Infection.
- Malignancy.

Hip

- Osteoarthritis.
- Referred pain—e.g. from a lumber spine abnormality.
- Trauma.
- Rheumatoid arthritis.

- Infection.
- Hernia.
- Tendonitis.
- Bursitis.

Shoulder

- Rotator cuff disorders (e.g. tendonitis, rupture, adhesive capsulitis/frozen shoulder).
- Referred pain—e.g. cervical, mediastinal, cardiac.
- Arthritis—glenohumeral, acromioclavicular.

Elbow

- Lateral epicondylitis (tennis elbow).
- Medial epicondylitis (golfer's elbow).
- Olecranon bursitis.
- Referred pain from neck/shoulder (e.g. cervical spondylosis).
- Osteoarthritis.
- Rheumatoid arthritis.

Mechanical/degenerative back pain

- Arthritis.
- Trauma.
- Disc prolapse.
- Osteoporosis.
- Infection.
- Ankylosing spondylitis.
- Spondylolisthesis.
- Lumbar spinal/lateral recess stenosis.
- Spinal tumours— especially metastases from lung, breast, prostate, thyroid, kidney.
- Metabolic bone disease.

Stiffness

This is a subjective symptom which must be explored in detail to establish exactly what the patient means.

Stiffness is the inability to move the joints after a period of rest. It may be due to mechanical dysfunction, local inflammation of a joint or a combination of both.

▸ If stiffness predominates over pain, consider spasticity or tetany. Look for hypertonia and other upper motor neuron signs ( p.354).

Ask the patient:

- When is the stiffness worst?
  - 'Early morning stiffness' is seen in inflammatory conditions (e.g. rheumatoid arthritis) whereas mechanical joint disease will become worse as the day progresses.
- Which joints are involved?… Or is the stiffness generalized?
  - A generalized stiffness may be seen in rheumatoid arthritis and ankylosing spondylitis.
- How long does it takes them to 'get going' in the morning?
- How is the stiffness related to rest and activity?
  - Mechanical joint diseases will be exacerbated by prolonged activity.

Locking

This is the sudden inability to complete a certain movement and suggests a mechanical block or obstruction usually caused by a loose body or torn cartilage within the joint (often secondary to trauma).

Swelling

Joint swelling can be due to a variety of factors including inflammation of the synovial lining, an ↑ in the volume of synovial fluid, hypertrophy of bone, or swelling of structures surrounding the joint.

This symptom is particularly significant in the presence of joint pain and stiffness. Establish:

- Which joints are affected (small or large)?
- Is the distribution symmetrical or not?
- What was the nature of onset of the swelling?
  - Rapid onset: haematoma or haemarthrosis (exacerbated by anticoagulants or any underlying bleeding disorder).
  - Slow onset is suggestive of a joint effusion.
- Are the joints always swollen or does it come and go (and when)?
- Is there any associated pain?
- Do the joints feel hot to touch?
- Is there erythema? (Common in infective, traumatic and crystal arthropathies).
- Have the joints in question sustained any injuries?

Deformity

Establish:

- The time frame over which the deformity has developed.
- Any associated symptoms such as pain and swelling.
- Any resultant loss of function? (What is the patient now unable to do with the joint in question?)

Acute deformity may arise with a fracture or dislocation.

Chronic deformity is more typical of bone malalignment and may be partial/subluxed or complete/dislocated.

Some common deformities are discussed later in this chapter.

Weakness

Always enquire about the presence of localized or generalized weakness which suggest a peripheral nerve lesion or a systemic cause respectively.

Consider that the weakness may be neurogenic or myopathic in origin.

Sensory disturbance

Ask about the exact distribution of any numbness or paraesthesia as well as documenting any exacerbating and relieving factors.

Loss of function

This is the inability to perform an action (disability) and is distinguished from the term ‘handicap’ which is the social/functional result or impact that disability has on the individual's life.

Loss of function can be caused by a combination of muscle weakness, pain, mechanical factors and damage to the nerve supply.

The questions you ask will depend partly on the patient's occupation. It is also essential to gain some insight into the patient's mobility (can they use stairs? How they cope with personal care such as feeding, washing and dressing? Can they manage shopping and cooking?).

Extra-articular features

Several locomotor disorders (e.g. rheumatoid arthritis) cause extra-articular or multisystem features, some of which are outlined below:

- Systemic symptoms: fever, weight loss, fatigue, lethargy.
- Skin rash.
- Raynaud's phenomenon.

- Gastrointestinal (e.g. diarrhoea and resultant reactive arthritis or enteropathic arthritis secondary to inflammatory bowel disease).
- Urethritis (Reiter's syndrome).
- Eye symptoms.
- Cardiorespiratory: breathlessness (pulmonary fibrosis?), pericardial and pleuritic chest pain, aortic regurgitation and spondyloarthropathies.
- Neurological: nerve entrapment, migraine, depression, stroke.

Box 11.4 Some terminology of joint deformity

Valgus

The bone or part of limb distal to the joint is deviated laterally.

For example, a valgus deformity at the knees would give 'knock knees' that tend to meet in the middle despite the feet being apart.

Varus

Here, the bone or part of limb distal to the joint is deviated medially.

For example, a varus deformity at the knees would give 'bow legs' with a gap between the knees even if the feet are together.

The rest of the history

Past medical history

Ask about all previous medical and surgical disorders and enquire specifically about any previous history of trauma or musculoskeletal disease.

Family history

It is important to note any FHx of illness, especially those locomotor conditions with a heritable element:

- Osteoarthritis.
- Rheumatoid arthritis.
- Osteoporosis.

Note that the seronegative spondyloarthropathies (e.g. ankylosing spondylosis) are more prevalent in patients with the HLA B27 haplotype.

Drug history

Take a full DHx including all prescribed and over-the-counter medications. Attempt to assess the efficacy of each treatment, including all those past and present.

Ask about any side effects of any drugs taken for locomotor disease including:

- Gastric upset associated with non-steroidal anti-inflammatory drugs.
- Long-term side effects of steroid therapy such as osteoporosis, myopathy, infections and avascular necrosis.

Ask also about medication with known adverse musculoskeletal effects including:

- Statins: myalgia, myosistis, and myopathy.
- ACE-inhibitors: myalgia.
- Anticonvulsants: osteomalacia.
- Quinolone: tendinopathy.
- Diuretics, aspirin, alcohol: gout.
- Procainamide, hydralazine, isoniazid: SLE.

▸ It is also worth bearing in mind that illicit drugs may ↑ the risk of developing infectious diseases such as tuberculosis, HIV, and hepatitis, all of which can cause musculoskeletal complaints.

Smoking and alcohol

As always, full smoking and alcohol histories should be taken (p.44).

Social history

This should form a natural extension of the functional enquiry and should include a record of the patient's occupation if not already noted, as well as ethnicity.

- ***Certain occupations*** predispose to specific locomotor problems. For example, repetitive strain injury, hand-vibration syndrome, and fatigue fractures seen sometimes in dancers and athletes.
- ***Ethnicity*** is relevant as there is an overrepresentation of lupus, and TB in the Asian population, both of which are linked to a variety of locomotor complaints.

- If the patient is an older person, make a note about the activities of daily living, how mobile the patient is and if there are any home adaptations such as a chair lift or railings.
- Remember to ask about home care or other supports.
- Where appropriate, take a sexual history. This is important because reactive arthritis or Reiter's syndrome may be caused by sexually transmitted diseases such as Chlamydia and gonorrhoea.

Outline examination

A full examination of the entire locomotor system can be long and complicated.

In this chapter, we have broken the examination down into the following joints/regions: elbow, shoulder, spine, hip, knee, ankle, and foot.

Box 11.5 Examination framework

The examination of each joint should follow the standard format:

- Look.
- Feel.
- Move*.
  - Passive.
  - Active.
- Measure.
- Special tests.
- Function.

▸ In a thorough locomotor examination, you should examine the joints 'above' and 'below' the symptomatic one. For example, for an elbow complaint, also examine the shoulder and wrist.

▸ The **hand examination** is discussed in Chapter 3, p.72.

The GALS screen

The overall integrity of the locomotor system can be screened very quickly by using the 'GALS' method of assessment.

You may use this to make quick, 'screening' examination of the whole locomotor system in order to identify which joints or regions to examine in more detail.

The GALS screen consists of 4 components:

- **G** = Gait.
- **A** = Arms.
- **L** = Legs.
- **S** = Spine.

Box 11.6 Modified GALS screen

The GALS screen was devised as a quick screen for abnormality in the absence of symptoms.* With apologies to the original authors, below is our slightly modified version:

Gait

- Watch the patient walk.
  - There should be symmetry and smoothness of movement and arm swing with no pelvic tilt and normal stride length. The patient should be able to start, stop and turn quickly.

Arms (sitting on couch)

- *Inspection:* look for muscle wasting and joint deformity at the shoulders, elbows, wrists and fingers. Squeeze across the 2nd-5th metacarpals—there should be no tenderness.
- *Shoulder abduction:* 'raise your arms out sideways, above your head'. Normal range 170-180°.
- *Shoulder external rotation:* 'touch your back between your shoulder blades'.
- *Shoulder internal rotation:* 'touch the small of your back'. Should touch above T10.
- *Elbow extension:* 'Straighten your arms out'. Normal is 0°.
- *Wrist and finger extension:* the prayer sign (📖 p.75).
- *Wrist flexion and finger extension:* the reverse prayer sign (📖 p.75).
- *Power grip:* 'make a tight fist'—should hide fingernails.
- *Precision grip:* 'put your fingertips on your thumb'.

Legs (lying on couch)

- *Inspection:* Look for swelling or deformity at the knee, ankle and foot as well as quadriceps muscle wasting. Squeeze across the metatarsals—there should be no tenderness.
- *Hip and knee flexion:* test passively and actively. Normal hip flexion is 120°, normal knee flexion is 135°.
- *Hip internal rotation:* normal is 90° at 45° flexion. See 📖 p.384.
- *Knee:* bulge test (📖 p.386) and patellar tap (📖 p.386).
- *Ankle:* test dorsiflexion (normal 15°) and planarflexion (normal 55°).

Spine (standing)

- *Inspection from behind:* look for scoliosis, muscle bulk at the paraspinals, shoulders and gluteals, level iliac crests.
- *Inspection from the side:* look for normal thoracic kyphosis and lumbar and cercial lordosis.
- *Tenderness:* feel over the mid-supraspinatus—there should be no tenderness.
- *Lumbar flexion:* 'touch your toes'. Normal is finger-floor distance <15cm. Lumbar expansion (Schober's test p.383).
- Cervical lateral flexion: 'put your ear on your shoulder'.

* Doherty *et al* (1992). Annals Rheum Dis **51**:1165-9.

Elbow

Look

Look around the bed for any mobility aids or other clues. Ask the patient to stand, make sure both upper limbs are exposed and look at the patient from top to toe.

Inspect the elbow joint from the front, side, and behind, and note:

- Malalignment of the bones.
- Scars.
- Skin change (e.g. psoriatic plaques).
- Skin or subcutaneous nodules.
- Deformities.

- Varus (‘cubitus varus’): can be caused by a supracondylar fracture.
- Valgus (‘cubitus valgus’): can be caused by non-union of a lateral condylar fracture.

- Muscle wasting.
- Swelling.

Feel

▸ Always ask about pain before getting started.

Palpate the joint posteriorly and feel for:

- Temperature.
- Subcutaneous nodules.
- Swelling.
  - Soft swelling may be due to olecranon bursitis.
  - Hard swellings suggests a bony deformity.
  - Boggy swelling suggests synovial thickening (e.g. secondary to RA).
- If fluid is present, attempt to displace it on either side of the olecranon.
- Carefully palpate the joint margin for tenderness and note if it is localized to the medial epicondyle (golfer's elbow) or the lateral epicondyle (tennis elbow).

Move

Check that there is good shoulder function before attempting to assess elbow movements.

▸ Remember to test passive movements (you do the moving) and active movements (the patient does the moving) at each stage.

- Ask the patient to place their arms on the back of their head.
- Next assess elbow flexion and extension with the upper arm fixed.
  - Remember to compare with the opposite side.
- With the elbows tucked into the sides and flexed to a right angle, test the radio-ulnar joints for pronation (palms towards floor) and supination (palms towards the sky).

Measure

Measure elbow flexion and extension in degrees from the neutral position (i.e. consider a straight elbow joint to be 0°).

Function

Observe the patient pour a glass of water and then put on a jacket.

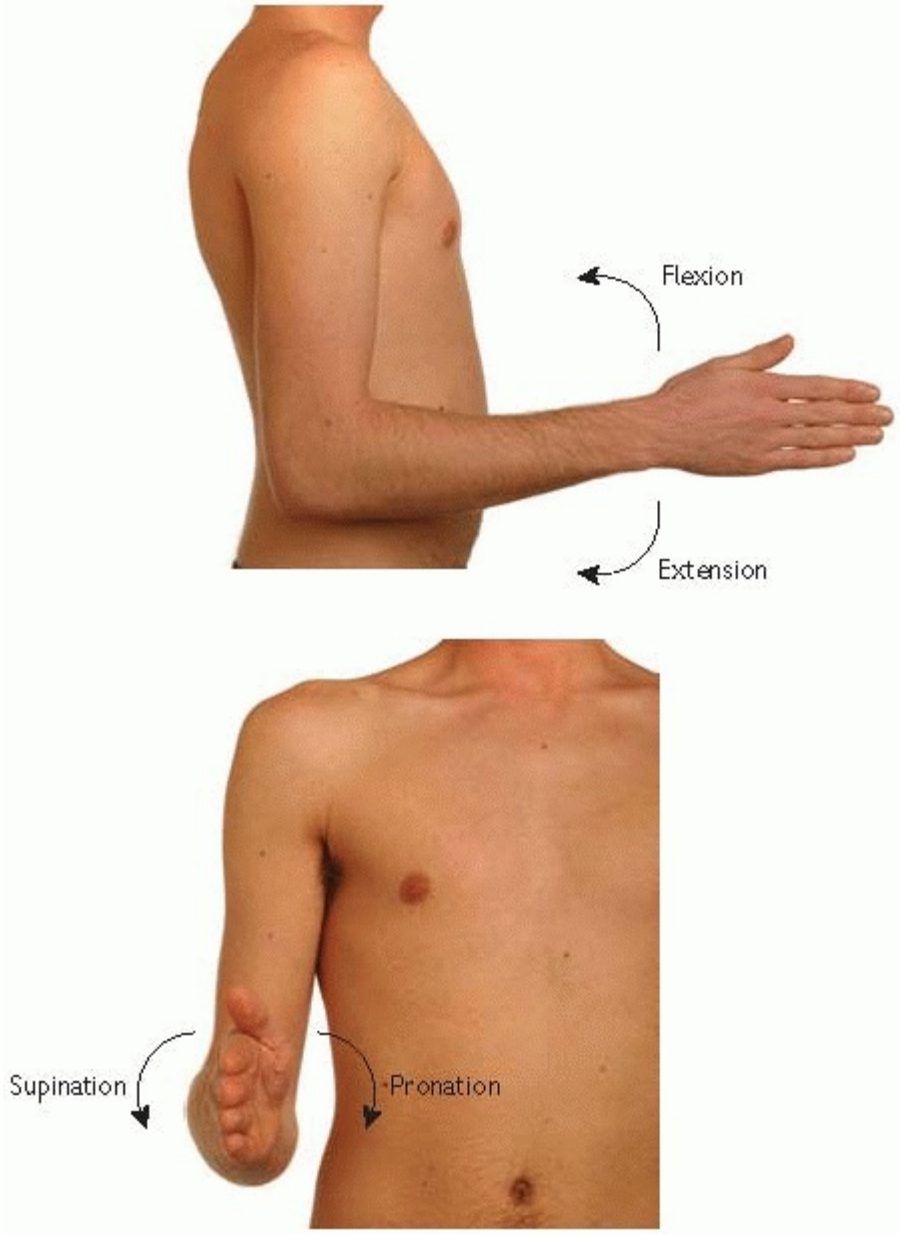

Fig. 11.2 Movements at the elbow joint. (a) Flexion and extension. (b) Pronation and supination (remember that pronation and supination require movement at the elbow as well as at the wrist).

Shoulder

Look

Look around for any aids or adaptations. Ask the patient to remove any covering clothing and expose both upper limbs, the neck, and chest. Scan the patient from top to toe. Inspect from the front, side, and behind.

Look especially for:

- Contours.
  - Make note of any obvious asymmetry or deformity such as winging of the scapula, prominence of the acromioclavicular joint and wasting of the deltoid or short rotators which overlie the upper and lower segments of the scapula.
- Joint swelling.
  - This is more obvious from the front and may be a clue to acute bleeds, rheumatoid effusions, pseudogout, or sepsis.
- Scars.
- Bruising or other skin/subcutaneous tissue changes.
- The position of both shoulders looking for evidence of dislocation.

- Posterior dislocation can be seen when the arm is held in an internally rotated position.
- Anterior dislocation can be seen easily with the arm is displaced in a forward and downward position.

- ▸ Remember to inspect the axillary regions.

Feel

▸ Always ask about pain before getting started.

Make note of any temperature changes, tenderness, or crepitus. Standing in front of the patient:

- Palpate the soft tissues and bony points in the following order: sternoclavicular joint, clavicle, acromioclavicular joint, acromial process, head of humerus, coracoid process, spine of scapula, greater tuberosity of humerus.
- Check the interscapular area for pain.
- Palpate the supraclavicular area for lymphadenopathy.

Move

▸ Remember to test passive movements (you do the moving) and active movements (the patient does the moving) at each stage. Quantify any movement in degrees (*measure*).

To test true glenohumeral movement, anchor the scapula by pressing firmly down on the top of the shoulder. After about 70° of abduction, the scapula rotates on the thorax—movement is scapulothoracic.

- ***Flexion:*** ask the patient to raise their arms forwards above their head.
- ***Extension:*** straighten the arms backwards as far as possible.
- ***Abduction:*** move the arm away from the side of the body until the fingertips are pointing to the ceiling.
- ***Adduction:*** ask the patient to move the arm inwards towards the opposite side, across the trunk.
- ***External rotation:*** with the elbows held close to the body and flexed to 90°, ask the patient to move the forearms apart in an arc-like motion in order to separate the hands as widely as possible.

- ***Internal rotation:*** ask the patient to bring the hands together again across the body. (Loss of rotation suggests a capsulitis.)
- ***Compound movements:*** these types of movements may be employed as screening tests to assess shoulder dysfunction, taking the place of a fuller examination if no abnormalities are detected. See Fig. 11.3.
  - Ask the patient to put both hands behind the head (external rotation in abduction).
  - Ask the patient to reach up their back with the fingers to touch a spot between their shoulder blades (internal rotation in adduction).

Special tests

Testing for a rotator cuff lesion/tendonitis: 'the painful arc'

Ask the patient to abduct the shoulder against light resistance.

Pain in early abduction suggests a rotator cuff lesion and usually occurs between 40°-120°. This is due to a damaged and inflamed supraspinatus tendon being compressed against the acromial arch.

Testing for acromioclavicular arthritis

If there is pain during a high arc of movement (starting around 90°) and the patient is unable to raise their arm straight up above their head to 180°, even passively, this is suggestive of acromioclavicular arthritis.

Function

Ask the patient to scratch the centre of their back or to put on a jacket.

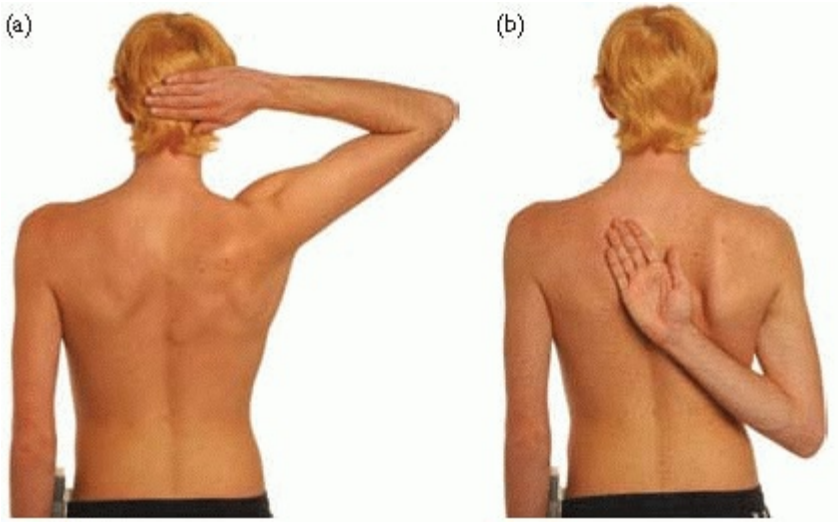

Fig. 11.3 Compound movements. (a) External rotation and abduction. (b) Internal rotation and adduction.

Box 11.7 A word about winging of the scapula

This arises due to weakness of serratus anterior as a result of damage to the long thoracic nerve, injury to the brachial plexus, injury or viral infections of C5-7 nerve roots and muscular dystrophy.

Winging only becomes obvious when the serratus anterior contracts against resistance such as pushing outstretched hands against a wall.

Spine

Look

Scan around the room for any clues such as a wheelchair or walking aids. Watch how the patient walks into the room or moves around the bed area. Study their posture, paying particular attention to the neck.

Ask the patient to strip down to their underwear. Inspect from in front, the side and behind in both the standing and sitting positions.

Look especially for:

- Scars.
- Pigmentation.
- Abnormal hair growth.
- Unusual skin creases.
- Asymmetry including abnormal spinal curvature:
  - *Kyphosis:* (convex curvature—normal in the T-spine).
  - *Lordosis:* (concave curvature—normal in the L- and C-spines).
  - *Scoliosis:* (side-to-side curvature away from the midline).

▸ A ‘question mark’ spine with exaggerated thoracic kyphosis and a loss of lumbar lordosis is classic of ankylosing spondylitis.

Feel

Palpate each spinous process noting any prominence or step and feel the paraspinal muscles for tenderness.

You should also make a point of palpating the sacroiliac joints.

Move

C-spine

Assess active movements of the cervical-spine first. These include flexion, extension, lateral flexion, and rotation. It is often helpful to demonstrate these movements yourself.

- *Flexion:* ask the patient to put their chin on their chest.
- *Extension:* ask the patient to look up to the ceiling.
- *Lateral flexion:* ask the patient to lean their head sideways, placing an ear on their shoulder.
- *Rotation:* ask the patient to look over each shoulder.

T- and L-spine

Movements at the thoracic and lumbar spine include flexion, extension, lateral flexion, and rotation.

- *Flexion:* ask the patient to touch their toes.
- *Extension:* ask the patient to lean backwards.
- *Lateral flexion:* ask the patient to bend sideways, sliding each hand down their leg as far as possible.
- *Rotation:* anchor the pelvis (put a hand on either side) and ask the patient to twist at the waist to each side in turn.

Measure

Schober's test

This is useful measurement of lumbar flexion.

- Ask the patient to stand erect with normal posture and identify the level of the posterior superior iliac spines on the vertebral column.
- These are located at ~L5.
- Make a small pen mark at the midline 5cm below and 10cm above this point.
- Now instruct the patient to bend at the waist to full forward flexion.
- Measure the distance between the 2 marks using a tape measure.
- The distance should have increased to >20cm (an increase of >5cm). If not, there is a limitation in lumbar flexion (e.g. found in ankylosing spondylitis).

Special tests

Sciatic nerve stretch test

- This test is used to look for evidence of nerve root irritation.
- With the patient lying supine, hold the ankle and lift the leg, straight, to 90°. Once there, dorsiflex the foot (Bragard test). If positive, pain will be felt at the back of the thigh. The pain may be relieved by knee flexion
- A positive stretch test suggests tension of the nerve roots supplying the sciatic nerve (L5-S2), commonly over a prolapsed disc (L4/5 or L5/S1).
- This test is partially age dependent. Most elderly people will struggle to flex their hip beyond 70°.

Femoral nerve stretch test

With the patient lying prone, abduct and extend the hip, flex the knee and plantar-flex the foot. The stretch test is positive if pain is felt in the thigh/inguinal region. See p.348.

Box 11.8 Neurological examination

Don't forget the neurological aspects of the examination. The femoral and sciatic stretch tests may uncover root irritation—but you should also examine for the neurological and functional consequences as in Chapter 10.

Hip

Look

Expose the whole lower limb. Look around the room for any aids or devices such as orthopaedic shoes or walking aids. If they have not done so already, ask the patient to walk and note the gait. Note if there is evidence of a limp or obvious pain.

With the patient in the standing position, inspect from the front, side, and behind. Look for:

- Scars.
- Sinuses.
- Asymmetry of skin creases.
- Swelling.
- Muscle wasting.
- Deformities.

Pay attention to the position of the limbs (e.g. external rotation, pelvic tilting, standing with one knee bent or foot held plantarflexed/in equinus).

Feel

Feel for bony prominences such as the anterior superior iliac spines and greater trochanters. Check that they are in the expected position.

Palpate the soft tissue contours and feel for any tenderness in and around the joint.

Move

Ask the patient if they have any pain before examining.

▸ Fix the pelvis by using your left hand to stabilize the contralateral anterior superior iliac spine since any limitation of hip movement can easily be hidden by movement of the pelvis.

With the patient *supine:*

- ***Flexion:*** ask the patient to flex the hip until the knee meets the abdomen, normal is around ~120°.
- ***Abduction:*** with the patient's leg held straight, ask them to move it away from the midline, normal is 30-40°.
- ***Adduction:*** with the patient's leg held straight, ask them to move it across the midline, normal is ~30°.

With the patient *prone:*

- ***Extension:*** asking the patient to raise each leg off the bed, normal is only a few degrees.
- ***Internal rotation:*** ask the patient to keep the knees tight together and spreading the ankles as far as possible.
- ***External rotation:*** ask the patient to cross the legs over.

Passive movements

Most should be assessed by the examiner as for active movements whilst the patient is in a relaxed state.

- *Passive external and internal rotation:* with the patient supine, flex the knee, stabilizing it with one hand whilst the other hand moves the heel laterally or medially so that the heel either moves away or towards the midline (internal and external rotation respectively).

Measure (limb length)

 True shortening, in which there is loss of bone length, must not be confused with apparent shortening due to a deformity at the hip, in which there is no loss of bone length.

Technique

- With the patient supine, place the pelvis square and the lower limbs in comparable positions in relation to the pelvis.
- Measure the distance from the anterior superior iliac spine to the medial malleolus on each side *(true length)*.
    - *Apparent length* is measured from a midline structure such as the xiphisternum to the medial malleolus.

Special tests

Trendelenberg test

This is useful as an overall assessment of the function of the hip and will expose dislocations or subluxations, weakness of the abductors, shortening of the femoral neck, or any painful disorder of the hip.

- Ask the patient to stand up straight without any support.
- Ask them to raise their left leg by bending the knee.
- Watch the pelvis (should normally rise on the side of the lifted leg).
- Repeat the test with the patient standing on the left leg.
- A *positive test* is when the pelvis falls on the side of the lifted leg indicating hip instability on the supporting side (i.e. the pelvis falls to the left = right hip weakness).

Thomas's test

A fixed flexion deformity of the hip (often seen in osteoarthritis) can be hidden when the patient lies supine by tilting the pelvis and arching the back. The Thomas test will expose any flexion deformity.

- With the patient lying supine, feel for a lumbar lordosis (palm upwards).
- With the other hand, flex the opposite hip and knee fully to ensure that the lumbar spine becomes flattened.
- If a fixed flexion deformity is present, the opposite leg flexes too (measure the angle relative to the bed).
- Remember to repeat the test on the other hip.

Function

Assess gait. See  p.350.

Knee

Look

Scan the room for any walking aids or other clues and inspect the patient standing. The lower limbs should be completely exposed except for underwear so that comparisons can be made.

Compare one side to the other and look for:

- Deformity (valgus, varus, or flexion).
- Scars or wounds to suggest infection past or present?
- Muscle wasting (quadriceps).
- Swelling (including posteriorly).
- Erythema.
- Look for loss of the medial and lateral dimples around the knees which suggest the presence of an effusion.

Feel

▸ Always ask about pain before getting started. Always compare sides. With the patient lying supine:

- Palpate for temperature using the back of the hand.

- Ask if the knee is tender on palpation.
- Feel around the joint line while asking the patient to bend the knee slightly.
- Palpate the collateral ligaments (either side of the joint).
- Feel the patellofemoral joint (by tilting the patella).

Examining for a small effusion—the 'bulge sign'

- Holding the patella still, empty the medial joint recess using a wiping motion of your index finger (Fig. 11.4a).
  - This will milk any fluid into the lateral joint recess.
- Now apply a similar wiping motion to the lateral recess and…
- Watch the medial recess (Fig. 11.4b).
  - If there is fluid present, a distinct bulge should appear on the flattened, medial surface and it is milked out of the lateral side.

Examining for a large effusion—the 'patellar tap'

If the effusion is large, the bulge sign is absent as you will be unable to empty either recess of fluid—use the patellar tap instead.

- Move any fluid from the medial and lateral compartments into the retropatellar space.
  - Apply firm pressure over the suprapatellar pouch with the flat of the hand and use your thumb and index finger placed either side of the patella to push any fluid centrally (see Fig. 11.5a).
- With the first one or two fingers of the other hand, push the patella down firmly (see Fig. 11.5b).
- If fluid is present, the patella will bounce off the lateral femoral condyle behind. You will feel it being pushed down and then 'tap' against the femur.

Move

▸ Remember to test passive movements (you do the moving) and active movements (the patient does the moving) at each stage. Quantify any movement in degrees (*measure*).

- Begin by the moving the joint passively and feel over the knee with one hand for any crepitus.
- *Flexion:* ask the patient to maximally flex the knee, normal ≈135°.
- *Extension:* ask the patient to straighten the leg at the knee.
- *Hyperextension:* assess by watching the patient lift the leg off the bed and then, holding the feet stable in both hands above the bed/couch and ask the patient to relax Ensure that you are not causing the patient any discomfort.

Measure

The visual impression of wasting of the quadriceps can be confirmed by measuring the circumference of the thighs at the same level using a fixed bony point of reference e.g. 2.5cm above the tibial tubercle.

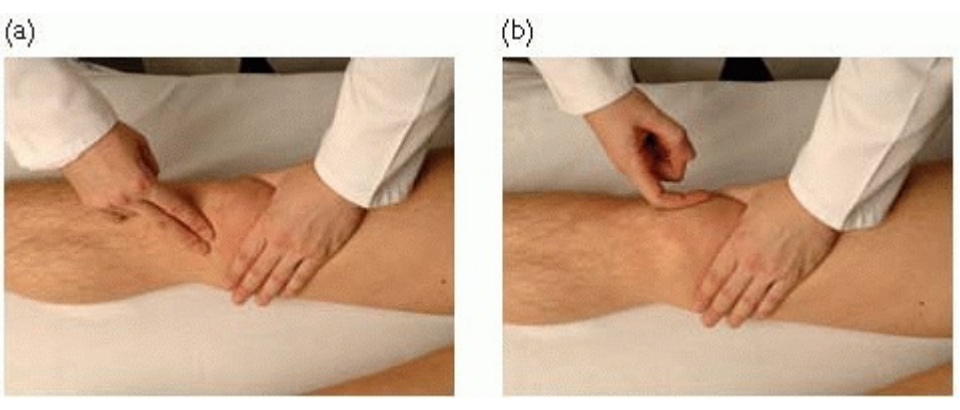

Fig. 11.4 Examining for the ‘bulge sign’. (a) Wipe any fluid from the medial joint recess. (b) Wipe the fluid back out of the lateral joint recess and watch the medial side.

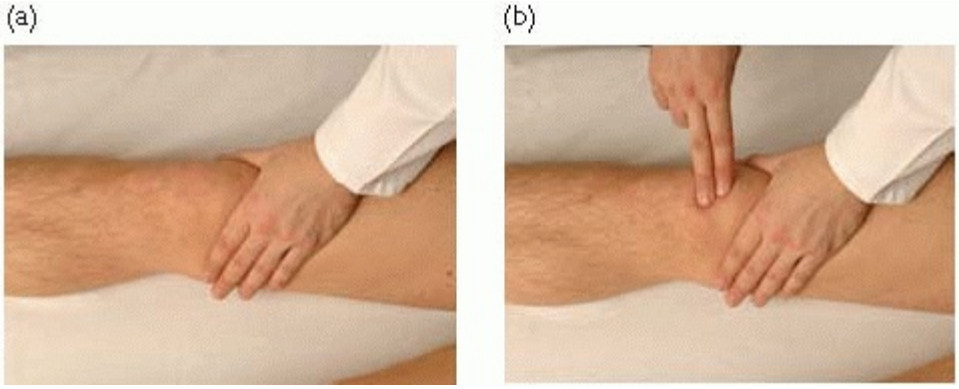

Fig. 11.5 Testing for ‘patellar tap’. (a) Use the palmar surface, thumb and index finger of one hand to move any fluid into the retropatellar space. (b) Attempt to ‘tap’ the patella on the femur using the other hand.

Special tests

Testing for medial and lateral collateral ligament instability

- Take the patient's foot under your right upper arm.
- Hold the patient's extended knee firmly with both hands.
- Attempt to bend the distal leg medially (varus).
  - This tests the lateral collateral ligament.
- Attempt to bend the distal leg laterally (valgus).
  - This tests the medial collateral ligament.
- Repeat the above with the knee at 30° of flexion.
  - Normally, the joint should move no more than a few degrees, excessive movement suggests a torn or stretched collateral ligament.

Anterior and posterior drawer tests

These test the anterior and posterior cruciate ligaments. These ligaments prevent the distal part of the knee moving anteriorly and posteriorly ‘drawer tests’.

- Ensure the patient is lying in a relaxed supine position.
- Ask the patient to flex the knee to 90°.
- You may wish to position yourself perched on the patient's foot to stabilize the leg. Warn the patient about this first!
- Wrap your fingers around the back of the knee using both hands, positioning the thumbs over the patella pointing towards the ceiling.
- Push up with your index fingers to ensure the hamstrings are relaxed.
- The upper end of the tibia is then pulled forwards and pushed backwards in a rocking motion.
  - Normally, there should be very little or no movement seen.
  - Excessive anterior movement reflects anterior cruciate laxity.

- Excessive posterior movement denotes posterior cruciate laxity.

McMurray's test

A test for meniscal tears.

- With the patient lying supine, bend the hip and knee to 90°.
- Grip the heel with your right hand and press on the medial and lateral cartilage with your left hand.
- Internally rotate the tibia on the femur and slowly extend the knee.
- Repeat but externally rotate the distal leg whilst extending the knee.
- Repeated with varying degrees of knee flexion.
    - If there is a torn meniscus, a tag of cartilage may become trapped between the articular surfaces, and cause pain and an audible click. You may also be able to feel the click with your left hand.

Apley's test

Another test for meniscal tears.

- Position the patient *prone* with the knee flexed to 90°.
- Stabilize the thigh with your left hand.
- With the right hand, grip the foot.
- Rotate or twist the foot and press downwards in a ‘grinding motion’.
    - This test should produce symptoms if a meniscus is torn.

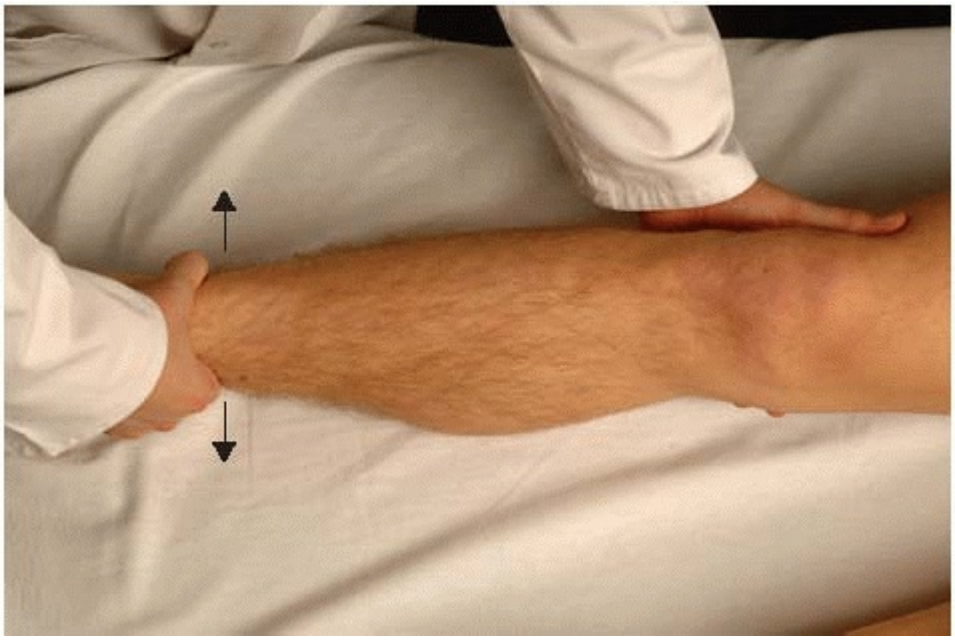

Fig. 11.6 Testing collateral ligament stability.

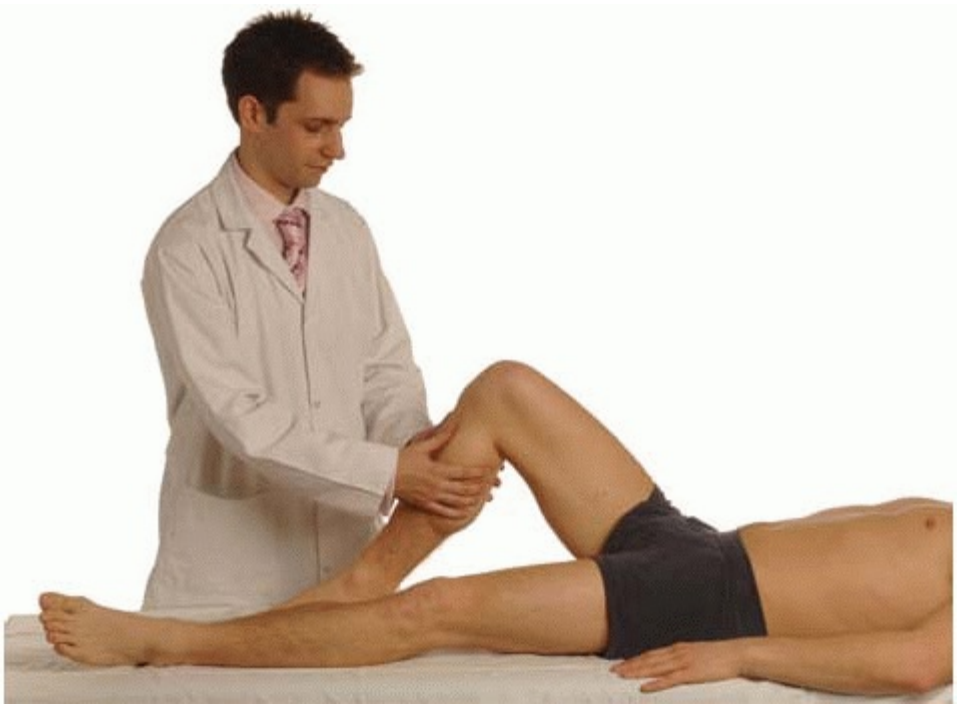

Fig. 11.7 Performing the anterior drawer test.

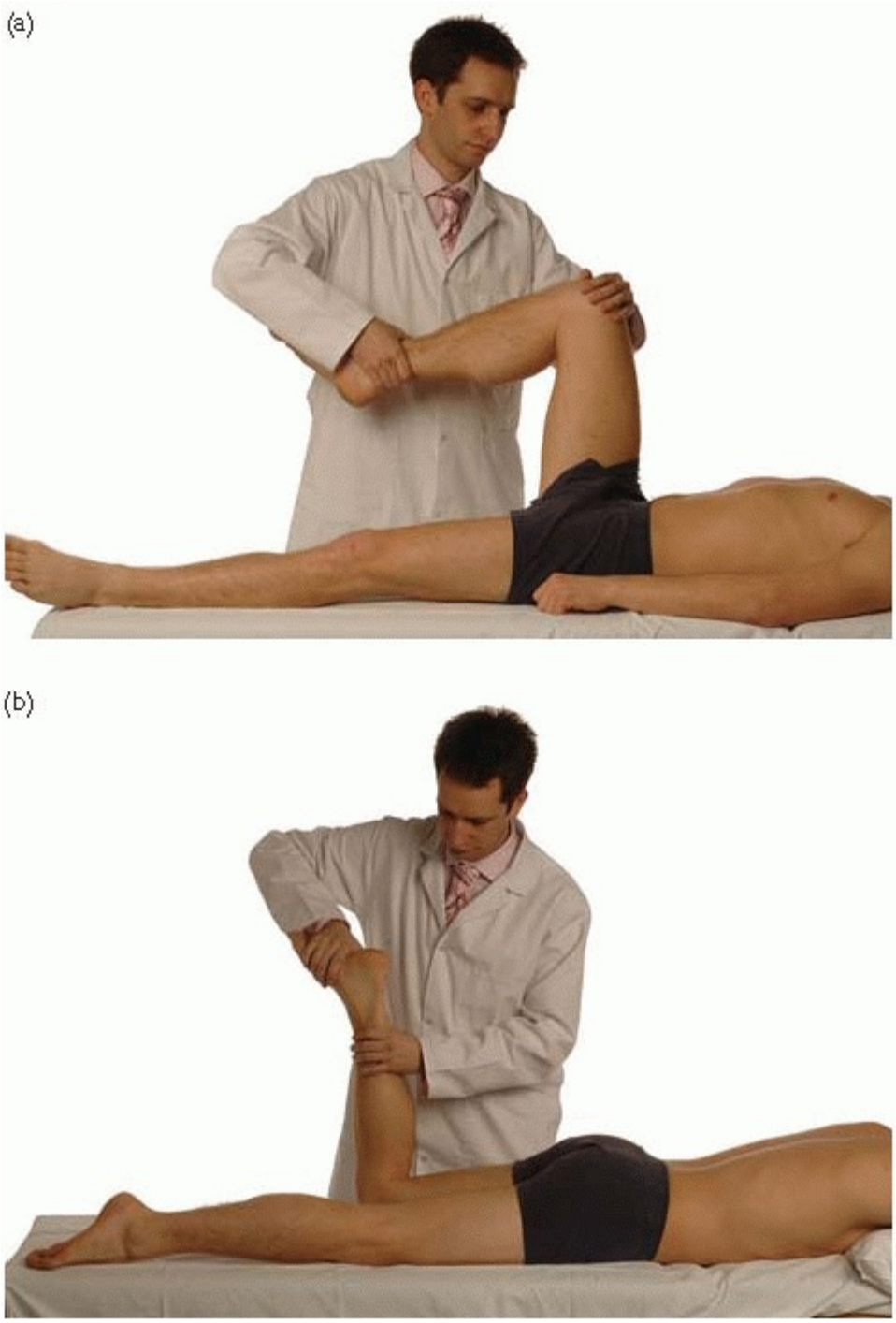

Fig. 11.8 Testing for meniscal tears. (a) McMurrays' test. (b) Apley's grinding test.

Ankle and foot

Look

Expose the lower limbs and make a note of any walking or other aids present. Take a moment to also examine the shoes carefully for any abnormal wear or stretching.

Examine the feet and ankles both when the patient is standing and, more carefully, with the patient lying on a couch or bed. Look for:

- Skin or soft tissue lesions including calluses, swellings, ulcers and scars.
- Muscle wasting at the calf and lower leg.
- Deformities, especially involving the arch.
  - Pes planus (flat foot).
  - Pes cavus (high-arched foot).

- Look for a bunion (bony deformity) at the 1st MTP joint.
- Look for a bunionette at the 5th MTP joint.
- Examine the nails carefully for any abnormalities such as fungal infections or in-growing toenails. ▸ Don't forget to look between the toes.

You may also wish to inspect for evidence of other abnormalities such as hammer toes, claw toes or clubbing of the feet (talipes equinovarus).

Feel

▸ Always ask about pain before getting started.

- Assess the skin temperature and compare over both the feet.
- Look for areas of tenderness, particularly over bony prominences (lateral and medial malleoli, MTP joints, interphalangeal joints and heel) as well as the metatarsal heads.
- Squeeze across the MTP joints and assess pain and movement.
- Remember to palpate any swelling, oedema, or lumps.

Move

The ankle and foot is a series of joints which function as a unit.

▸ Remember to test passive movements (you do the moving) and active movements (the patient does the moving) at each stage.

Active movements should be performed with the patient's legs hanging over the edge of the bed.

- ***Ankle dorsiflexion:*** ask the patient to point their toes at their head.
- ***Ankle planarflexion:*** ask the patient to push the toes down towards the floor 'like pushing on a pedal'.
- ***Inversion:*** (subtalar joint between the talus and calcaneum). Grasp the ankle with one hand and with the other, grasp the heel, thereby fixing the calcaneum and turn the sole inwards towards the midline.
- ***Eversion:*** as inversion but turn the sole outwards, away from the midline.
- ***Midtarsal joints:*** grasp the heel with one hand and attempt to move the tarsus up and down and from side to side with the other.
- ***Toe flexion:*** ask the patient to curl their toes.
- ***Toe extension:*** ask the patient to straighten the toes.
- ***Toe abduction:*** ask the patient to fan out their toes as far as possible.
- ***Toe adduction:*** ask the patient to hold a piece of paper between their toes.

Measure

Calf circumference can be measured bilaterally to check for any discrepancies which may highlight muscle wasting/hypertrophy. (e.g. 10cm below the tibial tuberosities).

Special tests

Simmond's test

This test is used to assess for a ruptured Achilles tendon.

- Ask the patient to kneel on a chair with their feet hanging over the edge. Squeezes both calves
- Normally the feet should plantarflex. If the Achilles tendon is ruptured, there will be no movement on the affected side.

Function

It is also helpful to observe the patient's gait with and without shoes. Be sure to ask the patient if they are able to do this.

Box 11.9 A word on inversion and eversion

Orthopaedic purists will say that the ‘ankle’ cannot invert or evert as it is mainly a simple hinge—the eversion and inversion tests are, therefore, a ‘failure only’ test. You should note that some eversion and inversion is possible in the normal state at the tarsal joints—as tested by the neurologists.

Orthopaedic practitioners test pathological inversion and eversion by watching the heels from behind as the patient stands on tip-toes.

Important presenting patterns

Rheumatoid arthritis (RA)

RA is a chronic inflammatory multisystem autoimmune disease mediated by pro-inflammatory cytokines such as tumour necrosis factor alpha (TNFα) and in some cases is characterized by the presence of the rheumatoid factor.

There is a strong association with HLA-DR4 and patients with DR4 tend to have more severe disease. It affects around 1-3% of the population in all racial groups, with peak age of onset in the 4^{th} and 5^{th} decades and a female:male ratio of 3:1.

The usual pattern of disease is insidious but can also be episodic with complete resolution between attacks (palindromic) or acute. The clinical features of RA can be divided into articular and extra-articular features and are summarized below.

Articular features

RA usually presents as a symmetrical polyarthritis affecting the wrists and small joints of the hands and feet. Occasionally, a patient presents with a monoarthritis of a larger joint such as the knee or shoulder. Common presenting symptoms are joint pain, stiffness, and swelling which are typically worse in the mornings and improves as the day progresses. The disease eventually leads to varying degrees of functional loss.

Signs of RA in the hands/wrist

Synovitis involving the wrists, metacarpo-phalangeal and proximal interphalangeal joints with sparing of the distal interphalangeal joints.

- Ulnar deviation of fingers (subluxation/dislocation at the MCP joints).
- Swan neck deformity: hyperextension of PIP joints with flexion of MCP and DIP joints.
- Boutonniere's deformity: flexion deformity of PIP joints with extension of DIP and MCP joints.
- Z deformity of thumb: flexed MCP joint with extended interphalangeal joint of thumb.
- Triggering of finger.
- Generalized wasting of small muscles of hand.
- Cutaneous vasculitis.

Signs of RA in the feet

- Forefoot synovitis with proximal phalangeal subluxation dorsally.
- Metatarsal head erosion and displacement towards the floor. Patient feels it ‘like walking on marbles’.
- Valgus deformities.
- Collapse of longitudinal arch.

Signs of RA in the spine

Atlanto-axial subluxation ± spinal cord compression

Extra-articular features of RA

- Rheumatoid nodules: common at sites of pressure (elbows and wrists). Associated with more severe disease and always rheumatoid factor positive.
- Tenosynovitis and bursitis.
- Carpal tunnel syndrome.
- *Anaemia:* causes include:

- Anaemia of chronic disease.
- GI bleeding associated with NSAID use.
- Bone marrow suppression secondary to disease-modifying anti-rheumatic drugs such as gold and penicillamine.
- Megaloblastic anaemia from folic acid deficiency (also secondary to methotrexate) or pernicious anaemia.
- Felty's syndrome (RA, splenomegaly, and leucopenia).

- *Lung features:*
  - Pleuritic pain.
  - Pleural effusions.
  - Pulmonary fibrosis.
  - Pulmonary nodules.
  - Obliterative bronchiolitis.
  - Caplan's syndrome (massive lung fibrosis in RA patients with pneumoniosis).
- *Neurological features:*
  - Peripheral nerve entrapment.
  - Mononeuritis multiplex.
  - Peripheral neuropathy.
  - Cervical myelopathy due to atlanto-axial subluxation.
- *Cardiac features:*
  - Pericarditis.
  - Pericardial effusions.
- *Eye features:*
  - Painless episcleritis.
  - Painful scleritis.
  - Scleromalacia perforans.
  - Keratoconjunctivitis sicca.
  - Sjogren's syndrome.
  - Cataracts (chloroquine, steroids).
- *Vasculitis:*
  - Nail fold infarcts.
  - Cutaneous ulceration.
  - Digital gangrene.
  - Cerebral and mesenteric infarction.
  - Coronary and renal vasculitis (rare).
- *Skin lesions:*
  - Palmar erythema.
  - Pyoderma gangrenosum.
- Amyloidosis (proteinuria, hepatosplenomegaly).
- Systemic features (fever, malaise, weight loss, and lymphadenopathy).

Osteoarthritis

Osteoarthritis is a chronic disorder of synovial joints characterized by focal cartilage loss and an accompanying reparative bone response.

It represents the single most important cause of locomotor disability with a prevalence which ↑ with age and a female preponderance.

The joints commonly affected include the hips, knees, spine, 1st carpometacarpal, 1st metatarsal, and distal interphalangeal joints.

Secondary causes include trauma, RA, infection, neuropathic (Charcot's) joints, and metabolic (e.g. Paget's disease, acromegaly, haemachromatosis, avascular necrosis, and hypoparathyroidism).

Clinical features

Common symptoms include swelling, deformity, stiffness, weakness, and pain which is normally worse after activity and relieved by rest.

Common signs include:

- Hard, bony swellings of the DIP joints (Heberden's nodes).
- Bony nodules at the PIP joints (Bouchard's nodes).
  - These are bony outgrowths at the joint margins (osteophytes).
- 'Square hand deformity' due to subluxation of the base of the thumb.
- Valgus and varus deformities.
- Crepitus.
- Wasting and weakness (especially of the quadriceps and glutei).
- Tilting of the pelvis.

Crystal arthropathies

Gout

A disorder of purine metabolism. Characterized by hyperuricaemia due to either overproduction or underexcretion of uric acid. Prolonged hyperuricaemia leads to the formation of urate crystals in the synovium, other connective tissues and the kidney.

Clinical features of acute gout

- Severe pain and swelling classically in the great toe MTP joint, worse at night and associated with redness.
- Occasionally multiple joints are involved.
- ± Systemic symptoms.

Clinical features of chronic (tophaceous) gout

- Tophus formation (soft tissue deposits of urate found especially in digits, helix of the ear, bursae and tendon sheaths).
  - ± Overlying necrotic skin with chalky exudate of urate crystals.

Pseudogout

Caused by deposition of calcium pyrophosphate crystals in the synovium, joint capsule and tendons. It is the commonest cause of an acute monoarthritis in the elderly and may present as either an acute synovitis or as a chronic arthritis.

Linked to chondrocalcinosis (calcification of articular cartilage). On examination, you may find a swollen, erythematous and tender joint (often knees, wrist, elbow, ankle, or shoulder and MCP joints especially the index and middle) associated with systemic upset.

Box 11.10 Causes of hyperuricaemia

- Drugs: diuretics, ethanol, low dose salicylates, pyrazinamide, ethambutol, nicotinic acid and ciclosporin).
- Chronic renal failure.
- Myeloproliferative and lymphoproliferative disorders (↑ purine metabolism).
- Obesity.
- Hypertension.
- Hypothyroidism.
- Hyperthyroidism.
- Familial.
- Excessive dietary purines.
- ▸ More common in the summer months due to reduced fluid intake and increased fluid loss.

Box 11.11 Conditions associated with gout

- Obesity.
- Type IV hyperlipidaemia.
- Hypertension.
- Impaired glucose tolerance/diabetes.
- Ischaemic heart disease.

Spondyloarthropathies

These include ankylosing spondylitis, psoriatic arthritis, reactive arthritis, and enteropathic arthritis. This is a group of related and overlapping forms of inflammatory arthritis which characteristically lack rheumatoid factor and are associated with HLA-B27. They present at any age, though young males are primarily affected.

They also share a number of key features which are:

- Enthesitis (an enthesis is the insertion of a tendon, ligament or capsule into a bone).
- Synovitis.
- Sacroiliitis.
- Dactylitis.
- Peripheral arthritis predominantly affecting the large joints.

Ankylosing spondylitis

Ankylosing spondylitis usually develops in early adulthood with a peak age of onset in the mid 20s and is 3 times more common in males.

Common symptoms

- Lower back pain and stiffness which is typically worse in the morning and after long periods of rest.
- Chest pain as a result of T-spine involvement as well as enthesitis at the costochondral joints.
- Tender sacro-iliac joints.
- Pain in peripheral joints such as the shoulders and knees.

Musculoskeletal features/signs

- ‘Question mark’ posture (loss of lumbar lordosis, fixed kyphoscoliosis of the T-spine, compensatory extension of the C-spine).
- Protuberant abdomen.
- Schober's test positive (see p.383).
- Achilles tendonitis.
- Plantar fasciitis.

Some extra-skeletal features

- Anterior uveitis.
- Aortic regurgitation.
- Apical lung fibrosis.
- AV block.
- Amyloidosis (secondary).

- Atlantoaxial dislocation.
- Traumatic fracture of a rigid spine.
- Hypoxia.
- Fever.
- Weight loss.

Psoriatic arthritis

Psoriatic arthropathy affects up to 10% of patients with psoriasis and may precede or follow the skin disease.

▸ Importantly the arthropathy does *not* correlate with the severity of the skin lesions.

There are 5 main subtypes of psoriatic arthropathy:

- Asymmetrical distal interphalangeal joint arthropathy.
- Asymmetrical large joint mono- or oligoarthropathy.
- Spondyloarthropathy and sacroiliitis.
- Rheumatoid-like hands (clinically identical to RA but seronegative).
- Arthritis mutilans (a severely destructive form with telescoping of the fingers).

Associated clinical features

- Psoriatic plaques (classically found on the extensor surfaces, scalp, behind the ears, in the navel and natal cleft).
- Nail involvement (pitting, onycholysis, discoloration, and thickening).
- Dactylitis (sausage-shaped swelling of the digits due to tenosynovitis).

Reactive arthritis

- An aseptic arthritis, strongly linked to a recognized episode of infection. Common causes are gut and genitourinary pathogens.
- It mainly affects young adults and usually presents with an asymmetric and oligoarticular arthritis with symptoms starting a few days to a few weeks after the infection.
- Enthesitis and dactylitis are other common features and patients may experience pain in the articular joints.

Associated extra-skeletal features

- Urethritis.
- Conjunctivitis.
- Skin and mucosal lesions.

Reiter's syndrome

A form of reactive arthritis associated with the classic triad of:

- Arthritis.
- Urethritis.
- Conjunctivitis.

It often follows dysenteric infections such as Shigella, Salmonella, Campylobacteria, and Yersinia or infections of the genital tract. Other findings which may be encountered are mouth ulceration, circinate balanitis, keratoderma blennorrhagica (pustular-like lesions found on the palms or soles) and persistent plantar fasciitis.

Enteropathic arthritis

Enteropathic arthritis is a peripheral or axial arthritis occurring in association with inflammatory bowel disease and does not typically correlate with the severity of bowel disease. However, the peripheral arthritis has been shown to improve if the affected bowel is resected.

Osteoporosis

Osteoporosis is a systemic skeletal disorder involving ↓ bone mass (osteopenia) and micro-architectural deterioration, resulting in an ↑ risk of fracture. Classification (and treatment) is based on measurement of the bone mineral density (BMD), with comparision to that of a young healthy adult. See  *OHCM6*, p.698.

The underlying pathology is related to an imbalance between the osteoblast cells producing bone and the osteoclast cells removing bone which ultimately produces a net loss of bone.

- *Type I:* accelerated (mainly trabecular) bone loss secondary to oestrogen deficiency and leads to fracture of vertebral bodies as well as the distal forearm in women in their late 60s and 70s.
- *Type II:* age-related cortical and trabecular bone loss occurring in both sexes and leads to fractures of the proximal femur in the elderly.

Clinical features

The process leading to established osteoporosis is asymptomatic and the condition usually presents only after bone fractures.

Features differ according to the fracture site. The most common clinical features include:

- Marked kyphosis.
- Loss of height.
- Protuberant abdomen.
- Spinal tenderness.

Paget's disease

A disorder of bone remodelling characterized by ↑ osteoclastic and osteoblastic cell activity, leading to accelerated but disorganized bone resorption and formation.*

Paget's disease is the 2nd commonest disease of bone after osteoporosis, is more common in males and affects around 3% of the population >40 years of age. It occurs more commonly in Britain than anywhere else in the world and there are thought to be up to one million sufferers in the UK. The exact aetiology remains unknown, however, a number of factors have been implicated, including a slow viral infection. 30% of Paget's patients have an affected 1st degree relative.

Important clinical features and complications:

- Enlargement of the skull.
- Hearing loss (ossicles are involved and VIII nerve compression).
- Optic atrophy and angioid streaks.
- Cardiac failure.
- Kyphosis, anterior bowing of the tibia, lateral bowing of the femur.
- ↑ bone warmth.
- ↓ mobility.
- Fractures.
- Sarcomatous change (rare).
- Cord compression.
- Cerebellar signs.
- Hypercalcaemia.

Box 11.12 Risk factors for osteoporosis

- Smoking.
- High alcohol consumption.
- BMI <19.
- FHx.
- Premature menopause.
- Prolonged immobilization.
- Prolonged secondary amenorrhoea.
- Primary hypogonadism.
- Low dietary calcium and vitamins.
- Older age.
- Female gender.
- Sedentary lifestyle.
- Caucasian or Asian origin.
- Chronic disorders such as:
  - Anorexia nervosa.
  - Malabsorption syndromes.
  - Primary hyperthyroidism.
  - Post transplantation.
  - Cushing's syndrome.
  - Chronic renal failure.

Box 11.13 Some important causes of a swollen knee

- RA.
- Ruptured Baker's cyst.
- Pseudogout.
- Gout.
- Oedamatous states (e.g. CCF, nephrotic syndrome).
- Trauma.
- Charcot's knee.
- Septic arthritis.
- Haemarthrosis.

The elderly patient

Rheumatological diseases represent a huge spectrum of illness in older people, often complicating and concurrent with other diseases—e.g. the impact of severe arthritis on COPD or heart failure or the effect of hip or knee arthritis on recovery after acute stroke. Arthritis and osteoporosis are two major factors in the 'geriatric giants' of immobility and instability—pertinent reminders of the widespread effect of locomotor illness with advancing age.

History

- ***Method of presentation:*** can vary, ranging from the fall that leads to a femoral neck fracture or a referral 'off legs' or with declining mobility. Older people will often have an existing diagnosis of some form of arthritis—the difficulty is not in the diagnosis, but understanding the impact on everyday life. Locomotor illnesses are a key *part* of such presentations, and attention to these illnesses is vital. However, it is important to remember that presentations such as falls are multifactorial—try to work out how locomotor illness contributes to mobility or falls risk
- ***Intercurrent illness:*** may often precipitate gout or particularly pseudogout. Equally important are those illnesses that disturb carefully balanced homeostasis, leading to a fall and fracture. Your task is not just to treat the consequence of the fall, but look at why it happened in the first place
- ***Septic joints:*** can be notoriously difficult to diagnose at times. Unilateral large joint swelling/acute arthritis should ring alarm bells instantly, especially if the patient is unwell. A myriad of causes contribute to back pain, but never forget deep seated infection such as discitis or osteomyelitis which may be a consequence of something innocuous as a urinary infection.
- ***DHx:*** as ever, a keystone of any assessment. Consider the side effect profile of NSAIDs, or whether gout has been precipitated by the

effects of diuretics or low dose aspirin. If your patient has sustained a fragility fracture due to osteoporosis, are they on appropriate treatment? Never forget the ↑ number of older people whose arthritis is successfully treated with disease modifying drugs—and understand the effects of such drugs (and the need to prescribe concurrent folic acid with methotrexate—don't forget!).

- ***Activities, occupation, and interests:*** overlaps with the functional history, a key message of these pages. Multi-disciplinary assessment is vital in terms of tailoring rehabilitation, aids and future care where appropriate. Ask too about hobbies and interests—improving balance, minimizing pain and maximizing function may allow patients to carry on with activities that are a key part of their lives (and might represent an opportunity for continued exercise or rehabilitation).

Examination

- ***General:*** the signs are often very clear, but despite this, easily overlooked. The need here is for a careful and thoughtful assessment of function as well as disease activity. Always be solicitous of your patient's comfort—and examine carefully, explaining what you wish to do to avoid misunderstanding and pain.
- ***Pattern of disease:*** is well described in the previous pages of this chapter. Look out for typical patterns of disease, and also single joint pathology. Look at ankles, feet, and back—it takes only a little more time to undertake a good examination, but is depressingly common to see patients with poor balance and falls with a clerking that details no locomotor assessment.
- ***Disease activity:*** be careful when palpating—but look to see if an acute exacerbation of joint disease may well have contributed to the current presentation.
- ***Gait and balance:*** often overlooked, but a vital part of the examination. Learn (e.g. from the ward physiotherapist) how to undertake the 'get up and go test', a well validated test of gait and balance (see below). This assessment should overlap with neurological assessment when appropriate.

Box 11.14 Get up and go

An easy test to do, and one which gives a wealth of information. In essence, ask you patient to rise to standing from a chair, walk 3 metres, turn and return to the chair. This is not a pure observer role for the clinician—you must make an assessment of safety and be on hand to support the patient if needed.

We thank Dr richard Fuller for contributing this page.

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Chapter 12

The Male Reproductive System

Applied anatomy and physiology

Anatomy

The male reproductive system consists of a pair of testes, a network of excretory ducts (epididymis, ductus deferens, and ejaculatory tracts), seminal vesicles, prostate, bulbo-urethral glands, and penis.

The penis

The penis consists of erectile tissue contained within 2 dorsally placed corpora cavernosa and the corpus spongiosum which lies on their ventral surface. The corpora are attached proximally to the inferior pubic rami. The corpus spongiosum expands distally to form the glans penis and surrounds the urethra.

The 3 corpora are contained within a fibrous tubular sheath of fascia and covered by freely mobile (and elastic) skin. A loose fold of skin, the prepuce or 'foreskin' extends distally to cover the glans penis.

The scrotum

This is a muscular out-pouching of the lower part of the anterior abdominal wall. It contains the testes, epididymis, and lower ends of the spermatic cords. The scrotum acts as a 'climate-control system' for the testes. Muscles in the wall of the scrotum, in conjunction with muscle fibres in the spermatic cord, allow it to contract and relax moving the testicles closer or further away from the body.

The testes

These are paired, ovoid organs measuring ~4 × 3 × 2cm, found within the scrotal sac. The testes are made up of masses of seminiferous tubules which are responsible for producing spermatozoa. Interstitial cells (Leydig cells) lying between these tubules produce the male sex hormones.

In the fetus, the testes develop close to the kidneys in the abdomen, then descend caudally through the inguinal canal to reach the scrotum at ~38 weeks gestation.

Each testis is covered by an outer fibrous capsule (tunica albuginea). Laterally and medially lies the visceral layer of the tunica vaginalis (a closed serous sac—an embryonic derivative of the processus vaginalis which normally closes before birth). The posterior surface of the testis is devoid of tunica vaginalis and is pierced by numerous small veins that form the pampiniform plexus. Also the seminiferous tubules converge here to form the efferent tubules with eventually give rise to the epididymis.

Spermatic cord

This suspends the testis in the scrotum and contains structures running between the testis and the deep inguinal ring (the ductus deferens, arteries, veins, testicular nerves, and epididymis).

The cord is surrounded by the layers of the spermatic fascia (internal spermatic fascia) formed from the transversalis fascia, the cremasteric fascia formed from fascia covering the internal oblique, and the external spermatic fascia formed from the external oblique aponeurosis).

The cremasteric fascia is partly muscular. Contraction of this (the cremaster muscle) draws the testis superiorly. The raising and lowering of the testis acts to keep it at a near-constant temperature.

Epididymis

This is a convoluted duct ~6cm in length lying on the posterior surface of the testis. It is a specialized part of the collecting apparatus where spermatozoa are matured before traveling up the vas deferens to join the ducts draining the seminal vesicles, known as the ejaculatory ducts.

The seminal vesicles are paired organs that lie on the posterior surface of the bladder and contribute the majority of the fluid that makes up semen along with fructose, ascorbic acid, amino acids, and prostaglandins.

Prostate gland

This is a firm, walnut-sized structure with lies inferior to the bladder, encircling the urethra. Many short ducts produce fluid which is emptied into the urethra and makes up a proportion of semen.

Bulbo-urethral glands

These are small, pea-sized glands located near the base of the penis.

In response to sexual stimulation, the bulbo-urethral glands secrete an alkaline mucus-like fluid which neutralizes the acidity of the urine in the urethra and provides a small amount of lubrication for the tip of the penis during intercourse.

Sex hormones

Three hormones are the regulators of the male reproductive system:

- FSH is produced in the anterior pituitary gland and stimulates spermatogenesis by its action on Sertoli cells.
- LH is produced in the anterior pituitary gland and stimulates the production of testosterone from Leydig cells.
- Testosterone is produced in the testis and adrenal gland and aids the development of male secondary sexual characteristics and spermatogenesis.

The male sexual response

There are 4 stages of the sexual response:

- ***Excitement or arousal:*** under control of the parasympathetic nervous system. During this, the penis becomes engorged with blood and stands out from the body. Other changes include an ↑ in heart rate, blood pressure, respiratory rate, and skeletal muscle tone.
- ***Plateau:*** continued sexual stimulation maintains the changes made in the arousal phase. This can last from a few seconds to many minutes.
- ***Orgasm:*** In males, this is the briefest stage and mediated by the sympathetic nervous system. Rhythmic contractions of the perineal muscles, the accessory glands and peristaltic contraction of the seminal ducts result in ejaculation. This is usually followed by a refractory period during which another erection cannot be achieved—this varies between individuals from minutes to hours and lengthens with advancing age.
- ***Resolution:*** blood pressure, heart rate, respiratory rate and muscle tone return to the un-aroused state. Accompanied by a sense of relaxation.

The sexual history

This can be awkward for both the patient and the history-taker. It should be undertaken in a sensitive, confident, and confidential manner. Before the discussion takes place, the patient should be reassured about the levels of privacy and confidentiality and that they are free to openly discuss their sexual life and habits.

Make no assumptions, remain professional and try to use the patient's own words and language. Beware of cultural and religious differences surrounding both sex and talking about it. See p.22 for further guidance.

You should approach a sexual history in a structured way as below.

Sexual activity

This should include an assessment of the risk of acquiring a sexually transmitted disease (STD).

You need to determine the number and gender of the patient's sexual partners, what *their* risk of having an STD is and what precautions (if any) were taken. Try asking the following questions:

- Do you have sex with men, women, or both?
- In the past 2 months, how many people have you had sex with?
- When did you last have sexual intercourse?
- Was it with a man or a woman?
- Were they a casual or regular partner?
- Where were they from?
- Do they use injected drugs?
- Do they have any history of STIs?
- How many other partners do you think they've had recently?
- In what country did you have sex?
- What kind of sex did you take part in? (e.g. vaginal, anal, oral)

- For each type of sex… did you use a condom?
- Does your partner have any symptoms?
- Have you had any other partners in the last 6 weeks?
    - If so, repeat the questions above for each partner.

Previous history

You also need to establish the history of STIs for the patient.

- Have you had any other STIs?
- Have you ever had a sexual check up?
- Have you ever been tested for HIV, hepatitis or syphilis?
- Have you ever been vaccinated against hepatitis A or B?

Psychological factors

Concerns over loss of libido and sexual functioning may point to a complex psychological cause for the symptoms. Explore this delicately and ask about:

- A history of sexual abuse.
- Problems with the relationship.
- Sexual partners outside the relationship.
- Any other cause for anxiety.
- A history of depression or anxiety.

Important symptoms

Urethral discharge

If the patient complains of discharge from the end of their penis, or 'mucus', establish:

- The amount.
- The colour.
- The presence of blood.
- The relationship between the discharge and urination or ejaculation.
- Is there any pain?
- Are there any other symptoms—such as conjunctivitis, arthralgia?
- Has the patient recently had symptoms of gastroenteritis?

You should also determine when this symptom was first noticed and how that relates to any sexual contacts that the patient has had and the possibility of exposure to STIs (see previous pages).

Rashes, warts, ulcers

Treat a genital skin lesion as you would any other rash (📖 p.88).

Ask also about:

- Similar lesions elsewhere (mouth, anus).
- Foreign travel.

You should determine the risk of recent exposure to STIs as previously.

Testicular pain

This is often felt as a deep burning and accompanied by nausea. Treat as pain in any other location as on p.39. Also ask about associated genital symptoms such as testicular swelling, dysuria, or haematuria.

Common causes include: testicular torsion, mumps, orchitis, and epididymitis. Remember the possibility of cancer.

Impotence

This term simply serves to confuse and is best avoided by doctors. Patients may use ‘impotence’ to mean a number of different sexual problems. Ask specifically if the patient means:

- Difficulty in either achieving or maintaining an erection (erectile dysfunction).
- Difficulty in ejaculating semen (ejaculatory dysfunction).
- Difficulty in reaching orgasm (orgasmic dysfunction).

▸ Remember that an erection is not necessary for men to reach orgasm or to ejaculate.

Erectile dysfunction

Erectile dysfunction is the inability to gain and maintain an erection for satisfactory completion of sexual activities.

If a patient complains of impotence, this needs to be explored in more detail. Establish particularly if the lack of function is related to a particular partner, a particular situation or is constant. Ask:

- Are you able to get an erection at all?
- Do you wake with an erection in the morning?
- Are you able to get an erection to masturbate?

If the cause is psychological, patients will often still wake with an erection (the so-called ‘morning glory’) but not be able to perform in a sexual situation. This can be tested with a sleep-study if necessary.

Psychological factors should be explored delicately.

Organic causes for erectile dysfunction include atherosclerosis, diabetes mellitus, multiple sclerosis, pelvic fractures, urethral injury, or other endocrine dysfunction.

DHx is important here. Drugs associated with erectile dysfunction include: barbiturates, benzodiazepines, phenothiazines, lithium, antihypertensives (e.g. ß-blockers), alcohol, oestrogens, methadone, heroin.

Loss of sexual desire (libido)

This can be the first sign of a pituitary tumour—but the cause is more often deeply rooted in the patient's psychology. Ask:

- How often do you shave your face?
- Has this changed recently?
- Do you have any muscle wasting or pain?
- …and explore any issues there may be surrounding the sexual partner and the patient's relationship with them.

Infertility

Around 10% of couples have difficulty in conception. Male infertility accounts for 1/3 of childless relationships. This is a huge topic and not within the scope of this book.

Relevant information to ascertain includes:

- The age of both partners.
- The length of time they have been trying to conceive.
- The presence of existing children belonging to both partners.
- Frequency and timing of intercourse.
- Any erectile, ejaculatory, or orgasmic dysfunction.

- DHx of both partners.
- Factors suggestive of endocrine malfunction as above under ‘loss of sexual desire’.
- Smoking and alcohol consumption.
- Menstrual history from partner.

Box 12.1 The rest of the history

A full history needs to be taken in all cases as described in  Chapter 2. The following may have particular relevance here.

PMH

Ask especially about:

- Sexually transmitted diseases (as previously).
- Orchitis.
- Inguinal, scrotal, and testicular injury/surgery.
- Urethral injury.

Smoking and alcohol

Detailed histories should be taken as described earlier in this book.

Examining the male genitalia

Explain to the patient that you would like to examine the penis and testes and reassure them that the procedure will be quick and gentle.

You should have a chaperone present, particularly if you are female*.

Ensure that the examination room is warm and that you are unlikely to be disturbed. With the patient on a bed or couch, raised to a comfortable height, ask them to pull their clothing down. You should be able to see the genitalia and lower part of the patient's abdomen at the very least.

The penis

Inspection

Make a careful inspection of the organ noting particularly:

- Size.
- Shape.
- Presence or absence of a foreskin.
- Colour of the skin.
- The position and calibre of the urethral meatus (see Box 12.2).
- Any discharge.
- Any abnormal curvature.
- Any scaling, scabbing or other superficial abnormality such as erythema or ulceration—particularly at the distal end (glans).

Palpation

Palpate the whole length of the penis to the perineum and note the state of the dorsal vein which is usually easily seen stretching the length of the penis at the dorsal midline. Note also any abnormalities of the underlying tissues (e.g. firm areas) which may not be visible—this may represent the plaques of Peyrone's disease.

Retract the foreskin to expose the glans penis and urethral meatus. The foreskin should be supple, allowing smooth and painless retraction. Look especially for any secretion or discharge and collect a specimen if possible. The patient may be able to ‘milk’ the shaft of the penis to express the secretion.

There is often a trace of smegma underlying the foreskin. This is a normal finding.

 Remember to replace the foreskin at the end of the examination.

▸ Note that in the presence of phimosis, the foreskin will be nonretractile and attempts may cause considerable pain.

Box 12.2 Hypospadias

Hypospadias is the abnormal, ventral, positioning of the urethral meatus. It is seen in ~1 in 250 males. In the vast majority, the hypospadias is slight. Patients may have a 'hooded foreskin' with the meatus at the very edge of the glans or a very slightly ventral meatus which is completely covered by a normal foreskin.

Slight hypospadias has no effect on sexual function but may be a cause of anxiety and embarrassment resulting in psychosexual problems once the patient is aware that his penis is 'different'.

The scrotum and contents

Examining the scrotum and scrotal contents is best done with the patient standing up.

Inspection

Make a careful examination of the scrotal skin. It is usually wrinkled, slightly more pigmented than the rest of the patient's body and should be freely mobile of the testes.

One testis usually hangs lower than the other. Remember to lift the scrotum, inspecting the inferior and posterior aspects.

Look especially for:

- Oedema.
- Sebaceous cysts.
- Ulcers.
- Scabies.
- Scars.

Palpation

The scrotal contents should be *gently* supported with your left hand and palpated with the fingers and thumb of your right hand. It may help to ask the patient to hold their penis to one side (see Fig. 12.1).

- Check that the scrotum contains 2 testes.
  - Absence of one or both testes may be due to previous excision, failure of the testis to descend or a retractile testis.
  - If there appears to be a single testis, carefully examine the inguinal canal for evidence of a discrete swelling that could be an undescended testis.
- Make careful note of any discrete lumps or swellings in the body of the testis.
  - Any swelling in the body of the testis must be considered to be suggestive of a malignancy.
- Compare the left and right testes, noting the size and consistency.
  - The testes are normally equal in size, smooth, with a firm, rubbery consistency. If there is a significant discrepancy, ask the patient if he has ever noticed this.
- Feel for the epididymis which lies at the posterolateral aspect of each testis.
- The vas can be distinguished from the rest of the cord structures, lying along the posterior aspect of the bundle and feels firm and wire-like. It runs from the epididymis to the external inguinal ring.

Scrotal swellings

If a lump is palpated…

- Decide if the lump is confined to the scrotum. Are you able to feel above it? Does it have a cough impulse? Is it fluctuant? (You will be unable to 'get above' swellings that descend from the inguinal canal.)
- Define the lump as any other mass as described on p.98.
- Transillumination is often important here. Darken the room and shine a small torch thought the posterior part of the swelling (see Fig. 12.2).
  - A solid mass remains dark while a cystic mass or fluid will transilluminate.

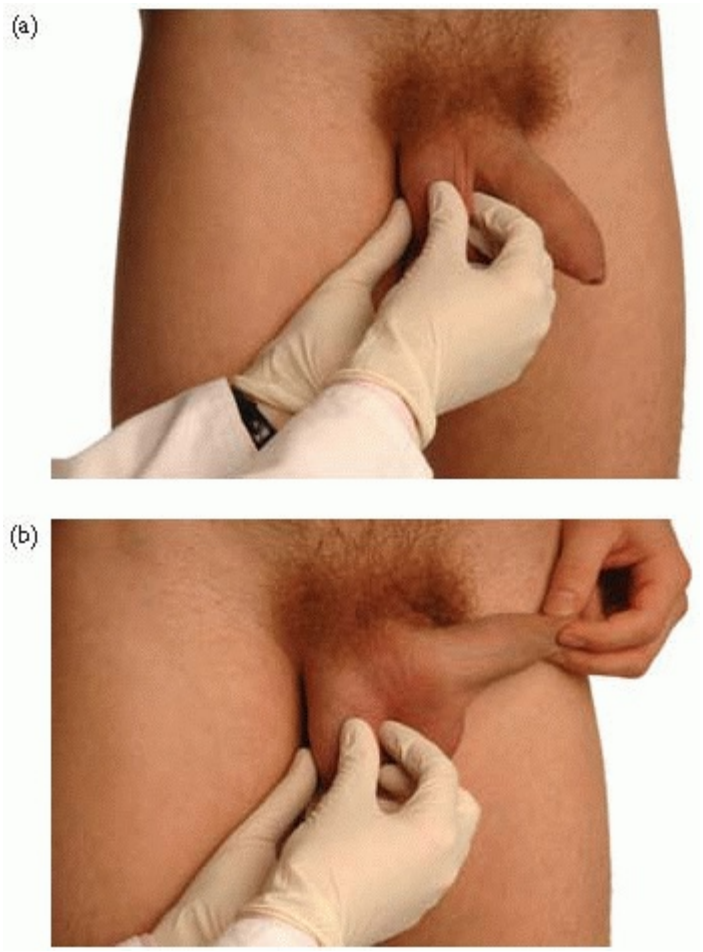

Fig. 12.1 (a) Examine the scrotum with the patient standing and use both hands. It is sometimes preferable to askthe patient to hold their penis aside (b).

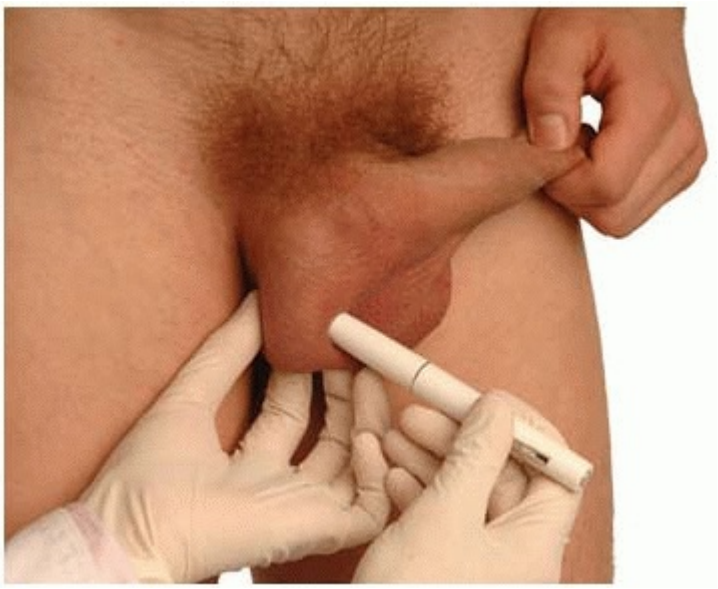

Fig. 12.2 Attempt to transilluminate any swelling by shining a small torch through it. NB Unlike the figure above, the room should be darkened.

The perineum and rectum

Don't overlook the perineum, anal canal and rectum. In particular, a digital rectal examination should be performed as described on p.264 with particular attention to feeling the prostate and seminal vesicles.

The local lymphatics

- Lymph from the skin of the penis and scrotum drains to the inguinal lymph nodes.
- Lymph from the covering of the testes and spermatic cord drains initially to the internal, then common, iliac nodes.
- Lymph from the body of the testes drains to the para-aortic lymph nodes—these are impalpable.
- Your examination is not complete without a careful palpation of the inguinal lymph nodes. This is best done with the patient lying comfortably on a bed or couch.
- If any swelling is found, it should be described in the same way as any lump (p.98).

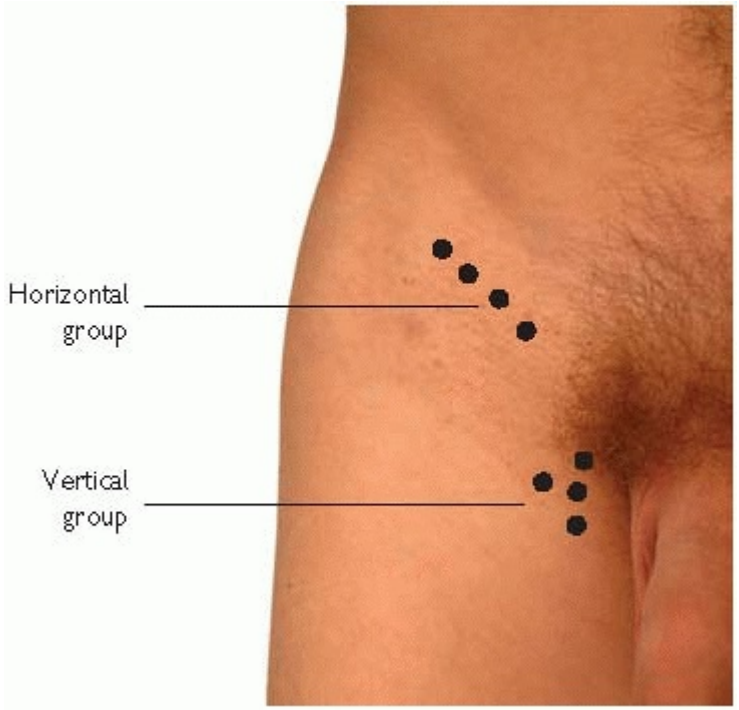

Fig. 12.3 Diagrammatic representation of the inguinal lymph nodes.

Important presenting patterns

Phimosis

This is a narrowing of the end of the foreskin which prevents its retraction over the glans penis.

This can cause difficulty with micturition and lead to recurrent balanitis. It may cause interference with erections and sexual intercourse.

Causes: congenital, infection, trauma, inflammation (balanitis).

Paraphimosis

In this case, the foreskin can be retracted but then cannot be replaced over the glans. This results in oedema which limits its movement still further. If left in this condition, it can become necrotic or gangrenous.

Commonly occurs in men 15-30 years old. A frequent complication of urinary catheterization if the practitioner fails to replace the foreskin after the procedure is performed.

Hypospadias

See also Box 12.2, p.413. A congenital abnormality in which the urethra opens on the ventral surface of the penis. Minor degrees result in a 'hooded' or dorsal foreskin and spraying of the urine during micturition.

Openings on the ventral surface of the shaft of the penis (secondary hypospadias) or openings at the scrotum or perineum (tertiary hypospadias) may cause serious difficulties with micturition and sexual function.

Balanitis and balanoposthitis

Balanitis is inflammation of the glans penis. Balanoposthitis is inflammation of the glans and the foreskin. Such inflammation presents as redness, swelling and pain of the affected parts, often with difficulty of retracting the foreskin.

Causes: *Candida albicans* (especially in patients with diabetes), herpes infection, carcinoma, drug eruptions, poor hygiene.

Priapism

This is a painful, persistent erection and a serious feature of sickle-crisis.

Other causes include: leukaemia, drugs (e.g. psychotropics), neurogenic (e.g. diseases of the spinal cord).

Penile ulcers

Conditions causing ulceration of the genitalia include herpes simplex (vesicles followed by ulceration), syphilis (non-tender ulceration), malignancy (e.g. squamous cell carcinoma—non-tender), Behçet's syndrome.

Hydrocoele

This is fluid entrapment in the tunica vaginalis causing usually painless swelling of the scrotum—the size of which can be considerable. Hydrocoeles will surround the testis making it impalpable. It will transilluminate.

As well as congenital abnormalities in the inguinal canal, hydrocoele can be caused by trauma, infection, and neoplastic disease.

Epididymal cysts

These are harmless, painless swellings arising behind, and separate from, the testis itself. The examiner may be unable to feel the rest of the epididymis. The testicle (anteriorly) should be normal. Epididymal cysts will transilluminate.

Varicocoele

Abnormal dilatation of the veins in the spermatic cord caused by incompetent valves in the testicular vein. They only become apparent when the patient is standing and almost disappear when the patient is supine. These are much more common on the left.

These are usually painless (although can cause a deep 'ache' in some men) and, in themselves, harmless. Varicocoele is, however, associated with a reduction of fertility (makes the testis abnormally warm). They are classically described as feeling like a 'bag of worms' on palpation.

Orchitis

Inflammation of the testis. The affected organ will hang higher in the scrotum, may be swollen and warm with redness of the overlying skin. It will be very tender to palpation. The patient may be systemically unwell with fever.

Testicular torsion

This presents in a very similar way to orchitis (above) and is often difficult to distinguish although the onset is much more sudden in torsion. Twisting of the testis on the spermatic cord ('torsion') will cause ischaemia and severe pain. Usually occurs in young adults and teenagers with a peak age of 14. Torsion is usually inward, towards the midline.

▸ This is a urological emergency. If the testicle is left in this condition without being untwisted (with appropriate analgesia), surgical removal of the testis may be necessary. Immediate surgical referral is advised if this is suspected.

Testicular carcinoma

This should be at the top of your list of differential diagnoses in the case of an intra-scrotal mass. Teratomas commonly occur between the ages of 20-30 years while seminomas are more common between 30-40 years.

There may be associated pain and tenderness or a dull aching, dragging sensation in the scrotum and groins. Look for constitutional symptoms suggestive of neoplastic disease such as malaise, wasting, and anorexia as well as leg swelling (venous or lymphatic obstruction), lymphadenopathy, or an associated abdominal mass.

The elderly patient

Many of the messages in this page overlap with those in the female reproductive system pages and we would encourage you to regard them as a 'whole'.

It is important to recognize that bladder carcinoma and diseases of the prostate are some of the most common urogenital problems faced by older men, remember to screen for such problems in any assessment. For prostate diseases, it is also important to be alert that awareness by patients is equally high, so expect questions and a wish to be involved in treatment decisions. Equally so, many of these problems are faced by patients with cognitive impairment, in whom history may be limited and thorough assessment is vital.

Retain an holistic outlook on male urogenital problems, and you're less likely to miss delirium because of acute epididymo-orchitis—a not uncommon presentation!

 It is also important to remember that studies report that 60% of men and 30% of women over the age of 80 still engage in some form of regular sexual activity. Avoiding these issues can cause major problems to be overlooked, with 70% of men over the age of 70 experiencing impotence—so try not to make assumptions when seeing older people with sexual health problems.

History

- ***Explore:*** the history. Even for patients with prostate disease, how will the effects of treatment (e.g. TURP) affect relationships or sexual activity? Keep your thought processes open when assessing continence problems—there may be an irritative/unstable bladder component alongside obstructive symptoms.
- **PMH***:* vascular diseases, metabolic, and neurological illnesses may all be underpinning diagnoses when faced with impotence. Could the new presentation of balanitis indicate diabetes?
- ***DHx:*** aside from obvious culprits (e.g. diuretics) consider the effects of anti-depressants, digoxin, and antihypertensives on both bladder and sexual function.
- ***SHx and sexual history:*** always take an appropriate functional history, particularly if there are continence problems. Consider alcohol and tobacco in relation to impotence, and undertake a detailed occupational history if the patient presents with haematuria (bladder cancer?). Have the confidence to take a sexual history if there are problems with erectile or ejaculatory dysfunction—as indicated earlier, many older people have active sex lives and you're more likely to be embarrassed about taking the history than they are recounting it.

Examination

- ***General:*** alongside the detailed examination considered earlier in this chapter, keep in mind the need for a general examination—focusing on mood and neurological assessment in particular.
- ***Cognition:*** a key part of this assessment, and particularly for continence and erectile dysfunction problems.

- ***Urogenital:*** think subtly: in older men, orchitis may present with declining mobility, delirium, or falls—so never forget to undertake a thorough examination in elders, even when there is apparently little to indicate it! For patients with urinary catheters, whether short or long-term, examination is a mandatory part of assessment.

Box 12.3 A note on (recurrent) urinary tract infections

Most readers will have already seen many older patients with this common (and often over-diagnosed) problem.

Whilst many diagnoses are made on clinical suspicion, it is vital to undertake urinalysis and obtain urine for microscopy and culture to confirm the presence of urinary tract infections (UTIs). The reasoning is two-fold—avoiding rushing to a label of UTI as the cause of delirium or mobility decline will reduce the chance of missing the correct diagnosis. Similarly, a proven culture diagnosis of UTI aids prescribing, and helps identify recurrent infections. Recognizing the latter may reveal underlying diagnoses and reduce discomfort or even hospitalization for some patients.

So, when faced with recurrent infections, be assiduous and request urine cytology (to look for bladder cancers), ultrasound (for structural abnormalities), and discuss the value of cystoscopy and rotating/long term antibiotics with urology colleagues.

We thank Dr Richard Fuller for providing this page.

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Chapter 13

The Female Breast

Applied anatomy and physiology

Anatomy of the breast

The 2 mammary glands are highly developed apocrine sweat glands. They develop embryologically along 2 lines extending from the axillae to the groin—the milk lines (see Fig. 13.1). In humans, only one gland develops on each side of the thorax although extra nipples with breast tissue may sometimes occur.

The breasts extend from the 2nd to the 6th ribs and transversely from the lateral border of the sternum to the midaxillary line.

For the purposes of examination, each breast may be divided into 4 quadrants by horizontal and vertical lines intersecting at the nipple. An additional lateral extension of breast tissue (the axillary tail of Spence) stretches from the upper outer quadrant towards the axilla (see Fig. 13.2).

Each mammary gland consists of 15-20 lobes separated by loose adipose tissue and subdivided by collagenous septa. Strands of connective tissue called the suspensory ligaments of the breast (Cooper's ligaments) run between the skin and deep fascia to support the breast. Each lobe is further divided into a variable number of lobules composed of grape-like clusters of milk-secreting glands termed alveoli and is drained by a lactiferous duct that opens onto the nipple. Myo-epithelial cells surround the alveoli which contract and help propel the milk toward the nipples.

The nipple is surrounded by a circular pigmented area called the areola and is abundantly supplied with sensory nerve endings. The surface of this area also contains the ‘sebaceous glands of Montgomery’ which act to lubricate the nipple during lactation.

Lymphatic drainage

Lymphatic drainage from the medial portion of the breast is to the internal mammary nodes. The central and lateral portions drain to the axillary lymph nodes which are arranged into 5 groups (see Fig. 13.7, p.437).

Physiology—normal breast changes in women

Puberty: during adolescence, oestrogen promotes the development of the mammary ducts and distribution of fatty tissue while progesterone induces alveolar growth.

The menstrual cycle: towards the 2nd half of the menstrual cycle, after ovulation, the breasts often become tender and swollen. They return to their ‘resting’ state after menstruation.

Pregnancy: high levels of placental oestrogen, progesterone, and prolactin promote mammary growth in preparation for milk production.

Postnatal: the sharply declining levels of oestrogen and progesterone permit prolactin to stimulate the alveoli and milk produced. Suckling stimuli ↑ secretion of prolactin as well as releasing oxytocin which stimulates myoepithelial cells to contract.

Menopause: the breasts become softer, more homogenous and undergo involutional changes including a ↓ in size, atrophy of the secretory portions, and some atrophy of the ducts.

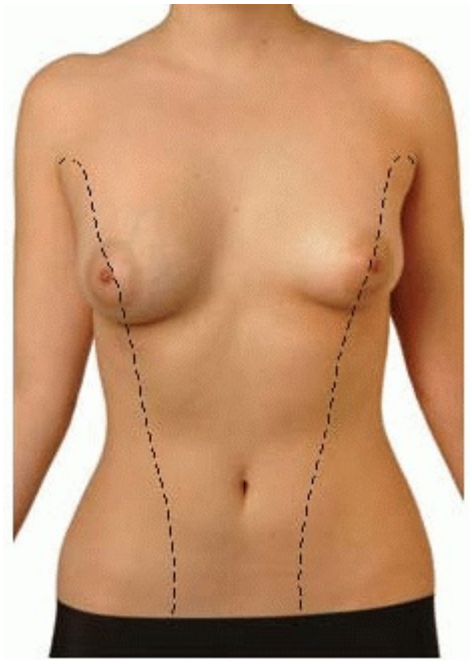

Fig. 13.1 Illustration of the 2 milk lines along which the nipples form—occasionally extra nipples can be found.

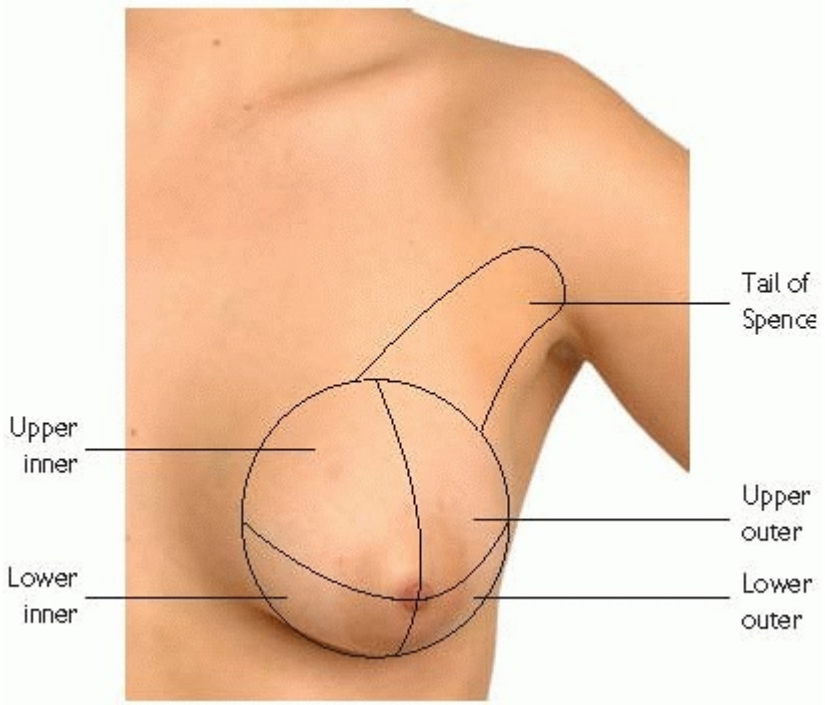

Fig. 13.2 Illustrations showing the 4 quadrants of the breast with the axillary tail of Spence.

Important symptoms

First steps

You should begin by establishing a menstrual history (see Box 13.1 and Chapter 14). You should also determine the date of the last period of menstruation. It is important to note that pre-existing disease in the breast is likely to become more noticeable during the 2nd half of the menstrual cycle—lumps often get bigger or become more easily palpable.

▸ You should bear in mind that seeking medical attention for a breast lump or tenderness can produce extreme anxiety and embarrassment in patients. Men with gynaecomastia are also likely to feel anxious about their breast development. Ensure that you adopt

an appropriately sensitive, sympathetic, and professional approach.

Breast pain (mastalgia)

As for pain at any other site, you should establish the site, radiation, character, duration, severity, exacerbating factors, relieving factors, and associated symptoms. Also ask:

- Is the pain unilateral or bilateral?
- Is there any heat or redness at the site?
- Are there any other visible skin changes?
- Is the pain cyclical or constant—and is it related to menstruation?
- Is there a history of any previous similar episodes?
- Is the patient breastfeeding?
- Is the patient on any hormonal therapy (especially HRT)?

The commonest cause of mastalgia in premenopausal women is hormonedependent change. Other benign causes include mastitis and abscesses. 1/100 breast cancers present with mastalgia as the sole symptom.

Nipple discharge

Important causes of nipple discharge include ductal pathology such as ductal ectasia, papilloma, and carcinoma.

Ask about:

- Is the discharge true milk or some other substance?
- The colour of the discharge (e.g. clear, white, yellow, blood-stained).
- Spontaneous or non-spontaneous discharge?
- Is the discharge unilateral or bilateral?
- Any changes in the appearance of the nipple or areola.
- Mastalgia.
- Any breast lumps.
- Periareola abscesses or fistulae indicating periductal mastitis.
    - This is closely linked to smoking in young women. Periductal mastitis is also associated with hidradenitis suppuritiva. Ask about abscess elsewhere e.g. axilla and groins. The symptoms are often recurrent.

 Remember that after childbearing, some women continue to discharge a small secretion of milk (galactorrhoea). However, in rare instances this can be the 1st presenting symptom of a prolactin-secreting pituitary adenoma. You should, therefore, in the case of true bilateral galactorrhoea also ask about:

- Headaches.
- Visual disturbance.

Box 13.1 The menstrual history

It is important to take a clear and accurate menstrual history as outlined in  Chapter 14. You should establish the:

- Age of first menses.
- Usual time between menstruation.
- Usual duration of menstruation.
- Usual quantity of menstruation.
- Age of menopause (if applicable).
- Number of pregnancies.
- Previous history of breast feeding.
- The date of the beginning of the last menstrual period.

Breast lumps

A very important presenting complaint with a number of causes—the most important of which is cancer. Establish:

- When the lump was first noticed.
- Whether the lump has remained the same size or enlarged.
- Whether the size of the lump changes according to the menstrual cycle.
- Is there any pain?
- Are there any local skin changes?
- Is there a history of breast lumps (ask about previous biopsies, the diagnoses, and operations).
- A full systems enquiry should include any other symptoms which might be suggestive of a neoplastic disease (loss of weight, loss of appetite, fatigue etc.) and metastatic spread to other organ systems (shortness of breath, bony pain etc.).

Age

A good clue as to the likely diagnosis of a lump is the age of the patient:

- Fibroadenomas are common between 20-30 years.
- Cysts are common between 30-50 years.
- Cancer is very rare <30 years but common in the >50 age group.

Box 13.2 The male breast

Gynaecomastia

This is enlargement of the male breast tissue which should not normally be palpable. There is an ↑ in the ductal and connective tissue.

A common occurrence in adolescents and the elderly. Gynaecomastia is seen in obese men due to increased adipose tissue.

In many patients, gynaecomastia is drug-related and the full causative list is long. Important drug causes include estrogen receptor binders such as oestrogen, digoxin, and marijuana as well as anti-androgens such as spironolactone and cimetidine.

▸ In the history, ask about drug and hormone treatment (e.g. for prostate cancer).

▸ You should also make a full examination of the patient looking for signs of hypopituitarism, chronic liver disease and thyrotoxicosis. Remember to make a careful examination of the genitalia.

Breast cancer in the male

The female:male ratio for breast cancer is ~100:1.

The appearance and pathology of breast cancer in the male is similar to that in the female.

The most common presentation in males is a hard, painless lump fixed to the skin or chest wall followed by nipple discharge.

Box 13.3 Risk factors for breast cancer in females

- Advancing age.
- Breast cancer in a 1st degree relative.
- *BRCA* genes (see *OHCM6*, p.434).
- Previous cancer in the other breast.
- Early menarche (<12 years).
- Late menopause (>55years).
- Nulliparity (no pregnancies).
- No previous breast-feeding.
- Previous radiotherapy—e.g. mantle radiotherapy for Hodgkin's disease.
- The oral contraceptive pill or HRT.

Box 13.4 Some causes of breast lumps

- Cyst.

- Fibroadenoma.
- Carcinoma.
- Fat necrosis.
- Hamartoma.
- Lipoma.
- Epidermoid cyst.
- Cystosarcoma.

Inspection of the breast

Before you start

- When examining the female breast, examiners should have a chaperone present. Ideally, the chaperone should be female.*
- The patient should be fully undressed to the waist and sitting on the edge of a couch with her arms by her side.
- You should be able to see the neck, breasts, chest wall, and arms.

* In the UK, official advice is that *all* doctors should have a chaperone when performing an intimate examination and the chaperone should be the same sex as the patient.

General inspection

Stand in front of the patient and observe both breasts, noting:

- Size.
- Symmetry.
- Contour.
- Colour.
- Scars.
- Venous pattern on the skin.
- Any dimpling or tethering of the skin.
- Ulceration (describe fully as on 📖 p.100).
- Skin texture: e.g. any visible nodularity.
    - An unusual finding, but one that should not be missed is the ‘orange peel’ appearance of *peau d'orange* caused by local oedema. Seen in breast carcinoma and following breast radiotherapy.

Nipples

Note whether the nipples are:

- Symmetrical.
- Everted, flat, or inverted.
- Scale (may indicate eczema or Paget's disease of the breast).
- Associated with any discharge.
    - Single duct discharge can indicate a papilloma or cancer.
    - Multiple duct discharge at the nipple suggests duct ectasia.

If abnormalities are present, make sure to ask if these are a recent or long-standing appearance.

Make note of any additional nipples, which can occur anywhere along the mammary line.

Axillae

Ask the patient to place her hands on her head and repeat the inspection process. Pay particular attention to any asymmetry or dimpling that is now evident. Examine the axillae for masses or colour change.

Manoeuvres

Finally, dimpling or fixation can be further accentuated by asking the patient to perform the following manoeuvres (see Fig. 13.3):

- Lean forward whilst sitting.
- Rest her hands on her hips.
- Press her hands against her hips ('pectoral contraction manoeuvre').

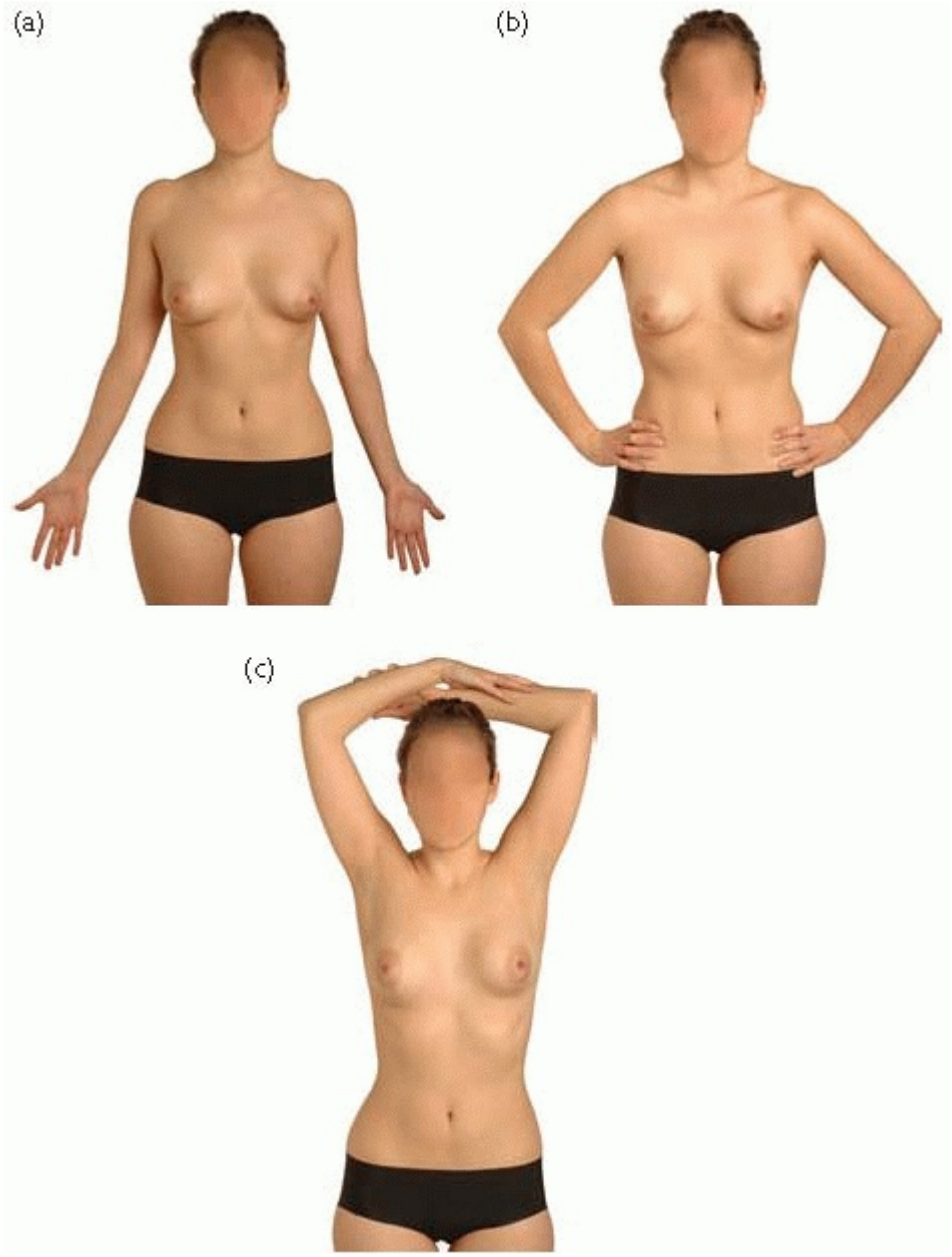

Fig. 13.3 Manoeuvres for breast inspection. (a) Anatomical position. (b) Hands on hips. (c) Arms crossed above the head.

Palpation of the breast

Before you start

Palpation of the breast should be performed with the patient lying supine on the couch. Initially, the patient should have her hands by her sides. Examination of the upper-outer quadrant is best performed with the hand on the side to be examined placed behind her head (Fig. 13.4).

Palpation

Ask the patient if there is any pain or tenderness—and examine that area last. Also ask her to tell you if you cause any pain during the examination.

You should begin the examination on the asymptomatic side, allowing you to determine the texture of the normal breast first.

Ask the patient to point out any areas of tenderness and come to these last.

The breast

Palpation should be performed by keeping the hand flat and gently rolling the substance of the breast against the underlying chest wall.

Most breasts will feel 'lumpy' if pinched.

You should proceed in a systematic way to ensure that the whole breast is examined. There are 2 regularly used methods (see Fig. 13.5) of which the authors favour the 1st:

- Start below the areola and work outwards in a circumferential pattern ensuring that all quadrants have been examined.
- Examine the breast in 2 halves working systematically down from the upper border.

▸ Do not forget to examine the axillary tail of Spence stretching from the upper-outer quadrant to the axilla.

Lumps

- If you feel a lump, describe it according to the method on p.98 noting especially: position, colour, shape, size, surface, nature of the surrounding skin, tenderness, consistency, temperature, and mobility.
- Next ascertain its relations to the overlying skin and underlying muscle.
- You must decide whether you are feeling a lump or a lumpy area.

Skin tethering

A lump may be described as tethered to the skin if it can be moved independently of the skin for a limited distance but pulls on the skin if moved further.

Tethering implies that an underlying lesion has infiltrated Cooper's ligaments which pass from the skin through the subcutaneous fat. Tethering may involve the skin itself with cancers or abscesses.

On inspection at rest, there may be puckering of the skin surface (as if being pulled from within) or there may be no visible abnormality.

To demonstrate tethering:

- Move the lump from side to side and look for skin dimpling at the extremes of movement.
- Ask the patient to lean forwards whilst sitting.
- Ask the patient to raise her arms above the head as in Fig. 13.3.

Skin fixation

This is caused by direct, continuous infiltration of the skin by the underlying disease. The lump and the skin overlying it cannot be moved independently. It is on a continuum with skin tethering. This may be associated with some changes of skin texture.

The relation of a lump to the muscle

The lump may be tethered or fixed to the underlying muscle (e.g. pectoralis major).

▸ Lumps that are attached to the underlying muscle can be moved to some degree if the muscle if relaxed but are less mobile if the muscle is tensed.

- Ask the patient to rest her hand on her hip with the arm relaxed.
- Hold the lump between your thumb and forefingers and estimate its mobility by moving it in 2 planes at right angles to each other (e.g.

up/down and left/right).

- Ask the patient to press her hand against her hip causing contraction of pectoralis major. Repeat the mobility exercise.

Immobile lumps

If a lump is immobile in all situations, it may have spread to involve the bony chest wall (e.g. in the upper half of the breast or axilla).

The nipple

If the patient complains of nipple discharge, ask her to gently squeeze and express any discharge, noting colour, presence of blood and smell.

Milky, serous or green-brown discharges are almost always benign. A bloody discharge may indicate neoplasia (e.g. papilloma or cancer).

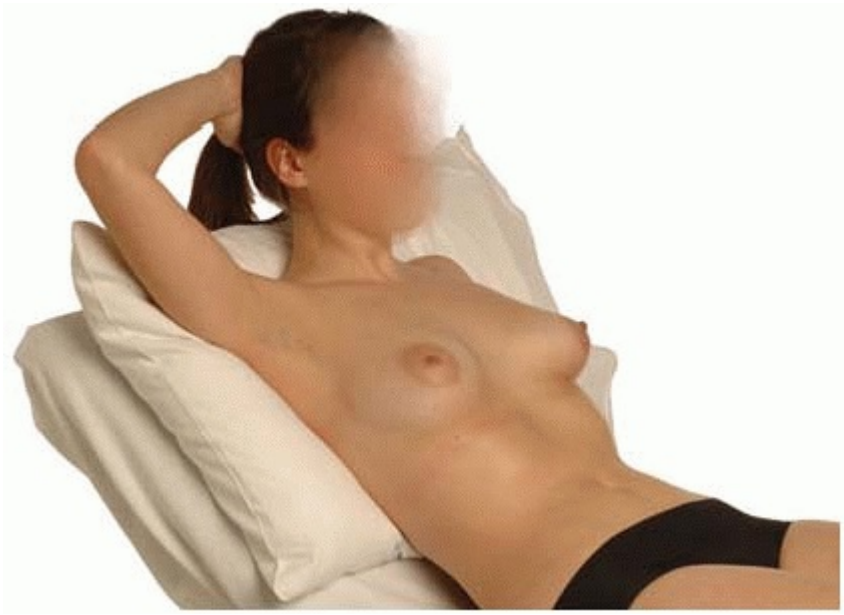

Fig. 13.4 Correct position of the patient for examination of the breast.

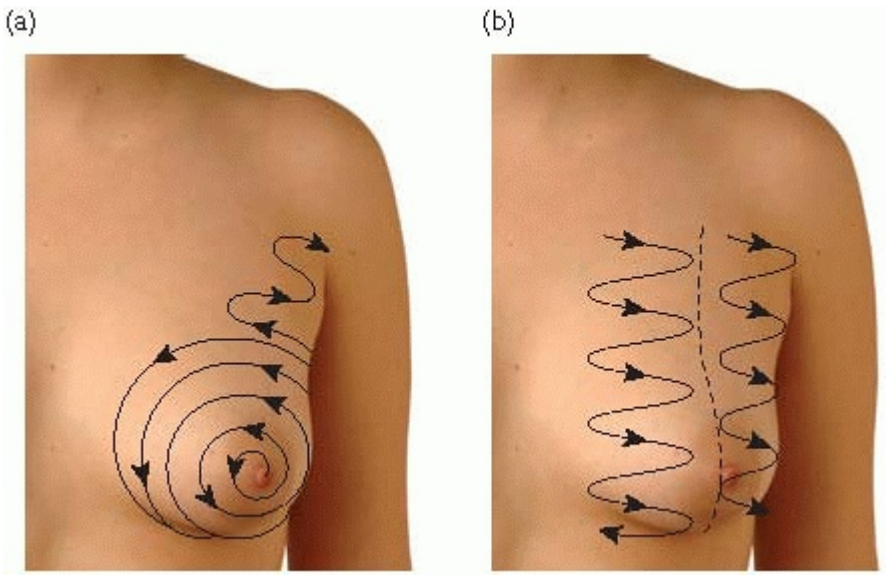

Fig. 13.5 Two methods forthe systematic palpation of the breast. (a) Work circumferentially from the areola. (b) Examine each half at a time, working from tope to bottom.

Examining beyond the breast

Lymph nodes

The technique is described in detail on p.68.

Support the patient's arm. For example, when examining the right axilla, abduct the patient's right arm gently and support it at the wrist with your right hand whilst examining the axilla with your left hand.

Examine the main sets of axillary nodes including:

- Central.
- Lateral.
- Medial (pectoral).
- Infraclavicular.
- Supraclavicular.
- Apical.

If you feel any lymph nodes, consider site, size, number, consistency, tenderness, fixation, and overlying skin changes.

Remember to also palpate for lymph nodes in the lower deep cervical lymph chain at the same time as the supraclavicular nodes.

The rest of the body

If cancer is suspected, is worth performing a full general examination, keeping in mind the common sites of metastasis of breast cancer. Examine especially the lungs, liver, skin, skeleton, and central nervous system.

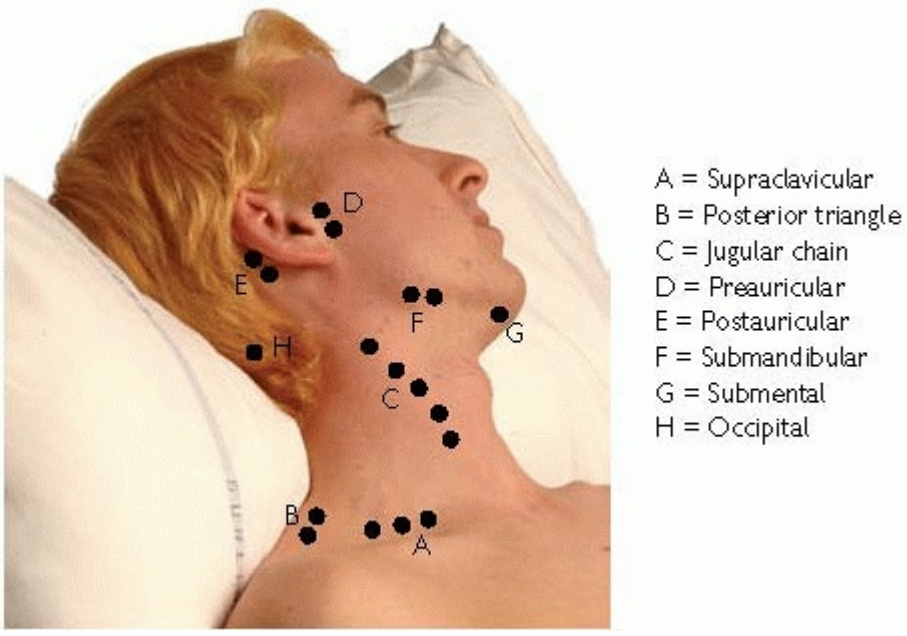

Fig. 13.6 Cervical and supraclavicular lymph nodes.

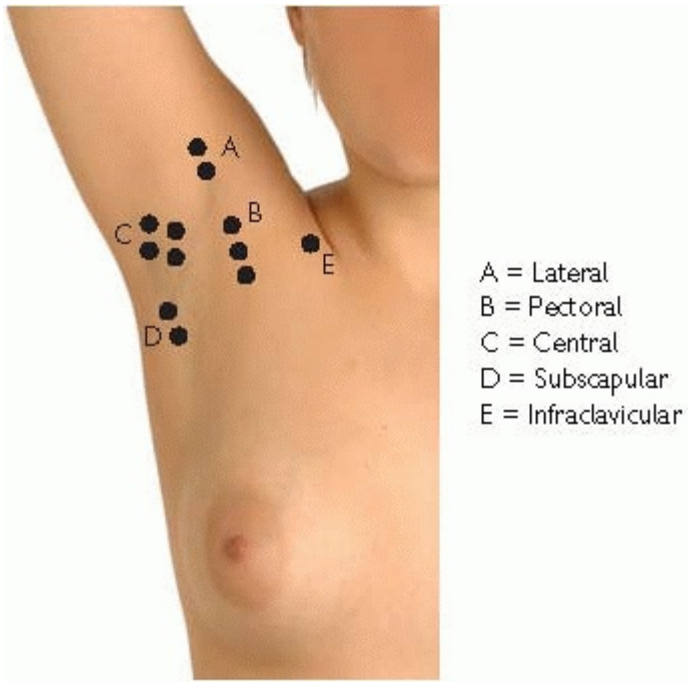

Fig. 13.7 Axillary lymph nodes.

Important presentations

Fibroadenoma

This is a benign neoplasm of the breast consisting of collagen arranged in 'swirls'. Usually found in young women <30 years old.

Presents as a painless, solid, mobile, well-circumscribed, rounded breast mass with a smooth, bosselated surface and rubbery consistency.

They may be multiple and bilateral with no axillary lymphadenopathy. Large fibroadenomas may be found in teenagers.

Fibrocystic disease

Another common benign breast condition consisting of fibrous (rubbery) and cystic changes in the breast.

Usually presents with pain or tenderness that varies with the menstrual cycle. Cysts and fibrous 'nodular' fullness can be found on examination. A solitary cyst is usually smooth, spherical, and tense. It is rarely possible to elicit fluctuation, a fluid thrill or transillumination. The axillary lymph nodes should not be palpable.

Fat necrosis

This can occur after trauma and the physical signs can mimic cancer (e.g. a firm hard lump with skin tethering).

Abscesses

Mainly occur during childbearing years and are often associated with trauma to the nipple during breast feeding.

Present with a painful, spherical lump with surrounding oedema. It often shows additional signs of inflammation (hot, red). The patient may have constitutional upset including malaise, night sweats, hot flushes and rigors.

Most recurrent or chronic breast abscesses occur in association with duct ectasia or periductal mastitis. The associated periductal fibrosis can often led to nipple retraction.

Abnormal nipple and areola

Diseases of the nipple are important because they must be differentiated from malignancy and cause concern to patients.

Unilateral retraction or distortion of a nipple is a common sign of breast carcinoma; as is blood-stained nipple discharge. The latter suggests an intraductal carcinoma or benign papilloma.

A unilateral red, crusted and scaling areola suggests an underlying carcinoma (Paget's disease of the breast) or, more commonly, eczema. Ask the patient if she has eczema at other sites and examine appropriately.

Breast cancer

Breast cancer is the commonest form of cancer affecting women in the UK.* It is rare under the age of 35, with the incidence steadily increasing per decade. There are a number of risk factors (📖 p.429).

There are 2 main types, which may be invasive or 'in situ':

- Ductal.
- Lobular.

In situ cancer is usually impalpable and diagnosed on mammography or biopsy.

There are a number of prognostic factors in breast cancer, mostly related to the histological appearance of the tumour. These are dealt with in detail in 📖 *OHCM6*, p.504.

Presenting symptoms

The presenting symptoms may be related to the primary lesion. For example:

- Palpable mass.
- Pain (1/100 cancers present with mastalgia only).
- Nipple discharge, retraction, or rash.
- Dimpling of the breast tissue.
- Local oedema.

The presenting symptom may be related to the effects of secondary spread. For example:

- Arm swelling (lymphatic or venous obstruction).
- Backache (skeletal infiltration).
- Malaise.
- Loss of weight.
- Dyspnoea.
- Cough.
- Nodules in skin.
- Jaundice.
- Mental changes.
- Fits/seizures.
- Symptoms of hypercalcaemia (e.g. thirst).

Editors: Thomas, James; Monaghan, Tanya
Title: *Oxford Handbook of Clinical Examination and Practical Skills, 1st Edition*

Chapter 14

The Female Reproductive System

Applied anatomy

The pelvis

The bony pelvis is composed of the 2 pelvic bones with the sacrum and coccyx posteriorly. The pelvic brim divides the 'false pelvis' above (part of the abdominal cavity) and the 'true pelvis' below.

- ***Pelvic inlet:*** also known as the pelvic brim. Formed by the sacral promontory posteriorly, the iliopectineal lines laterally and the symphysis pubis anteriorly.
- ***Pelvic outlet:*** formed by the coccyx posteriorly, the ischial tuberosities laterally and the pubic arch anteriorly. The pelvic outlet has 3 wide notches. The sciatic notches are divided into the greater and lesser sciatic foramina by the sacrotuberous and sacrospinous ligaments which can be considered part of the perimeter of the outlet clinically.
- ***The pelvic cavity:*** lies between the inlet and the outlet. It has a deep posterior wall and a shallow anterior wall giving a curved shape.

The contents of the pelvic cavity

The pelvic cavity contains the rectum, sigmoid colon, coils of the ileum, ureters, bladder, female reproductive organs, fascia, and peritoneum.

Female internal genital organs

Vagina

The vagina is a thin-walled distensible, fibromuscular tube that extends upwards and backwards from the vestibule of the vulva to the cervix. It is ~8cm long and lies posterior to the bladder and anterior to the rectum.

The vagina serves as an eliminatory passage for menstrual flow, forms part of the birth canal, and receives the penis during sexual intercourse.

The fornix

This is the vaginal recess around the cervix and is divided into anterior, posterior, and lateral regions which, clinically, provide access points for examining the pelvic organs.

Uterus

The uterus is a thick-walled, hollow, pear-shaped muscular organ consisting of the cervix, body and fundus. In the nulliparous female, it is ~8cm long, ~5cm wide, and ~2.5cm deep. The uterus is covered with peritoneum that forms an anterior uterovesical fold, a fold between the uterus and rectum termed the pouch of Douglas, and the broad ligaments laterally.

The uterus receives, retains, and nourishes the fertilized ovum.

Uterine orientation

In most females, the uterus lies in an anteverted and anteflexed position.

- Anteversion: the long axis of the uterus is angled forward.
- Retroversion: The fundus and body are angled backwards and therefore lie in the pouch of Douglas. Occurs in about 15% of the female population. A full bladder may mimic retroversion clinically.
- Anteflexion: the long axis of the body of the uterus is angled forward on the long axis of the cervix.
- Retroflexion: The body of the uterus is angled backward on the cervix

Fallopian tubes

The fallopian or 'uterine' tubes are paired tubular structures, ~10cm long. The fallopian tubes extend laterally from the cornua of the uterine body, in the upper border of the broad ligament and opens into the peritoneal cavity near the ovaries. The fallopian tube is divided into 4 parts:

- *Infundibulum:* distal, funnel-shaped portion with finger-like 'fimbriae'.
- *Ampulla:* widest and longest part of tube outside the uterus.
- *Isthmus:* thick-walled with a narrow lumen and therefore, least distensible part. Enters the horns of the uterine body.
- *Intramural:* that part which pierces the uterine wall.

The main functions of the uterine tube are to receive the ovum from the ovary, provide a site where fertilization can take place (usually in the ampulla) and transport the ovum from the ampulla to the uterus. The tube also provides nourishment for the fertilized ovum.

Ovaries

The ovaries are whitish-grey, almond-shaped organs measuring ~4cm×2cm which are responsible for the production of the female germ cells, the ova, and the female sex hormones, oestrogen and progesterone.

They are suspended on the posterior layer of the broad ligament by a peritoneal extension (mesovarium) and supported by the suspensory ligament of the ovary (a lateral extension of the broad ligament and mesovarium) and the round ligament which stretches from the lateral wall of the uterus to the medial aspect of the ovary.

Perineum

- The perineum lies inferior to the pelvic inlet and is separated from the pelvic cavity by the pelvic diaphragm.
- Seen from below with the thighs abducted, it is a diamond-shaped area bounded anteriorly by the pubic symphysis, posteriorly by the tip of the coccyx and laterally by the ischial tuberosities.
- The perineum is artificially divided into the anterior urogenital triangle containing the external genitalia in females and an anal triangle containing the anus and ischiorectal fossae.

Female external genital organs

These are sometimes collectively known as the 'vulva'. It consists of:

- ***Labia majora:*** a pair of fat-filled folds of skin extending on either side of the vaginal vestibule from the mons towards the anus.
- ***Labia minora:*** a pair of flat folds containing a core of spongy connective tissue with a rich vascular supply. Lie medial to the labia majora.
- ***Vestibule of the vagina:*** between the labia minora, contains the urethral meatus and vaginal orifice. Receives mucous secretions from the greater and lesser vestibular glands.
- ***Clitoris:*** short, erectile organ; the female homologue of the male penis. Like the penis, a crus arises from each ischiopubic ramus and join in the midline forming the 'body' capped by the sensitive 'glans'.
- ***Bulbs of vestibule:*** 2 masses of elongated erectile tissue, ~3cm long, lying along the sides of the vaginal orifice.
- ***Greater and lesser vestibular glands***.

Applied physiology

The menstrual cycle

Menstruation is the shedding of the functional superficial 2/3 of the endometrium after sex hormone withdrawal. This process, which consists of 3 phases, is typically repeated ~300-400 times during a woman's life. Coordination of the menstrual cycle depends on a complex interplay between the hypothalamus, the pituitary gland, the ovaries, and the uterine endometrium.

Cyclical changes in the endometrium prepare it for implantation in the event of fertilization and menstruation in the absence of fertilization. It should be noted that several other tissues are sensitive to these hormones and undergo cyclical change (e.g. the breasts and the lower part of the urinary tract).

The endometrial cycle can de divided into 3 phases…

Phases of the menstrual cycle

The first day of the menses is considered to be day 1 of the menstrual cycle.

The proliferative or follicular phase

This begins at the end of the menstrual phase (usually day 4) and ends at ovulation (days 13-14). During this phase, the endometrium thickens and ovarian follicles mature.

The hypothalamus is the initiator of the follicular phase. Gonadotrophinreleasing hormone (GnRH) is released from the hypothalamus in a pulsatile fashion to the pituitary portal system surrounding the anterior pituitary gland. GnRH causes release of follicle stimulating hormone (FSH). FSH is secreted into the general circulation and interacts with the granulosa cells surrounding the dividing oocytes.

FSH enhances the development of 15-20 follicles each month and interacts with granulosa cells to enhance aromatization of androgens into oestrogen and oestradiol.

Only one follicle with the largest reservoir of oestrogen can withstand the declining FSH environment whilst the remaining follicles undergo atresia at the end of this phase.

Follicular oestrogen synthesis is essential for uterine priming, but is also part of the positive feedback that induces a dramatic preovulatory leuteinising hormone (LH) surge and subsequent ovulation.

The luteal or secretory phase

The luteal phase starts at ovulation and lasts through to day 28 of the menstrual cycle.

The major effects of the LH surge are the conversion of granulosa cells from predominantly androgen-converting cells to predominantly progesterone-synthesising cells. High progesterone levels exert negative feedback on GnRH which, in turn, ↓ FSH/LH secretion.

At the beginning of the luteal phase, progesterone induces the endometrial glands to secrete glycogens, mucus, and other substances. These glands become tortuous and have large lumina due to ↑ secretory activity. Spiral arterioles extend into the superficial layer of the endometrium.

In the absence of fertilization by day 23 of the menstrual cycle, the superficial endometrium begins to degenerate and consequently ovarian hormone levels ↓. As oestrogen and progesterone levels fall, the endometrium undergoes involution.

If the corpus luteum is not rescued by human chorionic gonadotrophin (hCG) hormone from the developing placenta, menstruation occurs 14 days after ovulation. If conception occurs, placental hCG maintains luteal function until placental production of progesterone is well established.

The menstrual phase

This phase sees the gradual withdrawal of ovarian sex steroids which causes slight shrinking of the endometrium, and therefore the blood flow of spiral vessels is reduced. This, together with spiral arteriolar spasms, leads to distal endometrial ischaemia and stasis. Extravasation of blood and endometrial tissue breakdown lead to onset of menstruation.

The menstrual phase begins as the spiral arteries rupture, releasing blood into the uterus and the apoptosing endometrium is sloughed off.

During this period, the functionalis layer of the endometrium is completely shed. Arteriolar and venous blood, remnants of endometrial stroma and glands, leucocytes and red blood cells are all present in the menstrual flow.

Shedding usually lasts ~4 days.

History-taking in gynaecology

It is important to remember that many females can be embarrassed by having to discuss their gynaecological problems, so it is vital to appear confident, friendly, and relaxed.

Although there are parts particular to this history, most of it is the same as the basic outline described in  Chapter 2 and we suggest that readers review that chapter before going on.

Demographic details

Name, age, date of birth, occupation.

Presenting complaint

Ask the patient to tell you in her own words what she perceives the main symptom or symptoms to be. Document each in order of severity.

History of presenting complaint

More detailed questioning will depend on the nature of the presenting complaint—see the following pages. As described on  p.36 ascertain:

- The exact nature of the symptom.
- The onset.
  - When and how it began (e.g. suddenly, gradually—over how long?)
  - If longstanding, why is the patient seeking help now?
- Periodicity and frequency.
  - Is the symptom constant or intermittent?
  - If intermittent, how long does it last each time?
  - What is the exact manner in which it comes and goes?
  - ▸ How does it relate to the menstrual cycle?
- Change over time.
- Exacerbating and relieving factors.
- Associated symptoms.
- The degree of functional disability caused.

Menstrual history

- Age of menarche (first menstrual period).
  - Normally about 12 years but can be as early as 9 or as late as 16.
- Date of last menstrual period (LMP).
- Duration and regularity of periods (cycle).
  - Normal menstruation lasts 4-7 days.
  - Average length of menstrual cycle is 28 days (i.e. the time between first day of one period and the first day of the following period) but can vary between 21 and 42 days in normal women.
- Menstrual flow: whether light, normal, or heavy (see  p.448).
- Menstrual pain: whether occurs prior to or at the start of bleeding.
- Irregular bleeding.
  - E.g. intermenstrual blood-loss, post-coital bleeding etc.
- Associated symptoms.
  - Bowel or bladder dysfunction, pain.
- Hormonal contraception or HRT.
- Age at menopause (if this has occurred).

Past gynaecological history

Record all details of:

- Previous cervical smears, including date of last smear, any abnormal smear results, and treatments received.
- Previous gynaecological problems and treatments including surgery and pelvic inflammatory disease.

Contraception

It is also essential to ask sexually active women of reproductive age about contraception, including methods used, duration of use and acceptance, current method, as well as future plans.

Past obstetric history

- Gravidity and parity: see p.467 for a full explanation.

Document the specifics of each pregnancy:

- Current age of the child and age of mother when pregnant.
- Birth weight.
- Complications of pregnancy, labour, and puerperium.
- Miscarriages and terminations. Note gestation time and complications.

Past medical history

As described in Chapter 2. Pay particular attention to any history of chronic lung or heart disease and make note of all previous surgical procedures.

Drug history

As in Chapter 2. Ask about all medication/drugs taken (prescribed, over the counter and illicit drugs). Record dose, frequency, as well as any known drug allergies.

▸ Make particular note to ask about the oral contraceptive pill (OCP) and hormone replacement therapy (HRT) if not done so already.

Family history

Note especially any history of genital tract cancer, breast cancer and diabetes.

Smoking and alcohol

As always, document fully as described on p.44.

Social history

Take a standard SHx including living conditions and marital status.

This is also an extra chance to explore the impact of the presenting problem on the patient's life—in terms of their social life, employment, home life, and sexual activity.

Abnormal bleeding in gynaecology

Menorrhagia

This is defined as >80ml of menstrual blood loss per period (normal = 20-60ml) and may be caused by a variety of local, systemic, or iatrogenic factors. Menorrhagia is hard to measure, but periods are considered ‘heavy’ if they lead to frequent changes of sanitary towels.

As well as the standard questions for any symptom, ask about:

- The number of sanitary pads/towels used per day and the ‘strength’ (absorbency) of those pads.
- Bleeding through to clothes or onto the bedding at night (‘flooding’).
- The need to use 2 pads at once.
- The need to wear double protection (i.e. pad and tampon together).
- Interference with normal activities.
- ▸ Remember to ask about symptoms of iron deficiency anaemia such as lethargy, breathlessness, and dizziness.

Dysmenorrhoea

This is pain associated with menstruation—thought to be caused by ↑ levels of endometrial prostaglandins during the luteal and menstrual phases of the cycle resulting in uterine contractions. The pain is typically cramping, localized to the lower abdomen and pelvic regions, and radiating to the thighs and back.

Dysmenorrhoea may be primary or secondary:

- *Primary:* occurring from menarche.
- *Secondary:* occurring in females who previously had normal periods (often caused by pelvic pathology).

When taking a history of dysmenorrhoea, take a full pain history as on p.39, a detailed menstrual history (p.446), and ask especially about the relationship of the pain to the menstrual cycle. Remember to ask about the functional consequences of the pain—how does it interfere with normal activities?

Intermenstrual bleeding (IMB)

Intermenstrual bleeding is uterine bleeding which occurs between the menstrual periods.

As for all these symptoms, a full standard battery of questions should be asked (p.38), as full menstrual history (p.446), past medical and gynaecological histories (p.42) and sexual history (p.408).

Ask also about the association of the bleeding with hormonal therapy, contraceptive use and previous cervical smears.

Postcoital bleeding

This is vaginal bleeding precipitated by sexual intercourse. It can be caused by similar conditions to intermenstrual bleeding. Take a full and detailed history as above.

Box 14.1 Some causes of menorrhagia

- Hypothyroidism.
- Intra-uterine contraceptive device (IUCD).
- Fibroids.
- Endometriosis.
- Polyps—cervix, uterus.
- Uterine cancer.
- Infection (STIs).
- Previous sterilization.
- Warfarin therapy.
- Aspirin.
- Non-steroidal anti-inflammatory drugs (NSAIDs).
- Clotting disorders (e.g. von-Willebrand's disease).

Box 14.2 Some causes of secondary dysmenorrhoea

- Pelvic inflammatory disease.
- Endometriosis.
- Uterine adenomyosis.
- Fibroids.
- Endometrial polyps.
- Premenstrual syndrome.
- Cessation of OCP.

Box 14.3 Some causes of intermenstrual bleeding

- *Obstetric* pregnancy, ectopic pregnancy, gestational trophoblastic disease.
- *Gynaecological:* vaginal malignancy, vaginitis, cervical cancer, adenomyosis, fibroids, ovarian cancer.
- *Iatrogenic* anticoagulants, corticosteroids, antipsychotics, tamoxifen, SSRIs, rifampicin, and anti-epileptic drugs (AEDs).

Box 14.4 Some causes of post-coital bleeding

Similar to intermenstrual bleeding, as well as:

Vaginal infection with Chlamydia, gonorrhoea, trichomaniasis or yeast. Also cervicitis.

Amenorrhoea

This is the absence of periods and may be 'primary' or 'secondary'.

- *Primary:* failure to menstruate by 16 years of age in the presence of normal secondary sexual development or failure to menstruate by 14 years in the absence of secondary sexual characteristics.
- *Secondary:* normal menarche, then cessation of menstruation with no periods for at least 6 months.

▸ Amenorrhoea is a normal feature in prepubertal girls, pregnancy, during lactation, postmenopausal females, and in some women using hormonal contraception.

History-taking

A full and detailed history should be taken as described on p.446, and Chapter 2. Ask especially about:

- Childhood growth and development.
- If secondary amenorrhoea:
  - Age of menarche.
  - Cycle days.
  - Day and date of LM P.
  - Presence or absence of breast soreness.
  - Mood change immediately before menses.
- Chronic illnesses.
- Previous surgery (including cervical surgery with can cause stenosis and more obviously oophorectomy and hysterectomy).
- Prescribed medications known to cause amenorrhoea such as phenothiazines, domperidone and metoclopramide (produce either hyperprolactinaemia or ovarian failure).
- Illicit or 'recreational' drugs.
- Sexual history.
- SHx including any emotional stress at school/work/home, exercise and diet—include here any weight gain or weight loss.
- Systems enquiry: include vasomotor symptoms, hot flushes, virilizing changes (e.g. ↑ body hair, greasy skin etc), galactorrhoea, headaches, visual field disturbance, palpitations, nervousness, hearing loss.

Postmenopausal bleeding

This is vaginal bleeding occurring >6 months after the menopause. It requires reassurance and prompt investigation as it could indicate the presence of malignancy.

As well as all the points outlined above, ask about:

- Local symptoms of oestrogen deficiency such as vaginal dryness, soreness, and superficial dyspareunia (p.452).
- Itching (pruritus vulvae—more likely in non-neoplastic disorders).
- Presence of lumps or swellings at the vulva.

Cervical or endometrial malignancy

Often present with profuse or continuous vaginal bleeding or with a bloodstained offensive discharge.

Box 14.5 Some causes of amenorrhoea

- *Hypothalamic:* idiopathic, weight loss, intense exercise.
- *Hypogonadism from hypothalamic or pituitary damage:* tumours, craniopharyngiomas, cranial irradiation, head injuries.
- *Pituitary:* hyperprolactinaemia, hypopituitarism.

- *Delayed puberty:* constitutional delay.
- *Systemic:* chronic illness, weight loss, endocrine disorders (e.g. Cushing's syndrome, thyroid disorders).
- *Uterine:* mullerian agenesis.
- *Ovarian:* PCOS, premature ovarian failure (e.g. Turner's syndrome, autoimmune disease, surgery, chemotherapy, pelvic irradiation, infection).
- *Psychological:* emotional stress at school/home/work.

Box 14.6 Some causes of post-menopausal bleeding

- Cervical carcinoma.
- Uterine sarcoma.
- Vaginal carcinoma.
- Endometrial hyperplasia/carcinoma/polyps.
- Cervical polyps.
- Trauma.
- Hormone replacement therapy.
- Bleeding disorder.
- Vaginal atrophy.

Other symptoms in gynaecology

Pelvic pain and dyspareunia

As with any type of pain, pelvic pain may be acute or chronic. Chronic pelvic pain is often associated with dyspareunia.

Dyspareunia is painful sexual intercourse and may be experienced superficially at the area of the vulva and introitus on penetration or deep within the pelvis. Dyspareunia can lead to failure to reach orgasm, the avoidance of sexual activity and relationship problems.

Box 14.7 Gynaecological versus gastrointestinal pain

Distinguishing between pain of gynaecological and gastrointestinal origin is often difficult. This is because the uterus, cervix, and adnexa share the same visceral innervation as the lower ileum, sigmoid colon, and rectum. You should be careful in your history to rule out a gastrointestinal problem and keep an open mind.

When taking a history of pelvic pain or dyspareunia, you should obtain a detailed history as for any type of pain (📖 p.39) including site, radiation, character, severity, mode and rate of onset, duration, frequency, exacerbating factors, relieving factors, and associated symptoms.

You also need to establish the relationship of the pain to the menstrual cycle. Ask also about:

- Date of LMP.
- Cervical smears.
- Intermenstrual or post-coital bleeding.
- Previous gynaecological procedures (e.g. IUCD, hysteroscopy).
- Previous pelvic inflammatory disease or genitourinary infections.
- Previous gynaecological surgery (adhesion formation?).
- Vulval discharge.
- Bowel habit, nausea, and vomiting (📖 p.226).
- A detailed sexual history (📖 p.408) should also include contraceptive use and the degree of impact the symptoms have on the patient's normal life, and psychological health.

Vaginal discharge

Vaginal discharge is a common complaint during the child-bearing years. As well as the standard questions (📖 p.446) ask about:

- Colour, volume, odour, and presence of blood.
- Irritation.
- ▸ Don't forget to ask about diabetes and obtain a full DHx including recent antibiotic use—both of which may precipitate candidal infection.
- Obtain a full sexual history (📖 p.408). A full gynaecological history should include history of cervical smear testing, use of ring pessaries, and recent history of surgery (↑ risk of vesicovaginal fistulae).
- ▸ Lower abdominal pain, backache, and dyspareunia suggest PID.
- ▸ Weight loss and anorexia may indicate underlying malignancy.

Physiological vaginal discharge

Physiological discharge is usually scanty, mucoid, and odourless. It occurs with the changing oestrogen levels during the menstrual cycle (discharge ↑ in quantity mid-cycle and is a physiological sign of ovulation) and pregnancy.

It may arise from vestibular gland secretions, vaginal transudate, cervical mucus, and residual menstrual fluid.

Pathological vaginal discharge

This usually represents infection (trichomonal or candidal vaginitis) and may be associated with pruritus or burning of the vulval area.

- *Candida albicans:* the discharge is typically thick and causes itching.
- *Bacterial vaginitis:* the discharge is grey and watery with a fishy smell. Seen especially after intercourse.
- *Trichomonas vaginalis:* the discharge is typically profuse, opaque, cream-coloured and frothy. It also has a characteristic 'fishy' smell. This may also be accompanied by urinary symptoms, such as dysuria and frequency.

Box 14.8 Some causes of dyspareunia

- Scars from episiotomy.
- Vaginal atrophy.
- Vulvitis.
- Vulvar vestibulitis.
- PID.
- Ovarian cysts.
- Endometriosis.
- Varicose veins in pelvis.
- Ectopic pregnancy.
- Infections (STIs).
- Bladder or urinary tract disorder.
- Cancer in the reproductive organs or pelvic region.

Vulval symptoms

The main symptom to be aware of is itching or irritation of the vulva (pruritis vulvae). It can be debilitating and socially embarrassing. Embarrassment often delays the woman seeking advice.

Causes include infection, vulval dystrophy, neoplasia, and other dermatological conditions. Ask especially about:

- The nature of onset, exacerbating and relieving factors.
- Abnormal vaginal discharge.
- History of cervical intraepithelial neoplasia—CIN (thought to share a common aetiology with vulval intraepithelial neoplasia—VIN).
- Sexual history.
- Dermatological conditions such as psoriasis and eczema.
- Symptoms suggestive of renal or liver problems (📖 p.234).

- Diabetes.

Urinary incontinence

This is an objectively demonstrable involuntary loss of urine that can be both a social and hygienic problem.

The two most common causes of urinary incontinence in females are *genuine stress incontinence (GSI)* and *detruser over-activity (DO)*. Other less commonly encountered causes include mixed GSI and DO, sensory urgency, chronic voiding problems and fistulae.

When taking a history of urinary incontinence, ascertain under what circumstances they experience the symptom. See also p.236. Remember to ask about the functional consequences on the patient's daily life.

Genuine stress incontinence

Patients notice small amounts of urinary leakage with a cough, sneeze, or exercise. One third may also admit to symptoms of DO.

Ask about:

- Number of children (↑ risk with ↑ parity).
- Genital prolapse.
- Previous pelvic floor surgery.

Detruser over-activity

Urge incontinence, urgency, frequency and nocturia (see p.236). Ask about:

- History of nocturnal enuresis.
- Previous neurological problems.
- Previous incontinence surgery.
- Incontinence during sexual intercourse.
- DHx (see note under ‘the elderly patient’ p.484).

Overflow incontinence

Voiding disorders can result in chronic retention leading to overflow incontinence and ↑ predisposition to infection. The patient may complain of hesitancy, straining, poor flow, and incomplete emptying in addition to urgency and frequency.

Fistulae

Suspect if incontinence is continuous during the day and night.

Genital prolapse

Genital prolapse is descent of the pelvic organs through the pelvic floor into the vaginal canal. In the female genital tract, the type of prolapse is named according to the pelvic organ involved. Some examples include:

- Uterine: uterus.
- Cystocoele: bladder.
- Vaginal vault prolapse: apex of vagina after hysterectomy.
- Enterocoele: small bowel.
- Rectocoele: rectum.

Mild degrees of genital prolapse are often asymptomatic. More extensive prolapse may cause vaginal pressure or pain, introital bulging, a feeling of ‘something coming down’, as well as impaired sexual function.

Uterine descent often gives symptoms of backache especially in older patients.

There might be associated symptoms of incomplete bowel emptying (rectocoele) or urinary symptoms such as frequency or incomplete emptying (cystocoele or cysto-urethrocoele).

Box 14.9 Some causes of genital prolapse

- ***Oestrogen deficiency states:*** such as advancing age and the menopause (atrophy and weakness of the pelvic support structures).
- ***Childbirth:*** prolonged labour, instrumental delivery, fetal macrosomia, ↑ parity.
- ***Genetic factors:*** e.g. spina bifida.
- ***Chronic raised intra-abdominal pressure:*** e.g. chronic cough, constipation.

Box 14.10 Some other common vulval conditions

- ***Dermatitis:*** atopic, seborrhoeic, irritant, allergic, steroid-induced (itch, burning, erythema, scale, fissures, lichenification).
- ***Vulvovaginal candidiasis:*** itch, burning, erythema, vaginal discharge.
- ***Lichen sclerosus:*** itch, burning, dyspareunia, white plaques, atrophic wrinkled surface.
- ***Psoriasis:*** remember to look for other areas of psoriasis; scalp, natal cleft, nails.
- ***Vulval intraepithelial neoplasia:*** itch, burning, multifocal plaques.
- ***Erosive vulvovaginitis:*** erosive lichen planus, pemphigoid, pemphigus vulgaris, fixed drug eruption (chronic painful erosion and ulcers with superficial bleeding).
- ***Atrophic vaginitis:*** secondary to oestrogen deficiency (thin, pale, dry vaginal epithelium. Superficial dyspareunia, minor vaginal bleeding and pain).

Outline gynaecological examination

The gynaecological examination should include a full abdominal examination before proceeding to the pelvic, speculum, and bimanual examinations.

Explain to the patient that you would like to examine their genitalia and reproductive organs and reassure them that the procedure will be quick and gentle.

You should have a chaperone present, particularly if you are male*.

As always, ensure that the room is warm and well lit, preferably with a moveable light source and that you will not be disturbed.

The examination should follow an orderly routine. The authors' suggestion is shown below. It is standard practice to start with the cardiovascular and respiratory systems—this not only gives a measure of the general health of the patient but establishes a ‘physical rapport’ before you examine more delicate or embarrassing areas.

Box 14.11 Framework for the gynaecological examination

- General inspection.
- Cardiorespiratory examination.
- Abdominal examination.
- Pelvic examination
  - External genitalia—inspection.
  - External genitalia—palpation.
  - Speculum examination.
  - Bimanual examination (‘PV’ examination).

▸ Perform bedside urinalysis, if able.

* This is controversial at the time of writing—attitudes vary between countries. In the UK, official advice is that *all* doctors should have a chaperone when performing an intimate examination and the chaperone should be the same sex as the patient. In practice, male doctors performing an examination on a female and females performing an examination on a male should always have a chaperone present whilst the need for a chaperone in other situations is judged at the time.

General inspection and other systems

Always begin with a general examination of the patient (as described in  Chapter 3) including temperature, hydration, coloration, nutritional status, lymph nodes, and blood pressure. Note especially:

- Distribution of facial and body hair, as hirsutism may be a presenting symptom of various endocrine disorders.

- Height and weight.
- Examine the cardiovascular and respiratory systems in turn (see Chapters 7 and 8).

Breast examination is a routine part of the procedure in gynaecology in many countries. In the UK, it should be performed (Chapter 13) if there are symptoms or at first consultation in women over 45 years.

Abdominal examination

A full abdominal examination should be performed (see Chapter 9). Look especially in the periumbilical region for scars from previous laparoscopies and in the suprapubic region where transverse incisions from caesarean sections and most gynaecological operations are found.

Pelvic examination

The patient should be allowed to undress in privacy and, if necessary, to empty her bladder first.

Set-up and positioning

Before starting the examination, always explain to the patient what will be involved. Ensure the abdomen is covered. Ensure good lighting and remember to wear disposable gloves.

Ask the patient to lie on her back on an examination couch with both knees bent up and let her knees fall apart—either with her heels together in the middle or separated.

The lithotomy position, in which both thighs are abducted and feet suspended from lithotomy stirrups is usually adopted when performing vaginal surgery.

Examination of the external genitalia

- Uncover the mons to expose the external genitalia making note of the pattern of hair distribution.
- Apply a lubricating gel to the examining finger.
- Separate the labia from above with the forefinger and thumb of your left hand.
- Inspect the clitoris, urethral meatus, and vaginal opening.
- Look especially for any:
  - Discharge.
  - Redness.
  - Ulceration.
  - Atrophy.
  - Old scars.
- Ask the patient to cough or strain down and look at the vaginal walls for any prolapse.

Palpation

- Palpate the length of the labia majora between the index finger and thumb.
  - The tissue should feel pliant and fleshy.
- Palpate for Bartholin's gland with the index finger of the right hand just inside the introitus and the thumb on the outer aspect of the labium majora.
  - Batholin's glands are only palpable if the duct becomes obstructed resulting in a painless cystic mass or an acute Bartholin's abscess. The latter is seen as a hot, red, tender swelling in the posterolateral labia majora.

Speculum examination

Speculum examination is carried out to see further inside the vagina and to visualize the cervix. It also allows the examiner to take a cervical smear or swabs.

There are different types of vaginal specula (see Fig. 14.1) but the commonest is the Cusco's or bivalve speculum. It is important that you familiarize yourself with the operation of the speculum before examining a patient so that you can concentrate on the findings.

Inserting the speculum

- Explain to the patient that you are about to insert the speculum into the vagina and provide reassurance that this should not be painful.
- Warm the speculum under running water and lubricate it with a water-based lubricant.
- Using the left hand, open the lips of the labia minora to obtain a good view of the introitus.
- Hold the speculum in the right hand with the main body of the speculum in the palm (see Fig. 14.2) and the closed blades projecting between index and middle fingers.
- Gently insert the speculum into the vagina held with your wrist turned such that the blades are in line with the opening between the labia.
- The speculum should be angled downwards and backwards due to the angle of the vagina.
- Maintain a posterior angulation and rotate the speculum through 90° to position handles anteriorly.
- When it cannot be advanced further, maintain a downward pressure and press on the thumb piece to hinge the blades open exposing the cervix and vaginal walls.
- Once the optimum position is achieved, tighten the thumbscrew.

Findings

Inspect the cervix which is usually pink, smooth and regular.

- Look for the external os (central opening) which is round in the nulliparous female and slit-shaped after childbirth.
- Look for cervical erosions which appear as strawberry-red areas spreading circumferentially around the os and represent extension of the endocervical epithelium onto the surface of the cervix.
- Identify any ulceration or growths which may suggest cancer.
- Cervicitis may give a mucopurulent discharge associated with a red, inflamed cervix which bleeds on contact. Take swabs for culture.

Removing the speculum

▸ This should be conducted with as much care as insertion. You should still be examining the vaginal walls as the speculum is withdrawn.

- Undo the thumbscrew and withdraw the speculum.
  - The blades should be held open until their ends are visible distal to the cervix to avoid causing pain.
- Rotate the open blades in an anticlockwise direction to ensure that the anterior and posterior walls of the vagina can be inspected.
- Near the introitus, allow the blades to close taking care not to pinch the labia or hairs.

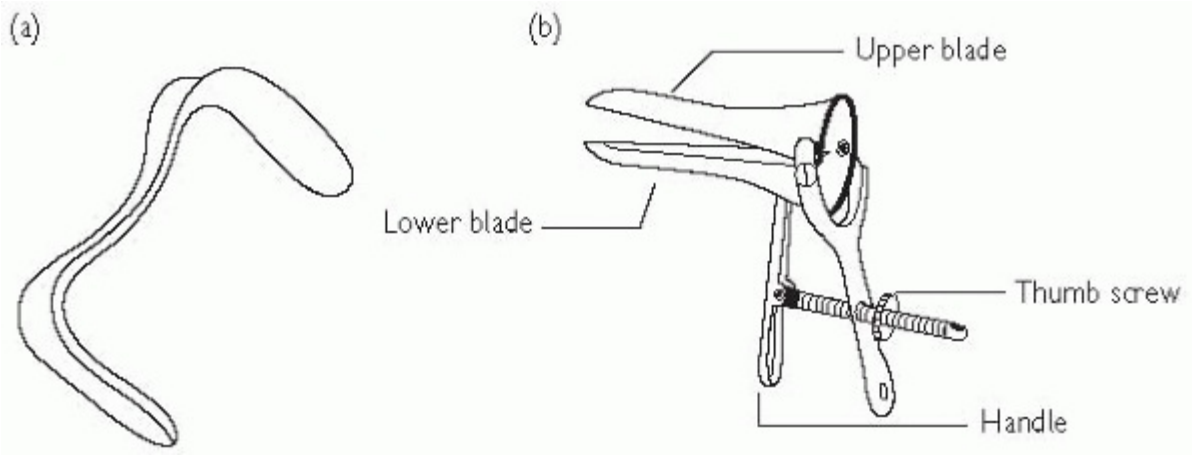

Fig. 14.1 (a) Sim's speculum—used mainly in the examination of women with vaginal prolapse. (b) Cusco's speculum.

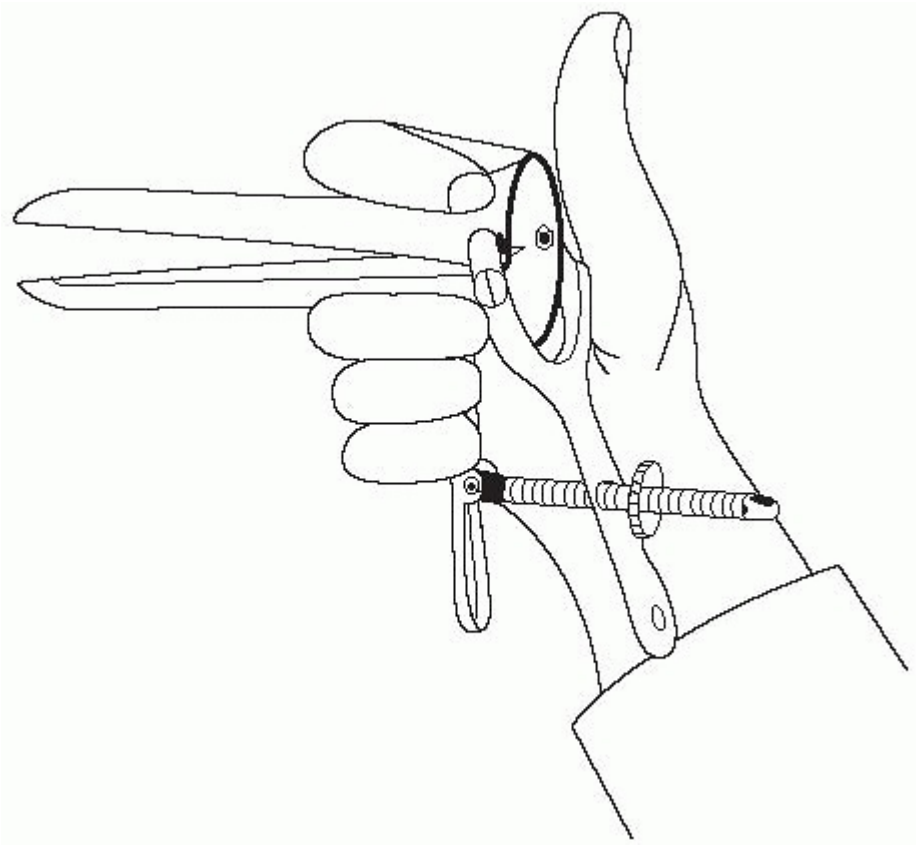

Fig. 14.2 Hold the speculum in the right hand such that the handles lie in the palm and the blades project between the index and middle fingers.

Box 14.12 A word about specula

Many departments and clinical areas now used plastic/disposable specula. These do not have a thumb-screw but a ratchet to open/close the blades. Take care to familiarize yourself with the operation of the speculum *before* starting the examination.

Bimanual examination

Digital examination helps identify the pelvic organs. Ideally the bladder should be emptied, if not already done so by this stage.

This examination is often known as *per vaginam* or simply 'PV'.

Getting started

- Explain again to the patient that you are about to perform an internal examination of the vagina, uterus, tubes, and ovaries and obtain verbal consent.
- The patient should be positioned as described on p.457.
- Expose the introitus by separating the labia with the thumb and forefinger of the gloved left hand.
- Gently introduce the lubricated index and middle fingers of the right hand into the vagina.
  - Insert your fingers with the palm facing laterally and then rotate 90° so that the palm faces upwards.
  - The thumb should be abducted and the ring and little finger flexed into the palm (see Fig. 14.3).

Vagina, cervix and fornices

- Feel the walls of the vagina which are slightly rugose, supple and moist.
- Locate the cervix—usually pointing downwards in the upper vagina.
  - The normal cervix has a similar consistency to the cartilage in the tip of the nose.
  - Assess the mobility of the cervix by moving it from side to side and note any tenderness ('excitation') which suggests infection.
- Gently palpate the fornices either side of the cervix.

Uterus

- Place your left hand on the lower anterior abdominal wall about 4cm above the symphysis pubis.
- Move the fingers of your right 'internal' hand to push the cervix upwards and simultaneously press the fingertips of your left 'external' hand towards the internal fingers.
  - You should be able to capture the uterus between your 2 hands.
- Note the following features of the uterine body:
  - Size: a uniformly enlarged uterus may represent a pregnancy, fibroid or endometrial tumour.
  - Shape: multiple fibroids tend to give the uterus a lobulated feel.
  - Position.
  - Surface characteristics.
  - Any tenderness.
  - ▸ Remember that an anteverted uterus is easily palpable bimanually but a retroverted uterus may not be.
- Assess a retroverted uterus with the internal fingers positioned in the posterior fornix.

Ovaries and fallopian tubes

- Position the internal fingers in each lateral fornix (finger pulps facing the anterior abdominal wall) and place your external fingers over each iliac fossa in turn.

- Press the external hand inwards and downwards and the internal fingers upwards and laterally.
- Feel the adnexal structures (ovaries and fallopian tubes), assessing size, shape, mobility and tenderness.
  - Ovaries are firm, ovoid and often palpable. If there is unilateral or bilateral ovarian enlargement, consider benign cysts (smooth and compressible) and malignant ovarian tumours.
  - Normal fallopian tubes are impalpable.
  - There may be marked tenderness of the lateral fornices and cervix in acute infection of the fallopian tubes (salpingitis).

Masses

It is often not possible to differentiate between adnexal and uterine masses. However, there are some general rules:

- Uterine masses may be felt to move with the cervix when the uterus is shifted upwards while adnexal masses will not.
- If suspecting an adnexal mass, there should be a line of separation between the uterus and the mass and the mass should be felt distinctly from the uterus.
- Whilst the consistency of the mass may help to distinguish its origin in certain cases, an ultrasound may be necessary.

Finishing the examination

- Withdraw your fingers from the vagina.
  - Inspect the glove for blood or discharge.
- Re-drape the genital area and allow the patient to re-dress in privacy-offer them assistance if needed.

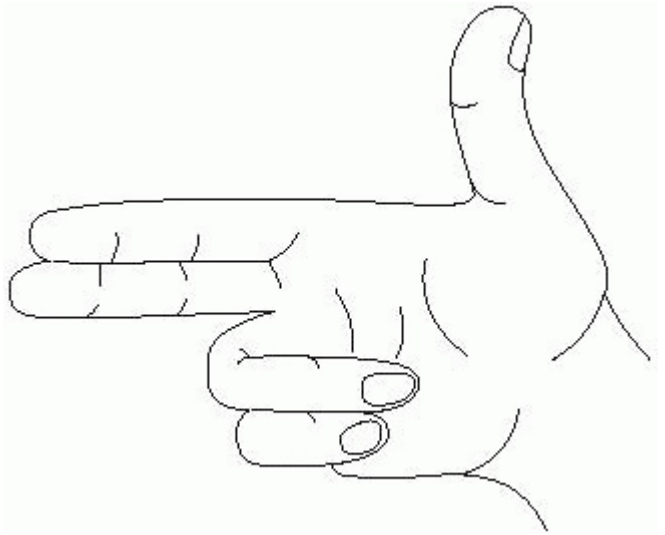

Fig. 14.3 Correct position of the fingers of the right hand for *per vaginam* examination.

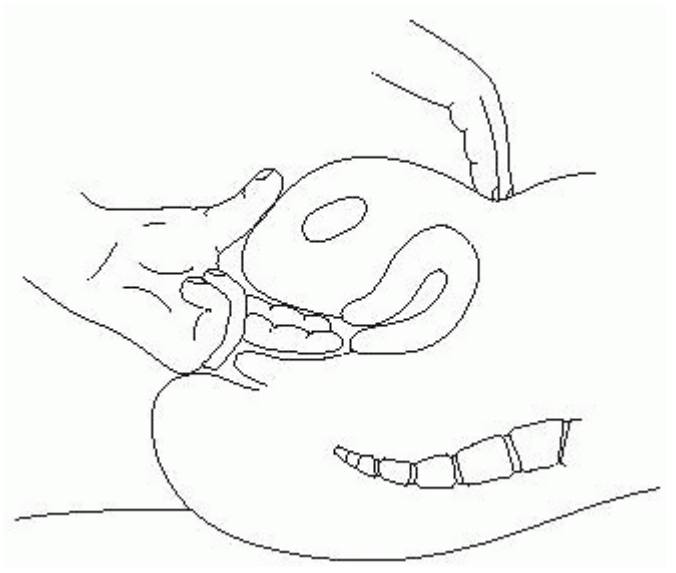

Fig. 14.4 Bimanual examination of the uterus.

Taking a cervical smear

Theory

The UK has a National Screening Program to detect pre-malignant conditions of the cervix. Women between the ages of 20 and 65 years receive an invitation to attend for screening every 3 years. A sample of cells from the squamo-columnar junction are obtained and a cytological examination performed to look for evidence of cerival intraepithelial neoplasia (CIN). This stage of the condition is easily and successfully treated.

The majority of Units are now using liquid based cytology (LBC) in order to minimize the number of inadequate samples.

Equipment

- Specula of different sizes.
- Disposable gloves.
- Request form.
- Sampling device-plastic broom (Cervex-Brush®).
- Liquid-based cytology vial-preservative for sample.
- Patient information leaflet.

Before you start

- Ensure the woman understands purpose of examination.
- Discuss how and when she will receive the results.
- Provide a patient information leaflet.
- Document the date of last menstrual period.
- Document the use of hormonal treatment (e.g. contraception, HRT).
- Record the details of last smear and previous abnormal results.
- Ask about irregular bleeding (e.g. post-coital or post-menopausal).
- Where appropriate, offer screening for Chlamydia infection (under 25 years, symptomatic).

Procedure

- Prepare woman as for vaginal examination remembering to make her comfortable and allow privacy—see p.460.
- Write the patient's identification details on LBC vial.
- Insert Cusco speculum to identify and visualize cervix as on p.460. Record any abnormal features of the cervix
- Insert the plastic broom so that the central bristles of the brush are in the endocervical canal and the outer bristles in contact with the ectocervix (see fig. 14.5).
- Using pencil pressure, *rotate the brush 5 times in a clockwise direction*.
  - The bristles are bevelled to scrape cells only on clockwise rotation.
- Rinse the brush thoroughly in the preservative (ThinPrep®) or break off brush into the preservative (SurePath®).
- Place in transport packaging with completed request form.
- Remove the speculum as p.460.
- Allow the patient to re-dress in privacy.

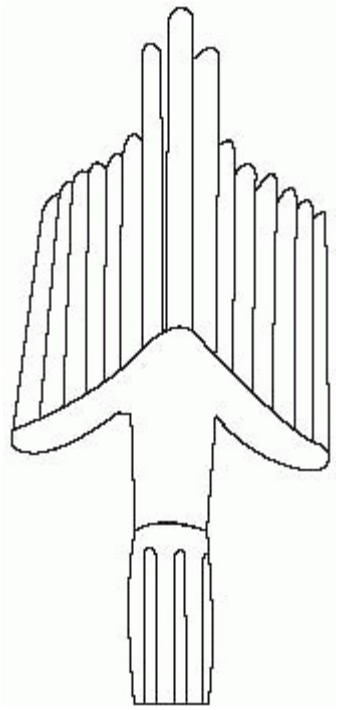

Fig. 14.5 The end of a typical cervex-brush®.

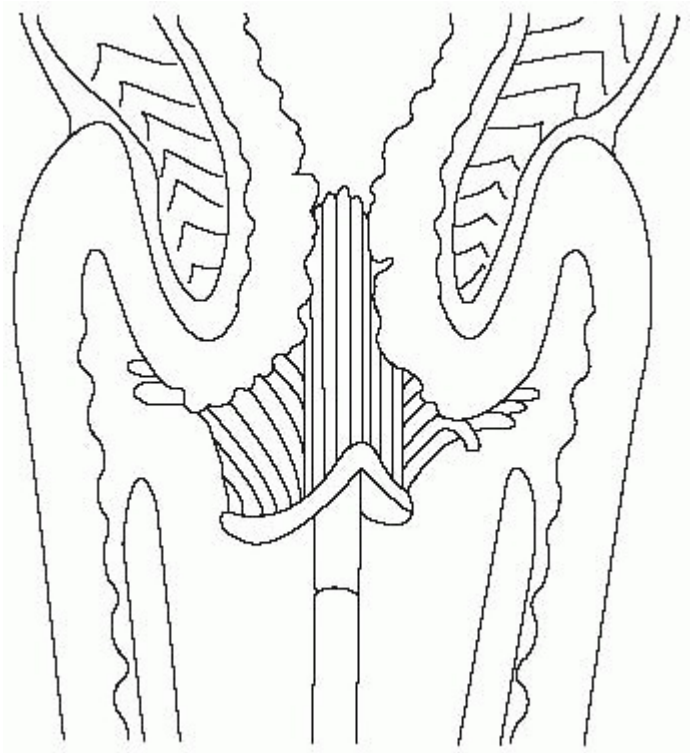

Fig. 14.6 Representation of how to use a cervex-brush®. Note that the longer, central bristles are within the cervical canal whist the outer bristles are in contact with the ectocervix.

Box 14.13 Cervical smears in pregnancy

Cervical smears should not be performed during pregnancy. The increase in cervical mucus (and resultant ↓ in the number of cells obtained) usually renders the sample inadequate and the results unreliable.

History-taking in obstetrics

Although there are parts particular to this history, most is the same as the basic outline described in Chapter 2 and we suggest that readers review that chapter before going on.

The parts of the history detail below are only those that differ from those described in Chapter 2 and earlier in this chapter (p.446).

Demographic details

- Name, age, and date of birth.
- Gravidity and parity—see Box 14.15.

Estimated date of delivery (EDD)

The EDD can be calculated from the last menstrual period (LMP) by Naegele's rule*, which assumes a 28-day menstrual cycle.

Box 14.14 Calculating the EDD

- Subtract 3 months from the first day of the LMP.
- Add on 7 days and 1 year.

If the normal menstrual cycle is <28 days, or >28 days, then an appropriate number of days should be subtracted from or added to the EDD. For example, if the normal cycle is 35 days, 7 days should be added to the EDD.

It is important to also consider at this point any detail that may influence the validity of the EDD as calculated from the LMP; such as:

- Was the last period normal?
- What is the usual cycle length?
- Are the patient's periods usually regular or irregular?
- Was the patient using the oral contraceptive pill in the 3 months prior to conception? If so, calculations based on her LMP are unreliable.

* Named after the German Obstetritian, Franz Naegele following its publication in his *Lehrbuch der Geburtshuelfe* published for midwives in 1830. The formula was actually developed by Harmanni Boerhaave. Boerhaave H. (1744) Praelectiones Academicae in Propias Institutiones Rei Medicae. Von Haller A, ed. Göttingen: Vandehoeck. 5 (part 2): 437.

Current pregnancy

About the patient's general health and that of her fetus. If there is a presenting complaint, the details should be documented in full as on p.38. and p.446. Also ask about:

- Fetal movements.
  - Not usually noticed until 20 weeks' gestation in the first pregnancy and 18 weeks' in the second or subsequent pregnancies.
- Any important laboratory tests or ultrasound scans.
  - Include dates and details of all the scans, especially the first scan (dating or nuchal translucency scan).

Box 14.15 Gravidity and parity

These terms can be confusing and, although it is worth knowing the definitions and how to use them, they should be supplemented with a detailed history and not relied on alone as you may miss subtleties which alter your outlook on the case.

Gravidity

The number of pregnancies (including the present one) to *any* stage.

Parity

The number of live births (at any stage of gestation) and stillbirths after 24 weeks' gestation.

Pregnancies terminating before 24 weeks' gestation can be written after this number with a plus sign.

Examples

- A woman who is currently 20 weeks pregnant and has had 2 normal deliveries:
  - Gravida 3, Para 2.
- A woman who is not pregnant and has had a single live birth and one miscarriage at 17 weeks:
  - Gravida 2, Para 1+1.
- A woman who is currently 25 weeks pregnant, has had 3 normal deliveries, one miscarriage at 9 weeks and a termination at 7 weeks:
  - Gravida 6, Para 3+2.

Twins

There is some controversy as to how to express twin pregnancies. Most people suggest that they should count as 1 for gravidity and 2 for parity—but you should check your local practice on this.

Past obstetric history

Ask about all of her previous pregnancies including miscarriages, terminations and ectopic pregnancies.

For each pregnancy, note:

- Age of the mother when pregnant.
- Antenatal complications.
- Duration of pregnancy.
- Details of induction of labour.
- Duration of labour.
- Presentation and method of delivery.
- Birth weight and sex of infant.

▸ Also enquire about any complications of the puerperal period. The puerperium is the period from the end of the 3rd stage of labour until involution of the uterus is complete (about 6 weeks).

Possible complications include:

- Postpartum haemorrhage.
- Infections of the genital and urinary tracts.
- Deep vein thrombosis.
- Perineal complications such as breakdown of the perineal wounds.
- Psychological complications (e.g. postnatal depression).

Past gynaecological history

- Record all previous gynaecological problems with full details of how the diagnosis was made, treatments received, and the success or otherwise of that treatment.
- Record the date of the last cervical smear and any previous abnormal results.
- Take a full contraceptive history.

Past medical history

Take a full PMH as on  p.42. Note especially those conditions which may have an impact on the pregnancy including:

- Diabetes.
- Thyroid disorders.

- Addison's disease.
- Asthma.
- Epilepsy.
- Hypertension.
- Heart disease.
- Renal disease.
- Infectious diseases such as TB, HIV, syphilis, and hepatitis.
  - Identification of such conditions will allow the obstetrician to consider early referral to a specialist for shared care.
- All previous operative procedures.
- Blood transfusions and receipt of other blood products.
- Psychiatric history—may extend beyond 'simple' post-natal depression.

Drug history

- Take a full DHx (p.44) which should include all prescribed medication, over-the-counter medicines, and illicit drugs.
- Record any drug allergies and their nature.
- If currently pregnant, ensure the patient is taking 400mcg of folic acid daily until 12 weeks' gestation to reduce the incidence of spina bifida.

Smoking and alcohol

A full history should be taken, as always (p.46).

Family history

FHx is an important aspect of the obstetric history and should not be overlooked.

- Ask about any pregnancy-related conditions such as congenital abnormalities, problems following delivery etc.
- Ask also about a FHx of diabetes.
- ▸ Ask especially if there are any known hereditary illnesses. Appropriate counselling and investigations such as chorionic villus sampling or amniocentesis may need to be offered.

Social history

A full standard SHx (p.48) should be taken. Ask about:

- Her partner—age, occupation, health.
- How stable the relationship is.
- If she is not in a relationship, who will give her support during and after the pregnancy?
- Ask if the pregnancy was planned or not.
- If she works, enquire about her job and if she has any plans to return to work.
- You may also use this opportunity to give advice on regular exercises and the avoidance of certain foods. e.g. tuna (high Mg content) soft cheeses (risk of listeria) calf's liver (high vitamin A content). See the *Oxford Handbook of Obstetrics and Gynaecology*[1] for more details.

[1] Arulkumaran (2005). *Oxford Handbook of Obstetrics and Gynaecology*. Oxford University Press, Oxford.

Box 14.16 A word about deliveries

The verb ‘to deliver’ is often misused by students of obstetrics as it is often misused by the population at large.

Babies are not delivered.

In fact, the mothers are ‘delivered of’ the child-as in being relieved of a burden.

Check your nearest dictionary!

Presenting symptoms in obstetrics

Bleeding—during pregnancy

Treat as any symptom. In addition, you should build a clear picture of how much blood is being lost, when and how it is affecting the current pregnancy.

After establishing an exact time-line and other details about the symptom, ask about:

- Exact nature of the bleeding (fresh/old).
- Amount of blood lost.
  - Number of sanitary pads used daily.
- Presence of clots (and, if present, size of those clots).
- Presence of pieces of tissue in the blood.
- Presence of mucoid discharge.
- Fetal movement.
- Associated symptoms such as abdominal pain (associated with placental abruption; placenta praevia is painless).
- Possible trigger factors—recent intercourse, injuries.
- Any history of cervical abnormalities—and the result of the last smear.

Abdominal pain

A full pain history should be taken as on p.39 including site, radiation, character, severity, mode and rate of onset, duration, frequency, exacerbating factors, relieving factors, and associated symptoms.

Take a full obstetric history and systems enquiry. Ask especially about a past history of pre-eclampsia, pre-term labour, peptic ulcer disease, gallstones, appendicectomy, cholecystectomy.

Remember that the pain may be unrelated to the pregnancy so keep an open mind! Causes of abdominal pain in pregnancy include:

- *Obstetric:* preterm/term labour, placental abruption, ligament pain, symphysis pubis dysfunction, pre-eclampsia/HELLP syndrome, acute fatty liver of pregnancy.
- *Gynaecological:* ovarian cyst rupture, torsion, haemorrhage, uterine fibroid degeneration.
- *Gastrointestinal:* constipation, appendicitis, gallstones, cholecystitis, pancreatitis, peptic ulceration.
- *Genitourinary:* cystitis, pyelonephritis, renal stones, renal colic.

Labour pain

This is usually intermittent, regular in frequency and associated with tightening of the abdominal wall.

Box 14.17 Some causes of vaginal bleeding in early pregnancy

We suggest the reader turns to the *Oxford Handbook of Obstetrics and Gynaecology*[1] for more detail.

Ectopic pregnancy

- *Symptoms:* light bleeding, abdominal pain, fainting if pain and blood loss is severe.
- *Signs:* closed cervix, uterus slightly larger and softer than normal, tender adnexal mass, cervical motion tenderness.

Threatened miscarriage

- *Symptoms:* light bleeding. Sometimes: cramping, lower abdominal pain.

- *Signs:* closed cervix, uterus corresponds to dates. Sometimes, uterus is softer than normal.

Complete miscarriage

- *Symptoms:* light bleeding. Sometimes: light cramping, lower abdominal pain and a history of expulsion of products of conception.
- *Signs:* uterus smaller than dates and softer than normal. Closed cervix.

Incomplete miscarriage

- *Symptoms:* heavy bleeding. Sometimes: cramping lower abdominal pain, partial expulsion of products of conception.
- *Signs:* uterus smaller than dates and cervix dilated.

Molar pregnancy

- *Symptoms:* heavy bleeding, partial expulsion of products of conception which resemble grapes. Sometimes: nausea and vomiting, cramping lower abdominal pain, history of ovarian cysts.
- *Signs:* dilated cervix, uterus larger than dates and softer than normal.

Information adapted from the WHO department of reproductive health research publication, 'Vaginal bleeding in early pregnancy'.

[1] Arulkumaran (2005). *Oxford Handbook of Obstetrics and Gynaecology*. Oxford University Press, Oxford.

Bleeding-after pregnancy

This is called 'post-partum haemorrhage' or PPH.

- ***Primary PPH:*** >500ml of blood loss within 24 hours following delivery.
- ***Secondary PPH:*** any excess bleeding between 24 hours and 6 weeks post delivery. (No amount of blood is specified in the definition).

▸ Take a full history as for bleeding during pregnancy on p.470. Ask also about symptoms of infection—an important cause of secondary PPH.

Hypertension

Hypertension is a common and important problem in pregnancy and you should be alert to the possible symptoms which can result from it such as headache, blurred vision, vomiting and epigastric pain after 24 weeks, convulsions or loss of consciousness.

Pregnancy-induced hypertension

Two readings of diastolic blood pressure 90-110, 4 hours apart after 20 weeks gestation. No proteinuria.

Mild proteinuric pregnancy-induced hypertension

Two readings of diastolic blood pressure 90-110, 4 hours apart after 20 weeks gestation and proteinuria 2+.

Severe proteinuric pregnancy-induced hypertension

Diastolic blood pressure 110 or greater after 20 weeks' gestation and proteinuria 3+. Other symptoms may include: hyper-reflexia, headache, clouding of vision, oligura, abdominal pain, pulmonary oedema.

Eclampsia

Convulsions associated with raised blood pressure and/or proteinuria beyond 20 weeks gestation. May be unconscious.

Box 14.18 Some causes of bleeding in 2nd/3rd trimesters (>24 weeks)

This is known as 'antepartum haemorrhage' (APH). See the *Oxford Handbook of Obstetrics and Gynaecology*[1] for more detail.

Placenta praevia

The placenta is positioned over the lower pole of the uterus, obscuring the cervix. Bleeding is usually after 28 weeks and often precipitated by intercourse. Findings may include a relaxed uterus, fetal presentation not in pelvis and normal fetal condition.

Placental abruption

This is detachment of a normally located placenta from the uterus before the fetus is delivered. Bleeding can occur at any stage of

the pregnancy. Possible findings include a tense, tender uterus, ↓ or absent fetal movements, fetal distress, or absent fetal heart sounds.

Box 14.19 Some causes of post-partum haemorrhage

Primary

- Uterine atony (most frequent cause).
- Genital tract trauma.
- Coagulation disorders.
- Retained placenta.
- Uterine inversion.
- Uterine rupture.

Secondary

- Retained products of conception.
- Endometritis.
- Infection.

Box 14.20 Risk factors for post-partum haemorrhage

Nulliparity, multiparity, polyhydramnios, prolonged labour, multiple gestation, previous PPH or APH, pre-eclampsia, coagulation abnormalities, genital tract lacerations, Asian or Hispanic ethnicity.

Box 14.21 Minor symptoms of pregnancy

These so-called 'minor' symptoms of pregnancy are often experienced by a number of woman as normal, physiological changes occur. This is not to say that they should be ignored as they may point to pathology.

Nausea and vomiting

The severity varies greatly and is more common in multiple pregnancies and molar pregnancies. Persistence of vomiting may suggest pathology such as infections, gastritis, biliary tract disease or hepatitis.

Heartburn/gastro-oesophageal reflux

Heartburn is a frequent complaint during pregnancy due partially to compression of the stomach by the gravid uterus. See p.228.

Constipation

Often secondary to ↑ progesterone. Improves with gestation (p.230).

Shortness of breath

Due to dilatation of the bronchial tree secondary to ↑ progesterone. Peaks at 20-24 weeks. The growing uterus also has an impact. Other possible causes (such as PE) need to be considered. See p.198.

Fatigue

Very common in early pregnancy, peaking at the end of the first trimester. Fatigue in late pregnancy may be due to anaemia.

Insomnia

Due to anxiety, hormonal changes and physical discomfort.

Pruritus

Generalized itching in the third trimester may resolve after delivery. Biliary problems should be excluded (p.234).

Haemorrhoids

May resolve after delivery.

Varicose veins

Especially at the feet and ankles.

Vaginal discharge

Exclude infection and spontaneous rupture of the membranes.

Pelvic pain

Stretching of pelvic structures can cause ligament pain which resolves in the second half of the pregnancy. Symphysis-pubis dysfunction causes pain on abduction and rotation at the hips and on mobilization.

Backache

Often first develops during the 5-7th months of pregnancy.

Peripheral paraesthesiae

Fluid retention can lead to compression of peripheral nerves such as carpal tunnel syndrome. Other nerves can be affected, e.g. lateral cutaneous nerve of the thigh.

Outline obstetric examination

Explain to the patient that you would like to examine their womb and baby and reassure them that the procedure will be quick and gentle.

You should have a chaperone present, particularly if you are male.

As always, ensure that the room is warm and well lit, preferably with a moveable light source and that you will not be disturbed.

As for the gynaecological examination, you should follow an orderly routine. The authors' suggestion is shown below. It is standard practice to start with the cardiovascular and respiratory systems-this not only gives a measure of the general health of the patient but establishes a 'physical rapport' before you examine more delicate or embarrassing areas.

Box 14.22 Framework for the obstetric examination

- General inspection.
- Cardiorespiratory examination.
- Abdominal inspection.
- Abdominal palpation.
  - Uterine size.
  - Fetal lie.
  - Fetal presentation.
  - Engagement.
  - Amniotic fluid estimation.
- Auscultation of the fetal heart.
- Vaginal examination.

▸ Perform bedside urinalysis (particularly protein) if able.

General inspection

Always begin with a general examination of the patient (as in Chapter 3) including temperature, hydration, coloration, nutritional status, lymph nodes, and blood pressure. Note especially:

- Any brownish pigmentation over the forehead and cheeks known as chloasma.
- Distribution of facial and body hair, as hirsutism may be a presenting symptom of various endocrine disorders.
- Height, weight, and calculate BMI (p.66).
- ▸ Blood pressure should be measured in the left lateral position at 45° to avoid compression of the IVC by the gravid uterus.
- Anaemia is a common complication of pregnancy so examine the mucosal surfaces and conjunctivae carefully (p.58).
- Examine the cardiovascular and respiratory systems in turn (see Chapters 7 and 8).
  - Flow murmurs are common in pregnancy and, although usually of no clinical significance, must be recorded in detail.
- A routine breast examination is not normally indicated unless a female patient complains of breast symptoms, in which case you must carefully look for any pathology such as cysts or solid nodules.

Abdominal examination

Inspection

Look for the abdominal distension caused by the gravid uterus rising from the pelvis. Look also for:

- Asymmetry.
- Fetal movements.
- Surgical scars.
  - Pubic hairline (transverse suprapubic Pfannenstiel incision).
  - Paraumbilical region (laparoscopic scars).
- Cutaneous signs of pregnancy including:
  - Linea nigra (black line) which stretches from the pubic symphysis upwards in the midline.
  - Red stretch marks of current pregnancy (striae gravidarum).
  - White stretch marks (striae albicans) from a previous pregnancy.
  - Other areas that can undergo pigmentation in pregnancy include the nipples, vulva, umbilicus and recent abdominal scars.
- Umbilical changes:
  - Flattening as pregnancy advances.
  - Eversion secondary to ↑ intra-abdominal pressure (e.g. caused by multiple pregnancies or polyhydraminios).

Palpation

Before palpating the abdomen, always enquire about any areas of tenderness and visit those areas last.

Palpation should start as for any standard abdominal examination (Chapter 9) before proceeding to more specific manoeuvres in an obstetric examination.

Uterine size

This provides an estimation of gestational age in weeks and is objectively measured and expressed in centimetres as the *symphysial-fundal height* (the distance from the symphysis pubis to the upper edge of the uterus).

Box 14.23 The symphysial-fundal height (cm) ≈ weeks of gestation

Between 16-36 weeks, there is a margin of error of ±2cm, ±3cm at 36-40 weeks, and ±4cm at 40 weeks onwards.

- You need a tape-measure for this-don't start without it!
- Use the ulnar border of the left hand to press firmly into the abdomen just below the sternum.
- Move the hand down the abdomen in small steps until you can feel the fundus of the uterus.
- Locate the upper border of the bony pubic symphysis by palpating downward in the midline starting from a few centimetres above the pubic hair margin.
- Measure the distance between the two points that you have found in centimetres using a flexible tape-measure.

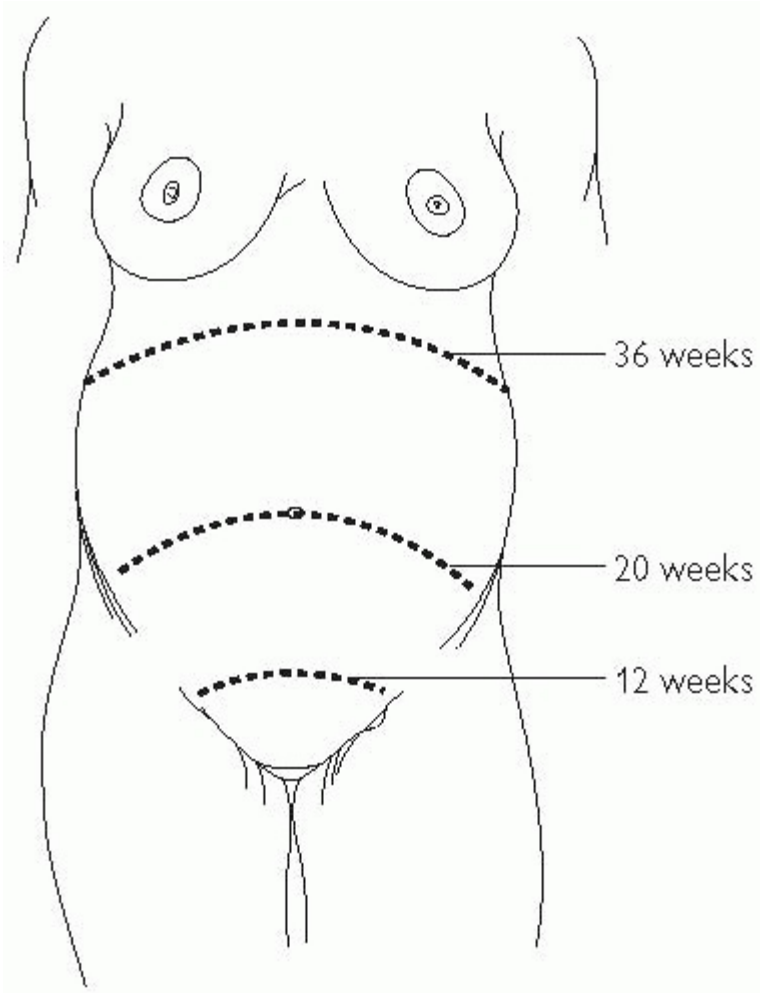

Fig. 14.7 The distance between the fundus (upper border of the uterus) and the pubic symphysis can be used as a guide to the number of weeks' gestation. You can also, therefore, judge whetherthe uterus is smaller or largerthan expected which may point to problems with the pregnancy.

Box 14.24 Uterine size—milestones

- The uterus first becomes palpable at 12 weeks' gestation.
- 20 weeks' gestation = at the level of the umbilicus.
- 36 weeks' gestation = at the level of the xiphisternum.

Fetal lie

This describes the relationship between the long axis of the fetus and the long axis of the uterus and, in general, can be:

- ***Longitudinal:*** the long axis of the fetus matches the long axis of the uterus. Either the head or the breech will be palpable over the pelvic inlet.
- ***Transverse:*** the fetus lies at right angles to the uterus and the fetal poles are palpable in the flanks.
- ***Oblique:*** the long axis of the fetus lies at an angle of 45° to the long axis of the uterus, the presenting part will be palpable in one of the iliac fossae.

Examination technique

The best position is to stand at the mother's right side, facing her feet.

- Put your left hand along the left side of the uterus.
- Put your right hand on the right side of the uterus.

- Palpate systematically towards the midline with one and then the other hand—use ‘dipping’ movements with flexion of the MCP joints to feel the fetus within the amniotic fluid.
- You should feel the fetal back as firm resistance or the irregular shape of the limbs.
- You should now palpate more widely using the 2-handed technique above to stabilize the uterus and attempt to locate the head and the breech.
    - The head can be felt as a smooth, round object that is ballotable—that is, it can be ‘bounced’ (gently) between your hands.
    - The breech is softer, less discrete and is not ballotable.

Fetal presentation

This is the part of the fetus that presents to the mother's pelvis. Possible presenting parts include:

- ***Head:*** ‘cepahalic’ presentation. One option in a longitudinal lie.
- ***Breech:*** ‘podalic’ presentation. The other option in a longitudinal lie.
- ***Shoulder:*** seen in a transverse lie.

Examination technique

- Stand at the mother's right side, facing her feet.
- Place both hands on either side of the lower part of the uterus
- Bring the hands together firmly but gently.
    - You should be able to feel either the head, breech, or other part as described above under ‘fetal lie’.

It is also possible to use a one-handed technique (Paulik's grip) to feel for the presenting part—this is best left to obstetricians. In this, you use a cupped right hand to hold the lower pole of the uterus. This is possible in ~95% of pregnancies at about 40 weeks.

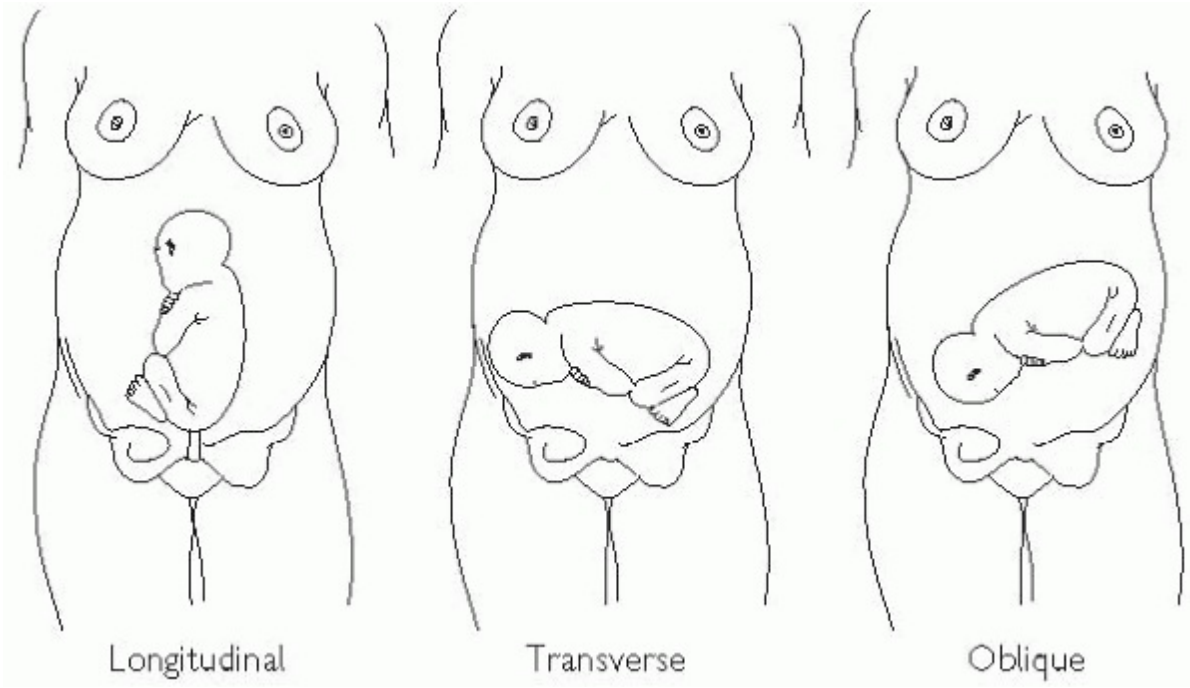

Fig. 14.8 Some examples of fetal lie.

Engagement

When the widest part of the fetal skull is within the pelvic inlet, the fetal head is said to be ‘engaged’.

In a cephalic presentation, palpation of the head is assessed and expressed as the number of fifths of the skull palpable above the pelvic brim. A fifth of a fetal skull is roughly equal to a finger breath on an adult hand.

- The head is engaged when 3 or more fifths are within the pelvic inlet—that is when 2 or less fifths are palpable.
- When 3 or more fifths are palpable, the head is not engaged.

Number of fetuses

The number of fetuses present can be calculated by assessing the number of fetal poles (head or breech) present.

- If there is one fetus present, 2 poles should be palpable (unless the presenting part is deeply engaged).
- In a multiple pregnancy, you should be able to feel all the poles except one—as one is usually tucked away out of reach.

Amniotic fluid/liquor volume estimation

The ease with which fetal parts are palpable can give an indication as to the possibility of ↓ or ↑ amniotic fluid volume.

- ↑ volume will give a large-for-dates uterus that is smooth and rounded. The fetal parts may be almost impossible to palpate.
- ↓ volume may give a small-for-dates uterus. The fetus will be easily palpable giving an irregular, firm outline to the uterus.

Percussion

This is usually unhelpful in an obstetric examination unless you suspect polyhydramnios (increased amniotic fluid volume), in which case, you may wish to attempt to elicit a fluid thrill (📖 p.260).

Auscultation

Auscultation is used to listen to the fetal heart rate (FHR). This is usually performed using an electronic hand-held Doppler fetal heart rate monitor and can be used as early as 14 weeks.

Using Pinard's fetal stethoscope

A Pinard's fetal stethoscope is not useful until 28 weeks' gestation. It is a simple-looking device rather like an old-fashioned ear-trumpet (Fig. 14.9).

- Place the bell of the instrument over the anterior fetal shoulder—where the fetal heart sounds are best heard.
- Press your left ear against the stethoscope so as to hold it between your head and the mother's abdomen in a ‘hands-free’ position or hold the instrument lightly with one hand.
- Press against the opposite side of the mother's abdomen with your other hand so as to stabilize the uterus.
- It should sound like a distant ticking noise. The rate varies between 110 and 150/minute at term and should be regular.
- ▸ Record the rate and rhythm of the fetal heart.

Vaginal examination

Vaginal examination allows you to assess cervical status before induction of labour. You should attempt this only under adequate supervision if you are unsure of the procedure.

This examination allows you to assess the degree of cervical dilatation (in centimetres) using the examining fingers.

Examination of the vagina and cervix should be performed under aseptic conditions in the presence of ruptured membranes or in cases with abnormal vaginal discharge.

Technique

The examination should be performed as described on 📖 p.462. The findings take experience to recognize. The student should not shy away from this examination due to its intimate nature.

Findings

Assess:

- Degree of dilation.
  - Full dilation of the cervix is equivalent to 10cm.
  - Most obstetric departments will have plastic models of cervices in various stages of dilatation which you can practice feeling.
- The length of the cervix.
  - Normal ~3cm but shortens as the cervix effaces secondary to uterine contraction.
- The consistency of the cervix which can be described as:
  - Firm.
  - Mid-consistency.
  - Soft (this consistency facilitates effacement and dilatation).
- Position.
  - As the cervix undergoes effacement and dilatation it tends to be pulled from a posterior to an anterior position.
- Station of the presenting part.
  - The level of the head above or below the ischial spines which may be estimated in centimetres.

Fig. 14.9 A Pinard's stethoscope.

The elderly patient

It is easy to be seduced into thinking that the principal focus should be on very ‘medical’ diagnoses such as urinary tract infections, which contribute to significant morbidity (and mortality) in older people.

Continence issues are sadly overlooked in most clinical assessments—despite costing the UK National Health Service £424m per annum (on figures from 2000). Large-scale surveys of prevalence have shown up to 20% of women over 40 reporting difficulties with continence; so whilst more common in older people, you should always be mindful of problems in younger adults too.

Although continence issues are one of the ‘Geriatric Giants’ of disease presentation, it is important to recall the physiology of the post menopausal changes—such as vaginal atrophy and loss of secretions—which can complicate urinary tract infections, continence and utero-vaginal prolapse in older patients.

Assessment

- ***Tact and understanding:*** although problems are common, patients may be reluctant to discuss them, or have them discussed in front of others. Engaging in a discussion about bladder and/or sexual function can seem daunting—but if done empathetically, remembering never to appear to judge, or be embarrassed—you may reveal problems that have seriously affected your patient's quality of life. Treating problems such as these, even with very simple interventions, can be of immeasurable value to the patient.
- ***Holistic assessment of urinary problems:*** learn to think when asking about bladder function, and work out a pattern of dysfunction—e.g. bladder instability or stress incontinence. Remember that bladder function may be disrupted by drugs, pain, lack of privacy. Continence issues may reflect poor mobility, visual and cognitive decline.
- ***Genital symptoms:*** never forget to consider vaginal or uterine pathology—view postmenopausal bleeding with suspicion. Discharges may represent active infection (if candida—consider diabetes) or atrophic vaginitis (see opposite).
- ***Past medical history:*** pregnancies and previous surgery in particular may help point to a diagnosis of stress incontinence. Are urinary tract infections recurrent—has bladder pathology been excluded?
- ***Drugs:*** many are obvious—diuretics and anticholinergics; some are more subtle—sedatives may provoke nocturnal loss of continence; Does your patient drink tea or coffee?
- ***Tailored functional history:*** the cornerstone of these pages—and of any assessment you perform. This largely relates to bladder function—is the lavatory up or down? How are the stairs? Does your patient already have continence aids—bottles/commodes/pads, and do they manage with them?

Box 14.25 A word on atrophic vaginitis

Up to 40% of postmenopausal women will have symptoms and signs of atrophic vaginitis and the vast majority will be elderly and may be reluctant to discuss this with their doctors. A result of oestrogen deficiency, the subsequent ↑ vaginal pH and thinned endometrium lead to both genital and urinary symptoms and signs. ↓ in vaginal lubrication presents with dryness, pruritus, and discharges, accompanied by an ↑ rate of prolapse. Urinary complications can result in frequency, stress incontinence, and infections.

Careful physical examination often makes the diagnosis clear with labial dryness, loss of skin turgidity, and smooth, shiny vaginal epithelium. A range of treatment options including topical oestrogens, simple lubricants, and continued sexual activity when appropriate are all key interventions to manage this common condition.

We thank Dr Richard Fuller for providing this page.

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Chapter 15

The Psychiatric Assessment

Approach to the psychiatric assessment

In taking a psychiatric history and assessing mental state, it is crucial to communicate to the patient with empathy, respect, competence, and interest in a non-judgemental fashion. This approach will create an atmosphere of trust that encourages the patient to talk honestly about their innermost feelings and thoughts.

Central to the psychiatric interview process is the art of active listening to what is said and an awareness of any unspoken feelings between patient and assessor.

Be prepared to spend anywhere from 30 minutes to an hour, depending on the circumstances, conducting an interview. This might seem a daunting task in the early stages, particularly as patients rarely find a narrative. However, staying on track is often made easier by remembering to write out the headings for parts of the assessment in advance. Use A4 paper and number the pages.

Preparation and preliminary considerations

The room

Before proceeding to questioning, adequate preparations should be made regarding the place where the assessment is to be carried out. An interview room should be a safe environment, especially when seeing a patient who is potentially violent.

- Inform your colleagues of your location.
- You should know where to locate, and how to use, the panic button.
- You should ideally be accompanied by a colleague if you are seeing a patient with a history of violence.
- Remove any objects that might pose a danger (i.e. those that can be used as a weapon).
- Know your nearest exit point and ensure that it is open or unblocked.
- Never allow the patient to come between you and the door.
- Ensure adequate privacy and lighting.
- Ideally, the patient should be sitting off-centre, so that all of their body may be seen but without the situation appearing too threatening.
- The height of your seats should be equal or similar.

Conduct of the interview

Begin by introducing yourself, explaining who you are and the purpose of your assessment. Use a handshake—a widespread sign of introduction and welcome. Establish whether or not the patient wishes a friend or relative to be present (and whether you feel it is appropriate).

The interview should generally start in an informal way to establish a friendly and concerned rapport, this might involve a short period of neutral conversation.

Try to avoid leading or direct questions. Remember to use relatively broad ‘open’ general questions and ↑ the level of specific ‘closed’ questions for further clarification. Allow breaks and digressions within reason, especially with sensitive individuals. At appropriate intervals, clarification of what the patient has said by repeating sentences and asking them to confirm is a useful strategy.

Examination

In psychiatry, you should be examining the patient's mental state and this is described later. However, don't overlook physical examination as this is often an important part of the assessment. Physical examination is described elsewhere in this book.

Box 15.1 Framework for the psychiatric assessment

History

- Name, age, marital status, occupation, ethnic origin, and religion.

- Source, mode, and reason for referral.
- Presenting complaint.
- History of presenting complaint.
- Risk assessment.
- Past psychiatric history.
- Past medical history.
- Drug history.
- Family history.
- Personal history.
  - Birth and early development.
  - School.
  - Occupational history.
  - Psychosexual history.
  - Marital history.
  - Children.
  - Forensic history.
- Premorbid personality.
- Social history.

Mental state examination

- Appearance and behaviour.
- Speech.
- Mood.
- Thought content.
- Perception.
- Cognitive functioning.
- Insight.

Physical examination

As appropriate—described elsewhere in the book.

History

The psychiatric history is very similar in structure to the standard medical history described in Chapter 2. Symptoms and issues should be dealt with in the same way (see Box 15.2).

Demographics

Start by making a note of the patient's name, age, marital status, occupation, ethic origin, and religion.

Source, mode, and reason for referral

Record here all the information you have about the patient from other sources—relatives, carers, social workers, counsellors, primary care team, and police.

- Who has asked for the individual to be seen and why?
- What was the mode of referral—informal or formal (under section of the Mental Health Act?).

Presenting complaint

Obtain a brief description of the principal complaint(s) and the time frame of the problem in the individual's own words.

This can, of course, be difficult if the patient is psychotic and does not believe a problem exists. In these cases, try to comment on the presenting complaint as described by an informant.

History of presenting complaint

This is a detailed account of the presenting problems in chronological order (as for any other kind of symptom as in Chapter 2) including:

- Onset of illness (when was the patient last well?)
- How did the condition develop?
- The severity of the patient's symptoms.
- Precipitating factors (including any significant life events preceding the onset of the symptoms).
- Exacerbating factors (what makes the symptoms worse?)
- Relieving factors (what makes the symptoms better?)
- How has it affected his/her daily life, pattern or routine (effect on interpersonal relationships, working capacity etc.).
- Treatment history. Include treatment tried during the course of the present illness, previous drug treatments, electroconvulsive therapy, and psychosocial interventions.
- Associated symptoms.
- Systematic enquiry. Similar to the standard medical history, run through other psychiatric symptoms and ask the patient if they have experienced them. Explore related symptoms-if the patient admits to a few depressive symptoms, ask about other symptoms of depression.

Box 15.2 The factors asked about for a physical or mental symptom

The following is repeated from Chapter 2. You should treat psychiatric symptoms in a similar way but remember that the patient may not regard their issue as a 'symptom' so tailor your language carefully.

- The exact nature of the symptom.
- The onset:
  - The date it began.
  - How it began (e.g. suddenly, gradually-over how long?)
  - If longstanding, why is the patient seeking help now?
- Periodicity and frequency:
  - Is the symptom constant or intermittent?
  - How long does it last each time?
  - What is the exact manner in which it comes and goes?
- Change over time:
  - Is it improving or deteriorating?
- Exacerbating factors:
  - What makes the symptom worse?
- Relieving factors:
  - What makes the symptom better?
- Associated symptoms.

Box 15.3 Tailoring the history

In the taking of a history in any specialty, you should mould your questions and the situation depending on what is said. Also, information may not be provided by the patient in the order you would like.

This is particularly true of psychiatry—if they are talking freely, you may find the patient providing information that comes under a number of different sub-headings in your history. You should be flexible, note the information in the appropriate places, and then 'fill in the gaps' with direct questions.

Risk assessment

This includes not only an assessment of self-harm but also of the possibility of harm to others. This should be broached in a serious and sensitive manner. Some useful questions in assessing suicide risk:

- How do you feel about the future?
- Does life seem worth living?
- Do you have thoughts of hurting or harming yourself?
- Have you ever thought of ending it all?

If suicidal thoughts are present, enquire as to how often they occur and if the patient has made a specific plan—and what the plan is.

- Ask about the means e.g. prescribed and over-the-counter drugs, guns, knives.
- Explore for feelings of excessive guilt and loss of self-esteem.

Previous history of self-harm

Ask about previous attempts—when, where, how, and why. Ask in detail about the most recent attempt:

- What events lead up to the attempt?
- Were there any specific precipitating factors?
- Was there concurrent use of drugs and alcohol?
- What were the methods used?
- Was it planned?
- Was there a suicide note?
- Were there any active attempts made to avoid being discovered?
- What is the meaning of the action (wanted to die, share distress)?
- Also ask about the circumstances surrounding discovery and how they were brought to medical attention (if at all).
- Was this what he/she expected?

Protective factors for suicide

The factors that would stop a person attempting suicide. Record what social supports are available to the person (friends, CPN, church, GP, etc.).

Assessing homicidal intent

▸ If faced with patient expressing homicidal intent, you should inform a senior colleague ± the police immediately.

Some helpful questions to assess a homicidal/violent patient include:

- Are you upset with anyone?
- Do you have thoughts of hurting anyone?
- Have you made plans to harm someone?
- How would you harm them? (It is important to establish whether the patient has actually made plans for carrying out the action.)

Box 15.4 Protective factors for suicide

- Strong family and social connections.
- Hopefulness, good skills in problem solving.
- Cultural or religious beliefs discouraging suicide.
- Responsibility for children.

Box 15.5 Factors that may precipitate suicide

The over-riding theme here is ‘loss’. Loss of occupation, independency, family member, friend, social supports, or freedom.

- Death, separation, or divorce.
- Imprisonment, or threat of.
- Humiliating event.
- Job loss.
- A reminder of a past loss.
- Unwanted pregnancy.

Box 15.6 Risk factors for suicide

Biological

- Age >40 years.
- Male sex.

Medical and psychiatric history

- Previous suicide attempts.
- Previous deliberate self harm.
- Psychiatric disorder (depression, substance misuse, schizophrenia, personality disorder, obsessive-compulsive disorder, panic disorder).
- Chronic physical illness.
- History of trauma or abuse.
- Substance misuse (including alcohol).

Personality

Impulsivity, poor problem solving skills, aggression, perfectionism, low self-esteem.

Family history

Suicide or parasuicide, depression, substance misuse.

Social

- Lack of social support, isolation.
  - Unemployment/retired.
  - Single/unmarried/divorced/widowed.
- High risk occupation: high rates in farmers, pharmacists, and doctors (especially psychiatrists and anaesthetists).
  - These occupations have lethal mean available (farmers = guns, doctors and pharmacists = drugs).

Access to means

This may be through occupation or social activities.

▸ For more information, we recommend the factsheets produced by the British mental health charity 'Mind' which can be found at http://www.mind.org.uk/lnformation/Factsheets/Suicide/.

Past psychiatric history

Explore in detail previous contact with psychiatry and other services for mental health problems. Include as far as possible:

- Dates of illness, symptoms, diagnoses, treatments, hospitalizations, previous outpatient treatment, compulsory treatment under the Mental Health Act.

Past medical history

This should be evaluated in the same way as in the general medical history but remember to ask in particular about obstetric complications, epilepsy, head injuries, and thyroid disorders.

Drug and alcohol history

- Ask about all current drug intake including prescribed and over-the-counter medicines.
- Take a detailed history of substance abuse if relevant, recording type, quality, source, route of administration, and cost.
- Remember to ask about alcohol, tobacco and any allergic reactions. If necessary, use the CAGE questionnaire (📖 p.241).

Family history

Explore family relationships in detail (parents, siblings, spouse, children).

It is useful to draw a family tree and record age, health, occupation, personality, quality of relationship, family history of mental illness including alcoholism, suicide, deliberate self-harm, as well as any other serious family illnesses.

Also record the details and times of certain important family events, such as death, separation, or divorce and their impact on the patient.

Personal history

The personal history is a chronological account of the individual's life from birth up to the present. This section, which is often lengthy, should be tackled under the following subheadings:

Birth and early development

- Begin with recording the place and date of birth, gestation at delivery, and any obstetric complications or birth injuries.
- Enquire about developmental milestones.
- Ask about ‘neurotic traits’ in childhood (night terrors, sleep walking, bedwetting, temper-tantrums, stammer, feeding difficulties).
- Ask about relationships with peers, siblings, parents, and relatives—particularly in adolescence.
- Record any adverse experiences (physical or emotional abuse).
- Note any significant life events such as separations and bereavement.

School

- Explore how the individual got on at school socially, academically, and athletically.
- Record the start and end of their education and qualifications.
- Ask about the type of school, relationships with peers, teachers, interest in games, and whether there was a history of truancy.

Occupational history

- Enquire about all previous jobs held, dates, and reason for change, level of satisfaction with employment and ambitions.
- Include present job and economic circumstances.

Psychosexual history

This can be a rather difficult section of the history to elicit and is often dependent on how willing the patient is to volounteer such intimate details. However, try not to avoid it. It may have to be excluded if judged inappropriate or likely to cause distress.

- Record the onset of puberty (and menarche if female).
- Sexual orientation (hetero-, homo-, or bisexual).
- First sexual encounter.
- Current sexual practices (including practice of safe sex?)
- Any sexual difficulties or sexual abuse.

Marital history

This includes a detailed account of number of marriages, duration, quality of relationships and personality, age and occupations of spouses, and reason for break-up of relationship(s).

Children

Sex, age, mental, and physical health of all children.

Forensic history

This may or may not be volunteered by the patient. Begin by asking nonthreatening questions. ‘Have you ever been in trouble with the law?’

- Ask about criminal record and any previous episodes of violence or other acts of aggression.

Premorbid personality

This is the patient's personality before the onset of mental illness. An independent account is especially important for this part of the history.

- It may help to ask the patient how they would describe themselves and how they think others would describe them.
- Ask about social relationships and supports.
- Include interests, and recreational activities.
- Enquire specifically about temperament-what's their mood like on most occasions.
- Ask the patient to describe the nature of their emotional reactions, coping mechanisms and character (e.g. shy, suspicious, irritable, impulsive, lacking in confidence, obsessional).
- What are their moral and religious beliefs?

Social history

Ask especially about finances, legal problems, occupation, dependants, and housing. If elderly, ask about social support such as home care, attendance at a day centre and how they cope with activities of daily living (hygiene, mobility, domestic activity).

Mental state examination

The mental state examination is a vital part of the psychiatric assessment. It is *your* assessment of the patient's mental state based on your observations and interaction. It begins as soon as you see the patient. Mental state features prior to the interview, whether described by the patient or by other informants, are considered part of the history.

Appearance and behaviour

This involves a brief descriptive note of your observations, both at first contact and through the interview process. It should include a description of:

- Dress and grooming.
  - Patients with depression, dementia and drug abuse may show evidence of self-neglect.
  - Flamboyant clothes with clashing colours may be worn by a manic patient.
  - Loose-fitting clothes, may indicate an underlying anorexia or other eating disorder.*
- Facial appearance.
- Eye contact.
- Degree of co-operation.
- Posture.
- Mannerisms.
- Motor activity.
  - Excessive movement indicating agitation?
  - Very little movement (retardation) suggesting depression?
- Abnormal movements.
  - E.g. tics, chorea, tremor, stereotypy—repetitive movements such as rocking or rubbing hands.
- Gait.
- Any other physical characteristics worthy of note.

Speech

Describe in terms of:

- Rate.

- Quantity (↑ = ‘pressure of speech’ and is often associated with flight of ideas, ↓ = known as ‘poverty of speech’).
- Fluency.
- Articulation (including stammering, stuttering and dysarthria).
- Form: this is the way in which a person speaks rather than actual content. (See Box 15.7).

Box 15.7 Some examples of abnormal speech/thought form

The following are examples of abnormal speech-however, the speech is a manifestation of the underlying thought processes. One could argue, therefore, that the following are abnormalities of thought form.

- ***Flight of ideas:*** associated with mania. Ideas flow rapidly but remain connected although sometimes by unusual associations. The patient's train of thought tends to veer on wild tangents.
- ***Derailment:*** loosening of association seen in formal thought disorders (e.g. schizophrenia) in which ‘train’ of thought slips of the ‘track’. Things may be said in juxtaposition that lack a meaningful association or the patient may shift from one frame of reference to another.
- ***Perseveration:*** mainly seen in dementia and frontal lobe damage. The patient finds moving to the next topic difficult, resulting in an inappropriate repetition of a response.
- ***Incoherence:*** a pattern of speech that is essentially incomprehensible at times.
- ***Echolalia:*** a feature of dementia. A repetitive pattern of speech in which a patient echoes words or phrases said by the interviewer.
- ***Neologisms:*** found mainly in schizophrenia and structural brain disease (see p.286). The invention of new words with no meaning.
- ***Circumstantiality:*** a long-winded pattern of speech loaded down with unnecessary detail and digression before finally getting to the point. The patient is, however, able to maintain the train of thought.

Mood and affect

Mood is a pervasive and sustained emotion that can colour the patient's perception of the world over long periods. ‘Affect’ is the patient's immediate emotional state, including the external expression of feeling.

Examining mood and affect involves consideration of the patient's subjective emotional state and your objective evaluation.

Abnormalities of mood include depression, elation, euphoria, anxiety, and anger. It should be noted whether mood is consistent with thought and action or ‘incongruous’.

Abnormalities of affect include:

- ***Blunting:*** the coarsening of emotions and an insensitivity to social context. This is commonly used synonymously with affective ‘flattening’.
- ***‘Flattening of affect’:*** this is a reduction in range and depth of outward emotion.
- ***Lability:*** superficially fluctuating and poorly controlled emotions. May be found in delirium, dementias, frontal lobe damage and intoxication.

Thought content

Preoccupations

These include phenomena such as *obsessional thoughts*, or ruminations which are characterized by an intrusive preoccupation with a topic. The patient cannot stop thinking about it even though they may realize that it is irrational.

Phobias represent a fear or anxiety which is out of proportion to the situation, cannot be reasoned or explained away and leads to avoidance behaviour.

Other types of ruminations particularly important to establish here include suicidal or homicidal thoughts in addition to morbid ideation (e.g. ideas of guilt, unworthiness, burden, and blame).

Abnormal beliefs

Overvalued ideas

These are isolated beliefs which are not obsessional in nature and preoccupy an individual to the extent of dominating their life. That is, the patient is able to stop thinking about them but they choose not to.

The core belief of anorexia nervosa—the belief that one is fat—is an example of an overvalued idea. Other examples include unusual sect or cult beliefs, forms of morbid jealousy, and hypochondriasis.

Delusions

These are fixed false beliefs which are based on an incorrect inference about reality, not consistent with a patient's intelligence and cultural background. Importantly, these cannot be corrected by reasoning. They can sometimes be difficult to differentiate from overvalued ideas. The difference is that the patient firmly believes the delusion to be true.

Delusions may be 'primary' with no discernable connection with any previous experience or mood (characteristic of schizophrenia) or 'secondary' to an abnormal mood state or perception. In this way, the content of the delusions can give a clue to the nature of the mental illness.

Box 15.8 Some examples of delusions and associated terminology

Mood congruent delusion: a delusion with content that has an association to mood. For example, a depressed person may believe that the world is ending.

Mood incongruent delusion: a delusion with content that has no association to mood. Seen in schizophrenia.

Nihilistic delusion: a false feeling that self, others, or the world is nonexistent or coming to an end.

Paranoid delusion: this is any delusion that is self-referent. In psychiatry, 'paranoid' does not carry the lay meaning of 'fearful/suspicious'.

Delusions of reference: a false belief that others are talking about you or that events are somehow connected with you. For example, the patient may believe that people on TV or radio are actually talking directly to them. The feelings and delusional messages received are usually negative in some way but the fact that the patient alone is being spoken to has a grandiose quality.

Delusion of grandeur: a person has an exaggerated perception of his or her importance, power or identity. Usually, patients believe that they have made an important achievement that has not been suitably recognized.

Delusions of control: a false belief that a person's will, thoughts or feeling are being controlled by external forces.

These include disorders of the possession of thought. *Thought broadcasting* is the false belief that the patient's thoughts can be heard by others. *Thought insertion* is the belief that an outside force, person or persons are putting thoughts in the patient's mind whilst *thought withdrawal* is the belief that thoughts are being removed. Thoughts being heard spoken aloud is called *thought echo. Thought blocking* is the experience of having your train of thought halted.

Passivity feelings: these are examples of delusions of control. They may include *'made acts and impulses'* where the individual feels they are being made to do something by another, *'made movements'* where patients believe their limbs are controlled by someone else, *'made emotions'* where they are experiencing someone else's emotions.

Erotomania: a belief that another person is in love with the patient. Patients often believe that innocent glances from another person have a deeper, sexual meaning.

The Capgras delusion[1]***:*** a belief that those around you (often loved ones) have been removed and replaced with exact replicas. They exist in a world of impersonators. The delusion may extend to include animals and objects—the feeling that they are in duplicated world, like a film-set. The patient may even believe that he is his own double.

Religious delusions: these are any delusions with a religious or spiritual content. Careful here! Beliefs that would be considered normal for a person's religious or cultural background (e.g. a Christian believing that God has cured their illness) are **not** classed as delusions.

[1] Capgras JMJ, Reboul-Lachaux J (1923). L'illusion des 'sosies' dans un délire systématisé chronique. *Bulletin de la Société clinique de médecine mentale*, **11**: 6-16.

Perception

Alterations in normal perception consist of changes to our normal, familiar awareness or ordinary experiences. These include sensory distortions (heightened or dulled perception), sensory deceptions (illusions and hallucinations) and disorder of self awareness (depersonalization, derealisation).

Disorders of self awareness

- *Depersonalization* is the feeling that the body is strange and unreal.
- *Derealization* is the perception of objects in the external world as being strange end unreal.

Both the above phenomena commonly occur in stressful situations, with drug intoxication, anxiety, depressive disorders, and schizophrenia. Many psychologically normal people can experience an element of derealization or depersonalization if sleep-deprived.

Illusions

An illusion is a misperception or misinterpretation of real sensory stimuli. It may affect any sensory modality. Enquire as to when they occur and what significance they have.

Illusions frequently arise from a sensory impairment such as partial sightedness or deafness and represent an understandable attempt at 'filling in the gap'. Most people have experienced some form of visual illusions—for example, mistaking a distant object for a person, particularly in poor lighting (e.g. at night).

Hallucinations

An hallucination is a false perception which is not based on a real external stimulus. It is experienced as true and coming from the outside world.

They may occur in any sensory modality, although visual and auditory hallucinations are the commonest (Box 15.9).

Importantly, not all hallucinations point to psychiatric disease. For example, some hallucinations occur in normal people, when falling asleep (hypnagogic) or on waking (hypnopompic) and, although the nature of dreams is heavily debated, it could be said that they are hallucinations. Note also the Charles Bonnet syndrome (Box 15.10).

Sensory distortions

This includes heightened perception with especially vivid sensations (e.g. hyperaccusis), dulled perception, and 'changed perception'. For example, patients may experience objects as having a changed shape, size, or colour.

Box 15.9 Some examples of hallucinations

Auditory hallucinations: false perception of sounds, usually voices but also other noises such as music. The hallucination of voices may be classed as *2nd person* where the voice is speaking to the patient ('you should do this') or *3rd person* where the voice or voices are talking about the patient ('he should do this').

Visual hallucinations: false perceptions involving both formed (e.g. faces, people) and unformed (e.g. lights, shadows) images.

Scenic or panoramic hallucinations: a form of visual hallucination involving whole scenes such as battles.

Olfactory hallucinations: the false perception of odours.

Gustatory hallucinations: the false perception of taste.

Tactile hallucinations: the false perception of touch or surface sensation (e.g. phantom limb; crawling sensation in or under skin in delirium tremens—formication).

Somatic hallucination: the false sensation of things occurring in or to the body, most often visceral in origin. Somatic hallucinations include *haptic* (touch, tickling, pricking), *thermic* (heat/cold), and *kinathetic* (movement and joint position).

Pseudohallucinations: these are recognized as not being 'real' by the patient, acquiring an 'as if' quality and have some degree of voluntary control.

Box 15.10 Charles Bonnet syndrome

This is a good example of hallucinations in a psychiatrically normal patient. In this syndrome, patients with some kind of visual impairment (usually older people) see visual hallucinations within the area of impaired vision. The hallucinations are often cartoon-like characters or faces. For example, the authors once came across a patient with a visual scotoma due to retinal injury. The voice of our Irish consultant would trigger the hallucination of a leprechaun dancing and cavorting within their blind spot.

The syndrome is also an example of pseudohallucination as, often, the patient realises that the visions are not real.

It was described by the Swiss philosopher Charles Bonnet in 1760, whose 87-year-old grandfather admitted seeing visions of buildings and people after developing severe cataracts in both eyes.

Charles Bonnet syndrome is likely much more common than most medical people realise. The elderly sufferers are often afraid to admit to it for fear of being diagnosed with a psychiatric disorder or being labelled 'mad'.

Cognitive function

Cognition can be described as the mental processes of appraisal, judgement, memory, and reasoning. Evaluation of cognitive functioning is important for detecting impairment, following the course of an illness and monitoring improvement or response to treatment.

The Mini Mental State Examination (MMSE)

The MMSE provides a brief quantitative measure of cognitive functioning and can be used in both a psychiatric or general medical setting. It tests attention, orientation, immediate and short-term recall, language, and the ability to follow verbal and written commands (see Box 15.11).

Notes on conducting the MMSE

It is important to remember that there are no half marks in this test-be strict and rigorous. The maximum total score is 30.

- *Orientation:* rather than asking for each part of the date in turn, ask the patient for today's date and then ask specifically for those parts omitted. Do the same for place ('where are we now?').
- *Registration:* say the name of the objects clearly and slowly, allowing about 1 second to say each. The first repetition determines the patient's score… but keep repeating the names of the object until the patient has got all three to enable testing of recall later.

- *Attention and calculation:* if the patient can't perform this mathematical task, ask them to spell the word 'WORLD' backwards. The score is the number of letters in the correct order (e.g. dlrow = 5, dlorw = 3).
- *Repetition:* allow one trial. Score 1 only if the repetition is completely correct. Make sure you say it slowly and clearly so that the patient can hear!
- *Three-stage command:* say all three stages of the command before giving the piece of paper to the patient. Do not prompt the patient as you go. Score 1 point for each part conducted correctly.
- *Reading:* say 'read this sentence and do what it says'. Score 1 point if the patient closes their eyes. No points if they simply read the sentence out loud.
- *Writing:* be sure not to dictate a sentence or give any examples. The sentence must make sense and contain a subject and a verb. Correct grammar, punctuation, and spelling are not necessary.
- *Copying:* all 10 angles must be present and 2 must intersect. Ignore mistakes from tremor and ignore rotation of the diagram.

Interpreting the final score

The MMSE score will vary within the normal population by age and the number of years in education (decreasing with advancing age and increasing with advancing schooling). The median score is 29 for people with 9 years of education, 26 for 5-8 years of education, and 22 for 0-4 years.[1]

As a rule of thumb, scores of <23 are taken to indicate mild, <17 moderate and <10 severe cognitive impairment. This is a non-linear scale, however. For more information, see the reference below.

[1] Crum RM, Anthony JC, Bassett SS, and Folstein MF (1993). Population-based norms for the minimental state examination by age and educational level, *JAMA*, **18**: 2386-91.

Box 15.11 The Mini Mental State Examination (MMSE)

Orientation (10)

(5) What is the (year), (season), (day), (date), (month)?

(5) Where are we: (country), (county), (town), (hospital) (floor/ward)?

Registration (3)

Name three unrelated objects. Allow one second to say each. Then ask the patient to repeat all three after you have said them. Give one point for each correct answer. (e.g. ball, car, man).

Attention and calculation (5)

Ask the patient to take 7 from 100, and again…total of 5 times. Give one point for each correct answer. Stop after five answers (93, 86, 79, 72, 65).

Alternatively, spell WORLD *backwards* giving one mark for each letter in the correct order—see notes opposite.

Recall (3)

Ask patient to recall the three objects previously stated. Give one point for each correct answer.

Naming (2)

Show patient a watch and ask them what it is. Repeat for a pen/pencil.

Repetition (1)

Ask the patient to repeat the following: 'No ifs, ands, or buts.'

Three-stage command (3)

Ask the patient to follow these instructions: 'take this paper in your left hand, fold it in half and put it on the floor'. Give the patient a piece of paper and score 1 for each stage completed correctly.

Reading (1)

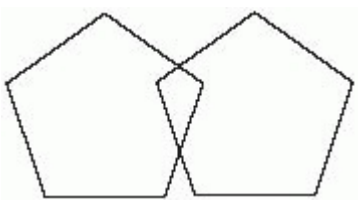
Write 'CLOSE YOUR EYES' on a piece of paper, ask the patient to read and obey what it says.

Writing (1)

Ask the patient to write a sentence.

Copying (1)

Ask patient to copy the following design.



Maximum total score = 30

Folstein MF, Folstein SE, and McHugh PR (1975). Mini-mental State: a practical method for grading the state of patients for the clinician. *Journal of Psychiatric Research,* **12:** 189-98.

Insight

This is how well the patient is able to understand or explain their condition. When assessing insight, ask:

- Does he/she recognize and accept that they are suffering from a mental or physical illness?
- Are they willing to accept treatment and agree to a management plan?

Note also whether an individual's attitudes are constructive or unconstructive, realistic or unrealistic.

If not accepting of a psychiatric diagnosis, to what does the patient attribute their difficulties or abnormal experiences?

Summary

At the end of the assessment, a summary should be made of history and mental state examination which should encompass a statement of diagnosis or differential diagnosis, aetiolgical factors, and a plan for further investigations and management.

Physical examination

A full physical examination, particularly that of the neurological system should be seen as an integral part of the assessment of a psychiatric patient.

You may want to tailor an examination to look for, or exclude, physical conditions that give rise to the psychiatric symptoms and signs that you have discovered. See the end of this chapter for details.

Important presenting patterns

Schizophrenia

The term schizophrenia* is often described as a single disease but the diagnostic category includes a group of disorders, probably with heterogeneous causes, but with somewhat similar behavioral symptoms and signs. It is a psychosis, characterized by 'splitting' of normal links between perception, mood, thinking, behaviour, and contact with reality.

The prevalence of schizophrenia is ~0.5% worldwide with equal incidence in both sexes. The onset is usually in adolescence or early adulthood. Symptoms tend to remit, although a return to baseline is unusual.

Clinical features

Schizophrenia is characterized by delusions and hallucinations with no insight. These symptoms are often followed by a decline in social functioning. Historically, several different diagnostic classifications have been developed, for the latest diagnostic criteria, see the ICD-10 or DSM-IV.

Bleuler's four As.

In 1910, Bleuler coined the term 'schizophrenia'. He went on to characterize the key features, summarized as the 'four As'.

- Associative loosening (disconnected, incoherent thought process).
- Ambivalence (the ability to experience two opposing emotions at the same time—e.g. loving and hating a person).
- Affective incongruity (affect disassociated with thought).
- Autism (self-absorption and the withdrawal into a fantasy world).

Crow's positive and negative symptoms

In 1980, Crow[1] suggested that the symptoms of schizophrenia could be divided into 2 distinct groups—those that are 'positive' and those that are 'negative'. This remains a useful way to think of the symptoms.

Crow went on to suggest that schizophrenia could be split into two syndromes, comprising mostly positive or negative symptoms respectively.

Positive symptoms

- Delusions (including ideas of reference).
- Hallucinations.
- Thought disorder.

Negative symptoms

- Blunted affect.
- Anhedonia (lack of enjoyment).
- Avolition (lack of motivation).
- Alogia (poverty of speech).
- Social withdrawal.
- Self-neglect.

[1] Crow TJ (1980). Molecular pathology of schizophrenia: More than one disease process? *British Medical Journal*, **280:** 66-8.

Schneider's first-rank symptoms

Kurt Schneider listed his 'first-rank' symptoms of schizophrenia in 1959[2]. One of these, Schneider said, is diagnostic of schizophrenia in the absence of organic brain disease or drug intoxication.

- Third person auditory hallucinations (running commentary, arguments, or discussions about the patient).
- Thought echo or 'Echo de la pensée'.
- Disorders of thought control (withdrawal, insertion, broadcast).
- Passivity phenomena.
- Delusional perception.
- Somatic passivity.

Schneider's criteria have been criticized for being 'too narrow', providing a snapshot of a patient at only one time and for not taking into account the long-term negative symptoms.

Box 15.12 Subtypes of schizophrenia

- ***Simple:*** negative symptoms tend to predominate.
- ***Paranoid:*** delusions and hallucinations are prominent and tend to include religious, grandiose, and persecutory ideas.
- ***Hebephrenic:*** affective incongruity predominates with shallow range of mood. Delusions and hallucinations tend to lack an organized theme.
- ***Catatonic:*** anhedonia, avolition, alogia, and poverty of movement are the key features. This may lead to a 'waxy flexibility' where the patient's limbs can be moved into, and stay in, certain positions.

For information on aetiology, treatment and prognosis, see appropriate other handbooks in this series.

[2] Schneider K (1959). *Clinical Psychopathology*. New York: Grune and Stratton.

[*] 'Schizophrenia' comes through Latin from the Greek *skhizein* 'to split' and *phrēn* 'mind'. The term 'phrenic', as readers will know, refers also to the diaphragm. This is because in ancient Greece, the mind was thought to lie in the diaphragm.

Delirium

Delirium or acute confusional state is a transient global disorder of cognition which is characterized by an acute onset and a fluctuating

course. It represents one of the most important and misdiagnosed problems in medicine and surgery. Delirium may occur in as many as 10-20% of hospital inpatients, with elderly patients being the most vulnerable. Approximately 60% of patients suffer delirium following hip fracture.

Below is a brief summary of the main features and causes. You should bear all the possible causes in mind and tailor your physical examination and investigations accordingly (see elsewhere in this book).

Predisposing factors (risk factors)

- Increasing age.
- Pre-existing cognitive defect.
- Psychiatric illness.
- Severe physical comorbidity.
- Previous episode of delirium.
- Deficits in hearing or vision.
- Anticholinergic drug use.
- New environment or stress.

Causes (precipitants)

Delirium is usually 'multifactorial' with a single cause difficult or impossible to identify. Some factors include:

Intracranial factors

- Trauma.
- Vascular disease (e.g. stroke).
- Epilepsy and post-ictal states.
- Tumour.
- Infection (meningitis, encephalitis, tuberculosis, neurosyphilis).

Extracranial factors

- Drugs—both prescribed and recreational, intoxication, and withdrawal.
- Electrolyte imbalances.
- Infection (e.g. urinary tract, chest, septicaemia).
- Endocrine (e.g. thyroid dysfunction, hypo- and hyperglycaemia).
- Organ failure (heart, lung, liver, kidney).
- Hypoxia.
- Acid/base disturbance.
- Nutritional deficiencies.
- Post-operative or post-anaesthetic states.
- Miscellaneous.
    - Sensory deprivation.
    - Sleep deprivation.
    - Faecal impaction.
    - Change of environment.

Symptoms include

- Fluctuating level of consciousness.

- Difficulty maintaining, or frequently shifting, attention.
- Disorientation (often worse at night).
- Illusions.
- Hallucinations (often simple, visual).
- Apathy.
- Emotional lability.
- Depression.
- Disturbance of the normal sleep/wake cycle.

Box 15.13 Confusion assessment method (CAM)

This is a method commonly used to assess delirium in the clinical setting.[*] The patient must display both features 1 and 2 *plus* either 3 or 4.

- **1** ***Acute onset and fluctuating course:*** often best obtained from a relative or member of ward staff. Onset is hours to days, lucid periods often in the morning.
- **2** ***Inattention:*** easily distracted, attention wanders in conversation.
- **3** ***Disorganized thinking:*** rambling or irrelevant conversation, illogical flow of ideas, unable to maintain a coherent stream of thought.
- **4** ***Altered level of consciousness:*** drowsy or over active, may fluctuate. May experience nightmares and hallucinations.

[*] Inouye S, van Dyck C, Alessi C, *et al*. (1990). Clarifying confusion: the confusion assessment method. *Annals of Internal Medicine,* **113**(12): 941-8.

Differential diagnosis

Dementia (often co-exists with delirium and ↑ risk of delirium 2-3 fold), depression, psychosis, AIDS-related complex.

Anxiety disorders[*]

Generalized anxiety disorder (GAD)

The main feature is excessive anxiety and worry about events or activities which the patient finds difficult to control—such as work or school performance. The symptoms must be present for more than 6 months and include 3 or more of:

- Restlessness or feeling ‘on edge’.
- Easily fatigued.
- Difficulty concentrating or ‘mind goes blank’.
- Irritability.
- Muscle tension.
- Sleep disturbance (insomnia and fatigue on waking).

Panic disorder

Spontaneous occurrence of severe panic attacks (periods of fear which peak within ~10 minutes).

These should be accompanied by 4 or more of tachycardia, sweating, trembling or shaking, shortness of breath, a feeling of choking, chest pain, dizziness, lightheadedness or presyncope, paraesthesia, depersonalization or derealization, nausea, abdominal pain, fear of dying, fear of losing control, and hot flushes.

Phobic disorders

A phobia is an irrational fear that produces an avoidance of the subject of the fear (an object, person, activity, or situation). A phobia is perceived by the patient as excessive (i.e. they have insight).

Agoraphobia

Agoraphobia[**] is not fear of wide open spaces *per se*, as is commonly thought, but is anxiety caused by being in places or situations from which escape may be difficult or in which help might not be available in the event of a panic attack. These situations may include being outside, home alone, being in a crowded place, or travelling on a bus or a train.

[**] Agoraphobia comes from the Greek '*agora*' meaning 'the marketplace'.

Social phobia

This is a fear of social situations in which the person is exposed to unfamiliar people or to possible scrutiny by others. The fear is of the resulting humiliation caused by a poor performance.

Avoidance behaviour, anticipation, or distress at the time of the social encounter leads to impairment in functioning at work or in school and can have a significant impact on the patient's life.

Other specific phobias

These are marked and persistent fears cued by the presence or anticipation of specific objects or situations. The list is manifold. Our favourites, from which no medic can suffer, include bromidrosiphobia (the fear of body odour), spermophobia (the fear of germs), belonephobia (the fear of needles), phronemophobia (the fear of thinking), iatrophobia (the fear of doctors), and, of course, pinaciphobia (the fear of lists).

Obsessive-compulsive disorder

- This is characterized by time-consuming obsessions ± compulsions which cause social impairment or mental distress.
- Obsessions are intrusive thoughts, feelings, ideas, or sensations. They are recognized by the patient as their own (compare with 'thought insertion')—the patient usually tries to ignore or suppress them.
- Compulsions are conscious, purposeful behaviours which attempt to neutralize or prevent a discomfort or dreaded event. Examples include repeated hand-washing, checking, and counting.
- The key here is that the obsessions and compulsions are recognized as coming from within the patient, they feel powerless to stop and are distressed by their presence.
- Severe obsessions and compulsions can occur in depression, schizophrenia, generalized anxiety disorder, panic disorder, and others.

Dementias

Dementia is usually a disease of older people and refers to a global deterioration of higher mental functioning without impairment in consciousness that is progressive and usually irreversible.

Dementia usually presents with a history of chronic, steady decline in short and long-term memory and is associated with difficulties in social relationships, work and activities of daily living. Important manifestations include disruption of language and intelligence as well as changes in personality and behaviour. Apathy, depression, and anxiety are frequently found and psychotic phenomena may be seen in a third of patients. A diagnosis of dementia is based on MMSE and information from other sources such as the patient's family, friends and employers.

Dementia may be 'primary' or 'secondary' to diseases such as:

- Chronic CNS infection: HIV, syphilis, meningitis, encephalitis.
- CNS trauma: anoxia, diffuse axonal injury, dementia pugilistica (repeated head injury—seen in boxers), chronic subdural haematoma.
- ↑ intracranial pressure: neoplasia, hydrocephalus.
- Toxins: heavy metals, organic chemicals, chronic substance abuse.
- Vitamin deficiencies: B_{12}, folate.
- Autoimmune disease: SLE, temporal arteritis, sarcoidosis.

Other possible causes include endocrinopathies, Wilson's disease, and lipid storage diseases.

Alzheimer's disease

The key pathological changes in Alzheimer's disease (AD) are reduced brain mass and ↑ size of the ventricles. There is neuronal loss and occurrence of amyloid plaques and neurofibrillary tangles. AD makes up ~50% of all cases of dementia and ~90% of all primary dementias. The main features are memory impairment and at least one of:

- Aphasia (📖 p.286).

- Apraxia (p.357).
- Agnosia (p.357).
- Abnormal executive functioning (planning, organizing, abstracting, sequencing).

Vascular dementia/multi-infarct dementia

This makes up about 20-30% of all cases of dementia. Onset may be abrupt and/or with a step-wise decline. Vascular dementia is associated with more patchy cognitive impairment then AD, often with focal neurological signs and symptoms such as hyperreflexia, extensor plantar responses, pseudobulbar, bulbar, or other cranial nerve palsies, gait abnormalities, and focal weakness.

The primary pathology is multiple small areas of infarction (cortex and underlying white matter). It is important to note vascular risk factors such as previous stroke, hypertension, heart disease, diabetes, and smoking.

Lewy body dementia

Lewy body dementia accounts for up to 20% of all cases. Patients with this show features similar to AD but also often have recurrent visual hallucinations, fluctuating cognitive impairment, parkinsonian features, and extrapyramidal signs.

Fronto-temporal dementia

This accounts for 75% of all dementia. Pick's disease is a form of frontotemporal dementia characterized by the presence of neuronal 'Pick's bodies' (masses of cytoskeletal elements). The predominance of frontal lobe involvement in evinced by profound personality changes, social impairment, and stereotyped behaviour. However, visuospacial skills are usually preserved. The patient may also show 'primitive reflexes' (p.334).

Huntington's disease

Huntington's is an autosomal dominant disease presenting as early as the third decade and is associated with a subcortical type of dementia. Apart from the movement disorder showing involuntary choreiform movements of the face, shoulders, upper limbs, and gait, the symptoms of the dementia include psychomotor slowing and personality alteration with apathy or depression.

Parkinson's disease

Patients with Parkinson's disease have cognitive slowing along with the signs described earlier (p.355). Dementia is seen in the later stages of the disease.

Creutzfeldt-Jakob disease (CJD)

Contrary to common perception, this is not a new disease or one that affects young people. The most frequently seen of this family of diseases is 'sporadic CJD' which has no known cause. Onset is usually between the forth and sixth decades of life and is associated with a very rapid progression of dementia, in addition to signs such as myoclonus, seizures, and ataxia—the time to death is typically a few months.

Variant CJD (vCJD) is a disease mainly confined to the UK, first reported in 1996, and is thought to have resulted from transmission of infection from cattle suffering from bovine spongeiform encephalopathy (BSE). The average age of onset is 27 years, presenting initially with behavioural symptoms. The duration is of a year or more. At the time of writing, there have been only 161 definite or probable vCJD cases.[1]

Affective disorders

Bipolar disorder

Bipolar disorder, previously known as manic-depression, is usually characterized by periods of deep, prolonged depression with periods of excessively elevated and irritable mood known as mania.

It is important to note that patients presenting only with mania, and no evident depression, are said to have bipolar disorder. There are 3 main patterns of disease:

- *Bipolar I disorder:* one or more episodes of major depression with episodes of mania.
- *Bipolar II disorder:* milder bipolar disorder consisting of recurrent periods of depression and hypomania but no manic episodes.
- *Cyclothymic disorder:* characterized by frequently occurring hypomanic and depressive symptoms that do not meet the diagnostic criteria of manic episodes or major depression.

Mania

Manic episodes are characterized by profound mood disturbance, consisting of an elevated, expansive, or irritable mood that causes impairment at work or danger to others. These patients may suffer delusions and hallucinations, the former usually involving power, prestige, position, selfworth, and glory. The key feature is 'disinhibitation'.

There may be several of the following:

- Inflated self-esteem or grandiosity.
- Reduced need for sleep.
- Racing thought, flight of ideas, and distractibility.
- Excessive talking or pressured speech.
- ↑ level of goal-focused activity at home, at work or sexually.
- Psychomotor agitation.
- Excessive involvement in pleasurable activities, often with unfortunate consequences (especially sexual indiscretions, unrestrained spending sprees).

Depression

Depressive disorders can be classified as bipolar or unipolar and as mild, moderate, or severe. They may include somatic symptoms and psychotic symptoms (delusions and hallucinations which are usually moodcongruent) in the case of severe depression.

The diagnostic criteria, treatment and prognosis can be found elsewhere in the Oxford Handbook series. Depression can cause significant social impairment and distress.

In general terms, features of major depression include:

- Depressed mood with feelings of worthlessness.
- Diminished interest or pleasure (anhedonia).
- Significant weight loss or gain.
- Insomnia or hypersomnia.
- Psychomotor agitation or retardation.
- Fatigue or loss of energy.
- Diminished ability to think or concentrate; indecisiveness.
- Recurrent thoughts of death, suicide, suicide attempts, or specific plans for suicide.

Hypomania

Hypomanic episodes are characterized by a persistently elevated, expansive, or irritable mood with similar features to mania. However, the episode is not severe enough to cause marked impairment in social or occupational functioning and delusions and hallucinations do not occur.

Box 15.14 Typical mental state examination in affective disorders

Mania

- *Appearance:* bright, colourful or garish clothing, hyperactivity, hypervigilance, restlessness.
- *Speech:* fast, pressured, flight of ideas.
- *Mood and affect:* joy, elation, jubilance, euphoria, annoyance, irritability.
- *Thought content:* expansive and optimistic thinking, excessively self-confident or grandiose, distractible, rapid production of ideas and thoughts.
- *Perception:* mood congruent and incongruent delusions. Fleeting auditory or, more rarely, visual hallucinations. Delusions of wealth, power, influence, or religious significance.
- *Cognitive functioning:* usually unaffected.
- *Insight* seriously impaired judgement and no insight.

Depression

- *Appearance:* reduced eye contact, poor grooming and hygiene, change in weight, psychomotor agitation, or retardation.
- *Speech:* slow, monotonous, or lacking in spontaneity.

- *Mood and affect:* sadness, numbness, irritability, anhedonia, reduced concentration, loss of energy, and motivation.
- *Thought content:* preoccupied with negative ideas and nihilistic concerns, overwhelmed or inadequate, helpless, worthless, hopeless, suicidal.
- *Perception:* delusions and hallucinations (usually mood congruent), especially 2nd person auditory hallucinations. For example, auditory hallucinations of voices calling the patient worthless.
- *Cognitive functioning:* poor memory and concentration but level of consciousness is normal. Depressed patients may score falsely low on an MMSE if they are not willing to answer the questions ('I don't know')—'pseudodementia'.
- *Insight* diminished judgement and insight.

Medical conditions with psychiatric symptoms and signs

There are many medical conditions that can give psychiatric clinical features. This can sometimes lead to failure of the underlying medical condition to be recognized and treated appropriately. It is important in psychiatry to consider possible 'organic' causes for the symptoms and signs before starting psychiatric treatment. Further, many medical disorders are associated with psychiatric diagnoses.

The following is a sample of such situations, aimed at illustrating the above points, rather than providing an exhaustive list.

Neurological disorders

- ***Seizure disorder:***
  - Ictal events, including status epilepticus, may mimic psychosis.
  - Automatisisms are seen in some temporal lobe seizures.
  - The pre-ictal prodrome can involve changes in mood, particularly irritability, and auras (including auditory and olfactory hallucinations) can be seen in temporal lobe epilepsy. These may also include epigastric sensations, *déja vu* or *jamais vu*.
  - The post-ictal state often involves confusion and disorientation.
- ***Parkinson's disease:*** patients may suffer from major depression, anxiety syndromes, hallucinations, and delusions.
- ***Brain tumours and cerebrovascular events:*** (depend on location).
  - Frontal: personality change, cognitive impairment, motor, and language disturbance.
  - Dominant temporal lobe: memory and speech, Korsakoff psychosis in bilateral lesions.
  - Occipital lesions: visual agnosis, visual hallucinations.
  - Limbic and hypothalamic: affective symptoms, rage, mania.
- ***MS:*** cognitive deficits, dementia, bipolar disorder, major depression.

Infectious diseases

- ***Neurosyphilis:*** primarily affects the frontal lobe (irritability, poor self care, mania, progressive dementia).
- ***Meningitis:*** especially with indwelling shunts, can cause acute confusion, memory impairment.
- ***Herpes simplex encephalitis:*** bizarre and inconsistent behaviour, seizures, anosmia, hallucinations (olfactory and gustatory), psychosis.
- ***HIV encephalitis:*** progressive subcortical dementia, major depression, suicidal behaviour, anxiety disorders, abnormal psychological reactions.

Endocrine disorders

- ***Hyperparathyroidism:*** delirium, sudden stupor and coma. Visual hallucinations with associated hypomagnesaemia.
- ***Hypoparathyroidism:*** psychosis, depression, anxiety.
- ***Hyperthyroidism:*** depression, anxiety, hypomania, psychosis.
- ***Hypothyroidism:*** depression, apathy, psychomotor retardation, poor memory, delirium and psychosis 'myxoedema madness'.

Rheumatological disorders

Systemic lupus erythematosus: delirium, psychosis, severe depression.

Metabolic disorders

- ***Hyponatraemia:*** confusion, depression, delusions, hallucinations, seizures, stupor, coma.
- ***Hypernatraemia:*** acute changes of mental state.
- ***Encephalopathy***
- ***Uraemia encephalopathy:*** memory impairment, depression, apathy, social withdrawal.

Vitamin deficiencies

- ***B_1 (thiamine):*** asthenia, fatigue, weakness, depression.
- ***B_{12} (cyanocobalamin):*** impaired cognitive function.

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Chapter 16

The Paediatric Assessment

History taking

Children and doctors

The speciality of paediatrics is very different to adult medicine. Children grow, change, and mature. Your style and approach to history taking and examination will very much depend on the child's age, independence, and understanding, so flexibility is essential. The most important thing to remember during your time as a student is that paediatrics should above all be pleasurable.

An approach to the child patient

The child needs to be put at ease and made to feel welcome.

- Make a complimentary remark about their clothes, or show them an interesting toy.
- Tell the child your name and ask theirs.
- Make friends with them by asking what their favourite lesson is at school or what they had for breakfast.
- Shake hands with children, even toddlers enjoy this formality.

The history

A structured approach to history taking is important to avoid forgetting things, but this must not become too rigid, as it is sometimes necessary to pursue a different line of questioning to gain essential information. The table opposite is a list of useful headings in paediatric history taking, and this should be memorized.

Talking to the child

Children should be asked to give their account of events with parental corroboration. Children under 5 years old will lack the vocabulary and communication skills to describe their symptoms, but will be able to point to parts that hurt.

Talking to the parents

Most of the history is likely to be gained from the parents or guardians.

- Ask if they have the infant medical record book—this contains information about height and weight centiles, immunizations, development, and illnesses in the first few years of life.
- Ask whether the parents have any views on what the cause of the child's trouble is. Listen carefully to the parents; they are acute observers of their children.
- Ensure that all terms used are appropriately defined—you should be gleaning information from the parents' observations and *not* their interpretation of the symptoms. For example, the word ‘wheeze’ is often used incorrectly and sometimes a demonstration can be helpful. Further, the parent may interpret a baby's cries as pain when, in fact, it is your task to establish the circumstances of the cries and, therefore, the cause.
- As children get older, the parents may have a hazy memory for early events. Establishing symptoms in relation to easily remembered events (e.g. first walked) may clarify the timeline.

Box 16.1 Outline of paediatric history

- Presenting complaint and history of presenting complaint.
- Birth history:
  - Place of birth.
  - Gestation and pregnancy.
  - Birth weight.

    - Delivery.
    - Perinatal events and SCBU admission.
- Feeding methods and weaning.
    - If bottle fed, note how the bottle feed is mixed (how many scoops/number of ounces).
- PMH including hospital admissions, infections, injuries.
- Developmental history.
- School progress.
- Immunizations.
- Drugs.
- Allergies.
- Family tree with sibling's ages, including deaths, miscarriages, and stillbirths.
- Parental age and occupation.
- Family illnesses and allergies.
- Housing.
    - This should include a discussion about the child's bedroom as they may spend 12 hours of each day there.
- Travel.
- Systems review.

> ‘You can do anything with children if you only play with them.’
>
> --Otto von Bismarck, 19th century.

The examination: an approach

Examination in children varies depending on the age and co-operation of the child. School-age children and babies may be examined on a couch with a parent nearby, whereas toddlers are best examined on the parent's lap. If the child is asleep on the parent's lap, much of the examination should be completed before waking them up.

Undressing

Let the parent undress the child: and only expose the part of the body you will be examining.

Positioning

Some children may prefer to be examined standing up. Only lay the child down when you have to, as this can be very threatening.

Putting the child at ease

Slowly introduce yourself to the child's space during the examination by exchanging toys, for example.

Explain what you are going to do and be repeatedly reassuring, children can be embarrassed by silence after a doctor's question, but will be comforted by endless nattering. And remember-don't ask permission, as this will often be refused!

The examination

Firstly, use a hands-off approach. Allow the child to look at you, and let them play in your presence. Watch the child. How do they interact with their parents? Do they look well or ill? Do they look clean, well nourished, and well cared for?

Kneel on the floor so that you are at the child's level. Use a style and language appropriate to the age of the child—a toddler will understand the word ‘tummy’ better than the word ‘abdomen’.

Be opportunistic

Do not adhere to a rigid examination schedule, e.g. you may have to listen to the heart first while the child is quiet, then look at the hands later. Never examine the presenting part only. Be thorough and train yourself to be a generalist.

Leave unpleasant procedures, such as examination of the tonsils, until last.

Presenting your findings

When presenting your findings, translate what you see into appropriate terminology. Informing a senior that a child ‘looks funny’ is not very helpful but the saying the child is dysmorphic, followed by a detailed description is acceptable. Describe in simple terms the relevant

features that make the child look unusual, e.g. low set ears, wide set eyes.

▸ There is no substitute for examining lots of normal children.

> ‘Paediatrics is a specialty bound by age and not by system.’
> --Apley

Box 16.2 Some distraction techniques to help with examination

- Playing peek-a-boo.
- Letting toddlers play with your stethoscope.
- Giving infants something to hold.
- Asking mum or dad to wave a bright toy in front of them.

Box 16.3 The mother's knee

Be cautious about taking any baby or young child to a couch. It is often better to leave them on their mother's knee for the majority of the examination.

▸ A baby should never be picked off their mother's knee if they are beyond 7-8 months of age—this will invariably result in screaming.

Box 16.4 Using this chapter

The examination routines in this chapter describe the techniques to employ and the signs to look for in each body system. For more detailed information about how to perform certain aspects, the reader should refer to the relevant organ system chapter elsewhere in the book (e.g. how to perform accurate percussion, where to listen to heart sounds and so on).

The respiratory system

Key points from the history

- Is the child short of breath or wheezy (remember to define terms)?
- Is there stridor or croup?
- Is there a cough? Does it disturb sleep?
- Does anything trigger the symptoms—sport, cold weather, pets?
- Has the child expectorated or vomited any sputum?
- Is the infant short of breath during breast or bottle feeding?
- Is there a possibility the child could have inhaled a foreign body?
- Is there any FHx of respiratory problems such as asthma or cystic fibrosis?
- Does the child have a fever—suggestive of infection?
- Has anyone else been unwell? Any contacts with tuberculosis?
- Has the child travelled abroad recently?
- How does the respiratory problem limit the child's life—how much school is missed, can they play sport, how far can they run, is sleep disturbed?

Examination

Inspection

Look around for any clues—is the patient on oxygen? Are there inhalers or nebulizers at the bedside?

General inspection

- Are they comfortable or in respiratory distress? Look for:
  - Nasal flaring.
  - Use of accessory muscles of respiration.
  - Intercostal recessions (sucking in of the muscles between the ribs) and subcostal recessions (drawing in of the abdomen).
  - Grunting (a noise at the end of expiration which is the infant's attempt to maintain a positive end expiratory pressure).
- Is the child running around or just sitting on the parent's knee?
- Are they restless or drowsy?
- Count the respiratory rate.
- Listen for wheeze or stridor (a harsh inspiratory sound caused by upper airways obstruction).
- What type of cough does the child have?
- Has the child coughed up any sputum (children under 5 years will swallow sputum, which is often vomited after a bout of coughing).

Hands

- Clubbing (cystic fibrosis, bronchiectasis).
- Measure the radial pulse—pulsus paradoxicus (📖 p.172) is an important feature of acute severe asthma in children.

Face

- Check the conjunctiva for anaemia.
- Look for central cyanosis in the tongue.
- Look for petechiae (non-blanching spots from small burst blood vessels) around the eyes from a prolonged bout of coughing.

Chest

- Look for chest movement. Is it symmetrical? Is the child splinting (failing to move) one side of the chest?
  - Children who splint their chest as a consequence of pneumonia often also have a slight spinal scoliosis.
- Look at the chest shape. Is there any chest wall deformity?
  - Harrison's sulcus: permanent groove in the chest wall at the insertion of the diaphragm in longstanding asthma.
  - Barrel chest: air trapping in asthma.
  - Pectus carinatum: ‘pigeon chest’ seen in longstanding asthma.
  - Pectus excavatum: normal variant.

Table 16.1 Normal respiratory and heart rates by age

Age	Heart rate	Respiratory rate
<1 year	120-160	30-60

1-3 years	90-140	24-40
3-5 years	75-110	18-30
5-12 years	75-100	18-30
12-16 years	60-90	12-16

Box 16.5 Pain

Children may complain of ‘pain’ when they wish to indicate distress or discomfort that their vocabulary will not allow. Remember also that diseases may present differently in children than in adults.

For example, children may often describe ‘chest pain’ with chest tightness and asthma.

Pneumonia often gives abdominal pain in children.

Box 16.6 Some childhood coughs

The following factors may give important clues as to the origin of the cough.

- ***Productive:*** cystic fibrosis, bronchiectasis, pneumonia.
- ***Nocturnal:*** asthma, cystic fibrosis.
- ***Worse on wakening:*** cystic fibrosis.
- ***Brassy:*** tracheitis.
- ***Barking:*** croup (laryngotracheobronchitis).
- ***Paroxysmal:*** pertussis, foreign body.
- ***Worse during exercise:*** asthma.
- ***Disappears when sleeping:*** habitual cough.
- ***During/after feeds:*** aspiration.

Palpation

- Feel the neck for enlarged cervical lymph nodes.
- Palpate the trachea to ensure that it is central.
- Then move onto the chest:
  - Feel for the apex beat. This may be displaced in effusion, collapse, or tension pneumothorax.
  - Assess expansion (see p.212) commenting on extent and symmetry.
  - In young children, you may be able to feel crackles.

Percussion

Percussion is rarely useful in infants and toddlers. Remember to also percuss for the normal cardiac dullness as well as the upper and lower borders of the liver.

- Dull = consolidation.

- Hyperresonant = air-trapping or pneumothorax.
- Stony dull = pleural effusion.

Auscultation

Before using a stethoscope on the child, pretend to auscultate the parent's chest or a less vulnerable part of the child's body (e.g. their leg).

▸ Remember to listen under the axillae as well as the anterior and posterior chest wall.

▸ Especially in young children, the upper airway noises may be transmitted to the chest, so if the child is old enough, ask them to cough to clear them.

Listen for:

- Breath sounds.
  - Are they vesicular (normal), absent or bronchial?
- Added sounds (e.g. wheeze or crackles—see adult pages for more details.
- Absent breath sounds in one area suggests a pleural effusion, pneumothorax, or dense consolidation.

Table 16.2 Some common respiratory conditions and signs

Condition	Age	Inspection	Auscultation
Bronchiolitis	<1 year	Pale, coryza, cough, recessions, tachypnoea	Wheeze and crackles throughout chest
Croup (laryngo-tracheobronchitis)	1-2 years	Stridor, hoarse voice, barking cough	Clear
Asthma	>1 year	Tachypnoea, recessions ± audible wheeze and use of accessory muscles	Wheeze, variable air entry throughout chest. Crackles in young children
Pneumonia	Infant	Tachypnoea, recessions, flushing due to fever, grunting	May be clear, reduced breath sounds over affected area, crackles
Pneumonia	Child	Tachypnoea, recessions, flushed, generally unwell	Abdominal pain (may be the only symptom), crackles and bronchial breathing over affected area

▸ ↑ respiratory rate and work of breathing are the most important signs of a lower respiratory tract infection in infancy, as sometimes palpation, percussion, and auscultation will be normal.

Ear, nose, and throat

ENT conditions are a common reason for children to present to the doctor.

Examination of this system should be left until last, as children find it unpleasant.

Key points from the history

- Does the child pull at their ears (suggests infection)?
- Does the child complain of earache or a sore throat?
- Are they coryzal (runny nose)?
- Does the child have a fever?
- Does the infant drool more than normal (suggests sore throat)?

Examination

Ears

- Sit the child on the parent's lap facing to the side.
- Ask the parent to hold the child's head against their chest with one hand, and to firmly hold the child's arms and upper body with the other hand (see Box 16.7).
- With an infant, gently pull the pinna back before inserting the auroscope. When examining an older child, pull the pinna upwards.
- Use the auroscope as in adults—see p.142.

Nose

- Examine the nose externally for discharge.
- The nose may be examined very gently using an auroscope.
  - Polyps are a common finding in asthma and cystic fibrosis.
  - Pale, boggy nasal mucosa suggests allergic rhinitis.

Throat

- Sit the child upright on the parents lap facing towards you.
- Ask the parent to hold the child's forehead with one hand, with the back of the child's head against their chest.
  - The parent should firmly hold the child's arms with their other hand.
- The difficulty now is encouraging the child to open their mouth!
  - Ask the child to open their mouth ‘as wide as a lion’.
  - Tempt an infant to open their mouth with a dummy.
  - Sometimes children will be more inclined to open their mouth if you promise not to use a spatula.
- When the child's mouth is open, gently depress the tongue with the spatula if it is obstructing the view of the tonsils.
- Decide whether the tonsils are:

- Normal: pink and small.
- Acutely inflamed: red, enlarged, sometimes with pus spots.
- Chronically hypertrophied: enlarged and pitted, but not inflamed.

Lymph nodes

- Always feel for cervical and supraclavicular lymphadenopathy.

Box 16.7 More on ear examination

While asking the parent to hold the child's head during the ear examination is the usual taught method, this often leads to a struggle.

It is equally appropriate to allow the child free movement of the head providing you splint the hand holding the auroscope against the child's face so that your hand (and auroscope) will move as the child's head moves. This can lead to a less distressing examination.

 In infancy, the pinna should be pulled forwards (not backwards) to straighten the auditory canal.

Table 16.3 Some common findings when examining the ear drums

Appearance of drum	Condition
Translucent, clear light reflex	Normal
Red, bulging, loss of light reflex	Acute otitis media
Retracted, loss of light reflex, dull	Glue ear (chronic otitis media with effusion)

The cardiovascular system

Key points from the history

- Does the child ever have blue spells (cyanosis)?
- Does the child ever become tired, pale, or sweaty (indicating heart failure)?
- If the patient is an infant, ask how long the child takes to feed from a bottle. Breathlessness may inhibit feeding.
- Is the child growing normally? Plot on a centile chart.
- Does the child suffer from recurrent chest infections?
- Does the child suffer from abdominal pain (caused by organomegaly)?
- Is there a history of fainting or collapse?
- Has the child ever complained of their heart racing (would imply an arrhythmia such as supraventricular tachycardia)?
- Is there a FHx of congenital heart disease?

Examination

Inspection

Search for evidence of heart failure: pallor, cyanosis, sweating, respiratory distress and tachypnoea.

Hands

- Clubbing—seen in cyanotic congenital heart disease.
- Search for signs of endocarditis, including splinter haemorrhages, Janeway lesions and Osler's nodes.

Face

- Anaemia in the conjunctiva.
- Central cyanosis (‘stick your tongue out!’).

Neck

The jugular venous pulse and pressure are difficult to appreciate in young children, due the relative shortness of the neck.

Peripheral pulses

Palpate the radial, brachial, and femoral pulses.

 The femoral pulse, although sometimes awkward to feel, must always be sought to ensure coarctation of the aorta is not missed.
Assess:

- Volume: is it full or thready? (You will need to practice feeling lots of pulses to appreciate the difference.) A thready, weak, or small volume pulse is indicative of hypovolaemia. Look for pulsus paradoxicus ( p.172).
- Rate: heart rate varies with age, activity, distress, excitement and fever (the pulse rate will ↑ by 10bpm with every temperature rise of 1°C).
- Rhythm:
  - Sinus arrhythmia: an ↑ in pulse rate on inspiration, with slowing on expiration. Very common in children.
  - Occasional ventricular ectopic beats: normal in children.
- Character:
  - Collapsing pulse in children is most commonly due to a patent ductus arteriosus.
  - Slow rising pulse suggests ventricular outflow obstruction.

Blood pressure

Blood pressure recordings in children are not easy, but they are important, so remember perform this test. The use of the correct cuff size is vital here to prevent inaccurate readings.

Anxiety and poor technique are the most common causes for raised blood pressure in children, so it should be measured several times.

Chest

Note the presence of:

- Precordial bulge: causes the sternum and ribs to bow forwards.
- Visible ventricular impulse: RV impulse may be visible under the xiphisternum. The LV impulse (apex beat) is often visible in thin children, and in children with true LV hypertrophy.
- Scars: indicative of previous heart surgery.

Palpation

- Feel the *apex beat* to determine its location and character. It is usually situated in the 4th intercostal space in the mid-clavicular line in infants or toddlers (often difficult to localize if they are plump), and in the 4th or 5th intercostals space in older children.
  - LV hypertrophy results in a diffuse, forceful, and displaced apex beat, felt as a 'heave'.
  - If the apex is impalpable, consider dextrocardia (inverted heart with apex pointing to the right) or pericardial effusion. 'Laevocardia' is the normal orientation of the heart to the left.
- *Right ventricular heave:* place your fingertips along the left sternal edge. If the child has right ventricular hypertrophy you will feel your fingers lift up with each impulse.
- Palpate in *the four valve areas* (aortic, pulmonary, tricuspid, and mitral) for thrills.
- *Palpate the abdomen* for hepatomegaly, which suggests heart failure (remember to percuss the upper border of the liver-a normal-sized liver may be displaced downwards by lung disease such as bronchiolitis. Raised JVP, pulmonary, and peripheral oedema is rarely seen in children.

Auscultation

Listen to the heart sounds in the 4 valve areas with the diaphragm and bell of the stethoscope (preferably paediatric size).

First heart sound (S_1)

Best heard at the apex with the bell.

- Loud S_1 heard with high cardiac output states (e.g. anxiety, exercise, fever).
- Soft S_1 heard with emphysema and impaired left ventricular function.

Second heart sound (aortic = A_2 and pulmonary = P_2)

Best heard at the base with the diaphragm. It is normally split in children.

- Soft P_2 heard with stenotic pulmonary valve (e.g. tetralogy of Fallot).
- Loud P_2 heard with pulmonary hypertension.
- Wide fixed splitting caused by atrial septal defect.

Third heart sound

Due to rapid ventricular filling.

- Causes include ↑ LV stroke volume (aortic or mitral regurgitation) and restricted ventricular filling (constrictive pericarditis, restrictive cardiomyopathy). It may be normal in children.

Forth heart sound

Due to forceful atrial contraction.

- Causes include hypertrophic cardiomyopathy and severe hypertension.

Murmurs

Auscultate for murmurs over the 4 valve areas, and at the back. About 30% of children have innocent murmurs.

Innocent murmurs

Patient asymptomatic. Systolic (except venous hum). No radiation or thrill. Change with altering patient posture.

- *Venous hum:* due to turbulent flow in the head and neck veins. A continuous murmur in diastole and systole heard below the clavicles

which disappears when child lies flat.

- *Ejection murmur:* due to turbulent flow in the outflow tracts of the heart. Heard in the 2^{nd}-4^{th} left intercostals spaces.

Pathological murmurs

Systolic or diastolic. May radiate. May have a thrill. Patient may be symptomatic.

- *Atrial septal defect:* soft ejection systolic murmur at the upper LSE due to ↑ RV outflow. Fixed wide splitting of the 2^{nd} heart sound may first be detected at school entry.
- *Ventricular septal defect* parasternal thrill. Loud pansystolic murmur at the lower LSE. Radiates throughout precordium. Signs of heart failure may be present.
- *Coarctation of the aorta:* ejection systolic murmur heard between the shoulder blades. Femoral pulses weak or absent.
- *Patent ductus arteriosus:* Collapsing pulse. Continuous 'machinery murmur' below the left clavicle.

Also see 🕮 p.185.

Table 16.4 Quick-spot guide to common paediatric murmurs

Symptom	Cause
Cyanosis + murmur	Usually tetralogy of Fallot
Cyanosis + murmur + operation	Possibly tetralogy of Fallot or transposition of the great arteries
Pink + loud systolic murmur	Probable ventricular septal defect (commonest form of CHD)
Pink + murmur + impalpable femorals	Coarctation of the aorta
Continuous low pitched murmur	Probable patent ductus arteriosus

The abdomen and gastrointestinal system

Key points from the history

Determine whether the child takes in sufficient calories for growth and has a well balanced diet. Ask about height and weight gain.

When taking a history, start at the head and work down to avoid missing things.

- Does the child have a good appetite?
- Does the child vomit?
  - How much?
  - Are they hungry afterwards?
  - Is it forceful or effortless?
  - Is it related to feeds?
  - What does it contain? Ask about coffee-grounds or other appearances of the vomit. (Bile-stained vomiting in an infant always indicates obstruction and must be considered as pathological.)
- Does the child suffer from abdominal pain?
- Does the child ever have a bloated abdomen?
- Are there any urinary symptoms?
- Ask about bowel habit—is the child constipated?
- Have there been any frequent or loose stools? Are the stools particularly offensive (suggests malabsorption).
- Is there a relevant FHx (e.g. Coeliac or inflammatory bowel disease)?

Examination

Inspection

Start with a general inspection of the patient, looking especially for:

- Visible liver edge or spleen.
- Peristalsis (important in diagnosing pyloric stenosis during a test feed).
- Jaundice.
- Observe for signs of liver disease (see 📖 p.270), including spider naevi, xanthomata, and purpura.
- Oedema over the tibia and sacrum.
- Whether the child is under- or overweight.
- Wasted buttocks (suggesting weight loss—typical of coeliac disease).

Hands

Clubbing, palmar erythema.

Face

- Check the conjunctivae for anaemia.
- Periorbital oedema (e.g. in nephrotic syndrome).

Abdomen

- Abdominal distension.
- Gross ascites may be evident—the abdomen will be distended and the umbilicus everted.
- Caput medusae (cutaneous collateral veins with blood flowing away from the umbilicus due to ↑ portal venous pressure— 📖 p.248).

Box 16.8 Causes of abdominal distension in a child

- Fat.
- Fluid.
- Flatus.
- Faeces.
- Organomegaly.
- Muscle hypotonia.
- Exaggerated lordosis (normal in young children).

Box 16.9 Detecting peritoneal inflammation

A further useful technique is to ask the patient to make their belly ‘as fat as possible’ and ‘as thin as possible’. Also ask them to cough. In the case of peritonitis, any of these manoeuvres may result in pain.

Palpation

Young children may resist abdominal examination. First try distraction techniques. If these fail, use the child's hand to guide yours around the abdomen. If there is doubt as to the significance of tenderness in a child's abdomen, listen with your stethoscope and gently apply more pressure. Often quite firm pressure can be tolerated in this way where there was previously tenderness.

The aims of palpation are to:

- Determine the presence of normal abdominal organs.
- Detect enlargement of the abdominal organs.
- Detect the presence of abnormal masses or fluid.

Procedure

- Ensure the child is relaxed and that your hands are warm.
- Enquire about pain before you begin.
- Palpate for tenderness (light palpation first, then deep palpation).
    - Feel for guarding (tensing of the abdominal muscles which may indicate underlying tenderness).
- Palpate the spleen. This is normally felt 1-2cm below the costal margin in infancy. It is soft and can be ‘tipped’ on inspiration. Begin palpation in the right iliac fossa and move towards the left upper quadrant to avoid missing a very large spleen. It may help to turn the child onto their right side.
- To palpate the liver, start in the right iliac fossa and move upwards in time with the child's respiratory movements until the liver edge meets your fingers. A liver edge 1-2cm below the costal margin is normal up to the age of 2 or 3 years.
    - Kidneys are not easy to palpate in children (they are easier to palpate in newborns), so if you can feel them they are probably enlarged. They are best palpated bimanually. The kidneys move with respiration, have a smooth outline, and one can get above them (unlike the liver and spleen).
- Palpate for other masses and check for constipation (usually felt as a hard, indentable, non-tender mass in the left iliac fossa).

Percussion

- Ascites. Test for shifting dullness (described on p.260) as a sign of ascites.
- Gaseous distension.
- Percuss to determine the size of the liver and spleen.

Auscultation

Bowel sounds.

Rectal examination

This is rarely indicated in children. However, it is often useful to inspect the perianal region for fissures, tags, soiling, and threadworms.

Male genitalia

Penis

True micropenis is rare. If the penis looks small, it is probably because it is buried in suprapubic fat.

Check the urethral orifice is at the tip of the glans. If not, is there epispadius (dorsal opening, very rare), or hypospadias (ventral opening)?

Scrotum

The child should be standing up.

- Inspect for normal rugosity of the scrotum.
- Palpate for the testes.
  - If they are not present in the scrotum, feel at the inguinal canal and, if found, try to milk the testis down.
  - Many undescended testes are subsequently found on re-examination as retractile testes, so be gentle in your approach to avoid provoking a cremasteric reflex!

Female genitalia

Inspect the female external genitalia if there are urinary symptoms.

Box 16.10 Confirming hepatomegaly

If in doubt, confirmation of liver enlargement can be made by:

- Placing the stethoscope over the xiphisternum.
- Gently rubbing the abdomen, progressing upwards from the right iliac fossa.
  - When the rubbing hand is over the liver, the sound will be heard through the stethoscope.

The nervous system

Key points from the history

- Detailed birth and perinatal history—including maternal drugs/illness.
- Careful history of the developmental milestones.
- Hearing or visual concerns. Did they pass the newborn hearing screen?
- Any change in school performance, personality or behaviour (e.g. aggression)?
- Ask about symptoms of raised intracranial pressure (e.g. headache, vomiting).
- Any change in gait or frank ataxia?
- Does the child have limited function-what can they do? What do they need help with?
- Relevant FHx of learning difficulties or genetic conditions.

Examination

Examination of the nervous system in school-age children should be performed as in adults. Young children cannot cooperate with a formal neurological examination, so assessment is opportunistic-observation becomes important. The assessment of a young child is described below.

Young children—where to start

- Palpate the anterior fontanelle when the child is quiet to:
- Determine the presence of raised intracranial pressure (felt as fullness or bulging).
    - Determine the degree of dehydration (felt as a sunken fontanelle).
- ▸ It is impossible to assess fontanelle tension in crying babies.
- ▸ Pulsation of the fontanelle is normal.
- Measure the maximum occipito-frontal head circumference and plot this on a centile chart.

Cranial nerves

It is not possible to systematically examine cranial nerves in infants or young children, below is a rough guide. (See  Chapter 10.)

- *I (olfactory):* very difficult.
- *II (optic):* ask parents—can the child see?
- *III, IV,* VI *(eye movements):* gain the infant's visual attention with an object and move it back and forth. Watch for the range of ocular movements as the child tracks the object. Pendular nystagmus may indicate a visual defect.
- *V (trigeminal):* rooting reflex
- *VII (facial):* facial palsy will become apparent when the child cries. Asymmetry will be more obvious. Does the child close both eyes?
- *VIII (vestibulocochlear):* formal hearing tests are performed at birth.
- *IX, X:* swallowing.
- *XI (accessory):* neck and shoulder movements.
- *XII (hypoglossal):* tongue movement.
- *Pupils:* check for size, shape, and reaction to light.
- *Fundi:* should be examined but should be left to the end of the examination, as it is unpleasant (and sometimes impossible).

Box 16.11 Assessment of a squint

Any squint persisting beyond the age of 6 weeks needs specialist assessment, as an untreated squinting eye may lead to amblyopia (cortical blindness).

- Ask when the squint is most apparent—latent squints may only be present when the child is tired.

Examination

- *Corneal light reflection test* shine a torch at a spot directly between the patient's eyes to produce a reflection in the cornea. The reflected light that you see should be at the same spot on each eye. If the reflection from the corneas is asymmetrical, a squint is probably present.
- *Eye movements:* to detect a paralytic squint (rare).
- *Cover test:* encourage the child to fix on a toy, and cover the normal eye with a piece of card. If the fixing eye is covered, the squinting eye moves to take up fixation.
    - *Manifest (constant) squint:* on removal of the cover, the eyes move again as the fixing eye takes up fixation.

Tone

- Observe the child's response to gravity (see  p.546).
    - *Hypotonic infant* slips through in ventral suspension (holding them up with a hand in each axilla), droops over your hand during ventral suspension, head lag when pulled to sit, ‘frog legs’ posture.
    - *Hypertonic infant* scissoring of lower limbs when the baby is picked up. Resistance to movement of limbs. Baby will seem to move ‘in one piece’.
- Move the limbs through their range of movements. Important areas to examine include:
- Arm flexors and extensors.

- Arm supinators and pronators (most sensitive for ↑ tone).
- Hip adductors.
- Leg extensors.
- Leg flexors.
- Ankle extension.

Power

Observe:

- Symmetry.
- Spontaneous movements. (Reduced movement is indicative of muscle weakness.)

Sensation

Difficult to assess. Only response to pain can be confidently elicited in young children, but please don't try this!

Reflexes

- *Tendon reflexes:* can be elicited by tapping the tendon with a finger in babies and using a tendon hammer in children.
  - The examiner should know the nerve roots responsible for the reflexes (📖 p.330).
  - ▸ Remember that the plantar response is upwards until 8 months.
- *Primitive reflexes:* the presence of 'primitive reflexes' beyond 6 months of age is abnormal and will inhibit normal development.
  - Persistence is indicative of an UMN lesion (e.g. cerebral palsy). See Table 16.5.

Coordination (assess in older children)

- Stand on one leg and then hop.
- Walk on tip toes.
- Walk on heels—a good test for co-ordination and overall neurological integrity. Patients with any kind of spasticity, for example, will be unable to do this.
- Finger-nose test and heel-shin test if the child is old enough.

Gait (assess in older children)

- *Spastic gait:* spasticity of extensor muscles causes a stiff gait on a narrow base. Toes catch the ground first (e.g. cerebral palsy).
- *Hemiplegic gait:* if the spasticity is unilateral, the affected leg drags stiffly and is circumducted as it is brought forward
- *Ataxic gait* broad based, unsteady, with frequent falls.
- *Lower limb weakness (distal):* affected leg is lifted over obstacles, then the foot returns to the ground with a slap.
- *Lower limb weakness (proximal):* waddling gait as the pelvis is thrown side to side, being poorly supported by the lower limbs (e.g. muscular dystrophy).
- *Limp:* has several causes, but always rule out dislocation of the hip.

Table 16.5 Some primitive reflexes and age of extinction.

Reflex	Age of extinction
Stepping reflex	2 months
Palmar grasp	3-4 months
Moro reflex	4-5 months
Asymmetric tonic neck reflex	6 months

Developmental assessment

Development is a continuous process, the rate of which varies considerably between normal children.

Development is divided into 4 areas:

- Gross motor.
- Fine motor and vision.
- Speech and hearing.
- Social.

Delay in all 4 areas is usually abnormal, but delay in one area may not be. For example, some children become expert at bottom shuffling and, having learned an effective means of travelling, the need to walk becomes less important.

Performing a developmental assessment

- Observation is key. Young children will often not cooperate. Take a history from the parents of which milestones the child has achieved.
- Be systematic and evaluate each of the 4 developmental areas in turn.
- Learn a few essential milestones, as it is difficult to remember them all.
- ▸ If an infant was born prematurely, allow for this by calculating their ‘corrected age’ from their expected due date.
- Limit distractions and present one task at a time.

Equipment for developmental assessment

- Wooden blocks: for assessing palmar grasp and building towers.
- ‘Hundreds and thousands’: for testing pincer grip.
- Pencil and paper: for assessing fine motor skills.
- Different coloured card/colourful book.

Table 16.6 Developmental warning signs

Age	Warning sign
Any	Regression in previously acquired skills or a halt in developmental progress.
8 weeks	No smiling.
6 months	Persistent primitive reflexes. Hand preference (this should not appear until 18 months).
12 months	No sitting. No pincer grip. No double babble.
18 months	Not walking. No words.
4 years	No words.

Table 16.7 Developmental milestones

Age	Gross motor	Fine motor	Language	Social
3 months	Head control, pushes up with arms	Opens hand	Laughs	Smiles (6 weeks)
6		Palmar grasp,	Babbles	

months	Sits	reaches, transfers	(monosyllabic—ba, ka, da)	Eats solid food
9 months	Crawls, pulls to stand	Pincer grip begins to develop	Double babble (dada, baba)	Stranger awareness, waves bye-bye
12 months	Walks	Developed pincer grip	Mummy, daddy—specifically	Peek-a-boo
18 months	Walks upstairs, jumps	Scribbles, 3 blocktower	2-word phrases	Mimics
2 years	Kicks, runs	Draws straight line, 6-8 blocktower	Beginning to use clauses (including verbs)	Uses spoon skilfully, undresses, symbolic play
3 years	Hops, walks upstairs adult-style	Draws a circle, builds a bridge with blocks	Says name, knows colours	Dresses, has a friend, dry nappies by day
4 years	Stands on one leg, hops	Draws a cross, makes 3 steps with blocks	Sentences of 5+ words	Does up buttons
5 years	Can ride a bicycle	Draws a triangle		Ties shoe laces, dry by night

The newborn

The vast majority of newborns have a normal intrauterine life, normal birth, and are physically normal. However, there is a wide variation in the spectrum of normal, and it is important to stress the value of examining a large number of neonates to appreciate the normal spectrum.

In the delivery room

All newborns should have a brief examination at birth to determine whether resuscitation is needed and to rule out any major abnormalities.

The APGAR score is used to gauge the need for resuscitation (Table 16.8).

Table 16.8 APGAR score

Sign	0	1	2
Appearance	White/blue	Blue extremities, pinktrunk	Pink
Pulse	Absent	<100bpm	>100bpm
Grimace on stimulation of foot	None	Frown/grimace	Cry
Activity, tone	Floppy	Some limb flexion	Active movement
Respiratory effort	Absent	Irregular, slow	Loud cry

On the post natal ward

A more thorough examination is carried out prior to discharge. At this stage, the baby is unrecognizable from the one you met in the delivery room—they will be pink, vigorous, and feeding well.

Ask briefly about whether the baby has passed urine and meconium (the first, black sticky stool), and enquire as to the progress of feeding, as well as a FHx of congenital anomalies. Of particular importance is a FHx of dislocated hips, renal abnormalities, and deafness.

- Examination should start at the top and work down, to ensure nothing is missed.
- Undress the baby yourself as the examination proceeds, to get a feel for how the baby handles.

General observation

First observe the baby without disturbing him/her.

- *Colour:* pink, pale, cyanosed, or jaundiced? Acrocyanosis (cyanosis of the hands and feet) is normal provided the lips and tongue are pink.
- *Rash:* a blotchy erythematous rash occurs in about half of all neonates, this is usually harmless and is called erythema toxicum.
- Peeling of skin is common, especially in post-dates babies.

Head and face

- *Shape of the head:* can vary widely in the first week.
- *Fontanelles:* they should be soft and flat. The size of the anterior fontanelle also varies widely, from 1-4cm in diameter. The posterior fontanelle may accept a little fingertip.
- *Cranial sutures:* are they fused?
- *Look for trauma from the birth:* such as caput succedaneum (oedema caused by pressure over the presenting part) and moulding (head changing shape as it passes through the birth canal), forceps marks, and subconjunctival haemorrhages. In general, these conditions will resolve within the first week.
    - A cephalhaematoma is a localized fluctuant swelling usually over the parietal bone, caused by subperiosteal bleeding. This will resolve over a few months.
- *Ears:* can be of different shape and size. Look for preauricular sinuses and ear tags, and observe their position.

Mouth

- *Palate:* look at it when the infant cries, then palpate it for a cleft with a clean finger.
    - 'Epstein's pearls' are small white cysts in the midline of the hard palate. They are normal and resolve spontaneously.
- *Jaw:* a small jaw (micrognathia) may be part of the Pierre Robin sequence (midline cleft, small jaw, posterior displacement of the tongue—can cause upper airway obstruction).
- *Tongue:* note the size. If it is large and protruding, this may indicate a number of syndromes (e.g. Down's syndrome).

Eyes

- Note position and size.
- Look for the red reflex with an ophthalmoscope to exclude a cataract, which would be seen as a white reflection.
    - To encourage the baby to open their eyes, wrap them in a blanket (a crying baby will not open their eyes) and sit them upright.
    - If this fails, give the baby something to suck on, or startle with the Moro reflex.
- Sticky eyes can be the result of ophthalmia neonatorum (purulent conjunctivitis in the first 3 weeks of life). Usually due to accumulation of lacrimal fluid due to incomplete drainage of the nasolacrimal duct.

Respiratory system and chest

- *Observe:* this is best done in a quiet baby (either sleeping or with the aid of a dummy).
- *Chest:* comment on size, symmetry, and shape.
- *Respiratory rate:* should be <60/minute. Note the work of breathing. Are there any subcostal or intercostal recessions? Is the baby grunting?
    - Normal newborn respiration should be quiet, effortless, and predominantly diaphragmatic (abdomen moves more than the chest).
- *Auscultate:* the lung fields to ensure symmetrical air entry. Crepitations may be normal in the first few hours of life.
- *Breasts:* engorgement is common in male and female infants.

Cardiovascular system

- *Observe:* note colour, respiratory effort and precordial heave.
- *Apex beat:* palpate and feel any thrills (not uncommon in neonates).
- *Femoral pulse:* this is extremely important; its absence may imply coarctation of the aorta. This requires a relaxed, still baby and lots of patience. Remember that too much pressure may obliterate it. A collapsing pulses suggest patent ductus arteriosus.
- *Heart rate:* should be between 100-160bpm.
- *Auscultate:* for the heart sounds and murmurs. Systolic murmurs are common, and usually best heard along the left sternal edge.

Abdomen

- *Observe:* distension could be bowel obstruction or abdominal mass.
- *Umbilical stump:* count the three vessels. Note any signs of infection such as an unpleasant smell, discharge, or periumbilical erythema.
  - The cord will spontaneously separate around the 4^{th} or 5^{th} day.
- *Palpate:* gently feel the abdomen for the intra-abdominal organs and exclude organomegaly. Use warm hands and a soother if necessary.
  - The liver edge is soft and easily missed.
- *Kidneys:* determine presence and size by balloting.
  - It is possible to palpate the lower poles of the kidneys in normal neonates.
- *Bladder:* palpate suprapubically. If felt, suggests outlet obstruction.
- *Anus:* infants with an imperforate anus may still pass meconium via a fistula, so check the anus is patent and in the correct position.

Male genitalia

- *Urethra:* identify the urethral orifice and exclude hypospadias.
- *Testes:* palpate gently. If they cannot be found in the scrotum, commence in the inguinal area and palpate downwards.
  - If a testis appears larger than normal, transilluminate the scrotum (p.413) to check for the common condition of hydrocele.
- *Inguinal herniae:* these are more common in preterm infants.
- Put the nappy back on quickly for obvious reasons.

Female genitalia

- *Labia minora:* may not be fully covered, especially in preterm infants.
- *Vaginal tags:* are common and resolve spontaneously in the first week.
- *Vaginal discharge:* and occasionally bleeding can occur, and is normal.
- Note ↑ pigmentation and clitoromegaly.

Limbs

- Ensure all joints have full range of movement to exclude any congenital contractures.
- Examine fingers and toes for syndactyly (fused digits) or polydactyly (extra digits).

Examination of the hips

- This is to detect congenital dislocation and instability of the hips, and should be left until last as it will make the baby cry.
- Observe for unequal leg length and asymmetry of skin creases.

- Hip examination is in 2 parts. Lay the infant supine on a flat surface with hips and knees positioned at 90°. Stabilize the pelvis with one hand, and with the other grasp the knee between thumb and palm, with the finger tips over the greater trochanter.
  - Barlow test: assesses whether the hip *can be* dislocated. Pull the hip up and then push downwards and laterally.
  - Ortolani test: assesses whether the hip *is* dislocated. Pull the hip upwards into the acetabulum (producing a 'clunk'), then the hip can be abducted. (Ortolani = out).

Feet

- Talipes equino varus: primary club foot. Usually a fixed structural deformity requiring early manipulation and fixation.
- Calcaneo valgus: common. Dorsum of the foot is in a position close to the shin. Resolves after about 2 months with ↑ calf muscle tone.
- Positional talipes is extremely common and involves no bony deformity. It is easily corrected by movement and treated with physiotherapy.

Spine

Lie the infant prone in one hand, and with the other palpate the spine, checking for spina bifida occulta or a dermal sinus.

Neurological examination

Because infants with little or no cerebral cortex can show normal reflexes and tone, you should observe the baby's state of consciousness throughout the examination. This should vary from quiet sleep to semiwakefulness to an alert state. A normal infant will be consolable when they cry, whereas it is very difficult to settle a neurologically abnormal infant.

Inspection of the spine

Any midline lesion over the spine requires immediate investigation. Even a single hair might indicate communication with the spinal column (spina bifida).

Posture

Generally flexor, although abnormal intrauterine positions can distort this, such as extended breech position.

Movements

Watch spontaneous limb movements noting the presence of ‘jitteriness’.

Tone

Assess and compare the flexor recoil of the limbs.

Evaluate tone in response to gravity:

- Pull to sit test. Let the baby grasp your fingers and pull them up to sit. The head should flex and follow the traction to an upright position and hold momentarily. Also observe the tone in the baby's arms.
- Ventral suspension is assessed by grasping the infant under each axilla. A normal infant will support themselves in this position by extending their back and hips, lifting their head, and flexing their arms and legs.

Primitive reflexes (See Box 16.12)

These are used to assess asymmetry of function, gestational age, and neurological function.

Vision

Assessment of vision should be carried out with the infant in an alert state. The baby will fix on an interesting object 20cm away, and will follow the target.

Hearing

This can be assessed by sounding a loud rattle outside of the infant's vision. The baby should still to the noise.

Head circumference and weight

Finally, measurement and plotting of head circumference and birthweight on a centile chart is of utmost importance.

Box 16.12 Primitive reflexes

- ***Palmar grasp:*** fingers close to hold an object placed in the palm.
- ***Rooting:*** when pressure is applied to the cheek, the head turns towards the pressure and mouth opens.
- ***Sucking:*** when a finger is place in the mouth, the infant will suck vigorously.
- ***Stepping:*** hold the infant with both hands and lower the feet onto a surface. The legs will move in a stepping fashion.

- ***Moro reflex:*** lay the infant supine on your hand and forearm. When the head is dropped a few centimetres, the upper limbs abduct, extend, and flex in a symmetrical flowing movement. A unilateral response indicates damage (usually transient) to the 5^{th} and 6^{th} cervical roots producing Erb's palsy.

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Chapter 17

Practical Procedures

Using this chapter

- This chapter describes those practical procedures that the junior doctor or senior nurse may be expected to perform.
- Obviously, some of these are more complicated than others—and some should only be performed once you have been trained specifically in the correct technique by a more senior colleague.
- Each procedure has a difficulty icon as follows:

 Requires no specific further training and all medical graduates should be competent to perform.

 Requires some skill. Doctors in their 2nd year after graduating should be able to perform with ease.

 More complex procedures which you may only come across in specialty jobs and will not be required to perform without specific guidance from seniors.

Rules are made to be broken

- Very many procedures and practical skills do not have a 'correct' method but have an 'accepted' method.
- These methods should, therefore, be abided by but deviation from the routine by a competent practitioner, when circumstances demand is acceptable.
- Many procedures have local variations—if in doubt, you should check the standard method that is used in your hospital or trust.

Infiltrating anaesthetic agents

A large number of procedures involve the infiltration of local anaesthetic agents. It is important that you deliver these safely—injection of a large amount of anaesthetic into a vein could lead to potentially fatal cardiac arrhythmias. It is also important, of course, to ensure that you do not damage any vessels.

Advance and withdraw

Whenever you inject anything, you should advance the needle and attempt to withdraw the plunger at each step-if you do not aspirate blood, you may *then* go ahead and infiltrate the anaesthetic.

Making a surface bleb

- Take the syringe of anaesthetic (e.g. 1% lidocaine) and a small needle.
- Pinch a portion of skin, insert the needle horizontally into the surface.
- Withdraw, as above, and inject a small amount of the anaesthetic—you should see a wheal of fluid rise.
- The area of skin will now be sufficiently anaesthetized to allow you to infiltrate deeper.

Sterility and preparation

Most equipment will come in pre-packed sterile wrapping. When performing a procedure where sterility is important, all packaging should be opened using a 'no-touch' technique.

Large ‘packs’ of equipment

Some equipment is available in pre-prepared sterile ‘packs’. For example, a ‘catheterization pack’ contains gauze, cotton balls and a sterile pot. These come wrapped in sterile tissue paper.

Any such packs should be placed on a trolley which has first been cleaned with antiseptic solution. You should then carefully open the pack out, touching the corners only—and using gloved hands.

The opened pack can then be used as a sterile surface on which to place additional sterile equipment.

Smaller pieces of equipment

Most equipment (e.g. needles, syringes) comes sterilized and wrapped in paper/plastic. These should also be opened using a no-touch technique if absolute sterility is needed.

For example, unwrap a needle by peeling back the packaging as if peeling a banana and allow the needle to drop onto the pre-prepared sterile surface.

Hand washing

Theory

Hand washing is the single most important procedure for preventing the spread of infections. It is underperformed in terms of frequency and quality. Hands should be washed before every episode of care that involves direct contact with a patient's skin, their food, invasive devices, or dressings, and after any activity or contact that potentially results in hands becoming contaminated.

Alcohol handrub should also be regularly used when entering or leaving a ward and before and after examining patients.

Equipment

- Soap/alcohol gel.
- Disposable paper towels.
- Moisturizer (if required).

Procedure

If hands are not visibly soiled, hand hygiene with alcohol is as, if not more, effective than handwashing.

When required to wash our hands we should use soap and warm water. Those parts often missed are the tips of fingers, thumbs, and between the fingers.

The following routine is advised in most trusts (Fig. 17.1):

- First, rub hands palm to palm (Fig. 17.1a).
- Rub right palm over the left dorsum.
- Rub left palm over the right dorsum.
- Wash palm to palm with the fingers interlaced (Fig. 17.1b).
- Wash the backs of the fingers with opposing palms with fingers interlocked (Fig. 17.1c).
- Perform rotational rubbing of the right thumb clasped with the left fist (Fig. 17.1d).
- Perform rotational rubbing of the left thumb clasped with the right fist.
- Wash the right palm with rotational rubbing using the fingers of the left hand.
- Wash the left palm with rotational rubbing using the finger of the right hand (Fig. 17.1e).
- Wash the space between the thumbs and first fingers by interlocking them and rubbing together (Fig. 17.1f).
- Rinse away all soap and pat dry using disposable paper towels.
- Apply moisturizer to protect the skin from the drying effects of regular washing.

▸ *Hints*

- Keep nails short, clean and polish free.

- Avoid wearing wrist watches and jewellery, especially rings with ridges or stones.
- Any cuts or abrasions should be covered with water-proof dressing.

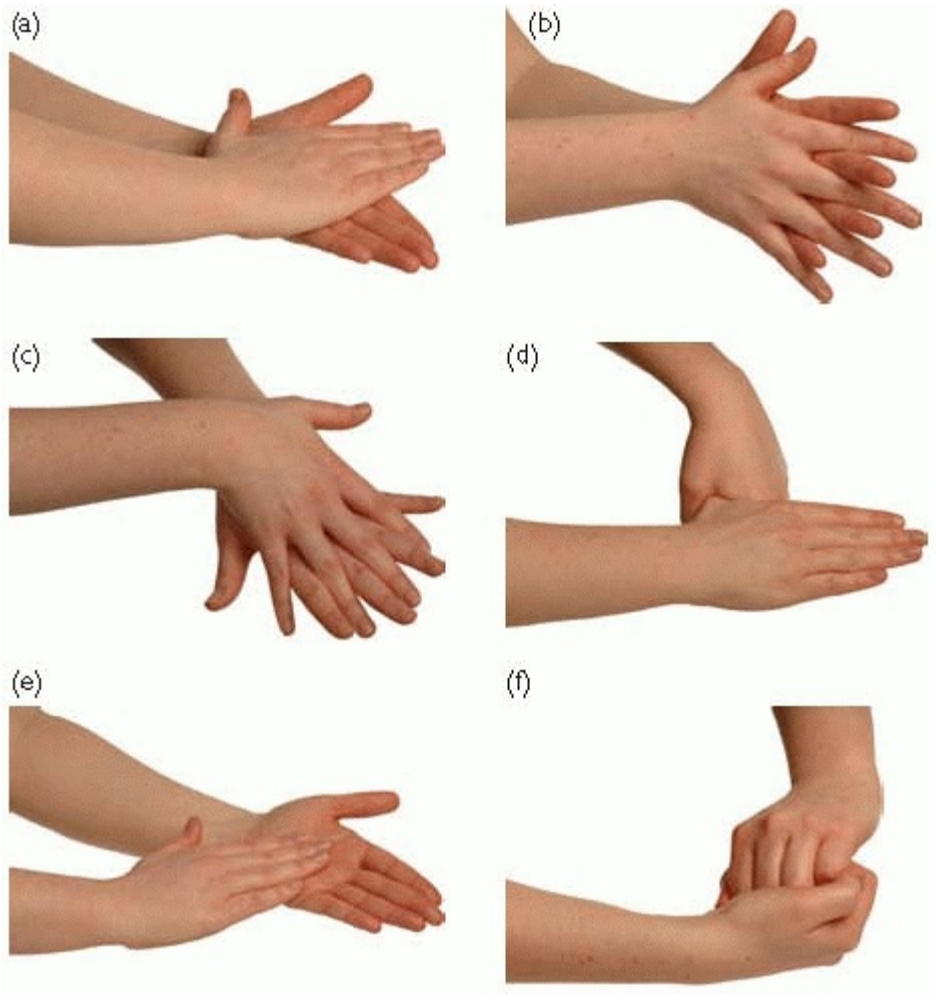


Fig. 17.1 The correct stages of hand washing as described opposite.

We thank Lyn Dean for her assistance with this topic.

Injections

Theory

This is an important and routine procedure which is often carried out by nursing staff, although doctors may be asked to administer medication at times. Good injection technique can make the experience for the patient relatively painless. Three commonly used routes of administration are subcutaneous (S/C), intramuscular (IM) and intradermal (ID).

Equipment

- Syringe (size depends on injection).
- Needles: 25-gauge for S/C route; 21-23-gauge for IM route.
- Extra 21-guage needle for drawing up dose.
- Alcohol swab.
- Gloves.
- Cotton wool.
- Sharps bin.

- Medication for injecting.

Procedures

Subcutaneous injections

The S/C route is used for a slow absorption of medication and is ideal for drugs such as insulin.

- Introduce yourself, confirm the identity of the patient, explain the procedure, and obtain verbal consent.
- Wash your hands and put on a pair of gloves.
- ▸ Always check you have the correct drug, correct dose, and that it is within date before injecting it.
- Draw up the medication using a 21-gauge needle and have a colleague double check the medication, dose, and expiry date.
- Expel any air in the syringe and replace with a 25-gauge needle.
- Clean the injection site with the alcohol swab.
- Pinch a fold of skin so as to lift the adipose tissue away from the underlying muscle.
- Insert the needle horizontally into the fold and draw back to ensure you are not in a vein.
- Now inject the medication.
- Withdraw the needle and apply the cotton wool to the site to mop up any bleeding.
- Suitable S/C sites include the forearm, triceps area, and abdomen.

Intramuscular injections

IM injections are administered through the epidermis, dermis, and S/C tissue into the muscle. They provide rapid systemic action and allow relatively large doses to be absorbed.

Suitable IM sites include the deltoid muscle, dorsogluteal site, ventrogluteal site, lateral thigh/vastus lateralis, quadriceps muscle, and the rectus femoris muscle.

Remember to avoid sites of inflammation, swelling, infection, or skin lesions.

- Introduce yourself, confirm the identity of the patient, explain the procedure, and obtain verbal consent.
- Wash your hands and put on a pair of gloves.
- ▸ Always check you have the correct drug, correct dose and that it is within date before injecting it.
- Draw up the medication using a 21-gauge needle and have a colleague double check the medication, dose, and expiry date.
- Expel any air in the syringe and replace with a 25-gauge needle.
- Inspect the proposed site for adequate muscle mass.
- Clean the injection site with the alcohol swab.
- IM injections should be given at a 90° angle to ensure the needle reaches the muscle, and to ↑ pain.
- A good way to ensure accuracy and avoid a needle-stick injury is to rest the heel of the palm on the thumb of the non-dominant hand.
- Pull the skin downwards or to one side at the intended site.
- Hold the syringe between the thumb and forefinger and insert the needle at full depth.
- Draw back on the syringe to ensure the needle is not in a vein.
- Slowly inject the medication.
- After needle insertion and injection allow 10 seconds before removing the needle to facilitate diffusion of the medication into the muscle.
- Withdraw the needle and wipe the area clean with cotton wool.

Intradermal injections

The ID route provides a local, rather than systemic effect and is used primarily for diagnostic purposes such as allergy or tuberculin testing.

This involves the same preliminary work-up above except a 25-gauge needle is inserted at a 10-15° angle, bevel up, just under the

epidermis.

Up to 0.5ml is injected until a wheal appears on the skin surface-just as you would when creating a bleb of local anaesthetic.

We thank Lyn Dean for her assistance with this topic.

Venepuncture

Two methods exist, the ‘traditional’ needle-and-syringe and the newer method of collecting blood directly into the tubes by Vacutainer®.

Equipment

- Gloves
- Sticky tape
- Alcohol swabs
- Gauze/cotton wool
- Tourniquet
- A needle (try 12G first), a syringe and blood collection bottle or…;
- A Vacutainer® tube, holder and blood collection needle.

Procedure

Using a needle and syringe

- Introduce yourself, confirm patient's identity, explain the procedure, and obtain verbal consent.
- The patient should be lying or sitting comfortably with the arm from which blood is to be taken resting on a pillow.
- Select a vein site—usually the antecubital fossa (see Fig. 17.2).
- Apply the tourniquet proximal to the puncture site and recheck the vein.
- Put on gloves and ask the patient to clench their fist a few times.
- Cleanse the area with an alcohol swab in spirals, inside to out.*
- Attach the needle to a syringe and unsheathe it.
- Use the thumb of your non-dominant hand to gently anchor the skin just below the puncture site.
- Warn the patient to expect a ‘sharp scratch’ and to not move their arm.
- Insert the needle firmly through the skin, bevel upwards, at an angle of 20-40® over the vein.
- With experience, you will feel a slight ‘give’ as the vein is entered and blood will visibly enter the hub (plastic portion) of the needle (‘flashback’).
- Carefully holding the needle in position, pull back on the plunger. There are several ways of doing this, the authors favour holding the needle and syringe in the non-dominant hand, once in place, and pulling back with the dominant hand.
- When enough blood is taken, release the tourniquet *before* removing the needle from the vein.
- Apply a clean cotton wool ball or folded gauze to the puncture site as the needle is withdrawn. Pressure should be applied for ~1min. (ask the patient to do this for you, if they are able).
- Apply a plaster to the site, thank the patient.
- Vacuum blood tubes are filled by puncturing the rubber top with the needle and allowing the blood to enter the tube.
- Remember to label the tubes correctly—ideally at the patient's bedside and dispose of sharps in a sharps bin.

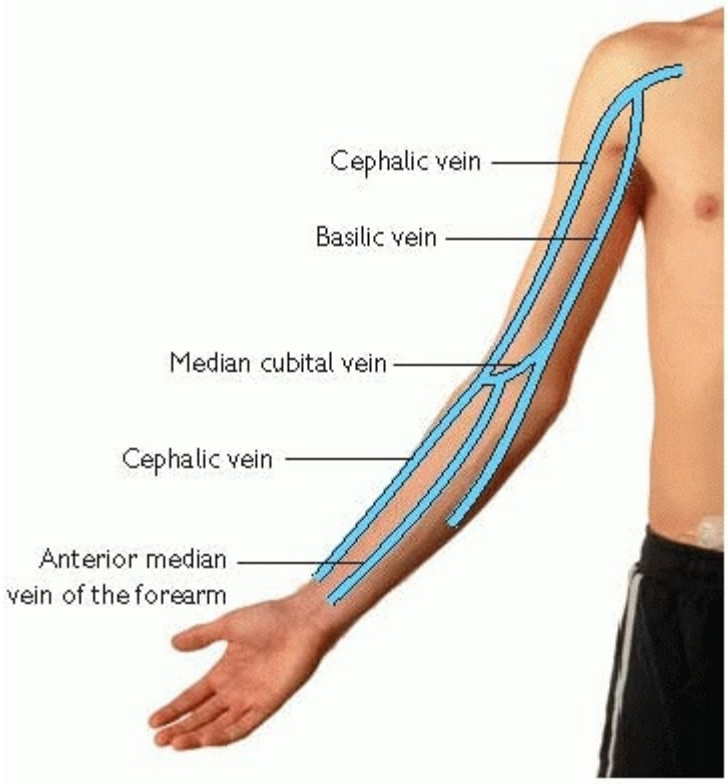


Fig. 17.2 Representation of peripheral veins of the upper limb.

* There is no solid evidence for benefit in using alcohol wipes unless there is *visible* dirt at the venepunture site. However, their use is 'policy' in most health care trusts and should be used accordingly.

Using a Vacutainer® system

Much of the procedure is the same…

- Vacutainer® needles are double-ended, one 'standard' needle and one needle covered by a rubber sleeve.
- Attach a Vacutainer® holder over the covered needle (see Fig. 17.3).
- The needle is inserted into the vein as above but no 'flashback' will be visible.
- Once in place, the Vacutainer® tubes are attached to the needle directly by pushing them onto the covered needle using the tube holder.
- Allow enough blood to enter tube (some tubes must be filledcheck local laboratory guidance).
- Multiple tubes may be filled by removing and replacing tubes whilst carefully holding the needle in position.

Inappropriate sites for venepunture

- Oedematous areas.
- Cellulitic areas.
- Haematomas.
- Phlebitis or thrombophlebitis.
- Scarred areas.
- Arm in which there is a transfusion or infusion.
- Arm on the side of previous mastectomy.
- Arms with AV fistulae or vascular grafts.

Hints

- If extraction of blood with Vacutainers is proving difficult, it may be easier to switch to the needle-and-syringe technique as this allows you more control over the flow of blood.
- Venepuncture can be performed at *any* peripheral vein-difficult to bleed patients in hospital often have blood taken from the back of their hands, feet or legs.
- In difficult to reach places, it is often easier to use a 'butterfly' needle. This is a smaller needle attached to a length of tubing which can be used with either technique. It allows for greater control of the needle.

Fig. 17.3 Vacutainer® blood collection system, ready for use.

Box 17.1 Taking blood from a central venous catheter

Theory

Central lines should only be used for taking blood if it is not possible to obtain a sample via the peripheral route. Do not risk catheter sepsis or a clotted line unless there are no alternatives!

Equipment

- 3 × 10ml syringes.
- 0.9% isotonic or heparinized saline.
- Chlorhexidine spray or iodine.
- Gauze.
- Sterile gloves.
- Drape.

Procedure

- Introduce yourself, confirm the identity of the patient, explain the procedure, and obtain verbal consent.
- Stop any infusions for at least one minute before sampling.
- Place the patient in a supine position.
- Ask the patient to turn their head away from the line site during the procedure.
- Drape the site in case of splash and put on a pair of sterile gloves.
- Spray the line chosen to withdraw blood with chlorhexidine solution.
- You may alternatively use a gauze dipped in iodine solution.
- Clamp the line before removing the cap.
- Connect a 10ml syringe to the line before unclamping.

- Withdraw 5-10ml of blood, clamp the line and remove the syringe.
- Discard the blood.
- Connect a new 10ml syringe to the line, unclamp it and withdraw a futher 10ml of blood.
- Clamp the line, remove the syringe (keep this sample).
- Fill a further syring with saline and attach to the line.
- Unclamp the line, instill the saline, and clamp the line again.
- Remove the syringe and replace the cap.

Peripheral IV cannulation

Theory

Peripheral IV cannulation is a 'generic' skill that the medical student should learn early. A thin tube 'line' is inserted into a vein allowing easy venous access which is used in many situations, including the administration of fluids and iv medication.

Equipment

- Gloves.
- Alcohol swabs.
- Tourniquet.
- Saline for injection.
- 5ml syringe.
- Sticky tape.
- Gauze/cotton wool.
- A cannula of appropriate size.

Procedure

- Introduce yourself, confirm patient's identity, explain the procedure, and obtain verbal consent.
- The patient should be lying or sitting comfortably with the arm in which the cannula is to be inserted resting on a pillow.
- Apply the tourniquet to the arm and identify a suitable vein (often those that can be *felt* are more reliable than those that are *seen)*. The vein should be superficial and have a straight course for a few centimeters.
- Put on the gloves and clean the overlying skin with the alcohol swab.
- Remove the cannula from its packaging.
- Ensure that the cannula is functioning properly by slightly withdrawing the needle and replacing it. Fold down the 'wings' and open and close the port on the top.
- Warn the patient to expect a 'sharp scratch' and to not move their arm.
- Insert the cannula firmly through the skin, bevel upwards, at an angle of 20-40° over the vein.
- With experience, you will feel a slight 'give' as the vein is entered and blood will visibly enter the hub (plastic portion) of the cannula ('flashback').
- Once the flashback is seen, hold the needle in place with one hand and slide the cannula off the needle—into the vein—with the other. Once the cannula is fully inserted, the needle should be sitting just within it, preventing blood from spilling.
- Release the tourniquet.
- Press over the vein at the tip of the cannula, remove the needle, and dispose of it safely in a sharps bin.
- Put the cap on the cannula and fix it in place with the sticky dressing.
- Draw up the saline into the syringe and 'flush' it through the cannula using the port on the top. Watch the vein—if the cannula is misplaced, the saline will enter the subcutaneous tissues causing swelling.
- ▸ Don't forget to do this—it confirms that the cannula is working and clears it of blood which would form a clot.

Box 17.2 Sizing cannulae

Like needles, cannulae are colour-coded according to size. Each is given a ‘gauge’ which has an inverse correlation to the external diameter.

The standard size cannulae is ‘green’ or 18G but for most hospital patients, a ‘pink’ or 20G cannula will suffice. Even blue cannulae are adequate in most circumstances unless fast flows of fluid are required.

Gauge	External diameter (mm)	Length (mm)	Approximate maximum flow rate (mL/min)	Colour
14G	2.1	45	290	Orange
16G	1.7	45	172	Grey
18G	1.3	45	76	Green
20G	1.0	33	54	Pink
22G	0.8	25	25	Blue

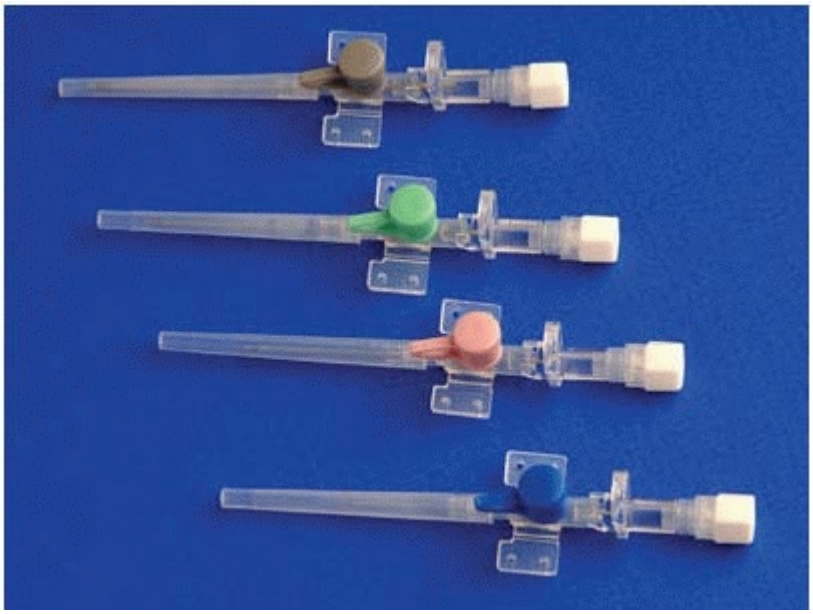


Fig. 17.4 A selection of standard IV cannulae.

Hints

- Try to avoid the antecubital fossa. Although this is often the easiest place to see and feel a vein, cannulae at that site can become kinked and blocked whilst causing pain for the patient on bending the arm.
- Avoid an arm with a fistula or AV shunt.
- Bring a selection of different sized cannulae to the bedside allowing you to choose a smaller gauge if you experience problems.

- Don't be afraid to ask for assistance from nursing or auxiliary staff if the patient is likely to move their arm during the procedure.

Setting up an infusion

Theory

Fluid therapy is one of the basic responsibilities of junior doctors and one of the core skills for nurses. Whilst it is usually the job of the nursing staff to set up the drip, junior medical staff should nevertheless be competent at this technique.

Equipment

- Gloves.
- An appropriate fluid bag.
- Giving set.
- Drip stand.

Procedure

- IV infusions require IV access—see p.564. Check the fluid in the bag and fluid prescription chart.
- Ask a colleague to double-check the prescription and the fluid and sign their name on the chart.
- Open the fluid bag and giving set, which come in sterile packaging.
- Unwind the giving set and close the adjustable valve.
- Remove the sterile cover from the bag outlet and from the sharp end of the giving set (see Fig. 17.5).
- Using quite a lot of force, push the giving set end into the bag outlet.
- Invert the bag, hang on a stand.
- Squeeze the drip chamber to half fill it with fluid.
- Partially open the valve to allow the drip to run, and watch fluid run through to the end (it might be best to hold the free end over a sink in case of spills).
- If bubbles appear, try tapping or flicking the tube.
- Once the giving set is filled with liquid, connect it to the cannula.
- Adjust the valve and watch the drips in the chamber.
- Adjust the drip rate according to the prescription (see Table 17.1).

We thank Lyn Dean for her assistance with this topic.

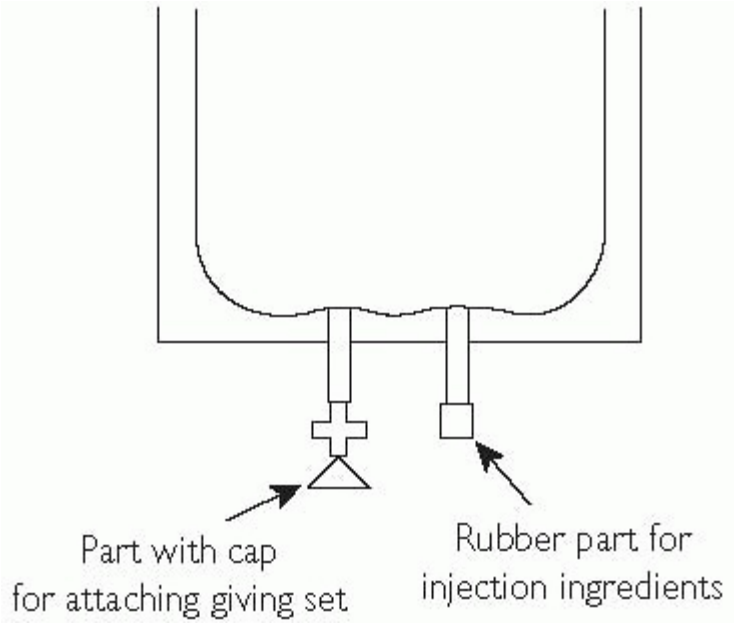


Fig. 17.5 Diagrammatic representation of the base of a fluid bag and the port in which the giving set should be inserted.

Box 17.3 Drip rate

Most infusions tend to be given with electronic devices which pump the fluid in at the prescribed rate. However, it is still important that health care professionals are able to set up a drip at the correct flow rate manually.

Using a standard giving set, clear fluids will form drips of about 0.05ml—that is, there will be approximately 20 drips/ml. You can then calculate the number of drips per minute for a given infusion rate as below.

Table 17.1 Infusion and drip rates

Prescription Number of hours per litre of fluid	Infusion rate (ml/hour)	Infusion rate (ml/minute)	Drip rate (drips/minute)
1	1000	16	320
2	500	8	160
4	50	4	80
6	166	3	60
8	125	2	40

10	100	1.6	32
12	83	1.4	28
24	42	0.7	14

External jugular vein cannulation

Theory

The external jugular vein lies superficially in the neck, running down from the angle of the jaw, across the sternocleidomastoid muscle before passing deep to drain into the subclavian vein. It is sometimes used to provide essential venous access in cardiac arrest and other emergency situations where no peripheral access is obtainable.

See Fig. 17.6 for the surface anatomy of the external jugular vein.

Equipment

- Antiseptic solution/antiseptic wipe.
- 2 × 5ml syringes.
- 1 25-gauge orange needle.
- 1 21-gauge green needle.
- 1% lidocaine.
- 14- or 16-gauge cannula.
- 0.9% saline flush.
- Dressing.
- Gloves.
- Sharps bin.

Procedure

- Introduce yourself, confirm the identity of the patient, explain the procedure and obtain verbal consent if possible.
- Wash your hands and don a pair of gloves.
- Tilt the patient to 10-15° head-down to facilitate venous filling.
- Once the external jugular vein is visible, clean the area with antiseptic solution.
- Attach a 25-gauge (orange) needle to a 5ml syringe and make a S/C bleb of 1-2ml of 1% lidocaine and infiltrate around the insertion site.
  Be careful not insert any anaesthetic into the vein.
- Position yourself at the head of the bed.
- Remove the cap from the cannula and attach a clean 5ml syringe.
- Turn the patient's head away from the side of insertion.
- Cannulate using the same technique as for peripheral venous access.

- ▸ Remember to aspirate as you advance the cannula. Correct placement will be confirmed once you are able to aspirate venous blood.
- Fix the cannula in place using a suitable dressing.
- Flush the cannula with 5ml of 0.9% saline solution.
- Dispose of all sharps in sharps bin, wash hands and help the patient to a comfortable position.
- Document details of procedure in notes.

Hints

In an emergency situation, you may forgo the anaesthetic as venous access may be needed swiftly.

Central venous cannulation

Theory

Central venous access is the placement of a catheter in a vein which leads directly to the heart. There are a number of central veins including the internal jugular, external jugular, subclavian, femoral, and antecubital.

For each of these, the basic equipment and preparation are the same. Central venous cannulation is performed for vascular access, TPN, infusion of irritant, vasoactive, and inotropic drugs, measurement of CVP, cardiac catheterization, pulmonary artery catheterization, transvenous cardiac pacing, and haemodialysis/plasmaphoresis.

Single and multi-lumen catheters are available and the type to be used should be decided prior to insertion depending on the anticipated use (e.g. concurrent CVP monitoring and multiple drug infusion).

Equipment

- Trolley.
- Sterile pack including sterile drapes.
- Sterile gown and gloves.
- Suture material e.g.-2/0 silk on a curved needle.
- Antiseptic solution.
- Local anaesthetic-approx 5ml of 1% lidocaine.
- Seldinger central venous line kit.
- 21-gauge green and 25-gauge orange needles.
- Saline or heparinized saline to prime and flush the line prior to and post insertion.
- Sterile dressing.
- Ultrasound machine.

Procedure—internal jugular vein

- Introduce yourself, confirm the identity of the patient, explain the procedure, and obtain verbal consent.
- Remember, this can be a potentially frightening procedure. Explanations and reassurance must be given before and during the procedure.
- Put on a sterile gown and gloves.
- Unwrap all equipment.
- Check that the wire passes through the needle freely. Attach 3-way taps to all ports of the catheter. Flush all the lumens with heparinized saline.
- Place the patient in a supine position, at least 15° head down.
    - This is usually quite easy on a tilting bed and is performed to distend the neck veins and reduce the risk of air embolism.
- Turn the head away from the venepuncture site.
- Cleanse the skin with antiseptic solution and drape the area.

- Stand at the head of the bed.
- Locate the cricoid cartilage and palpate the carotid artery lateral to it.
- The site for insertion is approximately 1/3 of the way up the sternocleidomastoid, just between its 2 heads.
- Use local anaesthetic to numb the venepuncture site once located.

- Infiltrate the skin and deeper tissues with a smaller orange needle and then replace with a green needle.
- Introduce the large calibre introducer needle, attached to an empty syringe, into the centre of a triangle formed by the 2 lower heads of the sternocleidomastoid muscle and clavicle.
- Keep your finger on the carotid artery and ensure the needle enters the skin *lateral* to the artery.
- Direct the needle caudally at an angle of 30-40° to the skin, towards the ipsilateral nipple. The vein is usually within 2-3cm of the skin.
- Aspirate as the needle is advanced. Once you see blood, cannulate the vein using the Seldinger technique …
- Remove the syringe, occlude the needle lumen with a thumb.
- Straighten the J tip of the spring guidewire, and advance into the vessel through the needle.
- Holding the spring-wire in place, remove the needle whilst maintaining a firm grip on the wire at all times.
- Enlarge the cutaneous puncture site with the cutting edge of the scalpel positioned away from the spring-wire guide.
- Use the dilator provided to enlarge the site and thread the tip of the catheter into the vessel using the spring-wire guide.
- Grasp the catheter near the skin and using a slight twisting motion advance into the vein.
- Make sure that before you push the catheter forward the wire is visible at the proximal end. *Hold the wire at all times*, to prevent it being lost inside the patient.
- Hold the catheter and remove the spring-wire guide.
- Check lumen placement by aspirating through the pigtails and flush with saline.
- Lock off the 3-way taps. The patient can now be levelled.
- Secure the catheter in place with a suture and cover with an adhesive sterile dressing. (Do not forget to anaesthetize suture sites as well).
- Request a CXR to verify correct catheter position and to exclude a pneumothorax.
  - Catheters have a radio-opaque strip for this purpose.
  - The catheter tip should lie in the SVC at the level of the carina.
- Dispose of your sharps and clear away the trolley.
- Document the details of the procedure in the notes.

Complications of internal jugular vein cannulation

- Pneumothorax.
- Haemothorax.
- Chylothorax.
- Air embolism.
- Arrhythmais.
- Carotid artery puncture.
- Infection.
- Thrombosis of vessel.
- Neural injury.
- Cardiac tamponade.
- AV fistula.
- Patient discomfort.

Guidelines produced by the National Institute for Clinical Excellence (NICE) in September 2002 encourage the routine use of 2-D (B-mode) ultrasound guidance for CVC insertion into the Internal jugular vein in adults and children in elective and emergency situations. There is however, limited evidence supporting ultrasound use for subclavian and femoral vein cannulation. Ultrasonography allows direct visualization of the anatomy before and during cannulation. Portable ultrasound machines can be used at the bedside. This is not discussed

here.

Procedure: femoral vein

The femoral vein lies medial to the femoral artery immediately beneath the inguinal ligament. It is commonly used in an ICU setting for placement of a double-lumen haemofiltration line and when central access is unfeasible by other routes. This is impractical for mobile patients and raises concerns regarding the sterility of the groin area.

- Introduce yourself, confirm the identity of the patient, explain the procedure, and obtain consent.
- Extend the patient's leg and abduct slightly at the hip.
- Adopt full asepsis.
- Locate the femoral artery, keep a finger on the artery, and introduce a needle attached to a 10ml syringe at 45°, 1.5cm medial to the femoral artery pulsation, 2cm below the inguinal ligament.
- Slowly advance the needle cephalad and posteriorly whilst gently withdrawing the plunger.
- When a free flow of blood appears, follow the Seldinger approach as detailed for the internal jugular vein.
- Ultrasound can be used to identify the vessels and ensure that the vein is punctured near the inguinal ligament where the artery and vein lie side by side.

Procedure: subclavian vein

The subclavian vein is preferred for central venous access if the patient has a cervical spine injury and is best for long-term parenteral nutrition, pacing wires, or Hickman lines. It is, however, associated with a higher incidence of incorrect line placement than internal jugular cannulation. Due to the local anatomy, pressure cannot be exerted on the subclavian artery if it is accidentally punctured.

The subclavian vein is a continuation of the axillary vein and runs from the apex of the axilla, behind the posterior border of the clavicle and across the first rib to join the internal jugular vein, forming the brachiocephalic vein behind the sternoclavicular joint.

- Introduce yourself, confirm the identity of the patient, explain the procedure, and obtain verbal consent.
- Place the patient in a supine position, head-down.
- Turn the head to the contralateral side.
- Adopt full asepsis.
- Introduce a needle attached to a 10ml syringe, 1cm below the junction of the medial 1/3 and outer 2/3 of the clavicle.
- Direct the needle medially, slightly cephalad, and posteriorly behind the clavicle toward the suprasternal notch.
- Slowly advance the needle while gently withdrawing the plunger.
- When a free flow of blood appears, follow the Seldinger approach as detailed earlier.
- The catheter tip should lie in the SVC above the pericardial reflection.
- Perform a CXR to confirm the position and exclude a pneumothorax.
- As before, ultrasound can be used to guide puncture of the vein using a more lateral approach.

Removing internal jugular venous catheters

- Remove any dressing and suture material.
- Ensure that all drugs and infusions have been stopped.
- Lie the patient down to reduce the risk of air embolism.
- Ask the patient to take a deep breath and fully exhale.
- Remove the line smoothly with a steady pull, while the patient is breath holding and apply firm pressure to the puncture site for at least 5 minutes to stop bleeding.
- Sit the patient up.
- If infection is suspected, send the tip of the line in a dry specimen pot for culture.

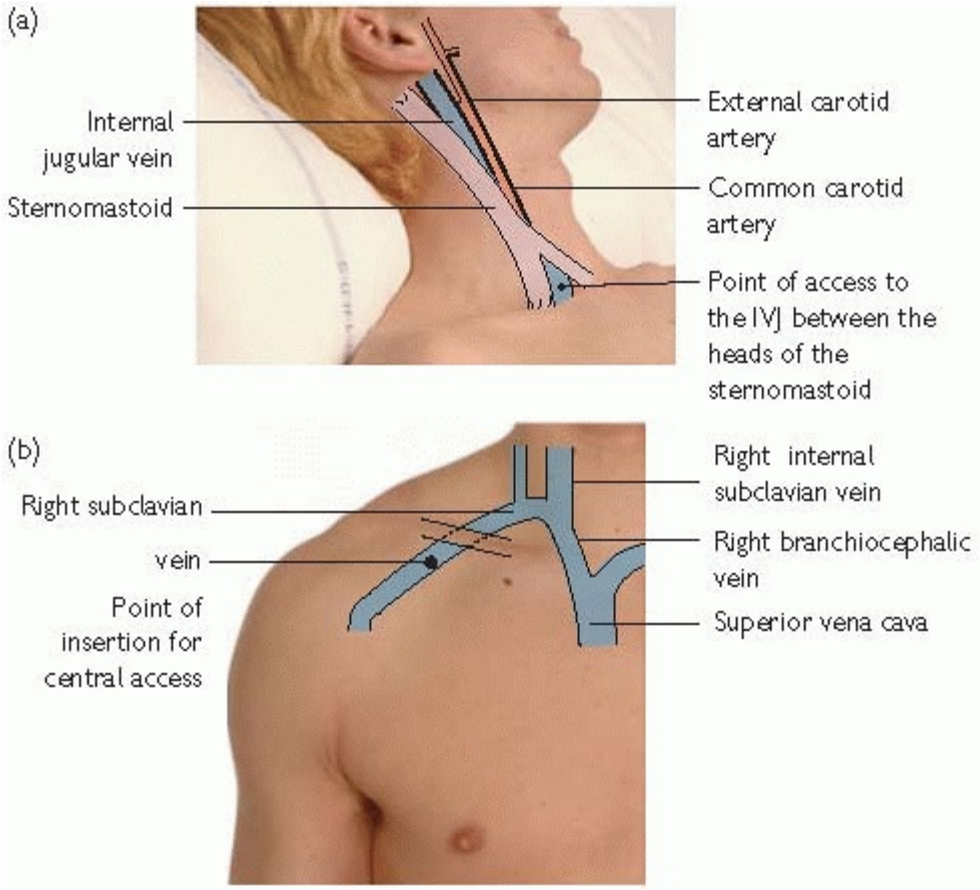


Fig. 17.6 The surface anatomy of the internal jugular vein (a) and subclavian vein (b).

Blood pressure measurement

Theory

BP is measured with a sphygmomanometer (sphyg)—usually at the brachial artery.

Machines, operated by nurses or health care assistants usually measure BP these days but these are not fool-proof and a good working knowledge of the 'manual' method of BP measuring is still essential.

A cuff is applied to the upper arm and inflated so as to cut off the arterial supply. The pressure is released slowly and a stethoscope used to listen for the blood flow. When the pressure in the cuff equals the systolic blood pressure, blood will audibly pulse through the artery. When the cuff pressure falls below the diastolic blood pressure, the blood will flow continuously and the sound of intermittent blood flow will disappear.

Equipment

- A (functioning) sphygmomanometer with …
- An appropriately sized cuff (see Table 17.2).
- Stethoscope.

Procedure

- Introduce yourself, explain the procedure, and obtain verbal consent.
- Check the sphyg is working and the dial reads '0'.
- The patient should be sitting, relaxed for 5 minutes beforehand.
- Apply the cuff to the upper arm with the air bladder anteriorly (over the brachial artery).
- Using your left arm, support the patient's arm so that it is held horizontally at the level of the mid-sternum.
- Close the valve (may be a screw or lever), monitor the patient's radial artery, and inflate the cuff until the radial pulse is no longer

palpable.

- Listen over the brachial artery at the antecubital fossa—using the diaphragm or the bell of the stethoscope—whilst deflating the cuff at a rate of 2-3mmHg/sec.
- Note the point at which the pulsation is audible (Korotkoff* phase I—the systolic BP)…
- And the point at which the sounds disappear (Korotkoff phase V—the diastolic BP).
- Record the BP as 'systolic/diastolic' to the nearest 2mmHg.

▸ *Hints*

- In some normal people, the sounds may not disappear completely. In this case, a distinct muffling of the noise (Korotkoff phase IV) should be used to indicate the diastolic BP.
- BP recording may be particularly difficult in a noisy hospital ward at the time of an emergency (which is when doctors are most often asked to record the BP) or when the BP is very low. In this case, a rough estimation of the systolic BP may be made by feeling for the return of the radial pulse as the cuff is deflated.

Table 17.2 BHS guidelines for choice of BP cuff

Indication	Width (cm)	Length (cm)	Bladder dimensions (cm)	Arm circum-ference (cm)
Small adult/child	10-12	18-24	12×18	<23
Standard adult	12-13	23-35	12×26	<33
Large adult	12-16	35-40	12×40	<50
Adult thigh cuff	20	42		<52

Recording a 12-lead ECG

Theory

The ECG is a recording of the electrical activity of the heart. Electrodes are placed on the limbs and chest for a '12-lead' recording. The term '12-lead' relates to the number of directions that the electrical activity is recorded from and is *not* the number of electrical wires attached to the patient!

Equipment

- An ECG machine capable of recording 12 leads.
- 10 ECG leads (4 limb leads, 6 chest leads)-should be attached to machine.
- Conducting sticky pads (‘ECG stickers’).

Procedure

- Introduce yourself, confirm the identity of the patient, explain the procedure, and obtain verbal consent.
- Position the patient so that they are sitting or lying comfortably with their upper body, wrists, and ankles exposed.
- Each electrode is attached by clipping it to the sticky pads and sticking them to the patient's skin.
- The leads are usually labelled. The limb leads are often colour-coded.
  - Right arm—red.
  - Left arm—yellow.
  - Right leg—green.
  - Left leg—black.
- The arm leads are of medium length and should be attached to a hairless part of the patient's wrists.
- The leg leads and longest are should be attached to the patient's ankles (the hairless part just superior to the lateral malleolus is ideal).
- Position the chest leads as below (see Fig. 17.7).
  - V1—4th intercostal space at the right sternal border.
  - V2—4th intercostal space at the left sternal border.
  - V3—midway between V2 and V4.
  - V4—5th intercostal space in the mid-clavicular line on the left.
  - V5—left anterior axillary line, level with V4.
  - V6—left mid-axillary line, level with V4.
- Turn on the ECG machine. These are usually self-explanatory require just 1 button to be pressed—marked ‘analyse’ or ‘record’.
- Check the calibration and paper speed:
  - 1mV should cause a vertical deflection of 10mm.
  - Paper speed should be 25mm/s (5 large squares per second).
- Ensure the patient's name, DOB, as well as the date and time of the recording are clearly recorded on the trace.
- Remove the leads, discard the sticky electrode pads.

▸ *Hints*

- Encourage the patient to relax as muscle contraction will cause interference.
- Ensure that you cleanse the area gently with an alcohol swab before attaching an electrode to ensure a good connection.
- The AC mains electricity may cause interference. If this is the case, try turning off the fluorescent lights.

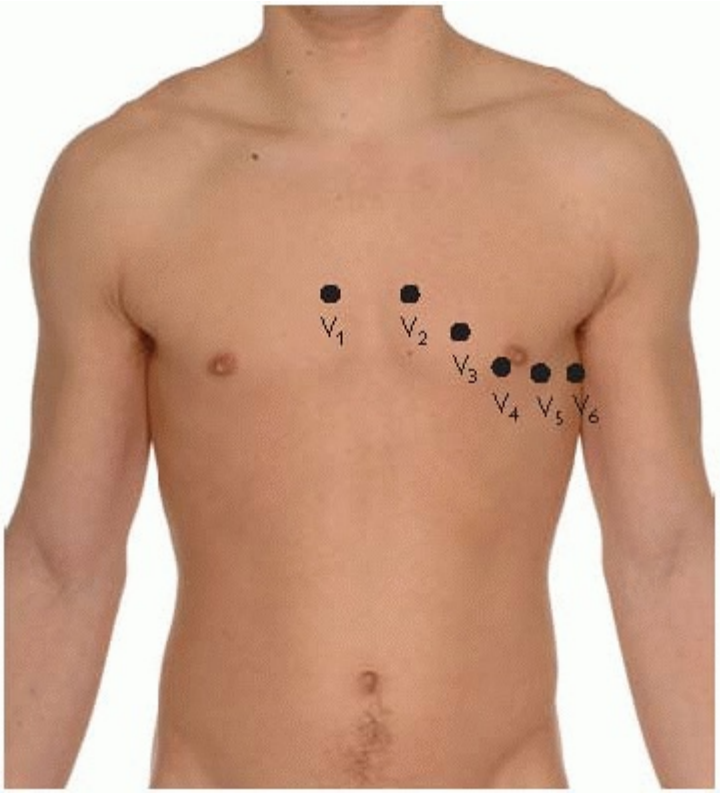


Fig. 17.7 Correct positioning of the electrodes for a standard 12-lead ECG.

Arterial blood gas sampling

Theory

An arterial sample is obtained to assess pH, PO_2, PCO_2, HCO^-_3 and base excess/deficit. Sometimes also used for rapid assessment of electrolytes.

Equipment

- ABG kit (usually contains heparin-filled syringe, needle and vented cap).
- Gauze or cotton ball.
- Tape.
- Sterile gloves.

Procedure

- Wash hands and put on gloves.
- Verify patient identity, explain procedure, and obtain verbal consent. Be sure to warn the patient of potential pain and ask them to keep as still as possible.
- Note patient's temperature and oxygen support.
- Chose site for arterial puncture.

Radial

(No adjacent nerves or vessels so is the most commonly used site.)

- Assess for adequate ulnar arterial circulation by obstructing the radial artery with finger-tip pressure.
- Ask the patient to make a tight fist, expelling blood from hand, maintaining pressure on radial artery.

- Ask them to open their hand and watch for flushing of the palm. This indicates adequate perfusion.
- Take the blood gas syringe, ensuring that the heparin has coated the inside by withdrawing and advancing plunger.
- Attach the needle and expel the excess heparin.
- Position the wrist in extension.
- Palpate the radial arterial pulse along its length using the middle and index fingers.
- Clean the skin.
- Having chosen a suitable spot, insert the needle with the bevel facing towards the direction of blood-flow, using an appropriate angle.
- Advance the needle until arterial pressure fills the syringe.
- Obtain a sample of 1-3ml and withdraw the needle.
- Apply pressure to the puncture site using gauze or cotton ball until bleeding has stopped (minimum 2 minutes).
- Remove and discard the needle with care and place a vented cap on the syringe. Holding vertically, expel any air through the vent.
- Mix sample gently and take to a blood gas analyser.

Femoral

- Position the patient with the hip extended and slightly internally rotated.
- Note that the femoral nerve is just lateral to the artery so maintain a medial approach.

- Procedure as above but use a 21-gauge needle, aiming at the pulsation positioned between your index and middle finger.

Brachial

- Position the elbow in extension.
- Watch for adjacent nerves (see below).

Appropriate angles for needle insertion.

- Radial artery—45°.
- Brachial artery—60°.
- Femoral artery—90°.

Hints

- The key to success is carefully lining up the needle over a palpable pulsation—take your time!
- If there is no flash-back, withdraw the needle slightly, change the angle and advance. Note that most pain is from puncturing the skin so do not remove needle fully when repositioning.
- If there will be some delay in analyzing the sample, store the bloodfilled syringe on ice.
- Sources of blood gas result errors:
  - Air in the sample.
  - Delay in analyzing sample or delay in icing.
  - Excess heparin in the syringe.
  - Accidentally obtaining a venous or mixed arterio-venous sample.
  - Alterations in temperature.

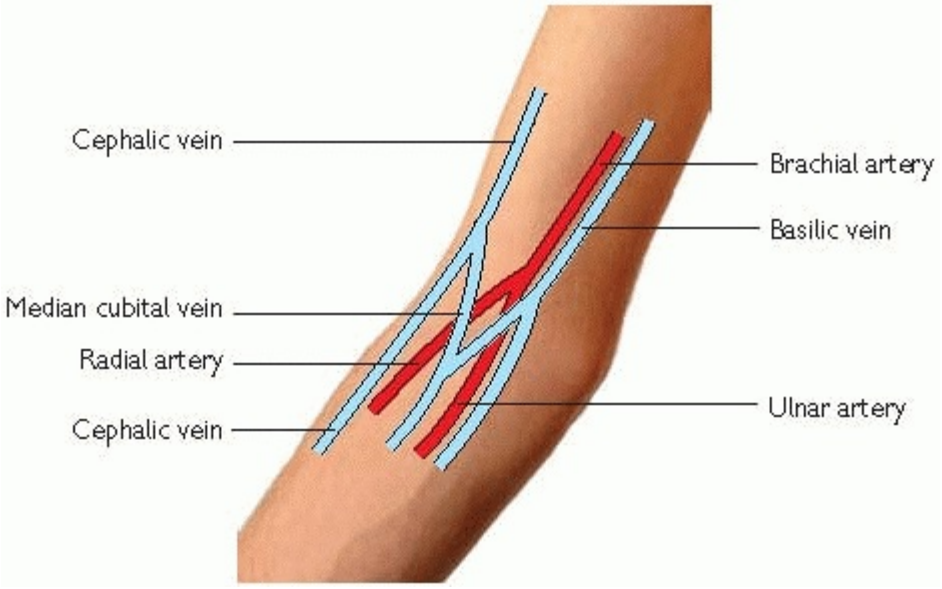


Fig. 17.8 Position of the brachial artery and surrounding structures at the antecubital fossa. Right arm is pictured.

Peak flow measurement

Theory

Full name 'peak expiratory flow rate' (PEFR) is a measure of the maximum *speed* of expiration. Expressed in 'litres/minute', is a simple and easy to administer test which is a useful indicator of airway calibre and may be performed before and after the administration of a bronchodilator to assess reversible airway obstruction.

Normal values are based on gender, age, and height.

Equipment

- A peak flow meter (see Fig. 17.9).
- A clean disposable mouth-piece.

Procedure

- Introduce yourself, explain the procedure, and obtain verbal consent.
- The patient should be standing or sitting upright.
- Ensure that the meter is set to '0'.
- Ask the patient to take a deep breath in, hold the mouthpiece in the mouth, and seal their lips tightly around it.
- The patient should blow out as hard and as fast as possible.
- ▸ The PEFR needs a *hard* and *short* maximal blow out. The patient does *not* have to blow out completely.
- Make a note of the reading achieved.
- The procedure should be repeated and the best of 3 efforts recorded.
- The result should be compared to the 'normal' value on the Nunn-Greggs Nomogram (see *OHCM6*, p.169).
- If the patient is to keep a record, be sure to explain how to record the readings appropriately. (Sometimes a 2-week diary is kept by the patient to assess for diurnal variation).

▸ *Hints*

- If the patient is having difficulty performing correctly, a brief demonstration often proves very useful.

- If a patient has very variable flow measurements, repeat your demonstration and go on asking for flows until 3 consistent readings have been recorded.

Fig. 17.9 A standard peak flow meter using the EU scale, adopted in 2005.

Inhaler technique

A person new to respiratory medicine may be surprised by the sheer number of different inhaler devices on the market. Each has its advantages and disadvantages and a different set of drugs that it can deliver.

On these pages, we outline the inhaler devices currently available and the instructions for use written as you would explain them to a patient. The authors suggest that students and respiratory nurse specialists become familiar with the different devices by asking your ward pharmacist if you can see placebo versions.

Devices are constantly changing. At the time of writing, the following inhalers are 'out of fashion' and are not described here: Clickhaler, Spinhaler, Aerohaler, Diskhaler, Rotahaler, Foradil inhaler, Pulvinal. We do, however, include the 'Handihaler' which is a relative newcomer.

Metered dose inhaler

This was the first device and is the one people think of as a 'typical' inhaler. It is small, cheap, and has many different drugs and doses. However, there is no dose counter and requires a good deal of coordination to use correctly—making it unsuitable for the very young, elderly, or those with arthritis or other ailments affecting the hands.

Instructions for use

- Take one dose at a time.
- Remove the cap and shake the inhaler several times.
- Sit upright, hold head up and breathe out.
- Place inhaler in mouth and seal lips around mouthpiece.
- Breath in, press the canister down to release the drug and continue to take a deep breath in. (The canister should be pressed *just after* the start of inhalation, not before).
- Remove inhaler and hold breath for as long as possible up to 10 seconds.
- Recover before taking the next dose, replace cap.

Fig. 17.10 A metered dose inhaler (MDI)—pictured is a Salbutamol inhaler.

Autohaler

This is one of the 'breath-actuated' inhalers, releasing a dose of the drug when a breath is taken. This eliminates the need for hand coordination and can reassure patients that a dose has been successfully administered.

Some people, however, may still find the priming lever hard to use or may have difficultly remembering to prime the device for each dose. Also, the puff and click during inhalation can be distracting.

Instructions for use

- Remove cap and shake inhaler several times.
- Prime the device—push the lever right up, keeping the inhaler upright.
- Sit upright, hold head up and breathe out.
- Seal lips around mouthpiece.
- Inhale *slowly* and *deeply*—don't stop when the inhaler clicks and continue taking a really deep breath.
- Remove inhaler and hold breath for as long as possible up to 10 seconds.
- Push lever down and replace cap.
- Recover before taking the next dose.
- (Remember to advise the patient, they won't feel the spray hitting the back of the throat—although there may be a slight taste disturbance).

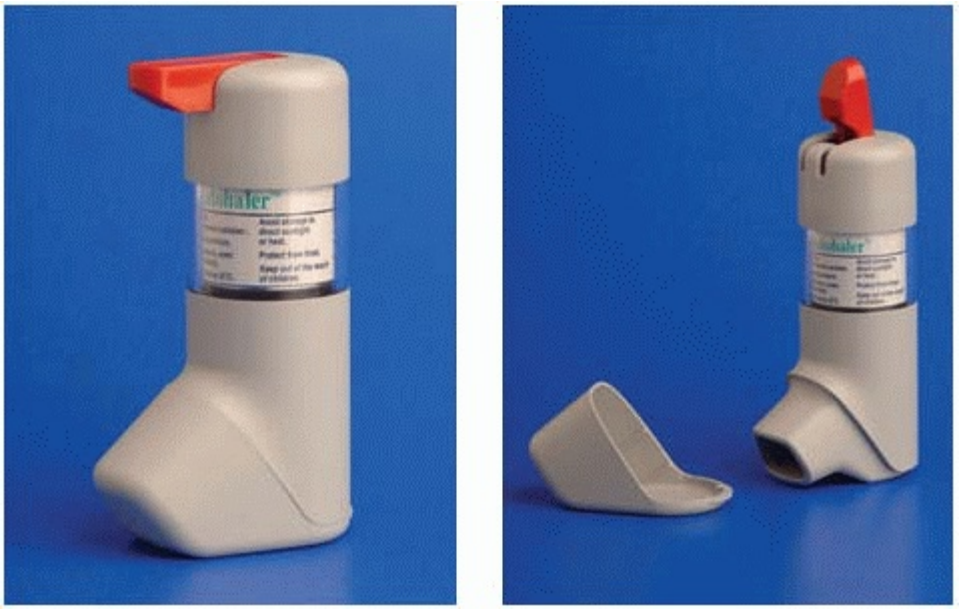


Fig. 17.11 Autohaler. Note the lever on the top. Remember that the inhaler must be primed for each dose.

▸ *Hints*

- Patients unable to push the lever up by hand can sometimes use the edge of a table to push it against.
- Patients should breathe in steadily, not as fast as possible.

Easibreathe

This is a breath-actuated inhaler, like the Autohaler on the previous page. Again, there is no need for hand coordination and no lever on top of the device. Instead, the inhaler is primed by opening the cap.

Some people will, however, still find it difficult to prime the device—and may forget that the cap must be closed and opened between each successive dose.

Instructions for use

- Shake the inhaler several times.
- Hold the device upright and prime by opening the cap.
- Sit upright, hold head up and breathe out.
- Seal lips around mouthpiece and be careful not to block the air holes on top with your hands.
- Inhale slowly and deeply. Don't stop when the inhaler puffs.
- Remove inhaler and hold breath for as long as possible up to 10 seconds.
- Close the cap, with the inhaler upright.
- Recover before taking the next dose.
- (Remember to advise the patient, they won't feel the spray hitting the back of the throat—although there may be a slight taste disturbance).

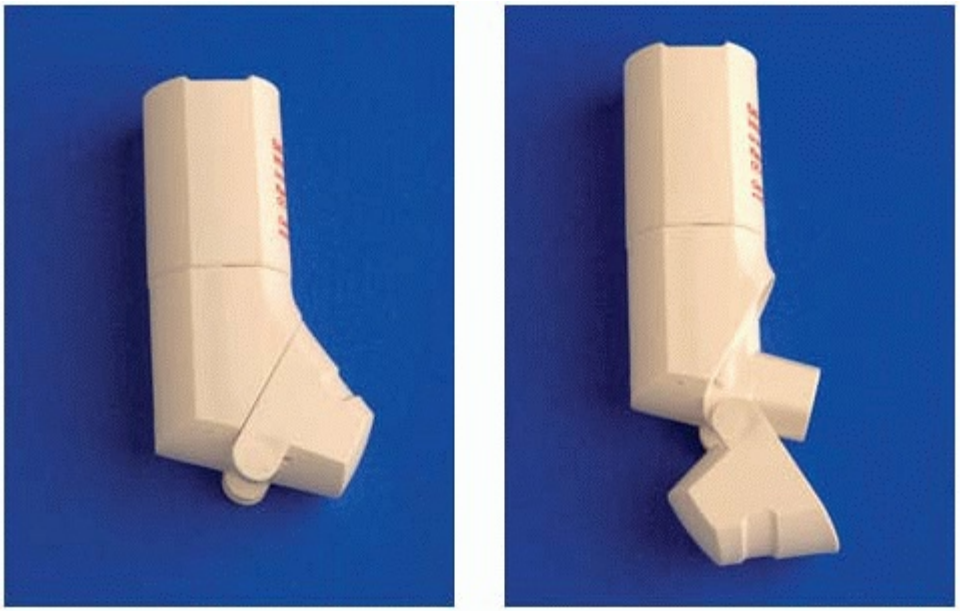


Fig. 17.12 Easibreathe. Note the air holes on the top which should not be blocked by your hand when taking a dose.

Hint

- Advise the patient not to dismantle the inhaler. Some people are prone to taking the top off and using like an MDI!

Accuhaler

This is one of the 'dry-powder' devices and has superseded the Diskhaler and the Rotahaler. Like most of the other inhalers, it is preloaded and has an integral cap. It also has a dose counter. However, it is more expensive than some of the other devices and has several-step priming mechanism that some may not be able to cope with.

Instructions for use

- Hold the outer casing and push the thumb grip away from you, exposing the mouthpiece, until you hear a click.
- Holding the mouthpiece towards you, slide the lever back until it clicks (the device is now primed and the dose-counter moves on one).
- Sit upright, hold head up and breathe out.
- Holding the Accuhaler lever, seal lips around mouthpiece.
- Inhale deeply and steadily.
- Remove inhaler and hold breath for as long as possible up to seconds.
- To close, slide thumbgrip towards you, so that the cover moves over the mouthpiece, until you hear a click.
- Recover before taking the next dose.
- (Remember to advise the patient, they won't feel the spray hitting the back of the throat—although there may be a slight taste disturbance).

Fig. 17.13 Accuhaler close and open. Note the thumbgrip, lever and mouthpiece.

Turbohaler

Another dry powder device with preloaded, tasteless drug. There is no dose counter, but a window that turns red after 20 doses—the device is empty when there is red at the bottom of the window. Some people find the lack of taste disadvantageous (they like to be sure the dose has been given) and, again, those with hand diseases or deformities may find it difficult to use.

Instructions for use

- Unscrew and remove the white cover.
- Hold the inhaler upright.
- Twist the grip clockwise then anticlockwise as far as it will go until a click is heard.
- Sit upright, hold head up and breathe out.
- Seal lips around mouthpiece.
- Inhale slowly and as deeply as possible.
- Remove the inhaler and hold breath for 10 seconds.
- Replace cover.
- Recover before taking the next dose.
- (Remember to advise the patient, they won't feel the spray hitting the back of the throat—although there may be a slight taste disturbance).
- **NB** There are devices available which can calculate whether a person has a sufficient inspiratory flow rate to deliver the drug into the airways.

Fig. 17.14 Turbohaler. Note the tiny dose-indicating window.

Handihaler

At the time of writing, this is relatively new to the market and only available for tiotropium. It is a dry-powder device with an integrated cap and requires a lower inspiratory flow rate than other devices. However, it is not preloaded, requiring a dose to be inserted via a capsule at each use requiring some dexterity. Some people also find the cap rather hard to open as it requires a moderate amount of strength to get right.

Instructions for use

- Open cap by pulling upwards exposing mouthpiece.
- Open the mouthpiece by pulling upwards exposing the chamber.
- Take a capsule from the blister-pack and insert it into the chamber.
- Replace the mouthpiece (make sure it clicks) and leave cap open.
- Press the side button in a few times to pierce the capsule (you can watch through the small window).
- Sit upright, hold head up and breathe out.
- Seal lips around mouthpiece.
- Breathe in deeply to a full breath (you should hear the capsule vibrate).
- Remove inhaler and hold breath for as long as is comfortable.
- Breathe out slowly.
- Remove the use capsule and replace the cap.

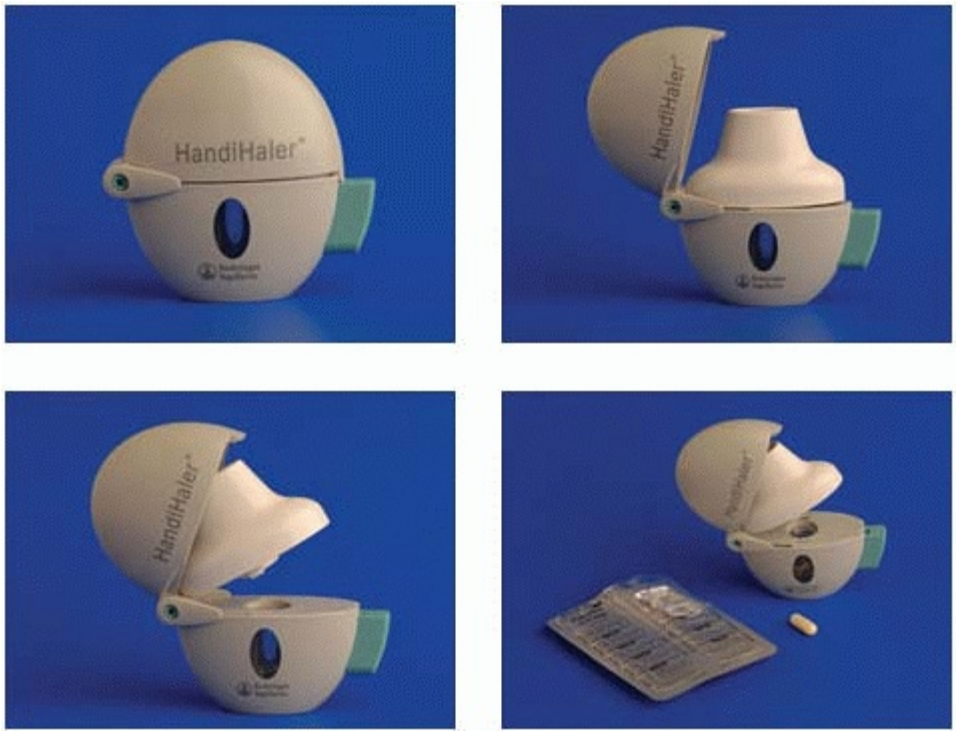


Fig. 17.15 Handihaler. Note the button at the side for piercing the capsule and the small window at the front.

Spacer devices

These are used with a standard MDI and allow the drug to be puffed into a chamber before it is inhaled. This reduces deposition of the drug in the upper airways (and the local side effects) and increases peripheral lung desposition. This also means that no coordination is required and the patient has more time to inhale the drug. These are particularly useful for the very young, elderly, or those with severe breathlessness.

These devices are, however, rather bulky which patients may find embarrassing. They do also require a certain amount of dexterity to put together.

There are a number of devices available but it is expected that, during the life of this edition, all will be replaced by the Aerochamber. We will, therefore, only discuss this device.

Instructions for use (Aerochamber)

- Remove cap of the MDI, shake the inhaler and insert into the back of the Aerochamber.
- Breathe out.
- Seal lips around mouthpiece.
- Press down the canister once to release the drug.
- Breathe in slowly and deeply—the Aerochamber will whistle if you breathe too quickly.
- Hold breath for 10 seconds.
- Breathe out through the mouthpiece and breathe in again (do not press the canister a second time). This may be repeated up to 4 or 5 breaths.
- If a second dose is required, relax for a minute and then repeat steps 3-5.
- Remover the inhaler and replace the cap.

Cleaning the Aerochamber

- The device must be rinsed daily in soapy water.
- Allow to air-dry on drainer—do not rub (creates static electricity).
- Aerochambers should be replaced by a new model every 6 months.

Fig. 17.16 Aerochamberwith MDI inserted in the end.

We thank Jeremy Robson for his help in the construction of the ‘Inhaler technique’ pages.

Oxygen administration

Theory

This is the administration of supplementary oxygen when tissue oxygenation is impaired.

The aim is to achieve adequate tissue oxygenation (without causing a significant ↓ in ventilation and consequent hypercapnia or oxygen toxicity) while minimizing cardiopulmonary work.

▸ Oxygen is a drug with a correct dosage and side effects which when administered correctly may be life saving.

The primary responsibility for oxygen prescription at the time of writing lies with the hospital medical staff. It is good practice to record:

- Whether delivery is continuous or intermittent.
- Flow rate/percentage used.
- What SaO_2 should be.

When to treat

- Tissue hypoxia is difficult to recognize as clinical features are non-specific—include altered mental state, dyspnoea, cyanosis, tachypnoea, arrhythmias, and coma.
- Treatment of tissue hypoxia should correct any arterial hypoxaemia (cardiopulmonary defect/shunt e.g. PE, pneumonia, asthma), any transport deficit (anaemia, low cardiac output), and the underlying causes.
- ▸ Remember SaO_2/PaO_2 can be normal when tissue hypoxia is caused by low cardiac output states.

Equipment

See 📖 p.592.

Procedure

- Explain what is happening to the patient and ask their permission.
- Chose an appropriate oxygen delivery device (see next page)
- Chose an initial dose …
  - Cardiac or respiratory arrest: 100%.
  - Hypoxaemia with $PaCO_2$ < 5.3kPa: 40-60%.
  - Hypoxaemia with $PaCO_2$ > 5.3kPa: 24% initially.
- Decide on the acceptable level of SaO_2 or PaO_2 and titrate oxygen accordingly.
- If possible, try to measure a PaO_2 in room air prior to giving supplementary oxygen. (This is not absolutely necessary especially if the patient is in severe respiratory distress/hypoxaemic.)
- Liaise with nursing staff, physiotherapist or outreach for support in setting up equipment.
- Apply the oxygen and monitor via oximetry (SaO_2) and/or repeat ABGs (PaO_2) in 30 minutes.
- If hypoxaemia continues, then the patient may require respiratory support either invasively or non-invasively—liaise with your seniors and/or the respiratory doctors.

- Stop supplementary oxygen when tissue hypoxia or arterial hypoxaemia has resolved.

Hints

- Only 10% of patients with COPD are susceptible to CO_2 retention with oxygen therapy. Use venturi style masks and monitor closely.
- Think about what is ‘normal’ for the individual.

Oxygen administration equipment

The method of delivery will depend on the type and severity of respiratory failure, breathing pattern, respiratory rate, risk of CO_2 retention, need for humidification and patient compliance.

Each oxygen delivery device comprises an oxygen supply, flow rate, tubing, interface ± humidification. (Humidification should be used for patient comfort, presence of thick tenacious secretions, or for flows >4L/min.)

Nasal cannulae

These direct oxygen via 2 short prongs up the nasal passage They:

- Can be used for long periods of time.
- Prevent rebreathing.
- Can be used during eating and talking.

Local irritation, dermatitis and nose bleeding may occur and rates of above 4L/min should not be used routinely.

Low flow oxygen masks

These deliver oxygen concentrations that vary depending on the patient's minute volume. At these low flow rates there may be some rebreathing of exhaled gases (they are not sufficiently expelled from the mask).

Fixed performance masks

These achieve a constant concentration of oxygen independent of the patient's minute volume.

The masks contain ‘venturi’ barrels where relatively low rates of oxygen are forced through a narrow orifice producing a greater flow rate which draws in a constant proportion of room air through several gaps.

Partial and non-rebreathe masks

Masks such as this have a ‘reservoir’ bag that is filled with pure oxygen and depend on a system of valves which prevent mixing of exhaled gases with the incoming oxygen.

The concentration of oxygen delivered is set by the oxygen flow rate.

High-flow oxygen

Masks or nasal prongs that generate flows of 50-120L/min using a high flow regulator to entrain air and oxygen at specific concentrations.

It is highly accurate as delivered flow rates will match a high respiratory rate in patients with respiratory distress. It should always be used with humidification.

We thank Heidi Ridsdale for her help in preparing this topic.

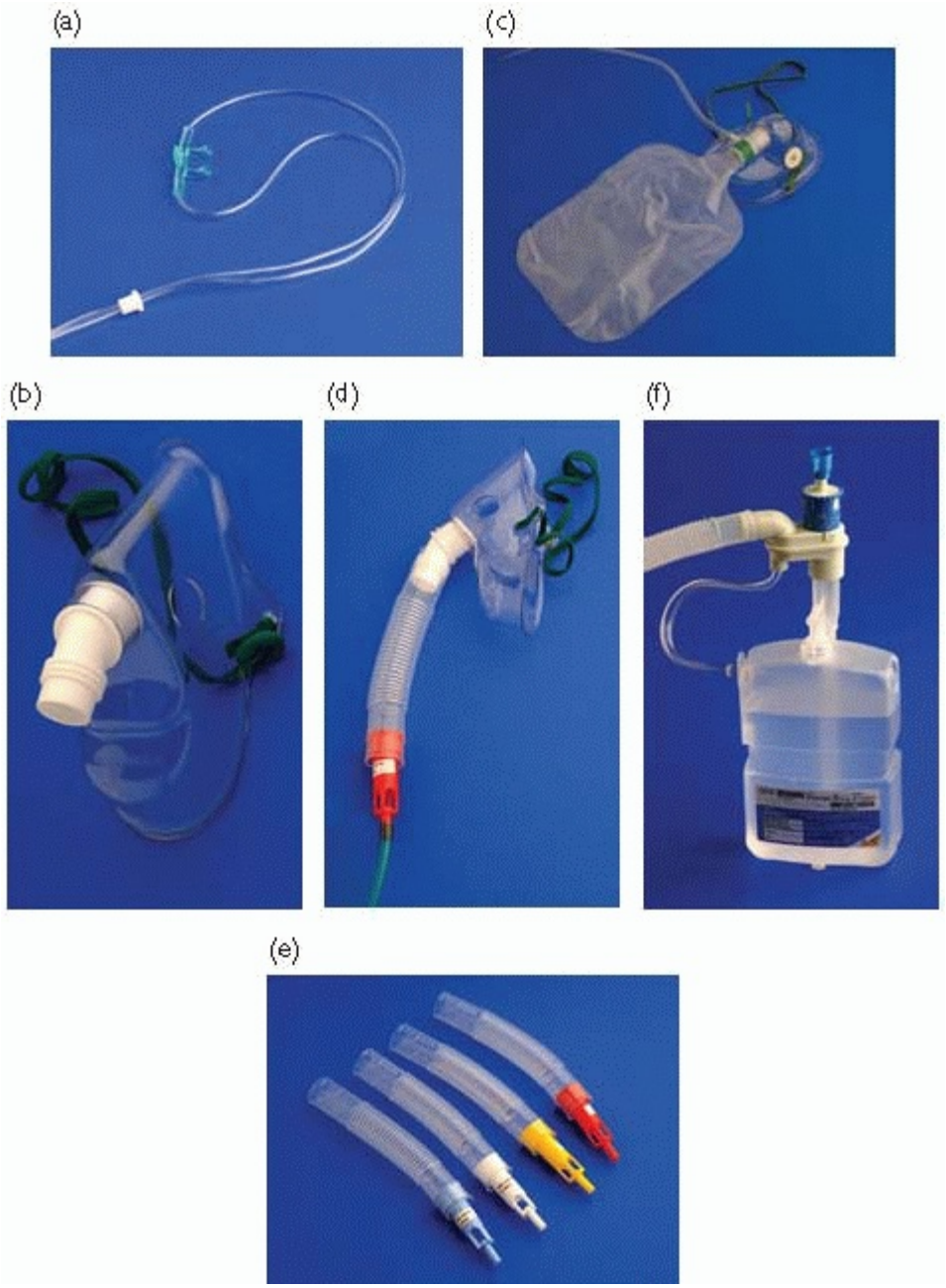


Fig. 17.17 (a) Nasal cannulae. (b) Low flow/variable concentration mask. (c) Non-rebreath mask. (d) Mask with venturi valve attached. (e) Selection of venturi valves. (f) Humidification circuit.

Basic airway management

Theory

An inadequate airway leads rapidly to hypoxaemia and, if uncorrected, brain damage and death. Endotracheal intubation remains the 'gold standard' for securing an airway and protecting the patient from aspiration.

Airway management without intubation is an important skill to master and consists of the use of one or more of the following; triple manoeuvre, facemasks, oropharyngeal and nasopharyngeal airways, laryngeal masks, and other supraglottic devices e.g. Combitube. It may

be carried out when intubation equipment or skills are unavailable, if intubation is difficult or on a patient with a partially obstructed airway.

Urgency is an important factor in planning and securing an airway in the most appropriate manner. This will depend on risk of vocal cord injury, degree of patient co-operation, anatomy of airway, equipment to hand and your own experience.

Before you start

▸ Think about simple positioning and the recovery position of the patient especially for airway protection alone.

- Assess for airway obstruction
  - LOOK (into mouth and for chest/abdominal movement).
  - LISTEN (snoring/gurgling/wheezing)
  - FEEL (expired air).
  - ▸ Complete airway obstruction is *silent*
- Make sure that you have:
  - Oxygen tubing.
  - Suction equipment.
  - Ambu-bag.
  - Rebreathe bag.

Hints

- A fully conscious talking patient is able to maintain his/her own airway and needs no further assessment.

 Do not use head tilt or chin lift in suspected cervical spine injury except as a last resort.

Box 17.4 Common causes of airway obstruction

- Tongue (due to unconciousness).
- Soft tissue swelling (trauma, tumor).
- Foreign material (blood, vomit).
- Direct injury.
- Secretions.
- Bronchospasm.

Box 17.5 Secure airways

A secure airway may be necessary in patients with the following:

- Apnoea.
- GCS < 9/15.
- High aspiration risk.
- Respiratory failure.
- Unstable mid-face trauma.
- Airway injuries.

Airway manoeuvres

The following are performed with the patient lying supine and all aim to open the airway with simple physical manoeuvres. These are useful as a first step in managing a patient with a compromised airway and are used in conjunction with an oxygen mask. Also useful in situations where the are no airway devices available.

If unsuccessful, you should go on to use additional equipment.

Head tilt

Place hands around patient's forehead and tilt backwards so as to achieve upper cervical extension (Fig. 17.18).

Chin lift

- Usually used with the head tilt.
- Place the tips of the index and middle fingers of your right hand under the front of the patients mandible.
- Lift upwards, pulling the mandible anteriorly (Fig. 17.19).

Jaw thrust

- Use this if there is suspicion of an injury to the cervical spine. A two-handed technique.
- Holding that patient from behind, place the fingers of both hands behind the angle of the mandible.
- Lift the mandible with these fingers whilst using your thumbs to displace the chin downwards, opening the mouth (Fig. 17.20).

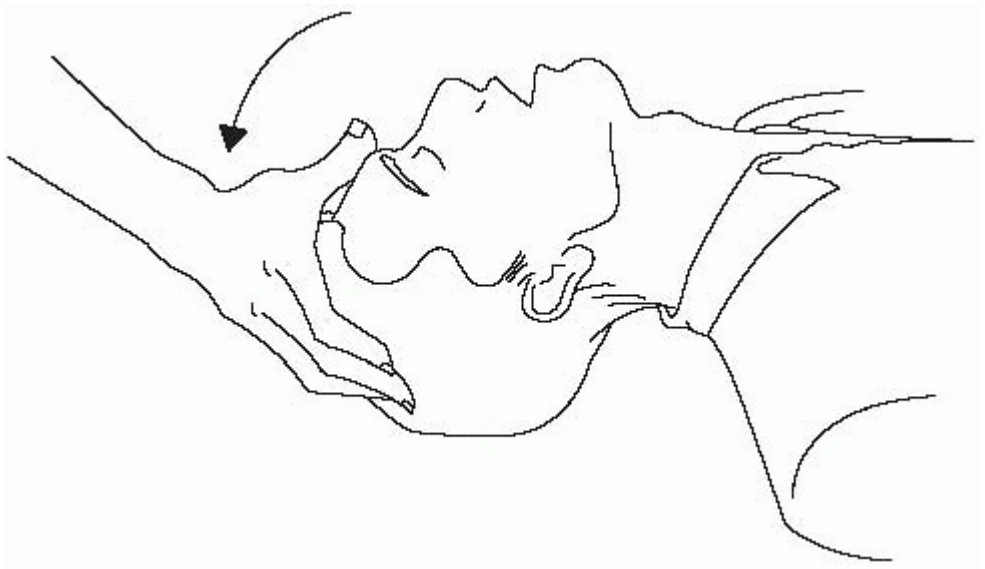


Fig. 17.18 Performing a head-tilt.

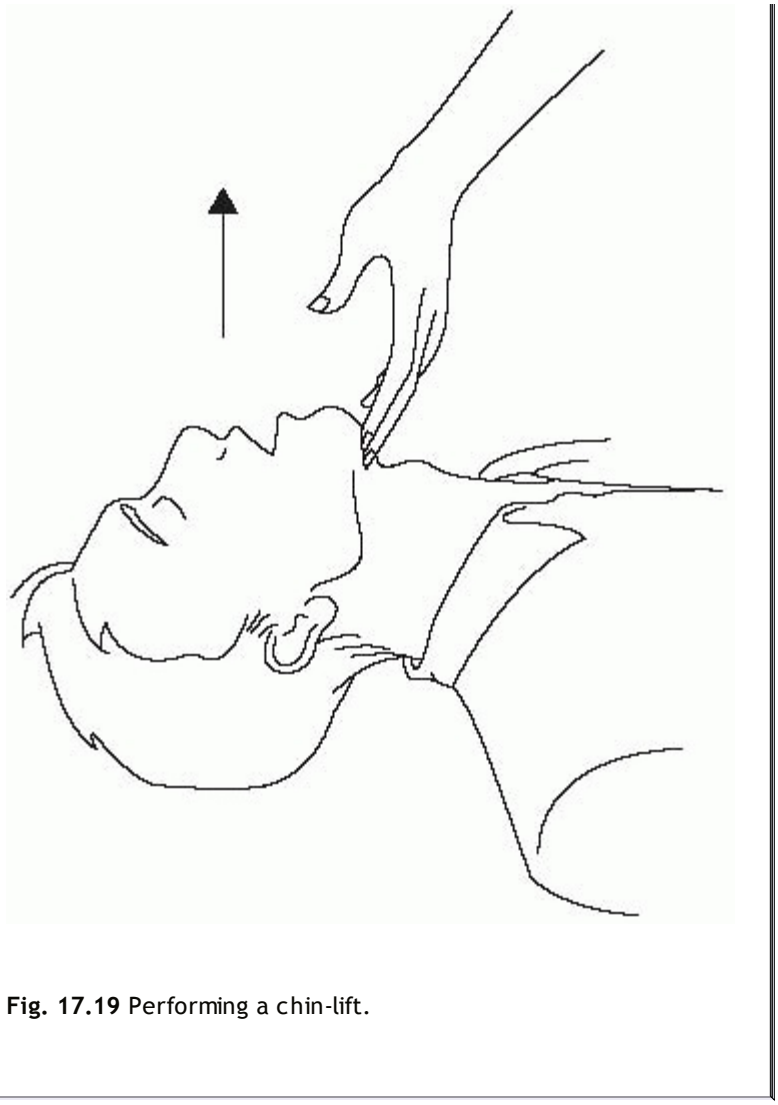


Fig. 17.19 Performing a chin-lift.

Fig. 17.20 Performing a jaw-thrust.

▸ ***Hint***

Do not use a head-tilt or chin lift in a patient with (or suspected to have) cervical spine injuries.

Airway devices

Facemasks

▸ Use the smallest fitting mask to fit over mouth and nose.

This is a simple mask that is fitted over the nose and mouth. You may use an airway to aid ventilation or to clear any obstruction.

One hand technique

- Place your thumb and index finger on the mask in a 'C' shape (see Fig. 17.21).
- Grasp the jaw with remaining fingers pulling the face into the mask.

Two hand technique

- Place your thumbs either side of the mount.
- Use your index fingers to support the body of the mask.
- Your other fingers can be used to lift the jaw and extend the neck (Fig. 17.22).

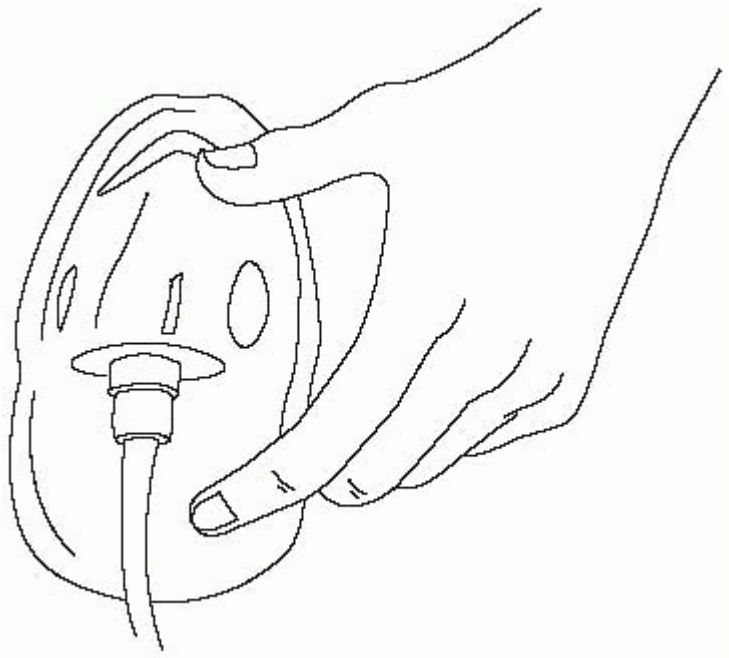


Fig. 17.21 Use of a face-mask, one-handed technique.

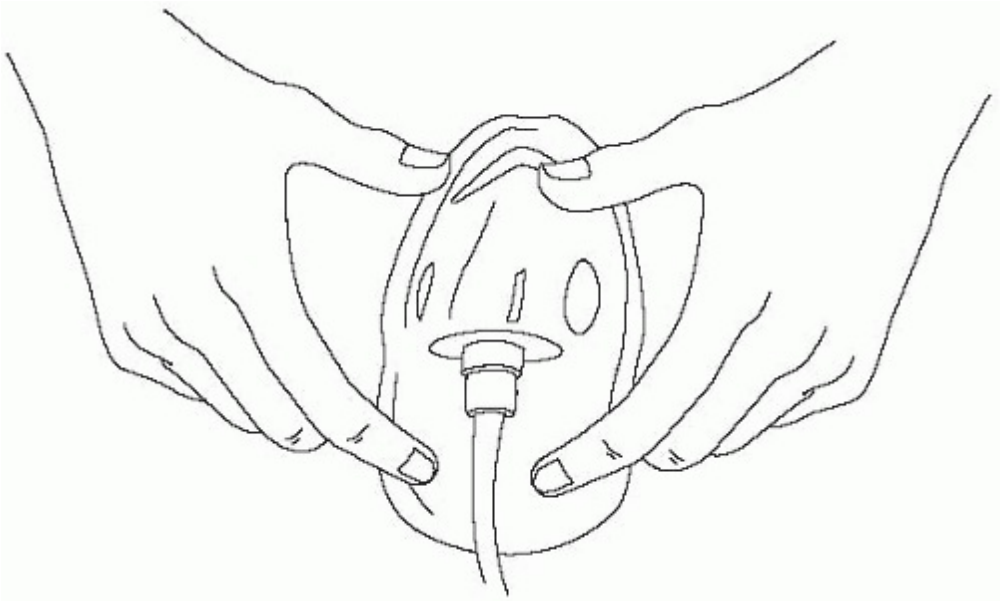


Fig. 17.22 Use of a face-mask, two-handed technique.

Oropharyngeal airway/Guedel airway

▸ Use when the patient is semi-conscious.

This consists of a flange (limits depth of insertion), bite portion (teeth of patient rest against this), and curved body (follows curvature of tongue) which has a lumen allowing passage of air and suction.

Different sizes are available and are colour coded. The correct size is determined by measuring the airway against the distance between the corner of the mouth and the angle of the jaw (Figs. 17.23 and 17.24).

Technique

- Lubricate and insert airway upside down.
- Once it is well into the mouth rotate 180° and advance to full position.
  - Alternatively, hold tongue down and forward with a tongue depressor until airway is in place.
  - Check for no gagging, snoring or vomiting and that air is moving in/out.
- Use a size 10/12/14 catheter for suction, if required.

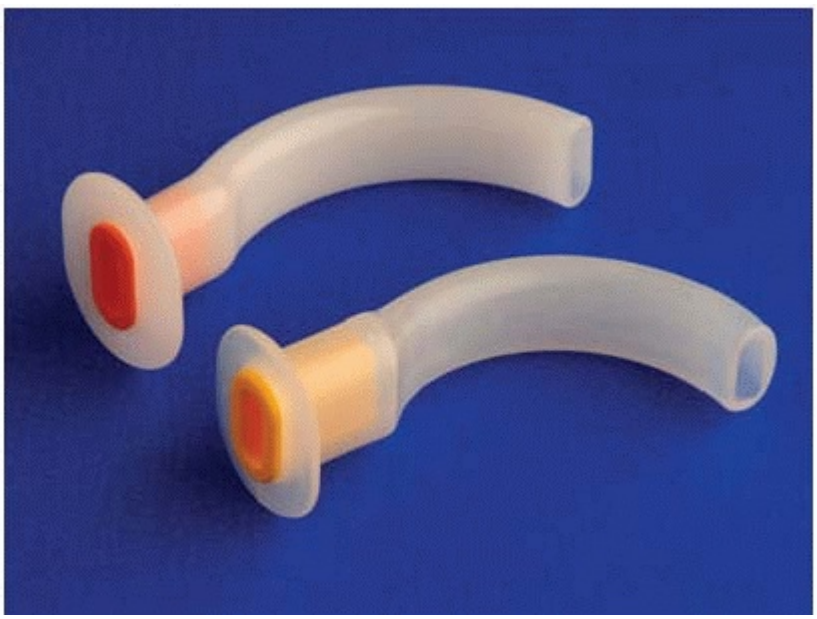


Fig. 17.23 Oropharyngeal airways.

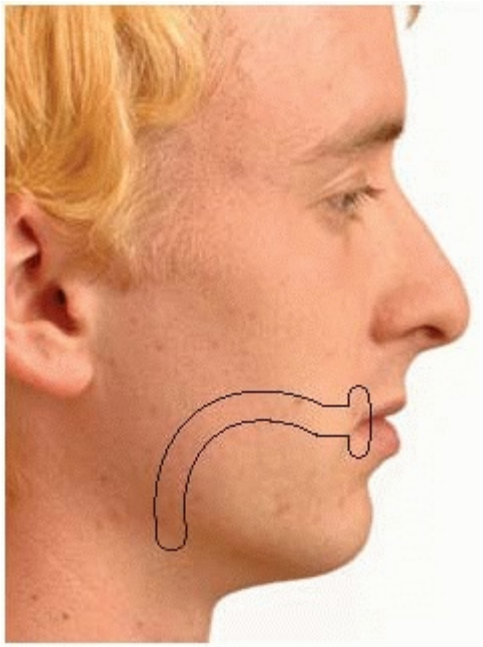


Fig. 17.24 Chose the size of the oropharyngeal airway by measuring from the patient's teeth to the angle of the mandible.

Nasopharyngeal airway

▸ Tolerated better than the oropharyngeal airway in alert patients.

This consists of a flange (limits depth of insertion). The pharyngeal end has a bevel to facilitate a non-traumatic insertion and curved body with lumen allowing passage of air and suction. Some airways come without an adequate flange, so a safety pin is used at the nasal end to prevent the airway falling back into the nose! (see figure).

Different sizes are available. Determine the correct size by comparing with the distance between the nostril and the tragus (see Fig. 17.25).

Technique

- The wider nostril is traditionally chosen but most airways are bevelled for introduction into the left nostril.
- Lubricate airway and pass directly into nasal passage passing along the floor of the nose or aiming for the back of the opposite eyeball.
- Use a size 10/12 catheter for suction, if required.

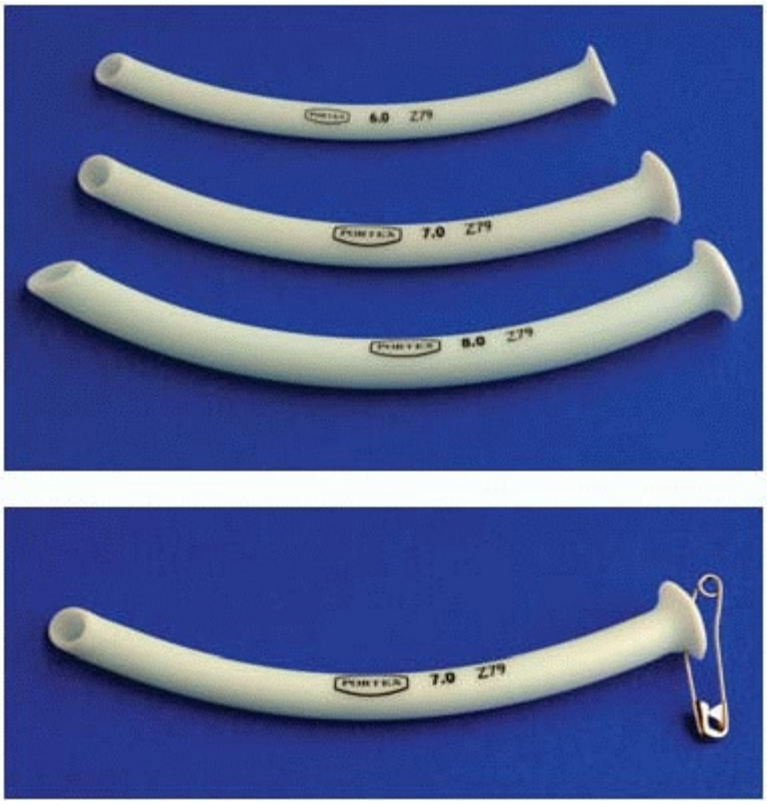


Fig. 17.25 Nasopharyngeal airways.

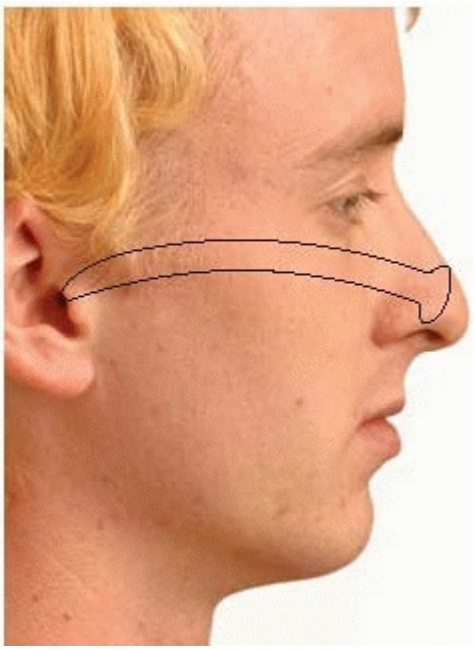


Fig. 17.26 Chose the size of the nasopharyngeal airway by measuring from the patient's nostril to the tragus.

Laryngeal mask airway (LMA)

This consists of a tube with an inflatable cuff which is designed to seal around the laryngeal opening. It requires the patient to be deeply unconscious.

Technique

- Maintain oxygenation by bag and mask.
- Deflate the cuff of the LMA using a 20ml syringe.
- Lubricate the outer cuff with aqueous gel. This part will not be in contact with the larynx.
- The patient should be in a supine position with the head and neck in alignment.
- Stand behind the patient or if this is not possible, from the front.
- Hold the tube like a pen and pass into the mouth with the distal aperture facing the feet of the patient.
- Push back over the tongue while applying the tip to the surface of the palate until it reaches the posterior pharyngeal wall.
- The mask is then pressed backwards and downwards until it reaches the back of the hypopharynx and resistance is felt.
- The black line on the tube should be aligned with the nasal septum.
- Inflate the cuff with usually 20-30ml of air.
- The tube should lift out of the mouth slightly and the larynx is pushed forward if it is in the correct position.
- Attach a breathing circuit and gently ventilate the patient with 100% oxygen.
- Confirm correct placement by auscultating the chest in the axillary regions and observe for bilateral chest movement.
- Insert a bite block or oropharyngeal airway alongside the tube and secure the airway with the bandage or tie provided.

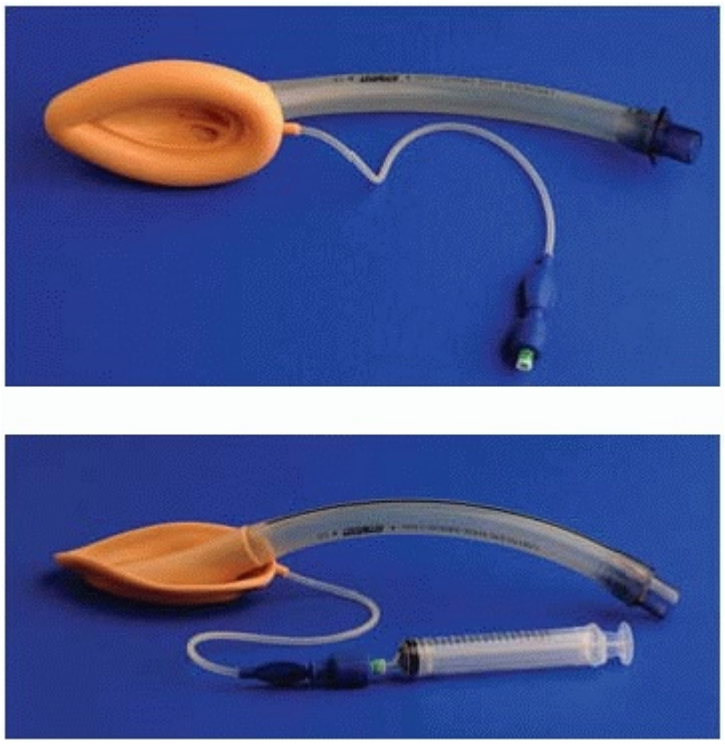


Fig. 17.27 A laryngeal mask airway.

We thank Heidi Ridsdale for her assistance with this topic.

Tracheostomy management

Theory

A tracheostomy is an opening in the anterior wall of the trachea below the larynx which can facilitate ventilation/respiration.

A mini-tracheostomy is a narrow diameter cuffless tube that is inserted into the trachea through which a catheter can be passed to stimulate a cough and/or suction. Is not a method for protecting the airway or delivering any kind of ventilatory support except emergency oxygen therapy.

Tracheostomy may be performed if the need for an endotracheal tube is prolonged, to facilitate weaning, identification of an inability to maintain/protect airway, and to secure and clear an airway.

Patients may have a permanent tracheostomy in place or even a stoma. These often do not need humidification or suction unless the patient is acutely unwell.

Equipment explained

The tracheostomy tube

The tracheostomy tube may be stitched in or secured around the neck and is either single or double lumen (has an inner tube that can be removed for cleaning). The tubes are either fenestrated or non-fenestrated.

Fenestrated tubes allow the passage of air and secretions into the mouth. These are good for weaning.

Humidification

Heated humidification is used for the short term and is the 'gold standard' A heat moisture exchanger (Swedish nose, thermovent) is used for patients with minimal secretions or who are mobile.

Procedures

Open suction

Aim is to remove secretions and prevent blockages in the tracheotomy tube, bronchial obstruction and alveolar collapse.

- Use a catheter with diameter no more than one 1/2 internal tracheotomy diameter. [(Size of outer diameter) -2×2… e.g. 8.0mm - 2 × 2 = 12].
- Negative pressure should be 100-150mmHg (13.5-20kPa).
- Wear gloves, apron, and protective eye wear.
- Attach a sterile catheter to suction equipment ensuring a good seal and leave most of the packaging in place.
- Place under non-dominant arm.
- Put a clean disposable glove on the dominant hand and do not touch anything other than the catheter tip.
- Pull the packaging away with non-dominant hand.
- Open the suction port.
- Introduce the tip of the catheter into the tracheotomy tube with the dominant hand gently but quickly.
- Depth of insertion should be 0.5-1.0cm beyond end of the tracheostomy tube (about 1/3 length of catheter).
- Insert until patient coughs.

- Withdraw tip 0.5cm and apply suction.
- Continue to withdraw slowly and continuously.
- Close suction port and discard glove and catheter into bin
- ▸ Suction should last no more than 15 seconds
- ▸ Allow sufficient time between passes for recovery.
- Repeat until secretions are cleared.
- After suction, ensure patient is reconnected to respiratory support and oxygen and that oxygen levels are returned to normal.

Tube occlusion

- Call for help.
- Reassure the patient.
- Ask the patient to cough or attempt to clear secretions via suction.
- Remove inner cannula and replace with new one.
- If no inner cannula, deflate cuff and administer oxygen facially, instill 2-5ml of 0.9% saline and suction to try to clear blockage.
- If unable to clear blockage, a total tube change may be required (try using smaller size if necessary).
- If tube insertion fails then consider mask to stoma ventilation (consider suction via stoma).
- ▸ If respiration stops all together, put out a 'crash call', call for anaesthetist, inflate the cuff and manually ventilate using catheter mount and rebreathe or ambu-bag.

Swallowing assessment

This should be performed by a competent practitioner (e.g. speech and language therapist).

- Sit the patient up.
- Suction via tracheotomy prior to cuff deflation.
- *Deflate* cuff fully if possible or the maximal the patient can tolerate or as agreed by the MDT.
- Ensure there is an appropriate inner cannula in place.
- Give the patient *sips* of water from a teaspoon and follow the procedure explained on p.316.
- Intermittently check for voice quality (ask the patient to say 'ah' or count out loud).
- Stop if patient deteriorates, fatigues, shows signs of persistent coughing, aspiration on suction, or persistently 'wet' voice.

Hints

- Practise techniques before needing them in an emergency.
- Stridor is a good indication that an airway is partially blocked.
- Always remember to humidify oxygen in tracheostomy patients.

We thank Heidi Ridsdale for her assistance with this topic.

Endotracheal (ET) intubation

Theory

There are 3 main indications for tracheal intubation: relieving airway obstruction, protecting the airway from aspiration, and facilitating artificial ventilation of the lungs.

▸ Remember, if you are inexperienced in this technique, never perform tracheal intubation unsupervized.

▸ In an emergency situation, it is safer to proceed to bag and mask the patient or use a laryngeal mask airway if one is available and await senior assistance.

Equipment

- Laryngoscope (check bulb)—usually size 3 is adequate.
- Selection of ET tubes (size 7 in most women and size 8 in most men).
- Sterile lubricant.
- 20ml syringe for cuff inflation.
- Tape to tie tube in place.
- Gum—elastic bougie, or rigid stilette.
- Self-inflating bag and oxygen supply.
- Stethoscope for confirming correct position of tube.
- Suction apparatus with a wide bore rigid suction end (Yankauer).

Procedure

- In the awake patient, introduce yourself, confirm the patient's identity, explain the procedure, and obtain verbal consent.
- Wash your hands and put on a pair of gloves.
- Pre-oxygenate the patient with a high concentration of oxygen for a minimum of 15 seconds.
- ▸ Remember, intubation must take no longer than 30 seconds.

- Position the neck such that it is distally extended and proximally slightly flexed position with a small pillow underneath the head—an exaggeration of the normal cervical lordosis.
    - ▸ If a cervical injury is suspected, the head and neck should be maintained in neutral alignment.
- Stand at the head of the bed and open the mouth.
- Inspect for loose dentures or foreign material—remove any if present.
- Hold the laryngoscope in the left hand and look down its length as you insert.
- Slide the scope into the right side of the mouth until the tonsillar fossa comes into view.
- Now move the blade to the left so that the tongue is pushed into a midline position.
- Advance, following the posterior edge of the soft palate until the uvula comes into view.
- Advance the blade over the base of the tongue and the epiglottis should pop into sight.

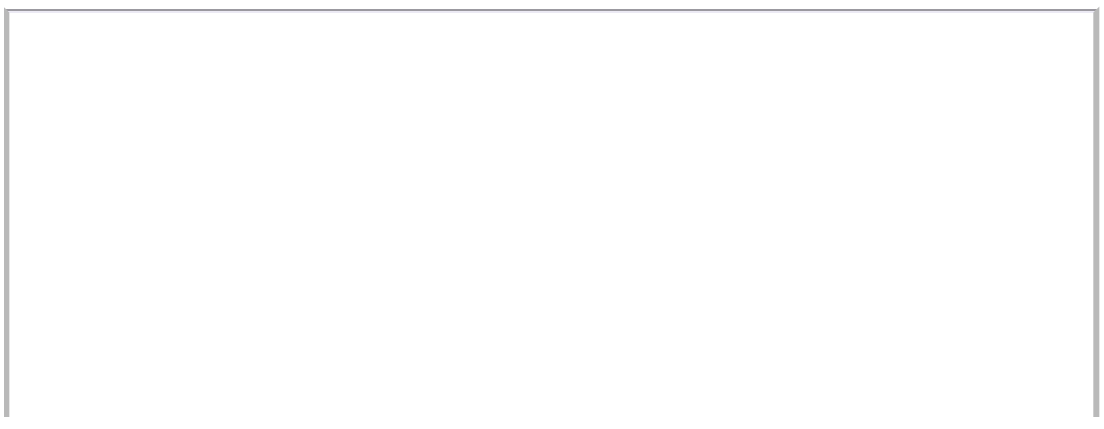
- With the blade positioned between the epiglottis and base of the tongue (vallecula), apply traction in the line of the handle of the laryngoscope.
    - This movement should lift the epiglottis and expose the v-shaped glottis behind.
- Once the triangular-shaped laryngeal inlet is in view, position the ET between the vocal cords so that the tube is just distal to them.
    - Use the mark on the tube above the cuff to indicate the correct position.
    - This is around 21cm in a female and 22cm in a male.
- If difficulty is experienced passing the ET tube into the larynx, pass a gum elastic bougie first and then try passing a lubricated ET tube over this.
- Once the ET tube is in position, inflate the cuff while ventilating through the ET with a self-inflating bag.
- Verify correct positioning of the tube by observing chest movement and auscultate at the sides of the chest in the mid-axillary line (both sides of the chest should move equally and you should hear breath sounds at both lung bases).
- Secure the ET tube with a tie.
- Obtain a CXR to confirm the tube position. The ET has a radio-opaque line within it.
- Document the details of the procedure in the notes.

Important note

▸ The insertion of the ET tube should take no more than 30 seconds from start to finish. If 30 seconds pass and the tube is not in the correct position, remove all the equipment and bag/mask ventilate the patient until you are ready to try again.

Some complications of ET intubation

- Trauma-to teeth, airway, larynx, or trachea.
- Aspiration.
- Airway obstruction.
- Tube misplacement.
- Hypoxia from prolonged attempts.
- Tracheal stenosis (late complication).



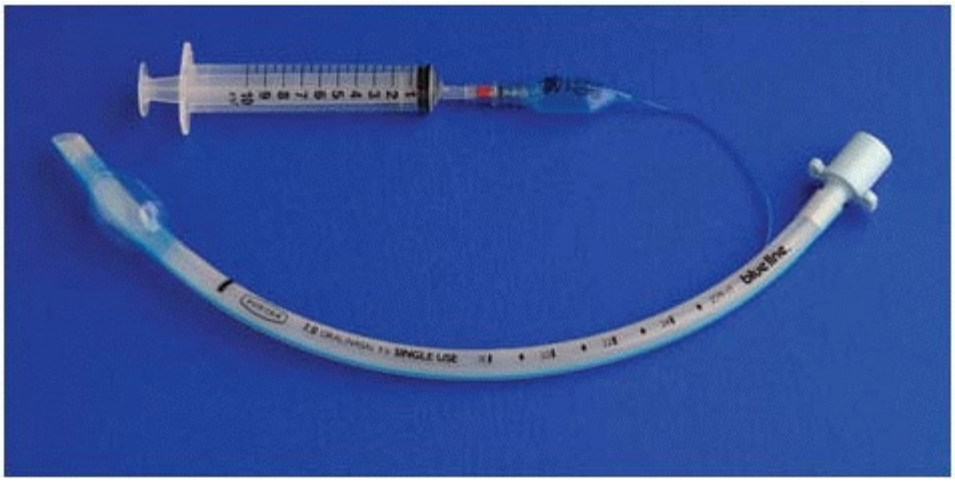


Fig. 17.28 ET tube with attached syringe. Note the cuff has been inflated to demonstrate.

Non-invasive ventilation (NIV)

Theory

NIV is the application of positive pressure ventilatory support via a facial or nasal interface and not via an airway (ETT, tracheostomy). NIV should be operated only by trained staff in an appropriate area. It may be used in acute conditions in hospital or in chronic conditions at home.

Patients need to be spontaneously breathing, maintaining their airway (i.e. conscious), and compliant. It is not a substitute for mechanical ventilation unless this has been decided as the 'ceiling' of treatment.

Pressures are usually documented in cmH_2O (rather than mmHg or kPa) and it is good practice that a decision of maximal pressure to be used is documented in the medical notes so that if a patient continues to deteriorate, the MDT has an appropriate management strategy in place.

Contraindications

Undrained pneumothorax and pulmonary haemorrhage. It is good practice to review a recent CXR to rule these out before beginning.

Cautions

Bullae, unstable cardiovascualar system, abscess, facial trauma, basal skull fracture, recent bronchial or oesophageal surgery, persistent vomiting, high bronchial tumour.

CPAP

Continuous **P**ositive **A**irways **P**ressure (CPAP) uses a single pressure continuously throughout both inspiratory and expiratory phases.

It is used in the treatment of type I respiratory failure (obstructive sleep apnoea, cardio-pulmonary oedema and occasionally in pulmonary embolus, pneumonia and weaning from ventilation).

BiPAP

Bilevel **P**ositive **A**irways **P**ressure (BiPAP) ventilation uses different pressures on expiration (EPAP) and on inspiration (IPAP). Higher EPAP increases FRC whilst higher IPAP augments tidal volume. The system is normally pressure driven but can be volume driven.

Used in the treatment of type II respiratory failure (i.e. hypoventilation, chronic neuromuscular conditions, exacerbations of COPD).

Box 17.6 Setting up NIV

This is not something that the student or early junior doctor will be expected to do. The following is a brief guide that should allow you to understand what is involved.

CPAP

Equipment

- Mask, head strap, PEEP valves (5-7.5-10cm H_2O).

- Circuit, safety ‘blow-off’ valve.
- High flow generator for oxygen and air.
- Heated humidification.

Procedure

- Explain all of the following to the patient and obtain verbal consent.
- Use measuring templates to assess appropriate size interface and minimize air leaks.
- Set oxygen and flow rate and ensure the PEEP valve opens a small distance only and never fully closes.
- Start with a low pressure and slowly increase for patient comfort and to gain compliance.
- Aim to reduce the work of breathing.
- Continuously monitor ABG/SaO_2, heart rate, and BP. Watch for abdominal distension.

BiPAP
Equipment

- Mask (facial/nasal), prongs, full face mask, head strap.
- Circuit, exhalation port.
- Entrained oxygen if required.
- Heated humidification.
- Ventilator (NIPPV1/2/3, Breas, BiPAP vision).

Procedure

- Explain all of the following to the patient and obtain verbal consent.
- Use measuring templates to assess appropriate size interface and minimize air leaks.
- Start with low pressures and slowly increase for patient comfort and to gain compliance. (Trial data in COPD is based on pressures of 20/5.)
- Setting inspiratory and expiratory times will need to be continuously reassessed as respiratory rate will change over time.
- Initially aim to match the patients own ventilatory pattern but eventually aim to ↓ respiratory rate and ↑ tidal volume/flow using the minimal pressures possible.
- Monitor ABG/SaO_2, heart rate and BP at 1 and 4 hours. Watch for abdominal distension.

We thank Heidi Ridsdale and Franco Guarasci for their assistance with this topic.

Pleural fluid sampling

Theory

After identifying a pleural effusion, a small volume of fluid may be aspirated and sent for biochemical, cytological, and microbiological analysis.

A neurovascular bundle runs on the inferior/inner aspect of each rib, to avoid this, needles for aspiration are inserted immediately *above* a rib.

Equipment

- Trolley.
- Sterile/wound care pack.
- Sterile gloves.
- Antiseptic solution.
- 5ml syringe.
- 20ml syringe.
- 1 vial of local anaesthetic (usually 1% lidocaine).
- Selection of needles (2 green, 1 orange/blue).
- 2 sterile sample containers.
- 1 pair of culture bottles.

- Biochemistry tube for glucose sample.

Procedure

- Introduce yourself, check the patient's identity, explain the procedure, obtain verbal consent, and unwrap the equipment.
- Position the patient comfortably sitting upright on the edge of the bed, leaning forward with arms raised—use a pillow on a raised bedside trolley for the patient to lean on (see Fig. 17.29).
- Percuss the upper border of the effusion posteriorly and choose a site 1 or 2 intercostal spaces below that.
- Mark the chosen spot at the upper edge of a rib with a pen.
- Wash hands and put on the sterile gloves.
- Clean the marked area using the antiseptic solution on a cotton ball. Work outwards in a spiralling fashion.
- Draw up 5ml of the lidocaine using the green needle.
- Swap the needle for an orange one and infiltrate the skin creating a surface bleb.
- Swap for a green needle and infiltrate anaesthetic deeper. Advance the needle in a step-wise manner-drawing back the syringe each time it is advanced to ensure vasculature is avoided and infiltrating anaesthetic before advancing again.
- Once you reach the pleural cavity, a flash-back of pleural fluid may be obtained.
- Take the 20ml syringe, attach a green needle and aspirate 20ml of pleural fluid, being careful to use the anaesthetized tract.
- Withdraw the needle and cover the wound with a suitable dressing (dry gauze and medical sticky tape will suffice).
- Put 4ml of fluid in each bottle and send to the laboratories for:
  - Biochemistry (pH, protein, LDH, amylase, glucose).
  - Cytology.
  - Microbiology (MC and S plus TB stain and culture if indicated).
- Request a CXR post-procedure to check for pneumothorax only if the procedure was difficult or high-risk.

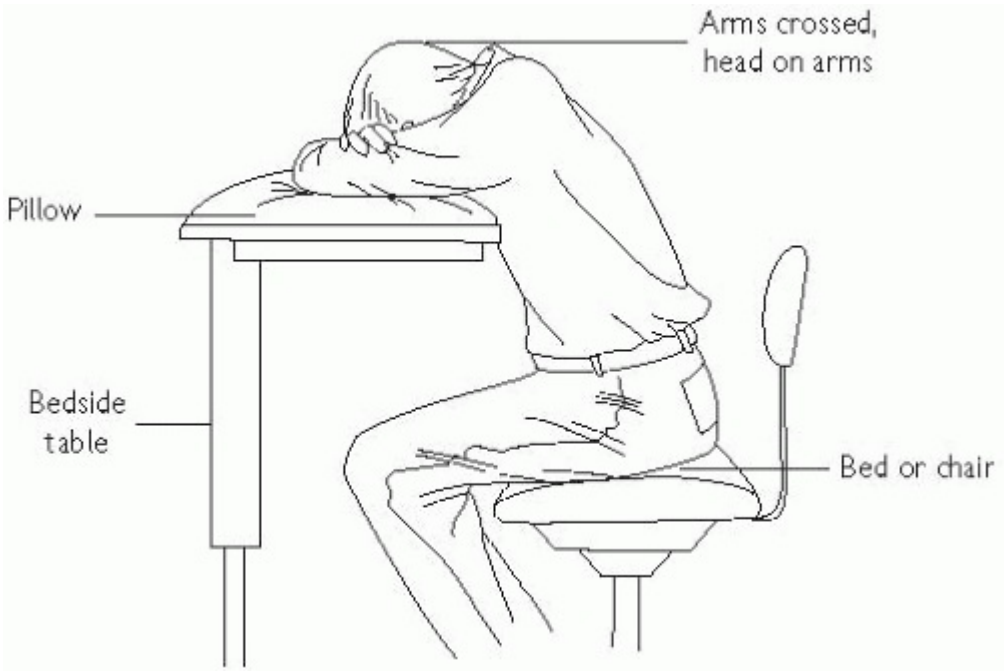


Fig. 17.29 Position a patient comfortably leaning forward—use a bedside trolley and pillow for them to lean on with their arms crossed.

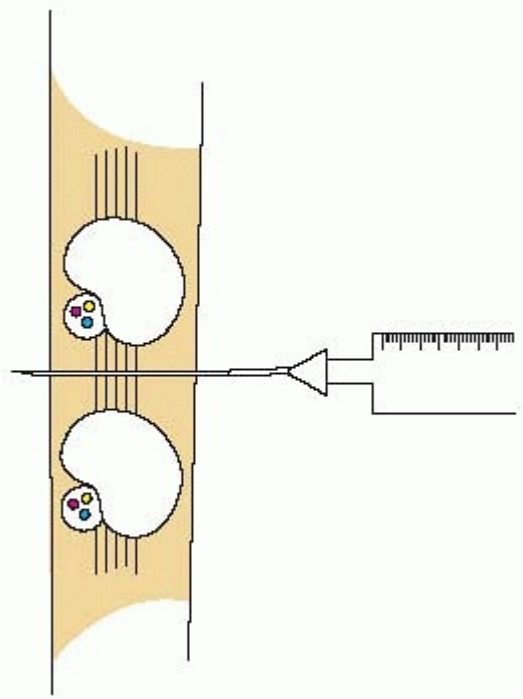


Fig. 17.30 Insert the needle just above a rib, at the lower border of the intercostal space to avoid the neurovascular bundle.

Hints

- ▸ If the lab is to measure pH, the sample must be sent to the laboratory immediately.
- Some laboratories will not measure pH—check before you begin. An alternative is to save a small amount of fluid, draw it up in a primed blood gas syringe and run it through a blood gas analyser to gain an instant pH measurement.
- In larger individuals, the pleural cavity may be at some depth from the skin. If this is the case, use a longer needle—needles of IV cannulae are often significantly longer despite being the same gauge.

Chest drain insertion

Theory

Drains are inserted to drain either fluid (pleural effusion/empyema) or air (pneumothorax) from the pleural cavity. In both cases, the insertion of the drain is almost entirely identical.

The drain is connected to a bucket with a small amount of water (creating an air-tight seal) so that there is no direct connection between the pleural cavity and the air. On inspiration, the negative intrathoracic pressure draws water up the tube (about 4cm); on expiration, the water level falls and (if draining a pneumothorax) air bubbles through the water. This one-way valve allows air or fluid to drain from the chest but not re-enter.

The method described below is the ‘Seldinger technique’. Other techniques exist for wide-bore drains but these are now only used in the setting of blunt trauma and cardiothoracic surgery—or for other problems such as extensive surgical emphysema overlying a pneumothorax.

Equipment

- Trolley.
- 10ml 1% lidocaine.
- 10ml syringe.
- 1 orange needle.
- 1 green needle.
- Sterile gloves.
- Sterile pack (containing cotton balls, container and drape).

- Incontinence pad.
- Suitable dressing (e.g. Hypofix® or drainfix®).
- Seldinger chest drain kit (containing chest drain, introducer, chest drain needle, syringe, scalpel, 3-way tap, guide wire).
- Suture (no. 15).
- Iodine solution.
- Chest drain tubing.
- Chest drainage bottle.
- 500ml sterile water.

Procedure

- Introduce yourself, confirm the identity of the patient, explain the procedure, and obtain verbal consent.
- Double-check the history and CXR to be *sure* of which side needs the drain.
- Position the patient sitting on a chair or the edge of their bed, arms raised. Instead of asking the patient to hold their arm over their head, it is often easier to ask them to cross their arms and lean on a bedside table with a pillow, raised level with their shoulders, (see Fig. 17.29).
- Triple-check the side by briefly examining the patient (tap out the dullness of an effusion or listen for the ↓ breath sounds of a pneumo).
- The usual site for insertion is in the mid-axillary line, within a triangle formed by the diaphragm, the latissimus dorsi and the pectoralis major. (Fig. 17.31). For apical pneumothoraces, you may wish to choose the 2nd intercostal space in the mid-clavicular line.
- Place the incontinence pad on the bed to mop up any spillages.
- Mark your chosen spot (just *above* a rib to avoid hitting the neurovascular bundle) with a pen.
- Sterilize the area with antiseptic solution or iodine on cotton balls working in a spiral pattern outwards from the insertion point.

- Using the syringe and the orange needle, anaesthetize the skin (see p.552) forming a subcutaneous bleb.
- Swap the orange needle for the green one and anaesthetize deeper, remembering to aspirate before injecting to ensure that you have not hit a vessel. Anaesthetize right down to the pleural cavity and only stop when you aspirate air or pleural fluid.
- Use the scalpel to make a small cut in the skin.
- Now use the drain-kit needle with the curved tip and syringe (in some kits, this has a central stilette which needs to be removed first). With the curved tip facing downwards (upward for a pneumothorax), advance through the anaesthetized route until you are aspirating either air or fluid again.
- Remove the syringe and hold the needle steady.
- Thread the guide-wire through the needle into the pleural cavity (this usually comes pre-coiled but often needs to be retracted slightly first to straighten the curve on the tip). See Fig. 17.32.
- Once the wire is half in the chest, discard the covering.
- Now withdraw the needle from the chest but **be sure to not remove the guide wire**—KEEP HOLD OF IT AT ALL TIMES.
- Thread the needle right off the end of the guide wire. You should now have the wire in the chest but nothing else.
- Thread the introducer over the guide wire and into the chest, twisting back and forth as you go to open up a tract for the drain's passage. You can then slide the introducer back off the wire—but be careful not to pull the wire out of the chest.
- The chest drain has a central stiffener in place, leave this in situ. Now thread the drain over the wire and into the chest, curving downwards. Always HOLD ON TO THE WIRE with one hand—you may need to pull the wire out of the chest slightly so that it protrudes from the end of the drain before you push the drain into the chest. You don't want to push it right into the chest and lose it!
- Once the drain is in place, you can withdraw the wire and the central stiffener.
- Quickly attach the 3-way tap and make sure all the ports are closed.
- Relax.
- You can now stitch the drain in place. This needn't be complicated—a simple stitch just above the drain will suffice with the ends then wrapped tightly around the drain knotted several times.
- Fix the drain in place with a ‘drainfix®’ or another suitable dressing.
- Attach the drain to the tubing and the tubing to the drain collection bottle which you have pre-filled with 500ml of sterile water.
- Open the 3-way tap. You should either see the fluid start to flow or air start to bubble in the collection bottle. Ask the patient to take a few deep breaths and watch the water level in the tubing to ensure it is rising and falling (‘swinging’).

- Warn the patient not to knock the bottle over and keep it below the level of their umbilicus.
- Request a post-insertion CXR.

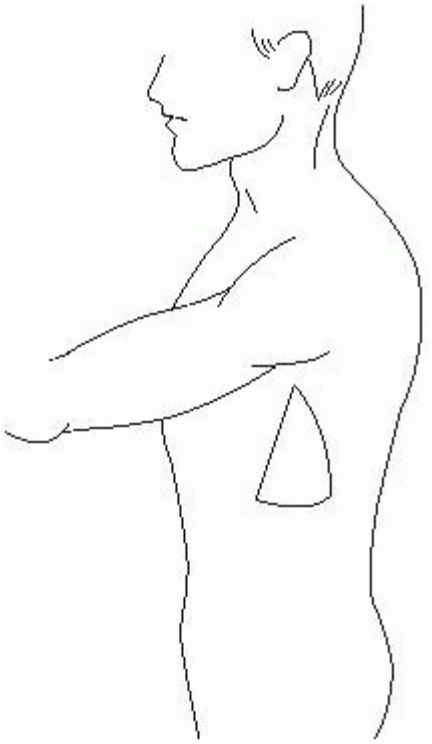


Fig. 17.31 Correct positioning of the patient for chest drain insertion and ideal site of drain insertion.

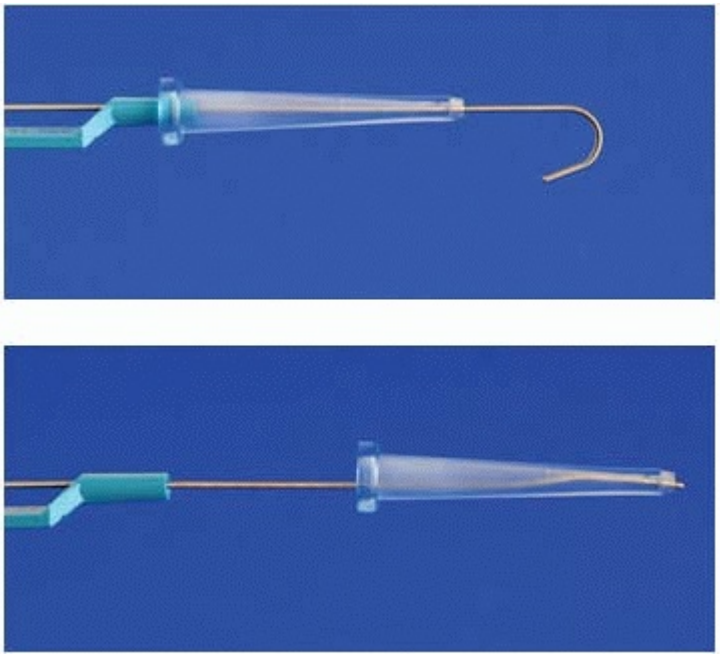


Fig. 17.32 Prepare the guide-wire before starting the procedure by retracting slightly so as to straighten the curved tip.

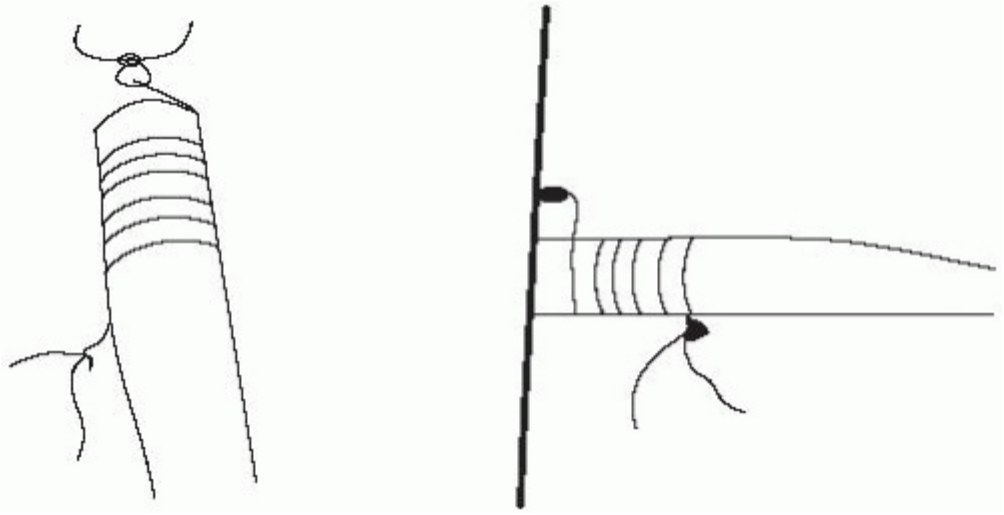


Fig. 17.33 Diagrammatic representation of a suitable stitch to hold the drain in place.

Nasogastric (NG) tube insertion

Theory

A plastic tube is inserted through the nose, down the back of the throat, oesophagus and into the stomach.

The 'bore' of the tube (large = 16, medium = 12, small = 10) is dictated by the tubes intended purpose. For short- or medium-term nutritional support in those with a defective swallow, a fine-bore tube is used. Larger bores are used to drain the stomach contents and decompress intestinal obstruction.

Contraindications: severe facial trauma and basal skull fractures.

Complications: aspiration, tissue trauma, electrolyte loss, tracheal or duodenal intubation, perforation of oesophagus or stomach.

Equipment

- Disposable gloves.
- Plastic apron.
- Drape.
- Lubricant gel.
- NG tube.
- Cup of water ± straw.
- 50ml syringe.
- Drainage bag (if necessary).
- Adhesive tape or steristrips and hypofix.
- Disposable vomit bowl.
- Paper towel.

Procedure

- Introduce yourself, confirm the patient's identity, explain the procedure, and obtain verbal consent.
- Wash hands thoroughly, put on gloves and plastic apron.
- Ideally, the patient should be seated upright (often the head tilted slightly forwards can aid insertion).
- Examine patient's nose for deformity/obstructions and decide which nostril to use.
- Use the tube to measure the distance xiphisternum → earlobe → tip of nose and note the distance.
- Lubricate the first 4-8cm of tube. You may also wish to use local anaesthetic spray on patient's throat if available.

- Pass the tube into the nostril, and then posteriorly, a short distance at a time. You will feel it turn the corner at the nasopharynx and another slight obstruction as it passes into the oesophagus.
- If the patient is able, they should be asked to swallow as the tube passes the pharynx—a brief sip of water may help here.
- Advance the tube as far as the pre-measured distance.
- To check for correct placement, you may wish to aspirate some stomach contents with the syringe and test the fluid's pH (should be <6).
- Secure the tube to the patient's nose with some tape, you may also wish to curl it back over their ear and secure it to their cheek.
- Request a CXR and confirm the tube's position *(below* diaphragm in the region of the gastric bubble) before using for feeding.
- Once the position is confirmed, remove the central guidewire before use and save this (use a plastic page-file and file in the nursing notes).
- Ensure that you record the procedure in the patient's notes.

▸ *Hints*

- If resistance if felt, try rotating the tube whilst advancing. Never force.
- Partially pre-freezing the tube can ease its passage.
- An alternate test of correct placement: insert a small quantity of air (20-30ml) down the tube using the syringe whilst listening to the epigastrium with the stethoscope. You should hear the air entering the stomach. (NB This technique is no longer considered appropriate in the UK and health care workers are advised against using it).

We thank Lyn Dean for her help with the preparation of this page

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Ascitic tap

Theory

A needle is inserted through the abdominal wall allowing the withdrawal of a small amount of fluid for diagnostic purposes.

Equipment

- 1 green needle.
- 1 orange needle.
- 10ml syringe.
- 20ml syringe.
- 5-10ml 1% lidocaine solution.
- Iodine or antiseptic solution.
- Microbiology culture bottles (anaerobic and aerobic).
- Sterile pack (including gloves, cotton balls and container).
- 2 sterile collection bottles.
- Biochemistry tube (glucose).
- Haematology tub.

Procedure

- Introduce yourself, confirm the identity of the patient, explain the procedure, and obtain verbal consent.
- Ensure that the patient has emptied their bladder.
- Position the patient lying supine or in the lateral decubitus position leaving the right side available—undress exposing the abdomen.
- Percuss the extent of the ascitic dullness (p.260).
- Mark your chosen spot in the region of the right iliac fossa (preferably) within the area of dullness.

- Clean the area thoroughly with antiseptic and don the sterile gloves.
- Infiltrate the skin and subcutaneous tissues with lidocaine via the orange needle and 10ml syringe and wait a minute for it to take effect.
- Attach the green needle to the 20ml syringe and insert into the abdomen, perpendicular to the skin. Advance the needle as you aspirate until fluid is withdrawn.
- Aspirate as much fluid as possible (up to the 20ml).
- Remove the needle and apply a suitable sterile dressing.
- Put ~4ml of fluid in each bottle and send to the laboratories for:
  - Biochemistry—standard collection bottle (albumin, LDH, amylase).
  - Biochemistry—accurate glucose collection tube (glucose).
  - Cytology.
  - Haematology (total and differential white cell count).
  - Microbiology (MC&S).

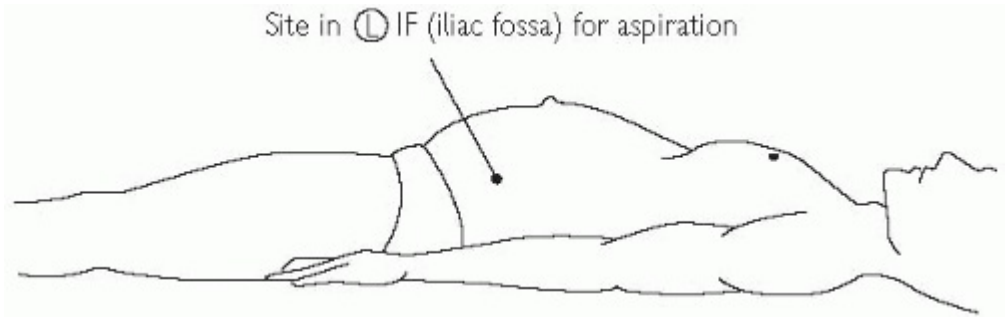


Fig. 17.34 Performing a diagnostic ascitic tap.

Abdominal paracentesis (drainage)

Theory

A drain is inserted into the abdominal cavity allowing drainage of large amounts of ascitic fluid for therapeutic purposes.

The procedure below relates to a Bonanno drainage kit—the essence is the same for other catheter kits although minor details may differ. You should refer to the manufacturer's instructions.

Equipment

- 1 orange needle.
- 1 green needle.
- 2 ×10ml syringes.
- 5-10ml 1% lidocaine solution.
- Iodine or antiseptic solution.
- Sterile pack (including gloves, cotton balls and container).
- Bonanno abdominal catheter pack (catheter, sleeve, puncture needle and adaptor clamp).
- Catheter bag.

- Catheter bag stand.
- Scalpel.

Procedure

- Introduce yourself, confirm the identity of the patient, explain the procedure, and obtain verbal consent.
- Ensure that the patient has emptied their bladder.
- Position the patient lying supine or in the lateral decubitus position leaving the right side available—undress exposing the abdomen.
- Percuss the extent of the ascitic dullness (📖 p.260).
- Mark your chosen spot in the region of the right iliac fossa (preferably) within the area of dullness.
- Clean the area thoroughly with antiseptic and don the sterile gloves.
- Infiltrate the skin and subcutaneous tissues with lidocaine via the orange needle and 10ml syringe and wait a minute for it to take effect.
- Attach the green needle to the other 10ml syringe and insert into the abdomen, perpendicular to the skin. Advance the needle as you aspirate until fluid is withdrawn.
- Prepare the catheter kit—straighten the catheter (which is curled in the pack) using the plastic covering sheath provided.
- Take the needle provided in the pack and pass through the sheath such that the needle bevel is directed along inside the curve of the catheter—continue until the needle protrudes from the catheter tip.
- Close off the rubber bung at the end of the catheter.
- Make a small incision in the skin using the scalpel.
- Grasp the catheter needle ~4' above the distal end and, with a firm thrust, push the needle through the abdominal wall to ~3cm deep.
- Disengage needle from the catheter hub and advance catheter until the suture disc is flat against the skin.
- Withdraw needle.
- Connect adaptor-clamp to the catheter hub and securely attach the rubber portion of the clamp into a standard drainage catheter bag.
- Carefully suture the catheter into the abdominal wall—you may also need to apply further tape to ensure the catheter won't fall out.
- Ensure the clamp is open to allow fluid to drain(!)

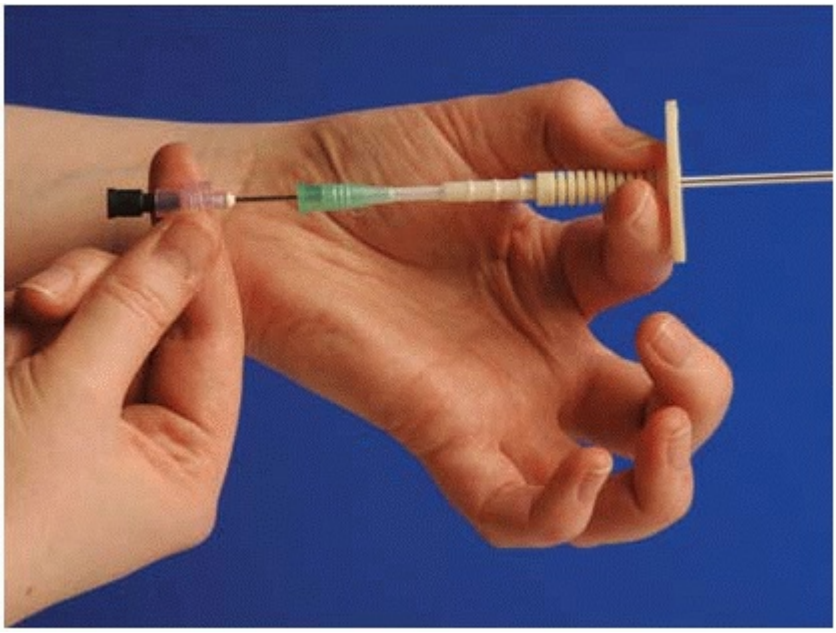


Fig. 17.35 The assembled catheter components.

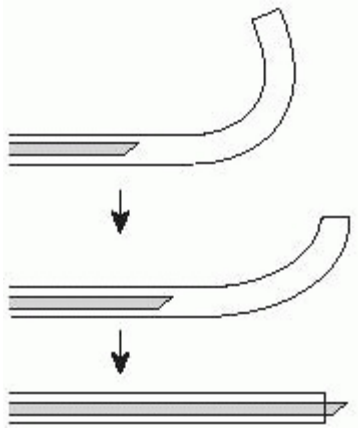


Fig. 17.36 Inserting the needle into the curved catheter.

Hints

- In cirrhotic patients, protein loss should be replaced (and haemodynamic stability maintained) by infusing human albumin solution (HAS) IV at a rate of 100ml of 20% HAS for every 2.5L of ascitic fluid drained—check local protocols with the gastroenterology dept.
- Usually catheters are not left in place for >24 hours.
- During drainage, the flow may stop suggesting that the drain is blocked—this may be positional and simply moving the patient may solve the problem.

Male urethral catheterization

Theory

A urinary catheter has a balloon near the tip which is inflated via a sidearm near the other end. Once inside the bladder, the inflated balloon prevents it falling—or being pulled—out.

Equipment

- A catheter pack (containing a kidney dish, a small pot with cotton balls, a sterile towel, sterile gauze, sterile gloves).
- Antiseptic solution or sachet of saline.
- 10ml 1% lignocaine/lubricant gel in pre-filled syringe (eg Instilligel®).
- 10ml water-filled syringe.
- A catheter bag (leg bag if situation is not acute).
- A male catheter (12F or 14F).

Procedure

- Wash hands thoroughly. Confirm the patient's identity, explain procedure, and obtain verbal consent.
- Unwrap all the equipment onto a trolley in an aseptic fashion and pour saline solution over the cotton balls.
- Position the patient supine with genitalia exposed. Raise bed to a comfortable height.
- Wash hands again and put gloves on. Create a hole in the centre of the towel and drape over the patient so the penis can be reached through the hole.
- From here on, use your non-dominant hand to hold the penis with some gauze.
- Clean the penis with the wet cotton balls, working away from the meatus. Remember to retract the foreskin and clean beneath.
- Lift penis to a vertical position, carefully position the nozzle of the lubricant gel inside the meatus and instill the full 10ml slowly. (If proving problematic, can be aided by gentle ‘milking’ action.)

- Position the kidney bowl between the patient's thighs to catch spillages later.
- The catheter will be in a plastic wrapper with a tear-away portion near the tip. Remove this portion, being careful not to touch the catheter.
- Insert the tip of the catheter into the urethral meatus and advance slowly but firmly by feeding it out of the remaining wrapper.
- On passing through the prostate, some resistance may be felt which, if excessive, may be countered by adjusting the angle of the penis by pulling it to a horizontal position between the patient's legs.
- On entering the bladder, urine should start to drain. Advance the catheter *to the hilt* to ensure the balloon is beyond the urethra.
- Inflate the balloon with the 10ml of saline via the catheter side-arm. Warn the patient to alert you to any pain and watch his face.
- Remove the syringe and withdraw catheter until resistance is felt.
- Attach draining tube and catheter bag.
- Replace the foreskin, clean and redress the patient as necessary.

Hints

- You may wish to verify the presence of a full bladder with a bladder-scanner before starting.
- Lack of urine drainage may be caused by: blockage of gel, empty bladder or catheter misplacement.
- Attempt to aspirate urine using a catheter-tipped syringe. Feel for a full bladder. If there is any doubt about the position of the catheter, remove immediately (deflating balloon first) and seek senior advice.
- Always record the residual volume—this is essential in cases of urinary retention.
- Consider the use of prophylactic antibiotics before the procedure.
- Complications: pain, infection, misplacement and trauma.
- Patients with prostate disease can often experience some mild haematuria following catheterisation. Don't worry but watch carefully and be sure the bleeding doesn't continue or form into clots.
- ▸ Beware latex allergy!

We thank Lyn Dean for her help with the preparation of this page.

Female urethral catheterization

Theory

A urinary catheter has a balloon near the tip which is inflated via a side-arm near the other end. Once inside the bladder, the inflated balloon prevents it falling—or being pulled—out.

Bear in mind that nurses tend to catheterize females if they are able—so if a doctor is asked to do it, expect the catheterization to be rather tricky! Always consider antibiotic prophylaxis.

Equipment

- A catheter pack (containing a kidney dish, a small pot with cotton balls, a sterile towel, sterile gauze).
- Sterile gloves.
- Saline solution.
- 5ml 1% lidocaine/lubricant gel in pre-filled syringe (e.g. Instilligel®).
- 10ml saline-filled syringe.
- A catheter bag.
- A female catheter (12F or 14F).

Procedure

- Wash hands thoroughly. Confirm the patient's identity, explain procedure, and obtain verbal consent.

- Unwrap all the equipment onto a cleaned (antiseptic) trolley in an aseptic fashion and pour saline over the cotton balls.
- Position the patient supine with knees flexed and hips abducted with heels together. Raise bed to a comfortable height.
- Wash hands again and put gloves on. Lay the towel and drape over the patient so the genitalia are exposed.
- From here on, use your non-dominant hand to hold the labia apart, approaching the patient from the right hand side, leaning over their ankles so as to reach the genitalia from below.
- Clean the genitalia with the wet cotton balls (using each once only), working in a pubis-anus direction.
- Carefully position the nozzle of the lubricant gel inside the meatus and instilling most of the 5ml.
- Position the bowl between the patient's thighs to catch spillages.
- The catheter will be in a plastic wrapper with a tear-away portion near the tip. Remove this portion, being careful not to touch the catheter and apply a little lidocaine gel to the catheter tip.
- Insert the tip of the catheter into the urethral meatus and advance slowly but firmly by feeding it out of the remaining wrapper.
- On entering the bladder, urine should start to drain. Advance the catheter fully to ensure the balloon is beyond the urethra.
- Inflate the balloon with the 10ml of saline via the catheter side-arm. Warn the patient to alert you to any pain and watch her face.
- Remove the syringe and withdraw catheter until resistance is felt.
- Attach draining tube and catheter bag.
- Clean and redress the patient as necessary.
- Record the residual urinary volume.

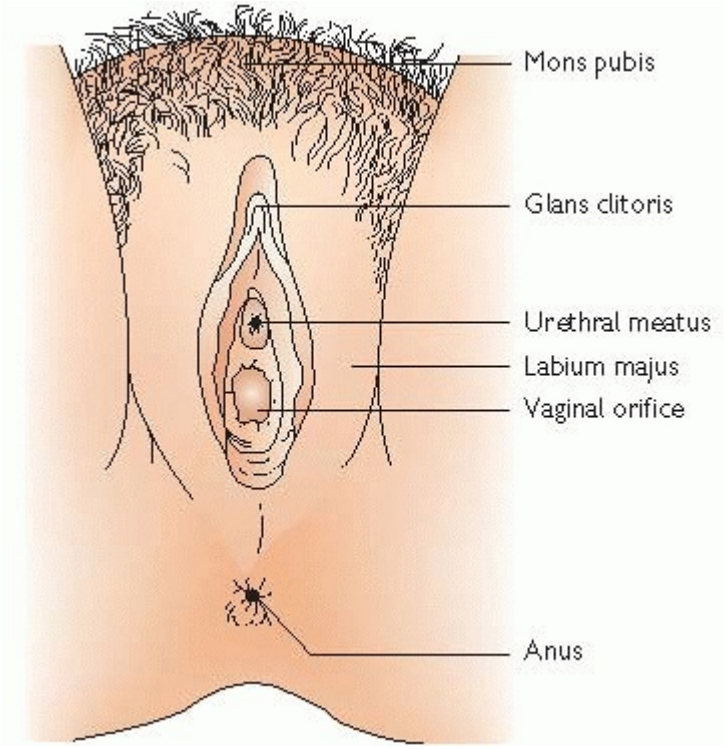


Fig. 17.37 Diagrammatic representation of the female external genitalia showing position of the urethral meatus.

Hints

- Some female patients are easier to catheterize in a different position—lying on their side with knees raised (seek experienced help).
- Lack of urine drainage may be caused by: blockage of gel, empty bladder or catheter misplacement.
- Attempt to aspirate urine using a catheter-tipped syringe. Feel for a full bladder. If there is any doubt about the position of the catheter, remove immediately (deflating balloon first) and seek senior advice.
- Complications:

- Pain.
- Infection.
- Misplacement and trauma.

- ▸ Beware latex allergy!

We thank Lyn Dean for her help with the preparation of this page.

Suprapubic catheterization

Theory

Suprapubic catheterization is sometimes seen as a safer and more efficient means of controlling bladder drainage than urethral catheterization—particularly if the patient has had treatment or surgery involving the vagina, urethra, ureter, or prostate. Patients often find this more acceptable than urethral catheterization. Also, it allows the assessment of when the patient is able to void spontaneously—without having to remove (and possibly replace) a urethral catheter.

The catheter is inserted directly into the bladder, through the abdominal wall just superior to the pubic symphysis. Safe placement under local anaesthesia requires a very full bladder.

The procedure below relates to the 'Bonanno' Suprapubic kit. The essentials of the technique will remain the same for other catheterization systems although small details may differ—refer to the pack instructions.

Many urologists currently favour the Bard Addacath system.

Equipment

- Trolley.
- 1 Bonanno Suprapubic catheter pack (contains puncture needle, catheter with sleeve and adaptor clamp).
- 1 drainage bag.
- Iodine or antiseptic solution.
- 2 × 10ml syringes.
- 5-10ml 1% lidocaine.
- 1 green needle.
- 1 orange needle.
- Sterile pack (containing gloves, swab, and container).
- Fine, non-absorbable suture.

Procedure

- Introduce yourself, confirm the identity of the patient, explain the procedure, and obtain verbal consent.
- Position the patient supine with genitalia exposed. Raise bed to a comfortable height.
- Unwrap all the equipment onto a trolley in an aseptic fashion and pour antiseptic solution over the cotton balls.
- Before commencing, make sure that the patient has a palpable bladder. If not, distend the bladder with 500-700ml of saline solution instilled via a urethral catheter (if urethral route available/feasible).
- ▸ If the bladder is not full **proceed no further.**
- Put on sterile gloves, prep the suprapubic area with antiseptic solution.
- The point of insertion is in the midline, 2 finger-breadths above the pubic symphysis and well below the upper edge of palpable bladder.
- Assemble the Bonanno catheter components (see Fig. 17.38).
- Advance the catheter sleeve along the course of the radio-opaque catheter from a proximal position adjacent to the suture disc to the distal end of the catheter to allow straightening of the coiled catheter.
- Infiltrate the insertion area with the lidocaine.

- Carefully insert the 18-gauge puncture needle into the catheter so that the heel of the needle bevel is directed along inside the curve of the catheter and move in a clockwise direction—until the bevel extends beyond the catheter tip (Fig. 17.38).

- Slide the straightener sleeve off the distal end of the catheter.
- Grasp the catheter needle ~9cm above the distal end and, with a firm thrust, push the needle through the abdominal wall—heading in a slightly caudal direction—until you feel resistance disappear.
- Check the position of the catheter in the bladder by removing the black vent plug and aspirate urine using a 10ml syringe.
- Disengage needle from the catheter hub and advance catheter until the suture disc is flat against the skin.
- Withdraw needle.
- Connect adaptor-clamp to the catheter hub and securely attach the rubber portion of the clamp into a standard drainage catheter bag.
- Carefully suture the catheter into the abdominal wall—you may also need to apply further tape to ensure the catheter won't fall out.
- Ensure the clamp is open to allow urine to drain(!)

Hints

- It may be easier to use a scalpel to make a small stab incision before inserting the needle.

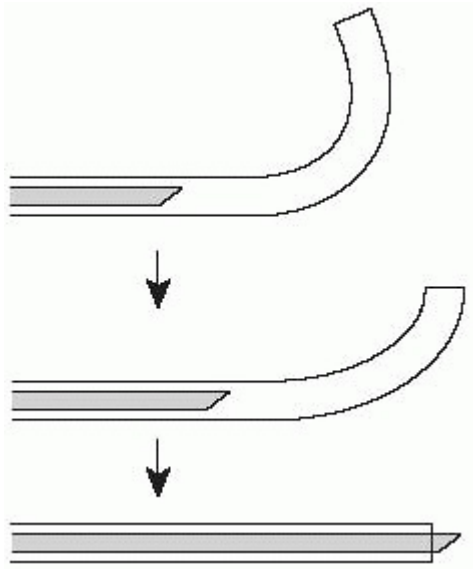


Fig. 17.38 Inserting the needle into the curved catheter.

Basic suturing

Theory

Basic suturing, or stitching, has many practical applications outside the field of surgery.

Whether you are called upon to suture a central line in place or are stitching up a laceration, it's a skill you should practise before you need to use it. There are many useful texts and articles which describe in more detail the fine art of suturing and we would refer you to these. Undoubtedly, the best way to learn is by watching a surgeon and then doing it yourself. In most clinical skills labs you should find the necessary equipment in order to practise these skills.

Equipment

- Trolley.
- Dressing pack.
- 21-gauge green needle.
- 25-gauge orange needle.
- 10ml and 20ml syringes.

- Gauze.
- Antiseptic solution.
- Sutures (selection depending on the site and nature of the wound).
- Tape.
- Sterile gloves.
- Sharps bin.
- Toothed forceps.
- Needle holder.
- Scissors.
- Scalpel.

Procedure

- Introduce yourself, confirm the identity of the patient, explain the procedure, and obtain verbal consent.
- First assess the wound and decide on the size of the suture material.
  - Remember that there are alternative ways to achieve wound closure such as glue, staples and steri-strips. Always consider the most appropriate means of closing a wound.
- Before suturing, irrigate the wound, and remove any foreign bodies and any non-viable or infected tissue.
- Use a needle holder such as toothed forceps where possible, to minimize the risk of needlestick injury.
- Hold the needle 2/3 of the way from the needle tip.
- Lift the skin edge farthest away without pinching or damaging it.
- Pierce the skin with the needle at 90°.
- Rotate your wrist to pass the needle into the middle of the wound.
- Release the forceps and clasp the needle again as it protrudes into the wound, rotating it out of the wound.
- Next press the near side with the closed forceps to evert the skin edge, and pass the needle through, taking a smooth semicircular course to exit at 90° to the wound edge. See Fig. 17.39.
- This method ensures a square bite and good eversion of the wound.
- Now perform a surgeon's knot.
  - Wrap the long end of the thread around the forceps, which is used to transfer the coil around the short end (grab the short tail and pull in towards you, pulling the long end away).
  - Repeat the cycle.

- Remember to cut the ends of the thread off, leaving a few mm so that they can be easily removed later.

▸ When removing sutures, clean the wound with antiseptic solution, use forceps or a blade and pull the suture out across rather than away from the wound.

The time taken to remove non-absorbable sutures depends on the location:

- Face: 5-7 days.
- Scalp: 7-10 days.
- Limbs and trunk: 12-14 days.

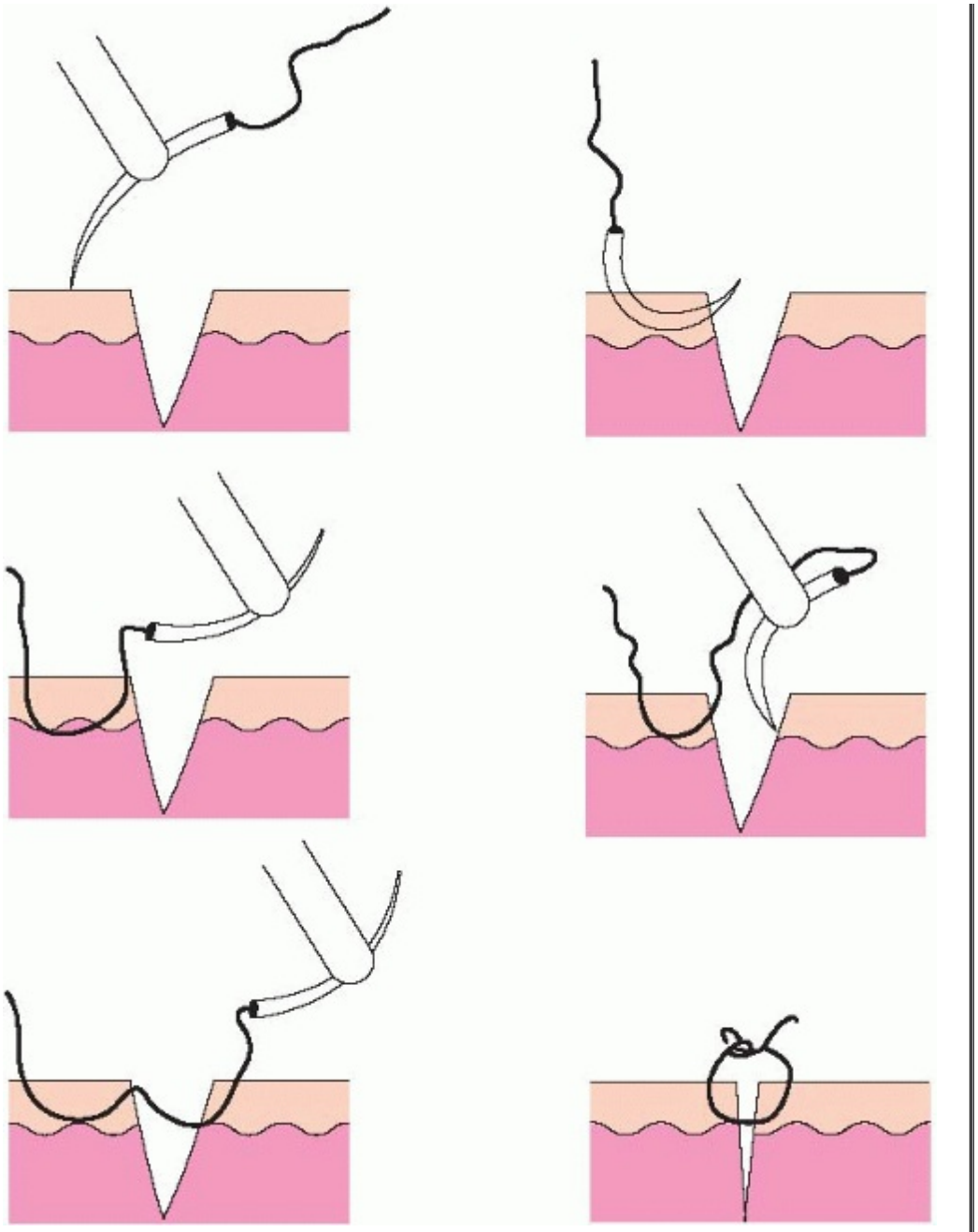


Fig. 17.39 Diagrammatic representation of the stages involved in a basic suture.

Lumbar puncture

Theory

A needle is introduced between the lumbar vertebrae at a level below the termination of the spinal cord. It then passes through the dura into the subarachnoid space and a sample of cerebrospinal fluid is obtained.

Used for diagnostic and therapeutic purposes too numerous to list.

Equipment

- Sterile gloves.
- Sterile pack (containing drape, cotton balls and container).
- Antiseptic solution.
- Sterile gauze dressing.
- 5-10ml 1% lidocaine.
- 2 × 10ml syringe.

- Biochemistry tube for glucose.
- Orange needle.
- Green needle.
- LP needle.
- LP manometer.
- 3-way tap (may be included in LP kit).
- 3 sterile collection tubes.

Procedure

- Introduce yourself, confirm the identity of the patient, explain the procedure, and obtain verbal consent.
- Position the patient lying on their left-hand side with the neck, knees and hips flexed as much as possible (if able, the patient may be asked to clasp their hands around their knees). Put a pillow between the patient's knees to prevent the pelvis tilting (see Fig. 17.40).
- Ensure that the patient can hold this position comfortably.
- Identify the iliac crest—the disc space vertically below this (as you are looking) will be ~L3-L4.
- Mark the space between the vertebral spines with a pen.
- Wash hands and put on sterile gloves.
- Unwrap all equipment and ensure it fit together correctly.
- Apply the drapes around the area and sterilize with the antiseptic solution and cotton balls in outward-spiral motions.
- Inject the lidocaine (using a 10ml syringe and the orange needle) at the marked site to raise a small wheal.
- Swap the orange needle for the green one and infiltrate the lidocaine deeper. Take care to aspirate before injecting to ensure blood vessels are avoided.
- Wait for ~1 minute for the anaesthetic to take effect.
- Introduce the spinal needle (22G usually) through the marked site at about 90° to the skin, heading slightly toward the umbilicus. Keep the bevel facing up the patient's spine.
- Gently advance the needle through the ligaments (to ~5cm depth).
- At this point, a further push of the needle should produce a ‘give’ as the needle enters the subarachnoid space (this takes a little practise to feel with confidence).
- If, at any point, the needle strikes bone and cannot be advance, withdraw slightly, re-angle and advance in a stepwise fashion until the gap is found.

- Withdraw the stilette from the needle … CSF should begin to drip out.
- Measure the CSF pressure—connect the manometer to the end of the needle via the 3-way tap (the CSF will rise up the manometer allowing you to read off the number).
- Open the tap and allow the CSF to drip into the 3 collection tubes—about 5 or 6 drips per tube. The tubes should be labelled ‘1’, ‘2’, and ‘3’, in order of collection. Collect a few more drips into the biochemistry tube for glucose measurement.
- Replace the stilette and remove the needle. Apply a sterile dressing.
- Send the fluid for analysis.
  - Cell count (bottles 1 and 3).
  - Microscopy, culture and sensitivities (bottles 1 and 3).
  - Biochemistry: glucose, protein (bottle 2).
- Advise the patient to lie flat for ~1 hour and ask nursing staff to check CNS observations at least twice during that time.

Hints

- Always use the smallest gauge spinal needle available.
- If the patient suffers a severe or prolonged headache after the procedure, it may be possible to inject ~20mL of venous blood into the LP site to produce a ‘blood patch’—ask for senior/anaesthetic advice!

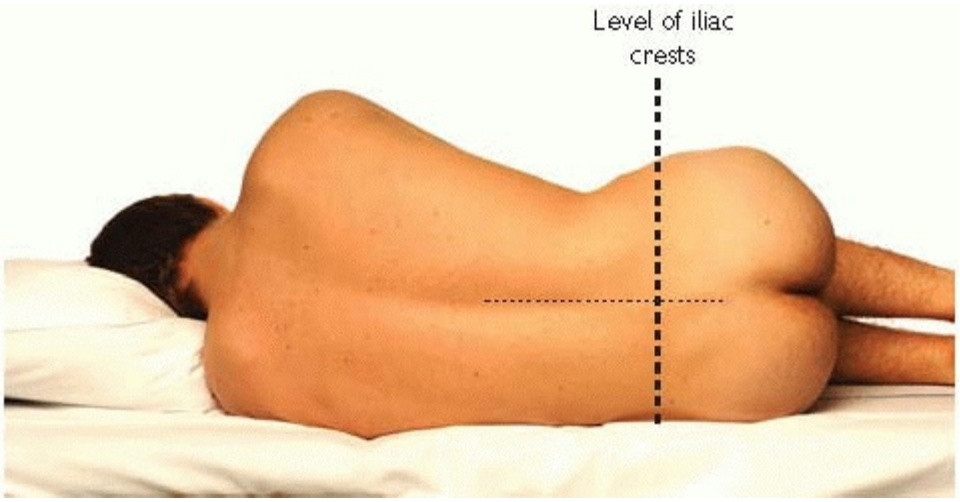


Fig. 17.40 Correct position of the patient for a lumbar puncture.

Pericardial aspiration

Theory

Emergency pericardial aspiration (drainage of fluid from the pericardial cavity) may be performed in cardiac tamponade or large pericardial effusions where there is haemodynamic compromise. This procedure can also be used to obtain diagnostic pericardial fluid.

Equipment

- Sterile gown and gloves.
- Antiseptic solution.
- Sterile towels.
- 10ml syringe.
- 50ml syringe.
- Three-way tap.
- ECG monitoring, defibrillator and resuscitation equipment.
- Local anaesthetic.
- Needles.
- 18-gauge cannula.

Procedure

- If conscious, introduce yourself, confirm the identity of the patient, explain the procedure, and obtain verbal consent.
- Establish IV access and connect ECG monitor with full resuscitation equipment to hand.
- Provide adequate sedation if necessary.
- Put sterile gloves and gown.
- If time permits, use local anaesthesia to infiltrate the insertion site.
- Attach the 18-gauge cannula to the 50ml syringe.
- Introduce needle at 45° to the skin immediately below and to the left of the xiphisternum to a depth of 6-8cm, in a direction aiming for the tip of the scapula.
- Aspirate continuously and watch the ECG.
    - If the needle touches the ventricle an injury pattern (depressed ST segment) or arrhythmia may be seen—withdraw the needle slightly.

- Aspirate pericardial fluid through the syringe and 3-way tap.
- Aspiration should produce immediate haemodynamic improvement.
- You can check if the fluid you are aspirating is pure blood if it clots quickly. Heavily bloodstained pericardial fluid does not clot.
- Perform a CXR and echocardiogram after the procedure.
- You may wish to insert a pericardial drain (seek senior advice).
- Document the details of the procedure in the notes.

Possible complications

- Pneumothorax.
- Arrhythmia.
- Myocardial puncture.
- Damage to the coronary arteries.

Defibrillation

Theory

Electrical defibrillation is the only effective therapy for cardiac arrest caused by ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT). The Resuscitation Council (UK) strongly recommends a policy of early attempted defibrillation. This is because the chances of successful defibrillation decline at a rate of 7-10% with each minute of delay.

In the hospital setting, 2 types of defibrillator may be encountered: the traditional manual defibrillator and the newer automated external defibrillators (AED).

There are two types of AED: most are semi-automatic and advise the need for a shock, but this has to be delivered by the operator when prompted. Some also have the facility to enable the operator to override the device and deliver a shock manually, without any prompts. A few fully automatic AEDs are also available.

Equipment

- Defibrillator.
- Gel pads.

Procedure for manual defibrillation (Fig. 17.42)

- Switch on the defibrillator, ensure the skin is dry and free of excess hair.
- Attach the ECG electrodes accordingly:
  - Red under right clavicle.
  - Yellow under left clavicle.
  - Green at the umbilicus.
- Ascertain that the ECG rhythm is shockable (VF/pulseless VT).
- Place the defibrillation gel pads on the patient's chest:
  - One just to the right of the sternum, below the clavicle.
  - The other just lateral to the cardiac apex.
- Shave chest hair only if it is excessive and will interfere with electrical contact.
- Select 360J on the defibrillator.
- Place the paddles firmly on the gel pads on the gel pads.
- Press the charge button on the paddles to charge the defibrillator and shout **'stand clear—charging'.**
- Check that all staff have stepped back (*including yourself*) and that no-one is touching the patient or their bed by carefully looking.

- ▸ Ensure high flow oxygen has also been removed.
- ▸ Check the monitor again to ensure a shockable rhythm.
- Shout **'stand clear—shocking'** and press both discharge buttons simultaneously to discharge the shock.
- Follow the protocol overleaf—return the paddles to the defibrillator before continuing with CPR.

Procedure for AEDs (Fig. 17.43)

- Switch on the defibrillator, ensure the skin is dry and free of excess hair and attach the electrode pads (same position as the gel pads in manual defibrillation opposite).
- Continue CPR while this is done if more than one assistant present.
- Make sure no one is touching the patient during ECG analysis by the AED.
- Follow the voice prompts.
  - These are usually programmable and the Resuscitation Council (UK) recommends that they be set as follows:
  - A single shock only when a shockable rhythm is detected.
  - No rhythm, breathing, or pulse check after the shock.
  - A voice prompt for immediate resumption of CPR after the shock.
  - Two minutes allowed for CPR using a ratio of 30 compressions to 2 rescue breaths before a voice prompt to assess the rhythm, breathing, or a pulse is given.
- ▸ If a shock is indicated shout **'stand clear'** and perform visual checks to ensure no personnel are in contact with the patient or their bed and that any oxygen has been removed.
- Push the shock button and continue as directed.

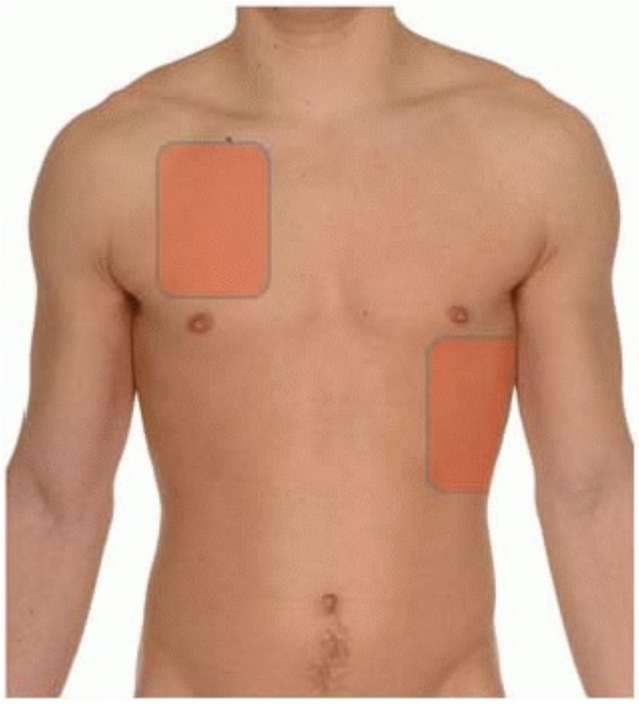


Fig. 17.41 Correct position of the gel pads or AED electrodes on the patient. Ensure that they are not touching or overlying any wires, oxygen tubing or any other conducting material. Ensure that the patient's chest is dry and shaved if particularly hairy.

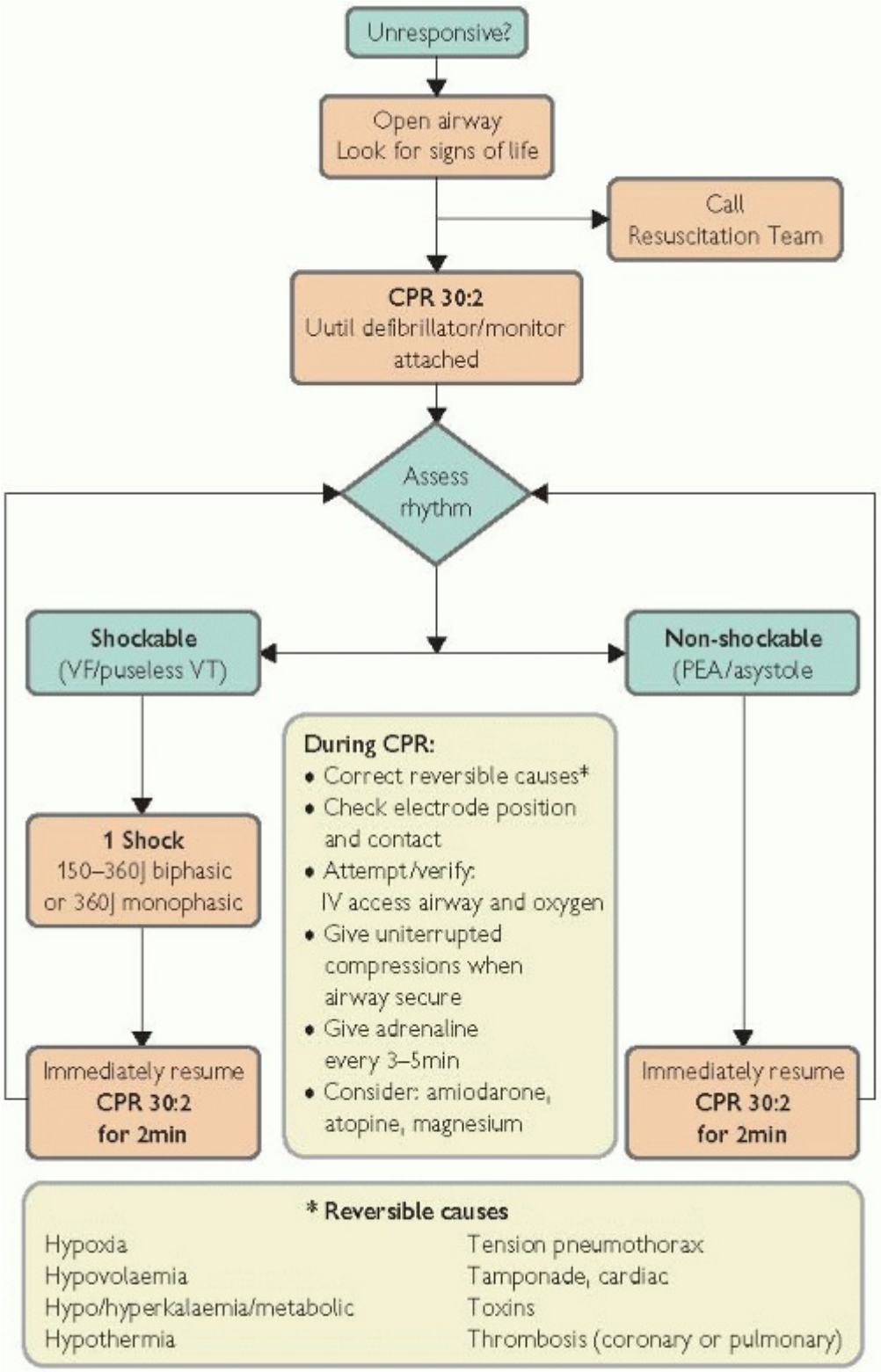


Fig. 17.42 Algorithm for advanced life support using a manual defibrillator. Reproduced with permission from the Resuscitation Council (UK) guidelines.

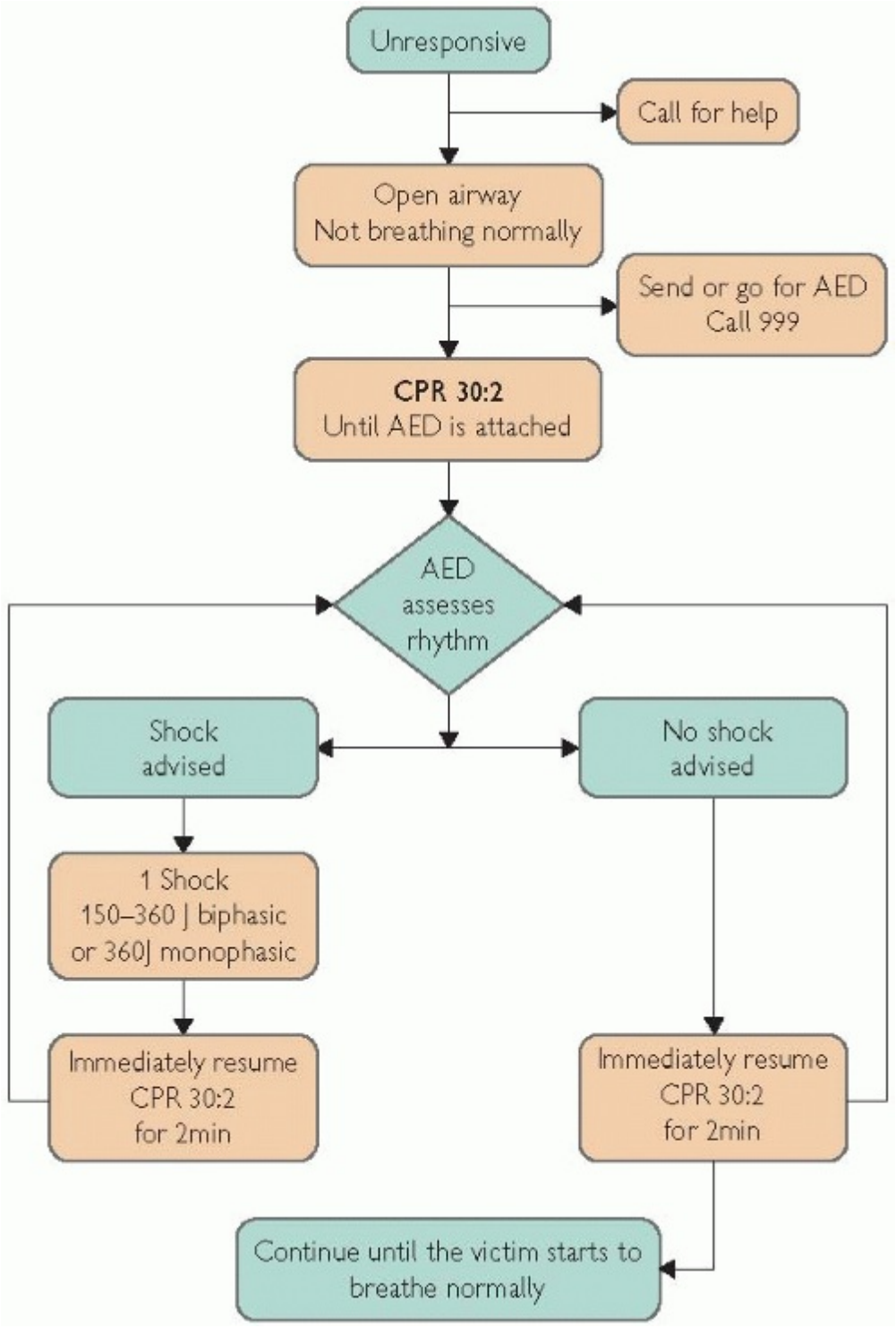


Fig. 17.43 AED algorithm. Reproduced with permission from the Resuscitation Council (UK) guidelines.

Knee joint aspiration

Theory

In the context of a swollen joint, a joint aspiration is performed for both diagnostic (to identify infectious and crystal arthropathies) and therapeutic (to relieve tense effusions and haemarthroses) purposes.

A sample of fluid may be removed and sent for microscopy, culture and sensitivity in addition to being examined for crystals under polarized light. See table showing important diagnostic features of joint aspirates in Chapter 18.

This same procedural approach may be used for joint injections (e.g. steroids and local anaesthetic to suppress inflammation).

Equipment

- 5-10ml 1% lidocaine.
- 1 × 20ml syringe.
- 1 × 5ml syringe.
- 1 × 21-gauge (green) needle.
- 1 × 25-gauge (orange) needle.
- Antiseptic solution.
- Sterile bottles.
- Sterile gloves.
- Dressing pack with cotton balls.

Procedure

- Introduce yourself, confirm the identity of the patient, explain the procedure, and obtain verbal consent.
- Ensure that the patient is relaxed and lying comfortably on the couch or bed with the knee exposed and slightly flexed.
- Palpate the outline of the patella and the medial joint line (aspiration is easier on the medial side).
- Wash your hands and don a pair of sterile gloves.
- Clean the site with the cotton balls and antiseptic solution.
- Infiltrate the insertion site with local anaesthetic. Use 1-2ml of 1% lidocaine using the 25-gauge orange needle and a 5ml syringe. Remember to aspirate before injecting.
- Take a 20ml syringe and attach a 21-gauge green needle.
- Insert the needle at an angle of around 45° in the gap between the lower border of the patella and the medial joint line.
- If the needle is in the joint space (about 2cm in), you should be able to freely aspirate synovial fluid. Aspiration can be aided by pressing on the opposite side of the joint with your free hand.
- Once the syringe is full, remove it from the joint and transfer the fluid into sterile specimen bottles.
- Send to microbiology for microscopy, culture and sensitivity and a further sample to the biochemistry department to look for crystals.
- Following aspiration, ask the patient to rest the knee for 24-48 hours.
- Record in the notes the procedural details including the colour of the synovial fluid and the investigations requested.

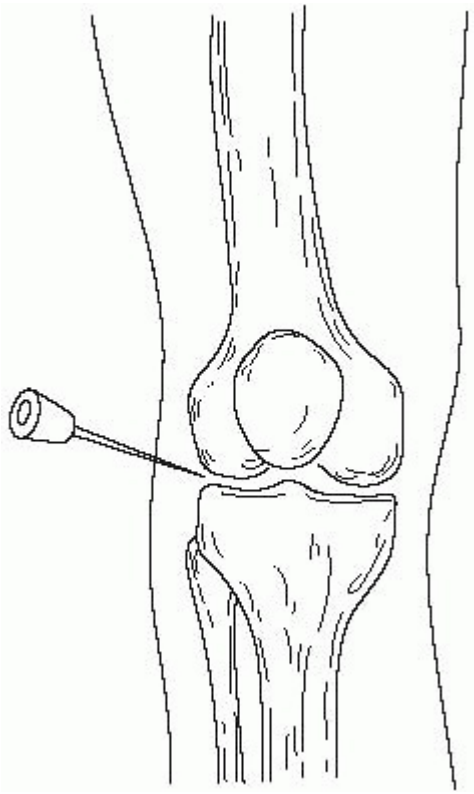


Fig. 17.44 Aspiration of the knee joint—insert the needle at about 45° heading distally below the patella.

Editors: Thomas, James; Monaghan, Tanya
Title: *Oxford Handbook of Clinical Examination and Practical Skills, 1st Edition*

Chapter 18

Data Interpretation

The ECG: an introduction

The first step in making sense of an electrocardiogram (ECG) printout is to understand the electrical conduction process in the normal heart.

Electrophysiology of the heart

Cardiac myocytes

In their resting state, the surface of cardiac myocytes (muscle cells) is polarized with a potential difference of ~90mV across the cell membrane (negatively charged intracellularly and positively charged extracellularly).

Depolarization (reversal of this charge) results in movement of calcium ions across the cell membranes and subsequent cardiac muscle contraction. It is this change in potential difference that can be detected by the ECG electrodes and represented as deflections on a tracing.

The basics of the tracing

It is easiest to imagine an electrode 'looking' at the heart from where it is attached to the body.

Depolarization of the myoctes that spreads towards the electrode is seen as an upwards deflection, electrical activity moving away from the electrode is seen as a downwards deflection and activity moving to one side but neither towards nor away from the electrode is not seen at all (see Fig. 18.1).

Electrical conduction pathway

In the normal heart, pacemaker cells in the sinoatrial (SA) node initiate depolarization. The depolarization first spreads through the atria and this is seen as a small upward deflection (the 'P' wave) on the ECG.

The atria and the ventricles are electrically isolated from each other. The only way in which the impulse can progress from the atria to the ventricles normally is through the atrioventricular (AV) node. Passage through the AV node slows its progress slightly. This can be seen on ECG as the isoelectric interval between the P wave and QRS complex, the 'PR interval'.

Depolarization then continues rapidly down the rapidly conducting Purkinje fibres—bundle of His, then down left and right bundle branches—to depolarize both ventricles (see Fig. 18.2). The left bundle has two divisions (fascicles). The narrow QRS complex on ECG shows this rapid ventricular depolarization.

Repolarization of the ventricles is seen as the T wave. Atrial repolarization causes only a very slight deflection which is hidden in the QRS complex and not seen.

▸ The P wave and QRS complex show the electrical depolarization of atrial and ventricular myocardium respectively, but the resultant mechanical muscle contraction—which usually follows—cannot be inferred from the ECG trace (e.g. in pulseless electrical activity (PEA)).

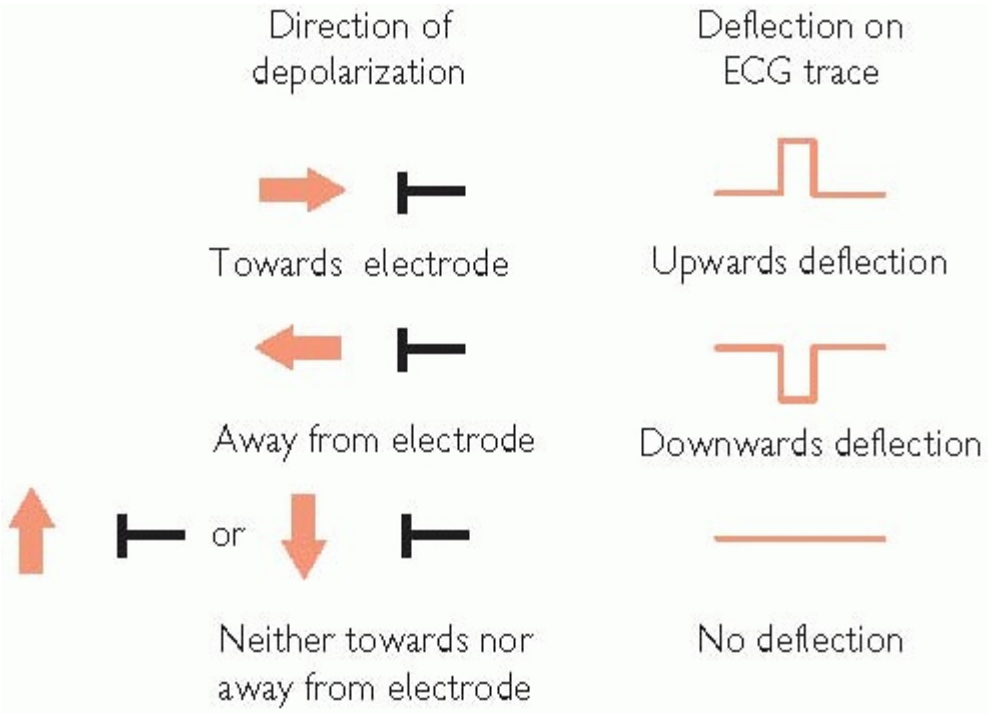


Fig. 18.1 Diagrammatic representation of how the electrodes monitor waves of depolarization.

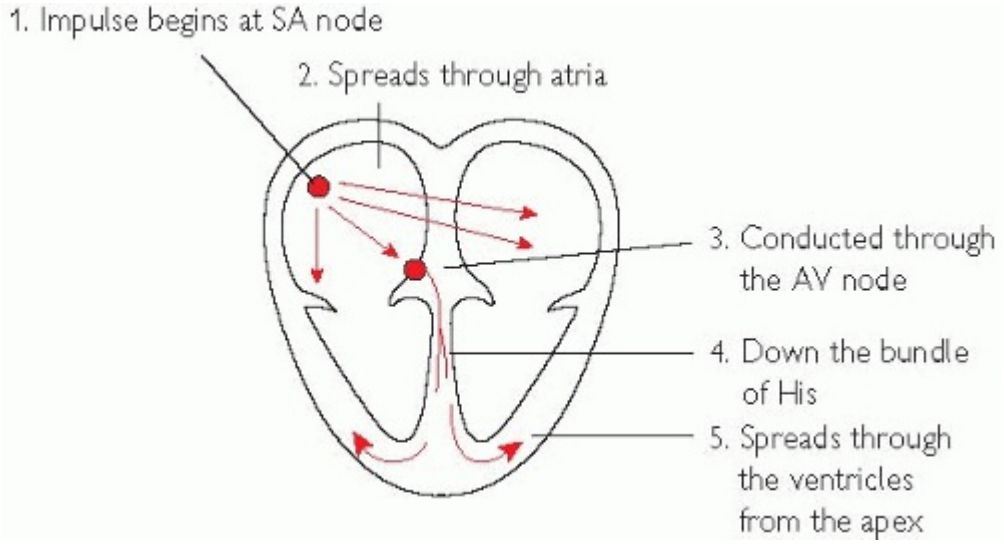


Fig. 18.2 Electrical conduction pathway in the normal heart.

The 12-lead ECG

Leads

Electrodes are placed on the limbs and chest for a '12-lead' recording. The term '12-lead' relates to the number of directions that the electrical activity is recorded from and is *not* the number of electrical wires attached to the patient!

The 6 chest leads (V_{1-6}) and 6 limb leads (I, II, III, aVR, aVL, aVF) comprise the 12-lead ECG. These look at the electrical activity of the heart from various directions. The chest leads correspond directly to the 6 electrodes placed at various points on the anterior and lateral chest wall (see Fig. 18.3). However, the 6 limb leads represent the electrical activity as 'viewed' using a combination of the 4 electrodes placed on the patient's limbs—e.g. lead I is generated from the right and left arm electrodes.

▸ Remember there are 12 ECG leads—12 different views of the electrical activity of the heart—but only 10 actual electrodes placed on the patient's body.

Additional leads can be used (e.g. V_{7-9} extending laterally around the chest wall) to look at the heart from further angles such as in suspected posterior myocardial infarction (MI).

ECG orientation

When a wave of myocardial depolarization flows towards a particular lead, the ECG tracing shows an upwards deflection. A downward deflection represents depolarization moving away from that lead. The key to interpreting the 12-lead ECG is therefore to remember the directions at which the different leads view the heart.

The 6 limb leads look at the heart in the coronal plane (see Fig. 18.4).

- aVR looking at the right atrium.*
- aVF, II and III view the inferior or diaphragmatic surface of the heart.
- I and aVL examining the left lateral aspect.

The 6 chest leads examine the heart in a transverse plane…

- V_1 and V_2 looking at the right ventricle.
- V_3 and V_4 at the septum and anterior aspect of the left ventricle.
- V_5 and V_6 at the anterior and lateral aspects of the left ventricle.

Although each of the 12 leads gives a different view of the electrical activity of the heart, for simplicity's sake when considering the standard ECG trace we can describe the basic shape common to all leads (see Fig. 18.5).

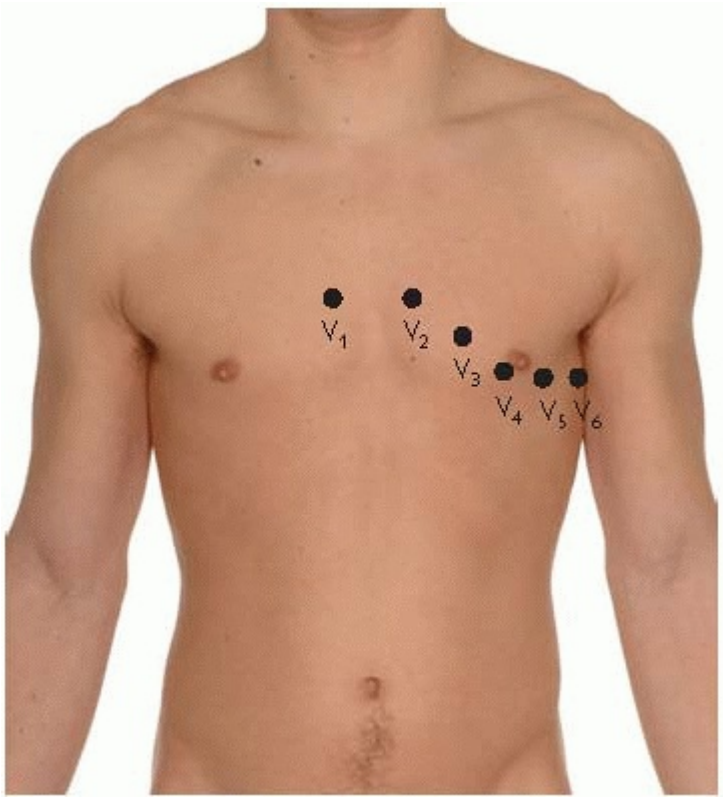


Fig. 18.3 V_{1-6} electrode placement on the chest wall.

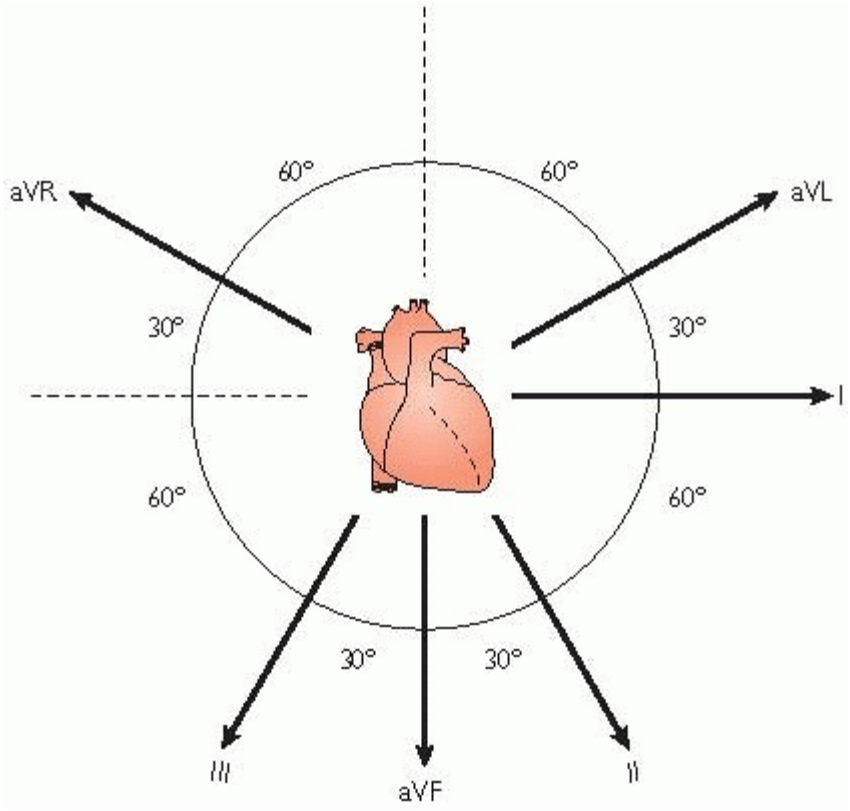


Fig. 18.4 The respective views of the heart of the 6 limb leads. Note the angles between the direction of the limb-leads—these become important when calculating the cardiac axis.

* All the vectors in lead aVR with be negative in the normal ECG.

The ECG trace

Waves

- P wave represents atrial depolarization and is a positive (upwards) deflection—except in aVR.
- QRS complex represents ventricular depolarization and comprises…
  - Q wave—so called if the first QRS deflection is negative (downwards). Pathological Q waves are seen in infarction— p.669.
  - R wave—the first positive (upwards) deflection—may or may not follow a Q wave.
  - S wave—a negative (downwards) deflection following the R wave.
- T wave represents ventricular repolarization and is normally a positive (upwards) deflection—concordant with the QRS complex.

Remember Q waves appear following myocardial infarction (MI) so should be absent from the normal ECG trace.

Rate

- The heart rate can be calculated by dividing 300 by the number of large squares between each R wave (with machine trace running at the standard speed of 25mm/sec and deflection of 1cm/10mV).
  - 3 large squares between R waves = rate 100.
  - 5 large squares = rate 60.
- Normal rate 60-100 beats/minute.
  - Rate <60 = bradycardia.

- Rate >100 = tachycardia.

Intervals and timing

- *PR interval:* from the start of the P wave to the start of the QRS complex. This represents the inbuilt delay in electrical conduction at the atrioventricular (AV) node. Normally <0.20 seconds (5 small squares at standard recording speed).
- *QRS complex:* the width of the QRS complex. Normally <0.12 seconds (3 small squares at standard rate).
- *R-R interval:* from the peak of one R wave to the next. This is used in the calculation of heart rate (see above).
- *QT interval:* from the start of the QRS complex to the end of the T wave. Varies with heart rate. Corrected QT = QT/square root of the R-R interval. Corrected QT interval should be 0.38-0.42 seconds.

Rhythm

- Is the rhythm (and the time between successive R waves) regular or irregular?
  - If irregular but in a clear pattern, then it is said to be 'regularly irregular' (e.g. types of heart block—see p.654).
  - If irregular but no pattern, then it is said to be 'irregularly irregular' (e.g. atrial fibrillation).

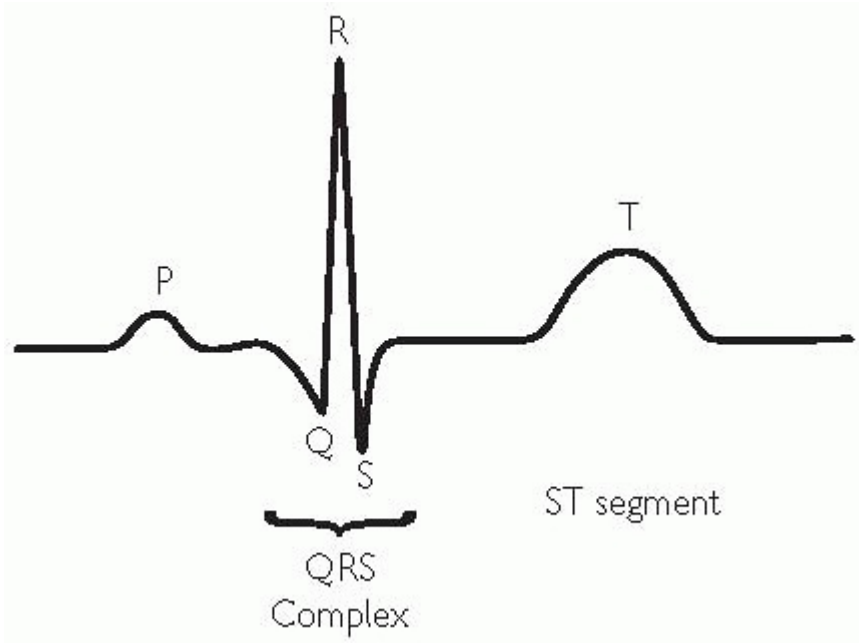


Fig. 18.5 The basic shape of the ECG trace.

ECG axis

Cardiac axis

The cardiac axis, or 'QRS axis', refers to the overall direction of depolarization through the ventricular myocardium in the coronal plane.

Zero degrees is taken as to the horizontal line to the left of the heart (the right of your diagram).

The normal cardiac axis lies between -30 and +90 degrees (see Fig. 18.6). An axis outside of this range may suggest pathology, either congenital or acquired.

Cardiac axis deviation may be seen in healthy individuals with distinctive body shapes—right axis deviation if tall and thin, left axis deviation if short and stocky.

Calculating the axis

Look at Fig. 18.4. Leads I, II and III all lie in the coronal plane (along with aVR, aVL, and aVF). By calculating the relative depolarization in each of these directions, one can calculate the cardiac axis. To accurately determine the cardiac axis, you should use leads I, II, and III as described below. There are less reliable short cuts, however.

- Draw a diagram like the one opposite showing the 3 leads—be careful to use the correct angles.
- Look at the ECG lead I. Count the number of mm above the baseline that the QRS complex reaches.
- Subtract from this the number of mm below the baseline that the QRS complex reaches.
- Now measure this number of centimetres along line I on your diagram and make a mark (measure backward for negative numbers).
- Repeat this for leads II and III.
- Extend lines from your marks, perpendicular to the leads (see Fig. 18.6).
- The direction from the centre of the diagram to the point at which all these lines meet is the cardiac axis.

Calculating the axis—short cuts

There are many shorter ways of roughly calculating the cardiac axis. These are less accurate, however.

An easy method is to look at only leads I and aVF. These are perpendicular to each other and make a simpler diagram than the one described above.

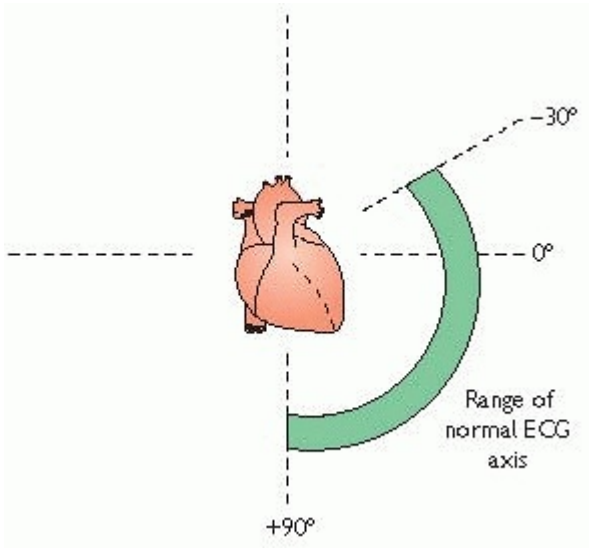


Fig. 18.6 The normal ECG axis.

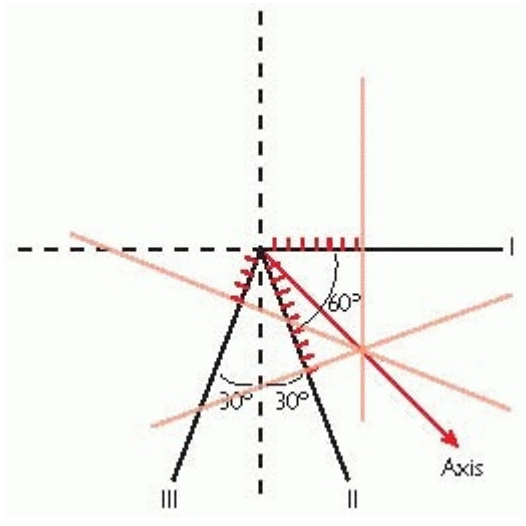


Fig. 18.7 Calculating the ECG axis using leads I, II and III. See text opposite.

Box 18.1 Causes of axis deviation

Left axis deviation (< -30 degrees)

- Left ventricular hypertrophy.
- Left bundle branch block (LBBB).
- Left anterior hemiblock (anterior fascicle of the left bundle).
- Inferior MI.
- Cardiomyopathies.
- Tricuspid atresia.

Right axis deviation (> +90 degrees)

- Right ventricular hypertrophy.
- Right bundle branch block (RBBB).
- Anterolateral MI.
- Right ventricular strain (e.g. pulmonary embolism).
- Cor pulmonale.
- Fallot's tetralogy (pulmonary stenosis).

Atrioventricular (AV) conduction abnormalities

In the normal ECG each P wave is followed by a QRS complex. The isoelectric gap between is the PR interval and represents slowing of the impulse at the AV junction. Disturbance of the normal conduction here, leads to 'heart block'.

Causes of heart block include ischaemic heart disease, idiopathic fibrosis of conduction system, cardiomyopathies, inferior and anterior MI, drugs: digoxin, ß-blockers, verapamil, and physiological (1^{st} degree) in athletes.

First degree heart block

PR interval fixed but prolonged at >0.20 seconds (5 small squares at standard rate). See rhythm-strip 1 (Fig. 18.8).

Second degree heart block

Not every P wave is followed by a QRS complex.

- *Möbitz type I:* PR interval becomes progressively longer after each P wave until an impulse fails to be conducted at all. The interval then returns to the normal length and the cycle is repeated (rhythm strip 2 (Fig. 18.8)). This is also known as the Wenckebach phenomenon.
- *Möbitz type II:* PR interval is fixed but not every P wave is followed by a QRS. The relationship between P waves and QRS complex may be 2:1 (2 P waves for every QRS), 3:1 (3 P waves per QRS), or random. See rhythm strip 3 (Fig. 18.8).

Third degree heart block

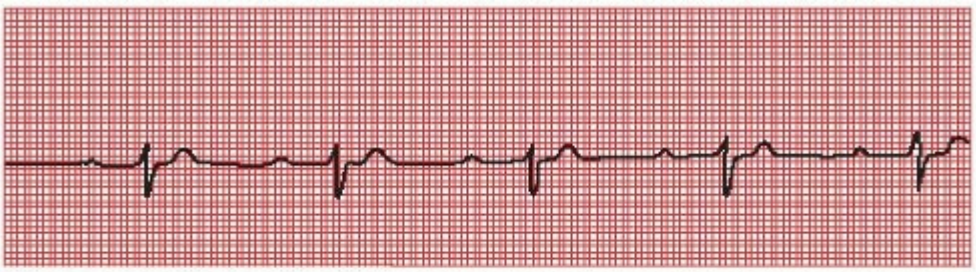
Also called complete heart block. See rhythm strip 4 (Fig. 18.8). There is no conduction of the impulse through the AV junction. Atrial and ventricular depolarization occur independent of one another. Each has a separate pacemaker triggering electrical activity at different rates (rhythm strip 4).

- The QRS complex is an abnormal shape as the electrical impulse does not travel through the ventricles via the normal routes (see ventricular escape).

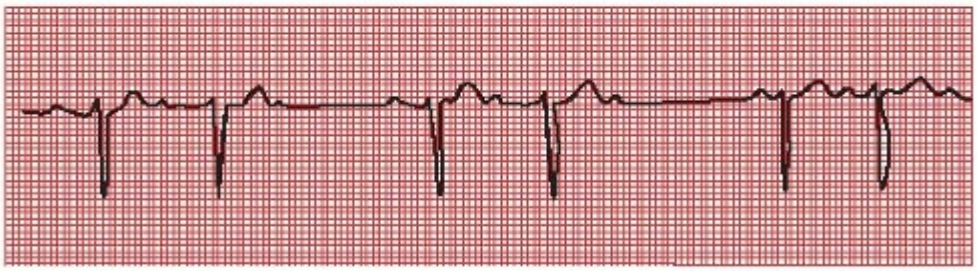
In third degree heart block P waves may be seen 'merging' with QRS complexes if they coincide.

If in doubt about the pattern of P waves and QRS complexes, mark out the P wave intervals and the R-R intervals separately, then compare.

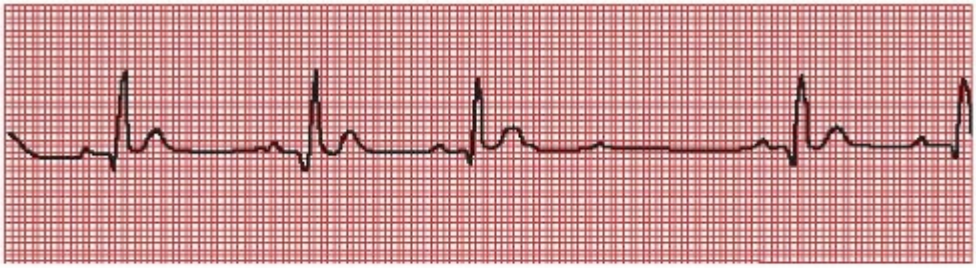
▸ P waves are best seen in leads II and V_1.



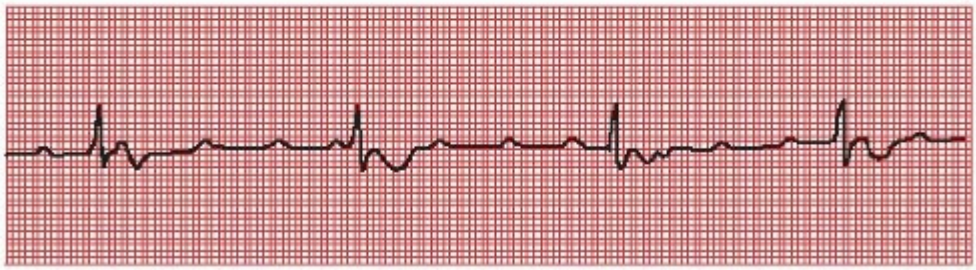
Rhythm strip 1—first degree heart block.



Rhythm strip 2—second degree heart block Möbitz type I.



Rhythm strip 3—second degree heart block Möbitz type II.



Rhythm strip 4—third degree (complete) heart block.

Fig. 18.8 Rhythm strips showing AV conduction abnormalities.

Ventricular conduction abnormalities

Depolarization of both ventricles usually occurs rapidly through left and right bundle branches of the His-Purkinje system (see Fig. 18.9). If this process is disrupted as a result of damage to the conducting system, depolarization will occur more slowly through non-specialized ventricular myocardium. The QRS complex—usually <0.12 seconds duration—will become prolonged and is described as a 'broad'.

Right bundle branch block (RBBB)

Conduction through the AV node, bundle of His, and left bundle branch will be normal but depolarization of the right ventricle occurs by the slow spread of electrical current through myocardial cells. The result is delayed right ventricular depolarization giving a second R wave known as R' ('R prime').

RBBB suggests pathology in the right side of the heart but can be a normal variant.

ECG changes

- 'RSR' pattern seen in V_1.
- Cardiac axis usually remains normal unless left anterior fascicle is also blocked ('bifascicular block') which results in left axis deviation.
- T wave ↓ in anterior chest leads (V_1-V_3).

Some causes of RBBB

- Hyperkalaemia.
- Congenital heart disease (e.g. Fallot's tetralogy).
- Pulmonary embolus.
- Cor pulmonale.
- Fibrosis of conduction system.

Left bundle branch block (LBBB)

Conduction through the AV node, bundle of His, and right bundle branch will be normal but depolarization of the left ventricle occurs by the slow spread of electrical current through myocardial cells. The result is delayed left ventricular depolarization.

LBBB should always be considered pathological.

ECG changes

- 'M' pattern seen in V_6.
- T wave ↓ in lateral chest leads (V_5-V_6).

Some causes of LBBB

- Hypertension.
- Ischaemic heart disease.
- Acute MI.
- Aortic stenosis.
- Cardiomyopathies.
- Fibrosis of conduction system.

LBBB on the ECG causes abnormalities of the ST segment and T wave. You should not comment any further on these parts of the trace.

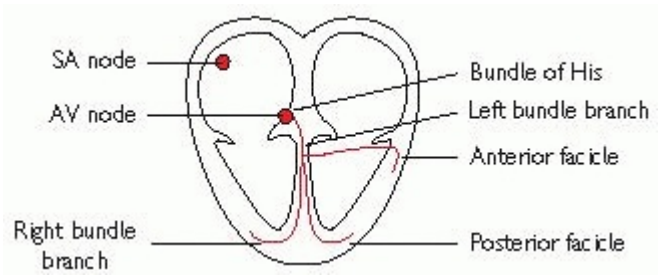


Fig. 18.9 Diagrammatic representation of the conducting system of the heart.

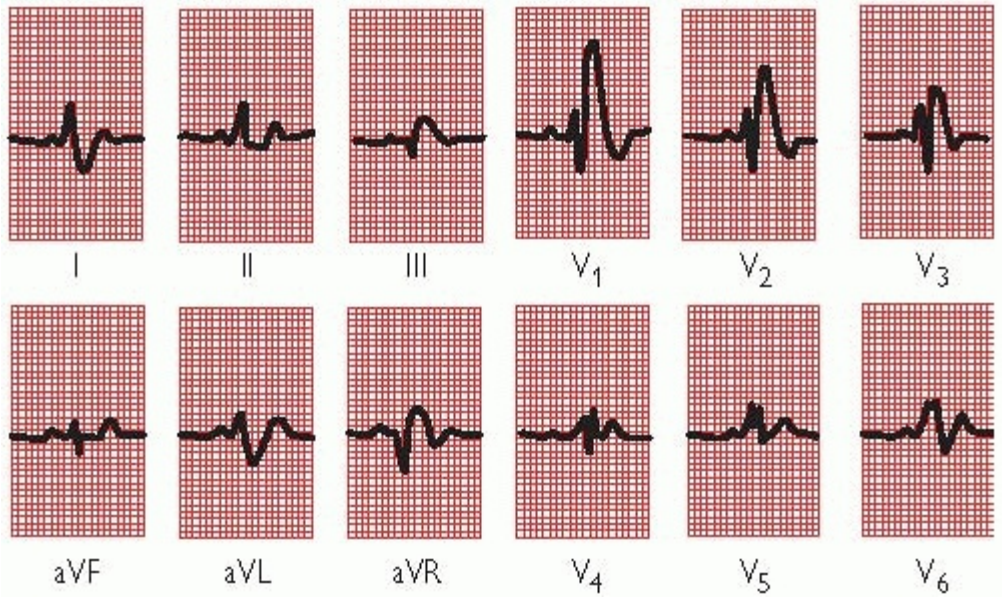


Fig. 18.10 Typical 12-lead ECG showing RBBB.

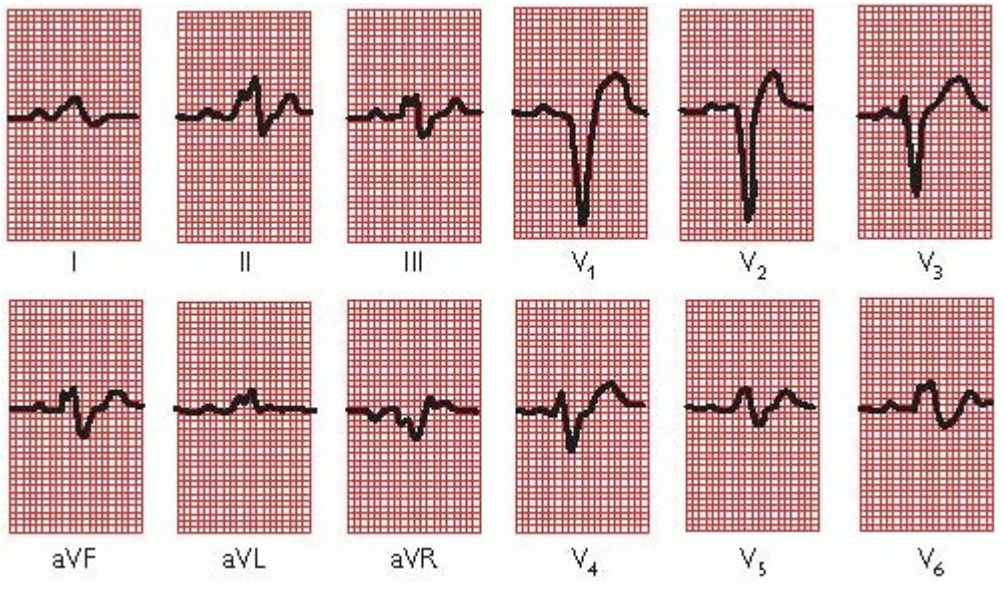


Fig. 18.11 Typical 12-lead ECG showing LBBB.

Box 18.2 Bundle branch block mnemonic

- In LBBB, the QRS complex in V_1 looks like a 'W' and an 'M' in V_6. This can be remembered as **'WiLLiaM'.** There is a W at the start, an M at the end and 'L' in the middle for 'left'.
- Conversely, in the case of RBBB, the QRS complex in V_1 looks like a 'M' and a 'W' in V_6. Combined with an 'R' for right, you have the word **'MaRRoW'**.

Sinus rhythms

Supraventricular rhythms arise in the atria. They may be physiological in the case of some causes of sinus brady- and tachycardia or may be caused by pathology within the SA node, the atria, or the first parts of the conducting system.

Normal conduction through the bundle of His into the ventricles will usually give narrow QRS complexes.

Sinus bradycardia

This is a bradycardia (rate <60 beats per minute) at the level of the SA node. The heart beats slowly but conduction of the impulse is normal.

Some causes of sinus bradycardia

- Drugs (ß-blockers, verapamil, amiodarone, digoxin).
- Sick sinus syndrome.
- Hypothyroidism.
- Inferior MI.
- Hypothermia.
- ↑ intracranial pressure.
- Physiological (athletes).

Sinus tachycardia

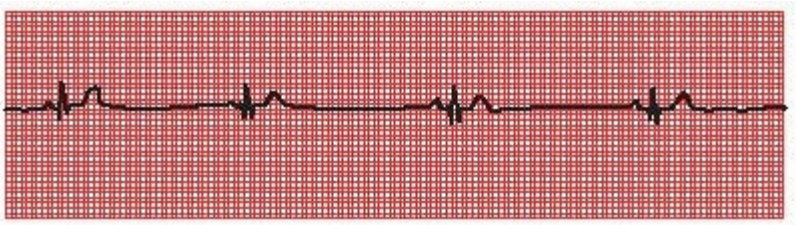
This is a tachycardia at the level of the SA node—the heart is beating too quickly but conduction of the impulse is normal. (Rhythm strip 1 (Fig 18.12)).

ECG features

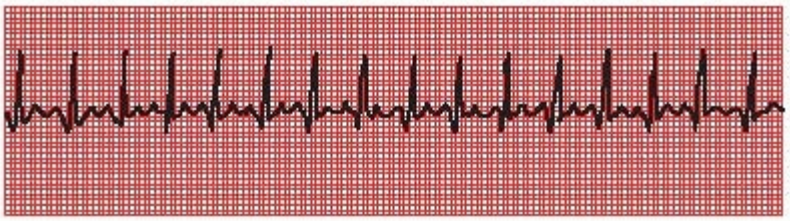
- Ventricular rate > 100 (usually 100-150 beats per minute).
- Normal P wave before each QRS.

Some causes of sinus tachycardia

- Drugs (epinephrine/adrenaline, caffeine, nicotine).
- Pain.
- Exertion.
- Anxiety.
- Anaemia.
- Thyrotoxicosis.
- Pulmonary embolus.
- Hepatic failure.
- Cardiac failure.
- Hypercapnia.
- Pregnancy.
- Constrictive pericarditis.



Rhythm strip 1—sinus bradycardia.



Rhythm strip 2—sinus tachycardia.

Fig. 18.12 Rhythm strips from lead II showing a sinus bradycardia (rhythm strip 1) and sinus tachycardia (rhythm strip 2).

Supraventricular tachycardias

These are tachycardias (rate >100bpm) arising in the atria or the AV node. As conduction through the bundle of His and ventricles will be normal (unless there is other pathology in the heart), the QRS complexes appear normal.

There are 4 main causes of a supraventricular tachycardia that you should be aware of: atrial fibrillation, atrial flutter, junctional tachycardia, and re-entry tachycardia. See *OHCM6*, p.128 for advice on clinical diagnosis and treatment.

Atrial fibrillation (AF)

This is disorganized contraction of the atria in the form of rapid, irregular twitching. There will, therefore, be no P waves on the ECG.

Electrical impulses from the twitches of the atria arrive at the AV node randomly, they are then conducted via the normal pathways to cause ventricular contraction. The result is a characteristic ventricular rhythm that is *irregularly irregular* with no discernable pattern.

ECG features

- No P waves. Rhythm is described as irregularly irregular.
- Irregular QRS complexes.
- Normal appearance of QRS.
- Ventricular rate may be ↑ ('fast AF')—typically 120-160 per minute.

Some causes of atrial fibrillation

- Idiopathic.
- Ischaemic heart disease.
- Thyroid disease.
- Hypertension.
- MI.
- Pulmonary embolus.
- Rheumatic mitral or tricuspid valve disease.

Atrial flutter

This is abnormally rapid contraction of the atria. The contractions are not disorganized or random, unlike AF, but are fast and inadequate for the normal movement of blood. Instead of P waves, the baseline will have a typical 'saw-tooth' appearance (sometimes known as F waves).

The AV node is unable to conduct impulses faster than ~200/min. Atrial contraction faster than that leads to impulses failing to be conducted. For example, an atrial rate of ~300/min will lead to every other impulse being conducted giving a ventricular rate (and pulse) of ~150/min. In this case, it is called '2:1 block'. Other ratios of atrial to ventricular contractions may occur.

A variable block at the AV node may lead to an irregularly irregular pulse indistinguishable from that of AF on clinical examination.

ECG features

- 'Saw-tooth' appearance of baseline.
- Normal appearance of QRS complexes.

Causes of atrial flutter

Similar to AF opposite.

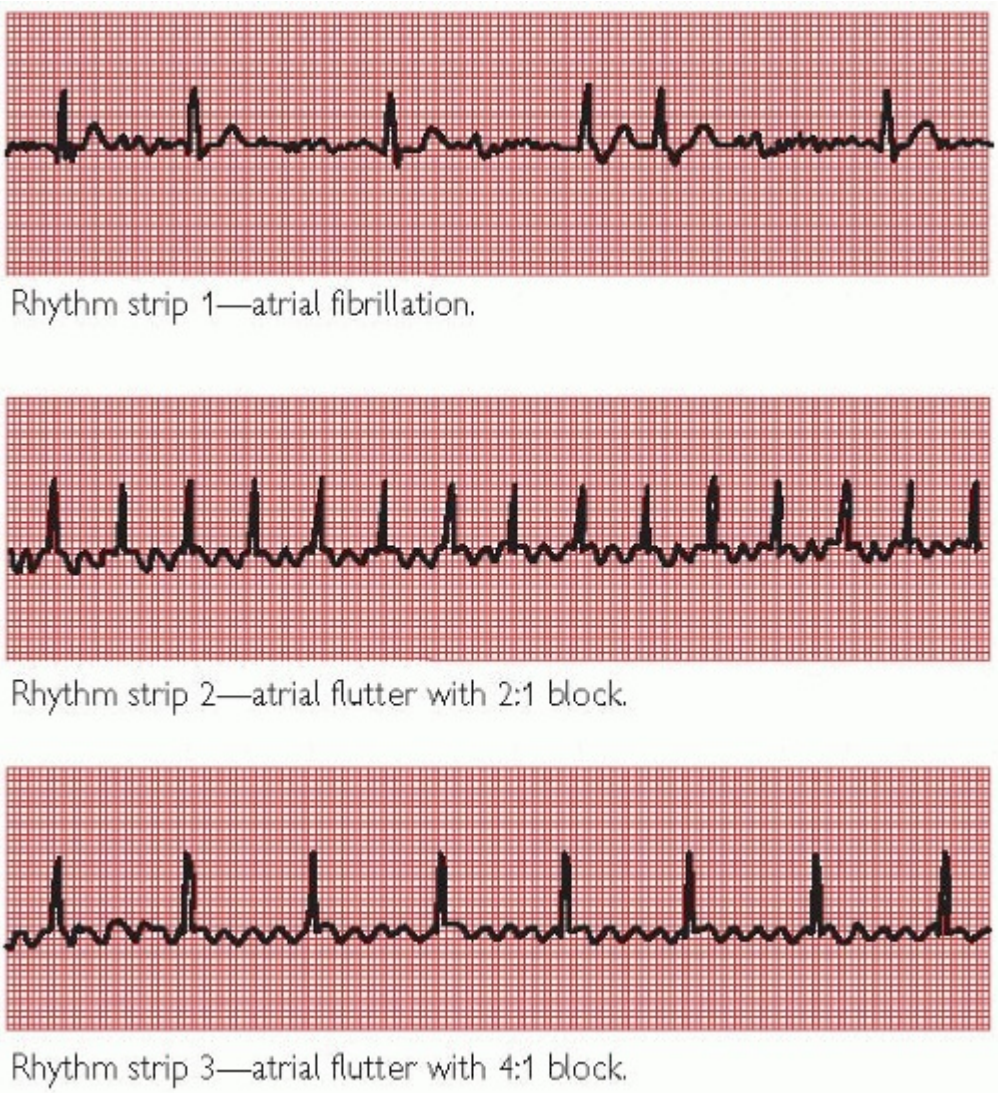


Fig. 18.13 Rhythm strips from lead II showing some supraventricular tachycardias.

Junctional (nodal) tachycardia

The area in or around the AV node depolarizes spontaneously, the impulse will be immediately conducted to the ventricles. The QRS complex will be of a normal shape but no P waves will be seen.

ECG features

- No P waves.
- QRS complexes are regular and normal shape.
- Rate may be fast or may be of a normal rate.

Some causes of junctional tachycardia

- Sick sinus syndrome (including drug-induced).
- Digoxin toxicity.
- Ischemia of the AV node, especially with acute inferior MI.
- Acutely after cardiac surgery.
- Acute inflammatory processes (e.g. acute rheumatic fever) which may involve the conduction system.
- Diphtheria.
- Other drugs (e.g. most anti-arrhythmic agents).

Wolff-Parkinson-White syndrome

In Wolff-Parkinson-White (WPW) syndrome, there is an extra conducting pathway between the atria and the ventricles (the bundle of Kent)—a break in the normal electrical insulation. This 'accessory' pathway is not specialized for conducting electrical impulses so does not delay the impulse as the AV node does. However, it is not linked to the normal conduction pathways of the bundle of His.

Depolarization of the ventricles will occur partly via the AV node and partly by the bundle of Kent. During normal atrial contraction, electrical activity reaches the AV node and the accessory pathway at roughly the same time. Whilst it is held up temporarily at the AV node, the impulse passes through the accessory pathway and starts to depolarize the ventricles via non-specialized cells ('pre-excitation'), distorting the first part of the R wave and giving a short PR interval. Normal conduction via the bundle of His then supervenes. The result is a slurred upstroke of the QRS complex called a 'delta wave' (see Figs. 18.14 and 18.15).

This is an example of a 'fusion beat' in which normal and abnormal ventricular depolarization combine to give a distortion of the QRS complex.

▸ *Re-entry tachycardia*

The accessory pathway may allow electrical activity to be conducted from the ventricles back up to the atria.

For example, in a re-entry tachycardia, electrical activity may be conducted down the bundle of His, across the ventricles and *up the accessory pathway* into the atria causing them to contract again…and the cycle is repeated. This is called a 're-entry circuit'.

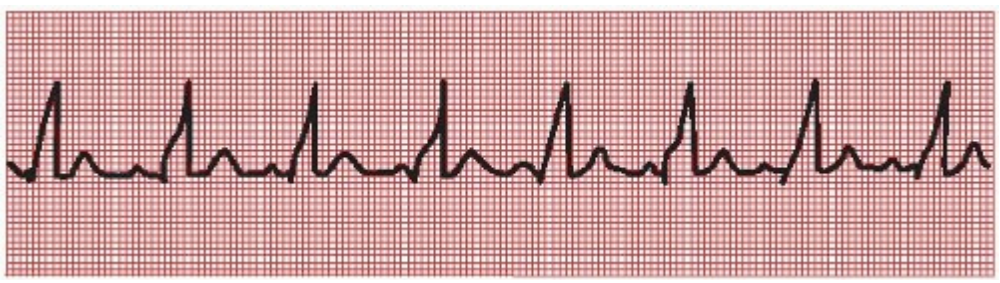


Fig. 18.14 Rhythm strip showing WPW syndrome.

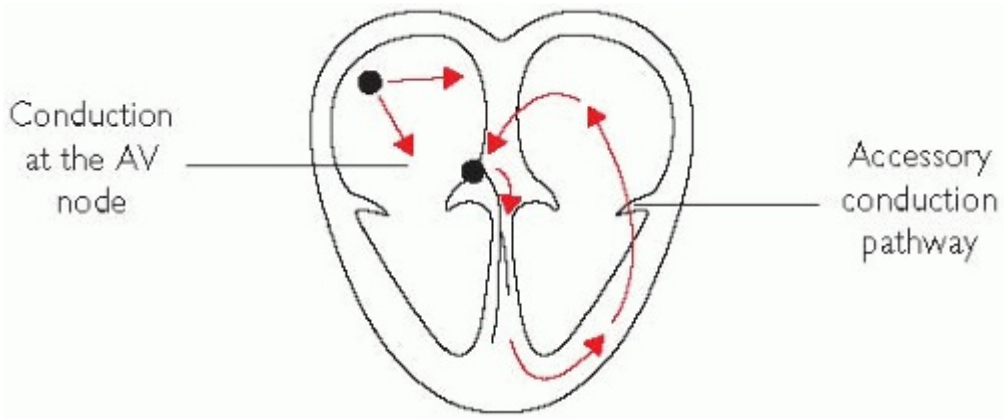


Fig. 18.15 Diagrammatic representation of re-entry tachycardia.

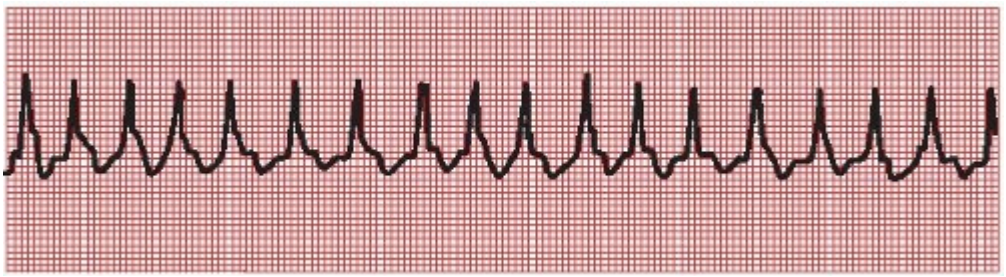


Fig. 18.16 Rhythm strip showing a re-entry tachycardia.

Box 18.3 Classification of Wolff-Parkinson-White syndrome

The bundle of Kent may connect the atria with either the right or the left ventricle. Thus, WPW is classically divided into 2 groups according to the resulting appearance of the QRS complex in the anterior chest leads. In practice, this classification is rather simplistic as 11% of patients may have more than one accessory pathway.

- ***Type A:*** upright delta wave and QRS in V_1.
  - May be mistaken for RBBB or posterior MI.
- ***Type B:*** downward delta wave and QRS in V_1, positive elsewhere.
  - May be mistaken for LBBB or anterior MI.

Ventricular rhythms

Most ventricular rhythms originate outside the usual conduction pathways meaning that excitation spreads by an abnormal path through the ventricular muscle to give broad or unusually shaped QRS complexes.

See *OHCM6*, p. 132 for advice on diagnosis and management.

Ventricular tachycardia (VT)

Here, there is a focus of ventricular tissue depolarizing rapidly within the ventricular myocardium. VT is defined as 3 or more successive ventricular extrasystoles at a rate of >120/min. ‘Sustained’ VTs last for >30 secs.

VT may be ‘stable’ showing a repetitive QRS shape (‘monomorphic’) or unstable with varying patterns of the QRS complex (‘polymorphic’).

It may be impossible to distinguish VT from an SVT with bundle branch block on a 12-lead ECG.

ECG features

- Wide QRS complexes which are irregular in rhythm and shape.
- A-V dissociation—independent atrial and ventricular contraction.
- May see fusion and capture beats on ECG as signs of atrial activity independent of the ventricular activity—said to be pathognomonic.
  - **Fusion beats:** depolarization from AV node meets depolarization from ventricular focus causing hybrid QRS complex.
  - **Capture beats:** atrial beat conducted to ventricles causing a normal QRS complex in amongst the VT trace.
- Rate can be up to 130-300/min.
- QRS concordance: all the QRS complexes in the chest leads are either mainly positive or mainly negative—this suggests a ventricular origin of the tachycardia.
- Extreme axis deviation (far negative or far positive).

Some causes of ventricular tachycardia

- Ischemia (acute including MI or chronic).
- Electrolyte abnormalities (↓ K, ↓ Mg).
- Aggressive adrenergic stimulation (e.g. cocaine use).
- Drugs—especially anti-arrhythmics.

Ventricular fibrillation (VF)

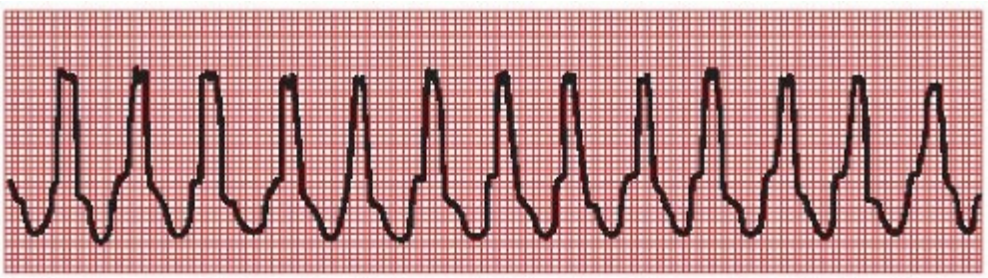
This is disorganized, uncoordinated depolarization from multiple foci in the ventricular myocardium.

ECG features

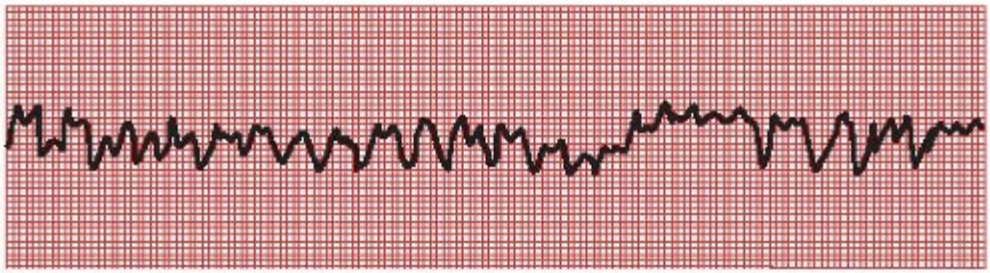
- No discernible QRS complexes.
- A completely disorganized ECG.

Some causes of ventricular fibrillation

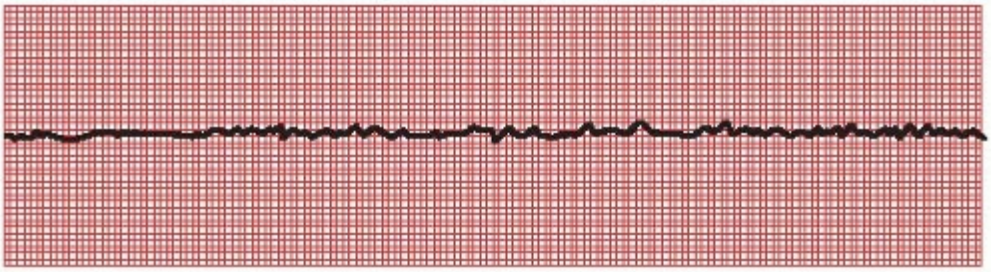
- Coronary heart disease.
- Cardiac inflammatory diseases.
- Abnormal metabolic states.
- Pro-arrhythmic toxic exposures.
- Electrocution.
- Tension pneumothorax, trauma, and drowning.
- Large pulmonary embolism.
- Hypoxia or acidosis.



Rhythm strip 1—monomorphic ventricular tachycardia (VT).



Rhythm strip 2—ventricular fibrillation (VF).



Rhythm strip 3—'fine' ventricular fibrillation.

Fig. 18.17 Rhythm strips showing ventricular rhythms.

Box 18.4 Fine VF

This is VF with a small amplitude waveform. It may resemble asystole on the ECG monitor (see Fig. 18.19), particularly in an emergency situation.

In a clinical situation, you should remember to increase the gain on the monitor to ensure what you think is asystole is not really fine VF as the managment for each is very different.

Other ventricular rhythms

Ventricular extrasystoles (ectopics)

These are ventricular contractions originating from a focus of depolarization within the ventricle. As conduction is via abnormal pathways, the QRS complex will be unusually shaped. See Fig. 18.19.

Ventricular extrasystoles are common and harmless if there is no structural heart disease. If they occur at the same time as a T wave, the 'R-on-T' phenomenon, they can lead to VF.

Ventricular escape rhythm

This occurs as a 'back-up' when conduction between the atria and the ventricles is interrupted (as in complete heart block).

The intrinsic pacemaker in ventricular myocardium depolarizes at a slow rate (30-40/min), see Fig. 18.19.

The ventricular beats with be abnormal and wide with abnormal T waves following them. This rhythm can be stable but may suddenly fail.

Asystole

This is a complete absence of electrical activity and is not compatible with life. See Fig. 18.19.

There may be a slight wavering of the baseline which can be easily confused with fine VF in emergency situations.

Agonal rhythm

This is a slow, irregular rhythm with wide ventricular complexes which vary in shape. This is often seen in the later stages of unsuccessful resuscitation attempts as the heart dies. The complexes become progressively broader before all recognisable activity is lost (asystole).

Box 18.5 Torsades de pointes

Torsades de pointes, literally meaning 'twisting of points', is a form of polymorphic VT characterized by a gradual change in the amplitude and twisting of the QRS axis. In the US, it is known as 'cardiac ballet'.

Torsades usually terminates spontaneously but frequently recurs and may degenerate into sustained VT and ventricular fibrillation.

Torsades results from a prolonged QT interval. Causes include congenital long-QT syndromes and drugs (e.g. anti-arrhythmics). Patients may also have ↓ K and ↓ Mg.

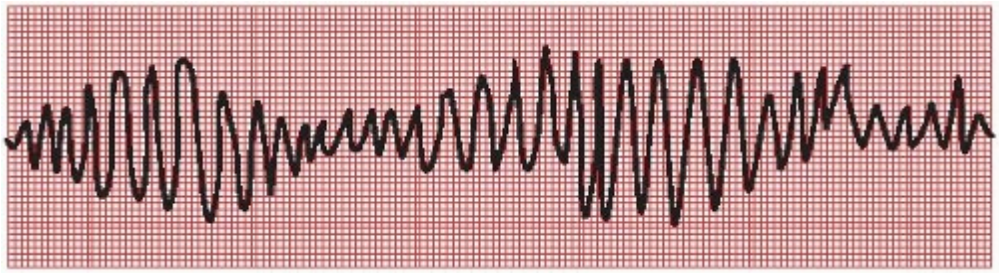
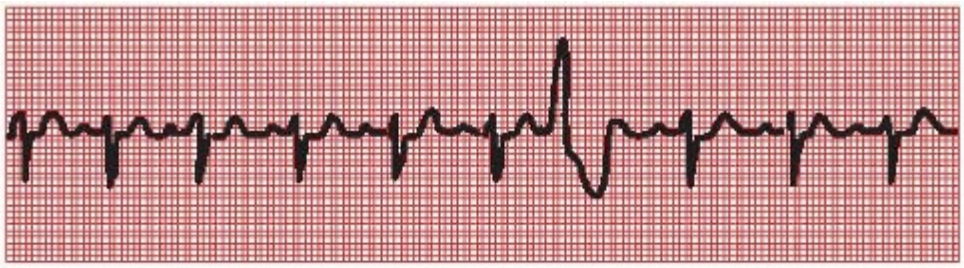
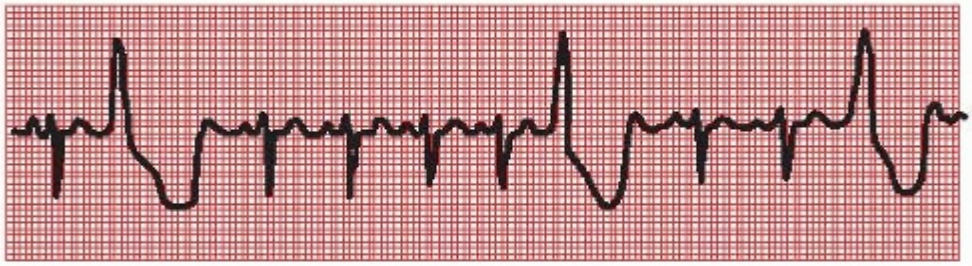


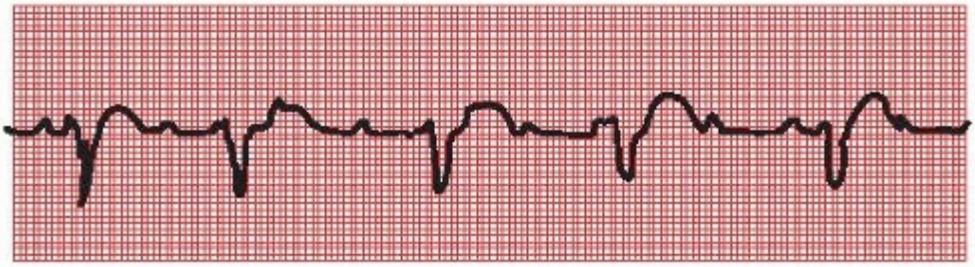
Fig. 18.18 Torsades de pointes. The axis of the QRS complex rotates—seen from a fixed position, there is a repeated change of amplitude.



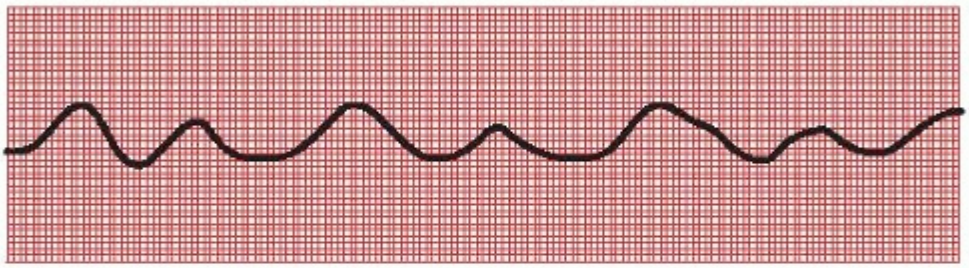
Rhythm strip 1—a single ventricular extrasystole.



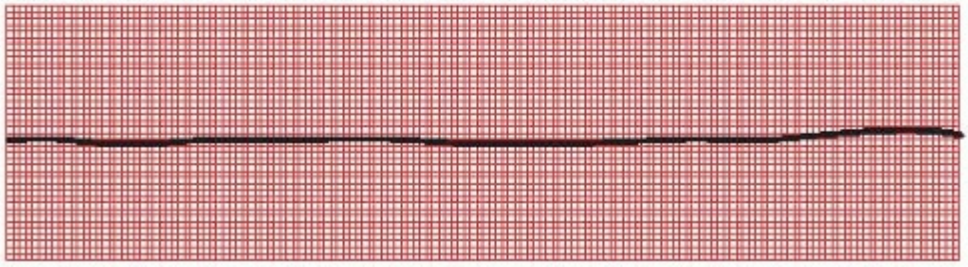
Rhythm strip 2—multiple, unifocal, ventricular extrasystole.



Rhythm strip 3—ventricular escape in the case of complete heart block.



Rhythm strip 4—agonal rhythm.



Rhythm strip 5—asystole.

Fig. 18.19 Rhythm strips showing some ventricular rhythms.

P and T wave abnormalities

The P wave

Represents depolarization of the small muscle mass of the atria. The P wave is thus much smaller in amplitude than the QRS complex.

Normal

- In sinus rhythm each P wave is closely associated with a QRS complex.
- P waves are usually upright in most leads except aVR.
- P waves are <3 small squares wide and <3 small squares high.

Abnormal

- Right atrial hypertrophy will cause tall, peaked P waves. Causes include pulmonary hypertension (in which case the wave is known as 'P pulmonale' and tricuspid valve stenosis).
- Left atrial hypertrophy will cause the P wave to become wider and twin-peaked or 'bifid'. This is usually caused by mitral valve disease—in which case the wave is known as 'P mitrale'.

The T wave

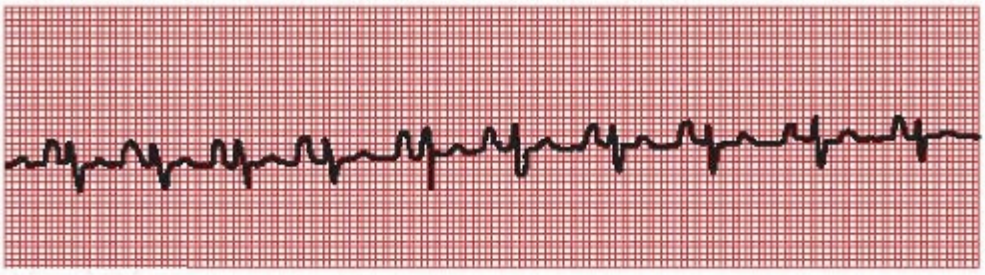
Represents repolarization of the ventricles. The T wave is most commonly affected by ischaemic changes. The most common abnormality is 'inversion' which has a number of causes.

Normal

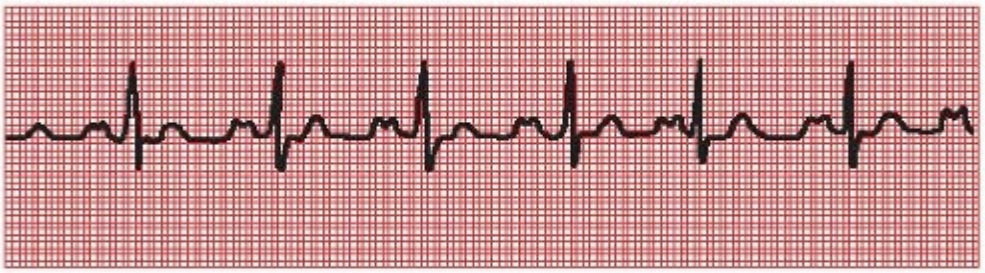
- Commonly inverted in V_1 and aVR.
- May be inverted in V1-V3 as normal variant.

Abnormal

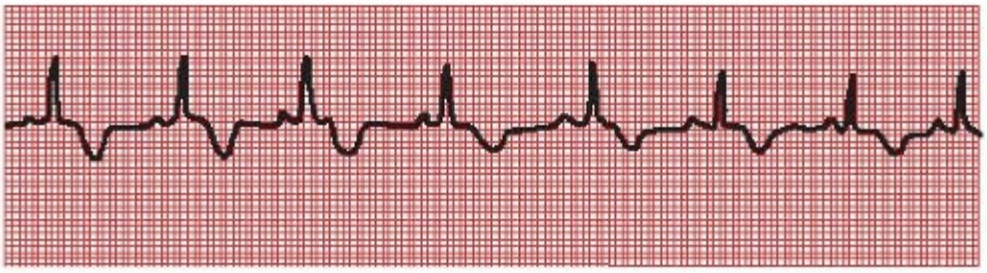
- Myocardial ischaemia or MI (e.g. non-Q wave MI) can cause T wave inversion. Changes need to be interpreted in light of clinical picture.
- Ventricular hypertrophy causes T inversion in those leads focused on the ventricle in question. For example, left ventricular hypertrophy will give T changes in leads V_5, V_6, II, and aVL.
- Bundle branch block causes abnormal QRS complexes due to abnormal pathways of ventricular depolarization. The corresponding abnormal repolarization gives unusually shaped T waves which have no significance in themselves.
- Digoxin causes a characteristic T wave inversion with a downsloping of the ST segment known as the 'reverse tick' sign. This occurs at therapeutic doses and is not a sign of digoxin toxicity. (See rhythm strip 4, Fig. 18.21).
- Electrolyte imbalances cause a number of T wave changes.
  - ↑ K can cause tall tented T waves.
  - ↓ K can cause small T waves and U waves (broad, flat waves occurring after the T waves).
  - ↓ Ca can cause small T waves with a prolongation of the QT interval. (↑ Ca has the reverse effect).
  - Other causes of T wave inversion include subarachnoid haemorrhage and lithium use.



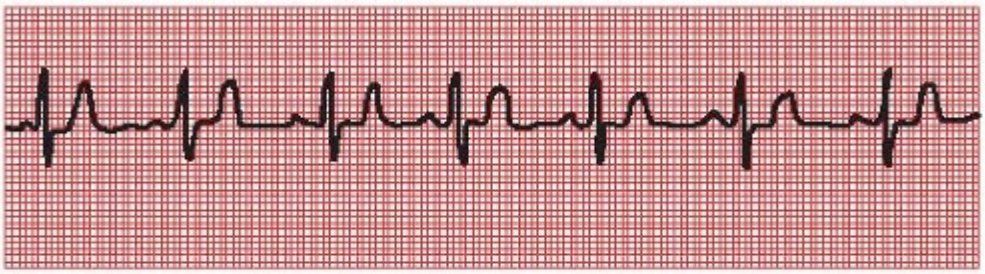
Rhythm strip 1—peaked P waves.



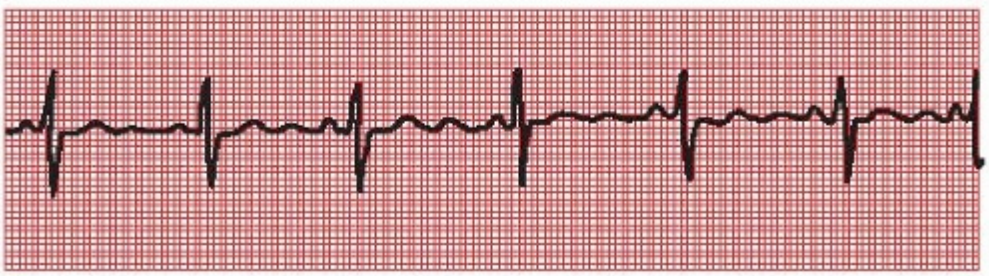
Rhythm strip 2—bifid P waves.



Rhythm strip 3—T wave inversion after myocardial infarction.



Rhythm strip 4—Hyperkalaemia with peaked T waves.



Rhythm strip 5—Hyporkalaemia with small T waves and U waves.

Fig. 18.20 Rhythm strips showing some P and T wave abnormalities.

The ST segment

This is the portion of the ECG from the end of the QRS complex to the start of the T wave and is an isoelectric line in the normal ECG (see Fig. 18.5, p.651.). Changes in the ST segment can represent myocardial ischaemia and, most importantly, acute MI.

ST elevation

The degree and extent of ST elevation is of crucial importance in ECG interpretation as it determines whether reperfusion therapy (thrombolysis or primary PCI) is considered in acute MI (see *OHCM6*, p.120).

Causes of ST elevation

- Acute MI—convex ST elevation in affected leads (the 'tomb-stone' appearance), often with reciprocal ST depression in opposite leads.
- Pericarditis—widespread concave ST elevation ('saddle-shaped').
- Left ventricular aneurysm—ST elevation may persist over time.

ST depression

ST depression can be horizontal, upward sloping, or downward sloping.

Causes of ST depression

- Myocardial ischaemia—horizontal ST depression and an upright T wave. May be result of coronary artery disease or other causes (e.g. anaemia, aortic stenosis).
- Digoxin toxicity—downward sloping ('reverse tick').
- 'Non-specific' changes—ST segment depression which is often upward sloping may be a normal variant and is not thought to be associated with any underlying significant pathology.

▸ In exercise ECG testing which looks to induce ischaemic changes, the extent, timing, and nature of ST changes can help quantify the probability of ischaemic heart disease (see *OHCM6*, p106).

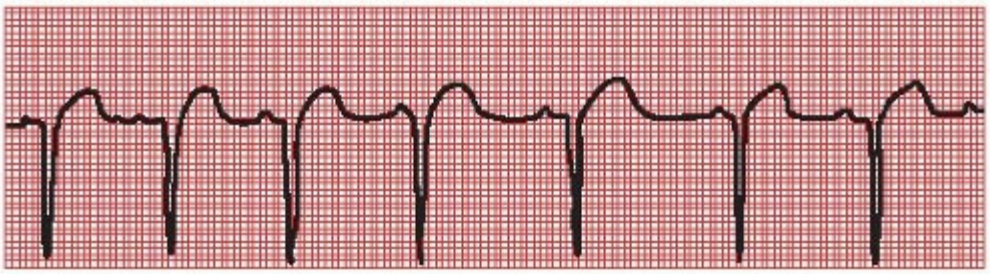
Myocardial infarction

In the first hour following a MI, the ECG can remain normal. However, when changes occur, they usually develop in the following order:

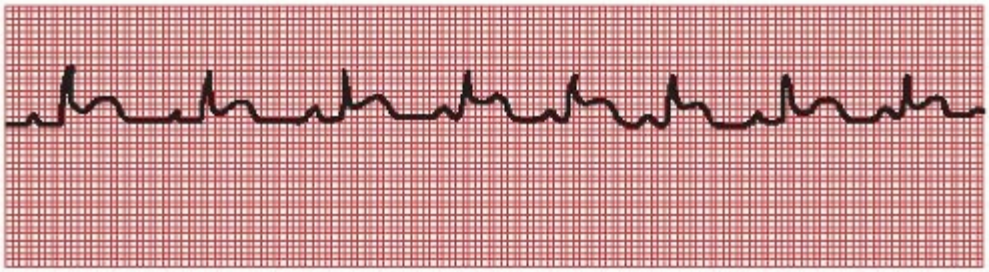
- ST segment becomes elevated and T waves become peaked.
- Pathological Q waves develop.
- ST segment returns to baseline and T waves invert.

The leads in which these changes take place allow you to identify which part of the heart has been affected and, therefore, which coronary artery is likely to be occluded.

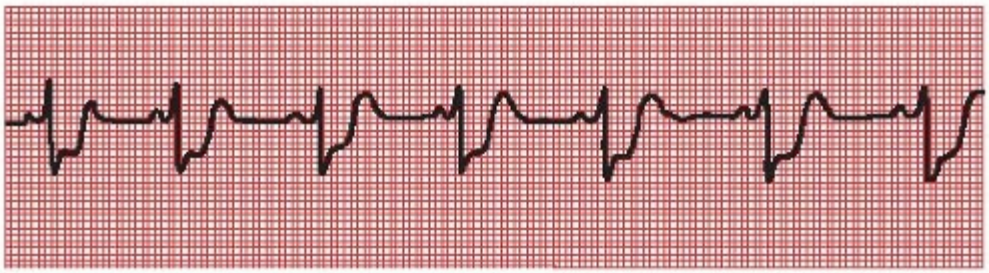
- ***Anterior:*** V_2-V_5.
- ***Antero-lateral:*** I, aVL, V_5, V_6
- ***Inferior:*** III, aVF (sometimes II also).
- ***Posterior:*** the usual depolarization of the posterior of the left ventricle is lost, giving a *dominant R wave in V1.* Imagine it as a mirror image of the Q wave you would expect with an anterior infarction.
- ***Right ventricular:*** often no changes on the 12-lead ECG. If suspected clinically, leads are placed on the right of the chest, mirroring the normal pattern and are labelled V_1 R, V_2R, V_3R, and so on.



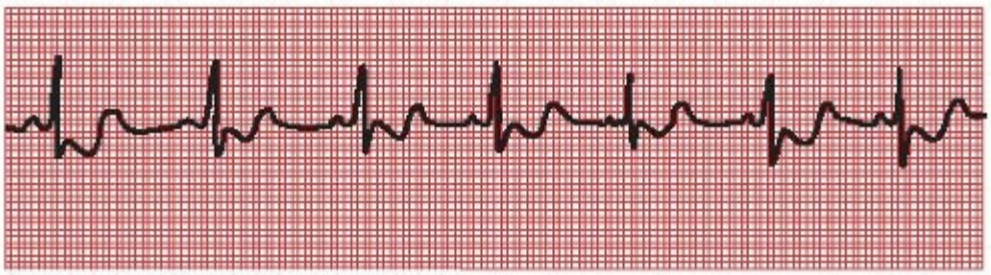
Rhythm strip—Lead V_2 showing acute myocardial infarction.



Rhythm strip—Pericarditis. The ST elevation is usually described as 'saddle-shaped'.



Rhythm strip—Ischaemia.



Rhythm strip—Digoxin use showing the 'reverse-tick'.

Fig. 18.21 Rhythm strips showing some ST segment abnormalities.

Hypertrophy

If the heart is faced with having to overcome pressure overload (e.g. left ventricular hypertrophy in hypertension or aortic stenosis) or higher systemic pressures (e.g. essential hypertension) then it will ↑ its muscle mass in response. This ↑ muscle mass can result in changes to the ECG.

Atrial hypertrophy

This can lead to changes to the P wave.

Ventricular hypertrophy

This can lead to changes to the cardiac axis, QRS complex height/depth, and the T wave.

Left ventricular hypertrophy (LVH)

- Tall R wave in V_6 and deep S wave in V_1.
- May also see left axis deviation.
- T wave inversion in V_5, V_6, I, aVL.
- Voltage criteria for LVH includes:
  - R wave >25mm (5 large squares) in V_6.
  - R wave in V_6 + S wave in V_1 >35mm (7 large squares).

Right ventricular hypertrophy

- 'Dominant' R wave in V_1 (i.e. R wave bigger than S wave).
- Deep S wave in V_6.
- May also see right axis deviation.
- T wave inversion in V_1-V_3.

Paced rhythms

Temporary or permanent cardiac pacing may be indicated for a number of conditions (see *OHCM6*, pp.134 and 762) such as complete heart block or symptomatic bradycardia. These devices deliver a tiny electrical pulse to an area of the heart, initiating contraction. This can be seen on the ECG as a sharp spike.

Many different types of pacemaker exist, and can be categorized according to:

- The chamber paced (atria or ventricles or both).
- The chamber used to detect the heart's electrical activity (atria or ventricles or both).
- How the pacemaker responds—most are inhibited by the normal electrical activity of the heart.

On the ECG look for the pacing spikes (see Fig. 18.22) which may appear before P waves if the atria are paced, before the QRS complexes if the ventricles are paced, or both.

Be careful not to mistake the vertical lines that separate the different leads on some ECG print-outs as pacing spikes!

▸ Paced complexes do not show the expected changes described elsewhere in this section. You are, therefore, unable to diagnose ischaemia in the presence of pacing.

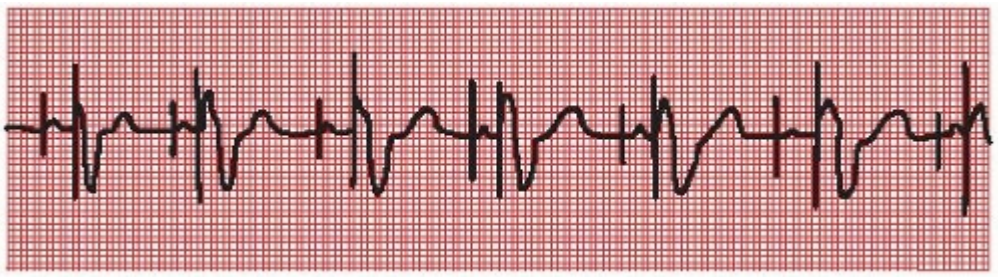


Fig. 18.22 Rhythm strip showing dual-chamber pacing.

Chest X-rays: an introduction

Senior clinicians and radiologists who look at a chest film and immediately give a diagnosis are able to do so through years or practice and the development of ‘pattern recognition’.

For the student or non-specialist, careful review of the chest radiographs requires a systematic examination of the film to include all systems and body parts therein.

There is no ‘correct’ method but you should ensure that all is examined. You could do this by region; we do this by organ system.

Box 18.6 Framework for reporting a chest film

- Name.
- DOB.
- Exam date.
- Technical considerations.
  - Type of film.
  - Position of patient.
  - Projection.
  - Orientation.
  - Rotation.
  - Exposure.
  - Inspiration.
  - Inclusion.
- Clinical review.

Technical considerations

▸ It should be remembered that the CXR is not a slice of the chest. It is a 2-dimensional *representation* of all the internal structures created by X-rays passing through them. Features can overlie each other hiding abnormalities or creating unusual features with ‘compound’ images. As such, the image can be influenced by a whole host of external factors.

Projection

This refers to the direction at which the X-ray beam passes.

- *PA (posterior-anterior):* the cassette is placed in front of the patient and x-rays pass from the back. This is a ‘standard’ CXR.
- AP *(anterior—posterior):* the cassette is placed at the back of the patient and the X-rays fired from in front. An AP film may be taken in the unwell patient who is unable to stand in front of the X-ray machine—for example, if they are bed-bound.
- *Lateral:* views from either the left or the right of the patient.

The direction of the beam has consequences for the appearance of the intrathoracic structures. As the X-rays leave the machine, they diverge. The result of this is that objects near the cassette will appear their true size but objects further from the cassette (i.e. nearer the source of radiation) will appear enlarged—Fig. 18.23. This is particularly important in the case of the heart—it is unwise (some would say impossible) to judge true heart size on an AP film as the heart lies much further from the cassette.

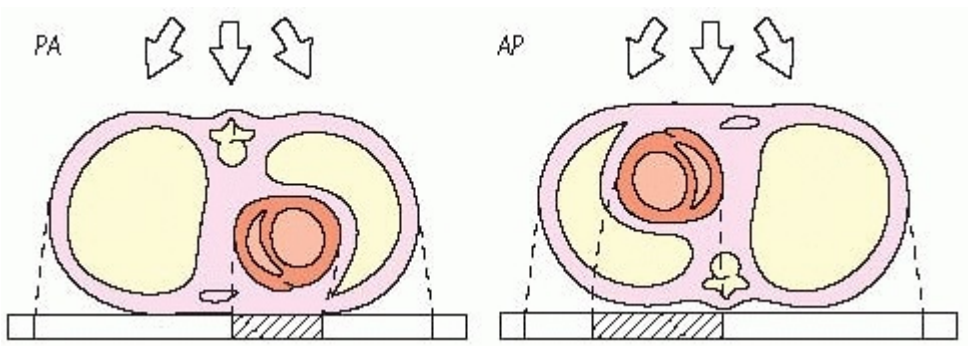


Fig. 18.23 Fig. showing an exaggeration of X-ray beam divergence. Note that when the heart is closerto the origin of the beam, it appears larger on the resultant image.

Position

Standard X-rays are taken with the patient standing. Any deviation from this will usually be marked on the film allowing you to learn more about the patient and adjust your assessment of the image.

- *Sitting:* suggests a severe illness as the patient is unable to stand
  - Diaphragm will be artificially raised, inflation of the lungs will be reduced, soft tissues at the front of the patient may be folded.
- *Supine or prone:* suggests a very severe illness (patient unable to sit).
  - Structures will be displaced within the chest, abnormal distribution of blood supply to the lungs, fluid levels will not be seen.

Orientation

- Films should have position markers. Often only one side is labelled (e.g. 'R' for the right of the patient).
- The image should be presented as if you are looking at the patient—that is, the patient's right on your left and vice versa.
- Ensure that the marker is on the correct side—beware the relatively rare cases of dextrocardia!

Rotation

Films should be taken with the patient facing directly away from the source (for PA films).

You should ensure that this is the case as rotation can cause abnormal appearances of the mediastinum and other structures.

- Look at the spinous processes of the thoracic vertebrae—these should appear at the centre of each bone.
- Look also at the distance between the medial ends of the clavicles and the spinous process of the nearest vertebra—these should be equal.

Exposure

The appropriate exposure depends largely on patient size. Too little will cause the lungs to appear too white, too much will make structures appear too dark and subtle signs may be lost.

- As rule of thumb, you should *just* be able to make out the thoracic vertebral bodies through the image of the heart.

Inflation

A CXR should be taken with fully inflated lungs. A poor inspiration can make the lungs appear more dense, draw the trachea to the right and make the heart appear abnormally large.

- There should be ≥9 ribs visible posteriorly.
  - Count from the top down but beware as it is often easy to confuse the first couple of ribs and the clavicles.
  - To be sure, find the anterior end of the first rib and trace it posteriorly, the 2nd rib often appears quite close below.

▸ See Fig. 18.25.

Inclusion

Does the film show all the structures that you wish it to? You should be able to see the apices of both lungs and both costophrenic angles.

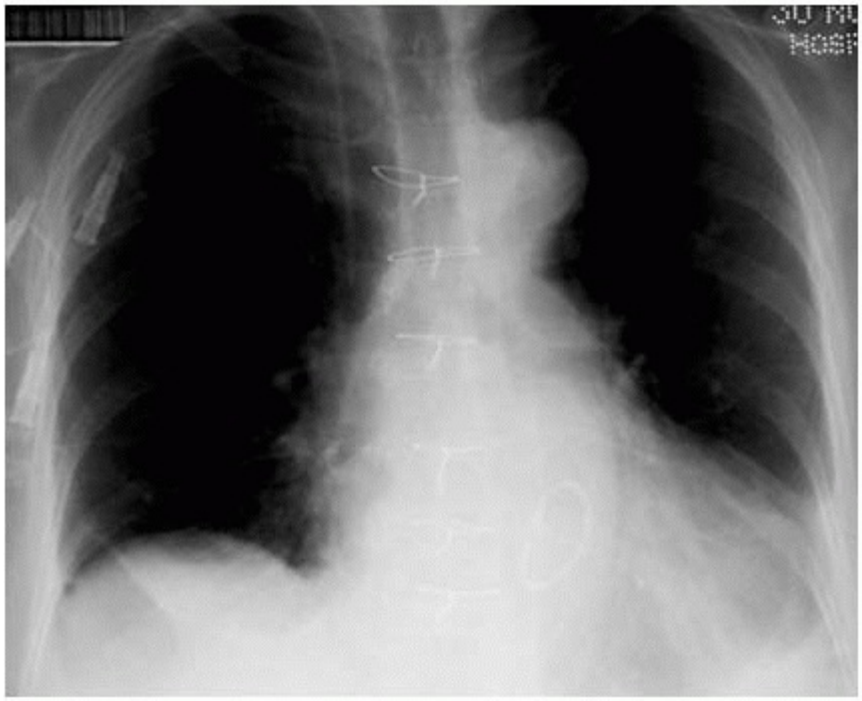


Fig. 18.24 Radiograph showing an inadequate image quality. The lung fields appear too black and the apices of the lungs are not included in the image.

Box 18.7 Densities

▸ Be aware that there are 5 basic densities on X-rays and these are:

- *Black:* gas.
- *Dark grey:* fat.
- *Light grey:* soft tissues and fluid.
- *White:* bone and calcification.
- *Intense white:* metal.

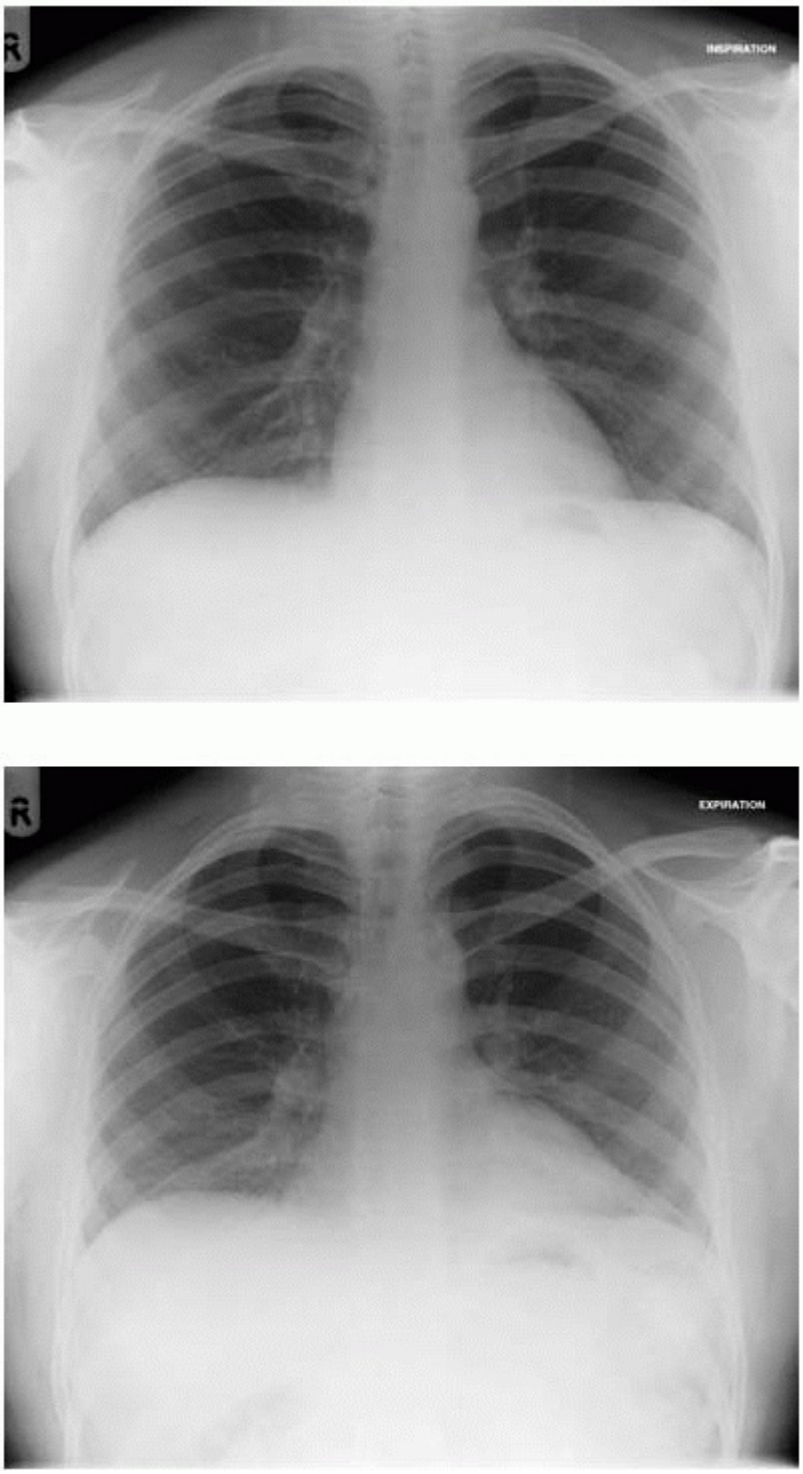


Fig. 18.25 Radiographs of the same patient showing the effect of inspiration. The upper image has a good inspiration, the lower is inadequate. Note how the lungs appear more dense in the lower image and the heart enlarged.

Clinical interpretation

Heart and mediastinum

The key to understanding the mediastinum is a knowledge of the normal anatomy. All the structures, apart from the trachea, appear as fluid or softtissue density and are hard to distinguish separately. Much can be learned by looking at the shape of the mediastinum.

Examine:

- The mediastinal border, looking for abnormality.
- The trachea and bronchi.
- The heart, looking for visible valves (metal heart valves will appear opaque on the CXR—see Fig. 18.52 p.698).

- Cardiac size: should be <1/2 of the thoracic width on a PA film—measure carefully (with a ruler).
- Look for masses, calcification, or free air (pneumomediastinum).

Abnormalities

- Venous engorgement: look at the lung fields.
- Hiatus hernia: may be seen as a rounded object sitting behind the heart or causing distortion of the normal mediastinal outline. There may be a visible fluid-level within it.
- Enlarged heart: enlargement of one or more chamber may be seen on the mediastinal outline or simply by enlargement of the heart size.
- Pericardial effusion: massive enlargement of the heart with a classical 'boot-shape'.
- LV aneurysm: seen as an enlarged left side of the heart—look for calcification which will appear as white.
- Pericardial calcification: seen as a patchy white outline of the heart.

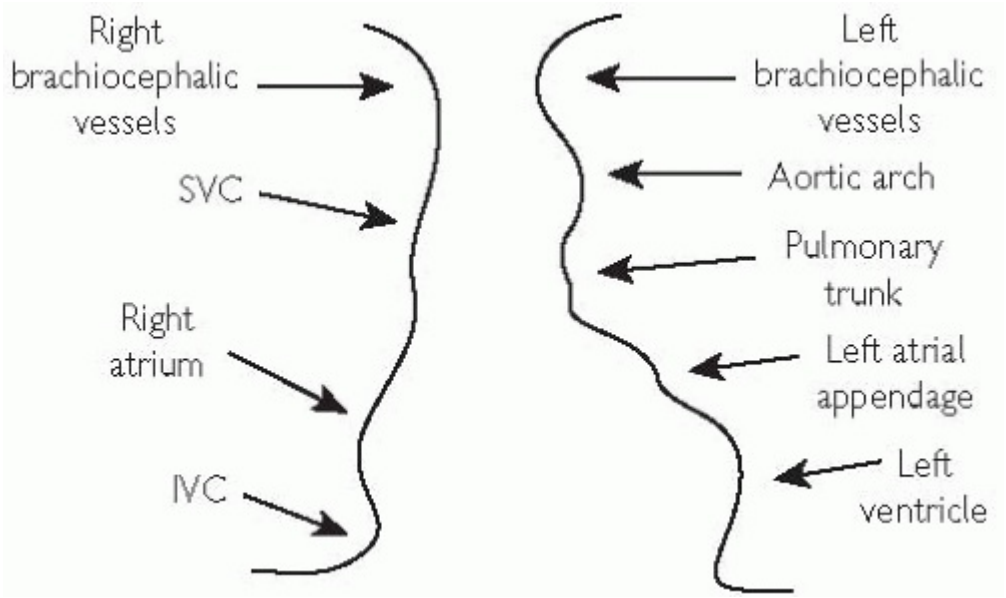


Fig. 18.26 Diagram illustrating the normal outline of the mediastinum and the structures giving rise to the shape.

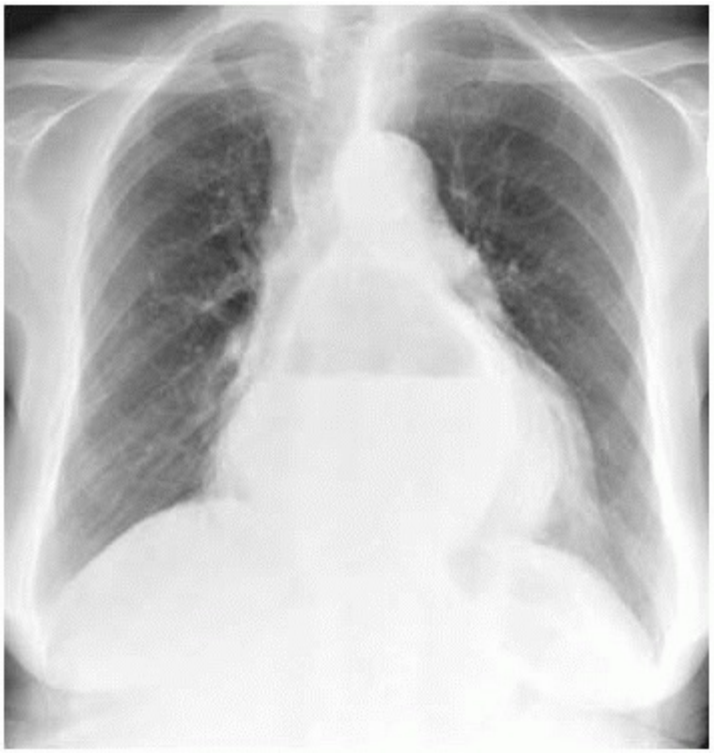


Fig. 18.27 Radiograph showing a large hiatus hernia. You can clearly see a fluid level within the herniated stomach behind the heart.

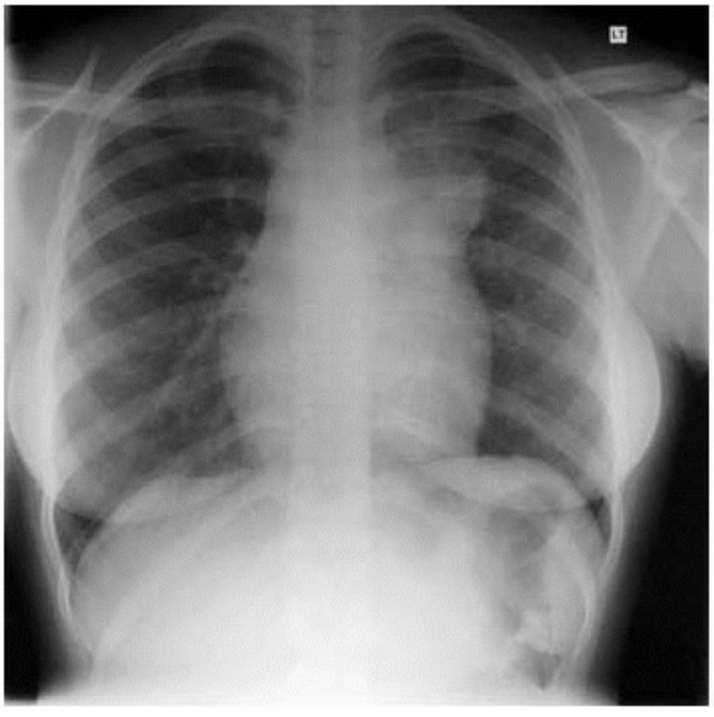


Fig. 18.28 Radiograph showing a mass in the upper portion of the mediastinum on the left.

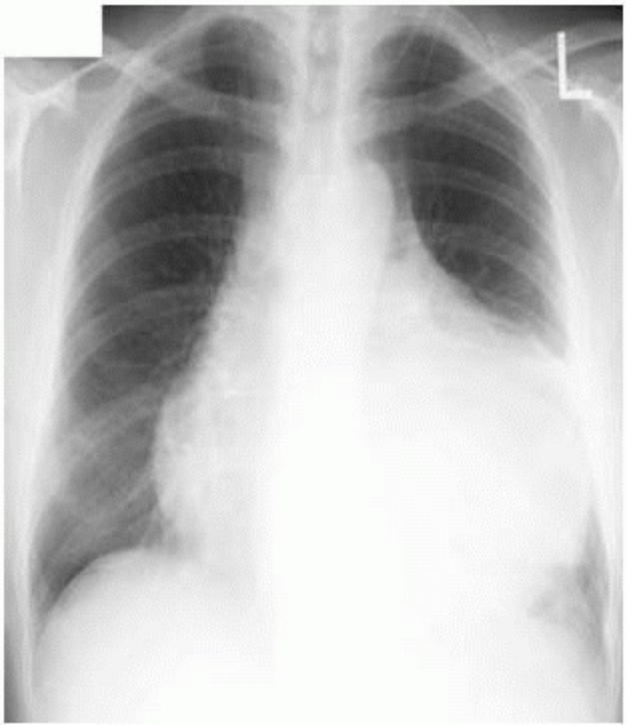


Fig. 18.29 Radiograph showing a large pericardial effusion. The heart appears grossly enlarged with a typical 'boot' shape.

Hila

The hila are the regions at which the lungs connect to the central circulation. They appear as opaque regions on the right and the left side of the mediastinum. Most of the image is created by the pulmonary arteries and veins coming to and from the heart.

The hila should be rounded and symmetrical. As the left pulmonary artery is slightly superior to the right, the left hilum appears ~1cm higher than the right. On each side, the bronchi appear as lucent structures.

Look for:

- Difference in density.
- Asymmetry.
- Loss of the normal concavity.
- ▸ Check for rotation of the film.

Causes of hilar enlargement

- Vascular: smoothly enlarged and irregularly shaped. Arterial enlargement due to congestion or aneurysm or pulmonary hypertension (usually bilateral). Pulmonary hypertension will also cause ↓ peripheral vasculature in the lung field.
- Lymph nodes: smoothly enlarged, regular masses within the hila. Causes: neoplastic (bronchial carcinoma, lymphoma, metastatic), infective (esp. TB—look also for peripheral abscesses or milliary shadowing), infiltrative (sarcoid—look also for pulmonary nodules).
- Other masses: irregular masses, sometimes with poorly defined edges.

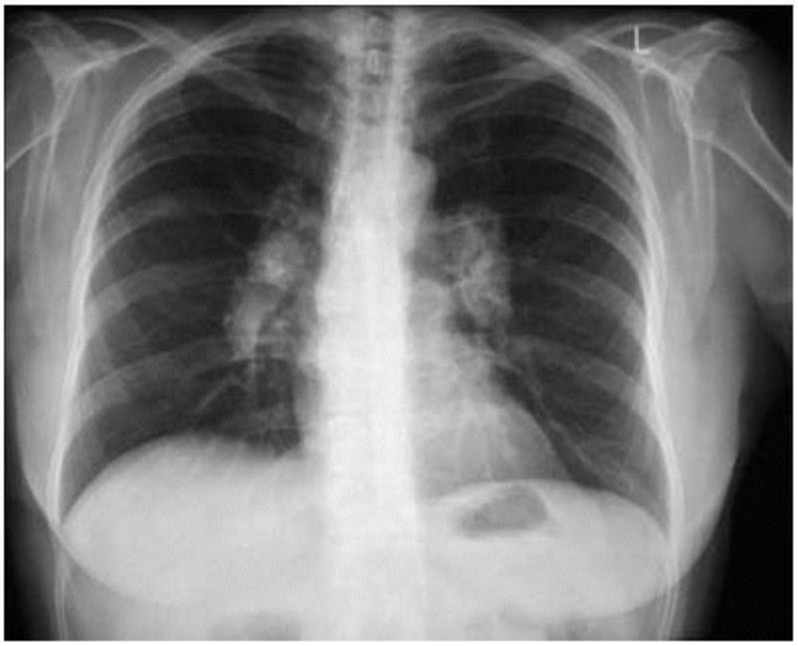


Fig. 18.30 Radiograph showing bilateral hilar lymphadenopathy—in this case, due to sarcoidosis.

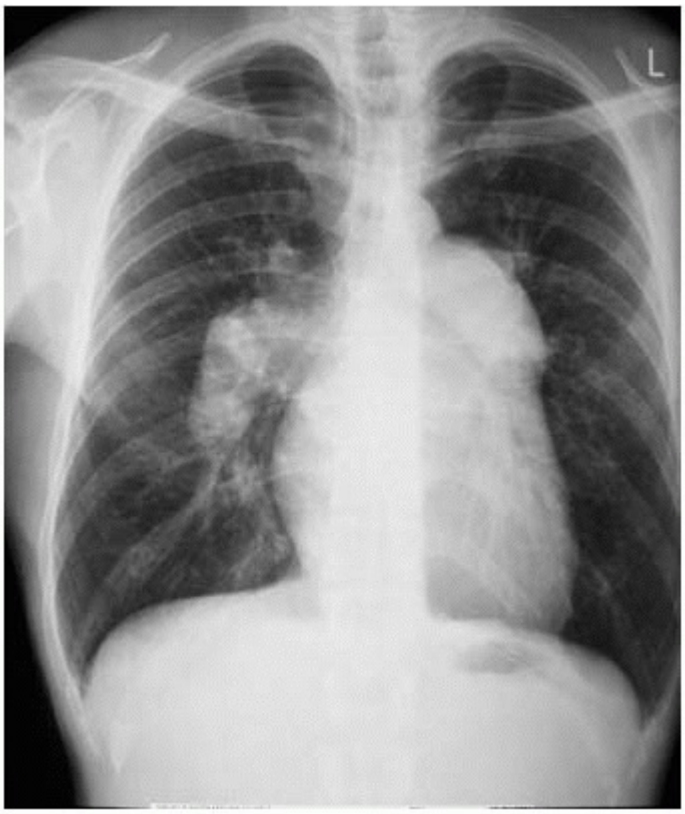


Fig. 18.31 Radiograph showing pulmonary hypertension. The engorged pulmonary arteries are clearly visible at both hila.

Bones

Look at each bone in detail including the shoulder girdles, ribs, clavicles, thoracic vertebrae. Look at the density and trabecular pattern. Look for:

- Lytic lesions.
- Sclerosis.

- Erosions.
- Fractures.
- Dislocation.

Vertebrae

Ensure that the size and density are uniform and the spaces equal. Remember to look for paraspinal soft tissue masses.

Ribs

- Trace each from the back to the front, note width of space between.
- New fractures appear as sharp lines—look for associated complications such as surgical emphysema and pneumothorax.
- Old fractures appear as widened areas of rib, often with a slight distortion of the line of the bone.

Lytic lesions

Bone looks as if it is smudged. This may be the only sign of metastatic lung disease.

Soft tissues

Remember that chest cavities are surrounded on all sides by soft tissue, including the front and the back. Look at all the regions visible. Look for calcification and free air.

Surgical emphysema

Air pockets in the soft tissues. Anterior or posteriorly, this can be difficult to see—and may only be seen indirectly by distortion of the image of the other structures. Laterally, side-on views of the pockets of air can be seen as radiolucent lozenge-shaped or tapering structures.

Breasts

Female and male breast tissue overlying the inferior part of the lung may give the appearance of an increase in lung density. Look for missing breasts as a clue to underlying disease.

Nipples

Nipples often appear as rounded opacities which could be mistaken for lesions within the lung.

Abdomen

One should be able to see air in the stomach as a bubble below the left hemidiaphragm. Often, there is an air-fluid level within it creating a shape with a rounded top and a horizontal lower edge.

A gastric-bubble on the right may indicate either situs inversus or mislabelling of the film!

Look below the diaphragm for free air (pneumoperitoneum)—often seen as a wisp of radiolucency between the liver and the right hemidiaphragm or above the gastric-bubble.

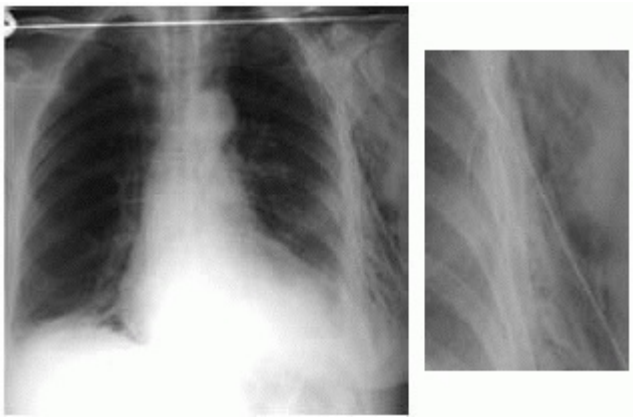


Fig. 18.32 Radiograph showing extensive surgical emphysema on the left of the chest. Insert shows a close-up—you can also see the chest drain that is the cause of the problem.

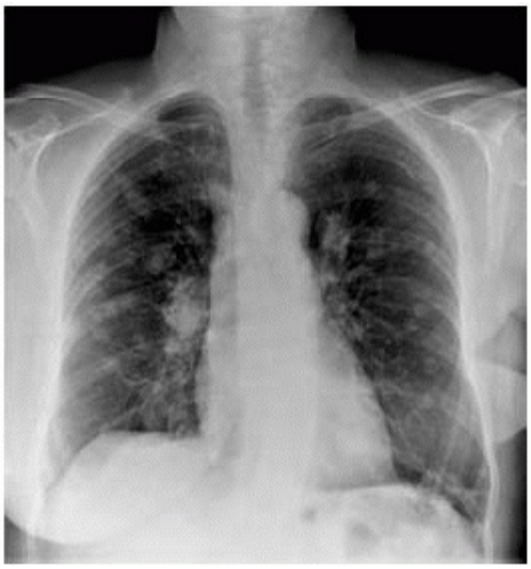


Fig. 18.33 Radiograph showing a patient with disseminated breast cancer. Aside from the multiple pulmonary lesions, there is a lack of breast shadow on the left indicating that this patient has had a left mastectomy.

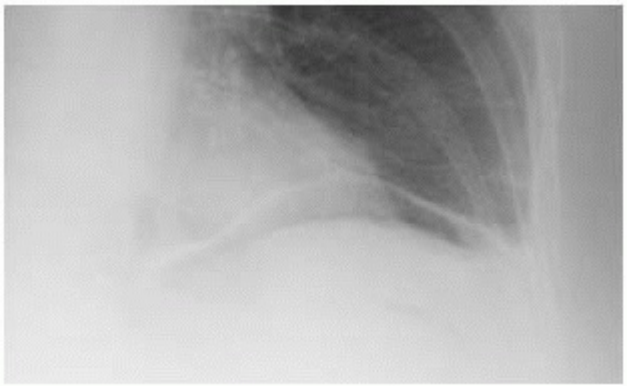


Fig. 18.34 Close-up showing free air below the left hemidiaphragm.

The lungs

The reader should remind themselves of the normal lung anatomy. The points pertinent to the physical examination are also relevant to the radiographic examination.

Normal

Seen head on, the oblique fissures are not visible. As the upper lobes lie largely anterior to the lower lobes, the lung field on the CXR is both the upper and lower lobes (on the left). Normal features include:

- Lung fields of equal density.
- Right hemidiaphragm slightly higher than the left.
- Sharp costophrenic angles and cardiophrenic angles.

- The horizontal fissure in the right lung passes horizontally from the midpoint of the right hilum to about the 6th rib in the axillary line.
- The pleura should be thin and symmetrical.

Examining the film

Scan the entire lung looking for areas which are too black, too white, or abnormally placed. Look for abnormal calcification.

▸ Look especially at the 1st ribs, behind the heart, and behind the diaphragms where lesions can often be missed.

▸ Ensure the lung markings extend to the periphery.

Collapse

Loss of air in a lobe or lung region which becomes dense. Look for:

- Changes to the normal anatomy and asymmetrical density.
- Mediastinal and tracheal shift (the volume loss in the affected lung will pull the mediastinum towards the lesion).
- Loss of clarity of the borders of the heart and the diaphragms (collapse nearby will cause a blurred outline).

Right upper lobe

Collapses upward and to the midline. The horizontal fissure may be elevated and the mediastinum shifted to the right. Appears as a white mass to the right of the upper part of the mediastinum.

Right middle lobe

Difficult to see on the PA film—may be seen only as a slight blurring of the right heart border and an elevation of the right hemidiaphragm.

Lateral film: seen as a triangle of white with its apex at the hilum and base anteriorly.

Right lower lobe

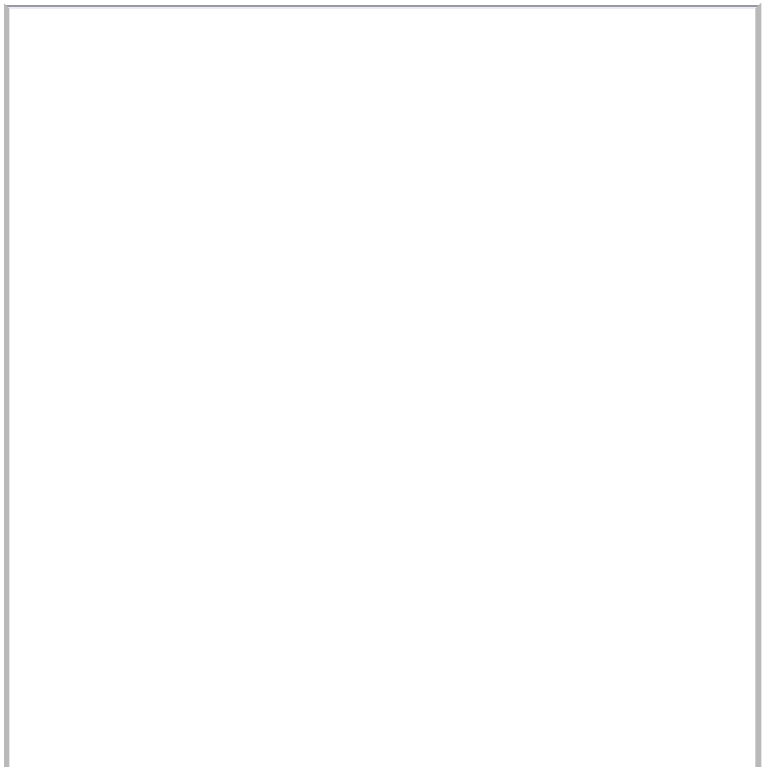
Triangular area of white between the right heart border and diaphragm causing both to be indistinct.

Left upper lobe

Difficult. Haziness to the left hemithorax with loss of clarity at the left heart border. Easier to see on a lateral film.

Left lower lobe

'Sail' shape of white—but hidden behind the heart border so difficult to spot. Look for a double-edge to the heart.



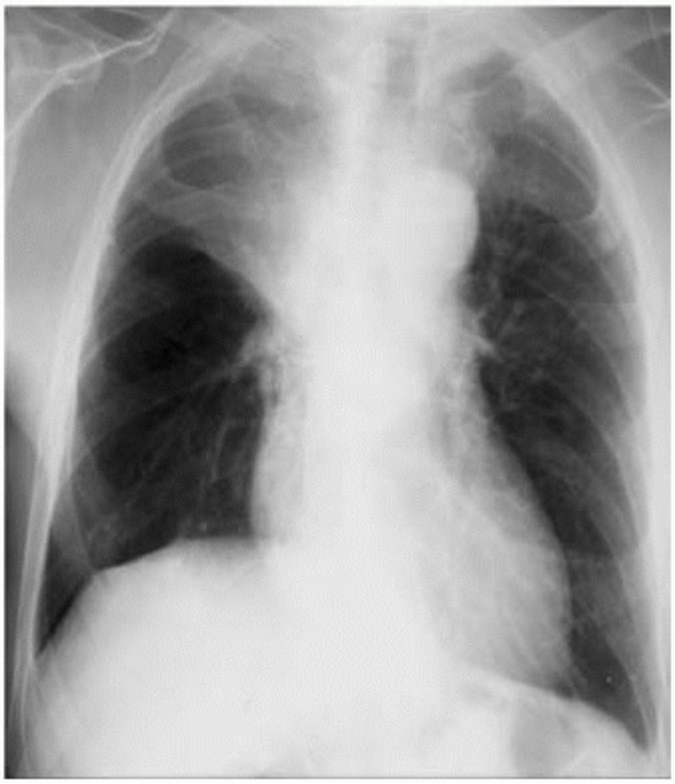


Fig. 18.35 Radiograph showing right upper lobe collapse.

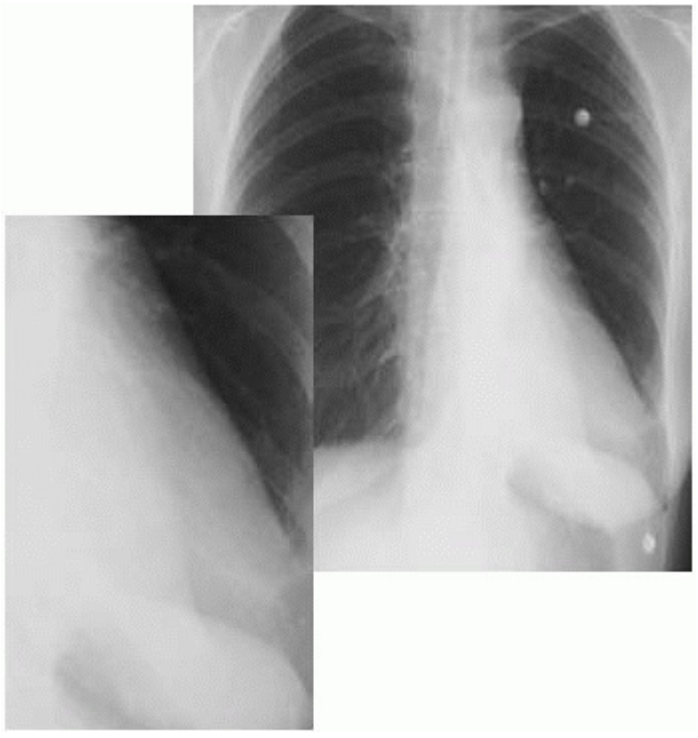


Fig. 18.36 Radiograph showing left lower lobe collapse—note the ‘sail-shape’ of white behind the heart.

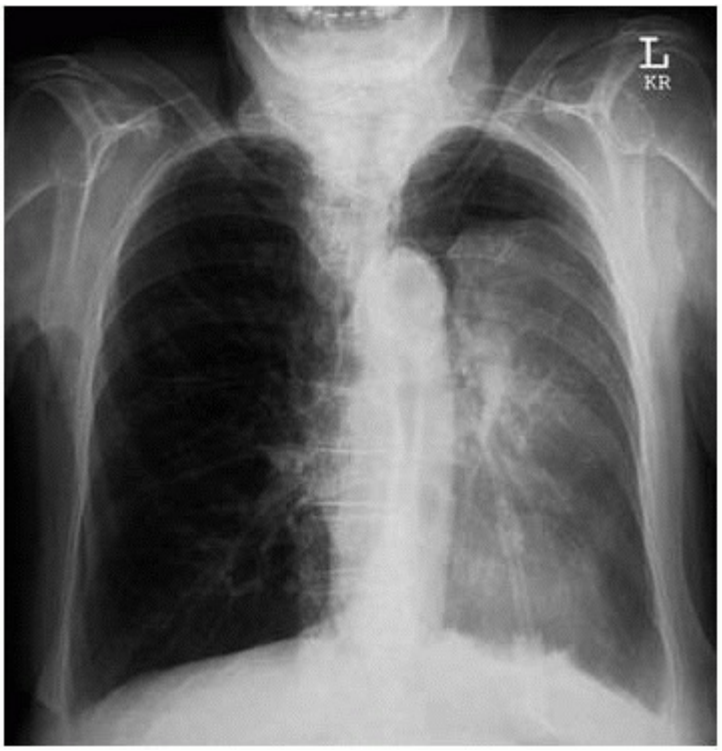


Fig. 18.37 PA radiograph showing left upper lobe collapse.

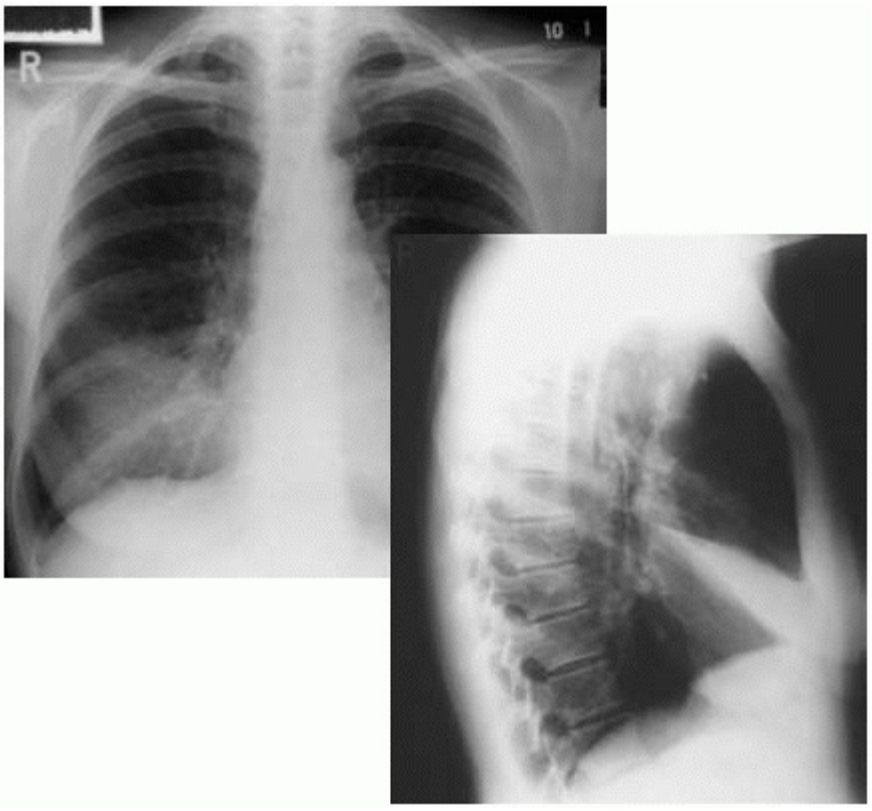


Fig. 18.38 Radiographs showing right middle lobe collapse. Seen only as a slight ↑ in density to the right of the heart on the PA film. Much easierto see than on the lateral film.

Consolidation

Consolidation is usually a sign of infection—fluid within the alveolar spaces making an area of the lung appear more dense (Fig. 18.39). Look

for:

- A focal area of ↑ density with indistinct margins.
- Heterogeneous ‘alveolar’ shadowing.
- Not present on previous X-rays (focal fibrosis may appear similar but is usually long-standing with a chronic course).
- There may be a sharp demarcation at lobar margins (right upper lobe consolidation may be seen with a sharp horizontal lower border where it meets the right middle lobe).
- *Air bronchograms:* as the airways remain patent, an air-filled bronchus passing through an area of consolidation may be seen as a radiolucent (black) tube.

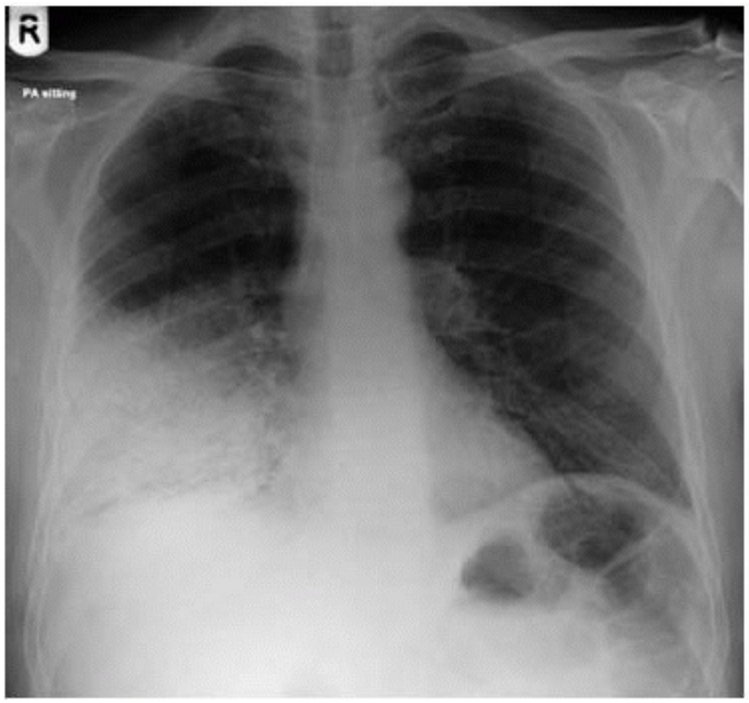


Fig. 18.39 Radiograph showing right lower lobe consolidation.

Box 18.8 Differentiating right lower zone consolidation

Consolidation at the base of the right lung may either be in the right middle lobe and/or right lower lobe. Look for blurring of the structures nearby.

- *The right middle lobe* does not touch the diaphragm—consolidation here will cause blurring of the right heart border whilst the diaphragm remains distinct.
- *The right lower lobe* lies against the diaphragm but very little of it touches the heart—consolidation here will cause blurring of the right hemidiaphragm but leave the right heart border relatively distinct.

Fibrosis

↑ density of the lung tissue in fibrosis causes a ‘reticulonodular’ (net-like with nodules) pattern of opacity. As it progresses, it may cause a ‘honeycomb’ appearance of the lung. You should be careful to differentiate fibrosis from oedema or consolidation.

Features of fibrosis (Fig. 18.41):

- Often bilateral, although not necessarily symmetrical.
- Causes ↓ lung volumes.
- If focal, draws the mediastinum towards the affected side.
- Causes blurring of the heart and diaphragm borders.
- Have a distinctive reticulonodular pattern.

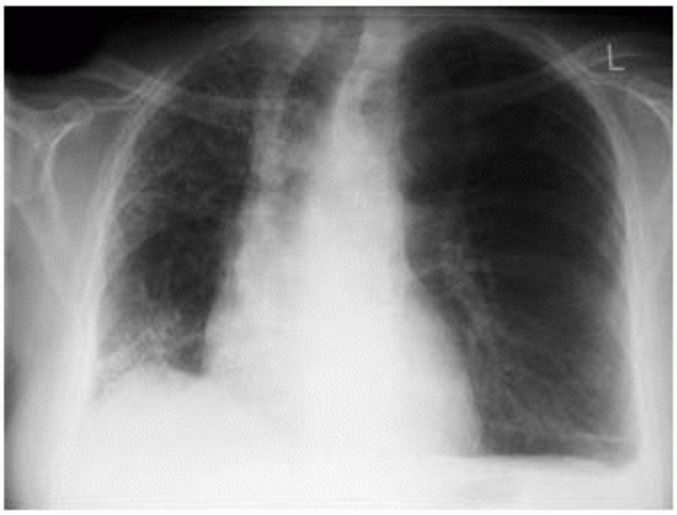


Fig. 18.40 Radiograph showing a patient post-lung-transplant. The right lung is the patient's native and is severely fibrosed—note the reticular shadowing and the ↓ volume. The left lung is the healthy donated lung.

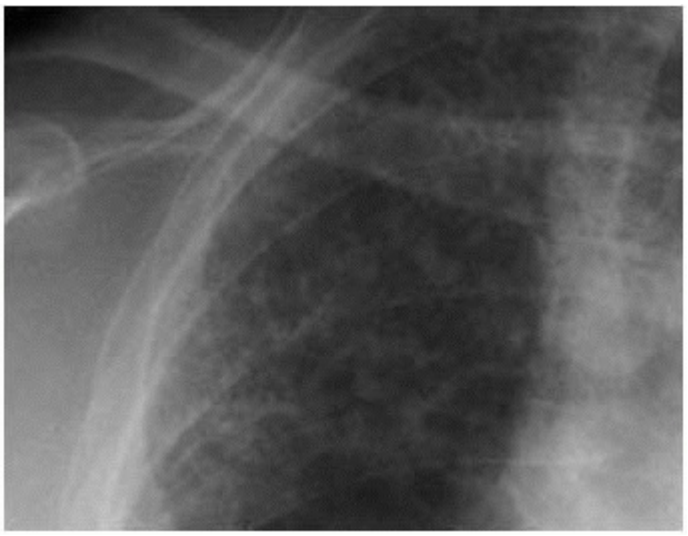


Fig. 18.41 Close-up of a radiograph showing pulmonary fibrosis.

Pulmonary oedema

This causes an alveolar pattern of shadowing. If due to congestive cardiac failure, there will also be changes in the appearance of the heart.

Features of pulmonary oedema secondary to heart failure (Fig. 18.42):

- Enlarged heart.
- Bilateral, although not necessarily symmetrical, lung shadowing.
- Classically, in the middle and upper zones causing a ‘bat's wing’ appearance.
- Pulmonary venous engorgement.
  - The vessels appear to extend further than normal into the lung field.
  - Vessels in the upper zone appearing larger than normal (often >5mm diameter) ‘upper lobe blood diversion’.

- *Kerley's B lines:* short, horizontal white lines close to the lung periphery usually extending horizontally into the lung field. Particularly found at the bases. Caused by oedema of the interlobular septa.

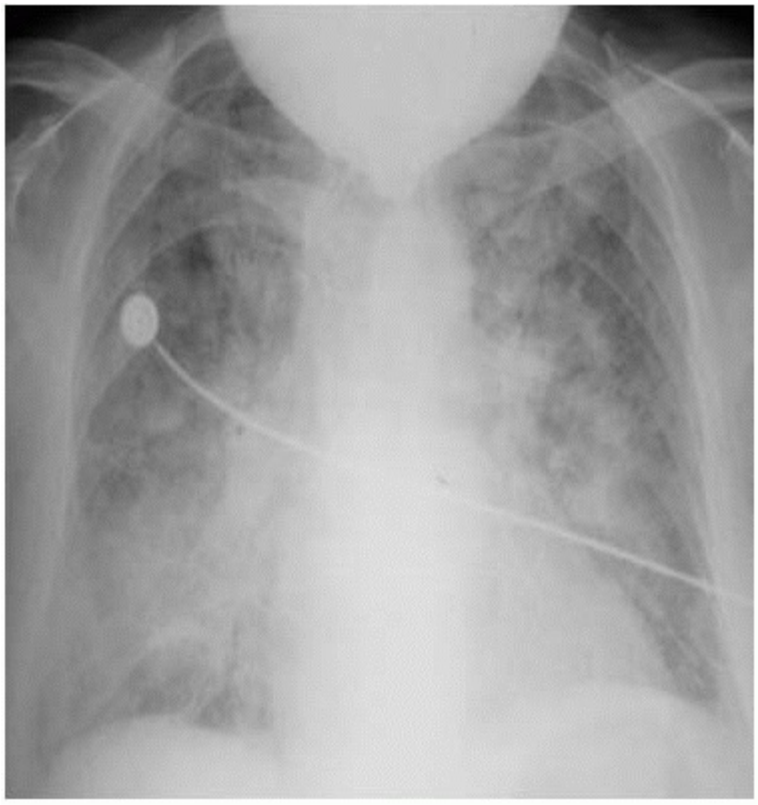


Fig. 18.42 Radiograph showing severe pulmonary oedema. There is extensive alveolar shadowing in both lung fields, the heart is enlarged. Note also the ECG lead attached to the patient's chest—a sign of the clinical severity!

Coin lesions

Lung masses often with well-demarcated borders. Could also represent a focal area of consolidation or a pleural lesion. Look for:

- Calcification within the lesion (rare if malignant).
- Air bronchogram within the lesion (indicating consolidation, not a mass).
- Cavitation.
- Changes in the bones and soft tissues nearby.
- Lymph node enlargement in the hila.
- Other lesions.

Cavitating lesions

A lung lesion with a central cavity creating a circular radiographic appearance. If there is fluid within the lesion, an air-fluid level may be seen. Look at the rest of the film for clues to the nature of the lesion (i.e. lymphadenopathy, other lesions, signs of infection). Possible causes include lung abscess, neoplasm, or focal area of infarction.

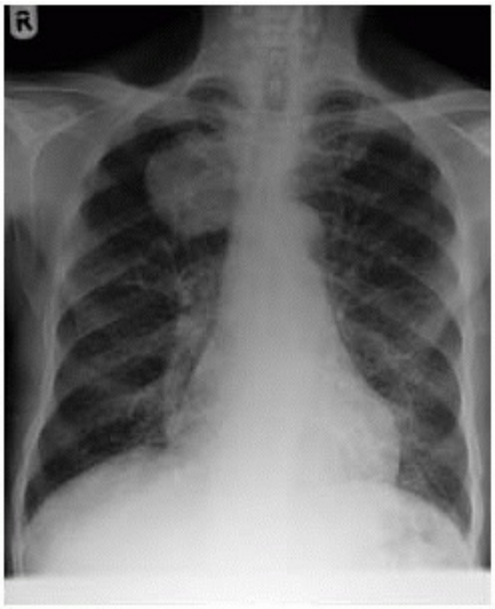


Fig. 18.43 Radiograph showing a right upperzone mass.

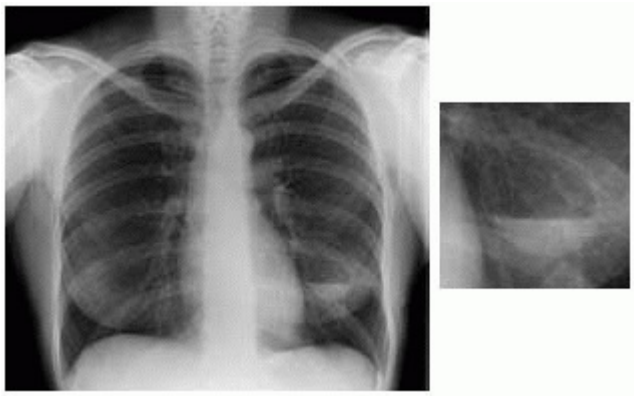


Fig. 18.44 Radiograph showing an abscess in the left lung—you can clearly make out the air-fluid level within the rounded lesion (insert).

Bronchiectasis

Can be difficult to diagnose on a plain x-ray. The combination of airway enlargement, airway wall thickening, and alveolar shadowing can, however, give a distinct appearance.

- *Ring shadows:* affected bronchi seen head on. Appear as small rings—several together give a 'bunch of grapes' appearance.
- *Tramline shadows:* affected bronchi seen side on. Appear as short parallel lines.

Pneumonectomy

Rare. The side from which the lung was removed will appear densely white (a 'white-out') (Fig. 18.45). As such, it should be carefully differentiated from a large pleural effusion or dense consolidation. There may also be:

- Mediastinal shift towards the affected side (an effusion would push the mediastinum the other way).
- A total absence of lung markings on the affected side.
- ↓ density of the remaining lung as it expands to fill the space available.

- Signs of previous surgery (e.g. rib resections, sutures).

Pleural lesions

Lesions in the pleura may be seen to overlie the lung field—or may be seen laterally at the lung edge. Pleural plaques caused by asbestos exposure are often seen as a heterogeneous lesion with irregular and spiculated edges ('holly-leaf' appearance).

Remember that the pleura also covers the diaphragm—look for the linear appearance of plaques seen side-on. Be careful not to mistake pleural lesions for lung lesions.

Pleural effusion

An effusion will appear as a white area at the base of the lung (if the patient is standing or sitting up). It often has a well-demarcated upper border with a 'meniscal' edge as the fluid tracks up lateral to the lung. As the overlying lung is unable to inflate fully, there may be the appearance of increased lung density with a blurred upper border of the effusion. Look for mediastinal shift (it will move away from the effusion, if large).

Small effusions may only be seen as a blunting of the costophrenic angle on the affected side—but remember that ~500ml of fluid can 'hide' behind the hemidiaphragm before it becomes visible on a PA chest film.

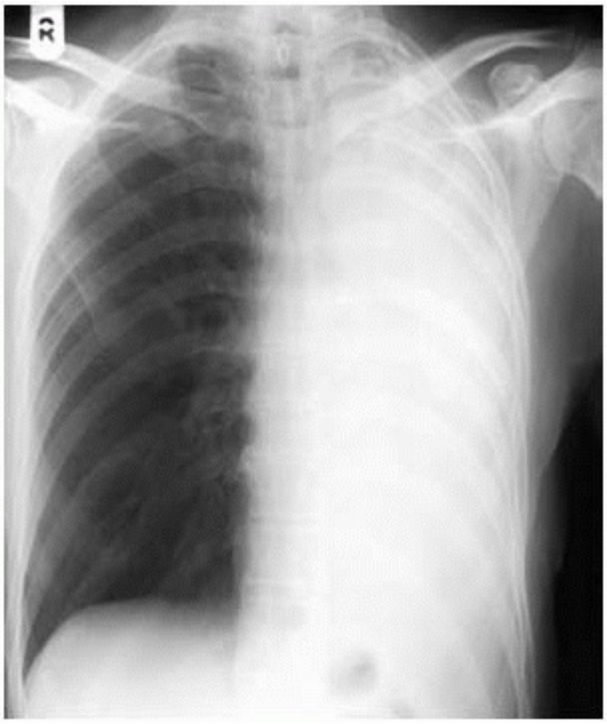


Fig. 18.45 Radiograph showing a previous left pneumonectomy. The mediastinum has shifted so far to the left that it is almost impossible to distinguish amid the ↑ density of the left side of the chest.

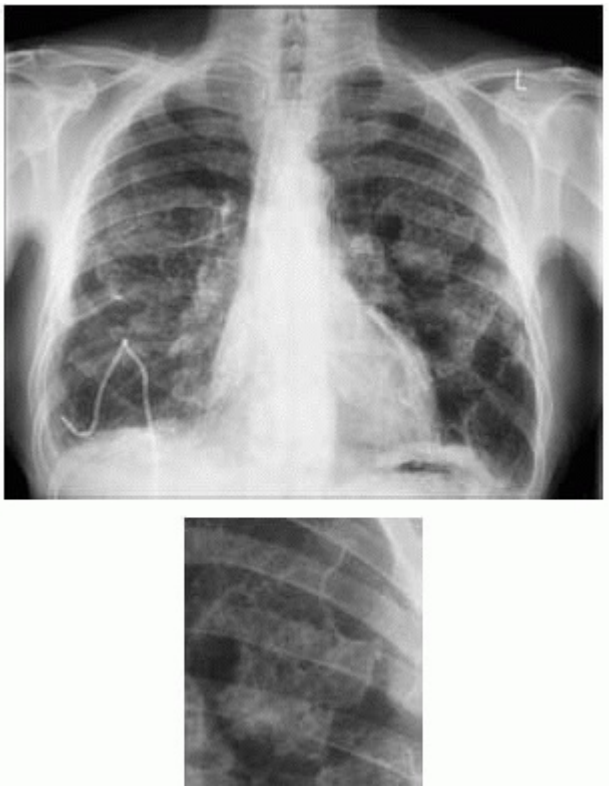


Fig. 18.46 Radiograph showing extensive pleural plaque disease. Close-up shows one of the lesions—note the characteristic holly-leaf appearance.

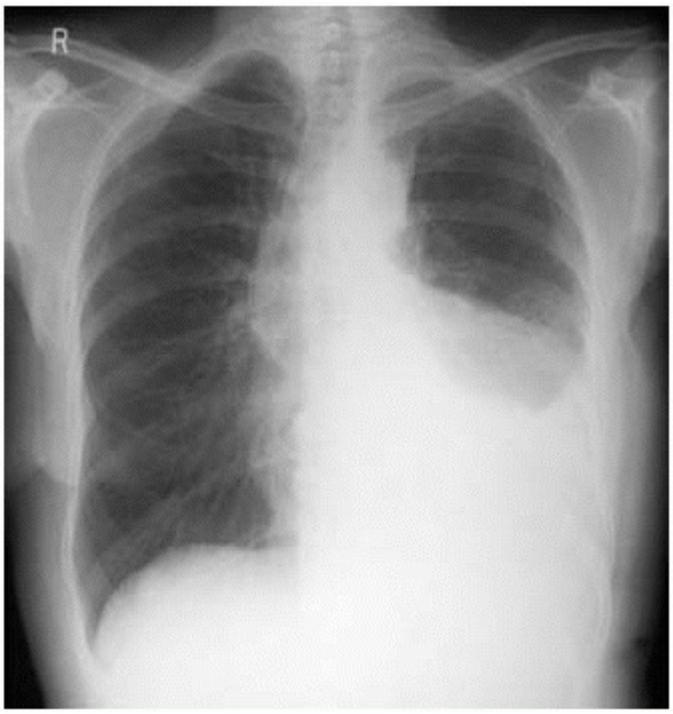


Fig. 18.47 Radiograph showing large pleural effusion on the left. Note the ‘meniscus’ as the fluid tracks laterally beside the lung.

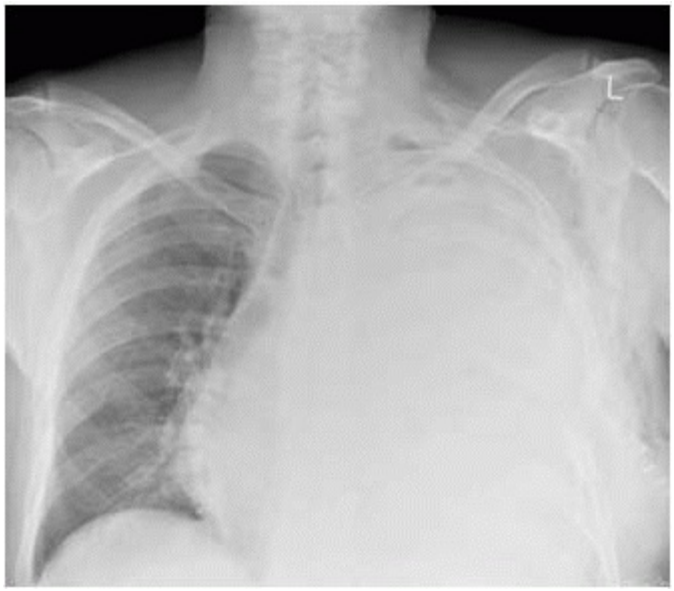


Fig. 18.48 Radiograph showing a massive pleural effusion causing almost a complete 'white-out' of the left lung field. Note how the mediastinum is shifted away from the affected side.

Pneumothorax

Air outside the lung will appear as a black area—often as a sliver between the lung and the thoracic wall. Look for:

- A darker area peripherally, often superior in an erect film.
- The lung markings do not reach the edge of the lung field—look carefully, do not mistake a bulla for a pneumothorax—examine the rest of the lung for similar areas.
- A visible, albeit thin, demarcation between the lung and the free air.

Small pneumothoraces can be hard to spot, try:

- Turning the film on its side (one can often see horizontal lines easier than vertical ones).
- Asking for an expiratory film (the lungs will appear more dense and the pneumothorax will take up relatively more of the lung field).

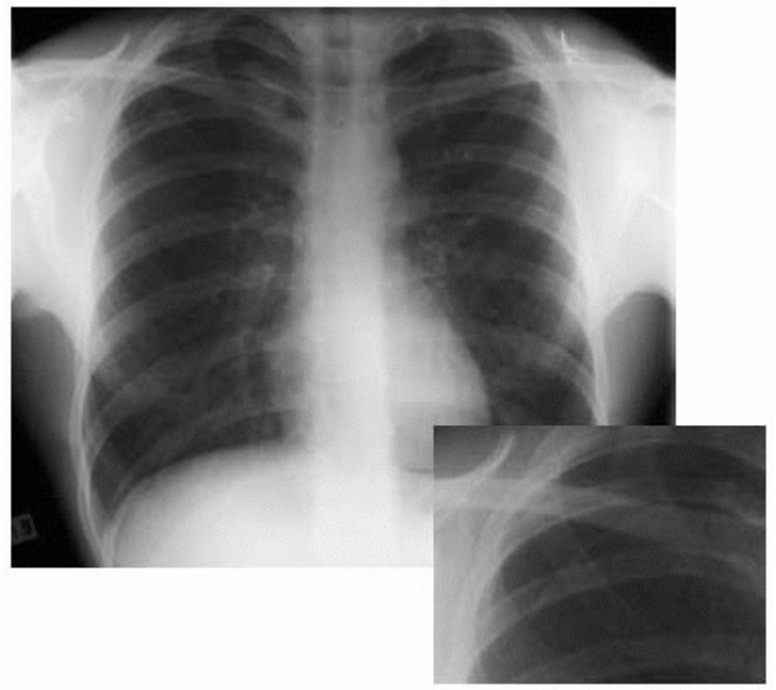


Fig. 18.49 Radiograph showing a right-sided pneumothorax. It is difficult to spot. Close-up shows the pleural edge as a tiny but distinct demarcation between the lung tissue and the free air.

Tension pneumothorax

A medical emergency as ↑ pressure in the chest reduces venous return to the heart.

- The lung may appear small and shrivelled within the lung field.
- Mediastinum shifted away from the affected side.
- Lowered hemidiaphragm on the affected side.

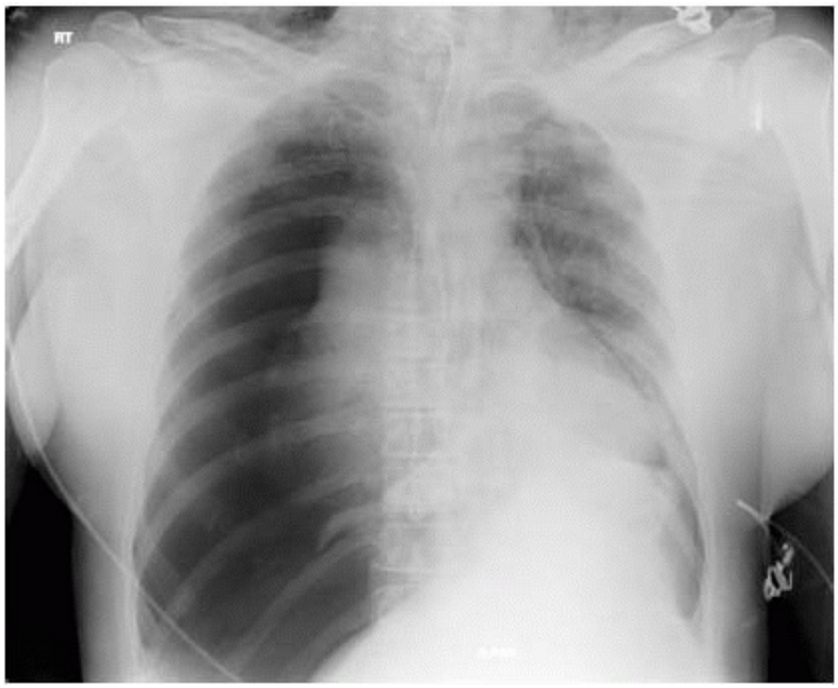


Fig. 18.50 Radiograph showing a right-sided tension pneumothorax. There are no lung markings on the right of the chest. The mediastinum is shifted to the left and the right hemithorax is overinflated.

COPD

Difficult to spot on a CXR. The lung fields may appear less dense than normal with reduced lung markings. The chest may be hyperinflated, often with an elongated-appearance to the mediastinum. Look for bullae and be careful to differentiate from a pneumothorax.

▸ The key sign is flattening of the diaphragms (Fig. 18.51).

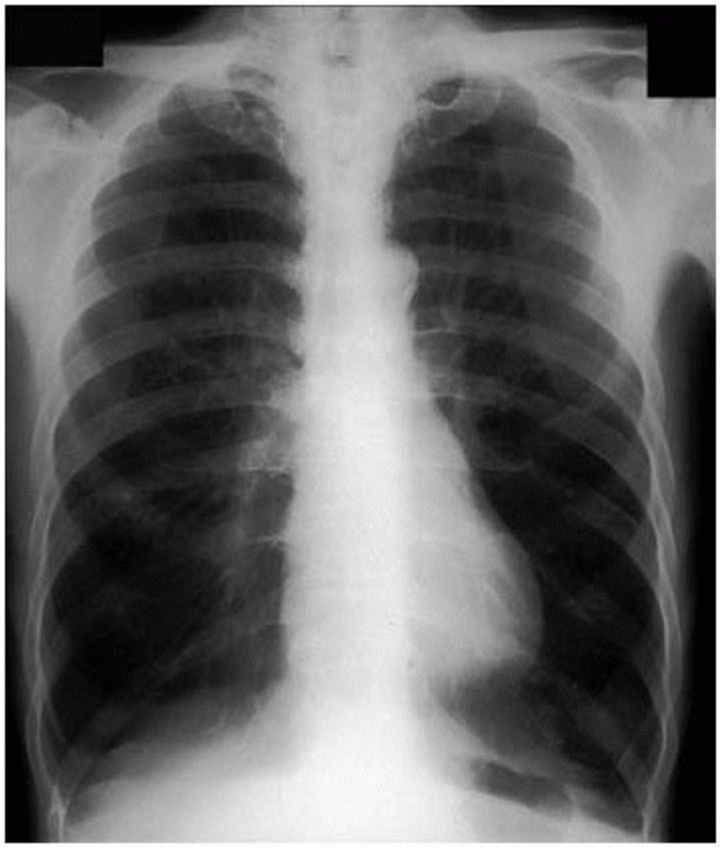


Fig. 18.51 Radiograph showing COPD. Note the ↓ density of the lung fields.

Iatrogenic features/foreign bodies

Clothes, buttons, zippers, or even a cigarette packet in a shirt pocket can give misleading and often amusing appearances on the chest film.

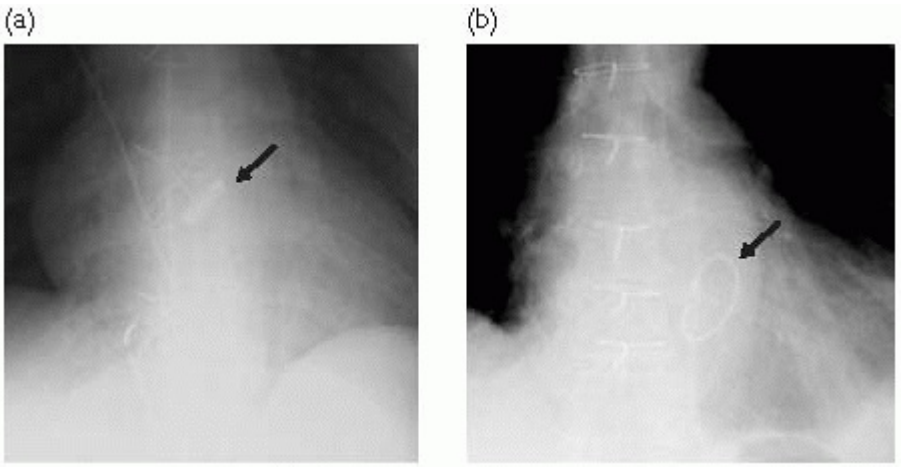


Fig. 18.52 Close-ups of radiographs showing: (a) Aortic valve replacement. (b) Mitral valve replacement.

NG tubes

A correctly placed NG tube will appear as a thin radio-opaque line descending through the mediastinum, below the diaphragm and ending somewhere in the region of the gastric bubble. If the tube does not reach the stomach but is seen to pass inferior to the carina, it is almost certainly in the oesophagus and will need to be inserted further rather than re-inserted. Tubes seen entering either major bronchus should be removed immediately.

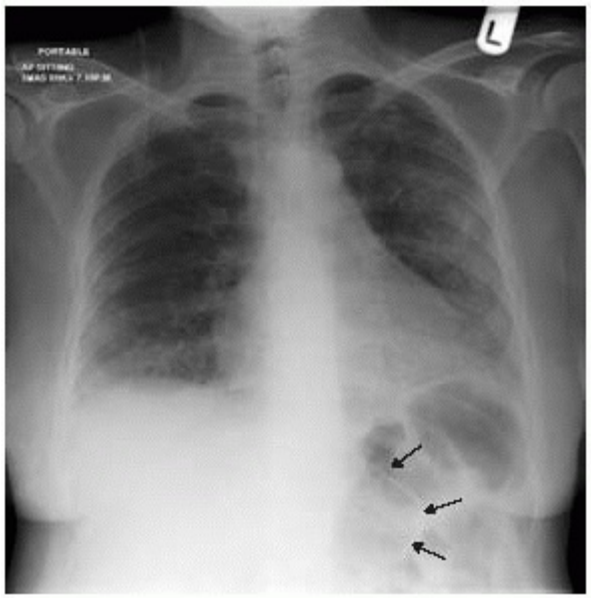


Fig. 18.53 Radiograph showing a correctly placed NG tube.

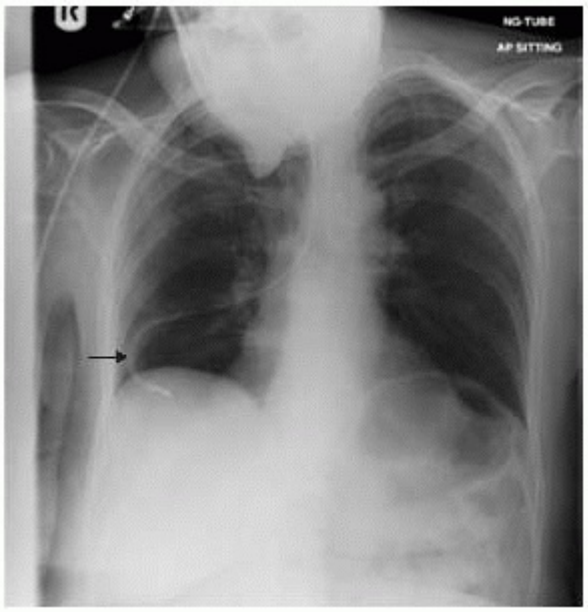


Fig. 18.54 Radiograph showing an NG tube in the right bronchus.

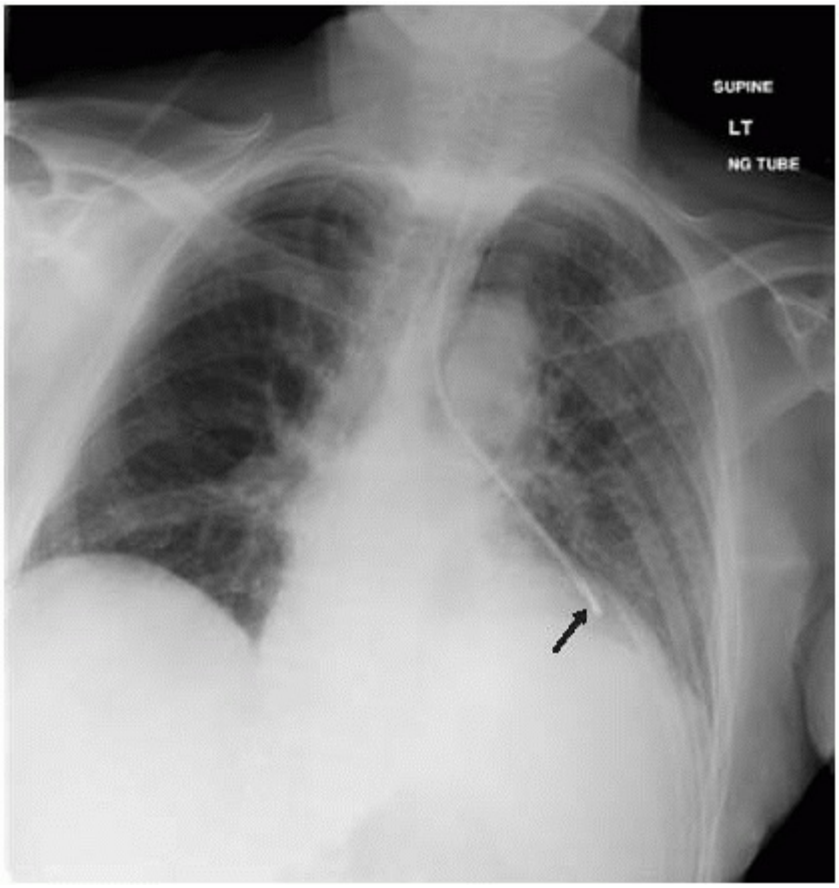


Fig. 18.55 Radiograph showing an NG tube in the left bronchus.

Abdominal X-rays: an introduction

A systematic viewing strategy will aid you in the interpretation of abdominal films. There are certain key points to inspect on every abdominal × ray. These are technical quality, gas pattern, calcification, bones, and soft tissue.

Technical assessment

Always begin your inspection by checking:

- Name of the patient.
- DOB.
- Age of the patient.
- Sex of the patient.
- Date when the film was taken.
- Left and right markers.
- Note the projection of the film.
    - Almost all abdominal X-rays are 'AP' but other views include erect, supine, or decubitus—these are usually marked on the film.

Views

- *Standard abdominal X-ray (AXR):* also known as a 'KUB' (kidneys, ureters, bladder) and is a supine view.
- *Erect film:* is useful in identifying the presence of free intraperitoneal air and intestinal air-fluid levels.
- *The left/right lateral decubitus*[*]: especially useful in severely ill patients who are suspected of having free intraperitoneal air and are unable to stand or sit.
    - Decubitus films can be identified by fluid levels lying parallel to the long axis of the body, as opposed to at right-angles to it on

conventional erect films.

Penetration

- A film that is too light (underpenetrated) or too dark (overpenetrated) is of little diagnostic value.
- As a general rule, if you can see the bones in the spine then the technical quality should suffice.
- With overexposed or overpenetrated films it is recommended that you inspect these areas with a bright light behind them (present on most viewing boxes).

Box 18.9 Densities

▸ Be aware that there are 5 basic densities on X-rays and these are:

- *Black:* gas.
- *Dark grey:* fat.
- *Light grey:* soft tissues and fluid.
- *White:* bone and calcification.

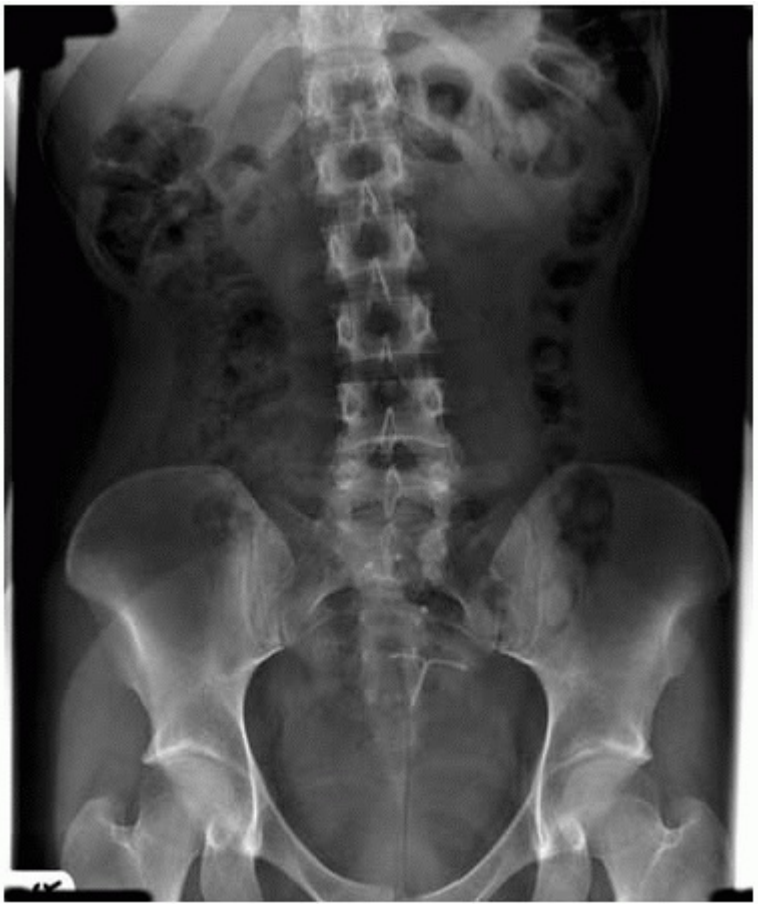


Fig. 18.56 A standard AXR showing a normal abdomen. Incidentally, this patient has a T-shaped copper contraceptive device in her uterus—which is visible on the film.

Gas pattern

The large bowel: lies around the periphery of the abdomen and normally shows haustral indentations. It should contain faeces and gas.

- In the large bowel, loops are considered dilated if the diameter is >5cm.

The small bowel: more central, shows mucosal folds across its width ('valvulae conniventes') and contains fluid and gas.

- The calibre of the normal small bowel should not exceed 2.5-3cm.

▸ Become familiar with the normal gas pattern on an AXR. Abnormal positioning of bowel loops may indicate the presence of an abdominal mass.

Fluid levels

Air-fluid levels are seen as a clear horizontal demarcation between an area of light-grey fluid and darker grey or black gas. Small fluid levels in the small and large bowel are a normal finding.

However, distended loops of bowel with air-fluid levels in a diffuse pattern is highly suggestive of a mechanical obstruction. The bowel loops frequently have a hairpin (180° turn) appearance.

To demonstrate fluid levels you need an erect or decubitus film.

Extraluminal gas

Carefully inspect for signs of extraluminal gas (outside the bowel lumen). This may be either free (pneumoperitoneum) or contained within an abscess cavity, the retroperitoneum, the bowel wall, or the biliary or portal venous systems of the liver.

Free intraperitoneal air suggests a ruptured viscus in the absence of immediate previous surgery.

A subtle sign to look for free gas is the *'double wall'* sign. Here both sides of the wall of loops of bowel become visible because of air on the inside and outside of the bowel.

Pneumoperitoneum

Remember to request an erect chest film in suspected cases of pneumoperitoneum and look for bilateral dark crescents of free gas under the hemidiaphragms.

Calcification

There are several structures which may become calcified, particularly with ↑ age of the patient. These may sometimes cause confusion if mistaken for intestinal structures:

- Costal cartilages.
- Aorta.
- Iliac and splenic arteries.
- Pelvic phleboliths (small round opacities sometimes containing lucent centres in the pelvic venous plexus).
- Mesenteric lymph nodes.

▸ Make careful note of abnormal calcifications such as biliary and urinary calculi, calcified aneurysms, tumours, pancreatic calcification in chronic pancreatitis, and calcified kidneys in nephrocalcinosis.

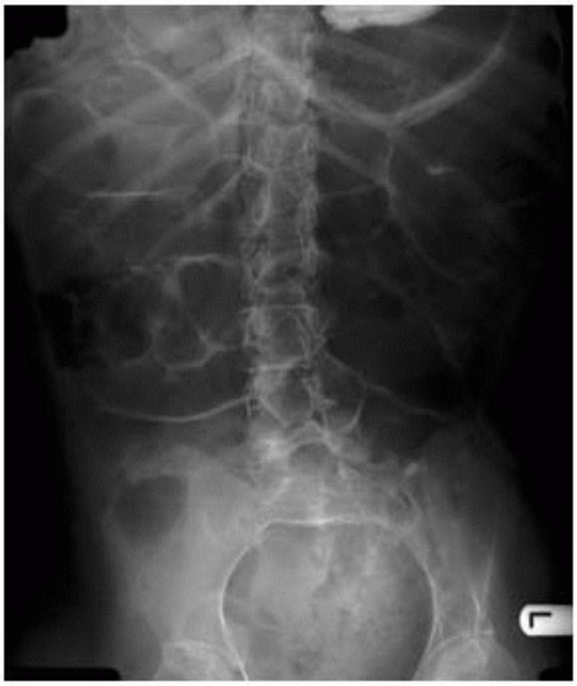


Fig. 18.57 An AXR showing a severe pneumoperitoneum. Note how the bowel wall can be clearly seen that it has air both on the outside and the inside.

Fig. 18.58 An AXR showing calcification in the gall bladder.

Bones

Examine the bones, looking for secondary malignant disease (possibly seen as rounded lucent lesions), degenerative changes, and osteoporosis (cortical thinning).

Remember to look at all the bones and joints within the field including:

- Pelvis.
- Vertebrae.

- Lowermost ribs.
- Sacroiliac joints.
- Femoral heads.

Soft tissues

Scan the film and identify:

- The lower borders of the liver and spleen.
- Renal outlines.
- Psoas muscles.

Masses

Abdominal masses are frequently revealed by the displacements or distortions of normal viscera.

Distended bladder is the most common mass encountered in the pelvis but a mass here may alternatively be ovarian or uterine pathology.

Gross liver enlargement may be indicated by a large mass in the right side of the abdomen which may extend to the iliac crest. Look for an absence of gut shadows and increased density here.

Gross splenic enlargement is seen as a mass extending inferomedially from the left upper quadrant. There may be elevation of the left hemidiaphragm and downward displacement of the left kidney and stomach. There may also be associated liver and lymph node enlargement.

Normal *renal* length is ~3-3.5 lumbar vertebrae plus their discs. The left kidney is usually higher and slightly larger than the right. A discrepancy of more than 1.5cm between the 2 sides should be viewed with suspicion.

▸ The loss of the psoas margin or renal outline generally indicates an inflammatory condition in the retroperitoneum.

Radiology: the pelvis

Normal

The symphysis pubis joint space should be no wider than 5mm and there should be alignment of the pubic rami. Look closely at the main pelvic ring and the two obturator rings to exclude any abnormality in the bony cortex. Examine the sacroiliac joints to ensure consistent width on either side. The sacral foramina should appear symmetrical and undisrupted.

Pelvic fractures

Stable

- Avulsion fractures: e.g. ischial tuberosity (hamstring avulsion).
- Iliac wing fracture: Duverney's.
- Sacral fractures: Denis classification.
- Ischiopubic rami fractures.
- Acetabular fractures.
- Coccygeal fractures: X-rays *not* indicated here as they will not change management.

 Avulsion fractures are often due to forceful muscle contraction.

Unstable

(Pelvic ring disrupted in 2 or more places).

- *Straddle fracture:* both obturator rings.
- *Bucket-handle fracture:* sacroiliac and contralateral ischiopubic rami.
- *Malgaigne fracture:* sacroiliac and ipsilateral ischiopubic rami.

- *Open book fracture:* both sacroiliacs and both ischiopubic rami.

Hints

▸ If you detect a fracture of the main pelvic ring, look hard to exclude a second disruption to the ring—either a fracture or joint widening at the symphysis pubis/sacroiliacs.

▸ Pelvic fractures can result in severe damage to internal organs.

Paediatrics

- *Normal development:* be careful not to misinterpret the normal cartilaginous junction (synchondrosis) between pubic and ischial bones as a fracture.

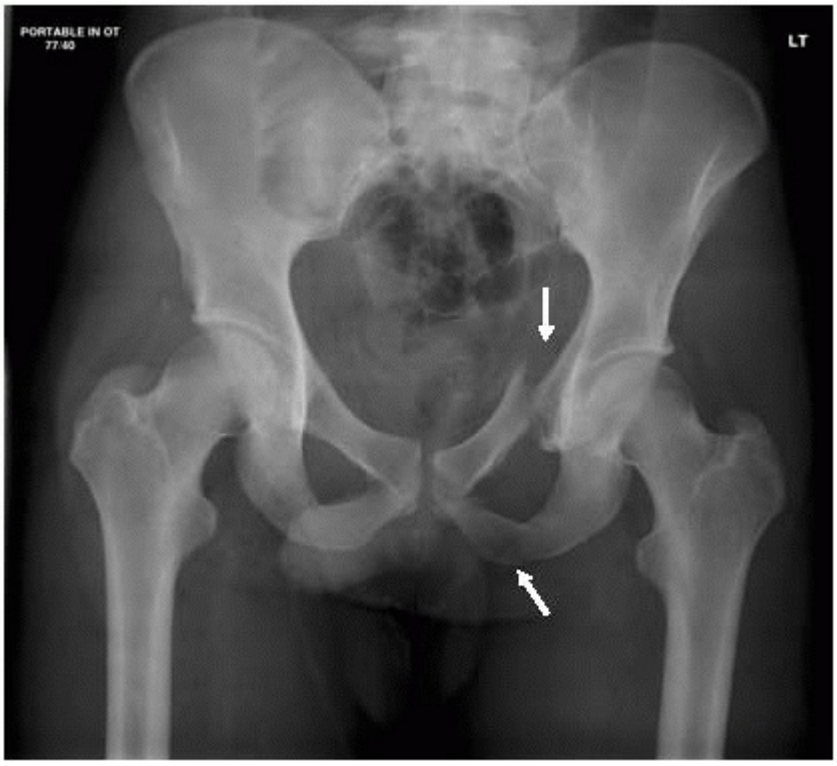


Fig. 18.59 Radiograph showing fractured pubic rami on the left. Note that both the superior and inferior pubic rami are broken—it is not usually possible to break only one.

Radiology: the hips and femurs

Normal

The joint space between the femoral head and acetabulum should not appear narrowed at any point. Examine the femoral neck for a normal trabecular (bony 'mesh') pattern and absence of sclerotic areas. Look for a smooth, undamaged cortex as you trace the outline of the proximal femur and femoral shaft.

Fractures

Fractures of the proximal femur are common in the elderly and carry significant mortality risk. Avascular necrosis is a potential complication if the fracture involves the femoral neck and results from disruption of the blood supply to the femoral head (retinacular vessels). Femoral neck fractures can be graded on their radiological appearance using the *Garden classification*.

Box 18.10 The Garden classification

- I—fracture line across neck stops short of inferior cortex. No displacement. Trabecular angulation across fracture.
- II—fracture extends right across neck. No displacement. No Trabecular angulation (no deviation of bony 'mesh' lines).

- III—fracture extends right across neck. Some displacement and/or rotation of femoral head.
- IV—femoral head severely (often completely) displaced.

Other types of proximal femur fractures include capital (head of femur), intertrochanteric (between the greater and lesser trochanters), and shaft of femur.

Hints

▸ Remember to ask for lateral, as well as AP views, when requesting hip X-rays.

'Hip fracture' is used loosely—but incorrectly—to describe all proximal femoral fractures. Strictly speaking it only relates to intracapsular fractures (those within the joint capsule).

Fractures described as subcapital (just below the femoral head) and transcervical (across the femoral neck) are both intracapsular fractures.

Degenerative changes

Classical X-ray changes in osteoarthritic degenerative disease at the hip—as with other joints—include:

- ↓ joint space.
- Osteophyte formation (bony spurs).
- Bone cysts.
- Subarticular sclerosis.

Dislocation of hip joint

90% are posterior dislocations, with the femoral head seen superior and lateral to the acetabulum. Check for sciatic nerve injury (10%).

Posterior dislocation strongly suggests major force applied—consider trauma elsewhere.

Paediatrics

- *Slipped femoral epiphysis:*—look for misalignment on lateral X-ray of shaft and neck of femur with the epiphysis (teenagers).
- *Perthes' disease:* (aseptic necrosis of femoral epiphysis)—look for sclerosis and flattening of epiphysis and cysts in metaphysic (pre-teens).
- *Developmental dysplasia of the hip:* ('congenital dislocation'). Femoral head fails to lie within acetabulum. If missed, osteoarthritis and gait defects ensue. Diagnosed neonatally with ultrasound.

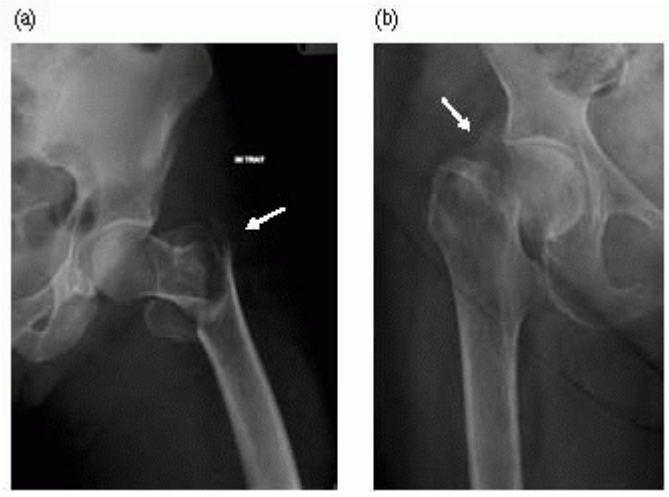


Fig. 18.60 Close-up of radiographs showing (a) an intertrochanteric fracture on the left; (b) fractured neck of femur.

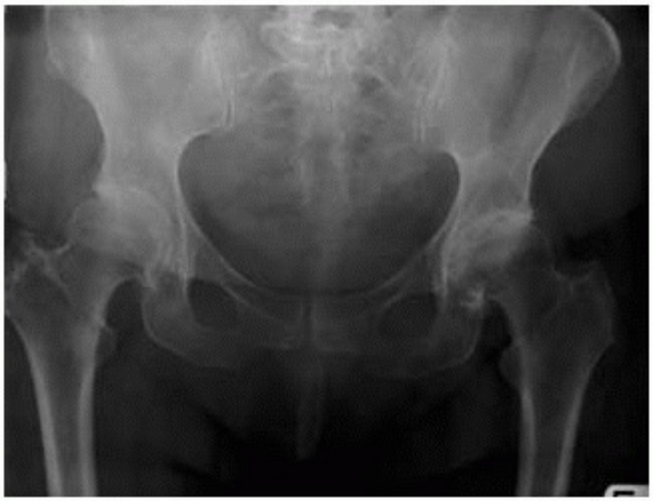


Fig. 18.61 Radiograph showing severe degenerative changes at both hips.

Radiology: the knees

Normal

AP and lateral views are provided as standard. The joint space should be maintained across the joint. On AP film the lateral tibial condyle margin should be no more than 5mm outside the femoral condyle margin. On lateral film the distance from the tibial tuberosity to the inferior aspect of the patella should be similar to the length of the patella.

Abnormal

Distal femur fracture

All readily identifiable on plain X-ray.

- Supracondylar.
- Condylar (medial or lateral).
- Intercondylar.

Proximal tibial fractures

Often as result of being hit by car. Lateral (80%) tibial plateau fractures more common (think impact site!).

On X-rays, look for an impacted sclerotic area and lateral displacement of tibial condyle, in addition to fracture itself. A lateral view of the knee can show a fat-fluid (marrow-blood) level in the suprapatellar bursa—which may be the only sign of intra-articular fracture.

- Depressed plateau fracture—lateral or medial.
- Split fracture—proximal fibula and lateral tibial condyle both affected.
- Combined split depressed fracture—combination of the 2 above.
- Medial condyle fracture—less common.
- Comminuted fracture of lateral and medial tibial condyles.
- Segond fracture—avulsion fracture superolateral aspect proximal tibia. Associated with ligamentous and meniscal injury.
- Intercondylar eminence fracture (tibial spine)—associated with anterior cruciate ligament injury.

Patella

Transverse, vertical, comminuted, or avulsion fractures. Oblique or skyline views may be required to identify some vertical fractures.

Patella dislocation is usually lateral. Rupture of patellar ligament leads to superior displacement of patella.

Neck of fibula fractures

Think about accompanying ligamentous (collateral and cruciate) injury.

Proximal third of fibula fractures

Look for associated ankle fracture.

Degenerative changes

Knee joint changes are similar to those at the hip.

Knee dislocation

75% posterior. Potential for significant injury to popliteal and peroneal blood vessels.

Hints

Most meniscal or ligamentous injuries have no X-ray changes.

▸ Remember to examine the hip when patients complain of a painful knee—it could be referred pain.

Paediatrics

- *Bipartite patella:* normal variant. Secondary ossification centre, usually in superiolateral region. May stay unfused throughout adulthood. Do not confuse with fracture.
- *Osgood-Schlatter disease:* recurrent minor trauma leads to swelling and pain at quadriceps tendon insertion. Avulsion of tibial tubercle may be seen on X-ray. Boys > girls.
- *Osteochondritis dissecans:* chronic trauma causes articular cartilage and subchondral bone disruption. Most commonly seen at lateral side of medial femoral epicondyle. Remember the mnemonic 'LAME'—'Lateral Aspect Medial Epicondyl'.

Radiology: the shoulder

AP and either axial (armpit view), lateral scapular (Y view), or apical oblique views are usually provided.

Abnormal

Glenohumeral dislocation

- *Anterior dislocations:* (95%) humeral head lies anterior and inferior to glenoid.
- *Posterior dislocation:* often missed. Look for 'light bulb' sign of rounded symmetrical humeral head (due to internal rotation).
- *Inferior dislocation (luxatio erecta):* rare (neurovascular damage more likely).

Fractures

- *Clavicle fracture:* readily identified on AP film. 80% lateral 1/3.
- *Fracture-dislocations:* humeral greater tuberosity often involved.
- *Humeral head/neck fracture:* oblique or impacted fractures seen on X-ray.
- *Humeral shaft fracture:* transverse, spiral, or comminuted.
- *Scapula fracture:* uncommon. May require trans-scapular view.
- *Supraspinatus rupture:* may see bony avulsion on X-ray.

Acromio-clavicular (AC) injury

On the AP view the inferior cortex of the clavicle and that of the acromion process should be aligned. Normal AC distance should be ≤8mm. Look for widening of AC joint and any associated fractures.

Graded according to degree of separation:

- *Grade I:* ligamentous involvement only. Slight separation.
- *Grade II:* subluxation but with some bony overlap.
- *Grade III:* AC joint fully dislocated. Coracoclavicular ligament ruptured.

Adhesive capsulitis (frozen shoulder)

On X-ray, joint space narrowing and osteoporotic changes due to disuse.

Sternoclavicular dislocation

Anterior dislocations more common. Risk of injury to large vessels and trachea with posterior dislocation.

Calcific tendonitis

Exquisitely painful, rapid relief with local steroidal injections. See abnormal calcium in soft tissues around the joint on X-ray.

Hints

- ▸ AP views of posterior dislocation may appear normal. Axillary view may be helpful.
- Fracture of glenoid can be a complication of anterior dislocation.
- Look for any fracture fragments with anterior dislocation.

Stress views (giving patient weight to hold on affected side) can make AC injury more obvious on X-ray.

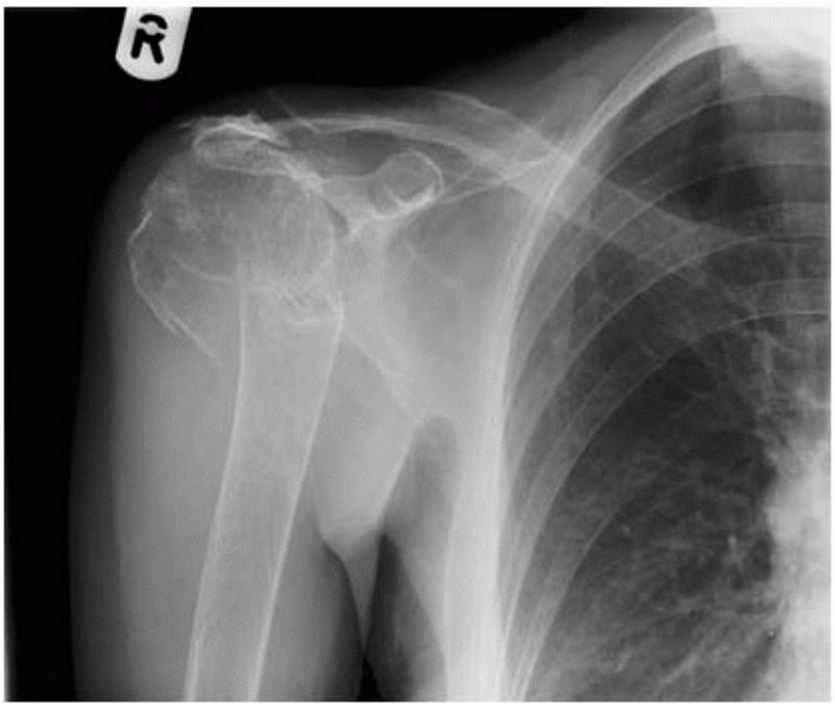


Fig. 18.62 Radiograph showing fractured neck of humerus on the right.

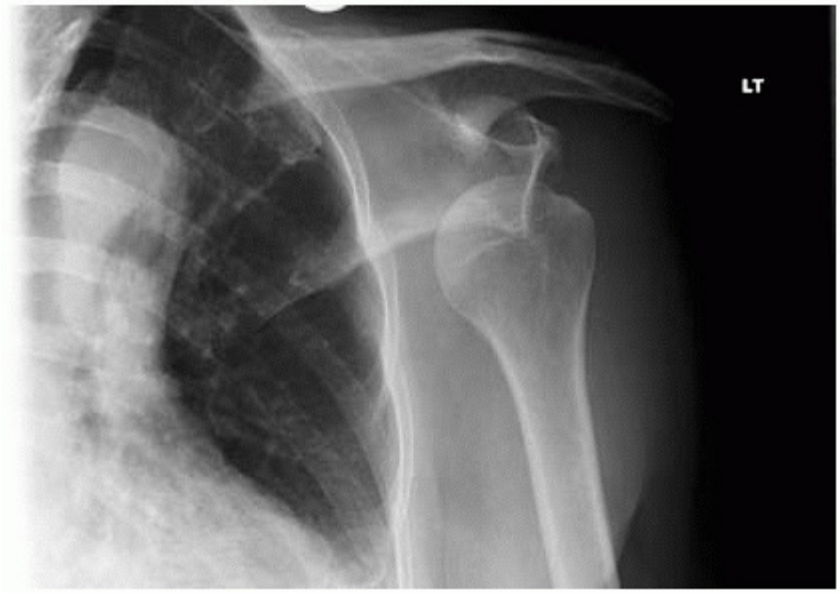


Fig. 18.63 Radiograph showing anterior dislocation of the humerus on the left.

Radiology: the cervical spine

Examining C-spine films

Three views are advisable—lateral, AP, and open-mouth (the latter to get a clear view of the odontoid peg). Remember, the peg is the upward extension of the body of C2—the 'axis'.

Lateral view

- Can you see all 7 cervical vertebrae including the top of T1?
- Trace 3 superimposed 'parallel' curved lines running down the neck to look for steps or misalignments (see Fig. 18.64).
- Check the gap between the anterior aspect of the peg and anterior arch of C1—should be ≤3mm in adults ≤5mm in children.
- Look for prevertebral soft tissue swelling. This may be only sign of a C-spine fracture (lack of soft tissue swelling does not exclude fracture).
- Inspect for narrowing of disc spaces.

AP view

- Ensure the spinous processes are aligned (note: some may be bifid).
- Measure the gaps between spinous processes. These should be approximately equal (but are allowed to be up to 50% wider than the gaps directly above or below).

Open-mouth (odontoid peg) view

- Check that the peg is intact.
- Are the lateral aspects of C1 and C2 aligned?
- Look for equal gaps on both sides of the peg.

C-spine injuries

Stable

- Unilateral facet dislocation: spinous processes misaligned on AP.
- Posterior arch of C1 fracture.
- Wedge fracture: ↓ height anterior vertebral body. Delayed instability possible.
- Extension teardrop fracture: avulsion fracture anteroinferior aspect C2.
- Burst fracture: compression fracture between C3-C7.
- Anterior subluxation: fanning (widening) seen between adjacent spinous processes on lateral view. Delayed instability possible.
- Clay-shoveler's fracture: oblique avulsion fracture lower spinous process.

Unstable

- Bilateral facet dislocation.
- Odontoid fracture.
- Hangman's: fracture of C2 pedicles.
- Jefferson's: fracture to anterior and posterior arches of C1.
- Flexion teardrop fracture: fracture-dislocation of vertebral body.
- Hyperextension fracture-dislocation: anterior displacement of vertebra common.
- Atlanto-occipital disassociation (remember ‘atlanto’ refers to C1).

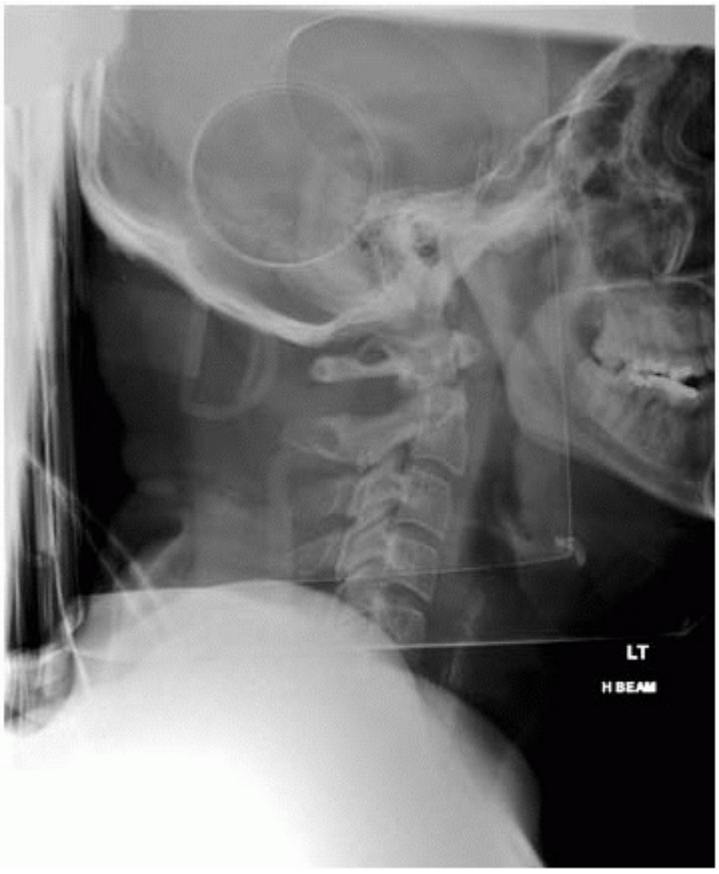


Fig.18.64 Radiograph showing a fractured odontoid peg. Note that the first cervical vertebra is displaced posteriorly. You can also see several additional lines on the film caused by the head and neck brace that the patient is in.

Paediatrics

- *Normal variant:* Particularly in children the base of C2 spinous process may lie ≤2mm posterior to the curved line linking the other bases.

- *Pseudosubluxation:* normal ligamentous laxity causes anterior displacement of C2 or C3.

Hints

▸ Above is only a guide to stability.

If you see one fracture look for another—20% are multiple.

- Most C-spine fractures occur near top (C1-C2) and bottom (C5-C7) of the neck.

Radiology: the thoracic and lumbar spine

Examining spinal films

Lateral and AP views are provided as standard.

- Examine the height of each vertebrae—should be approximately equal and the same front and back.
- Look for the normal concave shape of the back of each vertebral body. The gaps between vertebrae (the disc spaces) should be equal.
- Trace the lines of the anterior and posterior vertebral bodies. Look for any steps, ridges, or misalignments?
- On the AP view, the lumbar pedicles should become slightly further apart going down the spine.

Abnormalities

Thoracic and lumbar spine fractures

- Wedge (compression) fracture.
  - ↓ height of vertebral body with wedge shape.
  - Loss of concave shape to posterior vertebral body.
- Burst fracture: vertebral body crushed into >2 pieces (comminuted).
- Fracture-dislocations: high suspicion of spinal cord injury.
- Chance fracture: horizontal splitting of vertebra.
- Transverse process fracture.
- Spinous process fracture.

Spondylolysis

Degeneration of intervertebral discs leads to narrowing of disc space and osteophyte formation.

Spondylolisthesis

Anterior subluxation of vertebral body (congenital or after injury).

Hint

90% fractures at T11-L4.

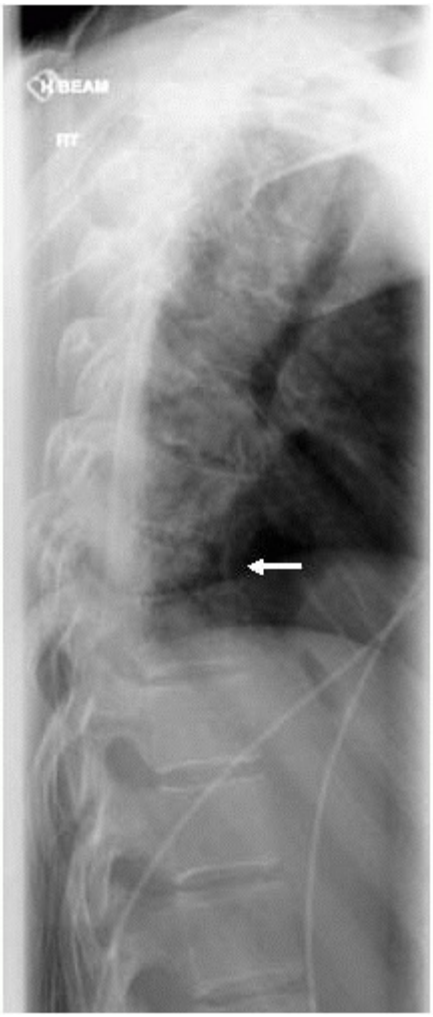


Fig. 18.65 Radiograph showing a wedge fracture of a thoracic vertebra.

Lung function tests

Simple lung function tests can help with the diagnosis and monitoring of common respiratory diseases such as asthma and chronic obstructive pulmonary disease (COPD). Recognizing patterns of abnormality of these tests is an essential skill in data interpretation.

Peak expiratory flow rate

Peak expiratory flow rate (PEFR) is the maximum flow rate recorded during a forced expiration. Predicted readings vary depending on age, sex, height and ethnicity (see Fig. 18.66).

Interpreting PEFR

PEFR readings less than the patient's predicted, or usual best, demonstrate airflow obstruction in the large airways.

PEFR readings are useful in determining the severity, and therefore the most appropriate treatment algorithm, for asthma exacerbations:

- PEFR <75% best or predicted—moderate asthma attack.
- PEFR <50% best or predicted—acute severe asthma attack.
- PEFR <33% best or predicted—life threatening asthma attack.

Note the diurnal variations in PEFR with asthma.

▸ Don't confuse PEFR with FEV_1.

Reversibility testing

Improvement in PEFR or FEV_1 ≥15% following bronchodilator therapy (e.g. salbutamol) shows reversibility of airflow obstruction and can help to distinguish asthma from poorly-reversible conditions such as COPD.

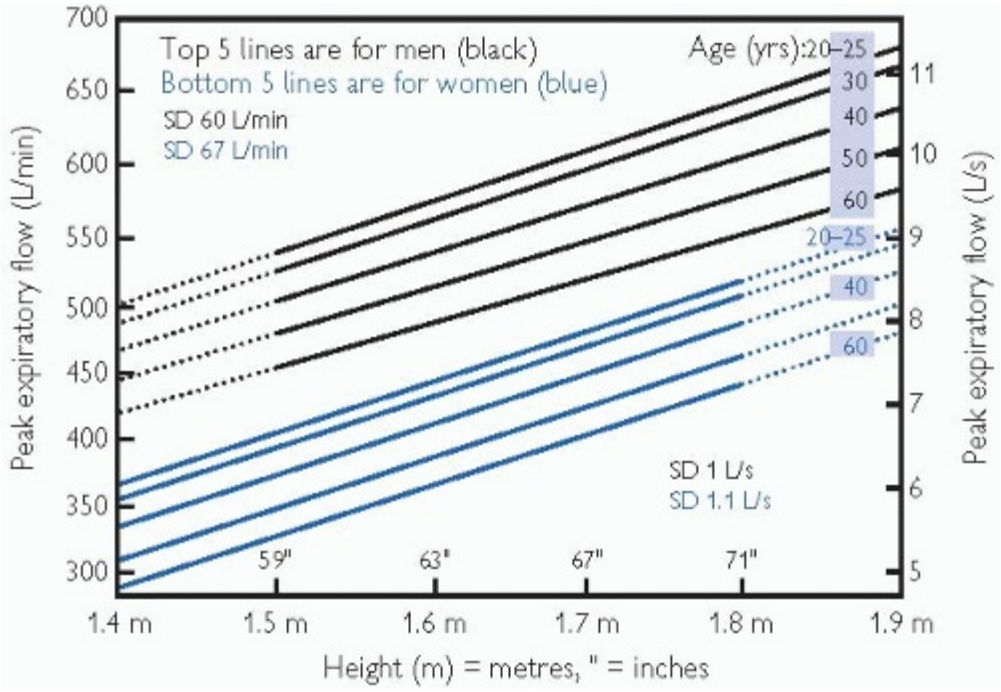


Fig. 18.66 Predicted values of peak expiratory flow rate according to age, sex, and height. Reproduced with permission from Longmore M *et al.* (2004). *Oxford Handbook of Clinical Medicine*, 6th edn. Oxford University Press, Oxford.

Spirometry

Spirometry measures airflow and functional lung volumes. Fig. 18.67 shows the pattern of lung volumes in a healthy individual during normal breathing, and maximum inspiration and expiration with a single breath.

Patients are asked to blow, as fast as possible, into a mouthpiece attached to a spirometer. This records the rate and volume of airflow. Unlike measuring PEFR, patients must continue blowing out for as long as possible. The test is repeated until 2 similar readings are achieved.

Two key values are:

- *FEV_1:* forced expiratory volume in the first second.
- *FVC:* forced vital capacity—the total lung volume from maximum. inspiration to maximum expiration, in forced exhalation.

Modern machines will often calculate the patient's predicted values for FEV_1 and FVC, which, like PEFR, are dependent on age, sex, height, and ethnicity. Usually 70-85% of the FVC is expired in the first second, giving the volume-time graph shown in Fig. 18.68.

Flow volume loops can also be generated from spirometry data and show the flow at different lung volumes. These are useful in distinguishing intra-and extra- thoracic causes of obstruction (see *OHCM6*, p.171) as well as to assess for small airways obstruction.

▸ Spirometry is valuable in assessing anesthetic risk prior to elective surgery and vital for the diagnosis of COPD.

Don't confuse PEFR with FEV_1. While these may correlate well in asthma, a normal PEFR does not exclude a reduced FEV_1 in COPD.

Gas transfer

Measuring the capacity of a gas to diffuse across the alveolar-capillary membrane can provide important information. DLCO (carbon monoxide diffusion capacity) measures the uptake from a single breath of 0.3% CO. Reduced in interstitial lung disease and emphysema.

Other tests

Specialized lung function centres can calculate static lung volumes with a body plethysmograph or using helium rebreath and dilutional techniques including:

- TLC—total lung capacity.

- RV— residual volume.

Both can help when identifying patterns of lung disease and help assess patients prior to lung surgery.

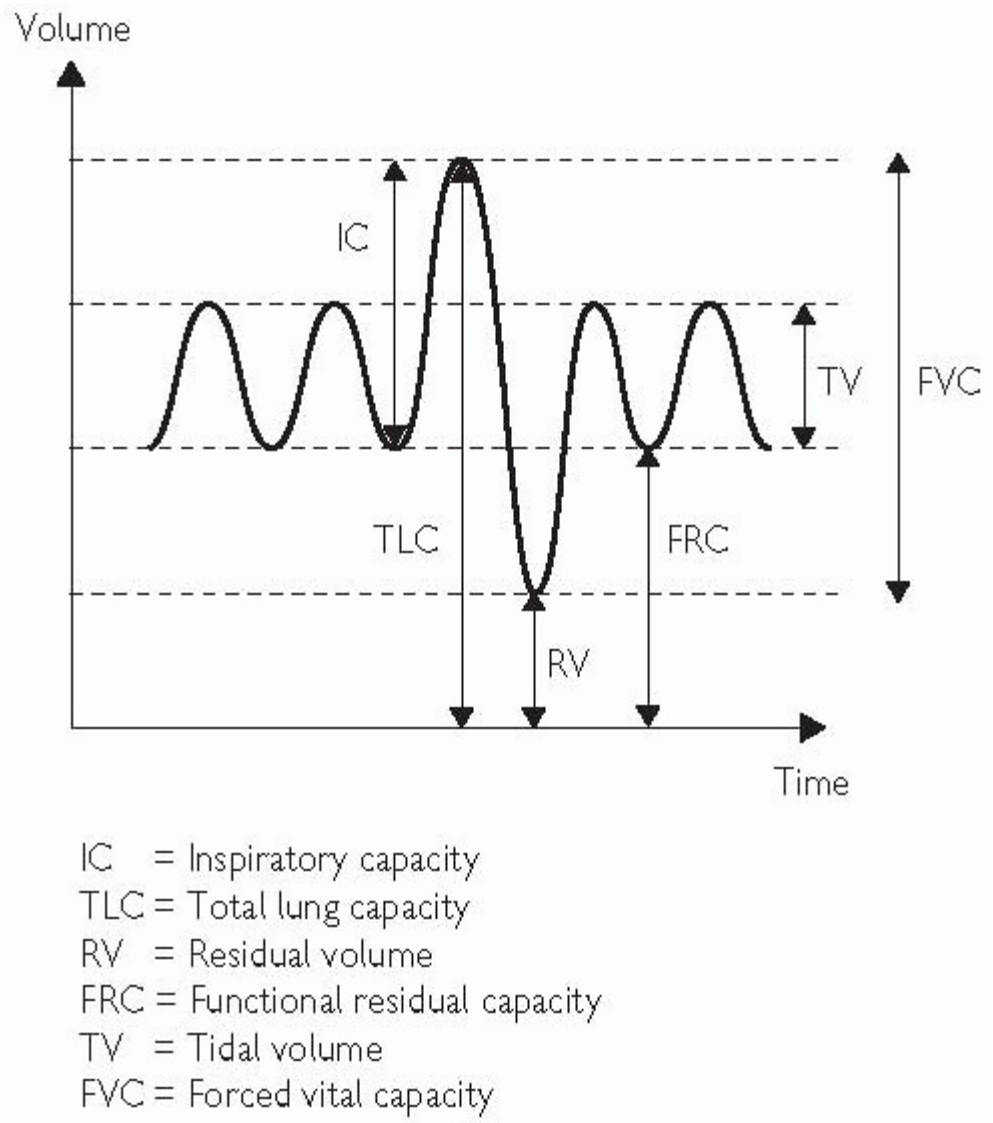


Fig. 18.67 Normal pattern of lung volumes.

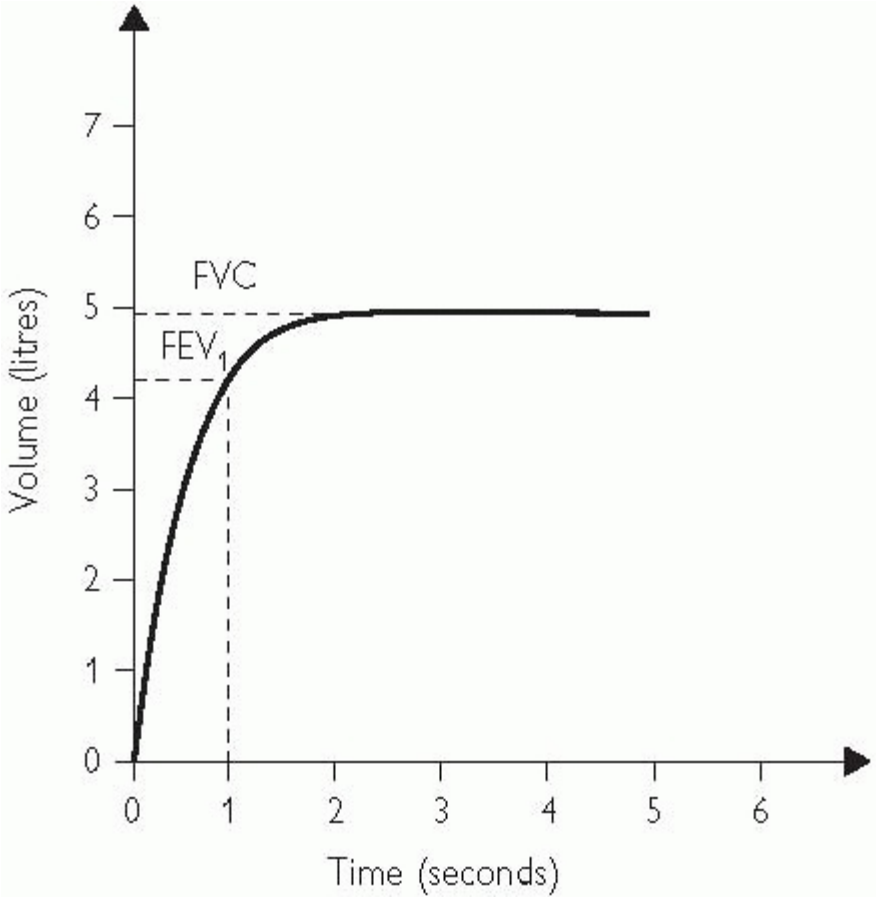


Fig. 18.68 Spirogram showing normal volume-time graph.

Common patterns of abnormality

Abnormal patterns of lung function fall broadly into two types, obstructive and restrictive.

Table 18.1 Obstructive v. restrictive spirometry results

Pattern	FEV_1	FVC	FEV_1/FVC ratio	TLC	RV
Obstructive	↓	↔/↓	<75%	↑(or ↔)	↑
Restrictive	↓	↓	>75%	↓	↓

See Fig. 18.69 for spirograms showing obstructive and restrictive volume-time graphs.

Obstructive

When airflow is obstructed, although FVC may be reduced, FEV_1 is much more reduced, hence the FEV_1/FVC ratio falls. It can also take much longer to fully exhale.

▸ Note that FVC can be normal in mild/moderate obstructive conditions Conditions causing an obstructive defect:

- COPD.
- Asthma.
- Bronchiectasis.
- Cystic fibrosis.
- Foreign bodies, tumours, and stenosis following tracheotomy (all localized airflow obstruction).

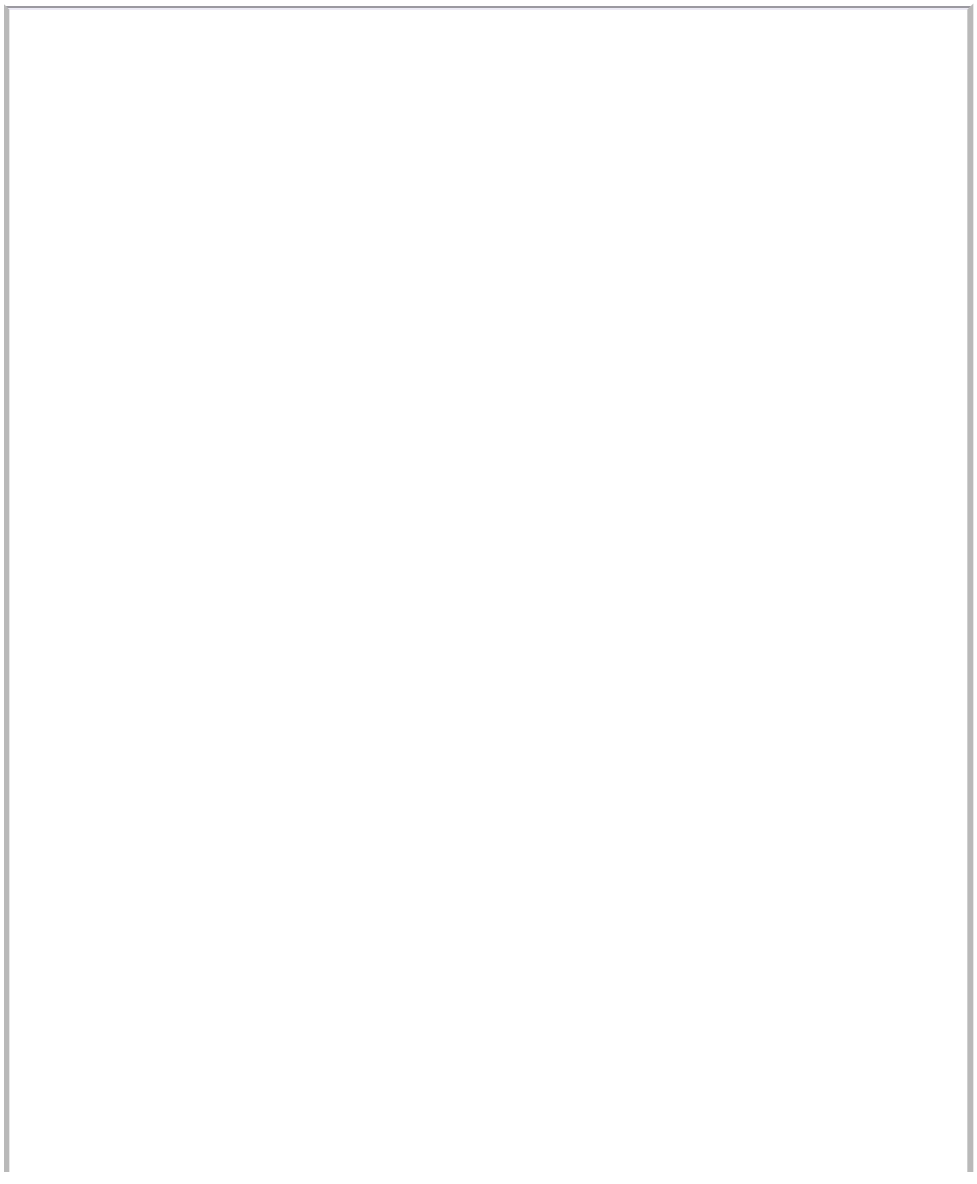
Restrictive

The airway patency is not affected in restrictive lung conditions, so the PEFR can be normal. But the FEV_1 and FVC are reduced due to the restrictive picture.

Conditions causing a restrictive defect:

- Fibrosing alveolitis of any cause.
- Skeletal abnormalities (e.g. kyphoscoliosis).
- Neuromuscular diseases (e.g. motor neuron disease).
- Connective tissue diseases.
- Late-stage sarcoidosis.
- Pleural effusion.
- Pleural thickening.

▸ Mixed obstructive and restrictive defects can also be found.



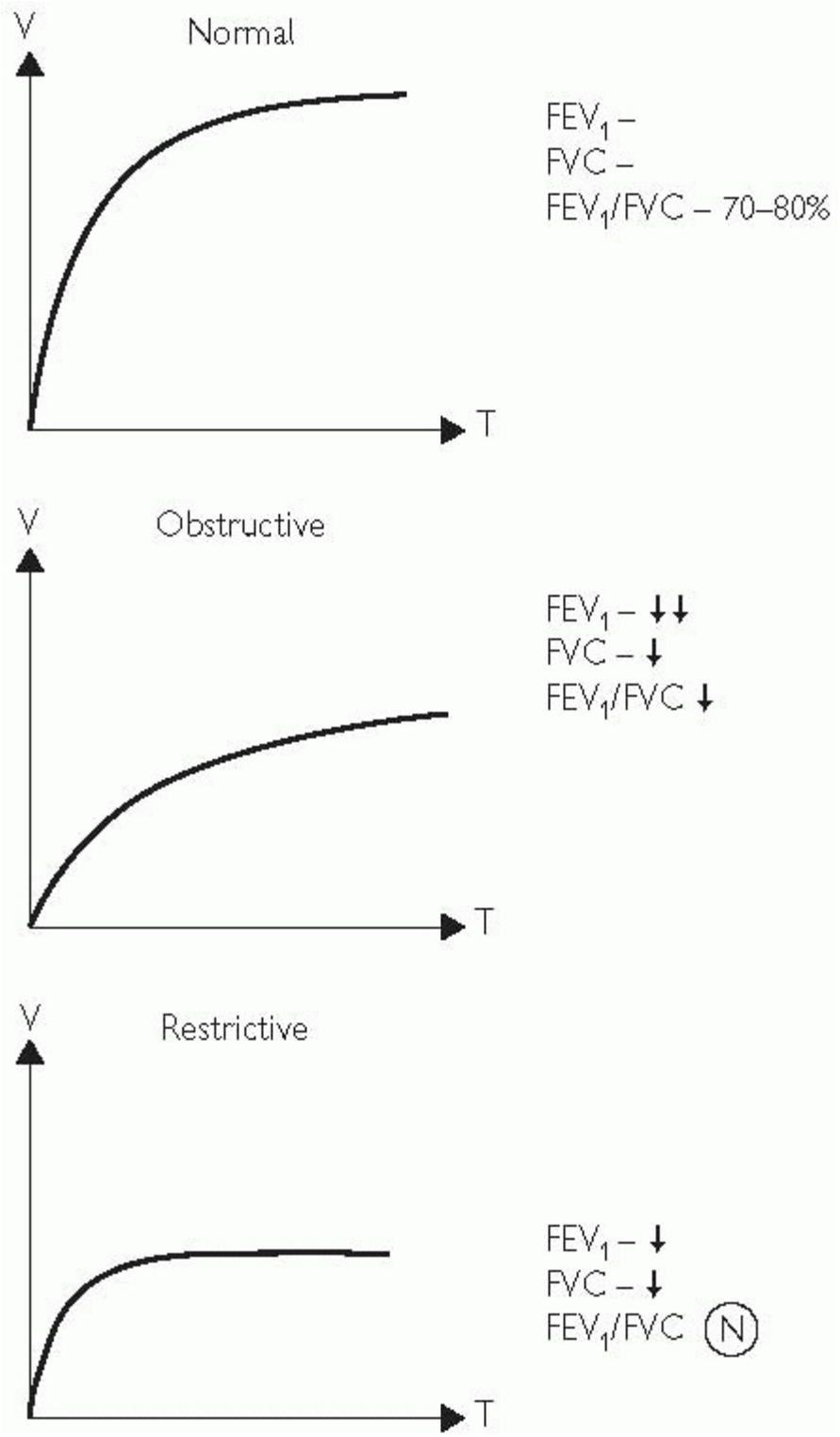


Fig. 18.69 Spirograms showing obstructive and restrictive volume-time graphs.

Arterial blood gas analysis

While clinical examination skills will allow you to detect many important conditions, at times we all need help from the analysis of blood and other fluids. Even the most experienced physician can't work out the $PaCO_2$ just by the laying on of hands (although they might tell you otherwise)!

A systematic approach

The printout from the ABG machine can have a bewildering number of results. Initially, just focus on the pH, $PaCO_2$, and HCO^-_3 in that order…

pH

Is it low (acidosis) or high (alkalosis)?

$PaCO_2$

(Remember, CO_2 is acidic!).

- If $PaCO_2$ is raised and there is acidosis (pH <7.35) you can deduce a *respiratory acidosis*.
- If $PaCO_2$ is low and there is alkalosis (pH >7.45) then the lack of acid gas has led to a *respiratory alkalosis*.
- Conversely, if $PaCO_2$ is low and there is acidosis then the respiratory system will not be to blame and there must instead be a *metabolic acidosis*.
  - Confirm this by looking at the **HCO^-_3,** it should be low—remember it is alkaline and acts as a buffer.
- If $PaCO_2$ is high or normal and there is alkalosis, then again the respiratory system will not be to blame and there must instead be a *metabolic alkalosis*.
  - Confirm this by looking at the **HCO^-_3,** it should be raised.

PaO_2

Note what FiO_2 the patient was breathing when the sample was taken. Hypoxia is PaO_2 of <8.0kPa and can result from V/Q (ventilation-perfusion) mismatch—e.g. pulmonary embolism—or from alveolar hypoventilation—e.g. COPD, pneumonia.

- *Type I respiratory failure:* hypoxia and $PaCO_2$ <6kPa.
- *Type II respiratory failure:* hypoxia and $PaCO_2$ >6kPa.

▸ If the PaO_2 is very low consider venous blood contamination.

Compensatory mechanisms

Mechanisms controlling pH are activated when acid-base imbalances threaten. Thus, renal control of H^+ and HCO^-_3 ion excretion can result in compensatory metabolic changes. Similarly, 'blowing off' or retaining CO_2 via control of respiratory rate can lead to compensatory respiratory changes. This can complicate interpretation of ABG results.

▸ A normal pH does not exclude acid-base disturbance—there may be adequate compensation or a mixed picture.

▸ A compensated picture suggests chronic disease.

Table 18.2. Some acid/base disturbances

Disturbance	pH	$PaCO_2$	HCO^-_3
Respiratory acidosis	↓	↑	↔ (↑ if compensated)
Metabolic acidosis	↓	↔ (↓ if compensated)	↓
Respiratory alkalosis	↑	↓	↔ (↓ if compensated)

Metabolic alkalosis	↑	↔ (↑ if compensated)	↑

Box 18.11 Reference ranges

- pH 7.35-7.45
- $PaCO_2$ 4.5-6.0kPa
- PaO_2 10.5-13.5kPa
- HCO^-_3 24-30mmol/l
- Base excess -2 to +2

The base excess and (standard) HCO^-_3 effectively measure the same thing.

Acidosis

A relative excess of cations (e.g. H^+), unless adequately compensated, will result in acidosis (more correctly acidaemia).

Respiratory acidosis

pH ↓, $PaCO_2$ ↑ (HCO^-_3 may be ↑ if compensated).

Conditions which can lead to respiratory acidosis:

- COPD, asthma, pneumonia, pneumothorax, pulmonary fibrosis.
- Obstructive sleep apnoea.
- Opiate overdose (causing respiratory depression).
- Neuromuscular disorders (e.g. Guillain-Barré, motor neuron disease).
- Skeletal abnormalities (e.g. kyphoscoliosis).
- Congestive cardiac failure.

In COPD, find the patient's normal $PaCO_2$—it may be abnormally high.

Metabolic acidosis

pH ↓, HCO^-_3 ↓ ($PaCO_2$ may be ↓ if compensated).

Anion gap: $(Na^+ + K^+) - (HCO^-_3 + Cl^-)$ Normal range 10-18mmol/l

It is useful to calculate the anion gap to help distinguish causes of metabolic acidosis. ↑ anion gap points to ↑ production of unmeasurable anions (see *OHCM6*, p.682).

Conditions which can lead to metabolic acidosis:

- ↑ anion gap.

- Diabetic ketoacidosis.
- Renal failure (urate).
- Lactic acidosis (tissue hypoxia or excessive exercise).
- Salicylates, ethylene glycol, biguanides.

- Normal anion gap.
  - Chronic diarrhoea, ileostomy (loss of HCO^-_3).
  - Addison's disease.
  - Pancreatic fistulae.
  - Renal tubular acidosis.
  - Acetazolamide treatment (loss of HCO^-_3).

Box 18.12 ***Example of respiratory acidosis (on room air)***
pH 7.29, $PaCO_2$ 7.9, PaO_2 7.0, HCO^-_3 35
Cause in this case: acute exacerbation of COPD.

Box 18.13 ***Example of metabolic acidosis (on room air)***
pH 7.15, $PaCO_2$ 2.5, PaO_2 15.1, HCO^-_3 10
Na^+ 135, K^+ 5.4, Cl^- 106 Anion gap: 24.4 (↑)
Cause in this case: diabetic ketoacidosis.

Alkalosis

A relative excess of anions (e.g. HCO^-_3), unless adequately compensated, will result in alkalosis (more correctly alkalaemia)

Respiratory alkalosis

pH ↑, $PaCO_2$ ↓ (HCO^-_3 may be ↓ if compensated). Conditions which can lead to respiratory alkalosis:

- Hyperventilation, secondary to:
  - Panic attack (anxiety).
  - Pain.
- Meningitis.
- Stroke, subarachnoid haemorrhage.
- High altitude.

▸ Sensory changes (tingling in hands, face, lips) with hyperventilation are due to ↓ ionized calcium.

Metabolic alkalosis

pH ↑, HCO^-_3 ↑ ($PaCO_2$ may be ↑ if compensated). Conditions which can lead to metabolic alkalosis:

- Diuretic drugs (via loss of K^+).
- Prolonged vomiting (via acid replacement and release of HCO^-_3).
- Burns.
- Base ingestion.

Mixed metabolic and respiratory disturbance

In clinical practice patients can develop a mixed picture where acid-base imbalance is the result of both respiratory and metabolic factors. For example, in critically ill patients hypoventilation leads to ↓ PaO_2, and O_2 depleted cells then produce lactic acid.

Box 18.14 ***Example of respiratory alkalosis (on room air)***

pH 7.53, $PaCO_2$ 3.1, PaO_2 15.1, HCO^-_3 25

Cause in this example: anxiety.

Box 18.15 ***Example of metabolic alkalosis (on room air)***

pH 7.50, $PaCO_2$ 5.9, PaO_2 11.8, HCO^-_3 35

Cause in this example: vomiting.

Box 18.16 ***Example of mixed respiratory and metabolic acidosis (on room air)***

pH 7.29, $PaCO_2$ 7.8, PaO_2 7.0, HCO^-_3 17

Cause in this example: septic shock.

Cerebrospinal fluid (CSF)

CSF is produced by the choroid plexus lining the cerebral ventricles and helps cushion and support the brain. Samples are usually obtained by lumbar puncture.

Normal adult CSF

- Pressure 6-20cm H_2O
- Red cells Nil
- Lymphocytes ≤5 × 10^6/L
- Neutrophils Nil
- Protein <450mg/L
- Glucose 2.5-4.0mmol/L (2/3 of blood glucose)
- IgG 5-45mg/L

▸ CSF glucose is abnormal if < 50% of blood glucose level

Premature babies, newborns, children, and adolescents have different normal ranges

Interpreting CSF

Table 18.3 Characteristics of CSF according to underlying pathology

Pathology	Appearance	Protein	Glucose (CSF:blood ratio)	Cells
Bacterial meningitis	Turbid	↑	↓	Neutrophils
Viral meningitis	Clear	↔/↑	↑/↔	Lymphocytes
Viral encephalitis	Clear	↔/↑	↓	Lymphocytes
TB meningitis	Fibrin webs	↑	↓ ↓	Lymphocytes Neutrophils
Fungal meningitis	Clear/turbid	↑	↓	Lymphocytes
Subarachnoid haemorrhage	Xanthochromia	↔/↑	↑	Red cells
Multiple sclerosis	Clear	↔/↑	↔/↑	Lymphocytes
Guillain-Barré syndrome	Clear	↑	↔/↑	
Cord compression	Clear	↑	↔	
Malignancy	Clear	↑	↓	Malignant

▸ This table is only a general guide. Be guided by CSF results together with the clinical picture.

Box 18.17 Further CSF tests

- Culture.

- Gram stain.
- Ziehl-Neelsen stain (TB).
- India ink (cryptococcus).
- Electrophoresis (oligoclonal bands in multiple sclerosis).
- Cytology (malignant cells).
- Serological tests (syphilis).
- Viral PCR.

Urinalysis

Bedside dipstick urinalysis offers speedy and non-invasive testing that can help with the diagnosis of common conditions such as UTIs and diabetes mellitus. Samples can be sent to the laboratory for further analysis, including MCS.

Box 18.18 Methods of collecting urine for analysis

- Random sample (contamination likely).
- Mid-stream sample (less contamination).
- First void urine (for intracellular organisms—e.g. Chlamydia).
- Early morning sample (to look for acid-alcohol fast bacilli—TB).
- Suprapubic aspiration.
- Via catheter bag.
- Via urine bag (paediatrics).
- 24 hour collection.

Dipstick

Dipstick testing gives semi-quantitative analysis of:

- Protein (normally negative).
- Glucose (normally negative).
- Ketones (normally negative).
- Nitrites (normally negative).
- Blood (normally negative).
- Leucocytes (normally negative).
- Bilirubin (normally negative).
- pH (normally acidic with range 4.5-8.0).
- Specific gravity (normal range 1.000-1.030).

▸ Test the urine within 15 minutes of obtaining the sample.

Colour vision must be intact to interpret the dipstick chart.

▸ Urine pregnancy testing is equally convenient and is indicated in females of child-bearing age who present with abdominal symptoms.

▸ Various foods (e.g. beetroot) and drugs (e.g. rifampicin, tetracyclines, levodopa, phenytoin, chloroquine, iron supplements) can change the colour of urine.

Microscopy, culture, and sensitivity (MCS)

Microscopy allows identification of bacteria and other microorganisms, urinary casts (formed in the tubules or collecting ducts from proteins or cells), crystals, and cells (including renal tubular, transitional epithelial, leucocytes and red blood cells). Organism growth and antibiotic sensitivities and can also be determined. See *OHCM6*, p.262.

▸ MCS may not be indicated for patients with uncomplicated UTI diagnosed on clinical history and dipstick, and commenced on antibiotics.

▸ Asymptomatic bacteriuria is more common in pregnancy (up to 7%) and can lead to pyelonephritis and potential fetal complications (see

OHCS6).

Characteristic urinalysis findings

- *UTIs:* nitrites, leucocytes.
- *Diabetes mellitus:* glucose.
- *Diabetic ketoacidosis:* ketones.
- *Cholestasis (obstructive jaundice):* bilirubin.
- *Pre-hepatic jaundice:* urobilinogen.
- *Glomerulonephritis:* protein, blood.
- *Renal stones:* blood.
- *Renal carcinoma:* blood.
- *Nephrotic syndrome*: protein ++.
- *Renal TB:* leucocytes, no organisms grown ('sterile pyuria').
- *Sexually transmitted infections (chlamydia, gonorrhoea):* sterile pyuria.

▸ Suspected UTIs in children must be investigated (see *OHCS6*).

Pleural and ascitic fluid

Pleural effusion

Fluid in the pleural space can be classified as *exudate* (protein content >30g/L) or *transudate* (protein content <30g/L). At borderline levels, if the pleural protein is >50% serum protein then the effusion is an exudate. Blood, pus, and chyle (lymph with fat) can also form an effusion. See p.614 for pleural tap guidance.

Box 18.19 Tests

- Microscopy, culture (conventional ± TB culture) and sensitivity (Gram stain, Ziehl-Nielsen stain).
- Cytology (malignant cells).
- Biochemistry.
  - Protein.
  - Glucose (↓ if rheumatoid or pneumonia related).
  - Amylase (↑ in pancreatitis).
  - LDH (lactate dehydrogenase—↑ in empyema, malignancy, rheumatoid).

- ▸ A large effusion needs a chest drain.
- ▸ If you suspect malignant pleural disease, consider pleural biopsy.
- Unilateral effusion suggests localized disease (malignancy, pneumonia).
- Pleural fluid may be purulent (e.g. empyema) and therefore difficult to aspirate.

Transudate causes

(↑ venous or ↓ oncotic pressure.)

- Heart failure.
- Hypoproteinaemia (liver failure, malabsorption, nephrotic syndrome).
- Hypothyroidism.
- Constrictive pericarditis.

- Meig's syndrome (ovarian fibroma + pleural effusion).

Exudate causes

(↑ capillary permeability.)

- Pneumonia.
- Empyema.
- Malignancy (lung, pleura, lymph).
- Pulmonary infarction.
- TB.
- Systemic lupus erythematosus (SLE).
- Rheumatoid arthritis.
- Dressler's syndrome (post MI).

Ascites

Fluid in the peritoneal cavity can result in abdominal distension and breathlessness. As with pleural fluid, analysis of an aspirated sample can aid diagnosis. See ascitic tap guidance.

Box 18.20 Tests

- MCS (bacterial peritonitis, TB).
- Cytology (malignant cells, macrophages in inflammatory diseases)
- Biochemistry (amylase, protein).

- ▸ Spontaneous bacterial peritonitis = neutrophils > 250/mm^3 (see *OHCM6*, p.230).
- ▸ Further tests you may consider for a patient with ascites include: LFTs, hepatitis serology, anti-mitochondrial antibodies (primary biliary cirrhosis), USS of liver/pelvis, OGD (varices).
- Useful to check platelets and clotting profile before inserting drain.
- Chylous ascites can result from obstruction of lymph drainage.

Causes

- Decompensated liver disease.
- Infection (bacterial peritonitis, TB).
- Malignancy (liver, ovary).
- Right sided heart failure.
- Pancreatitis.
- Portal vein occlusion.
- Nephrotic syndrome.

Serum/Ascites Albumin Gradient (SAAG)

Used to classify ascites as exudate or transudate.

SAAG = serum albumin concentration — Ascitic Fluid Albumin Concentration

SAAG >11g/Litre (Transudate)

- Cirrhosis with portal hypertension.
- Cardiac failure.
- Nephrotic syndrome.

SAAG <11g/Litre (Exudate)

- Malignancy.
- Pancreatitis.
- TB.

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