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# Oxford Handbook of CLINICAL EXAMINATION AND PRACTICAL SKILLS

EDITED BY James Thomas | Tanya Monaghan

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data interpretation, and communication skills

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2

# Oxford Handbook of
# Clinical Examination
# and Practical Skills

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# Oxford Handbook of **Clinical Examination and Practical Skills**

## 2nd edition

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# Preface

Since the publication of the first edition of this book, we have been heartened by the many positive comments and emails from readers and have been very grateful for the suggestions for improvements and modifications. We have tried to incorporate as many of these as possible.

We have tried hard to update the text to reflect modern practice and to make changes which, only with the 20–20 vision of hindsight, could we see were needed.

We have tried to keep an eye on OSCE examinations and the reader will find new 'skills stations' throughout the book to add to the existing examination frameworks.

Several chapters, including respiratory, paediatrics, skin, and locomotor have been rewritten from scratch.

We have incorporated new chapters on the eyes, the obstetric assessment.

The 'important presentations' section of each systems chapter has been greatly expanded and referenced to our sister publications the *Oxford Handbooks Clinical Tutor Study Cards*.

The practical procedures chapter has been significantly expanded and updated.

The photographs throughout the book have been updated to reflect modern healthcare dress codes.

There is a brand new chapter on 'other investigations' so that the reader can understand what is involved in common tests and how to prepare patients for them.

Finally, the chapter order has been changed to highlight the importance of the 'core' system examinations of the cardiovascular, respiratory, abdominal, and nervous systems.

As always, we welcome any comments and suggestions for improvement from our reader—this book, after all, is for you.

James D Thomas
Tanya M Monaghan
2013

# Acknowledgements

# Contents

# 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 presenting complaint. These are not exhaustive and are intended as guidance. The history parts of each chapter 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 by a more senior colleague.

### 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 dependent 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

| | |
|---|---|
| ↑ | increased |
| ↓ | decreased |
| ↔ | normal |
| ▶ | this fact or idea is important |
| ~ | approximately |
| ➔ | cross-reference |
| ⓘ | warning |
| ᕫ | website |
| ABG | arterial blood gas |
| ACL | anterior cruciate ligament |
| ACSM | American College of Sports Medicine |
| ACTH | adrenocorticotrophic hormone |
| ADH | antidiuretic hormone |
| ADP | adenosine diphosphate |
| AED | automated external defibrillator |
| AITFL | anterior inferior tibiofibular ligament |
| AMD | age-related macular degeneration |
| AMTS | Abbreviated Mental Test Score |
| ANCOVA | analysis of covariance |
| ANOVA | analysis of variance |
| ANTT | aseptic non-touch technique |
| AP | antero-posterior |
| APC | argon plasma coagulation |
| APH | ante-partum haemorrhage |
| APKD | adult polycystic kidney disease |
| APL | antiphospholipid |
| AS | ankylosing spondylitis |
| ASD | atrial septal defect |
| ASIS | anterior superior iliac spine |
| ATFL | anterior talofibular ligament |
| ATLS | advanced trauma life support |
| ATP | adenosine triphosphate |
| AV | atrioventricular |
| AVN | avascular necrosis |
| a-v$O_2$ diff | arterio venous difference in oxygen concentration |
| BLa | blood lactate |
| BMD | bone mineral density |

| | |
|---|---|
| BMI | body mass index |
| BMR | basal metabolic rate |
| BPV | benign postural vertigo |
| BSL | British Sign Language |
| CABG | coronary artery bypass graft |
| CAH | congenital adrenal hyperplasia |
| CDC | Centers for Disease Control |
| CFL | calcaneofibular ligament |
| CHD | congenital heart disease |
| CHF | congestive heart failure |
| CHO | carbohydrate |
| CISS | Comité International des Sports des Sourds |
| CJD | Creutzfeldt–Jakob disease |
| CKD | chronic kidney disease |
| CMCJ | carpometacarpal joint |
| CNS | central nervous system |
| CON | concentric |
| COPD | chronic obstructive pulmonary disease |
| CP | cerebral palsy |
| CP | creatine phosphate |
| CPAP | continuous positive airways pressure |
| CP-IRSA | Cerebral Palsy International Sport and Recreation Association |
| CPK | creatine phosphokinase |
| CPN | community psychiatric nurse |
| CPPD | calcium pyrophosphate dihydrate deposition disease |
| CPR | cardio-pulmonary resuscitation |
| CRP | C-reactive protein |
| CRVO | central retinal vein occlusion |
| CSF | cerebrospinal fluid |
| CT | computed tomography |
| CTD | connective tissue disease |
| CVD | cardiovascular disease |
| DCO | doping control officer |
| DCS | diffuse cerebral swelling |
| DEXA | dual energy x-ray absorptiometry |
| DIPJ | distal interphalangeal joint |
| DKA | diabetic ketoacidosis |
| DLCO | carbon monoxide diffusion capacity |
| DM | diabetes mellitus |
| DVT | deep vein thrombosis |

| | |
|---|---|
| ECC | eccentric |
| ECG | electrocardiogram |
| ECRB | extensor carpi radialis brevis |
| ECRL | extensor carpi radialis longus |
| ECU | extensor carpi ulnaris |
| EDD | estimated date of delivery |
| EEA | energy expenditure for activity |
| EIA | exercise-induced asthma |
| EIB | exercise-induced bronchospasm |
| EMG | electromyography |
| EMR | endoscopic mucosal resection |
| ENMG | electoneuromyography |
| EPB | extensor pollicis brevis |
| EPO | erythropoietin |
| ESR | erythrocyte sedimentation rate |
| ESRD | end stage renal disease |
| ET | endotracheal |
| ETT | exercise tolerance test |
| EVH | eucapnic voluntary hyperpnoea |
| EWS | early warning score |
| FABER | flexion abduction external rotation |
| FBC | full blood count |
| FCU | flexor carpi ulnaris |
| FDS | flexor digitorum superficialis |
| $FeCO_2$ | expired air carbon dioxide concentration |
| $FeO_2$ | expired air oxygen concentration |
| $FEV_1$ | forced expiratory volume in first second |
| FHR | fetal heart rate |
| FHx | family history |
| $FiO_2$ | fraction of inspired oxygen |
| FNA | fine needle aspiration |
| FPL | flexor policis longus |
| FSH | follicle stimulating hormone |
| FVC | forced vital capacity |
| GA | general anaesthetic |
| GAD | generalized anxiety disorder |
| GALS | gait, arms, legs, spine |
| GCA | giant cell arteritis |
| GCS | Glasgow Coma Scale |
| GFR | glomerular filtration rate |
| GH | growth hormone |

| GnRH | gonadotrophin releasing hormone |
| --- | --- |
| GOJ | gastro-oesophageal junction |
| GORD | gastro-oesophageal reflux disease |
| GTN | glyceryl trinitrate |
| Hb | haemoglobin |
| HCC | hepatocellular carcinoma |
| hCG | human chorionic gonadotropin |
| Hct | haematocrit |
| HDL | high density lipoprotein |
| HE | hepatic encephalopathy |
| HHT | hereditary haemorrhagic telangiectasia |
| HIS | International Headache Society |
| HMB | beta-hydroxy-beta-methylbutyrate |
| HR | heart rate |
| HRT | hormone replacement therapy |
| 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 |
| IJV | internal jugular vein |
| IMB | intermenstrual bleeding |
| INO | internuclear ophthalmoplegia |
| INR | international normalized ratio |
| IUCD | intra-uterine contraceptive device |
| IOC | International Olympic Committee |
| IOP | intraocular pressure |
| IPJ | interphalangeal joint |
| ITB | ilio-tibial band |
| ITBFS | ilio-tibial band friction syndrome |
| IVP | intravenous pyelogram |
| IZ | injury zone |
| JVP | jugular venous pressure |
| LBC | liquid-based cytology |
| LDH | lactate dehydrogenase |
| LDL | low density lipoprotein |
| LH | luteinizing hormone |
| LMA | laryngeal mask airway |

| | |
|---|---|
| LMN | lower motor neuron |
| LMP | last menstrual period |
| LOC | loss of consciousness |
| LRT | lower respiratory tract |
| LSE | left sternal edge |
| LV | left ventricle |
| LVH | left ventricular hypertrophy |
| MANOVA | multivariate analysis of the variance |
| MCD | minimal change disease |
| MCL | medial collateral ligament |
| MCPJ | metacarpophalangeal joint |
| MCS | microscopy, culture, and sensitivity |
| MDI | metered dose inhaler |
| MDT | multi-disciplinary team |
| MELD | model for end-stage liver disease |
| MEN | multiple endocrine neoplasia |
| MG | myasthenia gravis |
| MI | myocardial infarction |
| MMSE | Mini-Mental State Examination |
| MPHR | maximum predicted heart rate |
| MRI | magnetic resonance imaging |
| MRSA | methicillin-resistant *Staphylococcus aureus* |
| MSU | mid-stream urine sample |
| MTPJ | metatarsophalangeal joint |
| MUST | Malnutrition Universal Screening Tool |
| NASH | non-alcoholic steatohepatitis |
| NGB | National Governing Body |
| NIV | non-invasive ventilation |
| NPL | no perception of light |
| NSAIDs | non-steroidal anti-inflammatory drugs |
| NSF | nephrogenic systemic fibrosis |
| OA | osteoarthritis |
| OCD | osteochondritis dissecans |
| OCP | oral contraceptive pill |
| OGD | oesophagogastroduodenoscopy |
| *OHCM9* | *Oxford Handbook of Clinical Medicine* 9th ed. |
| *OHCS9* | *Oxford Handbook of Clinical Specialties* 9th ed. |
| ORIF | open reduction and internal fixation |
| OSA | obstructive sleep apnoea |
| OTC | over-the-counter |
| PC | presenting complaint |

| | |
|---|---|
| PCL | posterior cruciate ligament |
| PCOS | polycystic ovary syndrome |
| PCR | phospho-creatine (energy system) |
| PCS | post-concussion syndrome |
| PDA | patent ductus arteriosus |
| PEFR | peak expiratory flow rate |
| PFJ | patello-femoral joint |
| Pi | inorganic phosphate |
| PIN | posterior interosseous nerve |
| PIPJ | proximal interphalangeal joint |
| PMH | past medical history |
| PNF | proprioneurofacilitation |
| POMS | profile of mood states |
| PPH | post-partum haemorrhage |
| PRICE | protection, rest, ice, compression, elevation |
| PSC | primary sclerosing cholangitis |
| PSIS | posterior superior iliac crest |
| PSYM | parasympathetic |
| PTFL | posterior talofibular ligament |
| PTH | parathyroid hormone |
| PTHrP | parathyroid hormone related protein |
| PV | *per vaginam* |
| Q | cardiac output |
| QID | four times a day (quater in die) |
| QSART | quantitative sudomotor axon reflex tests |
| RA | rheumatoid arthritis |
| RAPD | relative afferent pupillary defect |
| RBBB | right bundle branch block |
| RCC | red cell count |
| ROM | range of movement |
| RPE | retinal pigment epithelium |
| 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 |
| SBAR | situation, background, assessment, recommendation |
| SBP | spontaneous bacterial peritonitis |

| SCAT | Standardised Concussion Assessment Tool |
| --- | --- |
| SCBU | special care baby unit |
| SEM | sports and exercise medicine |
| SFJ | sapheno-femoral junction |
| 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 computed tomography |
| STD | sexually transmitted disease |
| SV | stroke volume |
| SVCO | superior vena cava obstruction |
| SYM | sympathetic |
| TAVI | transcatheter aortic valve implantation |
| TBI | traumatic brain injury |
| TFCC | triangular fibrocartilage complex |
| TGA | transposition of the great arteries |
| TLac | lactate threshold (aerobic/anaerobic threshold) |
| TLC | total lung capacity |
| TNF-$\alpha$ | tumour necrosis factor alpha |
| TOE | trans-oesophageal echo |
| TUE | therapeutic use exemption |
| UCL | ulnar collateral ligament |
| ULTT | upper limb tension test |
| UMN | upper motor neuron |
| URT | upper respiratory tract |
| URTI | upper respiratory tract infection |
| US | ultrasound |
| UT | untrained |
| UV | ultraviolet |
| VA | alveolar ventilation |
| VE | minute ventilation |
| VEGF | vascular derived growth factor |
| VI | visually impaired |
| VIP | vasoactive intestinal polypeptide |
| VMO | vastus medialis obliquus |
| VO$_2$ | oxygen uptake |
| VT | ventricular tachycardia |
| WADA | World Anti-Doping Agency |
| WCC | white cell count |

# 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

Throughout this chapter, 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.

## 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 (see also Box 1.1)*
- 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.

**Becoming a good communicator**

*Learning*

As in all aspects of medicine, learning is a lifelong process. One part of this, particularly relevant to communication skills and at the beginning of your career, is watching others.

The student should take every opportunity to observe doctor–patient and other interactions. Look carefully at how patients are treated by staff that you come across and consider every move that is made . . . is that something that you could try yourself? Would you like to be treated in that way?

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 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. See Box 1.2 for notes on confidentiality.

## 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 no longer part of the medical culture in the UK. National guidance has widely been interpreted as 'bare below the elbow' with no long sleeves or jewellery. This does not mean that you should look scruffy, however. Many hospitals are now adopting uniforms for all their staff which helps solve some potential appearance issues. Fashions in clothing change rapidly but some basic rules still apply:

- Ensure you have a good standard of personal hygiene.
  - Any perfume or deodorant should not be overpowering
  - Many people believe men should be clean-shaven. This is obviously impossible for some religious groups and not a view shared by the authors. Facial hair should, however, be clean and tidy.
- Neutralize any extreme tastes in fashion that you may have.
- Men should usually wear a shirt. If a tie is worn, it should be tucked into the shirt when examining patients.
- 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 on a lanyard around the neck is acceptable depending on hospital policy.
  - Note that lanyards should have a safety mechanism which will allow them to break open if pulled hard. Most hospitals supply these—be cautious about using your own lanyard from a shop or conference
  - Wearing a name badge at the belt means people have to look at your crotch – not necessarily ideal!
- Stethoscopes are best carried—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 techniques to bond.

### 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 1.2 Confidentiality**

As a doctor, healthcare 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. 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. Withholding information from family 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, you should approach the patient and ask their permission, preferably within 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, however juicy the story is.

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 or persons involved.

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### 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. Be very careful to tease out the exact meaning of any pseudo-medical terms that the patient uses.

You may also think that some terms such as 'angina' and 'migraine' are well known—but these are very often misinterpreted.

### 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!

### 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.

### Remember the name

Forgetting someone's name is what we all fear but is 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.

### 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.

### 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. It may be inappropriate if the patient is unable to reciprocate through paralysis or pain. 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.

Remember also that members of some religious groups may be forbidden from touching a member of the opposite sex.

### Introductions

This is a potential 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.

*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.

Introducing yourself by the first name only seems too informal for most medical situations. Some young-looking students and doctors, however, may feel the need to introduce themselves using their title to avoid any misunderstanding of their role—particularly since the demise of the white coat. Perhaps worth considering is a longer introduction using both your names and an explanation along the lines of 'Hello, my name is Jane Smith, I am one of the doctors.'

# 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.

   Be sure that anyone else in the room has also been introduced by name.

### Style of questioning

*Open questions versus closed questions*

Open questions are those where any answer is possible. 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 by 'What sort of pain is it?' or '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, particularly if there are terms used which may have a different meaning to the patient than to you.

### Difficult questions

Apologise for potentially offensive, embarrassing, or upsetting questions ('I'm sorry to have you ask you this, but…').

### Reflective comments

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

### Staying on topic

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

### 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 change 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 your manner and speech according to the patient's educational level.

This can be extremely difficult—you should not make assumptions on intellect or understanding based 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 and remember that apparent aggression may be the patient's coping mechanism, born from a feeling of helplessness or frustration—it is not a personal insult or affront.

### 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, both in their understanding of disease, expectations of healthcare, and in other practices that may affect their health.

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

# 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 alternatively '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

Touching is 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

This refers to 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.

* Watch the first few minutes of the 1998 film 'Primary Colors' which demonstrates the different uses of touch during handshakes.

- *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.

# 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 you 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.

# 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 already mentioned, 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.

# 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.

- 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.

## SBAR

SBAR was created as an easy to remember mechanism to frame conversations and install some uniformity into telephone communication, particularly those requiring a clinician's immediate attention and action. There are 4 sections to help you order the information with the right level of detail and reduce repetition.

*S: Situation*
- Identify yourself (name and designation) and where you are calling from.
- Identify the patient by name and the reason you are calling.
- Describe your concern in one sentence.

Include vital signs where appropriate.

*B: Background*
- State the admission diagnosis and date.
- Explain the background to the current problem.
- Describe any relevant treatment so far.

You should have collected information from the patient's charts, notes, and drug card and have this at your fingertips. Include current medication, allergies, pertinent laboratory results, and other diagnostic tests.

*A: Assessment*
- State your assessment of the patient including vital signs, early warning score (EWS), if relevant, and your overall clinical impression and concerns.

You should have considered what might be the underlying reason for the patient's current condition.

*R: Recommendation*
- 'I think the problem is…'.
- Explain what you need and the time-frame in which you need it.
- Make suggestions and clarify expectations.
- 'Is there anything else I should do?'
- ► Record the name and contact details of the person you have been speaking to.
- ► Record the details of the conversation in the patient's notes.

# Other specific situations

### 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.

### Angry patients

Use body language to take charge of the situation without appearing aggressive. 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.

# 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?

### 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.

### 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:
  - 'What do you know so far?'
  - 'What have the other doctors/nurses told you?'
- You can also ask directly if they want to hear bad news. Say:
  - 'Are you the sort of person who likes to know all the available facts and details or would you rather a short version?'

### 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 stick to the rules above. If asked a direct question, you must be honest and straightforward.

### Warning shots

You should break the news step-wise, 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.

You could start by saying that the test results show things are more 'serious' than first thought and wait to see their reaction. If they ask what you mean, you can tell them more, and so on.

▶ 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.

### 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.

### Don't rush to the positive

When told of bad news, the patient needs a few moments to let the information sink in. 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.

### 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 and is perfectly acceptable to say so. Giving a figure will almost always lead to you being wrong.
- 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.

### Ending the conversation

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!

# Law, ethics, and consent

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-maleficence:* 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. There are a number of circumstances where confidentiality can, or must, be broken. The exact advice varies slightly between different bodies. See the links under 'further reading'. 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 three 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.

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 be, however, 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.

## Powers of attorney

People lacking mental capacity may need someone to manage their legal, financial, and health affairs. This is done through power of attorney as laid out in the Mental Capacity Act 2005.

### Enduring powers of attorney (EPA)
Before 2007, people could grant EPA so a trusted person could manage their finances. Those with EPA do not have the right to make other decisions on a person's behalf.

### Lasting powers of attorney (LPA)
*Property and affairs LPA*
Those with property and affairs LPA can make decisions regarding paying bills, collecting income and benefits, and selling property, subject to any restrictions or conditions the patient may have included.

*Personal welfare LPA*
This allows the 'attorney' to make decisions relating to living situation and other personal care. They can also make medical decisions *if this power has been expressly given in the LPA*.

## Further reading

There are many other complex topics in this area and the law varies between countries and even between regions within the UK. We suggest the following as a good start:
- The British Medical Association: ℜ http://www.bma.org.uk
- The Medical Defence Union: ℜ http://www.the-mdu.com
- The Medical Protection Society: ℜ http://www.medicalprotection.org
- The UK Ministry of Justice: ℜ http://www.justice.gov.uk/
- The UK Department of Health: ℜ http://www.dh.gov.uk

# The history

**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 standard headings. These are not exhaustive and are intended as guidance to supplement a thorough 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 into 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.

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**Box 2.1  The standard history framework**
- Presenting complaint (PC)
- History of presenting complaint (HPC)
- Past medical history (PMH)
- Drug history (DHx)
- Allergies/reactions
- Alcohol
- Smoking
- Family history (FHx)
- Social history (SHx)
- Systematic enquiry.

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The outline in Box 2.1 is the authors' favoured method—slight variations exist. Remember to record the history thoroughly (see Box 2.2). See also notes on collateral histories in Box 2.3.

▶ 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.

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

### 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 two phases:

First, ask an open question 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 in Box 2.4; the additional features regarding 'pain' are in Box 2.5.

See Box 2.6 for notes on long-standing symptoms.

At the end of the history of presenting complaint, you should have established a *problem list*. You should run through this with the patient, summarizing what you have been told and ask them if you have the information *correct* and if there 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 long-standing, 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 SOCRATES**
The questions to ask about the characteristics of pain can be remembered with the mnemonic 'SOCRATES':
- S: Site (where is the pain worse? Ask the patient to point to the site with *one finger*)
- O: Onset (how did it come on? Over how long?)
- C: Character (i.e. 'dull', 'aching', 'stabbing', 'burning', etc.)
- R: Radiation (does the pain move or spread to elsewhere?)
- A: Associated symptoms (e.g. nausea, dyspepsia, shortness of breath)
- T: Timing (duration, course, pattern)
- E: Exacerbating and relieving factors
- S: Severity (scored out of 10, with '10' as the worst pain imaginable).

**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.

# 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 in Box 2.7; see also the notes in Box 2.8.

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.

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**Box 2.7  PMH – ask specifically about:**
- Diabetes
- Rheumatic fever
- Jaundice
- Hypercholesterolaemia
- Hypertension
- Angina
- Myocardial infarction
- Stroke or TIA
- Asthma
- TB
- Epilepsy
- Anaesthetic problems
- Blood transfusions.

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**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, the patient 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.

# Drug history (DHx)

Here, you should list all the medications the patient is taking, including the dose, duration, and frequency of each prescription along with any significant side effects. 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/adherence—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/adherence 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 (record exactly what type of drug, route of administration, site, frequency of use, shared needles).

# Allergies and reactions

This should be documented separately from the 'drug history' 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.

► All true allergies should be clearly recorded in the patient's case notes and drug chart.

# Alcohol

Attempt to quantify, as accurately as you can, the amount and type of alcohol consumed daily/per week—and also establish if the consumption is spread evenly over the week or concentrated into a shorter period.

In the UK, alcohol is quantified in 'units' (1 unit = 10ml of alcohol).

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'.

Units can be calculated as in Box 2.9 and Box 2.10.

If there is a suspicion of excess alcohol consumption, you may wish to use the quick 'CAGE and 'FAST' questionnaires shown in Boxes 2.11 and 2.12.

## Recommended weekly alcohol consumption

- The Royal College of Physicians advises no more than 21 units per week for men and 14 units per week for women.
- The UK Department for Health advises alcohol consumption should not regularly exceed 3–4 units daily for men and 2–3 units daily for women.
- Both men and women should have at least 2 alcohol-free days per week.

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### Box 2.9  Calculating units

- You can work out how many units there are in any drink by multiplying the total volume of a drink (in ml) by its % alcohol by volume (ABV) or 'strength'. Divide the result by 1000
  - (Strength x volume)/1000
- Example:
  - 1 pint (568ml) of strong lager (ABV 5.2%)
  - = (5.2 x 568)/1000
  - = 2.95 units.

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### Box 2.10  Unit content of common drinks

- 1 unit = ½ pint of normal beer, single spirit shot
- 1.5 units = small glass of wine (125ml), bottle of alcopop
- 2 units = large bottle/can/pint normal beer, ½ pint of strong beer, medium glass of wine (175ml)
- 3 units = large bottle/can strong beer, large glass of wine (250ml)
- 9 units = bottle of wine
- 30 units = bottle of spirits.

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**Box 2.11 CAGE questionnaire**

A positive response to any of the four questions may indicate someone at risk of alcohol abuse. A positive answer to two or more questions makes the presence of alcohol dependency likely.

- C: Have you ever felt that you should <u>C</u>ut down your drinking?
- A: Have you ever become <u>A</u>ngry when someone suggested that you should cut down?
- G: Do you ever feel <u>G</u>uilty about your drinking?
- E: Do you ever need an '<u>E</u>ye-opener' in the morning to steady your nerves or get rid of a hangover?

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**Box 2.12 FAST questionnaire (Fast Alcohol Screening Test)**

- This questionnaire is used to identify hazardous drinking
- '1 drink' is defined as '1 unit' or ½ pint of beer, 1 glass of wine or 1 single spirit:

1. Men: How often do you have 8 or more drinks on one occasion?
1. Women: How often do you have 6 or more drinks on one occasion?

Never    Less than monthly    Monthly    Weekly    Daily

2. How often during the last year have you been unable to remember what happened the night before because you had been drinking?

Never    Less than monthly    Monthly    Weekly    Daily

3. How often during the last year have you failed to do what was normally expected of you because of drink?

Never    Less than monthly    Monthly    Weekly    Daily

4. In the last year, has a relative, or friend, or doctor, or other health worker been concerned about your drinking or suggested you cut down?

No    Yes, once    Yes, more than once

*Scoring*

- Question 1: never = not misusing alcohol; weekly/ daily = hazardous or harmful drinking. If other responses, go on to question 2
- Questions 1, 2, and 3: score each answer as 0, 1, 2, 3, 4 with never as 0 and daily as 4
- Question 4: no = 0; yes, once = 2; yes, more than once = 4
- Maximum score = 16.

The patient is misusing alcohol if the total score is more than 3.

# 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).
  - An alternative calculation which gets you the same result: (no. of cigarettes smoked per day x number of years)/20.
- 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. See Box 2.13 for notes on quantification.
- Remember to ask about passive smoking.
  - ❶ Be aware of cultural issues—smoking is forbidden for Sikhs, for example, and they may take offence at the suggestion.

## Health problems related to tobacco

*Cardiovascular*
- Coronary heart disease.
- Peripheral vascular disease.
- Abdominal aortic aneurysm.

*Respiratory*
- COPD.
- Bronchitis.
- Pneumonia.

*Neurological*
- Cerebrovascular disease.

*Sexual*
- Erectile and ejaculatory dysfunction.

*Neoplasias*
- Oral cavity.
- Laryngeal.
- Pharyngeal.
- Bronchial/lung.
- Oesophageal.
- Gastric.
- Pancreatic.
- Renal.
- Cystic.
- Cervical.
- Acute myeloid leukaemia.

*Other*
- Infertility.
- Pre-term delivery.
- Still-birth.
- Low birth weight.
- Sudden infant death syndrome.

### Some conditions where smoking can worsen symptoms

- Asthma.
- Chest infections including tuberculosis.
- Chronic rhinitis.
- Diabetic retinopathy.
- Optic neuritis.
- Hyperthyroidism.
- Multiple sclerosis.
- Crohn's disease.

### Some conditions which smoking increases the risk of

- Dementia.
- Optic neuropathy.
- Cataracts.
- Macular degeneration.
- Pulmonary fibrosis.
- Psoriasis.
- Gum disease.
- Tooth loss.
- Osteoporosis.
- Raynaud's phenomenon.

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**Box 2.13 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-judgemental 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 is worth noting that whilst many conditions run in families, some are due to a single gene disorder. If this is the case (such as Huntington's disease and cystic fibrosis) you should go back several generations for details of consanguinity and racial origins.

It may help to draw a family tree as shown in Box 2.14. 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 well-being.

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)?
- 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 (countries visited, travel vaccination, malaria prophylaxis)?
- Does the patient drive?

**Box 2.14 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 (●, ■).

See Figs 2.1 and 2.2 for examples of family trees.
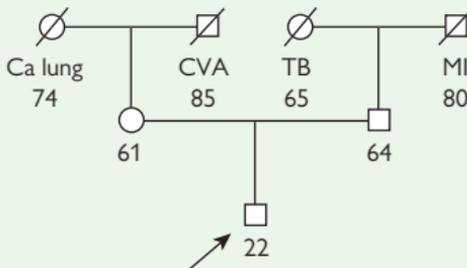


**Fig. 2.1** Our patient is an only child and has no children, his parents are alive but all his grandparents have died of different causes.
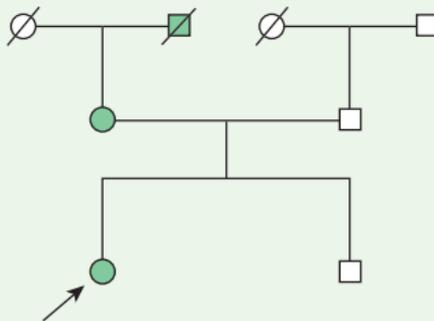


**Fig. 2.2** Our patient suffers from colon cancer and has no children. She has a brother who is well. Her parents are both alive and her mother also has colon cancer. Of her grandparents, only her paternal grandfather is alive. Her maternal grandfather died of colon cancer.

# 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.

*General symptoms*
- 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*
- Weight loss or gain.
- Abdominal pain.
- Indigestion.
- Dysphagia.
- Odynophagia.
- Nausea.
- Vomiting.
- Change in bowel habit, diarrhoea, constipation.
- PR blood loss.

*Genitourinary symptoms*
- Urinary frequency.
- Polyuria.
- Dysuria.
- Haematuria.
- Nocturia.

*Neurological symptoms*
- Headaches.
- Dizziness.
- Tingling.
- Weakness.
- Tremor.
- Fits, faints, 'funny turns'.
- Black-outs.
- Sphincter disturbance.

*Endocrine symptoms*
- Heat or cold intolerance.
- Neck swelling (thyroid).
- Menstrual disturbance.
- Erectile dysfunction.
- Increased thirst.
- Sweating, flushing.
- Hirsutism.
- Muscle weakness.

*Locomotor symptoms*
- Aches, pains.
- Stiffness.
- Swelling.

*Skin symptoms*
- Lumps/bumps.
- Ulcers.
- Rashes.
- Other lesions (e.g. skin colour or texture change).
- Itch.

# Sexual history

A detailed sexual history does not form part of the standard routine. However, if the patient complains of genitourinary symptoms, a full and thorough sexual history should be obtained.

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.

You should approach a sexual history in a structured way.

## 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 STDs?
- 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 for each partner.

## Previous history

You also need to establish the history of STDs for the patient.

- Have you had any other STDs?
- 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.

# The elderly patient

Obtaining a history from older people might be regarded as no greater a task than from any patient—however cognitive decline, deafness, acute illness, and the middle of a night shift 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 (see Boxes 2.15 and 2.16 for more).

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**Box 2.15  Learning to listen**

It can be tempting to ask lots of questions to obtain every fact in the history, particularly if you are rushed and faced with a clerking pro forma. 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.

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## Key points

- *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.
- *Past history:* as well as the traditional list of illnesses, remember to ask about recent admissions, whether to hospital or community/ intermediate care facilities. Do they see other disciplines in outpatients?
- *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 be 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 realize 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. Learn to consider not just the patient and their acute illness, but a wider context that involves home, family, and potential issues such as carer strain.

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**Box 2.16 A note on narratives**

Akin to 'learning to listen' is the 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. 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.

# General and endocrine examination

**The eye in endocrine disease**

Please turn to ➲ Chapter 9 for details of:
- Examination of the fundus.
- Eye signs in thyroid disease.
- The fundus in endocrine disease including diabetic retinopathy.

# Approaching the physical examination

## General conduct

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.

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.

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

## The format of the examination

### *The 'right' approach*

One important rule is that you should always stand at the patient's right hand side. It is thought this gives them a feeling of control over the situation (most people are right handed), although there is no hard evidence to this effect. 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.

### *The examination framework*

Each system examination is divided into 4 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.

# 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.

### Bedside 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 or the GCS.

# 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

Cyanosis refers to a bluish discoloration of the skin and mucous membranes and is due to the presence of at least 2.5g/dl of deoxygenated haemoglobin in the blood.

*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:* a bluish discoloration 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.

## 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 >35micromol/L).

Jaundice 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 discoloration of the skin, but the sclerae remain white.

## Other abnormalities of coloration

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 haemochromatosis, the silver-grey coloration in argyria (silver poisoning), the ↑ skin-fold pigmentation seen in Addison's disease, and the non-pigmented patches of vitiligo.

# 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
  - Rectal temperature is 0.5°C higher
  - Axillary temperature is 0.5°C lower.
- There is also a diurnal variation in body temperature.
  - Peak temperatures occur between 6pm and 10pm
  - Lowest temperatures occur between 2am and 4am.

### High temperature

- The febrile pattern of most diseases also follows the diurnal variation described. Sequential recording of temperature may show a variety of patterns which can be helpful in diagnosis.
  - Persistent pyrexia may be a sign of malignant hyperthermia, a drug fever (e.g. halothane, suxamethonium), typhus, or typhoid fever
  - An intermittent pyrexia can be suggestive of lymphomas and pyogenic infections such as miliary TB
  - A relapsing high temperature or Pel–Ebstein fever occasionally occurs in patients 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 considered characteristic of biliary sepsis or pyelonephritis, although can occur in the context of any sepsis.

### 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

You may already have obtained clues regarding hydration status 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 (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.
  - If normally hydrated, the skin will promptly return to its original position, whereas in dehydration (reduced skin turgor), 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.
- *Blood pressure:* check lying and standing blood pressure readings and look for a low blood pressure on standing (orthostatic hypotension) which may suggest dehydration.
- *JVP:* Assess the height of the JVP which is one of the most sensitive ways of judging intravascular volume (see ➲ Chapter 5).
  - The JVP is low in dehydration, but raised in fluid overload (e.g. pulmonary oedema).
- *Oedema:* another useful sign of fluid overload (think right heart failure, constrictive pericarditis, hypoalbuminaemia). Remember to test for both ankle and sacral oedema.

# Oedema

Oedema refers to fluid accumulation in the tissues, particularly the subcutaneous layer, 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.

Other causes of leg swelling are outlined in Box 3.1.

### Examination

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

▶ If oedema is present, note how far it extends proximally. What is the highest point at which you can detect oedema? Peripheral 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 fingertips.

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**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, deep vein thrombosis (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.

There 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, head of humerus, 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.

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

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 fat (waist:hip circumference ratio of >1.0 in men and >0.9 in women) is associated with higher 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.

**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).

**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. 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 in ➔ Chapter 7.

### 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 (see Fig. 3.1):* 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.
- *Axillae (see Fig. 3.2):* 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.
- *Inguinal (see Fig. 3.3):* with the patient lying supine, palpate their inguinal region along the inguinal ligament—the same position as feeling for a hernia (➔ Chapter 7) or the femoral pulse (➔ Chapter 5).
  - 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.

### Findings

Similar to the considerations to make when examining a lump (➜ Chapter 4), during palpation of lymph nodes, standard 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. See Box 3.2 for causes of generalized lymphadenopathy.

*Number*

- How many nodes are enlarged?
- Make a diagram 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.

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**Box 3.2 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').
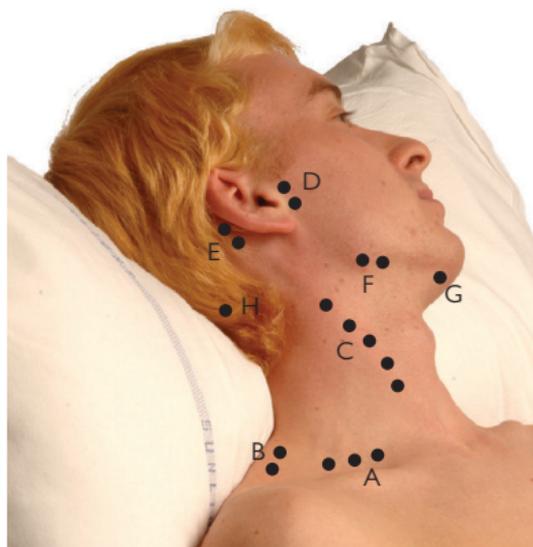
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A = Supraclavicular
B = Posterior triangle
C = Jugular chain
D = Preauricular
E = Postauricular
F = Submandibular
G = Submental
H = Occipital

**Fig. 3.1** Cervical and supraclavicular lymph nodes.
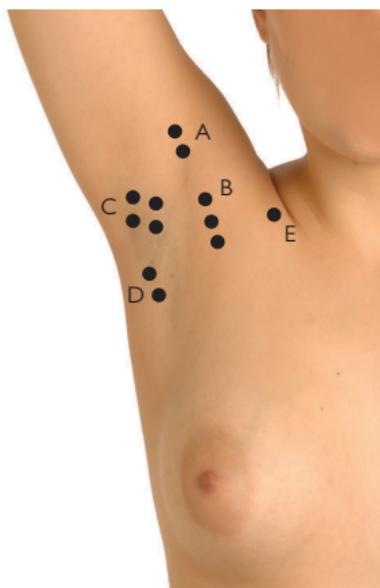


A = Lateral
B = Pectoral
C = Central
D = Subscapular
E = Infraclavicular

**Fig. 3.2** Axillary lymph nodes.
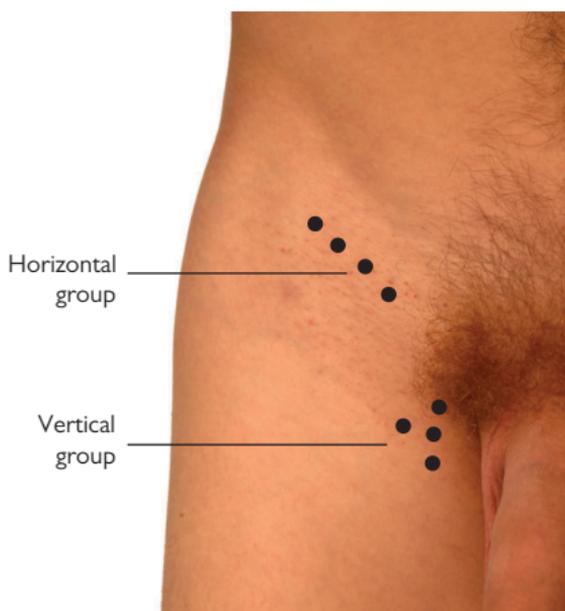
**Fig. 3.3** Inguinal lymph nodes.

# Hands and upper limbs

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 various 'hand signs' throughout this book. Detailed hand examination is described in ➲ Chapters 8 and 10, the nervous system and the locomotor system, so is not repeated here. See also ➲ Chapter 4 for details of skin and nail signs in the hands.

Be sure to include assessment of:
- Both the dorsal surface and the palm.
- Skin colour.
- Discrete lesions.
- Muscles.
- Joints.
- Bony deformities.
- Nails.
- Remember to palpate and test movement and sensation.

After examining the hands, palpate both the radial and ulnar pulses.

## 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.

# Recognizable syndromes

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

Certain physical features of conditions 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.
- *Limbs:* short fourth metacarpal or metatarsal, hyperplastic nails, lymphoedema, increased 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, kyphoscoliosis, aortic incompetence.
- *Other:* cystic disease of the lungs (spontaneous pneumothorax, bullae, apical fibrosis, aspergilloma and bronchiectasis), inguinal or femoral herniae.

## 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, periungual 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 Recklinghausen's disease—autosomal dominant.
- *Skin:* neurofibromata (single, lobulated or pedunculated, soft, firm, mobile, lumps or nodules along the course of nerves), café-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–Jeghers syndrome

- *Skin:* sparse or profuse small brownish-black pigmented macules on lips, around mouth and on buccal mucosa, hands, and fingers.

## Oculocutaneous albinism

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

## 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, 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.

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* More detail in ➜ Chapter 8.

# 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. 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), and Wernicke–Korsakoff syndrome.

### Vitamin $B_2$ (riboflavin)
- Found in wholemeal flour, meat, fish, and dairy produce. 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: dermatitis, diarrhoea, and 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$_9$ (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 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, depression, psychosis, and optic atrophy.

*Vitamin C (ascorbic acid)*
- Deficiency causes scurvy (perifollicular 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, neutropenia, impaired bone mineralization, Menkes' kinky hair syndrome (growth failure, mental deficiency, bone lesions, brittle hair, anaemia), sensory ataxia, muscle weakness, visual loss (optic neuropathy), peripheral neuropathy.
  - 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 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 such things as:
  - Skin lesions (malignant?)
  - New/isolated patches of 'psoriasis' (Bowen's disease?)
  - Asymptomatic peripheral arterial disease.

*Talk to your patient*
- During the examination as well as during the history.
- 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.
- Recognize the limits of early warning scores for older people, especially with chronic diseases as you may be falsely reassured.

*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?

*MMSE/AMTS*
- Should be mandatory for the majority of patients.

*Gait (where possible)*
- Akin to mental state examination, should be undertaken whenever possible. See ➲ Chapter 10 for the 'get up and go' test.

## Geriatric giants

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

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

## Information gathering

Faced with an acutely unwell, delirious patient, no old notes or GP letter and 'little to go on', it can be tempting to fall back on Isaacs' giants as a diagnosis. Make that extra effort to enquire of others for information which will reveal vital clues:
- Family and carers (e.g. care home staff): a real opportunity to update family, reassure or open up a conversation about other issues.
- IT: virtually all hospitals allow access to e-systems that record clinic letters, discharge notes, and results.
- GP surgeries: both for the Summary Care Record/current prescription and a discussion with one of the GPs, who is likely to know the patient better than you.
- Community services: you will often find that the rest of the multi-disciplinary team are ahead of you in liaising and gathering important information from homecare, district nurses, and intermediate care.

# 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 weigh themselves but may have noticed the consequences of weight change such as 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 endocrine disease such as:
- Diabetes mellitus.
- Cushing's syndrome.
- Hypoadrenalism.
- Hypothyroidism.
- Hypercalcaemia.

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

### Bowel habit

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

### Urinary frequency and polyuria

Endocrine causes might include:
- Diabetes mellitus.
- Diabetes insipidus.
- Hyperglycaemia caused by Cushing's syndrome.
- 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 hypo- or hyperthyroidism, Addison's disease, and Hashimoto's thyroiditis.
- ↑ *pigmentation:* Addison's disease, Cushing's syndrome.
- ↓ *pigmentation:* generalized loss of pigmentation in hypopituitarism.

### Hair distribution

See also 'Skin, hair, and nails', ⯈ Chapter 4.

*Hair loss*

Decreased adrenal androgen production and loss of axillary and pubic hair in both sexes can be caused by:
- Hypogonadism.
- Adrenal insufficiency.

*Hair gain*

Hirsutism or excessive hair growth in a female may be due to endocrine dysfunction. Consider:
- Polycystic ovarian syndrome.
- Cushing's syndrome.
- Congenital adrenal hyperplasia.
- Acromegaly.
- Virilizing tumours.

### 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 acropachy is seen only in hyperthyroidism due to Graves' disease. Features include 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.

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 over-growth. Patients may notice an increase in shoe size, glove size, or facial appearance (do they have any old photographs for comparison?).

### Changes in sexual function

*Women*

Altered menstrual pattern in a female may be an early symptom suggestive of pituitary dysfunction. See ⟳ Chapter 13 for more detail.

*Men*

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.

### 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.

# The rest of the history

A full history should be taken (see Box 3.3 for the history in patients with diabetes). 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 3.3).
- 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 3.4).

---

**Box 3.3 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 diabetic ketoacidosis (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?

If the patient is newly diagnosed with diabetes, ask about a history of weight loss (may differentiate type I and type II diabetes).

---

**Box 3.4 The MEN syndromes**

'Multiple endocrine neoplasias' which display autosomal dominant inheritance.

*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 endocrine 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.

See Box 3.5 for signs of tetany.

You may, however, perform a quick 'screening' general examination of a patient's endocrine status.

### Hands/arms

Look at size, subcutaneous tissue, length of the metacarpals, nails, palmar erythema, sweating, and 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 (⊙ Chapter 8).

### Axillae

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

### Face and mouth

Look for hirsutism, 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 ⊙ Chapter 9.

### Neck

Note any swellings or lymphadenopathy. 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, hirsutism. 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 and make note of any diabetes-related changes.

### Height and weight

Calculate the patient's BMI.

---

**Box 3.5 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 increased blood flow which is characteristic of the hyperthyroid goitre seen in Graves' 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.

---

### Skills station 3.1

*Instruction*

Clinically assess this patient's thyroid status.

*Model technique*

- Clean your hands.
- Introduce yourself.
- Explain the purpose of examination, obtain informed consent.
- Ask for any painful areas you should avoid.
- 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 acropathy (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 (➲ Chapter 9)
  - Hypothyroid features.
- Examine the eyes (➲ Chapter 9).
- Examine the thyroid and neck.
- Test tendon reflexes at the biceps and ankle (➲ Chapter 8).
- Test for proximal myopathy by asking the patient to stand from a sitting position.
- Look for pretibial myxoedema.
- Thank the patient and help them re-dress as necessary.

# 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 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.

## Framework for a thorough diabetic examination

*General 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, brownish-red margins, surrounding telangiectasia.
- Also look for granuloma annulare.

*Hyperlipidaemia*
- Eruptive xanthoma.
- Tendon xanthoma.
- Xanthelasma.

*Neurological examination*
- Visual acuity (⊃ Chapter 9).
- Fundoscopy (⊃ Chapter 9).
- Peripheral sensory neuropathy—evidence of injury, ulceration, and Charcot's joint formation.
- Test muscle strength (⊃ Chapter 8).
- 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.

*Using a 10g monofilament*
A small, thin plastic filament, designed such that it bends under approximately 10g of pressure.

- Apply the filament to the patient's skin at the spots shown in Fig. 3.4a.
- Press firmly so that the filament bends (Fig. 3.4b).
- 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.
- 🛑 Do not press on ulcers, callouses, scars, or necrotic tissue.
  - The patient's feet are 'at risk' if they cannot feel the monofilament at any of the sites.
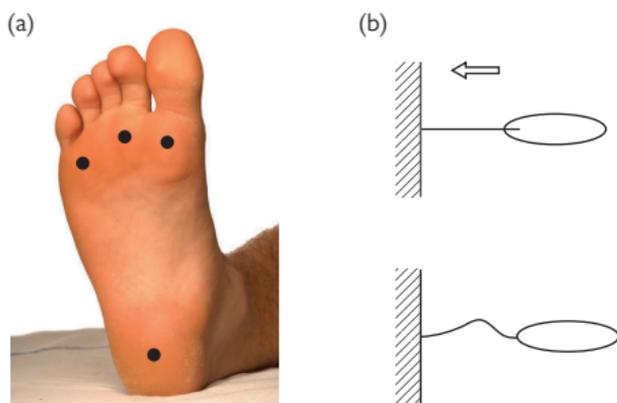


**Fig. 3.4** (a) Sites to test with a 10g monofilament in the diabetic patient. (b) Apply the monofilament to the skin with enough force to make it bend.

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**Skills station 3.2**

*Instruction*
Clinically assess the foot of this patient with diabetes.

*Model technique*
- Clean your hands.
- Introduce yourself.
- Explain the purpose of examination, obtain informed consent.
- Inspect, noting colour, ulceration, dryness, callous formation, evidence of infection.
- Evidence of injury—shoes rubbing?
- Are there any Charcot's joints?
  - Grossly abnormal and dysfunctional joints due to repeated minor trauma and poor healing due to a loss of pain sensation.
- 10g monofilament test.
- Test light-touch sensation, pain sensation, vibration sense, and proprioception.
- Palpate the peripheral pulses (dorsalis pedis and posterior tibial).
- Note the temperature of the skin on the dorsum and sole.
- Record capillary filling time.
- Thank the patient and help them re-dress as necessary.

# Important presentations

### Hypothyroidism

*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 or breast cancer).
- Pituitary adenoma.

*Symptoms*
- Tiredness.
- Weight gain.
- Anorexia.
- Cold intolerance.
- Poor memory.
- Depression.
- Reduced libido.
- Goitre.
- Puffy eyes.
- Brittle hair.
- Dry skin.
- Arthralgia.
- Myalgia.
- Muscle weakness.
- Constipation.
- Menorrhagia.

*Signs*
- *General:* croaking voice, mental and physical sluggishness, pseudodementia, 'myxoedema madness'.
- *Inspection:* coarse cool dry skin (look for yellowish tint of carotenaemia '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, xanthelasma.
- *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

*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.
- Increased appetite.
- Irritability.
- Restlessness.
- Muscle weakness.
- Tremor.
- Breathlessness.
- Palpitations.
- Sweating.
- Heat intolerance.
- Itching.
- Thirst.
- Vomiting.
- Diarrhoea.
- Eye complaints (Graves' ophthalmopathy).
- Oligomenorrhoea.
- Loss of libido.
- Gynaecomastia.

*Signs:*

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

## Glucocorticoid excess (Cushing's syndrome)

- *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, decreased 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 disease)

- *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)

- *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, dysmenorrhoea, 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: loss of libido, erectile dysfunction, infertility, galactorrhoea. If before puberty, 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 asymptomatic. 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 causes 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).

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

# Skin, hair, and nails

# Introduction

The skin is a highly specialized organ with various physiological roles including protection from trauma, infection, and ultraviolet (UV) radiation; regulation of body temperature and fluid balance; detection of sensory stimuli; and synthesis of vitamin D.

The skin also plays a key function in social interaction so what might seem to be a trivial disease in terms of physical health, such as acne, can have devastating psychosocial consequences.

## Anatomy and physiology

The skin comprises two layers: the epidermis and dermis. The fat layer below the skin is known as the subcutis.

### Epidermis

This is a keratinizing, stratified squamous epithelium. The keratinocyte is the principal cell. The basal layer of the epidermis is made up of cuboidal basal cells, which continually divide, generating new cells which migrate upwards towards the skin surface, from which they are eventually shed. This process takes 30–50 days, varying with body site. The epidermis forms a protective layer of keratin on the surface.

Other cell types in the epidermis include melanocytes, Langerhans cells, and Merkel cells.

- *Melanocytes:* reside in the basal layer and secrete melanin pigment into surrounding keratinocytes via long projections. Melanin helps to protect the skin from UV radiation. The number of melanin granules determines skin colour.
- *Langerhans cells:* important in the immune response, acting as antigen-presenting cells.
- *Merkel cells:* thought to be involved in touch sensation.

### Dermis

This is a layer of connective tissue consisting of collagen and elastic fibres, which give rise to much of the tensile strength and elasticity of the skin. It is here where the skin appendages (hair follicles, sebaceous glands, eccrine and apocrine glands), nerves, and blood and lymphatic vessels lie.

- *The follicle:* a specialized tubular structure which opens on to the skin surface and produces hair. Follicles are present over virtually the entire body; some sites, such as the scalp, contain very dense numbers of large follicles. The arrector pili muscle connects the follicle to the dermis; its contraction pulls the hair more perpendicular to the skin.
- *Sebaceous glands:* attached to hair follicles. These secrete lipid-rich sebum, which waterproofs and lubricates the skin and hair.
- *Eccrine glands:* responsible for the production of sweat.
- *Apocrine glands:* structurally similar to eccrine glands. Their role in humans is not clear, but they are important in scent production in some mammals.

*Subcutis*

The subcutis acts as a lipid store and helps with insulation. It also contributes to body contour and shape.

## Hair

Hair is comprised of keratin, a different form from that on the skin surface. Scalp hair is an example of *terminal* hair, which is coarse and pigmented, in contrast to fine vellus hair, found at sites such as the female face.

*Hair growth*

Hair growth is cyclical, with an active anagen phase, followed by the involutional catagen phase and then a resting telogen phase, at the end of which the hair is shed. At any given time, most hairs are in anagen phase, which typically lasts for upwards of 3 years. Although hair abnormalities may not seem particularly consequential to health, hair is of great importance in social interaction and sensitivity is required when dealing with patients with either too little or too much of it.

## Nails

The nail plate is a sheet of keratin. Its main functions are protection of the fingertip and to improve dexterity. It is produced by the nail matrix, which lies mainly beneath the proximal nail fold, but is just visible in some nails as the pale lunula (or 'half-moon'). Nails grow continuously at an average rate of around 3mm per month though fingernails grow faster than toenails. Nail changes can provide clues to diagnosis of both dermatological and systemic disease.

# The dermatological history

It is estimated that there are over 2000 different dermatological conditions. These can present with all manner of skin, hair, and nail changes and it is therefore difficult to be prescriptive when describing how to take a good dermatological history. It is hoped that the following will at least serve as a guide to elucidating the important elements, which include the **time course of the complaint**, its **evolution**, the **main symptoms**, **exacerbating or relieving factors**, and **associated features** or disease.

If an eruption is present, it is not necessary to elicit a detailed description of it from the patient (and rashes are notoriously difficult to describe anyway). However, if an eruption comes and goes, the history in some cases may still be diagnostic and should at the least provide important clues.

## History of the presenting complaint

- When did the problem start?
- Where did it start?
  - Where is affected now?
- How have things changed since?
  - Is it a continuous or intermittent problem?
- Is it evolving (if so, how?) or stable?
- Is there any discharge or bleeding?
- Is there pain, itch, or altered sensation?
- Is there dryness or itching?
- Are there any obvious factors which trigger or exacerbate the problem? Possibilities include:
  - Sunlight
  - Extremes of temperature (itching is often worsened by heat)
  - Contact with certain substances (e.g. latex, rubber, metals, hair dye)
  - Work (e.g. occupational allergy or wet work leading to irritant contact dermatitis).
- Does anything relieve the symptoms?
  - e.g. sunlight, topical treatments, systemic drugs.
- What treatments have been tried?
  - What was effective or ineffective?
- Are there any systemic symptoms such as fever, malaise, joint pain, weight loss, or sore throat?

## Past medical history

- Previous skin problems?
- Ask also about diabetes, connective tissue disease, inflammatory bowel disease, atopy (eczema, especially as a baby, asthma, hayfever)?

## Allergies

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

## Drug history

- Which drugs is the patient taking and for how long? See Box 4.1.
- If a drug reaction is possible, ask about recent courses of drugs not taken regularly (e.g. antibiotics, over-the-counter analgesics).
  - 🕛 Bear in mind that there may be a delay of a few days to months before a drug eruption occurs
  - Immunosuppression can increase the risk of skin cancer.

## Family history

- Ask especially about atopic diseases, psoriasis, skin cancer.

## Social history

- Occupation (consider wet work, sun exposure, exposure to chemicals or plants).
- Hobbies.
- Pets (including pets of close friends and relatives).
- Living conditions—how many share the house/living space?
- Recent travel? Were appropriate vaccinations taken before leaving?
- Insect bites?
- Risk factors for sexually transmitted diseases? Take a full sexual history if relevant. 🕛 Be delicate.

## Psychosocial impact

- Ask about how the condition is affecting the patient.
- Physical symptoms such as pain or worsening in sunlight might curtail usual activities.
- Self-consciousness and embarrassment in a physically asymptomatic condition can still lead to enormous social handicap.

---

**Box 4.1 Common culprits for drug eruption**

Rashes can be caused by virtually any drug and mostly occur 1–2 weeks after the drug is started. The following are frequently responsible and should be viewed with suspicion in a patient with a rash compatible with drug eruption:

- Anticonvulsants
- Sulphonamides (e.g. trimethoprim, co-trimoxazole)
- Penicillins
- Allopurinol
- Non-steroidal anti-inflammatory drugs (NSAIDs).

# Hair and nail symptoms

### Hair loss

Alopecia is a clinical sign meaning hair loss: it is not a diagnosis. See Box 4.2 for more. In the history consider:

- Sudden or gradual?
  - Bear in mind that it is normal to lose up to 150 hairs per day from the scalp.
- Areas affected (scalp and/or body hair?).
- Diffuse or localized?
- Other scalp symptoms e.g. scaling, itch, soreness, crusting.
- Rashes elsewhere (e.g. lichen planus, cutaneous lupus).
- Other medical problems (e.g. systemic lupus, severe trauma, psychological stress or febrile illness in last few months).
- Family history of hair loss.

### Excessive 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.
- DHx.
- For women, ask about the menstrual cycle (when was the last period? Are they regular or erratic?) and symptoms of virilization (e.g. voice change, clitoromegaly, new-onset acne).

### Nail symptoms

The history for nail changes should be treated as other dermatological conditions.

However, seek evidence of skin or systemic conditions which can involve the nails (e.g. psoriasis, eczema, fungal infections). See also Box 4.3.

---

**Box 4.2 Important disorders of hair loss**

- *Male-pattern baldness:* commonly occurs from the 3rd decade. Hair is lost first from the frontotemporal region, then the crown
- *Female-pattern hair loss:* tends to occur post-menopause, but sometimes in much younger women; leads to thinning over the crown with preservation of the frontal hairline
- *Alopecia areata:* associated with organ-specific autoimmune disorders and tends to begin in the 2nd or 3rd decade. Sharply demarcated, non-inflammatory bald patches on the scalp. There may be pathognomonic 'exclamation mark' hairs which are a few millimetres long and thinner at the base. Eyebrows, beard, and body hair can also be affected. Nails may be slow-growing and pitted. Extreme examples include:
  - Alopecia totalis: loss of all scalp hair
  - Alopecia universalis: loss of all hair including on the body.
- *Telogen effluvium:* severe illness, high fever, or childbirth may synchronize all the hair follicles causing all hairs to enter the telogen phase at the same time, 3–6 months later. This leads to dramatic shedding and near-total hair loss, which then resolves
- *Scarring alopecia:* inflammatory lesions causing hair loss include lichen planus, burns, and infection. Scarring alopecia causes destruction of the follicles and is therefore permanent.

---

**Box 4.3 Important nail disorders/signs**

See also Chapter 3.

- *Splinter haemorrhages:* tiny, longitudinal streak haemorrhages under the nails caused by micro-emboli or trauma. Distal lesions can be a normal finding especially in manual workers
- *Pitting:* tiny indentations in the surface of the nail. A feature of psoriasis, lichen planus, and alopecia areata
- *Onycholysis:* separation of the nail plate from the nail bed; seen in psoriasis. Also caused by trauma, thyrotoxicosis, and certain drugs
- *Leukonychia:* white nails, seen in hypoalbuminaemia
- *Beau's lines:* transverse depressions in the nail. These represent arrested nail growth during a period of acute severe illness
- *Paronychia:* infection or inflammation of the nail folds, causing pain, redness, and swelling
- *Koilonychia:* spooning (concave indentation) of the nail, associated with severe iron deficiency
- *Clubbing:* see ➲ Chapter 3
- *Onychomycosis:* fungal nail infection causing the nail to become thickened, opaque, crumbly, and yellow. This may be indistinguishable from psoriatic nail changes
- *Longitudinal melanonychia:* a pigmented streak in the nail which may represent subungual melanoma, especially if Hutchinson's sign (pigmentation extending on to the proximal nail fold) is positive.

# 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, to 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.

## 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 focal lesion

▶ See also 'examining a lump' if the lesion is raised.

Inspect each lesion carefully and note:
- Grouped or solitary? If grouped, is there a pattern?
- Site.
- Distribution/location (symmetrical/asymmetrical? Peripheral? In only light-exposed areas? Dermatomal?).
- Colour.
- Shape.
- Size (diameter).
- Surface.
- Border.
- 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.)

If the lesion is pigmented, some special considerations apply (see Box 4.4).

## 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—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 (➔ Chapter 3).

---

### Box 4.4 Describing pigmented lesions

When faced with a pigmented lesion, the key is to decide whether there is a possibility of melanoma, a potentially fatal cancer. See ➔ *OHCM9*. If so, the patient should be referred to a dermatologist or plastic surgeon for consideration of excision biopsy. Melanoma can arise in an existing naevus or *de novo*.

A useful system which can serve as a guide to whether a melanocytic lesion is clinically 'suspicious', as well as providing a framework for a description, is the ABCDE method. If one or more features are 'positive', melanoma is more likely. However, a history of change over time is also important. If in doubt, refer to a skin specialist.

*ABCDE*
- *A:* **a**symmetry
- *B:* irregular **b**order
- *C:* irregular **c**olour
- *D:* **d**iameter >6mm
- *E:* (new) **e**levation.

Bear in mind that many pigmented lesions are not melanocytic. For example, seborrhoeic keratosis usually demonstrates a warty, fissured surface, and is well defined to the point of looking 'stuck on'. If you reach the end of ABCDE and feel that you haven't mentioned all the salient points, think of E also as 'Everything Else'.

# Pictorial glossary of terms

A careful description often clinches the diagnosis in dermatology. All lesions should be documented in accepted dermatological terms (Figs 4.1–4.3).

**Flat, non-palpable changes in skin colour**
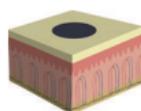
**Macule**
Small flat, non-palpable change in skin colour ≤ 0.5–1cm diameter 'Freckles' are pigmented macules
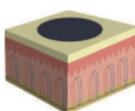
**Patch**
Large, flat, non-palpable change in skin colour

**Elevation due to fluid in a cavity**
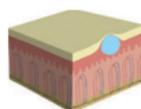
**Vesicle**
Small blister (0.5–1cm) that contains clear fluid
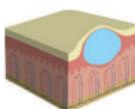
**Bulla**
Large blister that contains clear fluid
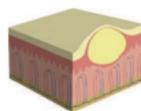
**Pustule**
Visible collection of pus
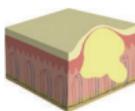
**Abscess**
Localized collection of pus in cavity >1cm diameter

**Elevation due to solid masses**
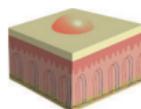
**Papule/papular**
Small, solid, raised lesion ≥ 0.5–1cm in diameter, usually dome-shaped
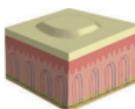
**Plaque**
Larger superficial flat-topped raised area
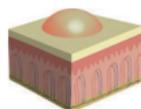
**Nodule**
A dome-shaped solid lump, >0.5–1cm in diameter, that may project or be deep in the skin
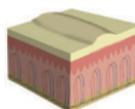
**Wheal (weal)**
Pale area of dermal oedema, usually <2cm diameter, often surrounded by an erythematous flare

**Loss of skin**
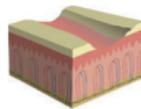
**Erosion**
Partial epidermal loss Heals without scarring
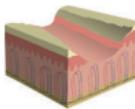
**Ulcer**
Complete loss of epidermis and some dermis, may scar when heals
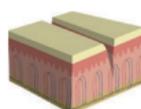
**Fissure**
A linear crack
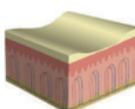
**Atrophy**
Thinning of the epidermis and/or dermis

**Fig. 4.1** Primary lesions. Images by Dr Ravi Kothari.

(a)
**Surface changes**
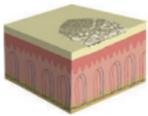


**Scale**
White flaking of superficial horny layer (indicates epidermal pathology)
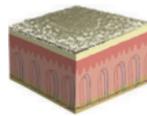
**Crust**
Dried blood or tissue fluid

**Callus**
Hyperplastic epidermis, found in areas of excessive friction/use
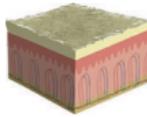
**Lichenification**
Thickening of the epidermis with exaggerated skin markings (bark-like) usually due to repeated scratching or rubbing

(b)
**Vascular changes**
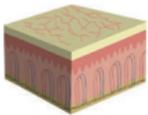
**Telangiectasia**
Easily visible superficial blood vessels (blanches)

**Spider naevus**
A single telangiectatic arteriole in the skin
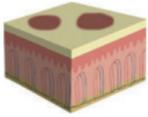
**Purpura**
(non-blanching): extravasation of blood into skin (usually around 2mm in diameter)
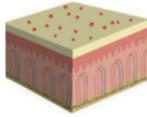
**Petechiae**
Pin-head sized areas of purpura
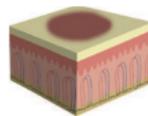
**Ecchymosis**
A 'bruise'. Purpura >2mm diameter
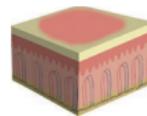
**Erythema**
Blanching reddening of the skin due to local vasodilatation

**Fig. 4.2** (a) Secondary lesions. (b) Vascular lesions. Images by Dr Ravi Kothari.

(a)



| Linear | Arciform | Annular |
| Serpiginous | Target | Gyrate |
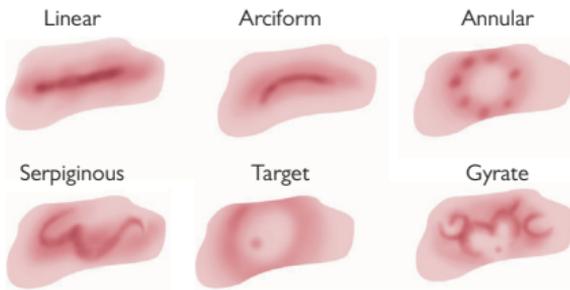
(b)
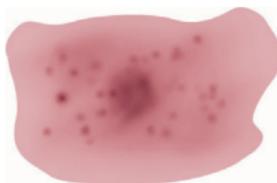
**Fig. 4.3** (a) Descriptive terms for lesion shapes and patterns of grouped lesions. (b) Confluence of grouped lesions. Note how the smaller lesions coalesce to form a larger lesion. Images by Dr Ravi Kothari.

# 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.

## 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 (see ➲ Chapter 7).
- *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 (see ➲ Chapter 6) 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 ➲ Chapter 7).
- *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'.
- *Reducibility:* a feature of herniae. 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 large lump, you could gain important clues regarding its origin and contents. Listen especially for:

- Vascular bruits.
- Bowel sounds.

# Widespread skin eruptions

### The 'DCM' method

'DCM' stands for distribution, configuration, and morphology, which are best presented in this order.

*Distribution*

- Where does the rash affect?
- Is there a pattern to it (e.g. predominantly extensor or flexor surfaces, photo-exposed distribution)?
- Is it broadly symmetrical?

*Configuration*

- If there are multiple lesions which comprise the rash, is there a pattern in the way these are aligned?
  - Are the lesions arranged in a line (linear), a ring (annular), or another recognizable shape?
  - Are there clusters of lesions, with spared skin in between?
- Bear in mind that most rashes will not display a specific configuration, but if you see one, it can be an important clue to the diagnosis.

*Morphology*

- Describe the actual features of the rash. For example, is it macular or are there papules or plaques?
- What colour is it?
- Are there discrete components and do they coalesce, or is there just a confluent area affected?

Finish by commenting on other features if relevant, such as abnormalities in the nails, hair, or mucous membranes.

# Examining an ulcer

The approach to examining an ulcer is similar to any other skin lesion. Consider the site and size, as well as whether there are single or multiple lesions. If the shape of the ulcer, or position, is unusual or difficult to describe, make a drawing! See Box 4.5 for notes on leg ulcers.

## Border

Assess the morphology of the border. See also Fig. 4.4. Some examples include:

- *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.

Most venous ulcers have a sloping border; arterial ulcers classically look 'punched out'; pyoderma gangrenosum (PG) and some pressure sores manifest an undermined border, meaning that the process extends beneath the edges of the actual ulcer. The border in PG also has a characteristic violaceous hue. If there is a rolled or heaped up edge, consider the possibility of a neoplastic ulcer: most types of skin cancer and some benign neoplasms can present with ulceration.

## Depth

- Ulcers are loosely divided into superficial or deep; visible bone or tendon at the base certainly implies a deep ulcer, but use your judgement.

## Base

- Healing ulcers have granulation tissue at the base; this appears moist, beefy red, and usually forms a cobble-stoned surface.
- Some ulcers will have surface slough, yellow or brown material which is sometimes mistaken for pus.

## Surrounding skin

- Look for signs of chronic venous disease (e.g. peripheral oedema, varicose veins, haemosiderin deposition, lipodermatosclerosis, atrophie blanche) and arterial insufficiency (loss of hair, shiny, erythematous skin, cool peripheries).
  - Check peripheral pulses and capillary refill time if arterial disease is suspected.
- Assess the quality of surrounding skin: there may be incipient ulceration elsewhere or other damage to the skin such as blistering.

- Check that there is no cellulitis, but bear in mind that eczema (gravitational or contact) is very common around chronic leg ulcers.
  - If there is scaling and itch, this is a far more likely diagnosis than infection, and treatment should reflect this.
- If arterial or venous disease are possibilities, the ankle brachial pressure indices should be checked to confirm or refute an arterial component and to establish whether compression can be used safely.
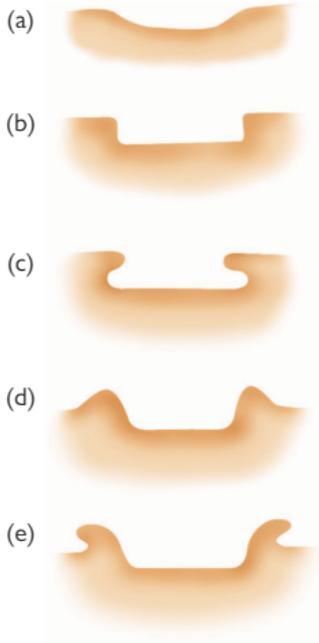


**Fig. 4.4** Representation of some ulcer borders. (a) Sloping, (b) punched out, (c) undermined, (d) rolled, (e) everted.

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**Box 4.5 A word on leg ulcers**

Leg ulcers are often a result of mixed venous and arterial disease; however, one pathology may predominate.

*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 discoloration (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, 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 ageing 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 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 ichthyosis 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 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.
- *MRSA:* Has the patient received decolonization treatment as appropriate? Could the new rash reflect an allergy to administered topical treatment?
- *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? Xerosis 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:* avoid 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. Look out for pigmentation change and ensure emollients are prescribed. For patients who may receive compression bandaging/hosiery—check peripheral pulses/ankle brachial pressure index (ABPI) carefully. Carefully describe any ulceration present.
- *ECG stickers:* if you perform an ECG—remove the stickers 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.

# Presenting patterns

## Skills station 4.1

*Instruction*

Describe your clinical findings and give the most likely diagnosis (Fig. 4.5).
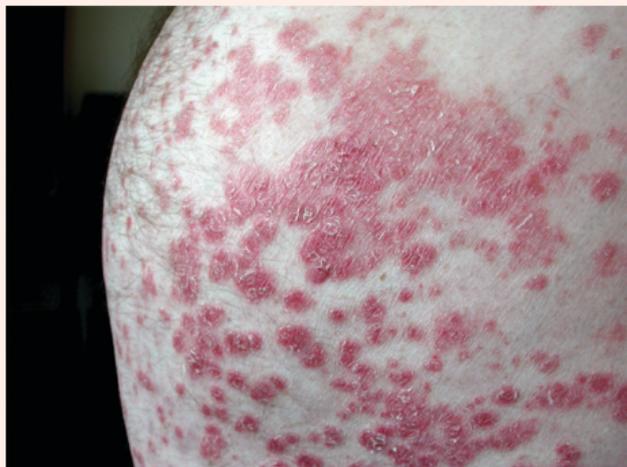


**Fig. 4.5** Skin skill 1.

*Model answer*

This is a generalized, broadly symmetrical eruption mainly affecting the trunk with fewer lesions on the limbs and sparing of the face.

There is no specific configuration.

Morphologically, there are erythematous plaques with overlying silvery scale. These are small (5–10mm diameter), but are coalescing into larger plaques at some sites.

The diagnosis is guttate psoriasis.

**Skills station 4.2**

*Instruction*

Describe your clinical findings and give the most likely diagnosis (Fig. 4.6).



**Fig. 4.6** Skin skill 2.

*Model answer*

This is a widespread, symmetrical eruption, which affects the trunk, arms, and face.

The lesions are markedly annular and polycyclic [having varying curves].

They comprise erythematous wheals of various sizes.

The diagnosis is urticaria.

**Skills station 4.3**

*Instruction*

Describe your clinical findings and give the most likely diagnosis (Fig. 4.7).



**Fig. 4.7** Skin skill 3.

*Model answer*

This symmetrical eruption is confined to the dorsal aspect of the hands. The changes are diffuse and ill-defined so there is no specific configuration. There is xerosis, with some mild erythema, fine scaling, and some tiny fissures.

These changes are eczematous and the most likely diagnosis is irritant contact dermatitis.

### Skills station 4.4

*Instruction*

Describe your clinical findings and give the most likely diagnosis (Fig. 4.8).



**Fig. 4.8** Skin skill 4.

*Model answer*

This is a solitary, pigmented lesion on the upper back. It is asymmetrical with an irregular but clearly demarcated border; it has various colours including grey, dark brown, blue-black, and black; the size is 18x11mm and the lesion is elevated.

Other features comprise a moist, eroded surface and some surrounding erythema.

The lesion looks highly suspicious for malignant melanoma.

**Skills station 4.5**

*Instruction*

Describe your clinical findings and give the most likely diagnosis (Fig. 4.9).



**Fig. 4.9** Skin skill 5.

*Model answer*

This is a solitary, pigmented lesion.

It is broadly symmetrical, with a somewhat irregular but clearly defined border. There is a mid-brown, macular base studded with darker brown papules, the largest of which is central. It measures 15x8mm in diameter.

This is probably a benign, congenital naevus spilus (speckled naevus).

**Skills station 4.6**

*Instruction*

Describe your clinical findings and give the most likely diagnosis (Fig. 4.10).

*Model answer*



**Fig 4.10** Skin skill 6.

This is a solitary 15mm lesion which straddles the left medial lower eyelid margin. It is a nodule with a rolled, pearly edge and some overlying telangiectasia. There is a small crusted area centrally.

The diagnosis is basal cell carcinoma.

**Skills station 4.7**

*Instruction*

Describe your clinical findings and give the most likely diagnosis (Fig. 4.11).

*Model answer*
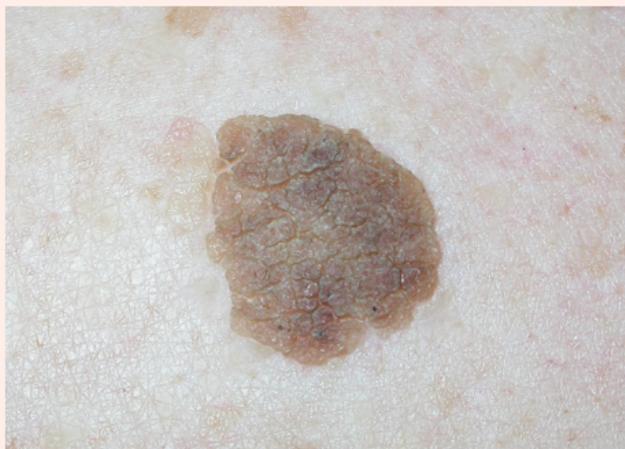


**Fig. 4.11** Skin skill 7.

This is a pigmented lesion. It is symmetrical with a slightly irregular border, which is sharply defined to the extent that the lesion has a 'stuck on' appearance.

The colour is uniform mid-brown throughout the main part of the lesion, though there is also a skin-coloured component abutting this on the left.

The diameter is 12mm and the lesion is morphologically a plaque (i.e. elevated). The surface is fissured and warty.

This is a seborrhoeic keratosis.

# The cardiovascular system

# Introduction

The cardiovascular 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. This 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'.

### Jugular venous pulse

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.

### 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 baseline, 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 usually possible to determine the probable cause of the pain from the history.

As for any other type of pain, the history must include the standard 'SOCRATES' questions (see ➲ Chapter 2):

- 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. See Box 5.1 for the classic features.

'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. Some patients may experience angina pain *only* in the arm or jaw, for example.

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

In patients with known angina, a *change* in the nature of the symptoms is important.

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### Box 5.1 Classic features of angina

- 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.

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### Myocardial infarction (MI)

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 shoulder blades.
- Persistent, most severe at onset.
- Patient is usually hypertensive and 'marfanoid'.

### Pleuritic (respiratory) pain

This is covered in more detail in ➲ Chapter 6. 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 6. 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; this gives 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 5.2. 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*

This is episodes of breathlessness occurring at night—usually thought to be due to pulmonary oedema. 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.

Ask:

- 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?

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**Box 5.2  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).

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# 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 reduced cardiac output and decreased 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 decrease 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?

# Palpitations

To have palpitations is to have an awareness of one's own heart beating. 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 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 heartbeat 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'?

# 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.

# 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. They should not then be repeated again later in the clerking.

- *Age:* increased risk with age.
- *Gender:* risk in males > females.
- *Obesity:* how heavy is the patient? (Calculate their BMI.)
- *Smoking:* quantify in pack-years. Don't be caught out by the 'ex-smoker' that gave up yesterday!
- *Hypertension:* find out when was it 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.

# Outline cardiovascular examination

The full examination framework is shown in Box 5.3. 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.

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**Box 5.3  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.

# General inspection and hands

### 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?
- 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 discoloration 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.
  - 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 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
  - A rare feature of bacterial endocarditis.

# Peripheral pulses

For each peripheral pulse, 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 illustrated in Fig. 5.1.

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, medial to the radial styloid process at the wrist.

### Brachial artery

- Feel at the medial side of the antecubital fossa, just medial to the tendinous 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.
  - ❶ 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 undressed 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. 5.1** 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 and calculate the rate by multiplication. Commonly, people count for 15 seconds and multiply 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 decrease with inspiration and increase 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 practice to master and it may be useful to imagine a graph such as those shown in Fig. 5.2. Some examples are:

- *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 coexist.
- *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).

| | | |
|---|---|---|
| | Normal | |
| | Slow rising | e.g. aortic stenosis |
| | Bisferiens | e.g. aortic stenosis mixed with aortic regurgitation |
| | Collapsing | e.g. aortic regurgitation |
| | None | e.g. occluded artery, death |
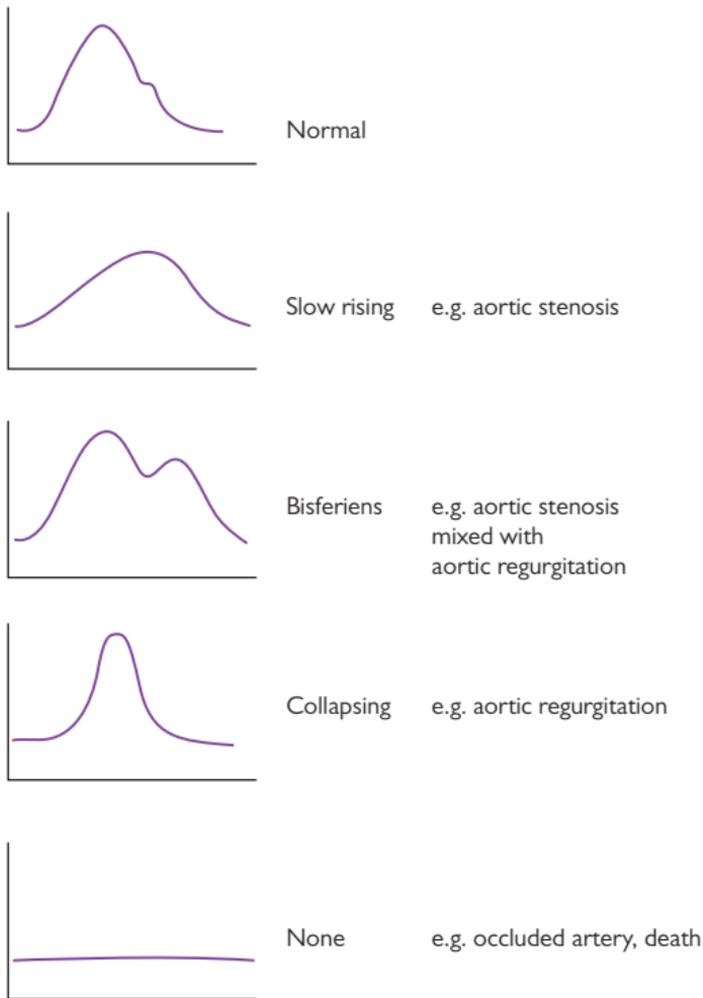
**Fig. 5.2** Graphical representation of arterial pulse waveforms and their causes.

### 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 discoloration of the sclera.
- *Anaemia:* seen as an unusually pale conjunctiva (practice 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 discoloration 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 in the routine, the carotid pulse should be examined.
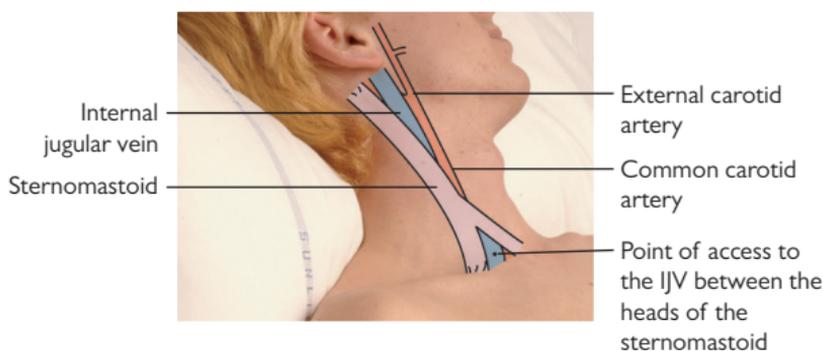


**Fig. 5.3** The surface anatomy of the vasculature in the neck. Note that the IJV is partly hidden by the sternocleidomastoid at the base of the neck.

## 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.

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. 5.3). 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.

*Examination*

- 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 (see Fig. 5.4).
  - 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.
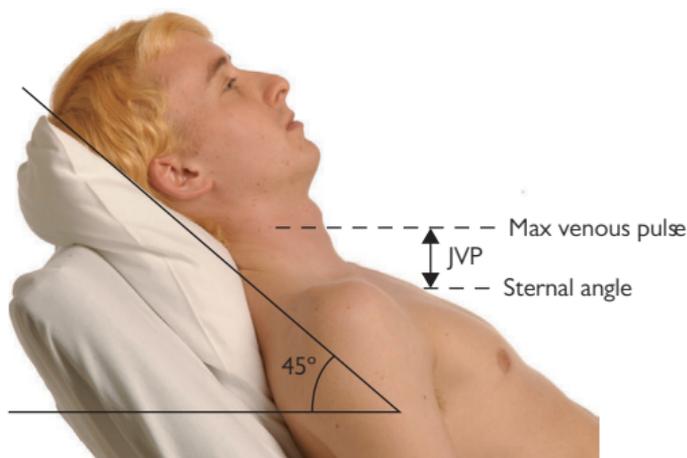


**Fig. 5.4** Measuring the JVP. Measure the *vertical* distance.

*Differentiating the jugular and carotid pulsations*

The rules for differentiating the jugular and carotid pulsations 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. See Table 5.1.

Table 5.1 Jugular and carotid pulsation

| 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 2cm, the carotid pulse will not.

*Character of the jugular venous pulsation*

This is rather difficult without experience (Box 5.4). The jugular pulsation has 2 main peaks (see Fig. 5.5). 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 descent:* atrial relaxation.
- *v wave:* tricuspid closure and atrial filling.
- *y descent:* ventricular filling as tricuspid valve opens.

*Findings*
- *Raised JVP:* right ventricular failure, tricuspid stenosis, tricuspid regurgitation, superior vena cava obstruction, PE, fluid overload.
- *Large a waves:* caused usually by 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 through an incompetent tricuspid valve.
- *Sharp y descent:* characteristic of constrictive pericarditis.
- *Sharp x descent:* characteristic of cardiac tamponade.

*Kussmaul's sign*

The JVP will reduce 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.
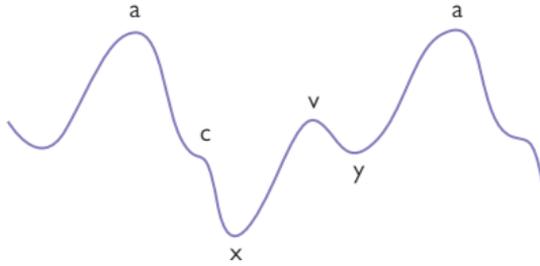
**Fig. 5.5** Representation of the normal jugular venous pulsation.

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**Box 5.4  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.

# The precordium: inspection and palpation

The 'precordium' refers to that part of the chest overlying the heart.

### 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.
- Pacemaker or implantable defibrillator.
  - Usually implanted over the left pectoral region.
- Any visible pulsations.

### Palpation

Explain what you are doing and gain consent before touching.

*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 feel any pulsations?
- *'Heave'*: a sustained, thrusting pulsation usually felt at the left sternal edge.
  - Indicates 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
  - If diastolic: mitral stenosis.

*Palpating the apex beat*

This is the lowermost lateral point at which a definite pulsation can be felt. Usually at the 5th intercostal space in the mid-clavicular line (Fig. 5.6).
- Abnormal position of the apex beat: usually more lateral.
  - 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.
  - Try asking the patient to lean forwards or laterally.
- 🛈 Beware of dextrocardia. If no beat is felt, check on the right.

*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.
  - Causes include sepsis, anaemia.
- *Sustained:* impulse 'longer' than expected.
  - Causes include left ventricular hypertrophy, aortic stenosis, hypertrophic cardiomyopathy, and 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.
  - Possible causes include emphysema, obesity, pericardial effusion, death.



**Fig. 5.6** Surface anatomy of the heart and most common location of the apex beat.

# The precordium: auscultation

### Technique

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.

- The 'bell' of the stethoscope is used to detect lower-pitched sounds, the diaphragm higher-pitched.
- You should auscultate at each of the four standard areas (Box 5.5, Fig. 5.7).

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**Box 5.5  The four 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.

🔴 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.

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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 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.)

### 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:* reduced 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 the 2nd heart sound*
- *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 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 reduced compliance—or increased 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 hypertension.



A = Aortic
P = Pulmonary
T = Tricuspid
M = Mitral
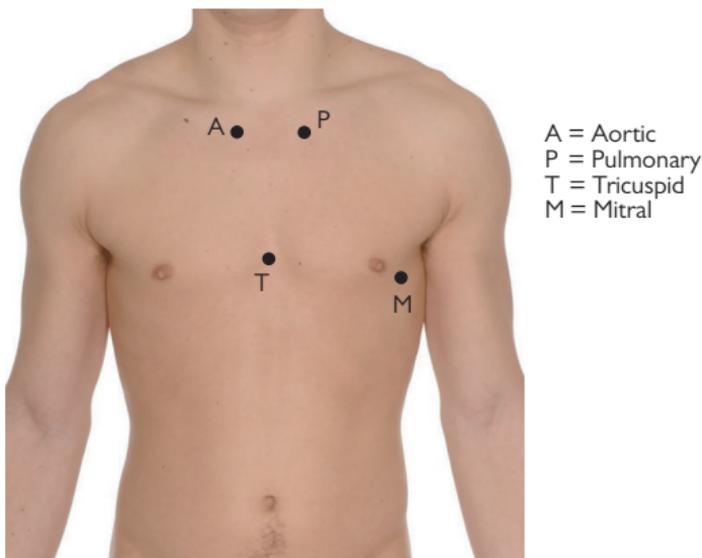
**Fig. 5.7** The four standard areas for auscultation of the precordium and the valves that are best heard at each area.

# 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 (see Box 5.6).
- 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.
- 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 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 mimic this 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 Steell murmur*
- Pulmonary regurgitation secondary to pulmonary artery dilatation caused by increased 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.

For example, the murmur of aortic stenosis will radiate up to the carotids, a mitral regurgitation murmur may be heard in the left axilla.

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**Box 5.6 Grading the volume of a murmur**

The experienced examiner should be able to give the murmur a 'grade' according to its loudness.

- 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 and heard over the whole of the precordium. 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

*Respiration*

- Right-sided murmurs (e.g. pulmonary stenosis) tend to be louder during inspiration and quieter during expiration (increased venous return). 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.
- Replicate by asking the patient to blow into the end of a syringe, attempting to expel the plunger.
  - This will reduce 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.

# **Extra sounds**

These are added sounds that are often associated with a specific murmur—see Table 5.2.

### **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. This 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 5.2** A selection of cardiac abnormalities and the expected clinical findings

| 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. 131) | |
| 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.

# Examining beyond the chest

### The lung bases
See � Chapter 6. Look especially for crackles or sign of effusion.

### The abdomen
See also ➲ Chapter 7. 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 increase in tissue fluid resulting in swelling—its causes are multiple 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).
- 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.
- Venulectasias ('venous stars'): intradermal thread veins.
- Atrophie blanche: white skin scarring without ulceration.
- Evidence of venous hypertension:
  - Lipodermatosclerosis (sclerosis of the skin and subcutaneous fat results in tapering of the legs above the ankle)
  - Venous eczema
  - Skin discoloration: brown pigmentation of haemosiderin deposition
  - Venous ulcers.

*Palpation*
- There may be pitting oedema at ankle.
- Examine for tenderness or heat over the veins (thrombophlebitis).
- Phalen's test: palpable tender defects in deep fascia along course of vein at medial border of tibia.
- Check for a cough impulse.
- 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.

*Auscultation*
- Listen over non-collapsible or large groups of varicosities to exclude an arteriovenous fistula.

*Ultrasound*
- The most reliable method of diagnosing and locating primary reflux, replacing the older clinical tests shown in Box 5.7.
- The probe is placed over the saphenofemoral junction (SFJ) 3–4cm below and lateral to pubic tubercle and saphenopopliteal junction whilst applying pressure to the calf and listening for reflux of 1–2 seconds.

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### Box 5.7 Older tests for varicose veins

▶ Doppler ultrasound has replaced these older, less reliable tests. These should not be performed and are presented here for historical interest.

*Tap test*
- With one hand resting on the medial calf along the course of a varicose vein, tap or flick the saphenofemoral junction (SFJ).
  - Transmission of the wave suggests intervening incompetent valves.

*Trendelenburg test*
- With the patient supine, empty the superficial venous system
- Press over the SFJ and ask the patient to stand whilst holding your finger in place.
  - If the veins do not fill, the site of incompetence is the SFJ
  - If the veins fill, there are incompetent perforators lower down.

*Tourniquet test*
- Uses the same principle as the Trendelenburg test, but a tourniquet is applied just below the SFJ and then caudally in a step-wise fashion until the vein-filling is prevented (this is the site of incompetence).

*Perthe's test*
- With a tourniquet over the SFJ, ask the patient to walk on tiptoes. The varicosities improve if the deep venous system is functional.

# Important presentations

## Valvular disease

*Mitral stenosis*
- *Symptoms:* dyspnoea, reduced exercise tolerance, cough productive of frothy (pink?) sputum, palpitations (often associated with atrial fibrillation and resultant emboli), dysphagia (oesophagus compressed by enlarged left atrium).
- *Signs:* palmar erythema, malar flush, 'tapping' apex beat, left parasternal heave, giant v waves in JVP, loud $S_1$, high-pitched early-diastolic (Graham Steell) murmur ± opening snap.

*Mitral regurgitation*
- *Symptoms:* acute dyspnoea and pulmonary congestion.
- *Signs:* thrusting apex beat displaced to the left (volume overload), possible systolic thrill at the apex, soft $S_1$, loud $S_2$ (pulmonary component), pansystolic murmur heard at the apex radiating to left axilla (best heard in the left lateral position) ± mid-systolic click.
- *Signs of decompensation:* small volume pulse, raised JVP, displaced thrusting apex with a systolic thrill, left parasternal heave, 3rd heart sound (Ken-tuc-ky), mid-diastolic flow murmur, bibasal crackles, peripheral oedema.

*Mitral prolapse*
- Displacement of an abnormally thickened mitral valve leaflet into the left atrium during systole, mimicking and leading to mitral regurgitation.
- Occurs in approximately 5% of the population.
- More common in females.
- *Signs:* mid-systolic click, late systolic murmur heard best at the apex.
  - Squatting delays the click and standing increases the murmur.

*Aortic stenosis*
- *Symptoms:* angina, syncope, dyspnoea, sudden death.
- *Signs:* small volume slow rising pulse ('pulsus tardus et parvus'), narrow pulse pressure, sustained and powerful apex beat (displaced if ventricular dysfunction and dilatation present), ejection systolic (crescendo-decrescendo) murmur heard at the left sternal edge and (loudest leaning forward at end-expiration) radiating to carotids, soft $S_2$.
- *Signs of decompensation:* raised JVP, left parasternal heave, gallop rhythm, bibasal crackles, peripheral oedema.

*Aortic regurgitation*
- *Symptoms:* similar to aortic stenosis.
- *Signs:* large volume 'collapsing' pulse which is exaggerated at the radial artery if you hold the patient's arm up ('waterhammer 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), you may also hear a 'pistol shot' sound over the femoral artery with severe aortic regurgitation. See Box 5.8.

**Box 5.8 Eponymous signs in aortic regurgitation**
- *Quincke's sign:* capillary nail bed pulsations
- *Corrigan's sign:* visible carotid pulsations
- *De Musset's sign:* head nodding with each heartbeat
- *Muller's sign:* pulsation of the uvula
- *Duroziez's sign:* diastolic femoral bruit when compressed distally
- *Traube's sign:* 'pistol shot' femorals
- *Austin Flint murmur:* mid-diastolic murmur heard at the apex, thought to be caused by a regurgitant jet interfering with the opening of the anterior mitral valve leaflet, mimicking the murmur of mitral stenosis
- *Rosenbach's sign:* pulsatile liver
- *Gerhardt's sign:* enlarged spleen.

*Showing off*
- Abraham Lincoln probably had Marfan's syndrome with his tall stature and long arms. In addition, a study of old photographs reveals that he had de Musset's sign. At the time when cameras had longer shutter times, President Lincoln's head-nodding caused his face to appear blurred in photographs when compared with those sitting around him
- *Prof. Heinrich Quincke:* German physician 1842–1922, also introduced the therapeutic lumbar puncture
- *Sir Dominic John Corrigan:* 1802–1880, was five-times President of the College of Physicians in Dublin
- *Alfred de Musset:* notable because de Musset's sign is named after the patient, not the doctor. De Musset was a French poet and novelist who died of syphilitic aortic regurgitation in 1857.

*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:* auscultation similar to that of mitral stenosis, hepatomegaly, pulsatile liver, and venous congestion.

*Tricuspid regurgitation*
- *Signs:* dilated neck veins, prominent v wave in JVP, pansystolic murmur louder on inspiration with a loud pulmonary component of $S_2$, left parasternal heave, pulsatile liver, peripheral and sacral oedema, ascites.

*Pulmonary stenosis*
- *Signs:* 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*
- *Signs:* loud $S_2$ which may be palpable, early diastolic murmur heard at the pulmonary area and high at the left sternal edge.

## Ventricular septal defect (VSD)

*Large*
- *Symptoms:* infant with breathlessness, poor feeding, and failure to thrive.
- *Signs:* As the pulmonary vascular resistance falls, a large defect may present with cardiac failure in the first few months of life.
  - Associated features include: low volume pulse, mid-diastolic murmur due to high flow through the mitral valve.

*Small*
- *Symptoms:* usually asymptomatic and called 'Maladie de Roger'.
- *Signs*: normal pulse, normal JVP, harsh pansystolic murmur at the lower left sternal edge, no evidence of decompensation.

## Atrial septal defect (ASD)

The commonest congenital lesion and often an asymptomatic finding discovered on investigating a murmur.

*Variants*
- *Ostium primum:* 15% of cases may be associated with mitral and tricuspid regurgitation or a VSD. Usually identified early in childhood, associated with congenital syndromes (Down's, Noonan's, Klinefelter's), ECG shows RBBB with left axis deviation.
- *Ostium secundum*: 70% of cases. Usually central fossa ovalis defects, occasionally associated with mitral valve prolapse, ECG shows RBBB with right axis deviation.
- *Sinus venosus:* 15% of cases, defect in upper septum involving inflow from SVC or IVC, associated with defects of pulmonary drainage.

*Symptoms*
- *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 of endocarditis.
- *Symptoms of secundum defect:* asymptomatic if small. Fatigue, dyspnoea, palpitations (atrial arrhythmias), recurrent pulmonary infections, right heart failure. Also migraine and paradoxical emboli.

*Signs*
- Irregularly irregular pulse (AF), apex beat undisplaced and palpable, fixed splitting of $S_2$, ejection systolic murmur at upper left sternal edge.
- *If haemodynamically significant:* irregularly irregular pulse (AF), apex beat displaced laterally, left parasternal heave (RV overload), systolic thrill over the pulmonary area, wide fixed splitting of $S_2$, ejection systolic murmur in the pulmonary area with ejection click (pulmonary artery dilatation), mid-diastolic rumble over tricuspid area (increased flow through the tricuspid valve from the large left-to-right shunt), pulmonary regurgitation.

*Holt–Oram syndrome*
- Triphalangeal thumb/other upper limb anomalies associated with ASD.
- Autosomal dominant (incomplete penetrance).

### 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:* bounding pulse, wide pulse pressure, displaced heaving apex beat, 'machinery' (continuous) murmur heard all over the precordium, $S_2$ not heard, systolic or diastolic thrill in the 2nd 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 and leg fatigue are also features. The coarctation may also cause the heart to strain and give symptoms of congestive cardiac failure.
- *Signs:* large volume radial pulse, radio-radial or radio-femoral delay, blood pressure discrepancy between upper and lower limbs, superficial collateral vessels on the chest wall, 'heaving' undisplaced apex beat, thrill over the collaterals and in the suprasternal notch, systolic or continuous murmur heard in the left infraclavicular area anteriorly and the left infrascapular area posteriorly, weak femoral pulses. May also have underdeveloped lower limbs.
- *After surgical correction:* left thoracotomy scar, normal right radial pulse, weak left radial pulse, normal heart sounds, no radio-femoral delay.

### 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, ascites ('pseudo-cirrhosis').

### Pericardial effusion

- Pulsus paradoxus, raised JVP, impalpable apex beat, soft heart sounds, hepatomegaly, ascites, peripheral oedema.

### Infective endocarditis

Bacteraemia can follow a wide range of events including dental work, brushing teeth, IV drug use, iatrogenic.

- *Symptoms:* malaise, lethargy, fevers, anorexia, weight loss, myalgia, arthralgia, heart failure, embolic stroke.
- *Signs:* pyrexia, petechial rash, splinter haemorrhages, Osler nodes (small, red/purple, raised, tender lesions often on finger pulps), Janeway lesions (irregular, flat, red, non-tender macules on palmar aspect of hands/feet), Roth spots ('cotton wool' spots on the retina), digital infarcts, digital clubbing, murmur, hepatosplenomegaly.

## Hypertrophic cardiomyopathy

Inherited forms are usually autosomal dominant.
- *Symptoms:* can present with sudden cardiac death. Otherwise asymptomatic usually. If outflow obstruction: dyspnoea, reduced exercise tolerance, palpitations, syncope, chest pain.
- *Signs:* 'jerky' peripheral pulse with a steep upstroke, 'a' wave seen at JVP, forceful apex beat, ejection systolic murmur at the left sternal edge radiating to the axilla (does not radiate to the neck) and increases in intensity on Valsalva, 4th heart sound (in some cases).
- *Poor prognostic factors:* young age at diagnosis, family history of sudden death, syncopal symptoms, ventricular arrhythmias documented on ambulatory monitoring.

## 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. It is usually described in terms of 'left' and 'right' heart failure but there is usually an element of both ('biventricular').

*Left ventricular failure (LVF)*
- *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, tachycardia, thready pulse, low blood pressure, narrow pulse pressure, displaced apex beat (murmur of an underlying valvular abnormality?), 3rd and 4th heart sounds, tachypnoea, crepitations at the lung bases.

*Right ventricular failure (RVF)*
- *Symptoms:* as LVF with peripheral oedema and facial swelling.
- *Signs:* many of those under LVF. Also raised JVP, hepatomegaly, ascites, peripheral (sacral?) oedema, pulsatile liver (if tricuspid regurgitation).

## Subclavian steal syndrome

Subclavian steal 'phenomenon' is flow reversal in the left vertebral artery due to left subclavian artery occlusive disease proximal to the origin of the vertebral artery. If associated with transient neurological symptoms due to cerebral ischaemia, it is termed 'subclavian steal syndrome'.
- *Symptoms:* exercising the left arm produces muscle cramping, dizziness, vertigo, dysarthria, syncope, diplopia, nystagmus.
- *Signs:* evidence of left upper limb ischaemia (pallor, cyanosis, ulcers), left radial and brachial pulses weak, reduced systolic blood pressure in the left arm, supraclavicular bruit on the left.

## Deep vein thrombosis (DVT)

Often confused with cellulitis and ruptured popliteal (Baker's) 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.

**See also:**
More information regarding the presentation and clinical signs of cardiovascular diseases to aid preparation for OSCE-type examinations and ward rounds can be found in the *Oxford Handbooks Clinical Tutor Study Cards*.

*'Medicine' Study Card set:*
- Mitral stenosis and regurgitation
- Aortic stenosis and regurgitation
- Mixed valve disease
- Prosthetic valves
- Ventricular septal defect
- Atrial septal defect
- Hypertrophic cardiomyopathy
- Coarctation of the aorta
- Persistent ductus arteriosus
- Fallot's tetralogy
- Dextrocardia
- Infective endocarditis.

*'Surgery' Study Card set:*
- Thoracic outlet syndrome
- Subclavian steal
- Carotid artery disease
- Superior vena cava obstruction
- Axillary vein thrombosis
- Deep vein thrombosis
- Ischaemic ulcer
- Abdominal aortic aneurysm
- Thrombophlebitis obliterans
- Raynaud's phenomenon
- Surgical arteriovenous (AV) fistulae
- Lymphoedema.

*'Practical Procedures' Study Card set:*
- Venepuncture
- Sampling from a central venous catheter
- Femoral venous catheter insertion
- Central venous access
- Arterial line insertion
- Recording a 12-lead ECG
- Carotid sinus massage
- Vagal manoeuvres
- Defibrillation
- DC cardioversion
- Temporary external pacing
- Pericardiocentesis
- Exercise tolerance testing.

# The elderly patient

Geriatricians are equally interested in cardiovascular disease—with an ageing population, the prevalence of cardiac, peripheral vascular, and stroke disease continues 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. Exertional breathlessness may reflect an arrhythmia.
- *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 beta-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 (check oxygen saturations!), the GTN spray close at hand, etc.
- *Auscultate 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, CABG, or TAVI 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.

# The respiratory system

# Introduction

### Anatomy

The respiratory tract extends from the nostrils to the alveoli but also includes the pulmonary parenchyma and vasculature and the musculoskeletal structures required for ventilation. It is often divided for convenience 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', at about the level of the sternal angle. There are about 25 further divisions before reaching the alveoli.

*Lungs*

The right lung has 3 lobes (upper, middle, and lower) whilst the left lung has 2 (upper and lower) to make room for the heart, but the lingular division of the upper lobe is effectively a 'left middle lobe '. Note that the oblique fissures run downwards from the back. Here's what this means when auscultating (see Fig. 6.1).

The diaphragm slants such that the inferior border of the lungs is at the 6th rib anteriorly but extends down to the 12th posteriorly.

### Physiology

This is a complex system, the outline here is an aide-mémoire only.

*Ventilation*

- Central processing.
  - Pacemaker respiratory centre
  - Influence from higher voluntary centres, emotional centres, and circulating endocrine factors.
- Sensors.
  - Brainstem and aortic arch chemoreceptors
  - Lung stretch and cough receptors.
- Effectors.
  - Diaphragm
  - Intercostal muscles
  - Accessory muscles (e.g. sternocleidomastoids).

Contraction of effector muscles increases thoracic volume and air is drawn in, expiration is largely passive with air being expelled as the lungs recoil under their innate elasticity. During physiological stress, ventilation increases first by increasing tidal volume then by increasing rate: to fit more breaths into a minute, expiration must therefore become active.

*Gas transfer*

Getting enough air in is the first step, but we must also extract oxygen and get rid of carbon dioxide. Anything that impedes gas transfer has clinical implications:

- Inadequate global ventilation (e.g. muscular dystrophy).
- Inadequate local perfusion of ventilated area (e.g. PE).
- Inadequate local ventilation of perfused area (e.g. pneumonia).
- Thickened barrier to diffusion (e.g. pulmonary fibrosis).

Note that the degree of ventilation–perfusion mismatch will be altered by a patient's position and cardiac output.

*Defence*

Cough receptors in the pharynx and lower airways initiate a deep inspiration followed by expiration against a closed glottis and a sudden glottal opening. This causes a rapid, forceful expulsion of air.

Larger inhaled particles will impact on airway walls going round the many corners of the respiratory tract. Particles smaller than this might have time to sediment out from the air deep in the lungs (like inhaled medications), before they can be exhaled.

Most of the respiratory tract is lined with mucus secreted from goblet cells that catches these inhaled particles. This is continuously swept upwards like an escalator by cilia, towards the larynx where the mucus is swallowed (yes, we all do it).

In the smaller airways and alveoli, macrophages and a variety of secreted defensive proteins act against microbes at a microscopic level.
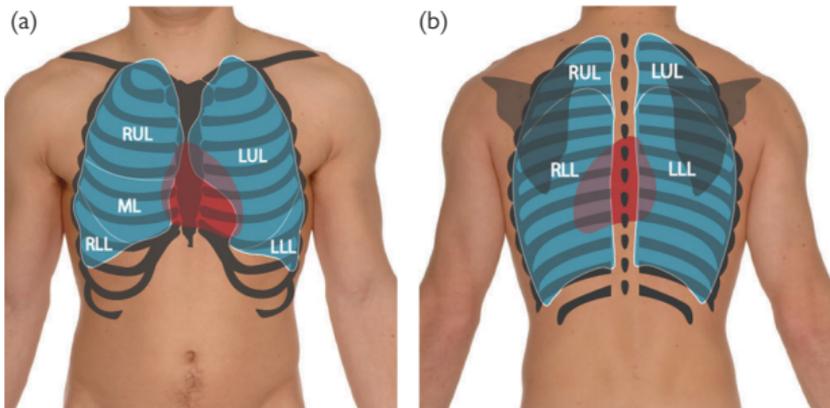
**Fig. 6.1** Surface anatomy of the lungs. UL: upper lobe, ML: middle lobe, LL: lower lobe.

# 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'.

This is NOT the same as 'hypoxia'. A person can be breathless but have normal oxygen levels. Marathon runners crossing the finish-line are breathless but not blue.

'Tightness' is often described and may relate to airway narrowing as in asthma or may be chest pain, as in cardiac disease. Tease out exactly what the patient means.

⚠ Pleuritic and musculoskeletal chest pain is worse at the height of deep inspiration and patients may say 'I am not able to get my breath'. Thus, seemingly complaining of breathlessness, their actual problem is pain on inspiration. Ask if they feel unable to breathe deeply and for what reason (is it pain or some other sensation?). If all else fails, ask the patient to take a deep breath and watch what happens.

### Onset and duration

How quickly did the SOB come on? (see Box 6.1)

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**Box 6.1 Some causes of dyspnoea by onset**

- Abrupt
  - Pulmonary embolus
  - Pneumothorax
  - Acute exacerbation of asthma.
- Days/weeks
  - Asthma exacerbation
  - Pneumonia
  - Congestive cardiac failure.
- Months
  - Pulmonary fibrosis.
- Years
  - Chronic obstructive pulmonary disease.

---

Slower onsets are poorly reported. The patient often reports the onset of a *worsening* of breathlessness or when the breathlessness stopped them doing their benchmark daily activity. Ask when they were last able to run up the stairs and the real duration of breathlessness becomes apparent.

The nature of progression of breathlessness is also crucial: asthma may be long-standing and fluctuate greatly whereas fibrosis inexorably gets worse (often in a step-wise fashion).

### Severity

Several classifications exist (see Box 6.2) but the key is to quantify in terms of progressive functional impairment whilst trying to keep it in context for the patient, e.g. 'Can you still mow the lawn without resting?', 'Do you have

to walk slower than your friends?', 'Are you breathless getting washed and dressed in the morning?'

🔔 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? Remember orthopnoea (➔ p. 104) is not specific for heart failure: breathing whilst lying relies heavily on the diaphragm and also increases perfusion of the upper lobes (usually most badly damaged in COPD) so many people with dyspnoea are more breathless doing this.

What makes the dyspnoea better? Do inhalers or a break from work help?

### Hyperventilation

Dysfunctional breathing, and particularly hyperventilation, is common generally and more so in people with genuine respiratory pathology. Hyperventilation ↓ blood $CO_2$ and so increases pH. This leads to symptoms of dyspnoea of rapid onset then:

- Early.
  - Paraesthesia in the lips and fingers
  - Light headedness
  - Chest pain or 'tightness'.
- Prolonged episode.
  - Bronchospasm
  - Post episode hypoxia ($SpO_2$ can be <85%).

---

**Box 6.2  MRC Dyspnoea Score**

- **1** = Not troubled by breathlessness except on strenuous exercise
- **2** = Short of breath when hurrying or walking up a slight hill
- **3** = Walks slower than contemporaries on level ground due to breathlessness, or has to stop for breath when walking at own pace
- **4** = Stops for breath after walking 100m or less on level ground
- **5** = Too breathless to leave the house, or breathless when dressing or undressing.

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# Cough and expectoration

## Cough

A common, often overlooked and potentially miserable symptom in respiratory disease, usually caused by upper respiratory tract infection (URTI) and/or smoking. Duration of cough is important, as well as character, exacerbating factors, and whether any sputum is produced. See Tables 6.1a and 6.1b for some causes of cough.

Note that cough may be the only reported symptom of asthma.

### Localizing the cough

This is not particularly useful, however patients are often keen to try to point out where they feel the cough originates.

Beyond the larynx, sensory innervation is such that localization is not possible. Patients often, therefore, point to their throat as the source of the cough.

### Chronic cough

'Chronic cough' is that lasting >8 weeks and is often multi-factorial: common contributors are initial viral infection, asthma, post-nasal drip, gastro-oesophageal reflux disease, and medications (though it can be the first manifestation of interstitial lung disease or even lung cancer).

🔔 Smokers will have a chronic cough, particularly in the mornings, so a history of a change is important.

## 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 and 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 grey in cigarette smokers. Yellow or green 'purulent' sputum is largely caused by inflammatory cells so usually indicates infection; although eosinophils in the sputum of asthmatics also discolour sputum, producing rubbery yellow plugs. See Table 6.2.

## Haemoptysis

The coughing up of blood can vary from streaks to massive, life-threatening bleeds ('massive' haemoptysis = >500ml in 24 hours). Establish amount, colour, frequency, and nature of any associated sputum.

Haemoptysis is 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 infection, bronchiectasis, carcinoma, pulmonary embolus, and pulmonary vasculitis. 'Infective' causes will often produce blood-stained sputum as opposed to pure haemoptysis.

Table 6.1a Some clues to the origin of a cough (acute)

| Cause | Character |
| --- | --- |
| Laryngitis | Cough with a hoarse voice |
| Tracheitis | Dry and very painful |
| Epiglottitis | 'Barking' |
| LRTI | Purulent sputum, perhaps with pleuritic chest pain |

Table 6.1b Some clues to the origin of a cough (chronic)

| Cause | Character |
| --- | --- |
| Asthma | Chronic, paroxysmal, worse after exercise and at night |
| Oesophageal reflux | Dry and nauseating. Often first thing in the morning, after eating, or with prolonged talking |
| Pulmonary oedema | Clear sputum, worse on lying flat |
| Postnasal drip | Tickly, often with nasal blockage |

Table 6.2 Some classical characteristics of sputum

| Age | Heart rate |
| --- | --- |
| White/grey | Smoking |
| Green/yellow | Bronchitis, bronchiectasis |
| Green and offensive | Bronchiectasis, abscesses |
| Sticky, rusty | *Streptococcus pneumoniae* infection |
| Frothy, pink | Congestive cardiac failure |
| 3 layers (mucoid, watery, rusty) | Severe bronchiectasis |
| Very sticky, often yellow | Asthma |
| Sticky, yellow but with large plugs | Allergic bronchopulmonary aspergillosis |

# Other respiratory symptoms

### Wheeze

This is a whistling 'musical' sound emanating from narrow smaller airways. Occurs in inspiration and expiration, but usually louder and more prominent in the latter. Airway calibre is dynamic, and the external pressure in expiration means this is when airways are narrowest and when you'll hear wheeze. Cause may be any process that ↓ airway calibre:

- Airway muscle contraction: asthma.
- Reduced airway support tissue: COPD.
- Airway oedema: heart failure.
- Airway inflammation/mucus: bronchiectasis.

### Stridor

A harsh 'crowing', predominantly inspiratory, sound with a largely constant pitch. Signals large airway narrowing, usually at the larynx or trachea, (e.g. vocal cord palsy, post intubation stenosis). Can precede complete airway obstruction (e.g. epiglottitis) so is treated as a medical emergency if the cause is unknown.

### Chest pain

Chest pain is explored fully in ⟶ Chapter 5.

*Pleuritic pain*

Pain arising from respiratory disease may be 'pleuritic' in nature: usually arising from the parietal pleura (the lungs have no pain fibres). It is felt as a severe, sharp pain at the height of inspiration or on coughing localized to a small area of chest wall. Note that patients will avoid deep breathing and may complain of 'breathlessness'.

*Lung parenchymal pain*

Pain from lung parenchymal lesions may be dull and constant. This is a sinister sign of malignancy spreading into the chest wall. Remember, though, that the stress placed through the chest wall by increased respiratory effort in other airways disease may cause ill-defined chest wall pain.

*Diaphragmatic pain*

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.

*Musculoskeletal pain*

In general, muscular and costal lesions will be tender to touch over the corresponding chest wall and exacerbated by twisting movements—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.

*Nerve root pain*

May be due to spinal lesions or herpes zoster.

### Somnolence

Sleepy people are often seen by respiratory physicians as the commonest pathological cause (obstructive sleep apnoea) usually requires commencement of nocturnal non-invasive ventilation.

Differentiate sleepiness from fatigue: think of how you feel after exercise and how you feel after being awake a long time (e.g. after a long-haul flight). Quantify how sleepy the patient is (see Epworth Score in Box 6.3).

*Obstructive sleep apnoea (OSA)*

This is caused by upper airway obstruction in susceptible individuals (overweight/retrognathic/relative macroglossia) as the palatal muscles become flaccid during REM sleep. Partial obstruction causes snoring then brief hypoxia as the obstruction becomes complete. Hypoxia is sensed and the patient wakes enough to return tone to their muscles and open their airway. This cycle is repeated many times per hour (sleepiness), the patient is restless and noisy (sleepy, irritated partner), and blood pressure doesn't fall at night (can give resistant hypertension).

Severe OSA leads to carbon dioxide retention, worsening somnolence, and early morning headaches.

*Narcolepsy*

Narcolepsy is less common than OSA but disabling and the diagnosis is often missed for years. Initially, patients experience weakening at the knees when experiencing sudden emotion (e.g. the punchline of a joke). This 'cataplexy' progresses to become more marked and widespread, sleep episodes suddenly occur at any time (e.g. mid-conversation), and dreams intrude into wakefulness. Strong genetic linkage.

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#### Box 6.3 Epworth Sleepiness Scale

Ask the patient to choose a numbered grade for each situation and then add the numbers to give an overall score:

*Grading*

0 = would never doze or sleep
1 = slight chance of dozing or sleeping
2 = moderate chance of dozing or sleeping
3 = high chance of dozing or sleeping.

*Situations*

- Sitting and reading
- Watching TV
- Sitting inactive in a public place
- Being a passenger in a motor vehicle for an hour or more
- Lying down in the afternoon
- Sitting and talking to someone
- Sitting quietly after lunch (no alcohol)
- Stopped for a few minutes in traffic whilst driving.

*Results*

0–10 = Normal; 10–12 = borderline; 12–24 = abnormally sleepy

# The rest of the history

## Other key symptoms

*Fever*

Particularly at night may be a sign of infection such as TB, but remember fever is caused by inflammation so may arise from malignancy, PE, or a connective tissue disorder.

*Weight loss*

A common symptom of cancer, COPD, and chronic infection. Attempt to quantify any loss (how much in how long).

*Peripheral oedema*

Oedema manifesting as ankle swelling at the end of the day may be a sign of fluid retention due to chronic hypoxaemia ± hypoxia or right heart failure secondary to chronic lung disease (cor pulmonale). Older smokers with COPD often have coexisting cardiac disease.

## Past medical history

- Vaccination for respiratory illnesses, particularly BCG.
- Previous respiratory infections especially TB before 1950 when surgery 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.
- Multisystem disorders that affect the chest e.g. rheumatoid.

## Drug history

Many medications can cause respiratory pathology – if unsure consult resources such as Pneumotox ($\wp$ http://www.pneumotox.com).

- What inhalers are used and how often? Check inhaler technique.
- Previous successful use of bronchodilators and steroids.
- Immunosuppressives including oral steroids predispose to (often atypical) infection.
- ACE inhibitors cause a dry cough.
- If $O_2$ therapy—cylinders or concentrator? How many hours a day?
- Illicit drug use (cannabis causes emphysema, many others are associated with respiratory disease).

## Family history

- Asthma, eczema, and allergies.
- Inherited conditions (e.g. alpha-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. 20 cigarettes is roughly the same risk exposure as 0.5oz (12.5g) of tobacco.

Ask about previous smoking as many will call themselves ex-smokers if they gave up on their way to see you!

Remember to ask about passive smoking.

### Alcohol

Alcoholics are at greater risk of chest infections and bingeing may result in aspiration pneumonia.

### Social history

*Pets*

Animals 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 *Legionella* can be caught from water systems and air-conditioning in developed countries. Pathogens common in other developed countries may be different to those in the UK (e.g. histoplasmosis in the USA) or show extensive antibiotic resistance.

*Occupation*

This is hugely important. Individual occupational diseases might be uncommon but collectively they represent a vast number of cases. Be alert to exposure to asbestos, coal, animals, metals and ores, cement dust, and organic compounds.

Trace the occupational history back as there may be a lag of >20 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 well have been exposed to asbestos in the past, as might their families, e.g. by washing clothes.

See Health and Safety Executive (HSE) website ℅ http://www.hse.gov.uk for more information.

# 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, make sure you have introduced yourself and have clean hands.

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? Spacer device?
- Nebulizer? NIV machine?
- Is the patient receiving $O_2$ therapy? If so, how much and by what method (i.e. face mask, nasal cannula, etc.)?
- Sputum pot? – look inside!
- 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? or after undressing/walking in?
- Count the respiratory rate. At rest, this should be <15/minute.
  - Pretend to be checking the pulse if you think your observation is changing the patient's breathing pattern.
- Are the breaths of normal volume? (Patients with neuromuscular or fibrotic disease have more shallow and rapid breathing.)
- Expiration should be shorter than inspiration (about 2:1), but this will be reversed in obstructive lung diseases as the patient tries to prevent airway collapse from external pressure.
- Are they breathing through pursed lips? (increasing the end-expiratory pressure—an indication of chronic obstructive lung disease.)
  - Patients with airway obstruction have a high residual volume (↑ airway radial traction/incomplete expiration due to airway collapse).
- Are they using the accessory respiratory muscles (e.g. sternomastoids) or bracing their arms to splint their chest? (The classic position is sitting forwards, hands on knees.)
- Does the abdomen move out in inspiration? Or is a weakened diaphragm being drawn up and hence the abdomen inward (abdominal paradox)?

*Abnormal breathing patterns*
- Kussmaul's respiration: deep, sighing breaths. Systemic acidosis.
- Cheyne–Stokes breathing: a waxing and waning of breath amplitude and rate. Due to failure of the normal respiratory regulation in response to blood $CO_2$ levels. Commonly seen after cerebral insult (poor prognostic sign) or in heart failure (patient often relatively well).
- Other characteristic neurogenic ventilation patterns are described but are far less common.

## Listen before 'auscultating'

- Is the speech limited by their breathlessness? If so, can they complete a full sentence?
- Listen for hoarseness as well as the gurgling of excess secretions.
- A nasal voice may indicate neuromuscular weakness.
- Listen for coughing (see previous pages) as well as stridor and wheeze.

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### Skills station 6.1

*Instruction*

Examine this patient's respiratory system.

*Model technique*

- Clean your hands.
- Introduce yourself.
- Explain the purpose of the examination, obtain informed consent.
- Ask for any painful areas that you should avoid.
- Note the patient's general appearance and demeanour.
- Note any bedside clues.
- Ask the patient to undress to the waist and sit comfortably at 45°.
- Measure the patient's respiratory rate and breathing pattern.
  - Some practitioners like to do this whilst pretending to feel the patient's radial pulse. In this way, the patient does not become self-conscious and breathes as they normally would.
- Examine the hands.
  - Note staining, cyanosis, clubbing, radial pulse
  - Assess for tremor.
- Examine the JVP.
- Look in the nose, mouth, and eyes.
- Feel for cervical, supraclavicular, and axillary lymph nodes.
- Inspect the chest.
- Assess mediastinal position and chest expansion, front and back.
- Percuss front and back, comparing sides.
- Auscultate front and back, comparing sides.
- You may wish to consider other bedside tests such as PEFR or simple spirometry.
- Thank the patient and help them re-dress if necessary.

# 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, evident when >2.5g/dl of reduced haemoglobin is present ($O_2$ sat. 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 is caused by peripheral vascular disease and vasoconstriction.

### Digital 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.

The most important respiratory causes are carcinoma and lung fibrosis but it is also seen in chronic sepsis (bronchiectasis, abscess, empyema, cystic fibrosis).

### Pulse
Rate, rhythm, character. A tachycardic 'bounding' pulse = $CO_2$ retention.

### Tremor
- *Fine tremor:* caused by use of β-agonist drugs (e.g. salbutamol).
- *Flapping tremor (asterixis):* flapping when holding the hands dorsiflexed with the fingers abducted (Fig. 6.2). Identical to the flap of hepatic failure. Late sign of $CO_2$ retention, so uncommon.

### Blood pressure
Pulsus paradoxus. Causes: pericardial effusion, severe asthma (but there should be some other clues to severe asthma!).

### JVP
See Raised in pulmonary vasoconstriction or pulmonary hypertension and right heart failure. Markedly raised, without a pulsation, in superior vena cava obstruction with distended upper chest wall veins, facial and conjunctival oedema (chemosis).

### Nose
Examine inside (nasal speculum) and out, looking for polyps (asthma), deviated septum, and lupus pernio (red/purple nasal swelling of sarcoid granuloma).

### Mouth
Look especially for candidiasis (common in those on inhaled steroids or immunosuppressants).

### Eyes
- *Conjunctiva:* evidence of anaemia?
- *Horner's syndrome:* caused by compression of the sympathetic chain in the chest cavity (tumour, sarcoidosis, fibrosis).
- *Iritis:* TB, sarcoidosis.
- *Conjunctivitis:* TB, sarcoidosis.

### Lymph nodes
See ➔ Chapter 3 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. 6.3).



**Fig. 6.2** Looking for a flapping tremor. Wrists are dorsiflexed and fingers abducted.
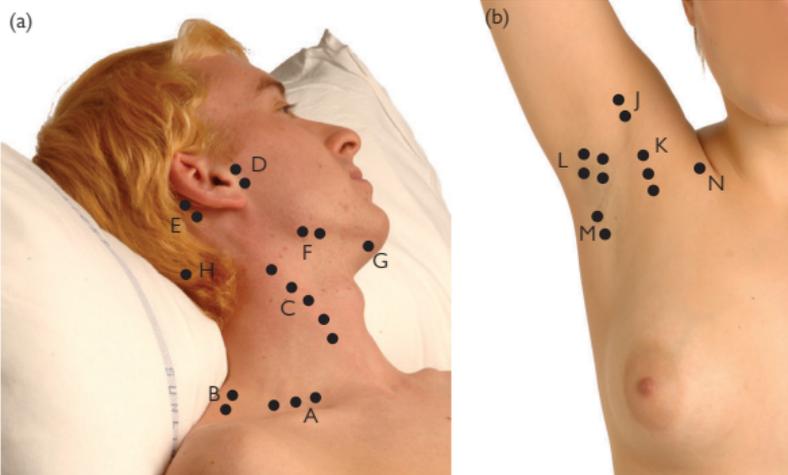


**Fig. 6.3** Cervical, supraclavicular, and axillary lymph nodes. A: supraclavicular, B: posterior triangle, C: jugular chain, D: preauricular, E: postauricular, F: submandicular, G: submental, H: occipital, J: lateral, K: pectoral, L: central, M: subscapular, N: infraclavicular.

# Inspection of the chest

Look at the shape and movement of the chest up-close.

## Surface markings

*Scars*

May indicate previous surgery. Look especially in the mid-axillary lines for evidence of past chest drains. Remember a pnemonectomy can be undertaken with a relatively small lateral scar.

*Radiotherapy*

Radiotherapy will often cause lasting local skin thickening and erythema. Sites are usually marked with tattoo dots.

*Veins*

Look for unusually prominent surface vasculature suggesting obstructed venous return.

## 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 chronic obstructive lung disease.
- *Pectus carinatum:* also called 'pigeon chest'. Sternum and costal cartilages are prominent and protrude from the chest. Can be 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, not usually of any clinical significance.
- *Surgical emphysema:* air in the soft tissues will appear as a diffuse swelling in the neck or around a chest drain site and will be 'crackly' to the touch.

## Breathing pattern

Again, note the rate and depth of breathing as you did at the end of the bed (you only need formally time it once).

## 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 usually indicates lung disease on that side.
- ↓ movement globally is seen in COPD or neuromuscular conditions.
- Harrison's sulcus is a depression of the lower ribs just above the costal margins and is occasionally seen in the context of severe childhood asthma.

# Palpation

## Mediastinal position

*Trachea*

The trachea will shift as the mediastinum is pulled or pushed laterally (e.g. by fibrosis or mass). It should lie in the midline deep to the sternal notch.

You'll need to push down as well as back otherwise you are just checking the position in the neck: so warn the patient it will be uncomfortable. Use two fingers and palpate the sulci either side of the trachea at the same time. They should feel of identical size (Fig. 6.4).

The trachea often feels central even if there is pathology, but if you do feel a deviation it may be instructive and other signs should be sought.

*Apex beat*

Normally at the 5th intercostal space in the mid-clavicular line. However, the apex beat is difficult to localize in the presence of hyperexpanded lungs and it may be shifted to the left if the heart is enlarged.

## Chest expansion

🛈 It is important to explain what you are doing here before grabbing hold of the patient's chest! See also Box 6.4.

*Antero-posterior diameter*
- Put both hands lightly on the anterior wall of the patient's chest above the nipples, fingers toward the clavicles.
- Ask the patient to breathe all the way out, then take a deep breath in: your hands should move equally.

*Lateral diameter (from the front)*
- Place both hands on the chest wall just below the level of their nipples, anchoring your fingers laterally at the sides (Fig. 6.5).
- Extend your thumbs so that they touch in the midline when the patient is in full expiration (or as near as you can if you have small hands or a large subject).
- Ask the patient to take a deep breath in. 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.

Most sources recommend testing lateral expansion at the front and back: this is almost testing the same thing twice but is a good way of ensuring you had the right answer initially.

To test expansion posteriorly it is easiest to ask the patient to lean forward and place your hands on the chest wall with the thumbs pointing *down*. The procedure can then be repeated.

## Tactile vocal fremitus

This is the vibration felt on the chest as the patient speaks. It gives the same information as vocal resonance testing so is now rarely tested.

> **Box 6.4 A word on the female chest**
>
> Breasts come in different shapes and sizes. 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, rather than trying to reach below them.



**Fig. 6.4** Palpating the trachea. Methods vary and students are taught to use either one, two, or even three fingers. We suggest using two fingers to palpate the sulci either side of the trachea at the sternal notch. These should feel symmetrical.



**Fig. 6.5** Placement of the hands for testing chest expansion. Anchor with the fingers and leave the thumbs free-floating.

# **Percussion**

### **Technique**

This takes some practice to master so serves as a sound indicator of how much time a student has spent on the wards (it does also give extremely useful information in clinical practice!).

The aim is to tap the chest and listen to and feel for the resultant vibration (see Figs 6.6 and 6.7). For a *right-handed* examiner:

- Place the left hand on the chest wall, fingers separated and middle finger lying between the ribs.
- Press the middle finger firmly against the chest (students often don't press hard enough).
- Using the middle finger of the right hand, strike the middle phalanx of the middle finger of the left hand (Fig. 6.6). You'll have to hit yourself harder if the left hand is not firmly applied.
- 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).

🛈 Students quickly learn to keep the middle fingernail of their right hand well-trimmed!

- Practise on yourself, friends, and on objects around the house. You'll soon learn the different feel and sound produced by percussing over hollow and dense objects like the lung and the liver.
- In clinical practice, one should percuss each area of the lung, each time comparing right then left.
- 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 is 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' is heard/felt over areas of ↑ density (consolidation, collapse, alveolar fluid, pleural thickening, peripheral abscess, neoplasm).
- 'Stony dullness' is the unique extreme dullness heard over a pleural effusion.
- 'Hyper-resonance' indicates areas of ↓ density (emphysematous bullae or pneumothorax).
  - COPD can create a globally hyper-resonant chest.

*Normal dull areas*

- There should be an area of dullness over the heart which may be diminished in hyperexpansion states (e.g. COPD or asthma).

The liver is manifested by an area of dullness below the level of the 6th rib anteriorly on the right. This will be lower with hyperinflated lungs.
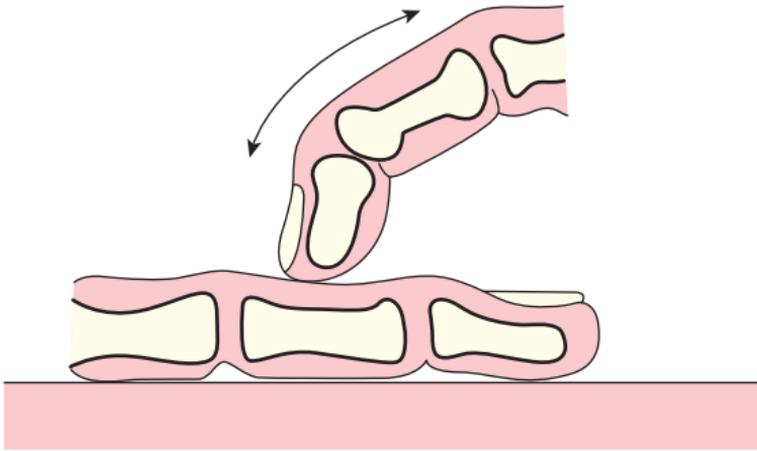
**Fig. 6.6** Strike the middle phalanx of the middle finger of the left hand with the middle finger of the right hand. Withdraw the striking finger quickly so as not to muffle the sound and feel.



**Fig. 6.7** Areas of the chest to percuss. Test right versus left for each area, front and back. You may examine the apices by percussing directly on the patient's clavicles—this does hurt a little, though.

# Auscultation

## Technique

The diaphragm of the stethoscope 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'. See also Box 6.5.
- Listen to the whole of 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 (see Table 6.3 for specific lobes).
- Listen for the breath sounds and any added sounds—and note at which point in the respiratory cycle they occur.

---

**Box 6.5  Patient performance**

Many patients have difficulty performing correctly here. They may take one deep breath and hold it, may breathe 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.

▶ Remember also that taking maximal forced breaths in and out will create additional noises in many healthy individuals, and will lead to symptoms from hyperventilation by the end of the examination. You may need to calm down your enthusiastic patients.

---

## 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. Can be heard over squashed lung above a pleural effusion.

*Added sounds*

- *Wheeze (rhonchi):* musical whistling sounds caused by narrowed airways. Heard 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.

- *Crackles (crepitations, rales):* caused by air entering collapsed airways and alveoli producing an opening snap or by mucus moving. 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. They sound like the tear of 'Velcro®' and can also be reproduced by rolling the hair at your temples between the thumb and forefinger. Usually fluid or fibrosis
  - The 'deciduous' crackles of bronchiectasis are of predominantly coarse type but fall away in volume and depth of note on inspiration
  - ❗ 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 crunch of a footstep in fresh snow—once you've heard it you'll remember. Heard best at the height of inspiration, and may be very well localized. 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.
- Sound transmitted though solid material (consolidated or collapsed lung) travels much better than through healthy air-filled lung, so phonation is more clearly heard.
- Ask the patient to say 'ninety-nine' or 'one, one, one' and listen over the same areas as before.
- Lower pitched sounds transmit particularly well so create a vocal 'booming' quality (this is why the original German 'neun und neunzig' works better than 'ninety nine').
- Marked ↑ resonance, such that a whisper can be clearly heard is termed 'whispering pectoriloquy'.

Table 6.3 Auscultating specific lung lobes

| Lobe | Where to listen |
| --- | --- |
| Upper | Anteriorly, nipple level and above |
| Right middle/left lingula | Anterolaterally |
| Lower | Posteriorly |

# Important presentations

### Pneumonia

*Inspection*
- Look for sputum pot at bedside.
- Tachypnoeic, tachycardic, or hypotensive?
- Warm peripheries.
- Bounding pulse.
- Sweaty and clammy.

*Palpation*
- Reduced expansion on the affected side.
- Increased tactile vocal fremitus if consolidation.

*Percussion*
- Dull.

*Auscultation*
- Coarse crackles, localized.
- Bronchial breathing (possible).
- Whispering pectoriloquy.
- Reduced air entry.
- Increased vocal resonance.

### Lobar collapse

*Palpation*
- Mediastinal shift towards the abnormality.
- Potentially ↓ chest wall movement locally.

*Percussion*
- Dullness to percussion restricted to affected lobe.

*Auscultation*
- ↓ breath sounds usually.

### Pleural effusion

*Inspection*
- Reduced chest expansion unilaterally (if large).

*Palpation*
- Trachea may be pushed away from the effusion.
- Apex beat:
  - A large right effusion will displace the cardiac apex to the left
  - A large left effusion may make the apex beat difficult to palpate.

*Percussion*
- 'Stony dull'.

*Auscultation*
- Markedly reduced breath sounds.
- Reduced vocal resonance.
- Collapsed or consolidated lung above the effusion may produce an overlying region of bronchial breathing.

### Pneumothorax

*Inspection*
- No mediastinal shift (only occurs with a tension pneumothorax).
- Chest wall asymmetry may be evident with a large pneumothorax (greater volume on affected side).

*Percussion*
- Hyper-resonant.

*Auscultation*
- ↓ breath sounds on affected side.
- ↓ vocal resonance on affected side.

### Interstitial fibrosis

*Inspection*
- Patients may be cyanosed.
- There may also be signs of connective tissue disease or skin changes of radiotherapy.
- Clubbing is common.

*Palpation*
- Trachea may move towards the fibrosis in upper lobe disease.
- ↔ or ↓ chest wall movement.

*Percussion*
- ↔ percussion note.

*Auscultation*
- ↔ breath sounds.
- ↔ vocal resonance usually, may be increased if dense fibrosis.
- Fine 'Velcro®' crackles.

### COPD

*Inspection*
- Inhalers at the bedside.
- Sputum pot?
- Thin skin with bruising (use of steroids).
- Use of accessory muscles/brace position.
- Tachypnoea.
- No mediastinal shift.
- Chest hyper-expanded with little additional excursion.
- Prolonged expiration and pursed lip breathing.

*Percussion*
- May be globally hyper-resonant to percussion.

*Auscultation*
- ↓ breath sounds globally, may be additional polyphonic wheeze.
- ↓ vocal resonance usually in the upper lobes (where bullae are commonest).
- Heart sounds often quiet.

### Bronchiectasis

*Inspection*
- Often copious sputum (usually purulent, may contain blood).
- Digital clubbing may be present.
- Low BMI.

*Palpation*
- No mediastinal shift.
- Chest wall expansion equal.

*Percussion*
- Percussion resonant.

*Auscultation*
- Mixed, predominant coarse crackles.
- Often additional polyphonic wheeze.
- Vocal resonance normal.

### Neuromuscular insufficiency

Intrinsic muscle weakness or damaged innervations.

*Inspection*
- Non-respiratory signs of neuromuscular illness (e.g. altered phonation, limited mobility).
- Rapid shallow breathing, sometimes with abdominal paradox.

*Palpation*
- Chest wall expansion equal but limited excursion.

*Percussion*
- Percussion note resonant.

*Auscultation*
- Breath sounds normal.
- Basal crackles common from atelectasis (impaired cough).

**See also:**

More information regarding the presentation and clinical signs of respiratory diseases, to aid preparation for OSCE-type examinations and ward rounds, can be found in the *Oxford Handbooks Clinical Tutor Study Cards*.

*'Medicine' Study Card set:*
- Chronic obstructive pulmonary disease
- Interstitial lung disease
- Lobectomy
- Pleural effusion
- Pneumothorax
- Previous tuberculosis
- Pneumonia
- Obstructive sleep apnoea
- Cystic fibrosis
- Kartagener's syndrome
- Bronchiectasis
- Superior vena cava obstruction
- Chronic cor pulmonale.

# 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 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:* under-nutrition 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 (under-nutrition, 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 under-treated. Spirometry is a key investigation.
- *Tuberculosis:* increased in the elderly—through reactivation, chronic illness, under-nutrition. Presents non-specifically—cough, lethargy, weight loss.
- *Smoking status:* The patient may be on long-term oxygen, but may still smoke! Consider nicotine withdrawal as a cause of agitation in hospitalized older patients. They may not be able to go out to smoke, and nicotine patches (on discussion with senior nursing and medical staff) may be very helpful.

# The abdomen

# Introduction

The abdomen includes the perineum, external and internal genitalia, and the inguinal regions. The male and female genitalia are discussed later in this book.

### 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 7th to 12th 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, gallbladder 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 nine portions for descriptive purposes. Four imaginary lines can be drawn (see Fig. 7.1):
- One horizontal line between the anterior superior iliac spines.
- One horizontal line between the lower border of the ribs.
- Two vertical lines at the mid-clavicular point.

To make life easier, the abdomen can also be simply divided into four quadrants by imagining one horizontal and one vertical line crossing at the umbilicus (see Fig. 7.2).
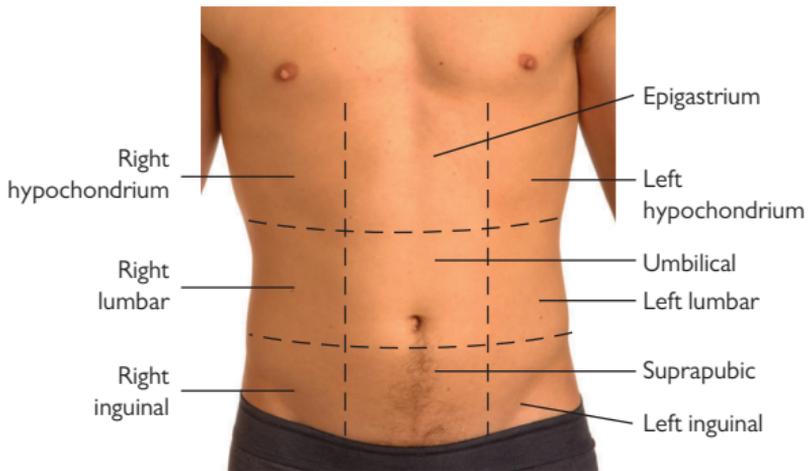
**Fig. 7.1** The nine segments of the anterior abdominal wall. Students should familiarize themselves with these, along with the organs lying in each area.
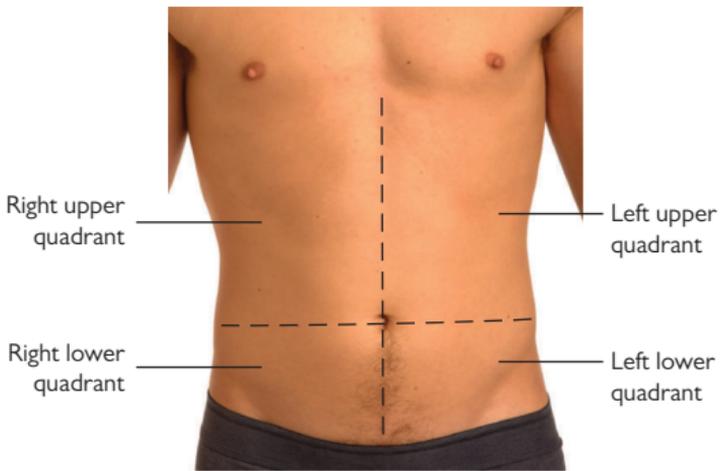


**Fig. 7.2** The four quadrants of the abdomen.

# Swallowing symptoms

## Physiology of swallowing

The swallowing process is controlled by the medulla initially and by an autonomous peristaltic reflex coordinated by the enteric nervous system in the mid- and distal-oesophagus. This complex process can be divided into three phases:

*Oral phase*
- Food enters the oral cavity.
- Mastication and bolus formation.

*Oro-pharyngeal phase*
- Tongue elevates and propels the bolus to the pharynx.
- The soft palate elevates to seal the nasopharynx.
- The larynx and hyoid bone move anteriorly and cranially.
- Epiglottis moves posteriorly and caudally to close the respiratory tract.
- Respiration pauses.
- Pharynx shortens.

*Oesophageal phase*
- Upper oesophageal sphincter relaxes.
- Bolus passes into the oesophagus.
- Oesophagus contracts sequentially (peristalsis).
- Lower oesophageal sphincter relaxes.
- Bolus enters the stomach.

## Dysphagia history

This is difficulty swallowing and is the principal symptom of oesophageal disease. When a patient complains of dysphagia you should establish:
- *Level of obstruction:* where does the patient feel the obstruction.
  - 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 may progress rapidly over a few months
  - Benign peptic stricture may give very long history of GORD with slowly progressive dysphagia.
- *Time course:* is the symptom intermittent or constant?
  - Present for only the first few swallows: lower oesophageal ring, spasm?
  - Progressive: cancer, stricture, achalasia.
- *Solids/liquids:* solids, liquids or both affected?
  - Both solids and liquids being affected equally suggests a motor cause (achalasia, spasm)
  - Solids affected more than liquids suggests 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).

### Types of dysphagia

See Box 7.1 for a list of causes.

*Oropharyngeal*

Also called 'high' dysphagia. Patients have difficulty initiating a swallow and often feel as though the cervical/neck area is the level of apparent obstruction.

Symptoms relate to both the dysphagia itself and likely underlying causes. These may include:
- Difficulty initiating swallow.
- Nasal regurgitation.
- Coughing.
- Nasal speech.
- Diminished cough reflex.
- Choking.
  - Due to laryngeal penetration and aspiration. Note that aspiration may occur without coughing in the neurologically impaired.
- Dysarthria and diplopia.
  - Due to underlying neurological diagnosis. Ask about other neurological symptoms and perform a full neurological examination (see ➲ Chapter 8).
- Halitosis.
  - Caused by large residue-containing Zenker's diverticulum. Also seen in advanced (end-stage) achalasia or long-term obstruction.

*Oesophageal*

Also called 'low' dysphagia. Patients find the site of apparent obstruction difficult to localize and may often point to their neck when the obstruction is actually within the distal oesophagus (e.g. in achalasia).

---

**Box 7.1  Causes of dysphagia**

*Oropharyngeal*
- Mechanical and obstructive: infections (e.g. retropharyngeal abscess), enlarged thyroid, lymphadenopathy, Zenker's diverticulum, reduced muscle compliance (e.g. myositis, fibrosis), malignancy, large cervical osteophytosis
- Neuromuscular: stroke, Parkinson's disease, bulbar palsy, motor neuron disease, multiple sclerosis, myasthenia gravis, muscular dystrophy
- Other: poor dentition, oral ulcers, xerostomia.

*Oesophageal*
- Mucosal disease: peptic stricture, oesophageal rings and webs (e.g. Plummer–Vinson syndrome), oesophageal tumours, chemical injury, radiation injury, infectious oesophagitis, eosinophilic oesophagitis
- Mediastinal disease: tumours, infection (e.g. TB, histoplasmosis), cardiovascular (e.g. vascular compression)
- Smooth muscle/innervation disease: achalasia (e.g. idiopathic, Chagas disease), scleroderma, post-surgical (e.g. post-fundoplication, antireflux devices, gastric banding).

### Odynophagia

This is pain on swallowing and usually reflects a severe inflammatory process involving the oesophageal mucosa or, rarely, the oesophageal musculature.

The character may range from a dull retrosternal ache to a sharp, stabbing pain with radiation through to the back. Severity can be such that patients feel unable to swallow their own saliva. Causes are shown in Box 7.2.

---

**Box 7.2 Causes of odynophagia**

- Chemical irritation
  - Acid
  - Alkali.
- Drug-induced oesophagitis
  - Antibiotics (e.g. doxycycline)
  - Potassium chloride
  - Quinidine
  - Iron sulphate
  - Zidovudine
  - NSAIDs.
- Radiation oesophagitis
- Infectious oesophagitis
- Healthy patients: *Candida albicans*, herpes simplex
- Immunocompromised patients: fungal (*Candida*, histoplasmosis), viral (herpes simplex, cytomegalovirus, HIV, EBV), *Mycobacteria* (*tuberculosis, avium-complex*), protozoan (*Cryptosporidium, Pneumocystis jiroveci*), idiopathic ulceration
- Severe ulcerative oesophagitis secondary to gastro-oesophageal reflux disease (GORD)
- Oesophageal carcinoma.

---

### Globus sensation

This is the sensation of a 'lump' or tightness in the throat and is usually not related to swallowing. Patients may describe this as a 'tightness', 'choking', or 'strangling feeling' as if something is caught in the throat.

- Present between meals.
- Swallowing solids or large liquid boluses may give temporary relief.
- Exacerbated by emotional stress.
- 🛈 Dysphagia and odynophagia are not present.

The cause of globus sensation is often unclear and may be a combination of physiological and psychological factors. Anxiety, panic disorder, depression, and somatization are often present. Physiological tests of oesophageal motility are often normal.

A combination of biological factors, hypochondriacal traits, and learned fear following an episode of choking can increase the symptom.

### 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. See also Box 7.3.

*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 reduce the GOJ sphincter pressure.

🛑 Hiatus hernia is another important cause of reflux symptoms—be sure to enquire about this in the history.

---

**Box 7.3  Causes of heartburn**

*Decreased lower oesophageal sphincter pressure*
- Foods
  - Fats, sugars, chocolate, onions, coffee, alcohol.
- Cigarette smoking
- Medication
  - Calcium channel blockers, nitrates, diazepam, theophylline, progesterone, anti-cholinergics.

*Direct mucosal irritation*
- Foods
  - Citrus fruits, tomato-based foods, spicy foods, coffee.
- Medication
  - Aspirin, NSAIDs, tetracycline, quinidine, potassium chloride, iron.

*Increased intra-abdominal pressure*
- Bending over
- Lifting
- Straining at stool
- Exercise.

*Other*
- Lying supine
- Lying on the right
- Red wine
- High emotion.

---

* The salivary glands can produce 10ml of saliva/minute. The 'oesophageo-salivary response'.

# Nausea and vomiting

### Definitions

*Nausea*
- A feeling of sickness—the inclination to vomit.
- Usually occurs in waves.
- May be associated with retching or heaving.
- Can last from seconds to days depending on the cause.

*Vomiting (emesis)*
- The forceful expulsion of the gastric contents by reflex contractions of the thoracic and abdominal muscles.
- Usually follows nausea and autonomic symptoms such as salivation.

### Onset

Over what time period have the symptoms developed?
- *Acute:* cholecystitis, gastroenteritis, recreational drug use, pancreatitis.
- *Chronic:* metabolic disorders, gastroparesis, gastro-oesophageal reflux, pregnancy, medications.

### Timing

You should be clear on exactly when the vomiting tends to occur—particularly its relation to meals.
- *Before breakfast:* alcohol, raised intracranial pressure, pregnancy, uraemia.
- *During or immediately after eating:* psychiatric causes (also peptic ulcer disease, pyloric stenosis).
- *1–4 hours after a meal:* gastric outlet obstruction, gastroparesis.
- *Continuous:* conversion disorder, depression.
- *Irregular:* major depression.

### 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.
- *Undigested food:* achalasia, oesophageal disorders (e.g. diverticulum, strictures).
- *Partially digested food:* gastric outlet obstruction, gastroparesis.
- *Bile:* proximal small bowel obstruction.
- *Faeculent/malodorous:* fistula, obstruction.
- *Large volume:* >1.5L in 24hrs, more likely physical than psychiatric.

### Associated symptoms and their causes

- *Malignancy:* weight loss.
- *Viral:* diarrhoea, myalgia, malaise, headache.
- *Central neurologic:* headache, neck stiffness, vertigo, focal neurological signs/symptoms.
- *Gastroparesis:* early satiety, post-prandial bloating, abdominal discomfort.
- *Cyclical vomiting syndrome:* repetitive migraine headaches, symptoms of irritable bowel syndrome.

### Vomiting blood (haematemesis)

Presence of blood indicates bleeding in the upper gastrointestinal tract (oesophagus, stomach, duodenum). See Box 7.4. Ask about:

- The amount of blood and exact nature of it:
  - *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 may suggest a gastric or oesophageal source such as 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:* looking like small brown granules, this is the term used for blood that has been 'altered' by exposure to stomach acid, implying that the bleeding has ceased or is relatively modest.
- Previous bleeding episodes, treatment, and outcome (e.g. previous surgery?).
- Cigarette smoking.
- Use of drugs such as aspirin, clopidogrel, NSAIDs, and warfarin.
- 🚨 Remember to ask about weight loss, dysphagia, abdominal pain, and melaena (consider the possibility of neoplastic disease).

---

**Box 7.4 Causes of upper GI bleeding**

- Peptic ulceration
- Oesophagitis
- Gastritis/erosions
- Erosive duodenitis
- Varices
- Portal hypertensive gastropathy
- Malignancy
- Mallory–Weiss tear
- Vascular anomalies (e.g. angiodysplasia, AV malformation)
- Connective tissue disorders (e.g. Ehlers–Danlos syndrome)
- Vasculitis
- Bleeding diathesis.

---

### Vomiting bile

Assess the presence or absence of bile. Remember that bile comes largely in two 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).

---

[*] This is the answer to the age-old question 'why are there always carrots in vomit?' The orange globules are, in fact, dyed with bilirubin. We suggest saving that fact for your next dinner party.

# Abdominal pain

As with any pain, work through the 'SOCRATES' questions (see ⮞ Chapter 2) to establish the site, onset, character, radiation, associated symptoms, timing, exacerbating/relieving factors, and severity.

## Site

Pain from most abdominal organs cannot be felt directly—the sensation is 'referred' to areas of the abdominal wall according to the organ's embryological origin (see Box 7.5 and Fig. 7.3). See Box 7.6 for some characteristic pains.

- Ask the patient to point to the area affected.
  - Patients 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.

---

**Box 7.5   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, gallbladder, bile duct, ureter).
- *Burning:* may indicate an acid cause and is related to the stomach, duodenum, or lower end of the oesophagus.

## Exacerbating/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.
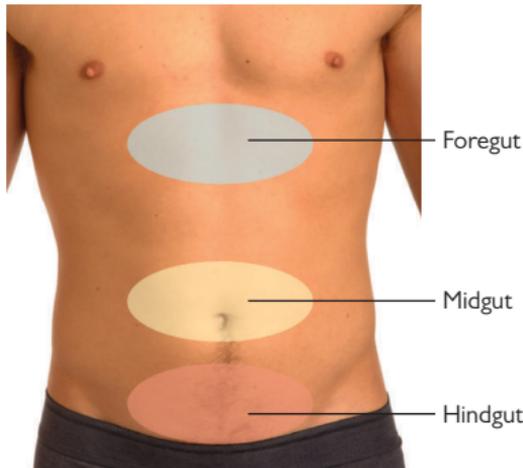
**Fig. 7.3** Typical sites of pain according to embryologic origin.

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**Box 7.6  Some characteristic pains**

- *Renal colic:* colicky pain at the renal angles and loins, which are tender to touch, radiating to the groins/testicles/labia. Typically, the patient writhes, 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
- *Small bowel obstruction:* colicky central pain associated with vomiting, abdominal distension, and/or constipation
- *Colonic pain:* as 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 three times/day to once every three days.

'Constipation' is the passage of stool <3 times/week, or stools that are hard or difficult to pass. Some causes are shown in Box 7.7.

A thorough history should include:
- Duration of constipation
- Frequency of bowel action
- 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)
  • Codeine, antidepressants, aluminium, and calcium antacids.
- Metabolic or endocrine diseases
  • Thyroid disorders, hypercalcaemia, diabetes, phaechromocytoma.
- Neurological problems
  • Autonomic neuropathy, spinal cord injury, multiple sclerosis, Hirschsprung's disease.

## 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. Some causes are shown in Boxes 7.8 and 7.9.

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 7.7 Some causes of constipation**
- Low-fibre diet
- Physical immobility (e.g. stroke, Parkinson's disease)
- Functional bowel disease (constipation-predominant irritable bowel syndrome)
- Drugs (e.g. opiates, iron, 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 7.8 Some causes of diarrhoea**
- *Malabsorption:* may cause steatorrhoea, a fatty, pale stool which is extremely odorous and difficult to flush
- *Increased intestinal motility:* hyperthyroidism, irritable bowel syndrome
- *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 (vasoactive intestinal polypeptide)-secreting tumour
- *Other:* drugs (especially antibiotics), laxative abuse, constipation and faecal impaction (overflow), small bowel or right colonic resection.

**Box 7.9 Fat malabsorption (steatorrhoea)**
A common feature of pancreatic insufficiency. Also caused by 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).

## Rectal bleeding and melaena

There are many causes of PR blood loss but, as always, a detailed history will help. See Box 7.10 for some causes. In taking the history, 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 stool 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.

---

**Box 7.10  Some causes of lower GI bleeding**
- Haemorrhoids
- Anal fissure
- Diverticular disease
- Colorectal carcinoma
- Colorectal polyp
- Angiodysplasia
- Inflammatory bowel disease
- Ischaemic colitis
- Meckel's diverticulum
- Small bowel disease (e.g. tumour, diverticula, intussusception, Crohn's)
- Solitary rectal ulcer
- Haemobilia (bleeding into the biliary tree).

---

## Mucus

Clear, viscoid secretion of the mucous membranes. Mucus contains 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 colonic bacterial fermentation of poorly absorbed carbohydrates.

Excessive flatus is a particular feature of:
- Hiatus hernia.
- Peptic ulceration.
- Chronic gallbladder disease.
- Air-swallowing (aerophagy).
- High-fibre diet.
- Lactase deficiency.
- Intestinal malabsorption.

### Tenesmus

This is the feeling of the need to open the bowels with little or no stool actually passed. The sensation may be constant or intermittent and is usually accompanied by pain, cramping, and involuntary straining.

Causes include:
- Inflammatory bowel disease.
- Anorectal abscess.
- Infective colitis.
- Colorectal tumours/polyps.
- Radiation proctitis.
- Irritable bowel syndrome.
- Thrombosed haemorrhoids.

# Generalized abdominal swelling

The five classic causes of abdominal swelling ('the 5 Fs'):
- Fat.
- Fluid (see also Box 7.11).
- Flatus.
- Faeces.
- Fetus.

To these, you should also add 'tumour' and another 'F': Functional (irritable bowel syndrome).

---

**Box 7.11 Ascites in decompensated liver disease**
- 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.

# Jaundice and pruritus

### Jaundice

Jaundice ('icterus') is a yellow pigmentation of skin, sclera, and mucosae caused by excess bilirubin in the tissue. See Box 7.12 for some causes and Box 7.13 for other causes of skin yellowing.

Jaundice results from interference in the normal metabolism of bilirubin (including uptake, transport, conjugation, and excretion). Ask about:
- The colour of the urine (dark in cholestatic jaundice due to renal excretion of conjugated bilirubin).
- The colour and consistency of the stools (pale in cholestatic jaundice).
- Abdominal pain (e.g. caused by gallstones).

Ask especially about:
- Previous blood transfusions.
- Past history of jaundice, hepatitis, pancreatitis, or biliary surgery.
- Drugs (e.g. antibiotics, NSAIDs, oral contraceptives, phenothiazines, herbal remedies, anabolic steroids). See also Box 7.14.
- Intravenous drug use.
- Tattoos and body piercing.
- Foreign travel and immunizations.
- Sexual history.
- FHx of liver disease.
- Alcohol consumption.
- Any personal contacts who also have jaundice.
- Occupational exposure to hepatotoxins.

---

#### Box 7.12  Causes of jaundice

*Pre-hepatic (unconjugated hyperbilirubinaemia)*
- Overproduction: haemolysis; ineffective erythropoiesis
- Impaired hepatic uptake: drugs (contrast agents, rifampicin), congestive cardiac failure
- Impaired conjugation: Gilbert's syndrome, Crigler–Najjar syndrome.

*Hepatic (conjugated hyperbilirubinaemia)*
- Infection: viral hepatitis, CMV, liver abscess, septicaemia
- Alcohol and toxins: carbon tetrachloride, fungi (*Amanita phalloides*)
- Drug-induced hepatitis: paracetamol, anti-tuberculosis drugs (isoniazid, rifampicin, pyrazinamide), statins, sodium valproate, halothane
- Metabolic: haemochromatosis, $\alpha_1$-antitrypsin deficiency, Wilson's disease, Rotor syndrome
- Vascular: Budd–Chiari, right-sided heart failure.

*Post-hepatic (conjugated hyperbilirubinaemia)*
- Luminal: gallstones
- Mural: cholangiocarcinoma, sclerosing cholangitis, primary biliary cirrhosis, choledochal cyst
- Extra-mural: pancreatic cancer, lymph nodes at porta hepatis
- Drugs: antibiotics (flucloxacillin, fusidic acid, co-amoxiclav, nitrofurantoin), steroids, sulphonylureas, chlorpromazine, prochlorperazine.

### Pruritus

This is itching of the skin and may be either localized or generalized. The mechanism is not fully understood but is likely due to increased bile acid levels secondary to cholestasis.

It has many causes—it is particularly associated with cholestatic liver disease (e.g. primary biliary cirrhosis, sclerosing cholangitis).

---

**Box 7.13  Some other causes of yellowing of the skin**

- Carotenodermia: excess ingestion of carotene (orange vegetables such as carrots, squash)
  - The pigment is concentrated on the palm, soles, forehead, and nasolabial folds but spares the sclerae.
- Lycopenodermia: excessive ingestion of lycopene-containing red foodstuffs (e.g. tomato)
- Quinacrine
- Excessive exposure to phenols.

---

**Box 7.14  Drug history in liver disease**

Think about drugs that can precipitate hepatic 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.

# 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. Be aware of triggers for concern (Box 7.15) and consider using the 'MUST' score (Box 7.16) for those at risk of malnutrition.

### Important notes on appetite and weight

- ▶ 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'.
- Ascites weighs 1kg/L and some patients with liver failure may have 10–20L of ascites, masking any 'dry weight' loss.
- The combination of weight loss with increased appetite may suggest malabsorption or a hypermetabolic state (e.g. thyrotoxicosis).
- In every case, you should calculate the patient's BMI (see ➲ Chapter 3).

---

**Box 7.15   Triggers for concern**

Closer nutritional assessment and follow-up should be considered if:
- Poor intake for longer than 1–2 weeks
- Weight loss of >10%.

---

### Questions to ask

- Ask the patient about their eating habit and average daily diet.
- 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 including:
  - Abdominal pain
  - Nausea
  - Vomiting
  - Fever
  - Menstrual irregularities
  - Low mood.

Ask also about:
- The colour and consistency of stools (e.g. steatorrhoea?).
- Urinary symptoms.
- Recent change in temperature tolerance.

**Box 7.16 The 'MUST' score**

The 'Malnutrition Universal Screening Tool' has been designed to help identify adults who are underweight and at risk of malnutrition, as well as those who are obese. This score was developed by the Malnutrition Advisory Group (MAG), a committee of the British Association for Parenteral and Enteral Nutrition (BAPEN).

Further information is available at ℘ http://www.bapen.org.uk

The five 'MUST' steps

1 Calculate body mass index (BMI) from weight and height.

| $Kg/m^2$ | Score |
|---|---|
| BMI >20 | = 0 (>30 = obese) |
| 18.5 – 20 | = 1 |
| <18.5 | = 2 |

2 Determine unplanned weight loss (%) in past 3–6 months.

| % | Score |
|---|---|
| <5 | = 0 |
| 5–10 | = 1 |
| >10 | = 2 |

3 Consider the effect of acute disease.

If patient is acutely ill and there has been or is likely to have been no nutritional intake for >5 days, score 2

4 Add scores from 1, 2 & 3 together to give overall risk of malnutrition.

| Total score = 0 | - low risk |
|---|---|
| = 1 | - medium risk |
| = 2 or more | - high risk |

5 Initiate appropriate nutritional management.

Using local management guidelines, prepare appropriate care plan.

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# Lower urinary tract symptoms

See also Box 7.17 for other points in the urinary history.

### Urinary frequency

This is the passing of urine more often than is normal for the patient.
- How many times in a day? How much urine is passed each time?
  - Is patient 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 five 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 increase 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 detrusor 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 caused by 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 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 detrusor dysfunction or prostatic disease.

### Intermittency

The disruption of urine flow in a stop–start manner. Causes include prostatic hypertrophy, bladder stones, and ureterocoeles.

### Oliguria

Oliguria is scanty or low-volume urination and is defined as the excretion of <400ml urine in 24 hours. Causes can be physiological (dehydration) or pathological (intrinsic renal disease, shock, or obstruction).

### Anuria

Anuria is the absence of urine formation and you should attempt to rule out urinary tract obstruction as a matter of 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.

---

**Box 7.17 Other points in the urinary history**
- Loin pain (urinary calculi, urinary retention, pyelonephritis, renal tumours)
- Back pain (e.g. bony metastases from prostate cancer)
- Systemic symptoms of acute kidney injury or chronic renal failure (anorexia, vomiting, fatigue, pruritus, peripheral oedema)
- Past medical history: neurologic diseases causing bladder dysfunction, previous abdominal or pelvic surgery causing bladder denervation
- Drug history (nephrotoxins)
- Family history of renal failure or polycystic kidney disease
- Occupational history (industrial carcinogens causing bladder cancer)
- Foreign travel (exposure to schistosomiasis).

# The rest of the history

As well as points from the history described elsewhere in this chapter under the individual symptoms, you should make note of the following in a full and thorough 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 haemochromatosis).

## Drug history

See notes on the drug history described previously under each presenting symptom.

## Smoking

Smokers are at increased risk of peptic ulceration, oesophageal cancer, and 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 ➲ Chapter 2.

## 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.

# 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 in Box 7.18. 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 7.18  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
Look 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.

# Inspection: hands

Take the patient's right hand in yours and examine carefully for the signs described here.

## Nails

See also ⟩ Chapter 4.

- *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 hypo-albuminaemic states including severe liver cirrhosis.
- *Digital clubbing:* Abdominal causes are cirrhosis, inflammatory bowel disease, and coeliac disease.
- *Blue lunulae:* a bluish discoloration of the normal lunulae seen in Wilson's disease.

## Palms

- *Palmar erythema:* 'liver palms'. A blotchy reddening of the palms of the hands, especially affecting the thenar and hypothenar eminences.
  - 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 4th and 5th metacarpals.
  - Can progress to a fixed flexion deformity of the fingers starting at the 5th and working across to the 3rd or 2nd
  - 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.

# Inspection: upper limbs

### 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 blanch with pressure. Possibly a sign of thrombocytopenia.
- *Muscle wasting:* seen as a decrease 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.
- *Iatrogenic features:* be careful not to miss arteriovenous (AV) fistulae or haemodialysis catheters.

### Hepatic flap (asterixis)

- Characteristic of encephalopathy due to liver failure. This is identical to the flap seen in hypercapnic states (see Box 7.19).
- Ask the patient to stretch out their hands in front of them with the hands dorsiflexed at the wrists and fingers outstretched and separated.
  - 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.

Hepatic encephalopathy in a patient with previously compensated liver disease 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 axillae

Examine carefully for lymphadenopathy and acanthosis nigricans (a thickened blackening of the skin, velvety in appearance. May be associated with intra-abdominal malignancy).

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**Box 7.19  Other causes of asterixis**

- Uraemia
- Azotaemia
- $CO_2$ toxicity
- Electrolyte abnormalities (hypoglycaemia, hypokalaemia, hypomagnesaemia)
- Drug intoxication (barbiturates, phenytoin, alcoholism).

# Inspection: face

### Eyes

Ask the patient to look ahead whilst you look at their eyes and orbits. Ask the patient to look up whilst you gently retract the lower lid with a finger, looking at the sclera and conjunctiva. Look especially for:

- *Jaundice:* a yellow discoloration of the sclera. Usually the first place that jaundice can be seen. Particularly useful in a patient with dark skin tones in whom jaundice would not be otherwise obvious.
- *Anaemia:* pallor of the conjunctivae.
- *Kayser–Fleischer 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—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 B12, and iron deficiencies.
- *Circumoral pigmentation:* hyperpigmented areas surrounding the mouth. Seen in Peutz–Jeghers 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.
- *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 B12, and folate deficiencies
  - *Macroglossia:* enlarged tongue. Causes include amyloidosis, hypothroidism, acromegaly, Down's syndrome, and neoplasia
  - *Leukoplakia:* a white thickening of the tongue and mucous 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 B2 (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.

# Inspection: neck and chest

## The neck

Examine the cervical and supraclavicular lymph nodes as in ➲ Chapter 3.

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 (Fig. 7.4)
  - 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. 7.4** Distribution of drainage to the superior vena cave and the area to look for spider naevi. The normal adult may have up to five such lesions.

# Inspection: abdomen

### Abdominal distension

Does the abdomen look swollen? Consider the 5 Fs and note the state of the umbilicus. (Everted? Deep?)

### Focal swellings

Treat an abdominal swelling as you would do any other lump 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 a 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 it in a 'milking' action
  - Release the pressure from one finger and watch for flow of blood
  - 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).

### 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.

### Skin discoloration

There are 2 classical patterns of bruising/discoloration indicating the presence of retroperitoneal blood (seen especially in pancreatitis):
- *Cullen's sign:* discoloration at the umbilicus and surrounding skin.
- *Grey Turner's sign:* discoloration at the flanks.

# Stomas

### Inspection

Use the following system to describe or identify a stoma.

- Site?
- Bag covering?
- Appearance:
  - Healthy mucous lining? What colour?
  - Spouted or flush to skin?
  - One orifice (end) or two (loop)?
- Content (e.g. urine, formed stool, semi-formed or liquid stool).
- Any other abdominal scars?
- Any drains or healed stoma sites?
- Look for evidence of complications:
  - Early: necrosis (black/brown discoloration), infection
  - Late: parastomal hernia, prolapse, stenosis, retraction, obstruction, skin erosions, bleeding.

### Common types of stoma

- *Ileostomy:* may be 'loop' or 'end'.
  - End: total/partial colectomy (e.g. inflammatory bowel disease, familial adenomatous polyposis, total colonic Hirschsprung's disease)
  - Loop: to protect distal anastomosis (e.g. partial colectomy, formation or ileorectal pouch).
- *Colostomy:* may be 'loop' or 'end'.
  - End: Hartmann's procedure, abdominoperitoneal resection
  - Loop: rectal trauma, colovaginal or perianal fistula.
- *Urostomy/ileal conduit:* one or both ureters are diverted to a short length of ileum which is disconnected and brought to the skin (usually follows radical lower urinary tract surgery).
  - Spouted, prominent mucosal folds, dark pink/red, right-sided
  - 🟡 Indistinguishable from ileostomy unless the output is seen.
- *Gastrostomy/duodenostomy/jejunostomy:* surgically or endoscopically created connection between the stomach/duodenum/jejunum and the anterior abdominal wall. For feeding and/or drainage (stomach).
  - Narrow calibre, flush to skin with little visible mucosa, usually at left upper quadrant. Fitted with indwelling tubes or access devices.

### Other stomas

- *Caecostomy or typhlostomy:* loop stoma in the caecum.
- *Appendicostomy or caecostomy tube:* used in paediatrics to allow administration of proximal enemas.
- *Cholecystostomy:* communication between the gallbladder and anterior abdominal wall (drainage of gallbladder contents).
- *Nephrostomy:* tube that exits through flank region and drains urine from the renal pelvis.

# Abdominal scars 1: open surgery

See Fig. 7.5 for common surgical scars.

## Upper- and lower-midline laparotomy

A standard elective and emergency incision.
- *Upper:* gastric, splenic surgery.
- *Lower:* gynaecological surgery (e.g. hysterectomy, oophorectomy), urological surgery (e.g. cystectomy, prostatectomy) or colorectal surgery (e.g. sigmoidectomy, anterior resection).
- *Full laparotomy:* performed in emergencies and for surgery that may involve both regions (e.g. right or left hemicolectomy).

## Roof-top or Mercedes-Benz incision

- Classically used for partial hepatectomies, liver transplants, or pancreatic surgery, e.g. Whipple's procedure or necrosectomy.

## Kocher incision

- Classical incision for an open cholecystectomy.

## Paramedian incision

- Now rarely used; previously used for colorectal surgery.
- Can take longer to perform and risks greater blood loss than midline laparotomy.
- Point at which the internal oblique aponeurosis splits round the rectus abdominis.

## Inguinal incision

- Inguinal hernia repair and orchidectomies.
- May be left- or right-sided.

## Gridiron and Lanz incisions

- Used for appendectomy.
- The Lanz is more transverse, extends more medially and is closer to the anterior superior iliac spine.

## Femoral incision

- Used to access the femoral triangle, especially the femoral artery.
  - If bilateral, often due to femoral–femoral bypass or endovascular aneurysm repair
  - If unilateral, look for further scars around the knee (for femoral–popliteal or femoral–distal bypass).

## Pfannenstiel incision

- Classically used for C-section or abdominal hysterectomy.

## Hockey-stick incision

- Classically used for renal transplants.
- The scar may not include the (vertical) laparotomy component depending on the surgeon.

## Nephrectomy incision

- Used for partial or complete nephrectomy.
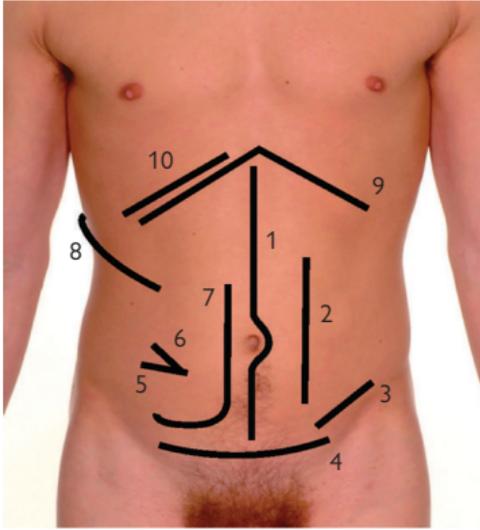
**Fig. 7.5** Common surgical scars. 1. Midline laparotomy. 2. Paramedian incision. 3. Left inguinal incision. 4. Pfannenstiel incision. 5. Lanz incision. 6. Gridiron incision. 7. Hockey-stick incision. 8. Nephrectomy incision. 9. Roof-top or, with additional superior incision, 'Mercedes-Benz' incision. 10. Kocher incision.

# Abdominal scars 2: laparoscopic

Laparoscopic or 'key-hole' surgery uses several small incisions for the insertion of the instruments. There will be a site for the camera insertion and at least 2 other 'ports' for the graspers.

The port site scars are often arranged in a loose semicircle around the internal operation site. See also Box 7.20.

If you work out where the camera and graspers are pointing, you can guess the possible operations based on your anatomy knowledge. See Fig. 7.6.

### Laparoscopic cholecystectomy
- This is now the most common method of removing the gallbladder. Complications can include bile leaks, retained stones in the common bile duct (CBD), damage to the CBD, and bleeding.

### Laparoscopic right hemicolectomy
- Left-sided port site placed at lateral margin of rectus abdominis.
- The RUQ port site is expanded to allow removal of the colon. The anastomosis may be performed through any port.

### Laparoscopic left hemicolectomy, sigmoidectomy, or anterior resection
- Right-sided port site placed at lateral margin of rectus abdominis.
- The LIF port site is expanded to allow removal of the colon and insertion of end-to-end circular stapler to join the rectum and cut end of bowel together again.

### Laparoscopic inguinal hernia repair
- Both the left and right inguinal canals are usually accessed through the same midline port sites.

### Laparoscopic appendicectomy
- The figure demonstrates one possible placement.
- Another common port placement is moving the LIF port to the RIF at the level of the umbilicus to allow traction of the appendix.

### Laparoscopic nephrectomy
- One of the port sites is expanded to allow removal of the organ.

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**Box 7.20  More on laparoscopic technique**
- In general an open technique or Hassan technique is used
  - A small infra-umbilical incision is made and the linea alba incised with direct visualization of the peritoneum. A finger is then inserted prior to the trocar to sweep away any adhesions
  - A pneumoperitoneum is maintained between 12–15mmHg.
- *Risks:* damage to other organs, diathermy burns to areas away from the operation site (perforations or bile leaks), converting to open surgery.
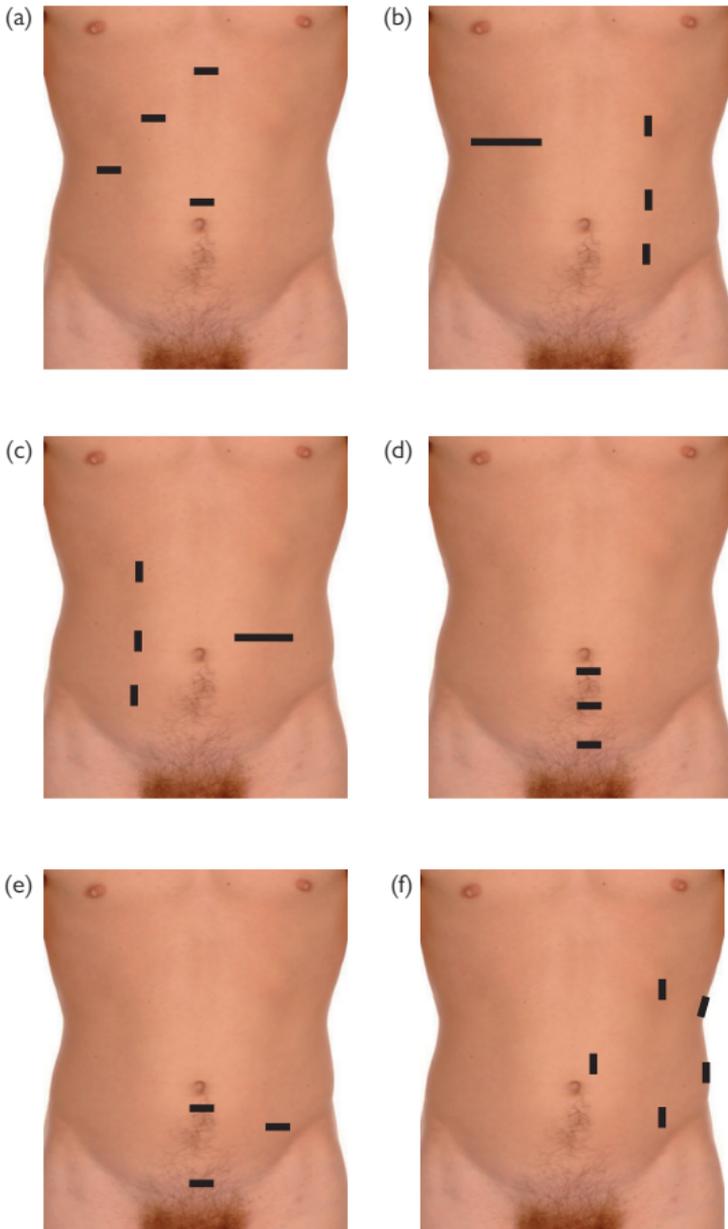
**Fig. 7.6** Common laparoscopic surgical scars. (a) cholecystectomy, (b) right hemicolectomy, (c) left hemicolectomy, sigmoidectomy, or anterior resection, (d) inguinal hernia repair, (e) appendicectomy, (f) nephrectomy.

# Palpation: general

### 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 four quadrants should be examined in turn with light, and then deep palpation before focusing on specific organs. 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 four 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 (see Boxes 7.21 and 7.22).
- It is often possible to detect the putty-like consistency of stool in the sigmoid colon.

---

**Box 7.21  Which layer is the mass in?**

- *Epidermal or dermal:* moves with the skin
- *Subcutis:* skin moves over the mass
- *Muscle/tendon:* moves with muscular contraction
- Ask the patient to raise their head and shoulders off the bed
  - Intra-abdominal: more difficult to palpate when abdominal muscles are tensed.
- *Bone:* immobile.

---

**Box 7.22 Characteristics of abdominal masses**
- *Consistency:* 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 opposite side may protrude
- *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.
- *Translucency:* darken the room and press a lit pen-torch to one side of the lump. It will 'glow' in the presence of water, serum, fat, or lymph
  - Solid lumps will not transilluminate.
- *Resonance:* only possible to test on large lumps. Percuss and listen (and feel) if the lump is hollow (gas-filled) or solid
- *Pulsatility:* consider carefully if the pulse is transmitted from an underlying structure or if the lump itself is pulsating
- *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 the same as 'reducibility'.
- *Reducibility:* A feature of herniae. Attempt to reduce the lump by maneouvring its contents back into the abdominal cavity. Ask the patient to cough and watch for the lump reforming.

# Palpation: aorta

The abdominal aorta may be palpated in the midline above the umbilicus, particularly in thin people. If felt:
- Place each hand either side of the outermost palpable margins.
- Measure the distance between your fingers. Normal = 2–3cm.
- Is it pulsatile/expansile in itself (in which case your fingers will move outwards) or is the pulsation transmitted through other tissue (in which case your fingers will move upwards)? See Fig. 7.7.
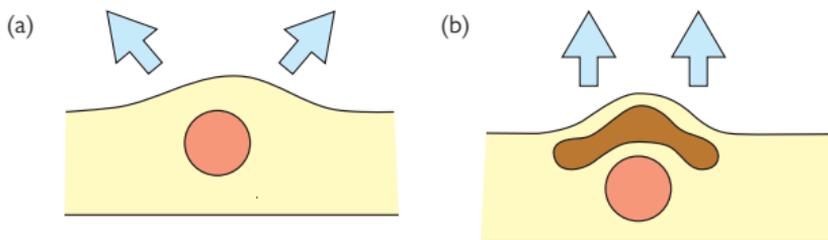


**Fig. 7.7** Palpating a pulsatile mass. If expansile (a), your fingers move outwards. If the pulsatility is being transmitted (b), your fingers will move upwards.

# Palpation: liver and gallbladder

### 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 it.

- 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 (Fig. 7.8).
- 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 ➲ p. 205).
- 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 (Fig. 7.9). Normally only palpable when enlarged due to biliary obstruction or acute cholecystitis (Box 7.23).

- 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 to lateral direction.

**Fig. 7.8** 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. 7.9** 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).

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**Box 7.23  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.

# Palpation: 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. Causes of splenomegaly, hepatosplenomegaly and hepatomegaly are outlined in Boxes 7.24, 7.25, and 7.26.

## Technique

- Palpated using a similar technique to that used to examine the liver (see Fig. 7.10).
- 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.
- 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.



**Fig. 7.10** 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 7.24 Causes of splenomegaly**

- *Infection:* EBV, CMV, HIV, viral hepatitis, any cause of septicaemia, subacute bacterial endocarditis, typhoid, brucellosis, tuberculosis, leptospirosis, histoplasmosis, malaria, leishmaniasis, trypanosomiasis
- *Haematological:* myeloid and lymphatic leukaemia, lymphoma, spherocytosis, thalassaemia, sickle cell (splenic infarcts may cause a small spleen in late disease), autoimmune haemolytic anaemia, idiopathic thrombocytopenic purpura
- *Infiltration:* glycogen storage diseases, Gaucher's disease
- *Congestive:* hepatic cirrhosis, congestive heart failure, portal vein thrombosis, splenic vein thrombosis, Budd–Chiari syndrome
- *Other:* amyloidosis, cysts, hamartomas, connective tissue disorders (e.g. RA, SLE, sarcoidosis).

**Box 7.25 Causes of hepatosplenomegaly**

- *Hepatic:* Chronic liver disease with portal hypertension (if cirrhotic, liver may be impalpable)
- *Infection:* EBV, CMV, viral hepatitis, infective endocarditis
- *Infiltration:* amyloidosis, Gaucher's disease
- *Haematological:* lymphoma, leukaemia, pernicious anaemia, myeloproliferative disease
- *Endocrine:* acromegaly, thyrotoxicosis
- *Granulomatous conditions:* tuberculosis, sarcoidosis, Wegener's granulomatosis
- *Other causes:* malaria, kala-azar, schistosomiasis.

**Box 7.26 Causes of hepatomegaly**

- Cirrhosis
- Congestive cardiac failure
- Neoplastic: secondary and primary (e.g. hepatoma)
- Infective: acute viral hepatitis, liver abscess, hydatid cyst
- Polycystic disease
- Tricuspid regurgitation (pulsatile hepatomegaly)
- Budd–Chiari syndrome
- Haemochromatosis
- Infiltrative: amyloidosis, sarcoidosis.

# Palpation: kidneys and bladder

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. Take care not to mistake splenomegaly for an enlarged kidney (see Table 7.1).

### Technique

- 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 (Fig. 7.11).
- 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 (Fig. 7.12).

### Findings

- Unilateral palpable kidney: hydronephrosis, polycystic kidney disease, renal cell carcinoma, acute renal vein thrombosis, renal abscess, acute pyelonephritis (see 'important presentations').
- Bilateral palpable kidneys: bilateral hydronephrosis, bilateral renal cell carcinoma, polycystic kidney disease, nephrotic syndrome, amyloidosis, lymphoma, acromegaly.

Table 7.1 Differentiating splenomegaly from 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 inferomedially 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 |

**Fig. 7.11** Palpation of the right kidney.



**Fig. 7.12** Palpation of the left kidney.

## Bladder

The urinary bladder is not palpable when empty. As it fills, it expands superiorly and may reach as high as the umbilicus 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.

# Palpation: herniae

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 herniae are usually caused by portions of bowel protruding through weakened areas of the abdominal wall. In the abdomen, herniae usually occur at natural openings or weak spots such as surgical scars. See Box 7.27 for some rarer hernia types.

Most abdominal herniae have an expansile cough impulse—asking the patient to cough will increase the intra-abdominal pressure causing a visible or palpable impulse.

*Strangulation*: herniae that cannot be reduced (irreducible) may become fixed and swollen as their blood supply is occluded causing ischaemia and necrosis of the herniated organ.

## An approach to herniae

- Determine the characteristics as you would any lump 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.

## Hernial orifice anatomy

### Inguinal canal

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.

The internal ring is an opening in the transversalis fascia lying at the mid-inguinal 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.

- 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 herniae). More likely to strangulate than direct inguinal herniae.

### Femoral canal

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 herniae 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.

## Examining an inguinal hernia

*Inspection*

- Look for visible swelling at both groins.
- Ask the patient to cough and watch for swellings.
  - Minor bulging of the inguinal canal is normal (Malgaigne's bulges).
- Look for scars in relation to previous open repair (groin) and laparoscopic repair (around umbilicus).

*Palpation*

- Ask the patient if there is any pain.
- Examine the scrotum and its content.
- Examine any lump carefully.
- Ask patient to cough and feel for an expansile cough impulse.
- Ask if the lump is normally reducible.
  - Lie patient down on couch to allow gravity to help reduction. Try to reduce hernia or ask patient to reduce it for you.

*Relate the swelling to the bony landmarks*

- Place a finger on the pubic tubercle and ask patient to cough.
  - Inguinal herniae will be superior and medial to your finger
  - Femoral herniae will be inferior and lateral to your finger.
- Place 2 fingers at the midpoint between anterior superior iliac spine and pubic tubercle (the internal ring). Ask patient to cough.
  - This will prevent an indirect inguinal hernia appearing, whereas a direct inguinal hernia will appear.

*Auscultation*

- Listen over any swelling for bowel sounds.

## Examining a femoral hernia

*Inspection*

- Lump in the groin.
  - Below and lateral to the pubic tubercle
  - Medial to the femoral artery (palpate for the pulsation).
- Flattening/obliteration of the inguinal skin crease.
- Overlying skin appears normal in colour/texture.

*Palpation*

- Firm smooth, spherical lump.
  - Tender if strangulated.
- Non-reducible.
- Often no cough impulse.

*Percussion and auscultation*

- A resonant percussion note and active bowel sounds imply strangulated bowel.
  - Femoral herniae commonly contain greater omentum.

## Incisional hernia

- Be sure to examine the patient standing and lying supine. Incisional herniae account for 10–15% of all herniae.
- Peak incidence is at 5 years following surgery.

*Inspection*

- A lump arising from the site of a previous incision.

*Palpation*

- A positive cough impulse is present.
- Note the size, site, shape, and constituents.
- Assess whether the hernia is reducible, if so palpate the edges to quantify the defect's size.
  - If irreducible, the lump may be an incarcerated or strangulated hernia.

*Auscultation*

- Active bowel sounds point to bowel in the hernial sac.

*Completion*

- A full abdominal examination should be conducted, looking for signs of deep infection and for a cause of raised intra-abdominal pressure.

## Paraumbilical hernia

- A hernia in the linea alba, just superior or inferior to the umbilicus
- Male:female ratio = 1:5.
- Risk factors include obesity, multiparity, and advancing age.

*Inspection*

- Bulge beside the umbilicus.
- The umbilicus is often distorted into a crescent shape.

*Palpation*

- The lump is separate from the umbilicus and should be reducible.
  - ► Examine the patient standing and make careful note of the size, site, shape, and constituents of the lump.
- A positive cough impulse may be present.
  - Absence of a cough impulse does not exclude a hernia.
- ► Now examine the patient lying supine.
- Assess if the hernia is reducible; if so, palpate the edges to quantify the defect's size.

*Percussion*

- Resonance to percussion implies bowel is present in the hernia sac.

*Auscultation*

- Active bowel sounds point to bowel in the hernia sac.

*Completion*

- A full abdominal examination should be conducted, looking for causes of raised intra-abdominal pressure.

### Spigelian hernia

- Also known as semi-lunar line herniae.
- Occurs through bands of the internal oblique muscle as it enters the semilunar line (sometimes called Spigel's line). Protrudes along the lateral border of the rectus sheath.
- Most occur below the umbilicus, adjacent to the line of Douglas.

*Inspection*

- A swelling at the lateral border of the rectus abdominis muscle.
  - Difficult to assess with the patient supine. Ask them to stand
  - May need patient to direct you to lump especially in the obese.

*Palpation*

- Positive cough impulse.
- Overlying skin is normal.
- May or may not be reducible.

*Percussion and auscultation*

- A resonant percussion note and active bowel sounds suggest herniated bowel.
  - If the hernia contains greater omentum only, there will be no positive findings on auscultation.

*Further examination*

- The whole abdomen should be examined, looking for potential causes of raised intra-abdominal pressure and abdominal masses.
- If the hernia contains strangulated bowel, there may be abdominal distension.

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**Box 7.27  Some other types of hernia**

- *Maydl's hernia:* two adjacent loops of bowel in the sac; the portion in the abdomen is at risk if strangulation occurs
- *Sliding (en-glissade) hernia:* the sac is partially formed by retroperitoneal tissue
- *Richter's hernia:* a knuckle of bowel wall is strangulated
- *Littre's hernia:* a protrusion of a diverticulum into a hernia sac (50% inguinal, 20% femoral). Classically contains Meckel's diverticulum
- *Amyand's hernia:* the appendix enters the hernial sac and can become occluded leading to appendicitis
- *De Garengeot's hernia:* a rare subtype in which the appendix is incarcerated within a femoral hernia.

# Percussion

In the examination of the abdomen, percussion is useful for:
- Determining the size and nature of enlarged organs or masses.
- Detecting shifting dullness.
- Eliciting rebound tenderness.

Organs or masses will appear as dullness whereas a bowel full of gas will seem abnormally resonant. Good technique comes with experience. Practise percussing out your own liver.

## 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 to laterally with the fingers spread and positioned longitudinally.
- Listen (and feel) for a definite change to a dull note.

There are then 2 specific tests to perform:

*Shifting dullness**
- Percuss centrally to 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.
- 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 (Fig. 7.13).
- 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.

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\* This is also the punch-line to the medical student joke: 'What's the definition of a ward round?'

**Fig. 7.13** 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 should not be skipped.

### 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 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 secondary 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.

As you proceed, explain each stage to the patient.

## 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 mucosa 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 two 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.

- *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.

### Procedure tips

- 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.

# Important presentations

## Hepatomegaly

Once hepatomegaly is found, examine for splenomegaly and evidence of other features which may help determine the cause. See also Boxes 7.28, 7.29, and 7.30.

*Examination findings*

- Pallor/anaemia.
  - Haemolysis, chronic liver disease, malignancy, marrow failure, infective endocarditis.
- Jaundice.
  - Haemolysis, chronic liver disease, hepatitis.
- Lymphadenopathy.
  - Lymphoma, metastatic disease, leukaemia, myeloproliferative disorders, connective tissue disorders, tuberculosis, viral hepatitis, infectious mononucleosis.
- Cachexia.
  - Malignancy, TB.
- Petechial rash.
  - Thrombocytopenia in cirrhosis, leukaemia.
- Herpes zoster, oral candidiasis.
  - Immunocompromised state in leukaemia, lymphoma, TB.
- Stigmata of chronic liver disease.
  - Spider naevi, palmar erythema, leukonychia, digital clubbing, gynaecomastia.
- Peripheral oedema.
  - Cirrhosis, right heart failure, hypoalbuminaemia.
- Raised JVP.
  - Right heart failure.

*Further examination: abdomen*

- Look for visible masses, prominent veins, caput medusa.
- Palpate in each quadrant for tenderness and masses.
- Palpate specifically for the kidneys, gallbladder, and presence of ascites.
- Auscultate for murmurs over the liver.

*Causes*

- Infective: EBV, CMV, hepatitis, liver abscess, malaria, leptospirosis, amoebiasis, hydatid cyst, actinomycosis.
- Neoplastic: hepatocellular carcinoma (HCC), metastasis, myeloma, leukaemia, lymphoma, haemangioma.
- Metabolic: haemochromatosis, amyloidosis, glycogen storage diseases (e.g. Hunter syndrome, Gaucher's disease, Niemann–Pick disease), fat.
- Congenital: Riedel's lobe, polycystic disease.
- Other: alcohol, right heart failure, Budd–Chiari syndrome, sarcoidosis.

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**Box 7.28  Causes of hepatomegaly by degree of enlargement**

*Mild*
- Infection: hepatitis, HIV, EBV, hydatid disease
- Other: biliary obstruction, and the causes below.

*Moderate*
- Haematological: lymphoma, myeloproliferative disorders
- Infiltration: amyloidosis
- Haemochromatosis, and the causes of massive hepatomegaly below.

*Massive*
- Malignancy: HCC, metastasis
- Haematological: myeloproliferative disorders
- Vascular: right-sided heart failure, tricuspid regurgitation
- Alcoholic liver disease and fatty infiltration.

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**Box 7.29  Causes of hepatomegaly by other examination findings**

*Irregular surface*
- Malignancy (e.g. HCC, metastasis), cirrhosis
- Hydatid cysts, amyloid, sarcoid granulomas.

*Pulsatility*
- Vascular: tricuspid regurgitation, vascular malformation e.g. AVM
- Malignancy: HCC.

*Tenderness*
- Infection: hepatitis, malaria, EBV, hepatic abscess
- Malignancy: HCC, metastasis (stretching of liver capsule)
- Vascular: right-sided heart failure, Budd–Chiari syndrome
- Biliary obstruction, ascending cholangitis.

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**Box 7.30  A word on chronic liver disease**
- *Cirrhosis* is a histological diagnosis that may also be suggested by non-invasive tests such as serum markers and ultrasonographic elastography
- The main prognostic tests of *function* are bilirubin, prothrombin time, creatinine, and sodium levels (as reflected in MELD and UKELD severity scores).

### Chronic liver disease

*Examination findings: general inspection*

Look for:

- Pallor (anaemia).
- Tattoos, needle track marks (may suggest viral hepatitis).
- Digital clubbing.
- Terry's nails (proximal 2/3 of nail plate white with distal 1/3 red).
- Muehrcke's lines.
- Palmar erythema.
- Spider naevi (number and size correlate with severity).
- Gynaecomastia.
- Generalized muscle wasting.
- Loss of body hair.
- Testicular atrophy.
- Evidence of alcohol misuse.
  - Dupuytren's contracture
  - Parotid enlargement
  - Cerebellar signs (past-pointing, ataxic gait).
- Signs of decompensated liver disease.
  - Jaundice, purpura, asterixis.

*Examination findings: abdominal inspection*

- Distension (ascites; a paraumbilical hernia may be visible).
- Caput medusa.
- Look also for scars, drain sites.

*Palpation, percussion, and auscultation*

- Palpate for hepatomegaly, splenomegaly.
- Percuss for liver span, spleen, shifting dullness, fluid thrill.
- Auscultate for either hepatic arterial bruit or venous hum (in portal hypertension; Cruveilhier-Baumgarten murmur).

*Aetiology*

- Toxins: alcohol and other drugs (e.g. amiodarone, methotrexate).
- Viral: hepatitis B and C, CMV, EBV.
- Metabolic: non-alcoholic steatohepatitis (NASH), haemochromatosis, Wilson's disease, $\alpha_1$-antitrypsin deficiency.
- Autoimmune: autoimmune hepatitis, primary biliary cirrhosis, primary sclerosing cholangitis.

*Clinical spectrum of alcoholic liver disease*

- Alcohol withdrawal syndrome including delirium tremens.
- Wernicke's encephalopathy.
  - Confusion, ataxia, and ophthalmoplegia
- Alcoholic fatty liver.
  - Fatty liver on ultrasound, abnormal liver enzymes on biochemistry with good synthetic function
- Alcoholic hepatitis.
  - Jaundice, raised liver enzymes, coagulopathy, encephalopathy
- Cirrhosis with portal hypertension.

### Hepatic encephalopathy
- Neuropsychiatric disorder in patients with liver dysfunction (personality changes, intellectual impairment, reduced consciousness).
- Diversion of portal blood into the systemic circulation via collaterals leads to a lack of hepatic detoxification.
- Exposure of the brain to excessive concentrations of ammonia can cause neurotoxicity.
- Severity can be graded by the West Haven classification (Box 7.31).
- 🛑 Ammonia levels don't always correlate with severity.
  - Other toxins implicated include mercaptans, short-chain fatty acids, and phenol.

*Epidemiology and prognosis*
- In decompensated cirrhosis, the risk of developing HE is 20% per year.
- At any time, 30–45% of people with cirrhosis exhibit evidence of HE.
- Development of HE is associated with a poor prognosis and strongly predicts short-term mortality in acute liver failure.

*Common precipitants*
- Increased nitrogen load: constipation, gastro-intestinal bleeding, blood transfusion, azotaemia, infection, hypokalaemia.
- Decreased toxin clearance: dehydration (fluid restriction, diuretics, abdominal paracentesis, diarrhoea, vomiting), hypotension (bleeding, systemic vasodilatation), anaemia, portosystemic shunts.
- Altered neurotransmission: benzodiazepines, psycho-active drugs.
- Hepatocellular damage: continued alcohol use, hepatocellular carcinoma.

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**Box 7.31  West Haven classification**
- *Grade 0:* encephalopathy without clinically overt cognitive dysfunction, can be demonstrated by neuropsychological studies
  - Minor memory problems, no changes in behaviour. No asterixis.
- *Grade 1:* trivial lack of awareness, altered mood/behaviour, sleep disturbance, shortened attention span, impaired performance of addition or subtraction
  - Tremor, constructional apraxia, incoordination.
- *Grade 2:* lethargy or apathy, disorientation, inappropriate behaviour, slurred speech
  - Asterixis, ataxia.
- *Grade 3:* somnolence to semi-stupor but responsive to verbal stimuli, significant confusion, gross disorientation
  - Asterixis usually absent, hyperreflexia.
- *Grade 4:* coma with or without response to painful stimuli
  - Decerebrate posture.

### Jaundice

Some causes of jaundice are shown in Box 7.32.

*Examination: inspection*

- Inspect the sclera and conjunctiva.
  - Using the left thumb, pull on the patient's lower eyelid and ask them to look towards the ceiling
  - Inspect the soft palate with a pen torch in cases of doubt (bilirubin is avidly taken up by tissues that are rich in elastin)
- Check for signs of chronic liver disease.
- Look for body piercings and tattoos (hepatitis risk).

*Examination: palpation*

- Palpate the abdomen for tenderness, masses, and organomegaly (including the gallbladder).

*Examination: percussion*

- Percuss for the liver, spleen, and presence of ascites.

*Examination: completion*

- Perform a digital rectal examination to look for pale stools (post-hepatic) or melaena (GI bleed complication).
- Examine the external genitalia for hair growth and testicular size (atrophic in chronic liver disease).
- Examine the hernial orifices.
- Carry out a urinary dipstick test for bilirubin (post-hepatic).

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**Box 7.32  Causes of jaundice**

*Pre-hepatic (unconjugated hyperbilirubinaemia)*

- Overproduction: haemolysis; ineffective erythropoiesis
- Impaired hepatic uptake: drugs (contrast agents, rifampicin), congestive cardiac failure
- Impaired conjugation: Gilbert's syndrome, Crigler–Najjar syndrome.

*Hepatic (conjugated hyperbilirubinaemia)*

- Infection: viral hepatitis, leptospirosis, liver abscess, septicaemia
- Alcohol and toxins: carbon tetrachloride, fungi (*Amanita phalloides*)
- Drug-induced hepatitis: paracetamol, anti-tuberculosis drugs (isoniazid, rifampicin, pyrazinamide), statins, sodium valproate, halothane
- Metabolic: haemochromatosis, $\alpha_1$-antitrypsin deficiency, Wilson's disease, Rotor syndrome
- Vascular: Budd–Chiari, right-sided heart failure.

*Post-hepatic (conjugated hyperbilirubinaemia)*

- Luminal: gallstones
- Mural: cholangiocarcinoma, sclerosing cholangitis, primary biliary cirrhosis, choledochal cyst
- Extra-mural: pancreatic cancer, lymph nodes at porta hepatis
- Drugs: antibiotics (flucloxacillin, fusidic acid, co-amoxiclav, nitrofurantoin).

### Gallstones

Some causes of gallstones are shown in Box 7.33.

*Background and epidemiology*
- Stones form due to the supersaturation of bile constituents, usually cholesterol.
- Affects 10% of the population (and 2% of children).
- Incidence increases with age (40% of women >80 years).
- Male:female ratio roughly 1:2.
- Typical patient: '5 Fs' (female, forty, fair, fat, fertile).

*Examination: inspection*

There may be no signs of gallstones. Look for risk factors and complications.
- Jaundice?
- Scars of open or laparoscopic cholecystectomy (see separate topics).
- If the patient has recently had surgery, a T-tube and drain may be visible.
  - A flexible tube arising from the right upper quadrant.

*Examination: palpation*
- Examine for a palpable gallbladder.

*Murphy's test*
- Using 2 fingers, palpate just below the costal margin in the right upper quadrant and maintain the position whilst the patient takes a deep breath. Note any tenderness.
- Repeat the procedure in the left upper quadrant.
- If the patient experiences tenderness only when the right side is palpated, the test is positive.
  - Indicates acute cholecystitis, ascending cholangitis, empyema.

---

**Box 7.33 Conditions predisposing to gallstones**
- *Haemolysis:* sickle cell, hereditary spherocytosis, thalassaemia, pernicious anaemia, prosthetic heart valves
- *Metabolic:* diabetes, obesity, pancreatic disease, cystic fibrosis, hypercholesterolaemia, hyperparathyroidism, hypothyroidism, pregnancy
- *Cholestasis:* hepatitis, Caroli's disease, parasitic infection, prolonged fasting (e.g. TPN), methadone use
- *Malabsorption:* (x10 risk of stone formation) inflammatory bowel disease (especially Crohn's), small bowel resection, bypass surgery
- *Other:* muscular dystrophy.

## Ascites

*Definition and aetiology*

- Ascites is more than 25ml of fluid within the peritoneal cavity.
- Common causes: cirrhosis with portal hypertension, peritoneal carcinomatosis.
- Less common causes: hepatocellular carcinoma, Budd–Chiari syndrome, congestive cardiac failure, pancreatitis, and tuberculosis.

*Ascites complicating advanced cirrhosis*

- Ascites often marks the first sign of hepatic decompensation.
  - Occurs in >50% over 10 years of follow-up, worsens the course of disease, and reduces survival substantially
  - If ascites becomes refractory to diuretics, 50% die within 1 year.
- Spontaneous bacterial peritonitis (SBP) is a frequent and serious complication of cirrhotic ascites and is defined as an ascitic neutrophil count >250 cells/mm$^3$.

*Examination: inspection*

- If there is gross ascites, the abdomen may be distended.
  - Look for bulging of the flanks in a supine patient (fluid accumulates in the paracolic gutters).
- Look for signs of chronic liver disease:
  - Hands: clubbing, leukonychia, bruising, palmar erythema, Dupuytren's contracture, hepatic flap, scratch marks
  - Face: anaemia, jaundice, xanthelasma, parotid enlargement, glossitis
  - Neck: Troisier's sign/Virchow's node (intra-abdominal malignancy)
  - Trunk: spider naevi, gynaecomastia
  - Abdomen: distended superficial veins, caput medusa.

*Examination: palpation*

- Palpate in each quadrant for tenderness and masses.
  - Palpation for organomegaly may be difficult in gross ascites.

*Examination: percussion*

- Percuss borders of the liver, spleen, bladder, and any masses.
- Shifting dullness.
- Fluid thrill test.

*Examination: completion*

- Examine the hernial orifices, lymph nodes and cardiovascular system (peripheral oedema and pleural effusion).

### Primary biliary cirrhosis

- Autoimmune liver disease characterized by progressive destruction of intrahepatic bile ducts with cholestasis, portal inflammation and fibrosis.
- May lead to cirrhosis, its complications, and eventually to liver transplantation or death.
- Predominantly affects females in their 5th to 7th decades.

*History*

- Majority are asymptomatic.
- Fatigue (multifactorial: autonomic dysfunction, sleep disturbance, and excessive daytime somnolence, depression).
- Pruritus (typically precedes onset of jaundice by months to years. Develops independently of degree of cholestasis and stage of disease).
- Vague right upper quadrant pain.
- Night blindness, bony pain, easy bruising, fat-soluble vitamin malabsorption (A, D, E, and K).

*Examination: inspection*

Look for:

- Scratch marks.
- Pigmentation.
- Digital clubbing.
- Arthropathy (involving small joints).
- Xanthelasma.
- Xanthoma.
- Evidence of decompensated liver disease:
  - Jaundice
  - Abdominal distension
  - Dilated abdominal veins
  - Hepatomegaly
  - Splenomegaly
  - Ascites
  - Encephalopathy
  - Flapping tremor (asterixis).

*Associated autoimmune conditions*

- Look for evidence of: rheumatoid arthritis, thyroid dysfunction, Sjögren's syndrome, scleroderma, systemic lupus erythematosus, coeliac disease.

## Haemochromatosis

- Autosomal recessive condition causing an abnormal accumulation of iron in the parenchymal organs, leading to organ toxicity.
- Gene for most genetic haemochromatosis (GH), called 'HFE', lies on short arm of chromosome 6. The two major mutations are C282Y and H63D. Defective hepcidin (iron-regulatory hormone) gene expression or function may underlie most forms of non-HFE associated GH.
- Carrier state ~1:10; frequency of homozygosity ~1:200/400 but penetrance is low (higher with co-factors such as excess alcohol).
- Male preponderance; menstruation has protective effect in females.

*Symptoms*
- Often vague and non-specific.
  - Weakness, fatigue, lethargy, apathy, weight loss.
- Organ specific: arthralgia, abdominal pain (hepatomegaly), amenorrhoea, loss of libido, impotence (pituitary dysfunction, hepatic cirrhosis), shortness of breath (CCF).
- Impaired memory, mood swings, and irritability.

*Examination findings*
A patient with haemochromatosis may show any of the following clinical features:
- Hepatomegaly.
- Cutaneous stigmata of liver disease (palmar erythema, jaundice, spider naevi).
- Signs of portal hypertension (splenomegaly, ascites).
- Arthritis and joint swelling.
  - Especially 2nd, 3rd metacarpophalangeal joints, wrists, hips, and knees.
- Shortness of breath, oedema, raised JVP (dilated cardiomyopathy).
- Arrhythmias (conduction abnormalities).
- Altered pigmentation.
  - Bronzing or 'slate-grey' due to iron and melanin deposition.
- Scars in cubital fossae (previous venesection).
- Hair loss.
- Hypogonadism (testicular atrophy, loss of axillary and pubic hair).
- Increased blood and urine glucose levels (diabetes mellitus secondary to iron toxicity of pancreatic beta cells).
- Signs of hypothyroidism (see separate topic).

### Wilson's disease

*Definition and epidemiology*
- Wilson's disease or hepatolenticular degeneration is a rare autosomal recessive inherited disorder of copper metabolism characterized by excessive deposition of copper in the liver, brain, and other tissues.
- Affects about 1 in 30,000 people, often manifesting as liver disease in children and adolescents and as a neuropsychiatric illness in young adults.

*Pathogenesis*
- The transport of copper by the P-type ATPase is defective due to one of several mutations (>300 identified) occurring within the ATP7B gene.
- This gene has been mapped to chromosome 13q14.3.
- Intestinal copper absorption and transport into the liver are intact, whilst incorporation into caeruloplasmin and excretion into bile are impaired.

*Clinical features*
Wilson's can cause a variety of clinical findings. A patient may present with any of the clinical features described here.
- Neurological.
  • Dysarthria
  • Dystonia
  • Tremor
  • Incoordination
  • Parkinsonian symptoms (rigidity, bradykinesia)
  • Poor hand-writing
  • Abnormal eye movements
  • Polyneuropathy.
- Hepatic.
  • Acute hepatitis, chronic active hepatitis
  • Cirrhosis
  • Fulminant hepatic failure.
- Psychiatric.
  • Irritability or anger, emotional lability
  • Hyperkinetic behaviour
  • Mania, depression
  • Psychosis
  • Impaired concentration
  • Personality changes.
- Ophthalmic: Kayser–Fleischer rings.
  • Deposition of copper in Descemet's membrane at the limbus of the cornea; greenish gold-brown, may be readily visible with the naked eye or can be identified on slit-lamp examination
  • Seen in nearly all with neurological signs of Wilson's disease
  • Sunflower cataracts (visible only with slit lamp).
- Other (<10% patients).
  • Endocrine, renal, cardiac, skeletal manifestations.

## Peutz–Jeghers syndrome

*Background and epidemiology*

- Peutz–Jeghers syndrome is due to germline mutations of the STK11 gene and is characterized by intestinal hamartomatous polyps and mucocutaneous melanocytic macules.
- Autosomal dominant, with variable penetrance. Incidence is estimated at between 1 in 50,000 to 1 in 200,000 live births.
- The disease carries an increased risk of gastrointestinal and other cancers, including pancreas, breast, cervix, ovary, uterus, lung, and testis.
- Patients have a 93% cumulative risk of cancer by the age of 64; roughly half die from cancer by the age of 57. Rapid increase in risk >50 years.
- Differential diagnosis is shown in Box 7.34.

*Examination: inspection*

- Pigmented macules of 1–5mm in diameter.
- Most commonly around the mouth, crossing the vermilion border of the lip.
- Dark brown to black (reminiscent of freckles).
- They may be present also on the hands and feet, and around the umbilicus, genitalia, and anus.
  - Similar lesions often occur on the buccal mucosa.
- A prolapsed rectal polyp may be seen.

*Examination: other systems*

- Abdomen: inspect for scars (e.g. resulting from surgery for intussusceptions or malignancy).
- A rectal mass due to a polyp may be palpable.
- Testicular masses also occur: examine the testicles and check the chest for gynaecomastia (as a result of a Sertoli cell tumour).
- In females, the breasts should be assessed for masses.

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**Box 7.34 Differential diagnosis of Peutz–Jeghers syndrome**

- *Laugier–Hunziker syndrome:* peri-oral and intra-oral pigmentation, but lacks systemic manifestations
- *Cronkhite–Canada syndrome:* intestinal polyposis and freckle-like lesions; presents in older individuals and the skin lesions are more extensive
- *Addison's disease:* hypotension, hyper-pigmentation of skin and gums, hyponatraemia, and hyperkalaemia
- *McCune–Albright syndrome:* precocious puberty, café-au-lait lesion, fibrous dysplasia of bones including the femur, tibia, pelvis, and skull
- *Familial adenomatous polyposis and Gardner syndrome:* numerous adenomatous colonic polyps. Gardner variant includes multiple extra-colonic polyps such as osteomas and desmoid tumours.

## Appendicitis

*Epidemiology*

- The diagnosis is clinical, therefore investigations are only needed to exclude other pathology.
- Lifetime risk 7%.
- Overall mortality 0.2–0.8% but higher in >70yrs due to diagnostic delay.
- After the first 36 hours, perforation risk is 16–36%, with 5% risk for every subsequent 12 hours.

*Examination: inspection*

- Fever.
- Pain on movement.
- Flexion of the right hip and a reluctance to extend it.
- Flushed appearance.
- Dry tongue.
- Fetor oris.
- Ask the patient to point to the site of pain with one finger.
  - Classically this is McBurney's point (2/3 of the way along a line between the umbilicus and the right anterior superior iliac spine).

*Examination: palpation*

- Tenderness and (guarding) at the right iliac fossa.
  - Maximal tenderness may be over McBurney's point.
- Percussion tenderness in the RIF.
- Rovsing's test.
  - Press the left iliac fossa and ask the patient if and where they feel pain
  - If pain is felt at the right iliac fossa, the test is positive.
- ▶ Rebound tenderness. Testing for this should be avoided as this is painful and does not add to the diagnosis.

*Other tests*

- PR or PV examinations: tenderness on the right may indicate appendicitis, but examination may be normal (see also Box 7.35).
- Psoas sign: extension of the hip stretches the psoas and is painful if irritated by a nearby inflamed appendix (especially retrocaecal).
- Obturator sign: suprapubic pain on flexion and internal rotation of the right hip. Due to obturator internus irritation by inflamed appendix.

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**Box 7.35 Findings by appendix position**

- *Retrocaecal (75%):* right loin/right iliac fossa pain and tenderness. Guarding may be absent. Psoas sign may be present
- *Pelvic (20%):* suprapubic pain, urinary frequency, diarrhoea. Bladder irritation may give haematuria and urinary leukocystosis. Abdominal tenderness may be minimal; rectal and vaginal tenderness predominate
- *Pre-ileal and post-ileal (5%):* may have few signs and symptoms. Ileitis may lead to vomiting.

## Ulcerative colitis (UC)

*Definition and epidemiology*
- UC is an idiopathic inflammatory bowel disease characterized by colonic mucosal inflammation and a chronic relapsing course.
- UC extends uninterrupted from the anal verge to involve part or all of the colon. Apart from backwash ileitis, the small bowel is not involved.
- Bimodal distribution with peaks at 15–30 years and in the 6th decade.
- Three times more common in non-smokers (some relapse on quitting).

*Associated conditions*
- Primary sclerosing cholangitis, cholangiocarcinoma, amyloidosis, uric acid renal stones.

*History*
- Depends on extent and activity of the disease.
  - Bloody diarrhoea, mucous discharge, faecal urgency, tenesmus, colicky abdominal pain, fever, malaise, anorexia, weight loss.

*Inspection: general*
Physical examination is often unremarkable, unless the patient is presenting acutely.
- Aphthous ulcers.
- Glossitis.
- Pallor (anaemia is common).
- Peripheral oedema.
- Digital clubbing.
- Ocular inflammation (uveitis, episcleritis, scleritis).
- Cushingoid features (if steroid use).
- Enteropathic arthropathy (large joint arthritis, seronegative spondyloarthropathy: sacroiliitis, ankylosing spondylitis).
- Erythema nodosum (15%).
- Pyoderma gangrenosum (1–2%).

*Inspection: abdomen*
- May be surgical scars (e.g. from hemicolectomy).
- Stomas or healed stoma sites.
- Abdominal drains or healed drain sites.

*Palpation*
- May find distended, tense abdomen.

*Percussion*
- Hyper-resonance (if abdomen distended).

*Auscultation*
- Tinkling bowel sounds (in obstruction).

## Crohn's disease

*Definition and epidemiology*
- Idiopathic inflammatory bowel disease characterized by transmural, granulomatous inflammation anywhere from mouth to anus (most common at ileocaecum). It has a chronic, relapsing/remitting course.
- Age of onset is bimodal with peaks at 15–30 and 60–80 years.
- Smoking increases risk x3–4.

*Intestinal complications*
- Malnutrition, fistulae, colorectal adenocarcinoma (Crohn's colitis), short bowel syndrome (following surgical resection).

*Extra-intestinal complications*
- Hepatic: fatty change, chronic active hepatitis, cirrhosis, amyloidosis.
- Biliary tract: gallstones, sclerosing cholangitis, cholangiocarcinoma.
- Renal: uric acid stones, oxalate stones.
- Musculoskeletal: enteropathic arthropathy, osteoporosis.
- Ocular: uveitis, episcleritis, scleritis.
- Dermatological: erythema nodosum, pyoderma gangrenosum.
- Haematological: anaemia (Fe, $B_{12}$, and folate deficiency), thrombosis.

*History*
- Abdominal pain, diarrhoea, weight loss, fever, malaise, anorexia.

*Inspection: general*
- Aphthous ulcers.
- Glossitis.
- Pallor (anaemia is common).
- Peripheral oedema.
- Digital clubbing.
- Ocular inflammation (uveitis, episcleritis, scleritis).
- Cushingoid features (if steroid use).
- Enteropathic arthropathy (large joint arthritis, seronegative spondyloarthropathy: sacroiliitis, ankylosing spondylitis).
- Erythema nodosum (15%), pyoderma gangrenosum (1–2%).

*Inspection: abdomen and perineum*
- Multiple surgical scars.
- Stomas or healed stoma sites.
- Enterocutaneous fistulae.
- Perianal skin tags, fissures, ulceration, sinuses.
- Abdominal drains or healed drain sites.

*Palpation*
- May find distended, tense abdomen, mass (especially in right iliac fossa), hepatomegaly.

*Percussion*
- Hyper-resonance (if abdomen distended).

*Auscultation*
- Increased bowel sounds (in acute exacerbations).

## Perianal disease

*Haemorrhoids*
- Hypertrophied endoanal cushions (causing symptoms).
- Clinical features: acute prolapse and inflammation with associated perianal lump, soreness, and irritation. May bleed. Chronic: bleeding and pruritus ani.
- Sites: primary (3, 7, 11 o'clock in the supine position; the locations of the main anal blood vessel pedicles).
- Classification (Lord's):
  • 1st degree: bleeding, no prolapse
  • 2nd degree: prolapse, spontaneous reduction
  • 3rd degree: prolapse, digital reduction.

*Fissure in ano*
- A superficial linear tear in the anoderm distal to the dentate line commonly caused by passage of hard stool. Almost always in the midline: 90% posterior, 10% anterior.
- Clinical features: identified on inspection, often too painful to perform PR. Causes acute severe, localized 'knife-like' pain during defaecation with associated deep throbbing pain for minutes or hours after (pelvic floor spasm). Blood is often seen on the paper when wiping.

*Perianal abscess*
- Definition: abscess within the soft tissues surrounding the anal canal.
- Clinical features: gradual onset, constant localized perianal pain. Associated swelling with tenderness and possible discharge.

*Fistula in ano*
- Definition: abnormal communication between the anorectal lining and the perineal or vaginal epithelium. Nearly always caused by a previous anorectal abscess. Other less common causes include trauma, Crohn's disease, carcinoma, radiation therapy, and tuberculosis.
- Clinical features: perianal or perineal pain, swelling and erythema of perianal skin, fever, tachycardia, discharge.
- Goodsall's rule: if the external opening is posterior to a line drawn transversely through the anus (in the supine position), the opening will be in the midline and thus have a curved tract. If the opening is anterior to this line it will have a radial tract.

*Rectal prolapse*
- Definition: protrusion of either the rectal mucosa or the entire wall of the rectum (complete prolapse). Mainly occurs in the elderly and young children.
- Clinical features: obvious, large perineal lump, dark red/blue with surface mucosa and, occasionally, some surface ulceration. Pain, constipation, faecal incontinence, mucous discharge, or rectal bleeding may occur.

### Nephrotic syndrome

Nephrotic syndrome is a clinical syndrome, not a diagnosis.

*Nephrotic syndrome is a tetrad of:*
- Proteinuria >3g/24 hours.
- Oedema.
- Hypoalbuminaemia.
- Hyperlipidaemia.

*Common causes of nephrotic syndrome in adults*
- Minimal change disease (MCD).
- Membranous nephropathy.
- Focal segmental glomerulosclerosis.
- Diabetic glomerulosclerosis.

*Other causes of nephrotic syndrome*
- Renal amyloidosis, lupus nephritis, mesangiocapillary glomerulonephritis, collapsing glomerulopathy (HIV-associated nephropathy), light chain deposition disease.

*Other causes of bilateral swollen legs*
- Right ventricular failure.
- Lymphoedema.
- Hypoalbuminaemia.
- Hepatic failure.

*Complications*
- Increased risk of thromboembolism (loss of anticoagulant factors in the urine).
  - Those with albumin <20g/L are often anticoagulated with warfarin.
- Renal vein thrombosis.
  - Suspect if the patient develops loin pain, haematuria, and an acute deterioration in their renal function.
- Pulmonary emboli.
- Infection (loss of immunoglobulin and complement).
- Hypercholesterolaemia.

*Examination findings*
- Extensive oedema.
- Periorbital swelling.
  - Typically worse in the morning.
- Bilateral pitting oedema of the lower limbs.
  - Usually symmetrical
  - May extend up to the abdomen.
- Also look for evidence of:
  - Pulmonary oedema
  - Pleural effusion
  - Ascites.

## Chronic kidney disease (CKD)

*Common causes in the UK*
- Diabetes mellitus.
- Glomerulonephritis e.g. IgA nephropathy.
- Reflux nephropathy.
- Obstructive uropathy.
- Renovascular disease.
- Hypertension.
- Polycystic kidney disease.

*Other causes*
- Myeloma, renal amyloidosis, systemic lupus erythematosus (SLE), vasculitis, tubulointerstitial nephritis, scleroderma, other inherited renal diseases e.g. Alport's disease, oxalosis, cystinosis.

*History*
Many patients with CKD are asymptomatic. Symptoms usually develop at an advanced stage and include:
- Fatigue, weakness (secondary to anaemia).
- Breathlessness (due to fluid overload, acidosis).
- Anorexia, vomiting, metallic taste in mouth (due to uraemia).
- Pruritus.
- Restless legs.
- Bone pain.
- Leg swelling.

*Examination findings*
A patient with CKD may have no specific clinical findings or they may have a number of clinical features:
- Pallor (due to chronic anaemia).
- A lemon tinge to the skin (due to uraemia).
- Scratch marks from pruritus.
- Hyertension.
- A pericardial rub (uncommon, due to uraemia).
- Pleural effusions.
- Palpable kidneys (causes include polycystic kidney disease, hydronephrosis).
- Bilateral lower limb oedema (fluid overload or heavy proteinuria).
- Distended bladder?

*Evidence that the patient is being prepared for dialysis*
- An arteriovenous fistula either at the wrist or in the antecubital fossa (usually in the non-dominant arm) for haemodialysis.
- A peritoneal dialysis catheter situated in the abdomen.

### Transplanted kidney
- A renal transplant is the most favourable and desired form of renal replacement therapy for patients with end-stage renal disease (ESRD).
- Simultaneous pancreas–kidney transplants are performed in patients with type 1 diabetes and stage 5 CKD (see Box 7.36).
- Following a renal transplant, patients are required to take life-long immunosuppression to prevent graft rejection.
- Transplanted kidneys may be from a deceased or a living donor.
- A transplanted kidney will lie in either the left or right iliac fossae.

*Examination: inspection*
- Look around the bedside for clues that the patient has diabetes.
- There may be lipoatrophy or lipohypertrophy at insulin injection sites.
- Look for other complications of severe diabetes including signs of visual impairment due to diabetic retinopathy and peripheral vascular disease.

*Examination: abdomen*
- A firm mass palpable deep to a diagonal scar.
  - It is possible that a patient may have had more than one transplant and could have a transplanted kidney in each iliac fossa
  - An extended Lanz incision in either iliac fossa is called a Rutherford–Morrison incision
  - The renal artery is usually anastomosed to the internal or external iliac artery and the renal vein to the external iliac vein. The ureter is attached separately to the patient's bladder.
- Looks for scars of previous nephrectomy (midline, chevron, or loin).

*Evidence of end-stage renal disease*
- An arteriovenous fistula either at the wrist or in the antecubital fossa (Cimino–Brescia fistula).
- Small scars near the umbilicus consistent with previous peritoneal dialysis catheter placement.
- Small scars beneath the clavicle on either side of the chest which might suggest previous tunnelled dialysis catheter placement.
- A scar at the base of the neck consistent with parathyroidectomy (for advanced renal bone disease).

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**Box 7.36 Simultaneous pancreas–kidney transplant (SPK)**
- A curved (bucket-handle) scar across the lower abdomen extending between the iliac fossae with a mass palpable in each
- The pancreas is usually transplanted into the right iliac fossa and the kidney into the left.

## Adult polycystic kidney disease

- Adult polycystic kidney disease (APKD) is the commonest inherited form of renal disease with a prevalence of around 1 in 1000.
- APKD is inherited in an autosomal dominant manner.
- 25–40% of patients will have no family history.
- Polycystic kidneys are usually not difficult to miss on examination.

*History*

- Flank or loin pain from enlarged or infected cysts.
- Nocturia and polyuria (loss of urinary concentrating ability).
- Hypertension.
- Low grade proteinuria, persistent microscopic haematuria +/− frank haematuria.
- Uraemic symptoms if the patient presents late.

*Examination findings*

- Bilaterally enlarged kidneys with irregular surfaces (see Box 7.37).
  • Ballotable flank masses which move caudally with inspiration and the examining hand can 'get above'.
- There may also be an enlarged liver with an irregular, lobulated edge.
- Look for evidence of end-stage renal disease.

*Evidence of advanced or end-stage renal disease (ESRD)*

- A scar in the iliac fossa and a mass consistent with a renal transplant.
- An arteriovenous fistula either at the wrist or in the antecubital fossa (usually in the non-dominant arm) for haemodialysis.
- A peritoneal dialysis catheter situated in the abdomen or a scar consistent with previous catheter placement.
- A scar at the base of the neck consistent with parathyroidectomy (for advanced renal bone disease).
- Small scars beneath the clavicle on either side of the chest which might suggest previous tunnelled dialysis catheter placement.

*APKD associations*

- Cysts of other organs notably the liver, spleen, and pancreas.
- Cardiac abnormalities: mitral valve prolapse, aortic regurgitation.
- Intracranial aneurysms which on rupture present as subarachnoid haemorrhage. Patients are screened for these only if there is a strong positive family history of intracranial bleeding.
- Colonic diverticula, abdominal and inguinal herniae.

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**Box 7.37 Other causes of bilateral renal enlargement**

- Bilateral hydronephrosis
- Amyloidosis (hepatosplenomegaly?)
- Tuberous sclerosis (adenoma sebaceum or shagreen patches?)
- Von Hippel–Lindau disease.

**See also:**

More information regarding the presentation and clinical signs of abdominal diseases to aid preparation for OSCE-type examinations and ward rounds can be found in the *Oxford Handbooks Clinical Tutor Study Cards*.

*'Medicine' Study Card set:*
- Chronic liver disease
- Hepatic encephalopathy
- Autoimmune hepatitis
- Primary biliary cirrhosis
- Haemochromatosis
- Wilson's disease
- Chronic kidney disease
- Lymphadenopathy
- Nephrotic syndrome.

*'Surgery' Study Card set:*
- Abdominal mass
- Inguinal hernia
- Femoral hernia
- Incisional hernia
- Paraumbilical hernia
- Spigelian hernia
- Abdominal stomas
- Appendicitis
- Jaundice
- Ascites
- Peutz–Jeghers syndrome
- Gallstones
- Perianal disease.

*Both the 'Medicine' and 'Surgery' Study Card sets:*
- Hepatomegaly
- Splenomegaly
- Ulcerative colitis
- Crohn's disease
- Adult polycystic kidney disease
- Transplanted kidney
- Single palpable kidney.

# 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? Many patients may describe themselves as having such diagnoses, but take the time to clarify what this means. Recent changes of bowel habit must always be treated seriously.
- *Constipation:* this can often lead to serious decline in patients. 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.
- *Drug history:* always consider the side effects of medication—analgesics and constipation, recent antibiotics, and diarrhoea – has the patient recently been in hospital (could the diarrhoea represent *C. difficile* infection?). 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 candidiasis 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.

# The nervous system

# Presenting symptoms

The history is key in 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, and associated symptoms.

- Ask about facial and visual symptoms. (Some different types of headaches are described in Box 8.1.)

*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 5.

*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–clonic 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.

See  Chapter 9 for more on visual symptoms.

# 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

- Headaches (see Box 8.1).
- Fits, faints and 'funny turns', and '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.

## Drug history

Ask especially about:

- Anticonvulsant therapy (current or previous), oral contraceptive pill, steroids, anticoagulants, 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.

**Box 8.1 Some characteristic headaches**

*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
- 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.

# 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. (See Box 8.2.)

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**Box 8.2 Examination framework**

The neurological examination can be complex and lengthy. The following is a brief outline of an approach to a 'full' neurological examination.

- Inspection, mood, conscious level
- Speech and higher mental functions
- Cranial nerves II–XII
- Motor system
- Sensation
- Coordination
- 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?
- Observe the gait as they approach the clinic room, if able.
- Any speech disturbance?
- What is their mood like?
  - A detailed mood assessment 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?

# Cognitive function

Neurological diseases may affect function such that patients' appearance or communication skills are at odds with their educational level—formal assessment allows for any future change to be noted and monitored.

## The Abbreviated Mental Test Score (10 points)

This serves as a brief screening tool (see Box 8.3). A more detailed, 30 point, score is shown in ➲ Box 16.12.

▶ Approach this gently—you may offend a patient by testing them without warning or explanation. Always explain the purpose of the questions—and ask their permission to proceed.

| Box 8.3  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.* 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 sometimes need a prompt here 'Like this: 20, 19, 18, and so on'. |
| *10.* Recognition | ● 'What job do I do?' and 'What job does this man/woman do?' |
| | • *Both* must be correct to score a point. |

## Hints and tips

● 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!

# 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 possibly 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 language function by asking the patient 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 or instructions, more complicated sentences, and finally by asking them to repeat complex phrases or tongue-twisters.
- Before jumping to conclusions, ensure that the patient is not deaf (or that their hearing-aid is working) and that they can usually understand the language you are speaking.

### Dysarthria

A defect of articulation with language function intact (writing will be unaffected). There may be a cerebellar lesion, an 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 myasthenia 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).

There are four main types of dysphasia:

*Expressive dysphasia*

Also called 'anterior', 'motor', or 'Broca's' dysphasia. (See Box 8.4.)
- 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.

---

**Box 8.4 Communicating in expressive dysphasia**

This can be very distressing for patients.

Relatives and others often try to get the patient to write things down, unaware that the problem is *language*, and are disappointed to find the attempts at writing are just as garbled as the speech.

Some helpful tips include:
- Do not pretend to understand, tell the patient what you can and cannot guess at and ask if you've got it right
- Encourage other means of communication including body language and hand signals. The patient is often still able to draw diagrams.

---

*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 substitution of one word with another.

*Conductive dysphasia*

Lesion in the arcuate fasciculus and/or other connections between the two 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 at the angular gyrus.
- Patient may function with 'circumlocution' (e.g. says 'that thing that I write with' if unable to say 'pen').

*Global dysphasia*
- Both Broca's and Wernicke's areas affected. The patient is unable to speak or understand speech at all.

# Cranial nerve examination

The following part of the chapter describes the individual examination of each cranial nerve in turn. In practice (and in exam circumstances), these are usually examined together in a fluid manner.

The student should know the ins and outs of each cranial nerve examination and develop their own method of going from one to the next that suits them and the patients.

# Cranial nerves II, III, IV, and VI

The examination of the II (optic), III (oculomotor), IV (trochlear), and VI (abducens) nerves is covered in detail in

Turn to the eye chapter for:

- Visual acuity.
- Visual fields.
- The pupils.
- Eye movements.
  - III, IV, and VI palsies
  - Other eye movement disorders.
- Ophthalmoscopy and the use of the ophthalmoscope.
  - Anterior segment examination
  - Posterior segment examination (including fundoscopy).

# 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. The 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:* mucus-blocked nostril, head trauma, subfrontal meningioma.

### ▶ Hints and tips

Peppermint, ammonia, and menthol stimulate the free trigeminal endings so are not a good test of cranial nerve I.

# Cranial nerve V: trigeminal

## Applied anatomy

*Sensory:* facial sensation in three branches—ophthalmic (V1), maxillary (V2), mandibular (V3). Distribution shown in Fig. 8.1

*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 three 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 warm 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).
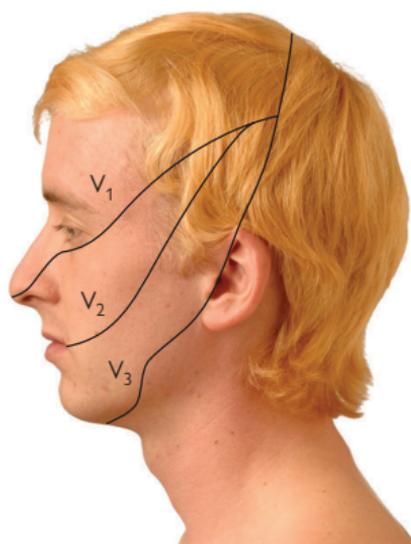
**Fig. 8.1** Distribution of the sensory branches of the trigeminal nerve.
$V_1$ = ophthalmic, $V_2$ = maxillary, $V_3$ = mandibular. Note that $V_1$ extends to the vertex and includes the cornea and $V_3$ does not include the angle of the jaw.

## 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 a 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 and tips

- 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 difficulty 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. (See Fig. 8.2.)

- 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).
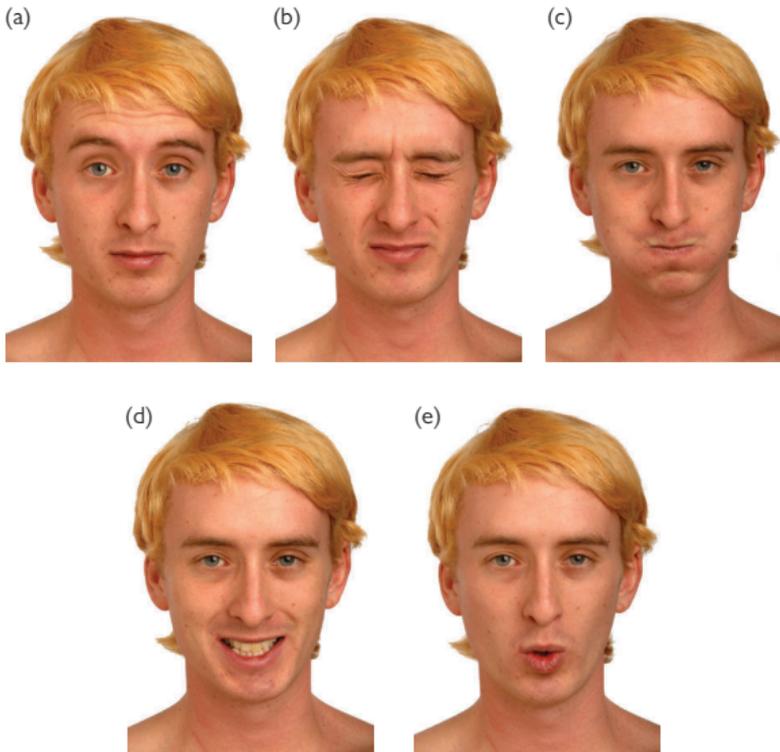
**Fig. 8.2** Testing the muscles of facial expression as described on ⮕ p. 254.
(a) Eyebrows; (b) eyelids; (c) puffing out the cheeks; (d) baring teeth; (e) whistle.

## 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, myasthenia 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 and tips

- *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 two 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 11.

## 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. 8.3a).
- Then apply the base of the tuning fork to the mastoid process (bone conduction)—see Fig. 8.3b.
- 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. 8.3c).
- 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.

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* This is the 'C' above 'middle C' for those who like to know such things.
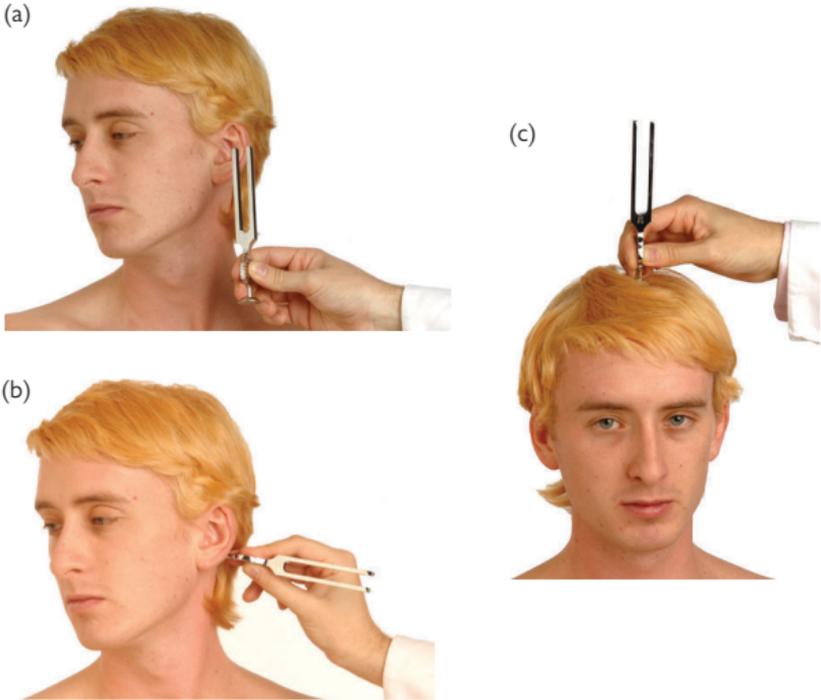
**Fig. 8.3** (a) Testing air conduction. (b) Testing bone conduction. (c) Position of the tuning fork for Weber's test.

## 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 lesion—sometimes 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:* stylopharyngeus
*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—two 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 × 3
  - Offer the patient a sip of water. Repeat × 3
  - Offer the patient the glass for a mouthful of water. Repeat × 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 arises 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, fasciculation, 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 a UMN lesion ipsilateral to the weak sternomastoid.

### Hints and tips

- 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. Lateral pressure to the jaw can cause pain and injury, particularly in the elderly and frail.

# 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

- An LMN neuron lesion will cause fasciculation 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 UMN lesion will rarely cause any clinically obvious signs.
- A bilateral UMN 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.

## ▶ Hints and tips

- Rippling movements may be seen if the tongue is held protruded for long periods. This is normal and should not be mistaken for fasciculation.

# 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. (See Box 8.5 also.)

## 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 in Fig. 8.4. 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 two 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 three pyramidal tracts:
- *Lateral corticospinal:* control of precise movement in the hands and feet and represents 90% of the UMN axons. These cross over (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 five 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.
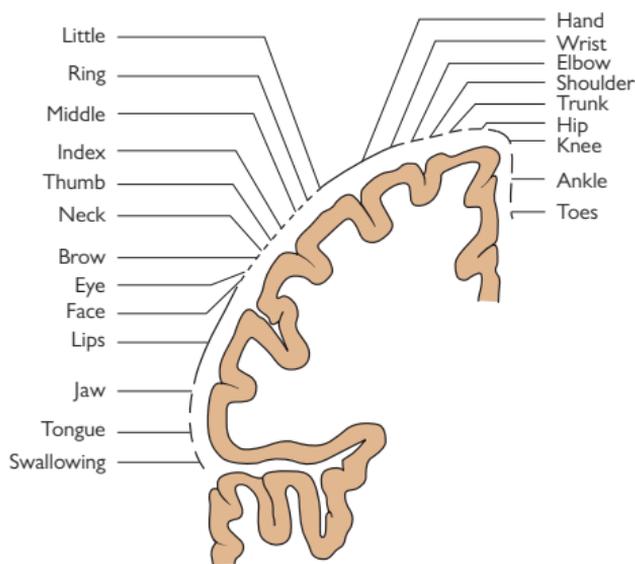
**Fig. 8.4** Coronal section through the motor cortex showing the representation of different muscle groups. Note the larger areas given to those muscles performing precise movements—hands, face, lips.

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**Box 8.5 Functional weakness**

Large parts of the neurological examination rely on the cooperation 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! 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?
- 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 practice and the feel of normal, ↓, or ↑ tone can only be learned through experience. (See Fig. 8.5.)

🛈 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. 8.5a). 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 on to the patient's knee and roll it from side to side (see Fig. 8.5b).
- *Knee:* with the patient in the same position, put your hand behind the patient's knee and raise it quickly (Fig. 8.5c). 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. 8.5d).

**Fig. 8.5** 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 to 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. 8.6).

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 in Box 8.6.

Examining the upper limbs also allows for both sides to be tested at once, allowing a direct comparison between left and right.

🕛 Be careful not to hurt frail and elderly patients or those with osteoarthritis, rheumatoid arthritis, 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. 8.6c) 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.



**Fig. 8.6** Testing power in the upper limbs. (a) Shoulder movements. (b) Elbow movements. (c) Finger flexion. (d) Finger extension. (e) Finger abduction. (f) Finger adduction.

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**Box 8.6  Medical Research Council (MRC) power classification**

- 5 = Normal power
- 4 = Movement against resistance but not 'full' normal power
- 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 = Muscle contractions but no movement seen
- 0 = No movement or muscular contraction

▶ The authors see an increasing number of power scores such as '4+' and '4–' in patients' notes. We find it hard to believe that someone can differentiate between 4+, 4 and 4–. There are either some very impressive clinicians about or people are attempting to be a little too precise in their scoring. Stick to whole numbers as described above.

# 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. (See Fig. 8.7.)

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. 8.7** 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

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 (Fig. 8.8). The patient should be relaxed (see notes on patient relaxation under 'inspection and tone' earlier in this chapter).

## 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 ↑. 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. 8.8** Testing tendon reflexes. (a) Biceps. (b) Triceps. (c) Supinator. (d) Fingers. (e) Knee. (f) Ankle. (g) Alternative method for ankle.

## Recording tendon reflexes

These are usually recorded as a list—or often by applying the numbers to the appropriate area of a stick-man sketch

- 0 = absent
- ± = present only with reinforcement
- 1+ = reduced/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.

## 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.
- Stroke the patient's sole—with an orange stick or similar disposable item (don't use your fingernail!).
- You should stroke from the heel, up the lateral aspect of the sole to the base of the 5th 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.

## 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 three beats of clonus—as long as the foot is held dorsiflexed—is abnormal.

## Abdominal reflex

(The upper segments are supplied by T8–T9, the lower by T10–T11.) This test relies on observing the abdominal muscles and 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, 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, 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 '+' and '–' respectively. ('±' for an intermediate response).

## 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.

# 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 three or four 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 8.10 and 8.11.

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 two main spinal pathways for sensory impulses. The clinical importance of these can be seen in spinal cord damage and is summarized later in this chapter.

### 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. 8.9). A lesion affecting one area will cause sensory loss in the corresponding body area on the contralateral side (see sensory pathways above).
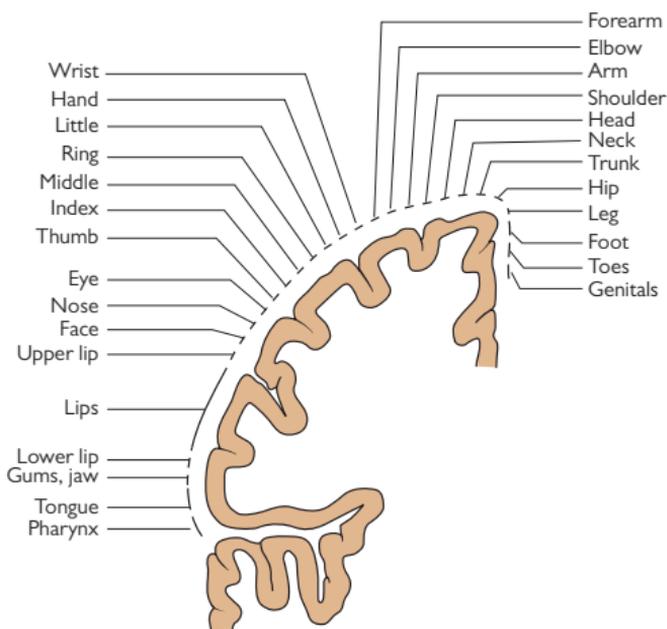
**Fig. 8.9** The sensory cortex map showing areas corresponding to the different parts of the body. Note the large areas given over to the fingers and lips.
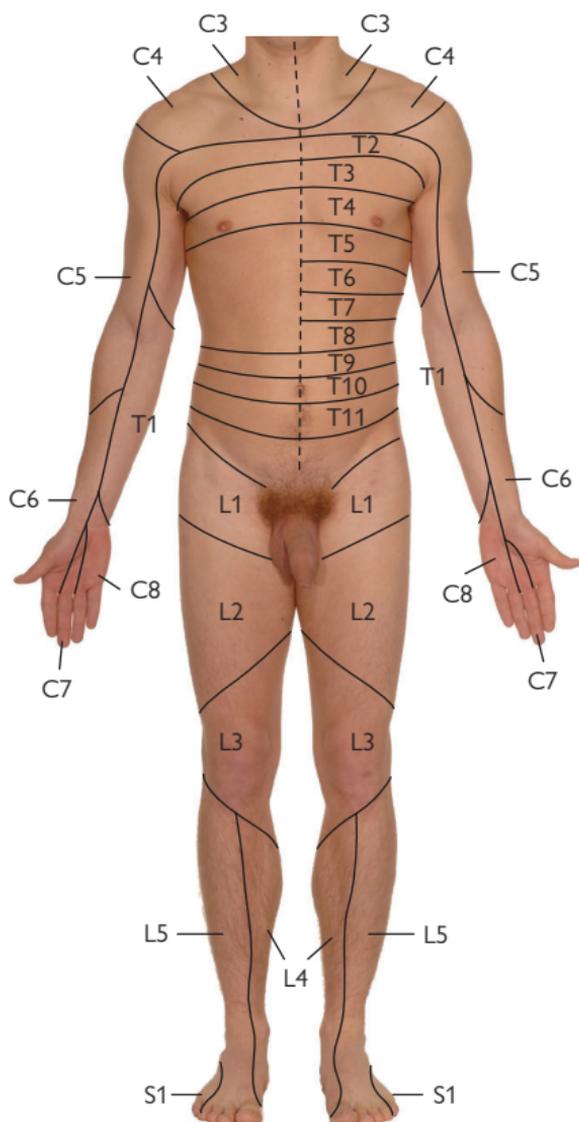
**Fig. 8.10** The dermatomes (anterior view). Students would do well to be familiar with these diagrams, particularly the limbs—note important landmarks to aid recall. (C7 covers the middle finger, T4 lies at the level of the nipples, T10 over the umbilicus.)
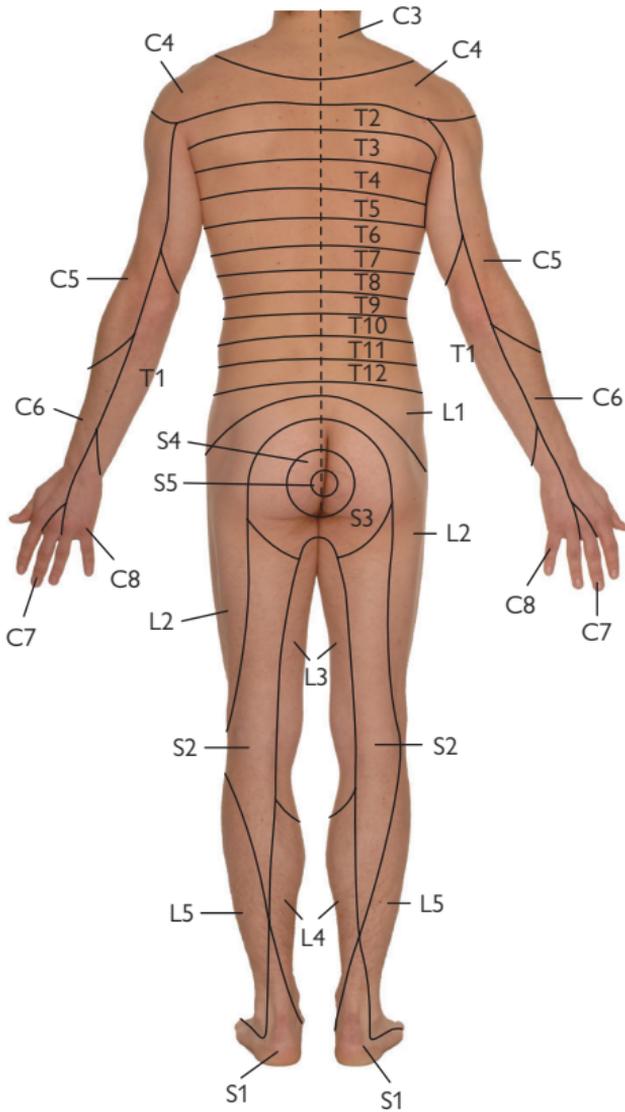
**Fig. 8.11** The dermatomes (posterior view).

# Sensory examination

This examination can be difficult—as it requires concentration and cooperation 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) is not noticed by the patient and revealing it 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 patient's 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 seem to, 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 (e.g. foot calluses).
- 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 not 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, metatarsophalangeal 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 phalanx 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.

*Pin-prick*

Use a disposable pin or safety pin—not a hypodermic needle as these break the skin.
- 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.

# Coordination

Coordination should be tested in conjunction with gait. Cerebellar lesions cause incoordination on the ipsilateral side. For each of the following, compare performance on the left and right. (See Box 8.7 for cerebellar syndrome.)

## 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. 8.12).
- 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. 8.13).
- Alternatively, ask them to mimic screwing in a light-bulb.
  - Slow and clumsy = dysdiadochokinesis.

🟢 This is the inability to perform rapidly alternating movements (dia-doke = Greek for succession).

*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 coordination, the patient will hit him/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.
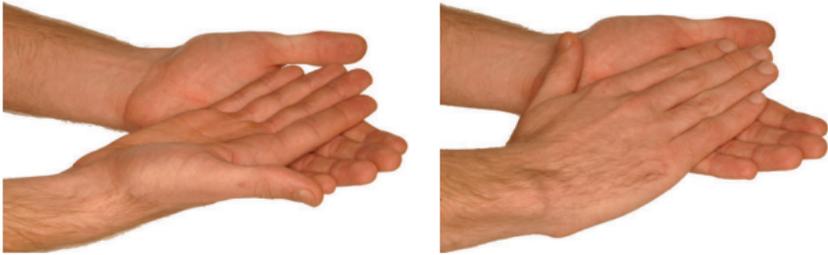
**Fig. 8.12** Testing rapidly alternating movements. This can be rather hard to describe to a patient—a brief demonstration is usually required.

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**Box 8.7 Cerebellar syndrome**

*Examination findings*

- Eye movements show a loss of smooth pursuit
  - This is the most subtle sign of cerebellar disease in the eyes.
- There is (may be) nystagmus
  - Note the direction of the gaze and the direction of the nystagmus.
- The patient is dysarthric
  - Note any dysarthria or the classical monotonous 'scanning' speech.
- Tone is reduced
  - Reduced in pure cerebellar disease but may be increased if cerebellar signs coexist with other disease (e.g. posterior circulation stroke, MS).
- Intention tremor
  - Perform finger–nose test.
- Dysdiadochokinesis
- Trunkal unsteadiness
  - Ask the patient to sit forward with their arms across their chest.
- Lower limb incoordination.
  - Ask the patient to place one heel on their knee on the other side, run it down their shin, pick it up and place it on their knee and repeat.

*Gait*

- Unsteady broad-based gait
  - If the patient is steady, assess tandem gait (walking with one foot placed in front of the other).

*Extras*

- Examine tendon reflexes
  - These may be reduced or pendular in cerebellar disease, or increased with coexisting disease (as above).

# 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 sections describe the signs following lesions of a selection of peripheral nerves. (See Boxes 8.8–8.10.)

## Upper limb

*Median nerve (C6–T1)*
- *Motor:* muscles of the anterior forearm, except flexor carpi ulnaris, and 'LOAF' (lateral two 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. 8.13).

*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. 8.13).

*Radial nerve (C5–C8)*
- *Motor:* triceps, brachioradialis, and extensors of the hand.
- *Sensory:* a small area over the anatomical snuff box—hard to test.
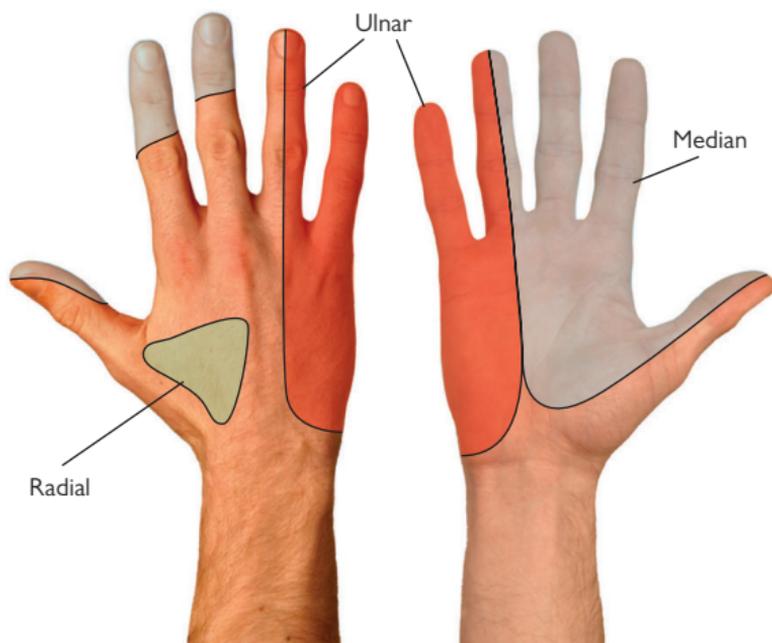


**Fig. 8.13** Sensory distribution of the major peripheral nerves in the hand. There is considerable overlap and the small area supplied by the radial nerve may not be detectable clinically.

**Box 8.8 Median nerve palsy**

*Inspection*
- Inspect the forearm carefully, looking for signs of rheumatoid arthritis, osteoarthritis, and any scars
- Wasting of the thenar eminence?
- Sign of benediction?
  - The index and middle fingers are held out straight despite flexion of the other fingers, rather like a Catholic blessing
  - Due to paralysis of flexor digitorum profundus.
- The thumb is held in abduction in the plane of the palm.
  - Also known as a 'simian thumb' or sometimes 'monkey paw'.

*Tone*
- Normal

*Power*
- Weakness of the thenar eminence
  - Abduction, flexion, and opposition of the thumb.
- Weakness of flexion at the terminal IP joint of the index finger
- The 'pen-touching test':
  - Place the patient's hand flat, palm up
  - Hold your pen (or similar object) just above the thumb and ask the patient to lift the thumb vertically to touch the pen, without moving the rest of their hand
  - In median nerve palsy, they will not be able to do this (weakness of flexor pollicis brevis).
- Oschner's clasping test:
  - Ask the patient to clasp their hands together with fingers interlocking
  - The index finger will fail to flex in median nerve palsy (weakness of flexor digitorum profundus).

*Sensation*
- Loss of sensation over the thumb, anterior index and middle fingers, and the radial side of the palm (see Fig. 8.13)

*Tinel's test*
- Percussion over the median nerve at the carpal tunnel causes tingling in the distribution of the nerve
  - Tinel's *sign* is paraesthesia in a nerve distribution caused by percussion of that nerve and applies to any nerve.

*Phalen's test*
- Ask the patient to hold both wrists flexed for 60 seconds
  - This produces an exacerbation of the paraesthesia – relieved by relaxing the wrist (positive in 50% of patients).

*Tourniquet test*
- A sphygmomanometer pumped to just above systolic pressure on the ipsilateral arm for 1–2 minutes reproduces the symptoms.

**Box 8.9 Ulnar nerve palsy**

*Inspection*
- Wasting of the dorsal interossei and hypothenar eminence
  - If the neuropathy is long-standing
- 'Ulnar claw'
  - Extension at the phalangeal-metacarpal joints and flexion at the interphalangeal joints – usually little finger first, then ring finger.
- The patient may be complaining of pain, usually in the forearm or elbow.

*Tone*
- Normal

*Power*
- Weakness of all the small muscles of the hand, except 'LOAF' (**l**ateral two **l**umbricals, **o**pponens pollicis, **a**bductor pollicis brevis, **f**lexor pollicis brevis)
- Weak abduction of the index finger
- Ulnar muscles in the forearm may be weak:
  - Flexor digitorum profundus of the fourth and fifth digits which flexes the distal phalanges
  - Flexor carpi ulnaris which flexes the wrist in the medial direction.
- **Froment's sign**: ask the patient to pinch a piece of paper between thumb and index finger or make a fist; this will be weak and the thumb will flex as it is unable to abduct (Fig. 8.14)
- Test thumb abduction to rule out a C8/T1 nerve root lesion.

*Reflexes*
- Normal tendon reflexes

*Sensation*
- Loss of sensation over the medial aspect of the hand, little finger, and medial half of the ring finger.
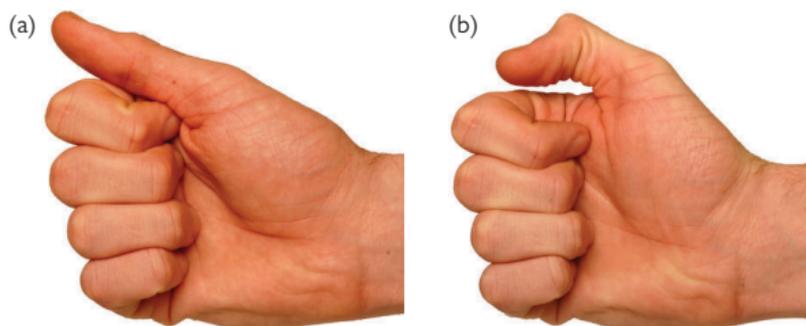


**Fig. 8.14** Froment's sign (a) normal. (b) Froment's positive. Jules Froment has the credit for this following his 1915 paper in La Press Medicale. The sign was actually described first in 1904 by Breeman.

### Box 8.10 Radial nerve palsy

*Inspection*
- Carefully inspect the upper limbs
- Radial nerve palsy is the classic cause of wrist drop.

*Tone*
- Normal

*Power*
- Weakness of triceps (elbow extension) in very proximal lesions
- Weakness of brachioradialis (elbow flexion with the forearm partially pronated)
- Test power in the extensor muscles of the wrist, fingers at the MCP joints, and thumb
  - Weakness in some or all of these
  - Sparing of finger abduction and thenar eminence muscles (thumb abduction at right angles to the palm—see median nerve palsy).
- Remember that to test finger abduction, fingers must be extended.
  - Place hand on a flat surface to test if there is finger drop.

*Reflexes*
- Absent or weak triceps reflex in very proximal lesions (and also C7 root lesions)

*Sensation*
- Sensory loss in a small area over the dorsal aspect of the hand at the anatomical snuff box
  - This is very hard to test and as there is considerable overlap in the sensory distribution to the hand, the small area supplied by the radial nerve may not be clinically detectable.

## Lower limb

*Lateral cutaneous nerve of the thigh (L2–L3)*
- Motor: none.
- Sensory: the lateral aspect of the thigh (see Fig. 10.15a).
- 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. Weakness of foot dorsiflexion and eversion. Preserved inversion. (Fig. 10.15b)
  - NB in an L5 lesion, there will be a similar deficit but also a weakness of inversion, hip abduction, and knee flexion.

*Femoral nerve (L2–L4)*
- *Motor:* quadriceps.
- *Sensory:* medial aspect of thigh and leg (see Fig. 10.15d).
- *Examining a lesion:*
  - Weakness of knee extension is only slightly affected—hip adduction is preserved
  - 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. 10.15c).
- *Examining a lesion:*
  - Foot drop and weak knee flexion
  - Knee jerk reflex is preserved but ankle jerk and plantar response 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.

**Fig. 8.15** 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. (See Box 8.11.)

A 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.
- *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.
- *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.

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**Box 8.11 Romberg's sign**

A further test of proprioception. Usually tested at the time of gait examination, as the patient will be standing at this point. When proprioception is lost in the lower limbs, patients can often stand and move normally as long as they can see the limb in question.

🔴 Perform this with care—only if you are able to prevent the patient falling and injuring themselves

- Ask the patient to stand. You stand facing them
- Ask the patient to close their eyes.

If there is loss of proprioception, the patient will lose their balance and start to fall—if so, ask them to open their eyes immediately, if they haven't already done so, and help them regain their balance without injury.

# The unconscious patient

## History
- Eye-witness account? State of clothing—loss of continence?
- Look for alert necklace/bracelet. Look in wallet, purse, etc.

## Examination
- *ABC:* (covered in detail in other Oxford Handbooks).
  - 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 four 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 four 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 and wrist, pronation of the forearm, 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 or AVPU (Boxes 8.12 and 8.13).
- *Neck:* do not examine if there may have been trauma. Test for meningeal irritation—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? 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, and added heart sounds.
- *Other bedside tests:* test urine, capillary glucose, and temperature.

**Box 8.12 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 three 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
Localizing 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 8.13 AVPU**

A much more simplified score used in rapid assessment of consciousness and often by non-specialist nurses in monitoring conscious level.

   A = Alert
   V = responds to Voice
   P = responds to Pain
   U = Unresponsive

# 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. (See Box 8.14 also.)

▶ None of the following tests should be conducted if there is suspicion of cervical injury or instability.

*Examination*
- 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.

## 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, fasciculation.
- ↓ tone.
- Flaccid weakness.
- ↓ tendon reflexes. Plantar response may be down-going or absent.

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### Box 8.14  Hemiplegia: examination findings

*Inspection*

- Are there any scars from a brain biopsy or craniotomy?

*Tone*

- Tone increased unilaterally

*Power*

- If examining the upper limbs, ask the patient to hold out their arms with their palms facing the ceiling. Ask them to close their eyes
  - Note any failure to fully raise and supinate the arm and any pronator drift with the eyes closed.
- Power is reduced in a pyramidal distribution:
  - Flexors stronger than extensors in upper limb
  - Extensors stronger than flexors in lower limb.

*Reflexes*

- Brisk tendon reflexes on the affected side
- Remember to examine for clonus which may be present on the affected side.

*Sensation*

- There may be sensory loss, usually on the side of the weakness
  - If crossed and dissociated pin-prick/vibration and joint position sense, this localizes the lesion to the brainstem.

*Gait*

- If the patient can walk, gait will be spastic on the affected side with a foot drop, difficulty flexing the knee resulting in swinging the leg round
- A pyramidal posture of the upper limb may be exaggerated.

## Motor neuron disease (MND)

See ➲ *OHCM9*. MND is a disease of the anterior horn cells of the motor pathway. It is progressive and eventually leads to respiratory failure and death. Most MND is sporadic but there are genetic forms of the disease. The most common is the autosomal dominantly inherited SOD mutation.

MND may present as 4 different phenotypes, described in Box 8.15.

*Inspection*
- Look around the patient for communication aids, walking aids, and wheelchairs.
- Look carefully at the limbs for muscle wasting and fasciculation.
- Is there a gastrostomy tube *in situ*?

*Cranial nerves*
- Patient may be dysarthric.
- Facial weakness.
- Weakness of neck flexion and extension.
- The tongue shows fasciculation.
  - Ask the patient to move the tongue quickly; a spastic tongue will not fasciculate but will be weak and move slowly.
- There is lip, tongue, and palatal weakness.
  - Ask the patient to say 'M, M, M', 'L, L, L', and 'K, K, K'.
- Jaw jerk is brisk.

*Peripheral nerves*
- On examination of peripheral tone, power and tendon reflexes, there is a mixture of upper and lower motor neuron signs.
  - Commonly muscle wasting and fasciculation with brisk reflexes and possibly extensor (up-going) plantar responses.
- Sensory examination is normal.

---

### Box 8.15  MND presentations

MND may *present* as four different phenotypes in the early stages:

*Amyotrophic lateral sclerosis*
- Clinical presentation described above
- Most common type with the classical mix of upper and lower motor neuron features.

*Bulbar presentation*
- Bulbar symptoms with preservation of limb function in the early stages; poor prognosis due to early respiratory involvement

*Progressive muscular atrophy*
- Purely lower motor neuron signs

*Primary lateral sclerosis*
- Purely upper motor neuron signs.

## Myotonic dystrophy

- The myotonic dystrophies are multisystem disorders in which myopathy and myotonia are prominent features (see Box 8.16).
- Myotonia is continued involuntary muscle contraction after voluntary effort has ceased.

*Inspection*
- Bilateral partial ptosis.
- Slack, open mouth due to jaw weakness.
- Frontal balding.
- Expressionless face.
- Cataracts (look with an ophthalmoscope).

*Tone*
- Normal.

*Power*
- Distal muscle weakness (especially hands and foot drop).
- *Myotonia:* ask the patient to squeeze their hand tightly shut and then quickly release it; note the slow relaxation of the muscles.
- *Percussion myotonia:* tap the thenar eminence with a tendon hammer; the abductor pollicis brevis will contract and very slowly relax.

*Reflexes*
- Reduced or absent.

*Sensation*
- Normal.

*Associated features*
- Cardiac conduction abnormalities and cardiomyopathy.
- Testicular atrophy.
- Endocrine disturbance (most commonly type II diabetes mellitus).
- Cognitive difficulties: intellectual and personality deterioration.
- Hypersomnolence.

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### Box 8.16 Myotonic dystrophies

*Myotonic dystrophy type 1*
- Autosomal dominant inheritance with genetic 'anticipation' (subsequent generations develop more severe symptoms earlier in life)
- It is an unstable trinucleotide CGT repeat on chromosome 19 in the myotonin protein kinase gene.

*Myotonic dystrophy type 2*
- Autosomal dominant inherited condition with a slightly different presentation to 'classic' MD
- Patients do not have facial weakness
- Limb weakness is proximal rather than distal
- Clinically milder than type 1, although patients also have cataracts and may have cardiac conduction abnormalities.

## Parkinson's disease

Parkinsonism is a pattern of symptoms comprising an akinetic–rigid syndrome. Parkinsonism has a number of causes including drug-induced and other intracranial pathologies.

Parkinson's disease (PD) is a neurodegenerative disease with loss of dopaminergic cells in the substantia nigra with Lewy body formation. PD is essentially a clinical diagnosis (currently). (See Box 8.17 for abnormal movements.)

*Inspection*
- 'Mask-like' facies with little or no expression.
- Reduced blink rate.
- Is there a head tremor?
  - yes–yes or no–no; associated with essential/dystonic tremor **not** Parkinson's disease.
- Speech is low-volume and monotonous.
- Examine for tremor with arms at rest and in posture.
  - PD tremor is asymmetrical
  - Usually said to be 'pill-rolling' and worse at rest, but can present with an asymmetrical postural tremor.

*Tone*
- Examine tone feeling for asymmetrical cogwheel rigidity.

*Power/function*
- Examine repetitive hand movements such as walking the thumb along the fingers.
  - Encourage the patient to make big, quick movements to be sure of eliciting bradykinesia if present.
- Use synkinesis (active movement of the opposite limb) to exaggerate tremor, cogwheeling or bradykinesia.

*Gait*
- Difficulty initiating gait.
- Loss of arm swing on one side.
- Shuffling gait.
- Difficulty turning.
- Unsteadiness/loss of postural reflexes.

*Extras*
- Examine pursuit and doll's eye movements.
  - Could this be a Parkinson's plus syndrome like progressive supranuclear palsy?

**Box 8.17 Abnormal movements**

- *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 of 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 as 'Brown-Sequard syndrome'.[*]
- Motor: below the level of the lesion, UMN pattern of weakness on ipsilateral side with brisk tendon reflexes.
- 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.)

*Anterior spinal artery occlusion*
- Loss of pin-prick and temperature sensation below the lesion.
- Intact light touch, vibration sense, and proprioception.

*Syringomyelia*

Longitudinal cavity (syrinx) in the central part of the spinal cord.
- Wasting of the small muscles of the hands, ulnar border of upper limb.
- Loss of pain and temperature sensation over the neck, shoulders, and arms in a 'cape' distribution (look for scars and cuts).
- Intact vibration sense, proprioception, and light touch.
- Atrophy and areflexia in the upper limbs.
- UMN weakness in the lower limbs.
- Look also for scoliosis due to weakness of paravertebral muscles.

[*] Charles Edward Brown-Sequard discovered this while studying victims of failed murder attempts amongst traditional cane cutters in Mauritius.

## Cauda equina syndrome

- The cauda equina is the name given to the descending nerve roots which extend from the termination of the spinal cord at the conus (which occurs at about L1) caudally to the final nerve root exits.
- Cauda equina syndrome is characterized by:
  - Pain in the lower back (although this is variable)
  - Bladder and bowel disturbance (as well as sexual dysfunction)
  - Saddle anaesthesia
  - Variable paralysis and sensory disturbance of the lower limbs.
- ▶ Acute herniation of a lumbar disc compressing the cauda equina and causing sphincter disturbance and paralysis is a surgical emergency and needs to be decompressed immediately to prevent long-term consequences.

*Inspection*

- Carefully inspect the lower limbs; with long-standing problems there may be muscle wasting.
  - Upper limbs will be normal.
- Note the presence of a catheter suggesting bladder dysfunction.

*Tone*

- Normal or reduced.

*Power*

- Normal in the upper limbs.
- Reduced in the lower limbs.
  - May be complete paralysis or weakness in a nerve root distribution
  - May be unilateral or bilateral.

*Reflexes*

- Tendon reflexes may be reduced or absent.
- Flexor (down-going) plantar responses.

*Sensation*

- Saddle anaesthesia (around the perineum and buttocks).
  - Compression of the sciatic nerve roots
  - May be unilateral or bilateral.

## Disturbance of higher functions

A selection of testable consequences of cortical lesions:

*Parietal lobe*

- Sensory and visual inattention.
- Visual field defects.
- Agnosias (lack of sensory perceptual abilities).
    - *Hemi-neglect*—patient ignores one side of their body
    - *Asomatognosia*—patient fails to recognize own body part
    - *Anosognosia*—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).

*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.

## Myasthenia gravis (MG)

MG is an autoimmune disease of the neuromuscular junction.

- Antibodies bind to the acetylcholine receptor, blocking acetylcholine.
- 50–60% of patients present with ocular symptoms, but only 10% of patients have isolated ocular MG.
- Disease onset is bimodal; young (15–30 years) or old (60–75 years).
- 90% develop generalized MG which can be fatal if the respiratory muscles are affected.

*Inspection*

- Look carefully at the eyes. Is there unilateral or bilateral ptosis? If so, check the pupils. In MG they are equal and reactive.
- Are the eyes conjugate as you inspect?
- Is there an eye patch indicating diplopia?
- Inspection of the limbs will usually be normal.

*Cranial nerves*

- Diplopia, worsening at the extremes of gaze.
  - Ask the patient if they have double vision and the direction of the double vision (i.e. horizontal, vertical, skewed).
- Is there any ophthalmoplegia and in which direction of gaze?
- Fatigable ptosis.
  - Hold your finger above the patient's eye-line and ask them to look up at it for 30 seconds
  - Watch their eyes
  - If fatigable ptosis and/or upgaze is present, their eyes will slowly fall back and the eyelids will begin to close.
- Facial weakness.
- Test by asking the patient to screw their eyes shut, purse their lips and open their jaw, all against resistance.
- Neck flexion and extension weakness.
- Dysarthria.
- Dysphagia.
  - Ask the patient about swallowing difficulties—do not test unless under controlled conditions.

*Power*

- Fatigable weakness of the proximal arm muscles.
  - Test power in shoulder abduction
  - Ask the patient to raise and lower their arms 20 times
  - Recheck power (will have weakened).

*Differential diagnoses*

- Unilateral ptosis and complex ophthalmoplegia: partial third nerve palsy.
- Bilateral ptosis: myotonic dystrophy.
- Bilateral facial weakness: Guillan–Barré syndrome, muscular dystrophy.
- Proximal muscle weakness: myopathy, muscular dystrophy.
- Dysarthria: stroke, motor neuron disease.

## Multiple sclerosis (MS)

- MS is a cell-mediated auto-immune condition characterized by repeated episodes of inflammation of the nervous tissue in the brain and spinal cord, causing loss of the insulating myelin sheath.
- UK prevalence is 100–140 per 100,000 with approximately 2500–3000 new diagnoses per year (or 50–60 a week).
- MS is more common in women with female-to-male ratio 2–3:1.
- MS is usually diagnosed in persons aged 15–45 years; however, it can occur in any age.
- 4% of people with a 1st degree relative with multiple sclerosis will develop the condition.
- 20% of MS patients have an affected relative.

*Examination*

- The presentation is highly variable depending on the site of the inflammation with lower and upper motor neuron signs, sensory system deficits, and cranial nerve palsies. A full and thorough systems examination is essential.

*Recognized patterns*

- *Relapsing/remitting:* symptoms come and go, 80% of patients at onset.
- *Secondary progressive:* gradually more or worsening symptoms with fewer remissions. 50% of those with relapsing/remitting MS develop secondary progressive MS during the first 10 years of the illness.
- *Primary progressive:* from the beginning symptoms gradually develop and worsen over time (10–15% of people at onset).

*Clinical symptoms and signs*

- *Motor:* weakness of variable severity including mono-, para-, hemi-, and quadri-paresis. Spasticity resulting in spastic gait. Facial myokymia (unusual fine undulating wave-like facial twitching).
- *Sensory:* dysaesthesic pain, paraesthesia, numbness, Lhermitte's sign, severe decrease or loss of vibratory sense and proprioception, positive Romberg's test.
- *Cranial nerves:* CN V most frequently involved followed by VII, III, and VIII. Isolated cranial nerve palsies are not frequent. Combined cranial nerve palsies are rare in MS. Symptoms might include trigeminal neuralgia. Signs such as taste and smell dysfunction frequently found if specifically looked for.
- *Cerebellar signs:* incoordination including dysdiadochokinesis, failure of heel–shin test, ataxic gait, scanning speech, loss of balance.
- *Ocular:* reduced visual acuity, colour blindness, complete loss of vision (1 in 35 cases), retrobulbar pain, blurred vision, diplopia, nystagmus, internuclear ophthalmoplegia, central scotomata and other visual field defects, oscillopsia, relative afferent pupillary defect (Marcus Gunn pupil), optic disc pallor and atrophy, Uhthoff's phenomenon (worsening of vision in optic neuritis during a fever, in hot weather, or after exercise).
- *Other:* neuropathic and musculoskeletal pain; bladder, bowel, and sexual dysfunction; fatigue; cognitive and emotional problems; heat sensitivity due to loss of thermoregulation manifesting as excess sweating, etc.

**See also:**

More information regarding the presentation and clinical signs of neurological diseases to aid preparation for OSCE-type examinations and ward rounds can be found in the *Oxford Handbooks Clinical Tutor Study Cards*.

*'Medicine' Study Card set:*

- Proximal myopathy
- Motor neuron disease
- Myasthenia gravis
- Cervical myelopathy
- Median, ulnar, and radial nerve palsies
- Wasting of the small muscles of the hand
- Syringomyelia
- Polymyositis
- Parkinson's disease
- Friedreich's ataxia
- Charcot–Marie–Tooth disease
- Subacute combined degeneration of the cord
- Brown-Sequard syndrome
- Tabes dorsalis
- Cerebellopontine angle syndrome
- Wallenberg's syndrome
- Cranial nerve palsies
- Bulbar palsy
- Pseudobulbar palsy.

# 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 also 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. 247; ➲ MMSE p. 513)—but remember no half marks! Note that the original format of the MMSE is now copyrighted, so may be less readily available.
- *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 and how they assess patients.

**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.

# The eyes

# Important symptoms

The ophthalmic history has extra sections: past ophthalmic history and family ophthalmic history (see Boxes 9.1 and 9.2). Specific symptoms related to the eyes include:

## Redness

Ask specifically about:
- Associated factors (discharge, watering).
  - Mucopurulent discharge suggests bacterial infection.
- Pain (sharp, dull).
  - Aching: anterior segment inflammation, acute glaucoma.
- Foreign body sensation.
  - Epithelial defect, foreign body.
- Photophobia.
- Blurred vision.
- Contact lens wear.
- History of trauma.

## Diplopia

See Box 9.3.
- Is it actually double or blurred?
  - Patients complain of 'double vision' without true diplopia.
- Duration/age of onset.
- Monocular or binocular?
- Variable or constant?
- Horizontal, vertical, or mixed?
  - Horizontal: III, VI nerve palsies
  - Vertical: IV nerve palsy
  - Variable: myasthenia gravis
  - Progressive: thyroid eye disease.

## Visual loss

- Unilateral or bilateral?
  - Uniocular: suggestive of ocular or optic nerve pathology
  - Binocular: lesions at or posterior to the optic chiasm.
- Extent.
  - Severe visual loss can occur with optic neuropathies
  - Unilateral, segmental visual loss: retinal disorders such as retinal detachment and branch retinal vein occlusion.
- Speed of onset.
  - Sudden suggests ischaemic causes
  - Gradual is more typical of compressive causes
  - Progression over a few hours to days can occur in optic neuritis.

## Colour vision abnormalities

- Often a feature of optic nerve disease.
- Congenital red–green colour discrimination deficiency is seen in 5–8%.
- Blue–yellow is rarely due to congenital colour deficiency so a causation should be sought.

### Flashing lights

- 'Photopsia' is the perception of light in the absence of a light stimulus.
- Monocular or binocular?
  - Monocular is typically due to vitreoretinal pathology
  - Binocular is usually a cortical phenomenon.

*Causes:*

- Mechanical retinal stimulation (posterior vitreous detachment, tears) or external compression.
- Subretinal pathology (choroidal neovascularization, uveitis, choroidal tumours).
- Cortical ischaemia (migraine or TIA).
- Visual hallucinations.

### Other symptoms

Ask also about:

- Glare.
- Haloes or starbursts.
- Floaters.
- Night-driving problems.
- Increased myopia.

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**Box 9.1 Some causes of pain**

- Gritty, sharp pain: corneal epithelial defect (abrasion, keratitis)
- Ache, photophobia: iritis
- Pain on eye movement: optic neuritis
- Scalp tenderness, jaw claudication: temporal arteritis
- Nausea, vomiting: acute angle closure glaucoma, raised intracranial pressure (papilloedema).

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**Box 9.2 Some causes of floaters**

- Weiss ring following posterior vitreous detachment
- Vitreous condensation
- Vitreous haemorrhage
- Liberated pigment cells associated with retinal tears
- Inflammatory cells
- Tumour cells
- Asteroid hyalosis.

# The rest of the history

### Systems enquiry

Use this to explore symptoms that may point to a systemic disease with ocular manifestations

- Multiple sclerosis: weakness, paraesthesia bladder dysfunction.
- Thyroid eye disease: heat intolerance, weight loss, irritability, anxiety.
- Myasthenia gravis: dysphagia, weakness worse at the end of the day.
- Embolic disease: atherosclerotic disease, arrhythmias.
- Acoustic neuroma: hearing loss, tinnitus, balance problems.
- Rheumatologic and collagen vascular diseases: arthralgia, rashes.

### Past medical history

- Diabetes.
- Hypertension.
- Atopy (allergic conjunctivitis).
- Rheumatologic disease (dry eye, corneal melt, scleritis).
- Neurological diseases (VII palsy, exposure keratopathy).
- Metabolic disease (hypercalcaemia).

### Past ocular history

- Past ophthalmic surgery.
  - Intraocular (endothelial dysfunction)
  - Refractive (post-laser-assisted stromal *in situ* keratomileusis [LASIK], dry eye, flap dehiscence).
- Does the patient wear glasses?
- Does the patient wear contact lenses?
  - Type
  - Overnight wear?
  - Cleaning regimen (including use of tap water)?
  - Swimming with lenses in?
- Trauma (physical, chemical, radiation).
- Infection.
  - Herpes simplex keratitis
  - Herpes zoster ophthalmicus.

### Drug history

- Topical steroid (cataract, glaucoma, herpetic geographic ulcer).
- Toxicity to preservatives/drop allergy.
- Ethambutol, isoniazid, amiodarone, and ciclosporin can cause optic neuropathy.
- Recreational drug use is important, particularly in atypical pupil abnormalities.

### Family history

- Family history of multiple sclerosis is common in patients with optic neuritis.
- Contact with infection, conjunctivitis.
- Inherited corneal dystrophies.
- Glaucoma.

### Family ophthalmic history
- Ask about any eye diseases which run in the family (e.g. glaucoma, inherited retinal dystrophies).

### Social history
Ask especially about:
- Occupation and hobbies: this is very important and needed to understand patient's visual requirements (sports, driving, reading).
- Country of previous residence (sun exposure, poor sanitation).
- Lead and carbon monoxide can cause optic nerve dysfunction.
- Possibility of sexually transmitted diseases (e.g. syphilis, HIV/AIDS).

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**Box 9.3  Some causes of diplopia**

*Horizontal: VI nerve palsy*
- Impairment of abduction due to lateral rectus palsy
- Horizontal diplopia worse on looking towards the side of the lesion.

*Vertical: IV nerve palsy*
- Diplopia worse on looking down due to superior oblique palsy
- Ipsilateral hypertropia, worse on looking away from the side of the lesion and on ipsilateral head tilt
- Reduced depression in adduction.

*Mixed: III nerve palsy*
- May be partial or complete
- Pupil involving or sparing, $\pm$ ptosis
- Palsy of all extraocular muscles except lateral rectus and superior oblique.

*Mechanical*
- Ductions and versions equally reduced
- Causes: thyroid eye disease, trauma (orbital wall/floor fracture), idiopathic orbital inflammatory disease, tumour.

*Myasthenia gravis*
- 'The great masquerader'
- Intermittent diplopia, variable severity
  - Often worse end of the day/after exercise
  - $\pm$ ptosis.
- Ice-pack test: recheck ptosis after placing ice pack on closed eyelid for 2 minutes. Positive if significant improvement of ptosis (>2mm).

*Decompensating phoria*
- Intermittent but with constant pattern

*Monocular*
- High refractive disparity between eyes (anisometropia, astigmatism)
- Corneal opacities or ectasias
- Lens subluxation
- Iris defects: trauma, laser peripheral iridotomies.

# Visual acuity

It is important to remember the path light takes that enables vision. Any interruption to this pathway can lead to loss of vision (see Box 9.4).

## Visual axis – applied anatomy

With an understanding of the anatomy, defects in the visual field enable the localization of a lesion within the visual pathway.

- Light passes through the cornea, anterior chamber, pupil, lens, and vitreous chamber before hitting the retina.
- 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 (decussate). 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 retina receives input from the temporal part of the visual field in each eye, whilst the temporal half of the retina receives input from the nasal half of the eye.
- 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.

## Testing visual acuity

*Snellen chart* (Fig. 9.1)

In good light conditions, stand the patient 6m from a Snellen chart.

- Test each eye in turn unaided or with the glasses they normally use for distance vision.
  - Repeat the test with a pinhole. Any improvement in vision implies an uncorrected refractive error (rather than ocular pathology).
- Record the lowest line that can be read (allow two errors per line).
  - The number associated with the letters indicates the distance from which a person with normal sight would be expected to read.
- Record the visual acuity as the distance from the chart followed by the number at the lowest letters read.
  - For example, if the patient has read the '36' line from a distance of 6 metres, the visual acuity is '6/36'.

*Poor vision*

If the patient is unable to see the Snellen chart at all, see if they can:

- Count fingers (CF).
- See hand movements (HM).
- See light (PL).
  - If the patient is unable to see light then record as 'NPL' (no perception of light).

**Box 9.4 Some causes of visual loss**

*Cornea*
- Dry eyes, corneal abrasion, corneal ulcer, herpetic keratitis, corneal oedema (acute angle closure glaucoma), keratoconus

*Anterior chamber*
- Iritis, hyphaema, hypopyon

*Lens*
- Cataract

*Vitreous chamber*
- Vitreous haemorrhage, vitritis

*Retina*
- Branch/central artery or vein occlusion, retinal detachment, macular degeneration, macular oedema, hypertensive retinopathy

*Optic nerve*
- Optic neuritis, ischaemic optic neuropathy, papilloedema

*Optic chiasm*
- Pituitary tumour, meningioma

*Optic tract*
- CVA, tumour

*Occipital cortex*
- CVA, tumour.

**Skills station 9.1**

*Instruction*
Examine this patient's optic nerve function.

*Model technique*
- Clean your hands
- Introduce yourself
- Explain the purpose of examination, obtain informed consent
- Sit facing the patient
- Measure visual acuity for distance and near
- Measure colour vision (e.g. Ishihara colour plates)
- Check for an RAPD (relative afferent pupillary defect)
- Examine the optic disc looking for any disc swelling, haemorrhage, atrophy, collateral vessels, and cupping
- Perform perimetry (confrontation, manual, automated) to detect any characteristic field defects
- Thank the patient.

**Fig. 9.1** Schematic example of a Snellen chart (reproduced with permission from *Oxford Handbook of Ophthalmology*).

*LogMAR chart*

This is an alternative method of checking distance vision. It has advantages over Snellen charts overcoming the 'crowding' phenomenon by having five letters on each line with equal spacing (see Fig. 9.2).



**Fig. 9.2** Schematic example of LogMAR chart (reproduced with permission from *Oxford Handbook of Ophthalmology*).

# Visual fields

### Testing the visual field

*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.
  - Ask the patient to look directly at you ('look at my nose')
  - Ask 'is any part of my face missing?'
  - 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
  - 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 report seeing only one hand when both are moving (compare with sensory inattention).

### Testing each eye

- In the same position as above, ask the patient to cover their right eye while you close your left and ask them to look into your right eye.
  - (If you were now to trace the outer borders of your vision in the air halfway between yourself and the patient, it 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
  - 🔔 Make sure they keep looking at your right eye
  - 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 two circles representing the patient's visual fields as shown in Fig. 9.3.

## Common visual field defects

Compare the defects below with the corresponding number on Fig. 9.3.

- *Tunnel vision:* a constricted visual field (glaucoma or retinal damage).
  - 'Tubular' vision is often functional.
- *Enlarged blind spot:* caused by papilloedema.
- *Unilateral visual loss:* (1) blindness in one eye caused by devastating damage to the eye, its blood supply, or optic nerve.
- *Scotoma:* a 'hole' in the visual field (macular degeneration, vascular lesion or toxins).
  - If bilateral, may indicate a very small defect in the corresponding area of the occipital cortex (e.g. 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 (rare).
- *Homonymous hemianopia:* (3) 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 (the macula) is spared, the lesion is likely in the optic radiation; without macula sparing, the lesion is in the optic tract.
- *Homonymous quadrantanopia:* corresponding quarters of the vision are lost in each eye.
  - Upper quadrantanopias (4) suggest a lesion in the temporal lobe
  - Lower quadrantanopias (5) suggest a lesion in the parietal lobe.
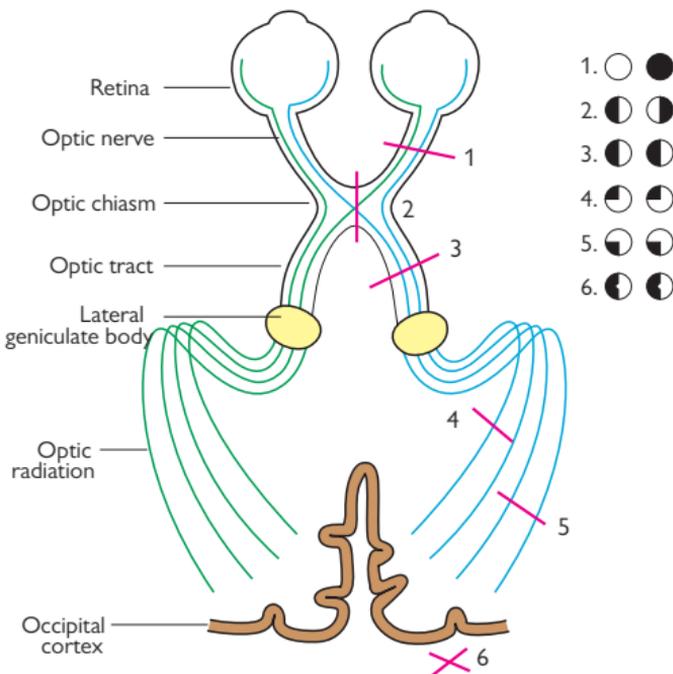


**Fig. 9.3** Representation of the visual pathway from the retina to the occipital cortex showing visual field loss according to site of lesion.

# The pupils

Pupillary abnormalities include irregular pupil size and impaired reflexes to light and accommodation. (See Boxes 9.5 and 9.6.)

## Applied anatomy

Pupil size is controlled by the autonomic nervous system.

- Pupil constriction (miosis) via parasympathetic nerves through innervation of the sphincter pupillae.
- Pupil dilation (mydriasis), via the dilator pupillae muscle is under sympathetic control.

*Light reflex (parasympathetic system)*

- Nasal retinal fibres cross at the chiasm and connect to the contralateral pretectal nucleus, which innervates both Edinger–Westphal nuclei (see Fig. 9.4).
- Temporal retinal fibres connect to the ipsilateral pretectal nucleus, which again innervates both Edinger–Westphal nuclei.
  - This quadruple innervation ensures that both pupils constrict when light is shone in either eye.
- The parasympathetic preganglionic motor fibres connect the Edinger–Westphal nucleus to the ciliary ganglion.
- The postganglionic fibres connect the ciliary ganglion to the sphincter pupillae via the short ciliary nerves.

*Relative afferent pupillary defect (RAPD)*

- Light shining into an eye will lead to constriction of the ipsilateral pupil, the '*direct*' reflex, but also constriction of the contralateral eye, the '*consensual*' reflex.
- If there is a defect in the afferent pathway (e.g. optic nerve damage), shining a light into the affected eye will cause both pupils to initially dilate.
- An RAPD can still be detected if one eye is pharmacologically dilated. One should observe the undilated pupil to assess whether an initial dilatation occurs.

*Near reflex (parasympathetic system)*

Looking at a near target activates the 'near reflex' which comprises accommodation, convergence, and miosis. The final pathways are as for the light reflex, e.g. third nerve, ciliary ganglion, and short ciliary nerves.

*Sympathetic system*

This involves three neurons:

- *Central:* descends from posterior hypothalamus through the ipsilateral brainstem to the ciliospinal centre of Budge in the intermediolateral horn of the spinal cord between C8 and T2 (see Fig. 9.5).
- *Preganglionic:* ascends close to the apical pleura to terminate in the superior cervical ganglion in the neck.
- *Postganglionic:* ascends along internal carotid artery, joining the ophthalmic division of the trigeminal nerve in the cavernous sinus. It terminates in the dilator pupillae muscle via the long ciliary nerve.

### Testing for RAPD

- With the patient sitting opposite, shine a pen-torch into one eye for 2–3 seconds.
- Quickly swing the light into the other eye.
- Watch for an initial pupillary dilatation (RAPD).

### Testing the accommodation reflex

- With the patient sitting opposite you, hold up an accommodative target (e.g. page of a book) approximately 30cm in front of the patient.
- Watch for pupil constriction.
- If in doubt, ask the patient to look at something distant over your shoulder and then at the page again.

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#### Skills station 9.2

*Instruction*

Examine this patient's pupils.

*Model technique*

- Clean your hands
- Introduce yourself
- Explain the purpose of examination, obtain informed consent
- Sit facing the patient
- Ask the patient to fixate on a distant target
- Look for any anisocoria (difference in pupil size), heterochromia (difference in iris colour), ptosis, or ocular deviation
- Dark response
  - Measure pupil sizes with ruler and then dim room lights and repeat measurements.
  - If anisocoria is present, the larger pupil in bright conditions is likely to be the abnormal one and vice versa
- Check direct pupil light response in each eye
  - Shine a torch and observe the pupil reaction in that eye
- Check consensual pupil response
  - Shine a torch into one eye and observe the pupil reaction in the other eye.
- Check for a relative afferent pupillary defect (RAPD)
- Check accommodation reflex
- Thank the patient and help them re-dress as necessary.

*In a full examination:*

- If possible, examine patient on slit lamp looking for vermiform iris movements (in Adie's pupil)
- Perform eye-movements examination looking for associated nerve palsies
- Perform full neurological examination, particularly checking for decreased deep tendon reflexes (in Holmes–Adie–Moore syndrome).

## Pupil abnormalities

*Relative afferent pupillary defect (RAPD)*

This results from lesions in the anterior visual pathway.

🛈 Corneal opacities or cataract do not cause a RAPD.

Causes include:
- Optic neuropathy (e.g. optic neuritis, compressive lesions).
- Gross retinal pathology (e.g. central retinal vein occlusion (CRVO), retinal detachment).
- Optic chiasm and tract lesions (infarcts, demyelination).

*Horner's syndrome*

Oculosympathetic palsy (interruption of the cervicothoracic sympathetic chain at a 1st, 2nd, or 3rd order neuron level). Multiple causes, depending on site of lesion.
- Unilateral mild ptosis.
- Ipsilateral anhydrosis (decreased sweating).
- Ipsilateral iris heterochromia (different or lighter coloured iris if the lesion is congenital or long-standing).
- Mild miosis.
- Normal or slight delay of pupillary dilatation.
- No relative afferent pupillary defect.

*Argyll Robertson pupil*

Caused by neurosyphilis.
- Constricted and irregular pupils (asymmetric).
- No reaction to light.
- Brisk constriction to accommodation.

*Holmes–Adie–Moore syndrome (or Adie's pupil)*

Denervation of the sphincter pupillae and ciliary muscles, probably following viral illness. Usually seen in middle-aged females.
- Dilated pupil (mydriasis).
  - Anisocoria (difference in pupil size) greater in the light.
- Poor response to light and accommodation.
- Deep tendon reflexes may be reduced or absent.

*Pupil involving 3rd nerve palsy*

Causes include subdural haematoma with uncal herniation, posterior communicating artery aneurysm, tumour, vasculitis.
- Fixed dilated pupil, ptosis, eye held 'down and out' with restricted eye movements.

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**Box 9.5 Light-near dissociation**

Absent or sluggish response to light but normal accommodation reflex
- Afferent conduction defect (e.g. optic neuropathy)
- Holmes–Adie–Moore pupil
- Argyll Robertson pupil
- Aberrant third nerve regeneration
- Myotonic dystrophy
- Parinaud dorsal midbrain syndrome.

## Box 9.6 Some other causes of abnormal pupils

*Pupil is constricted*
- Pharmacological (alcohol, opiates, antipsychotics)
- Chronic Adie's pupil
- Iritis.

*Pupil is dilated*
- Pharmacological (atropine, LSD, psilocybin mushrooms, cocaine, amphetamines, SSRI antidepressants)
- Iris trauma.



**Fig. 9.4** Light reflex pathway. Reproduced with permission from *Training in Ophthalmology* by Sundaram et al.



**Fig. 9.5** Sympathetic pupil pathway. Reproduced with permission from *Training in Ophthalmology* by Sundaram et al.

# Eye movements

Eye movements are controlled by three cranial nerves: oculomotor (III), trochlear (IV), and abducens (VI). There are also supranuclear centres that control conjugate eye movements or 'versions', where both eyes move in synchrony (see Fig. 9.6 and Box 9.7).

## Applied anatomy

*Third (III) nerve: oculomotor*
- *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.)
  - The III nerve has two motor nuclei: the main motor nucleus and the accessory parasympathetic nucleus (Edinger–Westphal nucleus).
- *Autonomic:* parasympathetic supply to the constrictor (sphincter) pupillae of the iris and ciliary muscles.
- The nuclear complex is in the midbrain at the level of the superior colliculus.

*Fourth (IV) nerve: trochlear*
- *Motor:* contralateral superior oblique.
  - It is the thinnest cranial nerve with the longest intracranial course and the only cranial nerve to exit from the dorsal brainstem.
- Nucleus lies inferior to the III nerve nuclei, at the level of the inferior colliculus in the midbrain. It receives input from the vestibular system and medial longitudinal fasciculus (MLF).

*Sixth (VI) nerve: abducens*
- *Motor:* ipsilateral lateral rectus muscle.
- The nucleus lies beneath the 4th ventricle. It connects with the nuclei of the III and IV cranial nerves through the medial longitudinal fasciculus.

*Horizontal eye movements*
- Horizontal eye movements are controlled by the horizontal gaze centre in the pontine paramedian reticular formation (PPRF). This connects to the ipsilateral sixth nerve nucleus to abduct the eye and also the contralateral third nerve nucleus via the contralateral median longitudinal fasciculus (MLF) to adduct the contralateral eye.

*Vertical eye movements*
- Vertical eye movements are controlled by the vertical gaze centre in the rostral interstitial nucleus of the MLF.

**Fig. 9.6** The nine positions of gaze, including looking straight ahead (neutral position).

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**Box 9.7 Normal and abnormal eye movements**

- *Ductions:* normal monocular movements including adduction, abduction, elevation, depression, intorsion, and extorsion
- *Versions:* normal binocular, conjugate eye movements where both eyes move in the same direction
- *Vergences:* normal binocular eye movements in which the eyes move synchronously in opposite directions (e.g. convergence)
- *Phorias:* eye deviations which are not obvious during normal binocular vision (when the retinal images are 'fused'). They can 'decompensate' when the patient is tired and become obvious or can be seen when binocular vision is prevented (e.g. covering one eye)
- *Tropias:* obvious deviations. For example:
  - Esotropia = inward deviation
  - Exotropia = divergent squint.

## Examining eye movements

*Observe*
- Position yourself opposite the patient and assess their head posture.
- Look for ptosis (drooping of the eyelid).
- Shine a pen-torch into each eye from a central position in front of the nose and look for asymmetry of the corneal reflections.
  - Both should be approximately central. If the reflection is nasal to the pupil the eye is exotropic. If it is temporal it is esotropic.

*Cover test*

This is used to assess for phorias (see Box 9.3). These are eye deviations which are compensated for during normal binocular vision.
- Ask the patient to look at a distant target.
- Cover each eye in turn with your hand and watch for movement of the other eye. If the non-covered eye moves to take fixation, there is a phoria and the direction of movement gives a clue as to the type:
  - Inward movement = exotropia (eye had been outward)
  - Outward = esotropia (eye had been inward)
  - Down = hypertropia (eye had been up)
  - Up = hypotropia (eye had been down).

*Uncover test*
- Remove the cover and watch for movement in the eye that is revealed.
  - See cover test above for interpretation of the movements.

*Alternate cover test*
- Repeatedly cover each eye for a few seconds moving quickly between each eye so one is always covered.
  - Watch for any eye deviation and recovery.

*Voluntary eye movements*
- Ask the patient if they have any diplopia in primary position (looking straight at you).
- Sitting opposite the patient, ask them to follow a target (e.g. your finger tip or pen-torch) without moving their head.
  - Sometimes, your hand on their chin helps to hold the head still.
- Examine the nine positions of gaze as in Fig. 9.6.
  - Avoid the extremes of gaze
  - Ask if they have double vision in each position
  - Watch for failure of eye movements or abnormal movements (e.g. nystagmus)
  - Perform a cover test in each position.

*Saccades*
- Hold two targets either side of the patient (your thumb of one hand and a finger of your other hand works well).
- Ask the patient to look rapidly between the two targets.
  - Often a quick demonstration helps
  - Movements should be accurate, smooth, and rapid.
- Repeat for the vertical meridian (holding targets above and below the midline).

*Convergence*
- Hold a target approximately 1m in front of the patient and ask them to fix on it.
- Slowly bring the target towards the patient and watch their eyes.
  - The eyes should converge slowly, symmetrically, and smoothly.

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**Skills station 9.3**

*Instruction*

Examine this patient's eye movements.

*Model technique*
- Clean your hands
- Introduce yourself
- Explain the purpose of examination, obtain informed consent
- Sit facing the patient
- Ask the patient if they have any visual problems
- Perform a brief examination of visual acuity and visual fields
  - Asking a blind patient to follow your finger will not work!
- Ask the patient to look straight at you
- Make note of the patient's head position and any evidence of ptosis
- Ask the patient to look at your nose
  - Look for any obvious asymmetry in eye position (strabismus)
- With the patient's eyes in neutral position, perform the cover–uncover test
- Examine voluntary eye movements
- Perform the cover–uncover test in each of the nine positions of gaze if necessary
- Test saccadic eye movements
- Test convergence
- Thank the patient.

# III, IV, and VI palsies

## Oculomotor (III) palsy

*Clinical features*
- Ptosis.
- Affected eye is exotropic and hypotropic ('down and out').
- Ophthalmoplegia in all directions other than laterally and inferiorly (Fig. 9.7).
- Mydriasis (pupillary dilation—variable).
  - The fibres which control pupil constriction lie near the surface of the nerve and are supplied by surface blood vessels, thus are vulnerable to compressive lesions. Pupil dilatation distinguishes 'medical' third nerve palsy from 'surgical' (compressive) nerve palsy.

*Causes*
- 20–45% microvascular causes (with diabetes and hypertension).
- 15–20% intracranial aneurysms (often posterior communicating artery).
- Trauma.
- Tumours.
- Demyelination.
- Vasculitis.
- Congenital.

## Trochear (IV) palsy

*Clinical features*
- Vertical diplopia (worse on downgaze).
- Slight external rotation of affected eye (head may be tilted to opposite side to compensate).
- Hypertropia (eye sits higher than contralateral side).
  - Worse on contralateral gaze and ipsilateral head tilt.
- Limitation of depression in adduction.

*Causes*
- 30–40% due to head trauma.
- 20% microvascular disease (often improves within 3–4 months).
- Congenital (common, although not usually symptomatic until adult life).
- Others: haemorrhage, infarction, demyelination, tumours, infection.

## Abducens (VI) palsy

*Clinical features*
- Inability to abduct the affected eye.
- Diplopia (worse when looking in direction of paretic muscle and worse for distance than near).

*Causes*
- Microvascular lesions (most common).
- Other causes: demyelination, infarction, raised intracranial pressure, tumours, meningeal infection, aneurysm (basilar artery), inflammatory processes.

**Fig. 9.7** A patient with right oculomotor (III) palsy, shown in five directions of gaze.

## Combined nerve palsies

### The cavernous sinus

All three nerves involved in oculomotor control, 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.

### Superior orbital fissure

The superior orbital fissure transmits all the nerves supplying the extra-ocular 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.

# Other eye movement disorders

### Internuclear ophthalmoplegia (INO)

An interruption of the medial longitudinal fasciculus (MLF) that interconnects the nuclei of cranial nerves III and VI on opposite sides.

The patient complains of horizontal diplopia due to impaired adduction on the affected side.

*Unilateral lesions*
- Impaired adduction in the ipsilateral eye.
- Nystagmus is often seen in the abducting eye (Box 9.8).

*Bilateral lesions*
- May appear bilaterally exotropic (eyes look laterally).
- Vertical nystagmus.
- Impaired visual pursuit.
- Convergence is intact in posterior lesions and absent in anterior and mesencephalic lesions.

### Parapontine reticular formation (PPRF) lesions

The PPRF is responsible for conjugate movements in horizontal gaze.
- Horizontal gaze paresis towards the affected side.
- Preservation of vertical gaze.

*Causes:* vascular disease, demyelinating disease, tumour.

### 'One and a half syndrome'

INO with the addition of a lesion in the PPRF or the ipsilateral VI nucleus.
- Conjugate gaze palsy to the side ipsilateral to the lesion (this is the 'one') and an inability to adduct the eye on the affected side when looking contralaterally (this is the 'half').

### Parinaud's (dorsal midbrain) syndrome

- Impaired upgaze in both eyes giving convergence, retraction of the globe into the orbit, and nystagmus.
- Light-near dissociation.
  - The near response is intact but the light response is slow.
- Lid retraction (Collier's sign).

*Causes:* demyelination, arteriovenous malformation, tumour, enlarged 3rd ventricle, vascular disease affecting the midbrain, meningitis.

### Box 9.8 Nystagmus

Nystagmus is involuntary rhythmic oscillation of the eyes and may have a number of appearances

- Direction: vertical, horizontal, upbeat, downbeat, rotatory
- Speed of away movement: slow, fast
- Speed of corrective movement
- *Pendular nystagmus:* oscillation is the same speed in both directions
- *Jerk nystagmus:* different speeds in different directions. The 'direction' of the nystagmus is determined by the fast phase.

*Sensory deprivation nystagmus*

- A pendular type of nystagmus due to a lack of visual stimulus
- Seen in a number of conditions including: congenital cataract, ocular albinism, aniridia, congenital optic nerve abnormalities.

*Motor nystagmus*

- Usually present at birth or very early in life. Not present whilst asleep. Decreases with convergence.

*Latent nystagmus*

- Bilateral jerk nystagmus which is only present when one eye is covered
- The fast phase of the jerk is away from the occluded eye.

*Dissociated nystagmus*

- Different pattern of nystagmus seen in the two eyes
- Causes include: INO, posterior fossa pathology.

*Downbeat nystagmus*

- A jerk nystagmus. Fast phase is down, slow upbeat. Null point in upgaze
- Associated with diseases of the craniomedullary junction: MS, stroke, syringomyelia, Arnold–Chiari malformation, lithium toxicity.

*Gaze-evoked nystagmus*

- Seen in particular directions of gaze and not in the primary position
- If physiological, it should be fatiguable and symmetrical
- Pathological causes: drugs, lesions of the brainstem and posterior fossa.

*Upbeat nystagmus*

- Seen in the primary position of gaze
- Causes: Wernicke's encephalopathy, drugs, lower brainstem lesions.

*Vestibular nystagmus*

- Another type of jerk nystagmus secondary to vestibular disease Often a rotary component. The fast phase away is from the side of the lesion. Check for associated tinnitus, vertigo, or hearing loss.

# Anterior segment examination

This is ideally performed with a slit lamp, but all the findings should be visible with an ophthalmoscope.

Set the dial to +10 to focus on the anterior segment.

Even without an ophthalmoscope you can gain a lot of information using a pen-torch and a blue filter (Box 9.9).

## Examination routine

- Check for RAPD (see ).
- General inspection.
  - General habitus
  - Facial asymmetry
  - Skin lesions (e.g. herpes).
- Lids.
  - Position: ptosis, entropion (inverted lid), ectropion (everted lid)
  - Examine the lashes, looking for blepharitis or other lesions
  - Look for lumps, erythema, swelling
  - Evert the upper and lower lids, looking at the conjunctiva and fornices. Look especially for foreign bodies, papillae, follicles, symblepharon (partial or complete adhesion of the palpebral conjunctiva of the eyelid to the bulbar conjunctiva of the globe).
- Conjunctiva.
  - Look for: hyperaemia, haemorrhage, chemosis (oedema), lumps, abrasions, foreign bodies, pterygia (benign growth of conjunctiva).
- Sclera.
  - Look for: colour, hyperaemia, swelling.
- Cornea.
  - Raise the lid to examine entire cornea
  - Instil a drop of 2% fluorescein and look for epithelial defects which will fluoresce green under a blue light.
- Anterior chamber.
  - Gauge the depth
  - Check for cells, fibrin, flare, blood (hyphaema), pus (hypopyon).
- Iris.
  - Note: colour, shape, movement, atrophy.
  - Use retroillumination to check for transillumination defects.
- Lens.
  - Look for: cataract, intraocular lens. Note position, movement.
- Anterior vitreous.
  - Focus behind the lens and ask the patient to look up, down, and then straight ahead to view the vitreous
  - Check for cells (small white deposits), 'tobacco dust' (pigment indicative of retinal tear), blood.

**Box 9.9 Using a slit lamp**

Only ophthalmologists will be expected to be proficient with a slit-lamp microscope. We include this for those that wish to impress:

- Check the slit lamp is plugged in and turn the switch on the left-hand side to get a light beam
  - If it is not working, check the bulb.
- Adjust the eye pieces to your interpupillary diameter (IPD) and dial in your own refraction for each eye piece
- Choose the light beam from the switch at the top (bright light, bright light with heat filter, normal light, green light, blue light)
  - Start with the normal light with a thin beam and then use the bright light with filter when required
  - Adjust the width, height, angle, and orientation of the light beam.
- Adjust the height of the patient's chair and your own to ensure they are at comfortable heights for you both
  - The height of the slit lamp can be adjusted with a lever under the table
- Ask the patient to place their chin in the rest and press their forehead against the bar. Ensure it is a comfortable height for them
- Adjust the height of the chin rest to align the patient's eyes with mark on the slit lamp
- Move the slit lamp slowly towards the patient and use the joystick for fine adjustments to get the eye in focus
  - You can twist the joystick to raise or lower the light beam
- Examine the anterior segment as on ⟳ p. 330.
  - You can increase the magnification by turning the switch beneath the eye pieces.

*Direct illumination*

- Using a coaxial light (angling the light) will allow greater evaluation of the cornea and anterior segment
- Check for cells.
  - Use a coaxial light and focus on the centre of the anterior chamber with the blackness of the pupil in the background
  - Cells will look like small dust particles visible in the light beam rising and falling in the aqueous flow.

*Retroillumination*

- Shorten the bright light beam and direct straight ahead through the pupil onto the retina. As the light is reflected you can assess lens opacities (dilated pupil) or iris transillumination defects.

# Posterior segment examination

The posterior segment is the part of the eye deep to the lens and includes the vitreous, optic disc, and the retina (see Box 9.10).

Direct ophthalmoscopic examination of the fundus is a vital part of any neurological examination but often avoided by students as it is considered difficult.

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.

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.

🔴 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.

## Using an ophthalmoscope

- Introduce yourself, explain the procedure to the patient, and gain informed consent.
- Ensure the room is dimly lit and, ideally, sit opposite the patient.
  - This can be performed with the patient lying down.
- Familiarize yourself with the ophthalmoscope. Choose a large-aperture light and adjust the brightness so as not to dazzle your patient.
- Ask the patient to focus on a distant object and keep their eyes still (relaxes accommodation as much as possible).
- Set the refraction to +10.
- 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.
- Approach the patient from 15 degrees keeping in mind the angle the optic nerve enters the eye.
  - Be careful not to block the view of their other eye which maintains fixation and allows them to keep their eyes still.
- With the +10 setting you can examine the anterior segment.
  - By gradually ↓ the power of the lens you can examine the cornea, iris, and lens in turn.
- Ask the patient to look up, down, and then straight ahead to view the vitreous.
- As you approach, dial the refraction to 0 or to your own refraction.
- Find a blood vessel and adjust the focus as necessary. Follow the blood vessel, as it increases in diameter to the optic disc. If you don't find it try following it in the other direction.

- Examine the optic disc, noting:
  - Cup:disc ratio
  - Colour
  - Shape
  - Margin
  - Rim
  - Abnormal vasculature.
- Examine all 4 quadrants.
- Examine the macula.
  - Ask the patient to look directly at the light.

---

**Box 9.10 What to look for on ophthalmoscopy**

*Vitreous*
- Cells (appear as small white particles)
- Pigment
- Blood
- Asteroid hyalosis (calcium deposits in the vitreous)
- Weiss ring (posterior vitreous detachment).

*Macula*
- Dot/blot haemorrhages
- Microaneurysms
- Exudates
- Cotton-wool spots
- Oedema.

*Vessels*
- Venous beading
- Venous loops
- Intraretinal microvascular abnormalities (IRMAs)
- New vessels (very thin, tortuous vessels)
- 'Silver wiring' (arteries appear to have a shiny, silver strip)
- Arteriovenous nipping (veins are 'pinched' as arteries pass over)
- Macroaneurysm.

*Peripheral retina*
- Degenerations
- Tears
- Retinal detachment
- Pigmentation
- Laser/cryotherapy scars
- Chorioretinal scars
- Tumours.

# Eye trauma

Being faced with a patient who has sustained trauma can be disconcerting but by maintaining a systematic approach you will not miss any important signs. It is critical in cases of assault that you document your findings accurately with drawings/images as cases may involve legal proceedings (see Box 9.11).

## The history

Take a complete history but specifically include.
- Date and time of injury.
- Mechanism of injury: blunt, sharp, high velocity.
- Visual symptoms: blurring, floaters, flashing lights, diplopia.
- Don't forget other facial/systemic injuries.

## Examination routine

- Visual acuity (best corrected and pin-hole).
- Pupils: equal and reactive?
- RAPD?
- Lids: swelling, bruising, laceration?
- Anterior segment examination.
  - In cases of suspected globe rupture, keep manipulation to the absolute minimum.
- Conjunctiva: laceration, subconjunctival haemorrhage?
- Cornea: is it clear?
  - Apply a drop of 2% fluorescein and examine under a blue light. Epithelial defects will fluoresce green (Siedel test).
- Anterior chamber: cells, blood (hyphaema), disparity in depth compared to the other eye.
- Lens: opacity, check for any movement as the patient moves their eye.
- Vitreous: haemorrhage?
- Retina: commotio retinae (white/blanched areas of retina), haemorrhage, retinal tears/detachment, choroidal rupture.
- Optic disc: check function and appearance.

**Box 9.11 Some clinical presentations of trauma**

*Chemical injury*

Alkalis are more dangerous than acids, causing greater cellular disruption and can penetrate deep into the stroma and beyond
- Check the pH in both eyes
- Irrigate first, complete the assessment later
- Check for limbal ischaemia (blanching of limbal conjunctival vessels)
- Check corneal clarity: haze develops with severe chemical injury
- Other clinical features: chemosis, epithelial defect, anterior uveitis.

*Foreign body*

These may be penetrating and perforating (enter and exit)
- Establish the risk of penetrating injury from the history
- Examine for signs of perforation: reduced visual acuity, epithelial defect (Siedel positive), irregular pupil, iris defect, lens defect
- Evert the lids, dilate, and examine for vitreous haemorrhage or retinal foreign body.

*Corneal foreign body*
- Patients complain of a sensation of something in their eye. Also photophobia, watering, blurred vision
- Look for: conjunctival hyperaemia, visible corneal foreign body ± rust ring or infiltrate, ± anterior uveitis.

*Corneal abrasion*
- Conjunctival hyperaemia, corneal epithelial defect, relief of pain with topical anaesthetic, improved vision with a pin-hole.

*Traumatic hyphaema*
- History of blunt trauma
- Blood in anterior chamber, ± traumatic mydriasis (dilated pupil), irregular pupil (pupil sphincter damage).

*Globe rupture: anterior*
- History of severe injury
- Subconjunctival haemorrhage, herniation of uveal tissue, shallow anterior chamber, hyphaema, ± corneal oedema, endophthalmitis.

*Globe rupture: posterior*
- History of severe injury
- Subconjunctival haemorrhage, deep anterior chamber compared to non-injured eye, vitreous haemorrhage, retinal haemorrhage, retinal detachment, endophthalmitis.

*Orbital fracture*
- History of blunt trauma (e.g. tennis ball)
- Clinical features: blurred vision, ± diplopia, periorbital bruising haemorrhage, hyphaema, painful restricted eye movements, infraorbital hypoaesthesia (infraorbital nerve damage associated with orbital floor fractures), enophthalmos, surgical emphysema.

# The red eye

## Approach
- A careful history must be taken including previous ophthalmic history, systems review, and family history of eye disease.
- Examine the eyes systematically:
  - Visual acuity
  - Pupil responses
  - Lids
  - Conjunctiva/sclera
  - Cornea (add fluorescein and examine under a cobalt blue light for an epithelial defect)
  - Anterior chamber
  - Iris
  - Lens.
- Always record the patient's visual acuity for distance and near with their appropriate prescription.
  - ▶ Don't forget to check both eyes.
- Can the vision be improved with a pin-hole?

## Red flags
- ▶ A red eye in a contact lens wearer should be assumed to be microbial keratitis until proven otherwise and should be seen by an ophthalmologist the same day.
- A significant reduction in visual acuity not corrected by a pin-hole or the patient's glasses should be regarded as sinister.

## Causes of the red eye

*Conjunctivitis*
See Box 9.12.

*Uveitis*
- Inflammation of the uveal tract (iris, ciliary body, and choroid).
- Anterior uveitis usually presents with circumcorneal injection and photophobia, watering, blurring of the vision.
- Aggressive uveitis may lead to the iris sticking to the lens. This (posterior synechiae) may give the pupil a small irregular appearance.
- In severe cases, pus may form in the anterior chamber (hypopyon).

*Microbial keratitis (corneal ulcer)*
- *Symptoms:* discomfort, often history of prolonged contact lens wear, photophobia, blurred vision.
- *Signs:* infected conjunctiva, corneal ulcer, ± anterior chamber cells.

*Herpes simplex keratitis*
- *Symptoms:* pain and photophobia. Often the eye waters, there may be a history of cold sores.
- *Signs:* a branching dendritic ulcer is visible on the surface of the cornea with fluorescein under a cobalt blue light.

*Episcleritis*
- Inflammation of the layer superficial to the sclera, deep to conjunctiva.
- *Symptoms:* bruised, tender feeling, watering.
- *Signs:* the inflamed vessels in the episclera are superficial and can be moved by touch unlike the deeper scleral vessels. Sometimes nodular.

*Scleritis*
- May be seen in association with connective tissue diseases (Wegener's granulomatosis, rheumatoid disease, polyarteritis nodosa).
- *Symptoms:* severe eye pain that keeps the patient awake at night.
- *Signs:* the sclera may thin revealing the choroid below as a blue tinge.

*Subconjunctival haemorrhage*
- Redness is usually the only symptoms and sign.
- Check for hypertension or a bleeding disorder.

*Acute angle closure*
- ▶ An emergency.
- Systemic features include nausea, vomiting, and headache.
  - A typical history includes blurred vision and haloes around lights.
- Clinical features: significant reduction in vision; red, infected eye. Fixed, mid-dilated and oval-shaped pupil, hazy cornea due to oedema.
- Raised intraocular pressure makes the eye feel hard (compare sides).

---

**Box 9.12 Conjunctivitis**

*Bacterial*
- *Symptoms:* acute red eye, grittiness, burning, discharge, no visual loss
- *Signs:* infected conjunctiva, mucopurulent discharge, clear cornea.

*Viral*
- *Symptoms:* acute red eye, watering, often starts in one eye and then spreads to other eye
- *Signs:* red eye, watering, ± chemosis, ± eyelid oedema, ± pseudo-membrane, corneal subepithelial opacities, tender lymphadenopathy.

*Chlamydial*
- *Symptoms:* unilateral or bilateral mucopurulent discharge, red eye (may become chronic), ± urethritis (may be asymptomatic)
- *Signs:* red eye, mucopurulent discharge, ± peripheral corneal infiltrates, tender lymphadenopathy.

*Allergic*
- *Symptoms:* itching is the key symptom, bilateral redness, watering, associated 'hay fever' symptoms (sneezing, nasal discharge)
- *Signs:* lid oedema, 'pinkish' conjunctivae, papillae.

*Ophthalmia neonatorum*
- Conjunctivitis occurring in the first month of life, commonly acquired during birth. It is a notifiable disease in the UK
- *Clinical features:* mucopurulent discharge, ± chemosis, lid oedema, keratitis.
  - *Chlamydial:* onset 4–28 days after birth
  - *Gonococcal:* hyperacute onset (1–3 days after birth).

# Eye signs in thyroid disease

### Examination

*Inspection*
- Look at the patient's eyes from the front, side, and from above (see Box 9.13).
- 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.

*Eye movements*
Test eye movements in all directions.

*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 increase in retro-orbital fat, oedema, and cellular infiltration.
- It can be formally assessed using 'Hertel's exophthalmometer'.

*Exophthalmos*
This is a 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 9.13 Eye signs of thyrotoxicosis and Graves' 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 thyrotoxicosis due to Graves' disease. However, the proptosis may persist once thyroid hormone levels have been normalized.

*Eye signs of thyrotoxicosis*
- Lid retraction
- Lid lag.

*Eye signs of Graves' disease (Graves' ophthalmopathy)*
- 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 should be performed.

# Important presentations

## Painful loss of vision

- Trauma.
  - Corneal foreign body, corneal abrasion, traumatic hyphaema, penetrating injury, globe rupture.
- Corneal ulcer.
- Herpes simplex keratitis.
- Anterior uveitis.
- Endophthalmitis.
- Scleritis.
- Giant cell arteritis (GCA).

## Acute painless loss of vision

*Vitreous haemorrhage*

- *Clinical features*: impaired or no fundal view, ± reduced red reflex.
- *Causes*: retinal detachment, diabetic retinopathy.

*Wet age-related macular degeneration*

- See elsewhere in this chapter.

*Central or branch retinal vein occlusion*

- *Clinical features*: widespread or hemispheric retinal haemorrhages, ± cotton-wool spots, exudates, disc swelling, macular oedema.
- *Common causes:* hypertension, diabetes, hyperlipidaemia.
- *Other causes:* vasculitis (e.g. Behçet's, sarcoidosis, systemic lupus erythematosus), clotting disorders (e.g. protein S or C deficiency, antiphospholipid syndrome), multiple myeloma, glaucoma.

*Central or branch retinal arterial occlusion*

- *Symptoms*: sudden loss of vision, may have symptoms of GCA,10% of patients have preceding amaurosis fugax.
- *Signs:* pale retina, cherry red spot, retinal vessel emboli.
- Causes:
  - Atherosclerotic: hypertension, diabetes, smoking, hyperlipidaemia
  - Embolic: carotid/aortic artery disease, cardiac valve disease
  - Haematological: protein C deficiency, antiphospholipid syndrome, lymphoma, leukaemia
  - Inflammatory: GCA, SLE, polyarteritis nodosa, Wegener's granulomatosis
  - Other: oral contraceptive, trauma, migraine.

*Anterior ischaemic optic neuropathy (AION)*

- May be arteritic (temporal arteritis or GCA) or non-arteritic (hypertension, diabetes, anaemia, smoking, hyperlipidaemia).

*Temporal arteritis or giant cell arteritis (GCA)*

- *Symptoms*: headache, scalp tenderness, jaw claudication, neck pain, fever, malaise, joint pains.
- *Signs*: tender, non-pulsatile temporal artery, relative afferent pupillary defect, swollen optic disc.
  - +/– central retinal arterial occlusion, cranial nerve palsies.

*Retinal detachment*
- *Clinical features:* floaters, flashing lights, loss of vision/visual field defect.
- *Risk factors:*
  - Ocular: history of trauma, cataract surgery, myopia
  - Systemic: Stickler syndrome, Marfan's syndrome, Ehlers–Danlos syndrome.

## Hypertensive retinopathy

*Clinical features*

▶ Findings depend on classification grade of retinopathy.
- Focal/generalized arteriolar constriction and straightening (Fig. 9.8).
- Arteriosclerosis leading to changes at arterio-venous (AV) crossing points: 'AV nipping' or 'nicking' (the arteriole crosses over the top of the vein) and silver-wiring of retinal arterioles.
- Microaneurysms.
- Cotton-wool spots indicating ischaemia.
- Retinal haemorrhages; usually flame shaped.
- Exudate; often in macular star pattern.
- Arterial macroaneurysms.
- Disc oedema.
- Tortuous vessels in malignant hypertension.

*Classification*
- *Grade 1:* mild generalized arteriolar constriction and narrowing.
- *Grade 2:* more severe narrowing with AV nipping.
- *Grade 3:* features of grade 1 and 2 plus retinal haemorrhages, exudates, microaneurysms, cotton-wool spots.
- *Grade 4:* all of the features described above with the presence of optic disc swelling ± macular oedema.
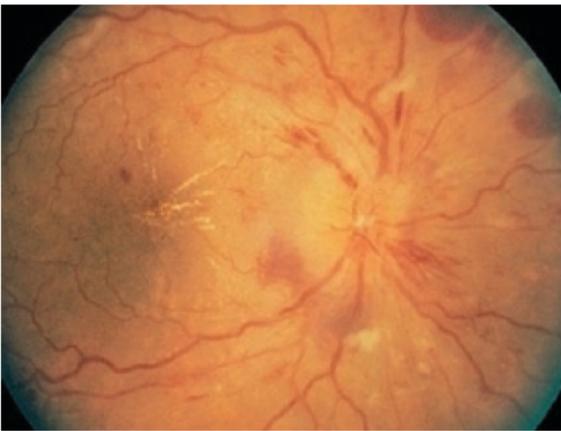


**Fig. 9.8** Hypertensive retinopathy.

### Diabetic retinopathy

Diabetes mellitus is the commonest cause of blindness in the working population. The best predictor of diabetic retinopathy is the duration of the disease (see Figs 9.9–9.11).

*Pathogenesis*

Microangiopathy primarily affecting pre-capillary arterioles, capillaries, and post-capillary venules. There is loss of pericytes, damage to the vascular endothelial cells, deformation of red blood cells with increased aggregation leading to microvascular occlusion and leakage.

*Clinical features*

Fundus findings are usually bilateral and broadly symmetrical. Abnormalities depend on the severity of disease and findings may include:
- Microaneurysms.
- Dot and blot haemorrhages.
- Lipid exudates.
- Venous beading.
- Intraretinal microvascular abnormalities (IRMAs).
- Cotton-wool spots (CWSs).
- New vessels at the disc or elsewhere (NVD/NVE).
- Tractional retinal detachment.
- Macular oedema and thickening (diabetic maculopathy).

*Classification*

The Early Treatment Diabetic Retinopathy Study provides a classification for the grading of diabetic retinopathy, dividing the disease into non-proliferative (NPDR) and proliferative (PDR) groups. The UK National Screening Committee classification is listed in brackets.

*Non-proliferative*

- *Mild:* at least one microaneurysm (R1).
- *Moderate:* haemorrhages or microaneurysms present, lipid exudates, venous beading, IRMAs (R2).
- *Severe (the '4:2:1' rule):* haemorrhages or microaneurysms in all four quadrants. Venous beading in two or more quadrants. IRMAs in at least one quadrant (R2).
- *Very Severe:* two or more of the 'severe' features above (R2).

*Proliferative*

- *Early PDR:* new vessels (NV) on the retina; either at the disc (NVD) or elsewhere (NVE) (R3).
- *High-Risk PDR:* new vessels on the disc (NVD) occupying 1/4 to 1/3 or more of the disc area. Any NV and vitreous or preretinal haemorrhage. (R3) Treatment is argon laser panretinal photocoagulation.

*Diabetic maculopathy*

- Retinal oedema within 500µm of the centre of the fovea.
- Hard exudates within 500µm of the fovea, adjacent retinal thickening.
- Retinal oedema ≥1 disc diameter in size within 1 disc diameter of the centre of the fovea.
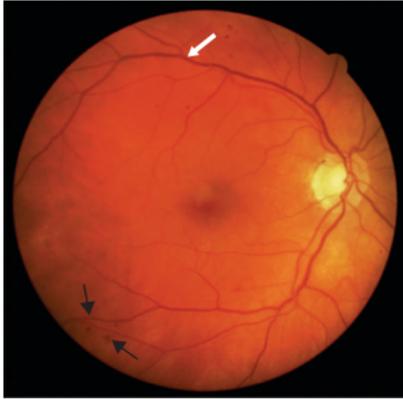
**Fig. 9.9** Non-proliferative diabetic retinopathy. White arrow shows a microaneurysm, black arrows show haemorrhages.
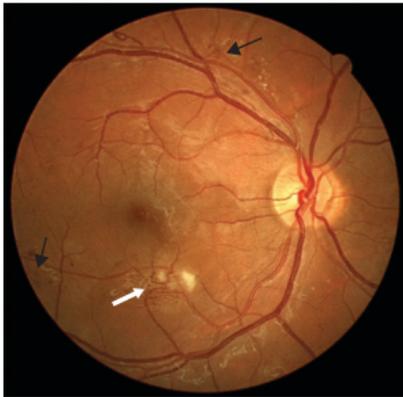


**Fig. 9.10** Proliferative diabetic retinopathy. White arrow shows new vessels growing into a cotton-wool spot. Black arrows show dot haemorrhages.
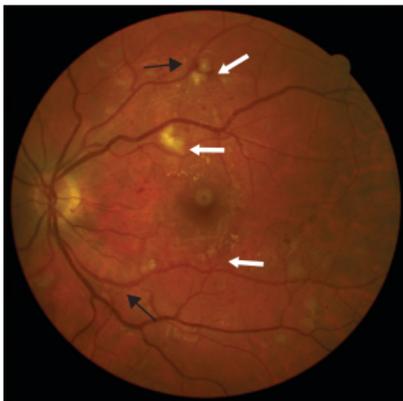


**Fig. 9.11** Diabetic maculopathy. White arrows show hard exudates, black arrows show haemorrhages. New vessels can also be seen growing into the macula.

## Glaucoma

Glaucoma is an optic neuropathy associated with raised intraocular pressures leading to irreversible optic nerve damage. It is usually asymptomatic and is often detected incidentally but can lead to blindness.

*Applied physiology*
- Aqueous humour is produced by the ciliary body and mainly drains through the trabecular meshwork but there is also drainage through uveoscleral routes.
  - Normal intraocular pressure = 8–21mmHg.

*Primary angle closure*
- Narrowing of the anterior chamber drainage angle prevents aqueous fluid outflow resulting in increased intraocular pressure (IOP).
- *Acute:* classically a red, painful eye; hazy cornea due to oedema; fixed, mid-dilated, oval pupil; reduced vision; headache, nausea, and vomiting.
- *Chronic/subacute:* may give a history of haloes around lights at night; often indistinguishable from open angle glaucoma unless anterior chamber viewed with gonioscopy.

*Primary open angle*
- Aqueous fluid outflow is reduced despite anterior chamber angle remaining open.
  - Most common form of glaucoma in patients >50 years.
- *Risk factors:* increased IOP, reduced central corneal thickness, Afro-Caribbean origin, increased age, affected 1st-degree relative.
- *Other risk factors:* hypertension, diabetes, myopia.

*Secondary glaucoma*
- Angle may be open or closed but the pathology results from a separate ocular condition or its treatment.

*Normal tension/pressure glaucoma*
- Open angle and glaucomatous field loss and disc changes despite an IOP falling within the 'normal' range.

*Conditions leading to secondary glaucoma*
- Uveitis:
  - Trabeculitis: chronic inflammation of the trabecular meshwork resulting in reduced aqueous outflow
  - Iris bombé: iris adhesions to the anterior capsule of the lens (posterior synechiae) through 360° following anterior uveitis can result in bowing forward of the iris, from trapped aqueous, closing the drainage angle.
- *Rubeosis*: ischaemic insults to the eye (retinal vein/artery occlusions, diabetes) can lead to new blood vessel growth/bleeding in the anterior chamber angle obstructing aqueous outflow.
- *Trauma*: damage to the drainage angle from blunt trauma can lead to scarring of the trabecular meshwork.

### Age-related macular degeneration (AMD)

AMD is the leading cause of blindness registration in the western world. There are two main types. The common dry form is associated with gradual visual loss, while the much less common wet or neovascular AMD is associated with rapid and more severe visual loss.

*Dry AMD*
- Progressive atrophic changes of the macula characterized by the presence of drusen, extracellular material deposited between the retinal pigment epithelium (RPE) and the underlying Bruch's membrane.
- There is resulting loss of the RPE and photoreceptor layers of the retina.

*Wet AMD*
- Accounts for 10% of cases of AMD and is the most common cause of blindness in the Western world.
- New vessels grow from the choroidal vasculature and enter the retina forming a choroidal neovascular membrane (CNV), which can leak fluid or blood at the macula leading to scarring and visual loss.

*Symptoms*
- Central visual loss (gradual or sudden).
- Distortion (metamorphopsia: straight lines look wavy and distorted).
- Scotoma.

*Signs*
- Hard drusen (well-demarcated yellow lesions).
- Soft drusen (ill-defined paler lesions which may become confluent).
- Pigmentation.
- Subretinal or intraretinal haemorrhage.
- Exudate.

*Risk factors*
- Age.
- Family history.
- Female sex.
- Caucasian.
- Smoking.
- Hyptertension.
- Cardiovascular disease.
- Hypercholesterolaemia.

## Cataract

The lens is a biconvex, transparent, avascular structure enclosed by a capsule. Lens fibres are continually laid down throughout life so the lens is the only eye structure that continues to grow. With time the lens loses its transparency leading to cataract formation, which is universal with increasing age (see Fig. 9.12).

*Common symptoms*
- Gradual deterioration of vision.
  - Difficulty reading.
- Glare from oncoming car headlights.

*Risk factors*

The majority of cataracts are senile, but cataracts may be associated with a number of systemic diseases including:
- Diabetes mellitus: usually cortical or posterior subcapsular cataracts. Patients are usually younger and the cataract can progress relatively quickly.
- Disorders of calcium homeostasis.
- Uveitis.
- Intraocular tumours.
- Angle closure glaucoma.
- Wilson's disease: so-called 'sunflower cataract' due to their shape. Green-brown in colour and secondary to copper deposition.

*Subtypes of acquired cataract*
- *Nuclear sclerosis:* central lens discoloration tends to affect distance vision to a greater extent than near vision.
- *Cortical cataract:* results from deterioration of younger lens fibres within the outer cortex of the lens. This may take on a variety of appearances including spokes and vacuoles.
- *Subcapsular cataract:* may form either anteriorly or posteriorly within the lens epithelial cells. Posterior tends to affect near vision more than distance vision.
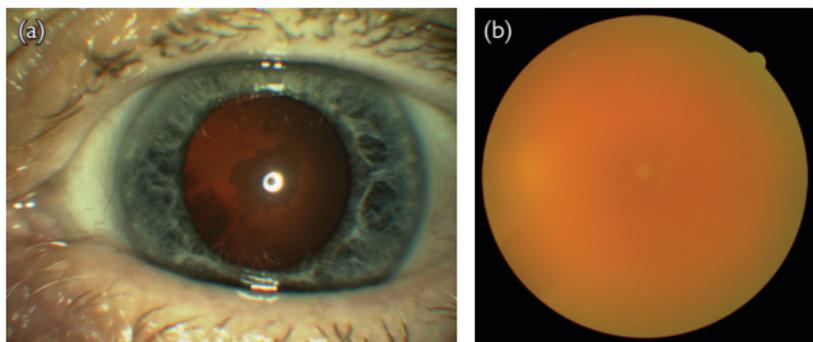
**Fig. 9.12** (a) Cataract seen externally. (b) Attempted fundoscopy through mature white cataract.

**See also:**

More information regarding the presentation and clinical signs of eye diseases to aid preparation for OSCE-type examinations and ward rounds can be found in the *Oxford Handbooks Clinical Tutor Study Cards*.

*'Medicine' Study Card set:*

- The red eye
- Diabetic retinopathy
- Hypertensive retinopathy
- Optic disc swelling
- Glaucoma
- Optic atrophy
- Cataracts
- Central retinal vein occlusion
- Retinal detachment
- Angioid streaks
- Age-related macular degeneration
- Cytomegalovirus retinitis
- Myelinated nerve fibres
- Asteroid hyalinosis
- Visual field defects
- Nystagmus
- Ptosis
- Holmes–Adie–Moore syndrome
- Argyll Robertson pupils
- Internuclear ophthalmoplegia.

# The locomotor system

# Introduction

## Joints

A joint 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 surfaces, and the degree of movement they allow.

There are three types of joints (Boxes 10.1 and 10.2):
- *Fibrous joints (syndesmosis):* held together by fibrous (collagenous) connective tissue, are 'fixed' or 'immoveable'. They do not have a joint cavity. Examples include sutures between the bones of the skull.
- *Cartilagenous joints (synchondrosis):* held together by cartilage, are slightly moveable, and again have no joint cavity. Examples include the symphysis pubis, and intervertebral discs.
- *Synovial joints (diarthrodial joint):* covered by cartilage with a synovial membrane enclosing a joint cavity. These are freely moveable, and are the most common joint type, being typical of limb joints.

## Synovial joints

### Cartilage

Hyaline articular cartilage covers the intra-articular surface of the bones, ↓ friction at the joint, and facilitates shock absorption. Not all intra-articular bone is covered by hyaline cartilage; the part not covered by hyaline articular cartilage is the bare area of the joint and is the target site for erosions in inflammatory arthritis (e.g. rheumatoid arthritis).

Some synovial joints have an additional fibrocartilaginous disc (e.g. meniscus at the knee), or a fibrocartilaginous labrum (e.g. hip, shoulder).

### Capsule and synovial membrane

A sleeve-like bag (fibrous capsule lined with synovial membrane) surrounds the synovial joint.

The inner synovial membrane secretes synovial fluid which has a number of functions including lubrication, and supply of nutrients to the cartilage (which is avascular and lacks pain fibres). The fluid contains phagocytic cells that remove microbes and debris within the joint cavity.

Ligaments are thickened portions of joint capsule. However, some ligaments may be distinct from the capsule.

### Entheses

The bony attachment of capsules, ligaments, or tendons is called the enthesis. Entheseal inflammation (enthesitis) is the hallmark of sero-negative inflammatory arthritis (e.g. psoriatic arthritis, ankylosing spondylitis).

### Tendon sheaths

Tendons e.g. those of flexor digitorum superficialis and profundus, biceps etc, are also covered by synovial sheaths called tenosynovium. This may get inflamed (tenosynovitis) due to mechanical (e.g. some cases of de Quervain's tenosynovitis), or autoimmune (e.g. lupus, rheumatoid arthritis) conditions. The Achilles tendon, one of the largest tendons in the body, does not have a tenosynovium, and is only covered by a thin layer of connective tissue: the *paratenon*.

### Box 10.1 Types of synovial joint

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 three axes: flexion/extension, abduction/adduction, rotation (e.g. 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 between 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).

### Box 10.2 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 with extended knee 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 with extended knee 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 dial anticlockwise
- *Supination:* moving the forearm as if turning a dial clockwise
- *Dorsiflexion:* moving the ankle to bring the dorsum of the foot towards the tibia (i.e. pointing the foot upwards)
- *Plantar flexion:* moving the ankle to bring the plantar surface in line with the tibia (i.e. pointing the foot downward)
- *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 backward.

# Important locomotor symptoms

As with any system, a carefully and accurately compiled history can be very informative and may point to a diagnosis even before examination or laboratory tests.

## Pain

Pain is the most common symptom in problems of the locomotor system, and should be approached in the same manner as any other type of pain.

Pain may arise from articular structures, peri-articular structures, or may be referred from other sites. (See Boxes 10.3–10.5.)

Determine the character, 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 due to mechanical/degenerative arthropathies increases with joint use, whereas pain due to inflammatory arthropathy improves with joint use.

- Pain in a joint is called arthralgia.
- Pain in a muscle is called myalgia.

*Character*
- Bone pain is typically experienced as boring, penetrating, and is often worse at night. Causes include Paget's disease, tumour, chronic infection, avascular necrosis, and osteoid osteoma.
- Pain associated with a fracture is usually sharp and stabbing in nature, and is often exacerbated by movement.
- Shooting pain is suggestive of nerve entrapment (e.g. disc prolapse).

*Onset*
- Acute-onset pain is often a manifestation of infections such as septic arthritis, or of crystal arthropathy (e.g. gout).
- Osteoarthritis, ankylosing spondylitis, and rheumatoid arthritis usually cause a slower (insidious) onset of pain.

*Site*
Determine the exact site of maximal pain if possible, and of any associated lesser pains. Ask the patient to point to the site of maximal pain.

Remember that the site of pain is not necessarily the site of pathology; often the pain is referred. Referred pain is due to inability of the cerebral cortex to distinguish between sensory messages from embryologically related sites. For example, hip pain is frequently referred down the thigh, towards the knee; and pain from the cervical spine is referred to the shoulder region. Referred pain is usually poorly localized, and is worsened by movement of the affected joint (not of the joint to which it is referred).

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### Box 10.3  Some causes of hip pain

- *Anterior (groin):* hip arthropathy, avascular necrosis, ilio-psoas bursitis, disc prolapse pressing on L1–L2 nerve root, inguinal or femoral hernia
- *Medial:* adductor enthesopathy, inguinal or femoral hernia
- *Lateral:* trochanteric bursitis, abductor enthesopathy (greater trochanteric pain syndrome), meralgia paresthetica
- *Posterior:* inflammatory back pain due to sacro-iliitis (frequently reported as buttock pain), ischial bursitis, spinal stenosis, and disc prolapse pressing on L3–L5, and sacral nerve roots.

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### Box 10.4  Some causes of knee pain

- *Chondromalacia patellae:* softening of the patellar articular cartilage and is felt as anterior knee pain 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
- *Other causes:* osteoarthritis, trauma, bursitis, tendonitis, rheumatoid arthritis, infection, malignancy.

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### Box 10.5  Some other causes of arthralgia

*Shoulder*
- Rotator cuff disorders (e.g. tendonitis, rupture, adhesive capsulitis/frozen shoulder)
- Referred pain: e.g. cervical, mediastinal, cardiac
- Arthritis: glenohumeral, acromioclavicular.

*Elbow*
- Epicondylitis (lateral = tennis elbow; medial = golfer's elbow)
- Olecranon bursitis
- Referred pain from neck/shoulder (e.g. cervical spondylosis)
- Osteo- and rheumatoid arthritis.

*Mechanical/degenerative back pain*
- Arthritis
- Trauma
- Infection
- Ankylosing spondylitis
- Spondylolisthesis
- Disc prolapse, lumbar spinal/lateral recess stenosis
- Spinal tumours
- Metabolic bone disease.

### Stiffness

This is a subjective inability or difficulty in moving a joint.

▶ If stiffness predominates without significant joint pain, consider spasticity or tetany. Look for hypertonia and other upper motor neuron signs.

Ask the patient:

- When is the stiffness worst?
  - 'Early morning stiffness', improving as the day goes by, is characteristically associated with inflammatory joint disease (e.g. rheumatoid arthritis, ankylosing spondylitis). This often takes hours before maximal improvement with activity
  - Early morning stiffness may be present in non-inflammatory joint diseases (e.g. osteoarthritis). In these cases, patients report a shorter duration of stiffness before improvement (often <20 minutes).
- Which joints are involved?
  - Stiffness predominates in hands and feet in rheumatoid arthritis; in shoulder and pelvic girdle in polymyalgia rheumatica; and in the buttocks and lower back in ankylosing spondylitis.
- How long does it take to 'get going' in the morning?
- How is the stiffness related to rest and activity?
  - Stiffness in inflammatory joint disease tends to worsen with rest, and improves with activity. However, in non-inflammatory joint disease, stiffness is exacerbated by activity and it is typically worse at the end of the day.

### 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, acute crystal synovitis, haemarthrosis (trauma, anticoagulants, or any underlying bleeding disorder)
  - Slow onset (over days to weeks) suggests inflammatory arthritis.
- Are the joints always swollen or does the swelling come and go?
- 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?
- Does the whole finger or toe swell up like a sausage? (Dactylitis.)

### Crepitus

A crunching sound and feeling on moving the joint.

Distinguish crepitus from other articular/peri-articular noises like cracking, clonking, popping, and snapping which are usually not pathological.

## Locking, triggering, and giving way

### *Locking*

Locking 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. This frequently occurs at the knee.

### *Triggering*

A similar phenomenon to locking. This may occur at the fingers, when there is an inability to actively extend the digit completely. This is usually due to thickening of the flexor tendon sheath but may occur in the context of trauma or other pathology to the extensor tendons. The triggering finger can be extended passively by the patient or the examiner.

### *Giving way*

Patients with degenerative arthritis affecting the lower limb joints (typically the knee or ankle) often report a feeling of instability when weight-bearing. This is described as a sensation of 'giving way', a subjective sensation which may or may not be associated with falls.

## Deformity

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. (See Box 10.6.)

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 unable to do now, which he or she could do before?).

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### Box 10.6 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.

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## Weakness

Always enquire about the presence of localized weakness (peripheral nerve lesion) or generalized weakness (systemic cause).

In some patients, subjective weakness may result from pain. This is often seen in patients with polymyalgia rheumatica who report 'weakness' around their hips and shoulders.

### 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?).

### Affect and sleep

Patients with chronic musculoskeletal conditions may have, or develop, low mood. Therefore, targeted enquiries should be made about the patient's mood.

Similarly, these patients may have reduced and/or poor quality of sleep. Chronic sleep deprivation results in increased pain sensitivity, increasing the perception of pain. If pronounced, this may result in chronic widespread pain (e.g. fibromyalgia).

You may enquire about sleep in the following ways:
- How is your sleep?
- Do you have problems falling asleep?
- Do you frequently wake up at night? If so, why?
- Is your sleep refreshing?

### Extra-articular features

Several locomotor disorders (e.g. rheumatoid arthritis, SLE) cause extra-articular or multisystem features, some of which are outlined below:
- Systemic symptoms: low-grade fever, weight loss, fatigue, lethargy.
- Skin rash: vasculitic, photosensitive, nail-fold infarcts, alopecia (see ➲ Chapter 4).
- Oral:
  - Dry mouth (Sjögren's syndrome)
  - Ulcers (non-scarring: SLE; scarring: Behçet's disease).
- Raynaud's phenomenon (primary, SLE, systemic sclerosis).
- Urethritis (Reiter's syndrome).
- Scarring oro-genital ulcers (Behçet's disease).
- Eye symptoms:
  - Dry eyes (Sjögren's syndrome)
  - Episcleritis, scleritis, scleromalacia perforans (rheumatoid arthritis)
  - Uveitis (sero-negative inflammatory arthritis).
- Cardiorespiratory: breathlessness (pulmonary fibrosis?), pericarditis, and pleurisy (rheumatoid arthritis); aortic regurgitation (ankylosing spondylitis).
- Neurological:
  - Nerve entrapment (rheumatoid arthritis)
  - Migraine, depression, psychosis (SLE)
  - Stroke (anti-phospholipid antibody syndrome).

# 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. (See Box 10.7.)

### Family history

It is important to note any FHx of illness, especially those locomotor conditions with a heritable element:
- Osteoarthritis.
- Rheumatoid arthritis.
- Osteoporosis.
- Psoriasis.
- SLE.
- Sero-negative spondarthropathy.

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 (including herbal) 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 skin thinning, osteoporosis, myopathy, infections, and avascular necrosis.

Ask also about medication with known adverse musculoskeletal effects including:
- Statins: myalgia, myositis, and myopathy.
- ACE inhibitors: myalgia.
- Anticonvulsants: osteomalacia.
- Quinolone: tendinopathy.
- Diuretics, aspirin, alcohol: gout.
- Procainamide, hydralazine, isoniazid: drug-induced lupus.

▶ 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 symptoms.

### Smoking and alcohol

As always, full smoking and alcohol histories should be taken.

### 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.
  - Mechanically demanding occupations result in osteoarthritis in the mechanically loaded joints
  - Repetitive strain injury is seen in office workers
  - Vibrating power tools predispose to hand-vibration syndrome
  - Fatigue fractures may be seen in 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.
- *Age:* 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. Where applicable, take a history of risk factors for HIV and hepatitis.

---

**Box 10.7 Screening history**

If the locomotor system is not the main area of concern, you may wish to take a very short screening history in the form of three questions:

- Do you have any pain or stiffness in your muscle, joints, or back?
- Are you able to dress yourself completely without any difficulty?
- Are you able to walk up and down stairs without any difficulty?

If any of these questions reveal a problem, the issue should be explored in more detail.

# Outline locomotor history

As with other systems, organizing the clinical data (and your thoughts) into a structured framework can be crucial in clinching the diagnosis (see Box 10.8).

---

**Box 10.8  Structure of a musculoskeletal history**

- Presenting complaint(s)
  - How many joints are affected?
  - Establish their locations and timeline
- What is the pattern of joint involvement?
  - Single episode
  - Episodic/intermittent
  - Progressive/additive
- Extra-articular features
- Past medical history
- Family history
- Allergies
- Drug history
- Smoking
- Alcohol
- Social history.

---

### Formulations

At the end of a locomotor history, you should be able to identify:
- Key locomotor symptoms.
- Number of joints involved (see Box 10.9).
- Location of joint involvement (see Box 10.10).
- Onset and progression of joint involvement (see Box 10.11).

This allows you to formulate a clinical diagnosis in most instances.

---

**Box 10.9  Diagnoses by number of joints involved**

*One joint (monoarthritis)*
- Crystal arthropathy
- Haemarthrosis
- Infection
- Degenerative
- Post-traumatic
- A mono-articular presentation of an oligo- or polyarthritis.

*2–4 joints (oligoarthritis)*
- Inflammatory arthritis (rheumatoid, psoriatic, reactive, ankylosing spondylitis)
- Infection (endocarditis, acute rheumatic fever)
- Osteoarthritis.

*>5 joints (polyarthritis)*
- Inflammatory (rheumatoid, psoriatic, SLE)
- Osteoarthritis.

**Box 10.10 Diagnoses by pattern of involvement**

*Rheumatoid arthritis*
- MCPJs, PIPJs, wrist, and MTPJs
  - Typically does not affect DIPJs & 1st CMCJ.

*Psoriatic arthritis*
- Typically affects DIPJs more commonly than other hand joints
  - May sometimes mimic RA.

*Ankylosing spondylitis*
- Sacro-iliac joints, spine, shoulder, and hips

*SLE*
- MCPJs and wrists

*Osteoarthritis*
- Knees, 1st CMCJ, DIPJ, PIPJ, spinal apophyseal joints, hips, and ankle

*Gout*
- 1st MTPJ, IPJs, knees, and ankles

*Calcium pyrophosphate dihydrate deposition disease (CPPD)*
- Knees, wrist, MCPJs.

**Box 10.11 Diagnoses by onset and progression**

*Single attack of acute arthritis*
- Reactive arthritis, first presentation of crystal or inflammatory arthritis, septic arthritis

*Multiple completely resolving episodes*
- Crystal synovitis (gout, acute CPP crystal synovitis), palindromic rheumatoid arthritis

*Persistent or progressive 'additive' joint involvement*
- Rheumatoid arthritis (typically symmetric), seronegative inflammatory arthritis (typically asymmetric)
  - Progression may be over months or years.

*Persistent or progressive sacro-iliac joint and spinal involvement*
- Sero-negative spondarthropathy
  - Peripheral large joint oligo-arthritis may also occur.

*One joint involved after the other over years*
- Osteoarthritis
  - Progressive involvement of several IPJs over a period of few months may occur at disease onset in 'nodal' OA. This typically occurs in post-menopausal women.

# Outline locomotor 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: hand (including wrist), elbow, shoulder, spine, hip, knee, ankle, and foot. (See Box 10.12.)

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**Box 10.12 Examination framework**

The examination of each joint should follow the standard format:
- Look
- Feel
- Move
  - Active
  - Passive.
- Special tests
- Function.

Limitation of active movement alone reflects underlying pathology of the tendons and muscle surrounding the joint, but limitation of both active and passive movement suggests an intrinsic joint problem.

▶ 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.

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# The GALS screen

The overall integrity of the locomotor system can be screened very quickly by using the 'GALS' method of assessment. (See Box 10.13.)

You may also use the GALS screen to make a quick, 'screening' examination of the whole locomotor system in order to identify which joints or regions require to be examined in more detail.

The GALS screen consists of four components:
- **G** = Gait.
- **A** = Arms.
- **L** = Legs.
- **S** = Spine.

**Box 10.13 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 a slightly modified version:

*Gait*
- Watch the patient walk, turn, and then walk back
  - 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
- *Wrist flexion and finger extension:* the reverse prayer sign
- *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
- *Knee:* bulge test and patellar tap
- *Ankle:* test dorsiflexion (normal 15°) and plantar flexion (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 cervical 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)
- Cervical lateral flexion: 'put your ear on your shoulder'.

*Doherty et al. (1992). *Annals Rheum Dis* 51: 1165–9.

# Hand

Hand examination is an important part of all examination routines. This section focuses on hand examination with an emphasis on the locomotor system. The reader should refer to ⮕ Chapters 4 and 8 on skin, hair, and nails and the nervous system for other components of hand examination (Fig. 10.3).

## Inspection

Look around the room, or at the patient for any functional aids, or adaptations. Begin by exposing the forearms up to the elbows, and sit facing the patient. Inspect the dorsal surface first, asking the patient to place their hands flat, palms down on a pillow with fingers and thumbs resting on the pillow. Then, inspect the palms. Look for:

*Skin and nails*
- Skin colour.
  - Erythema (acute gout, acute CPP crystal arthritis, infection)
  - Digital ischaemia: pallor or bluish discoloration.
- Skin thickness.
  - Thin skin: corticosteroid use
  - Callosities: mechanically demanding occupation
  - Tight shiny skin, sclerodactyly: systemic sclerosis.
- Discrete skin lesions.
  - Vasculitis
  - Psoriasis
  - Gottron's papules (dermatomyositis)
  - Ulcers (systemic sclerosis—typically on fingertips).
- Other soft-tissue lesions.
  - Rheumatoid nodules (along extensor tendons)
  - Gouty tophi (near interphalangeal joints on fingers).
  - Ganglion (near wrist).
- Scars.
  - Wrist fusion: dorsal median longitudinal scar on wrist
  - MCPJ replacement: dorsal longitudinal scar on MCPJs
  - Tendon transfer: dorsal longitudinal scar.
- Nails:
  - Clubbing: hypertrophic pulmonary osteo-arthropathy
  - Splinter haemorrhage: infective endocarditis, vasculitis, physical trauma (digital trauma in mechanically demanding occupations may also result in occasional splinters in the distal nail bed)
  - Onycholysis (psoriasis, hyperthyroidism)
  - Pitting of the nail plate (psoriasis)
  - Nail-fold capillary telangiectasia and peri-ungual erythema: dermatomyositis, SLE, systemic sclerosis.

*Muscle wasting:*
Look at the interossei and forearm muscles.

*Attitude, deformity, and swelling*
- Inspect each of the following joint groups sequentially (see Box 10.14):
  - Distal interphalangeal (DIP)
  - Proximal interphalangeal (PIP)
  - Metacarpophalangeal (MCP)
  - Wrist.
- You may also ask the patient to make a fist and look at the MCP heads.
  - Normally, there is a depression ('valley') between neighbouring MCP heads which become 'filled in' with MCPJ synovitis.
- Ask the patient to hold their hand in front of their chest with elbow and shoulders flexed and wrist extended. In this position, look for:
  - Volar subluxation of the carpus in RA
  - Wrist drop in radial nerve palsy
  - Finger drop due to ruptured finger extensor tendons.

*Palmar abnormalities*
As well as the features noted on the dorsal aspect, look for:
- Scar of carpal tunnel decompression: volar mid-line longitudinal scar.
- Thenar or hypothenar wasting.
- Palmar erythema (RA, liver disease, corticosteroid use).
- Nodules near the distal palmar crease (Dupuytren's contracture gives thickened palmar fascia).
- Tendon nodules: usually occur at the level of MCPJ heads, associated with triggering.
- Gouty tophi (fingertips).
- Calcinosis cutis (fingertips; seen in limited cutaneous systemic sclerosis).
- Digital pits (fingertips; seen in diffuse cutaneous systemic sclerosis).

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**Box 10.14 Some finger and wrist deformities**

- *Swan neck:* fixed flexion at the DIPJ and hyperextension at the PIPJs—associated with rheumatoid arthritis
- *Boutonnière:* hyperextension at the DIPJ and flexion at the PIPJs—associated with rheumatoid arthritis
- *Z-shaped thumb:* flexion at the MCPJ of the thumb, hyperextension at the interphalangeal joint—associated with RA
- *Ulnar deviation at MCPJs:* a feature of rheumatoid arthritis and other conditions, the fingers are deviated medially (toward the ulnar aspect of the forearm) at the MCPJs
- *Wrist subluxation:* volar subluxation of carpus (RA)
- *Wrist deviation:* radial deviation is frequently seen in RA
- *Heberden's nodes:* postero-lateral bony swelling (due to osteophytes) at the DIPJs—a feature of osteoarthritis
- *Bouchard's nodes:* similar to Heberden's nodes, but at the PIPJs—a feature of osteoarthritis
- *Dorsal bar:* merging of Heberden's or Bouchard's nodes at the IPJs
- *Telescopic fingers:* advanced destruction of IPJs. The finger may be elongated when it is pulled out passively. There is concentric wrinkling of the skin. Seen in advanced psoriatic arthritis.

## Palpation

🛈 Ask the patient if there is any tenderness and palpate those areas last.
  • Using the dorsum of your hands, feel for temperature change over
    the wrists, thumb-base, MCPJs, and IPJs.
    • As a rule, skin in the peripheries gets cooler distally.
  • Palpate any abnormalities identified on inspection.
  • Perform an MCPJ squeeze (as in GALS) to screen for MCPJ synovitis.
  • Depress the ulnar styloid (piano key sign) to check for the integrity of
    the inferior radio-ulnar joint and its supporting ligaments.
  • Assess for skin thickening.
    • Gently pick the skin on the dorsum of the hand between your
      thumb and index finger to make a skin fold
    • If you are not able to make a skin fold, try to move the skin over
      the underlying soft-tissue structures.
  • Palpate finger flexor tendons on the palmar aspect for thickening.

## Movement

Before assessing movement, ask the patient if they have any pain. Test
active, and then passive movements.

*Active movements*
  • *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. 10.1.
  • *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
    as in Fig. 10.2.
  • *Wrist abduction:* with elbows flexed at 90°, palm facing up, and
    forearm fixed, ask the patient to point their fingers outwards.
  • *Wrist adduction:* with elbows flexed at 90°, palm facing up, and
    forearm fixed, ask the patient to point their fingers inwards.
  • *Finger flexion:* ask the patient to make a fist.
    • Also observe for range of movement. Normally a patient should be
      able to dig their finger nails into their palms
    • Detailed assessment of IPJ flexion may be carried out if necessary.
      Fix the proximal phalanx to assess PIPJ flexion (flexor digitorum
      superficialis) and the distal phalanx to assess DIPJ flexion.
  • *Finger extension:* ask the patient to straighten their fingers out. Also
    tested with the prayer and reverse prayer positions. Triggering of the
    finger may be revealed during extension.
  • *Thumb movements:* assess flexion, extension, abduction, adduction,
    opposition (see ➲ Chapter 8).

*Passive movements*
  • Move each joint and assess the range of movement and watch for
    any pain.
  • Feel for crepitus, especially over the base of the thumb.

## 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.



**Fig. 10.1** The prayer position.



**Fig. 10.2** The reverse prayer position.

**Skills station 10.1**

*Instruction*

Examine this patient's hands.

*Model technique*
- Clean your hands
- Introduce yourself
- Explain the purpose of examination, obtain informed consent
- Ask for any painful areas you should avoid
- Ask the patient to expose the distal upper limb including the forearm, wrist, and hand
- Sit opposite and ask the patient to position their hands on a pillow, palms facing down
- Inspect the dorsum of the hand, wrist, and forearm. Look at the elbow
- Feel for temperature over the joint areas (proximal to distal) using the dorsum of your hands
  - Compare opposite sides simultaneously
- Palate the wrist, MCPJs, and IPJs for swelling and tenderness
- Ask the patient to turn their hands over
- Inspect the palmar surfaces of the hand, wrist, and forearm
- Assess active, then passive movements
- Examine sensation (see ➋ Chapter 8)
  - Examine the median, ulnar, and radial nerves specifically
- Examine power (see ➋ Chapter 8)
- Test reflexes (see ➋ Chapter 8)
- Check the radial and ulnar artery pulsations
- Assess function
  - Ask the patient to write and do up some buttons
- Thank the patient.
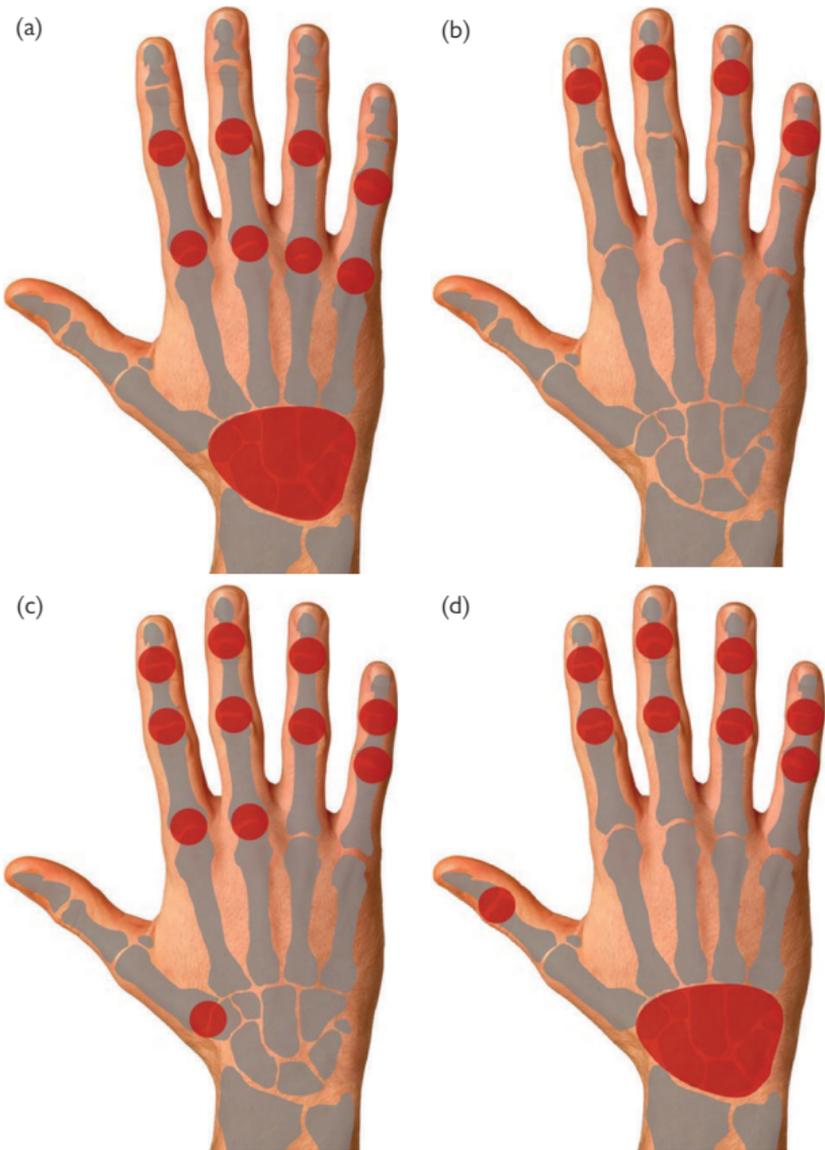
Fig. 10.3 Patterns of joint involvement. (a) Rheumatoid arthritis. (b) Psoriatic arthritis. (c) Osteoarthritis. (d) Gout.
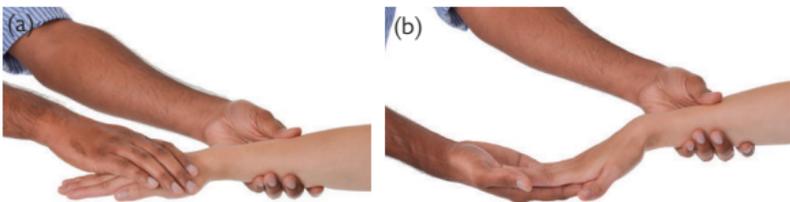


Fig 10.4 Assessing resisted active motion. (a) Wrist extension; (b) wrist flexion.

# 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 from the front, side, and behind, with the patient's arm hanging by side, the forearm supine, and note:
- Skin change (e.g. psoriatic plaques).
- Skin or subcutaneous nodules (e.g. rheumatoid nodules, gouty tophi).
- Scars.
- 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 or Turner's syndrome
  - Fixed flexion deformity (inability to straighten the elbow completely): can be caused by synovitis in inflammatory arthritis or by joint damage in inflammatory arthritis or osteoarthritis.
- Muscle wasting.
- Swelling.
  - Synovial swelling is seen on the lateral aspect of elbow, around the radial head, or around the posterior para-olecranon fossa (felt lateral and medial to the olecranon process)
  - Bursal swelling occurs at the olecranon bursa posteriorly.

### Feel

▶ Always ask about pain before getting started.

Palpate the joint posteriorly and laterally and feel for:
- Temperature.
- Subcutaneous nodules (rheumatoid nodules, gouty tophi).
- Swelling.
  - Soft swelling may be due to olecranon bursitis (e.g. sepsis, gout)
  - Boggy swelling suggests synovial thickening (e.g. rheumatoid)
  - Hard swelling suggests a bony deformity
  - If fluid is present, attempt to displace it on either side of olecranon.
- Tenderness.
  - Carefully palpate the joint margin and epicondyles. Note the exact location of any pain.
- Crepitus.
  - Palpate posterolaterally during flexion/extension and pronation/supination.
- Ulnar nerve.
  - Feel medially for ulnar nerve subluxation (occurs with a 'snap' during flexion/extension; palpate medially for this)
  - Palpate the ulnar nerve for any thickening.

### 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. Test active movements before passive movements.

- 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 ceiling).
  - This position fixes the upper arm, and prevents any trick movement of the upper arm across the abdomen.

*Resisted active motion*

- With the elbow flexed at 90° and prone, hold the patient's forearm still. Ask the patient to extend their wrist against resistance (Fig. 10.4).
  - This reproduces pain in lateral epicondylitis (tennis elbow).
- In the same position, ask the patient to flex the wrist against resistance.
  - This reproduces pain in medial epicondylitis (golfer's elbow).
- With the elbow flexed at 90° ask the patient to supinate, or flex their elbow against resistance.
  - This causes pain in distal bicipital tendonitis.

### Measure

- Measure elbow flexion and extension in degrees from the neutral position (i.e. consider a straight elbow joint to be 0°).
- The normal ranges of movements at the elbow are:
  - 0–150° for flexion/extension
  - 0–85° for pronation
  - 0–90° for supination.

### Function

Observe the patient pour a glass of water and then put on a jacket.

# Shoulder

### Look

Look around for any aids or adaptations. Ask the patient to remove any covering clothing and expose the upper limbs, neck, and chest. Scan the patient from top to toe. Inspect from the front, side, and behind.

Look especially for:
● Scars.
● Bruising or other skin/subcutaneous tissue changes.
● Contours.
   • Look for winging of the scapula, prominence of the acromioclavicular joint and muscle wasting in the deltoid or short rotators which overlie the upper and lower segments of the scapula (rotator cuff pathology)
   • Generalized atrophy of shoulder muscle suggests painful shoulder arthropathy, or brachial neuritis.
● Joint swelling.
   • This may be a clue to acute bleeds, effusions, pseudogout, or sepsis
   • Sub-deltoid/subacromial bursa swelling appears on the lateral aspect of the shoulder.
● Attitude/deformity: Look at the position of both shoulders looking for evidence of dislocation.
   • Posterior dislocation: the arm is held in an internal rotation
   • Anterior dislocation: the arm is displaced antero-inferiorly
   • In advanced glenohumeral arthropathy, the attitude of shoulder is internal rotation, and adduction.
● ▶ 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, glenohumeral joint, spine of scapula, greater tuberosity of humerus.
   ● Check the interscapular area for pain.
   ● Palpate the supraclavicular area for lymphadenopathy.

### Move

▶ Remember to test active movements (the patient does the moving) before passive movements (you do the moving) at each stage.

*Compound movements*

These may be employed as screening tests to assess shoulder dysfunction. See Fig. 10.5.
   ● Ask the patient to put both hands behind the head (flexion, external rotation, and abduction).
   ● Ask the patient to reach up their back with the fingers to touch a spot between their shoulder blades (extension, internal rotation, and adduction).

*Glenohumeral movements*

🛈 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.

Quantify any movement in degrees (measure).

• *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 external rotation suggests adhesive capsulitis).

## 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. Similar symptoms may also occur in subacromial bursitis.

*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.
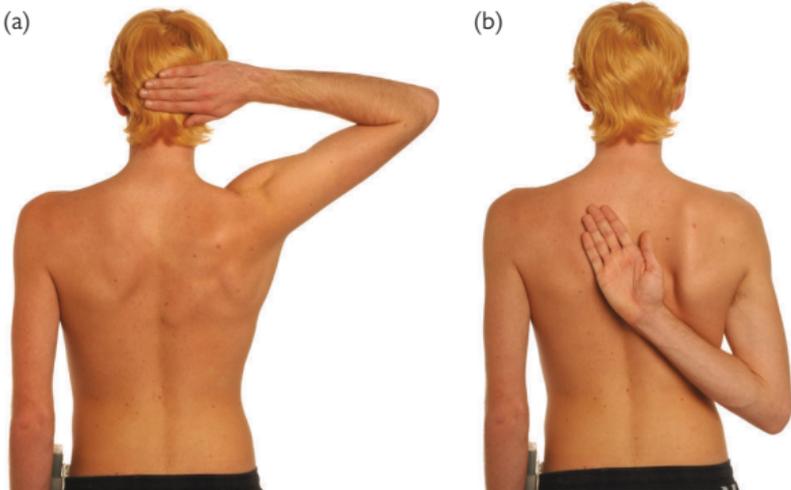


**Fig 10.5** Compound movements. (a) Flexion, external rotation, and abduction. (b) Extension, internal rotation, and adduction.

# 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. Ask the patient to walk, turn around, and walk back. 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.
- Muscle spasm.
- Height of iliac crest on each side.
- Asymmetry including abnormal spinal:
  - *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. This may be postural (corrects on anterior flexion) or structural (unchanged or worsened on flexion).

► 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 and spasm. Apply firm pressure using your thumb to elicit tenderness arising from facet joint arthritis.

You should also make a point of palpating the sacro-iliac 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 (0–80°).
- *Extension:* ask the patient to look up to the ceiling (0–50°).
- *Lateral flexion:* ask the patient to lean their head sideways, placing an ear on their shoulder (0–45°).
- *Rotation:* ask the patient to look over each shoulder (0–80°).

### T- and L-spine

- *Flexion:* ask the patient to touch their toes, keeping knees straight.
- *Extension:* ask the patient to lean backwards (10–20°).
- *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.
- Make a small pen mark at the midline 5cm below and 10cm above this.
- Now instruct the patient to bend at the waist to full forward flexion.
- Measure the distance between the two marks using a tape measure.
- The distance should have increased to >20cm (an increase of >5cm).

*Modified Schober's test*
- As above but only the 10cm segment above the level of posterior superior iliac spines is measured. Increase >3.5cm on flexion is normal.

*Chest expansion*
- See ⟶ Chapter 6. Expiration to peak inspiration should be ≥5cm.

*Occiput to wall distance*
- Ask the patient to stand upright with their heels and back touching the wall, looking straight forward with the chin at the usual carrying level.
- Ask them to try to touch the wall with the back of their head.
- Measure the distance between the occiput and the wall.
  - Any gap suggests thoracic kyphosis or fixed cervical flexion.

*Other measurements*

Students will not be expected to perform the following, but should be aware that they are used in patients with spondyloarthropathies.
- Tragus to wall distance.
- Cervical rotation (in degrees).
- Lateral flexion (measure of lumbar spine mobility).

## Special tests

*Sciatic nerve stretch test*

This test is used to look for evidence of nerve root irritation (Fig. 10.6).
- With the patient lying supine, hold the ankle and lift the leg, straight, to 70–80°. Once at 70–80°, or at an angle at which pain is felt, dorsiflex the foot (Bragard test). If positive, pain will be felt at the back of the thigh, radiating to below the knee.
  - The pain may be relieved by knee flexion, returning the foot to a neutral position, or by reducing the degree of flexion at the hip.
- A positive stretch test suggests tension of the nerve roots supplying the sciatic nerve, commonly over a prolapsed disc (L4/5 or L5/S1).

*Femoral nerve stretch test*

With the patient lying prone, 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.

*Sacro-iliac joint distraction test*
- With the patient lying supine, apply firm outward pressure to both iliac crests at the anterior superior iliac spines.
  - Pain in the buttocks suggests sacro-iliac joint arthropathy.

### Skills station 10.2

*Instruction*

Examine this patient's spine.

*Model technique*
- Clean your hands
- Introduce yourself
- Explain the purpose of examination, obtain informed consent
- Ask for any painful areas you should avoid
- Ask the patient to undress to underwear
- Ask the patient to walk. Watch the gait
- With the patient standing, inspect from back and side
- Palpate the spinous processes individually
- Test active movements
- Perform Schober's test
  - If positive, perform other tests to assess restriction in spinal movements
- Ask the patient to lie on the examination couch
- Perform the straight leg raise test
- Examine sensation and reflexes in the lower limbs
- Assess function
  - Ask the patient to pick something up from the floor
  - Ask about how they manage to turn in bed
- Thank the patient.
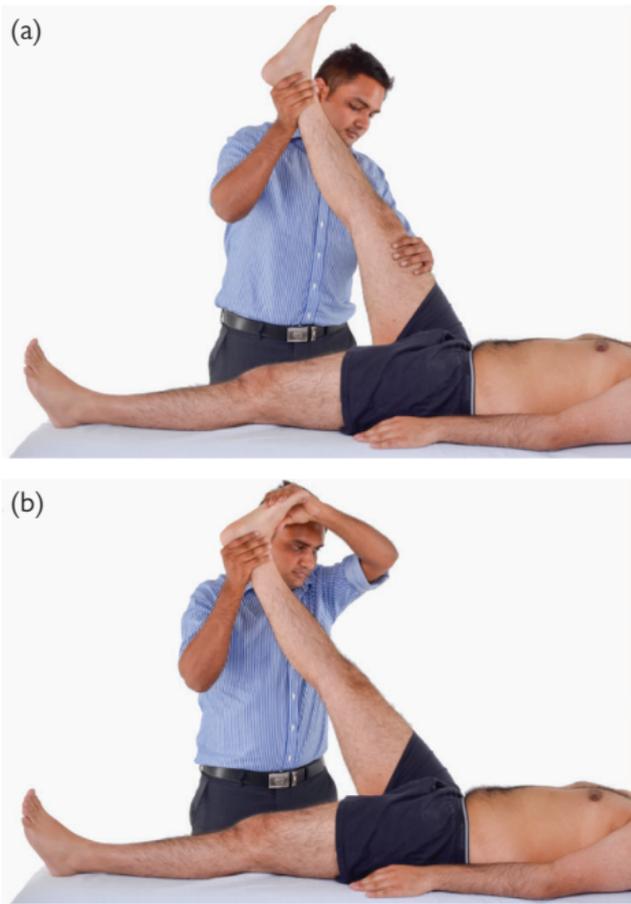
**Fig. 10.6** Sciatic nerve stretch test. (a) With the patient supine, hold the ankle and lift the leg, straight, to 70–80° or until pain is felt. (b) Dorsiflex the foot (Bragard test). If positive, pain will be felt at the back of the thigh, radiating to below the knee.

# 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.

Pay attention to the position of the limbs (e.g. external rotation, pelvic tilting, standing with one knee bent, or foot held plantar-flexed/in equinus).

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/attitude.
  - Patients with structural hip arthropathy (e.g. advanced osteoarthritis) tend to hold the hip joint in flexion, external rotation, and abduction. A similar posture is adopted in hip synovitis.

### 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 (usually elicited lateral to the femoral pulse) and over the greater trochanter.

### 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.

*Active movements*

With the patient *supine*:
- *Flexion:* ask the patient to flex the hip until the knee meets the abdomen, normal is around ~100–135°.
- *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°.
- *Internal rotation:* ask the patient to keep the knees together and point the feet towards each other, normal is 30°.
- *External rotation:* ask the patient to keep the knees together and point the feet as far apart as possible, normal is 15–30°.

With the patient *prone:*
- *Extension:* ask the patient to raise each leg off the bed, normal is 15–30°. (This movement is not routinely measured in clinical practice.)

*Passive movements*

Most should be assessed by the examiner as for active movements whilst the patient is in a relaxed state. With the patient supine:

- *Passive flexion:* flex the hip and knee simultaneously. (This relaxes the hamstrings and avoids performing a 'straight leg raising test'.)
- *Passive external and internal rotation:* flex the knee and hip to 90°, hold the knee with one hand, and move the ankle away or towards the midline with the other.
  - In hip arthropathy, internal rotation of the hip is the first movement to be restricted.
- *Passive abduction and adduction:* examine with the limb in neutral.

## 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.

*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 (or the umbilicus) to the medial malleolus.
- A difference of 1cm is considered abnormal.

## 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. Thomas's test will expose any flexion deformity.

- With the patient 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 affected leg flexes too (measure the angle relative to the bed).
- Remember to repeat the test on the other hip.

## Function

Assess gait. See ➲ Chapter 8.

# 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 (e.g. genu valgus, genu varus, fixed flexion, or hyperextension 'genu recurvatum').
- 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'*
- With the knee extended and the quadriceps relaxed, gently milk any synovial fluid from suprapatellar pouch downwards into the retropatellar space (Fig. 10.7).
- Now, holding the patella still, empty the medial joint recess using a wiping motion of your palm.
  - 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.
  - If there is fluid present, a distinct bulge should appear on the flattened, medial surface as it is milked out of the lateral side.

*Examining for a large effusion—the 'patellar tap'*
If the effusion is tense or 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 (Fig. 10.8).
  - 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.
- With the first one or two fingers of the other hand, push the patella down firmly.
  - 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 active movements (the patient does the moving) before passive movements (you do the moving) at each stage. Quantify any movement in degrees (measure).

- *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, ask the patient to relax. Ensure that you are not causing the patient any discomfort.
- *Passive movements:* Feel over the knee with one hand for any crepitus.

## 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. 25cm above the tibial tubercle.



**Fig. 10.7** 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.
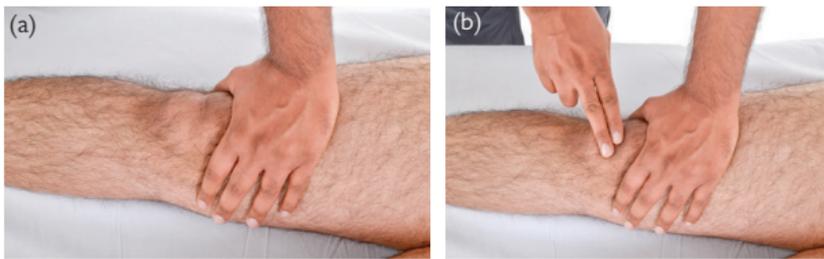


**Fig. 10.8** Testing for 'patellar tap'. (a) Use the palmar surface, thumb, and index finger of one hand to move any fluid into the retropatellar space. (b) Attempt to 'tap' the patella on the femur using the other hand.

## Special tests

*Testing for medial and lateral collateral ligament instability*
Normally, the joint should move no more than a few degrees laterally, excessive movement suggests a torn or stretched collateral ligament.
- With the patient's legs extended, take the foot in your right hand.
- Hold the patient's extended knee firmly with the other hand.
- Attempt to bend the knee medially (varus) whilst feeling the lateral knee joint line.
  - This tests the lateral collateral ligament.
- Attempt to bend the knee laterally (valgus), feeling the medial joint line.
  - This tests the medial collateral ligament.
- Repeat the above with the knee at 30° flexion.
  - In this position, only the lateral and medial collateral ligaments (and not the cruciates) contribute to varus–valgus stability at the knee.

*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.
- Ensure the patient is lying in a relaxed supine position.
- Ask the patient to flex the knee to 90°.
  - In an anterior cruciate ligament tear, the tibia sags posteriorly in this position ('sag sign').
- 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 (Fig. 10.9a).
- 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 a meniscus is torn, a tag of cartilage may become trapped and cause pain and an audible (and palpable) 'click'.

*Apley's test*
Another test for meniscal tears. If 'positive', the test will produce pain.
- Position the patient prone with the knee flexed to 90° (Fig. 10.9b).
- Stabilize the thigh with your left hand.
- With the right hand, grip the foot and rotate or twist it whilst pressing downwards in a 'grinding motion'.
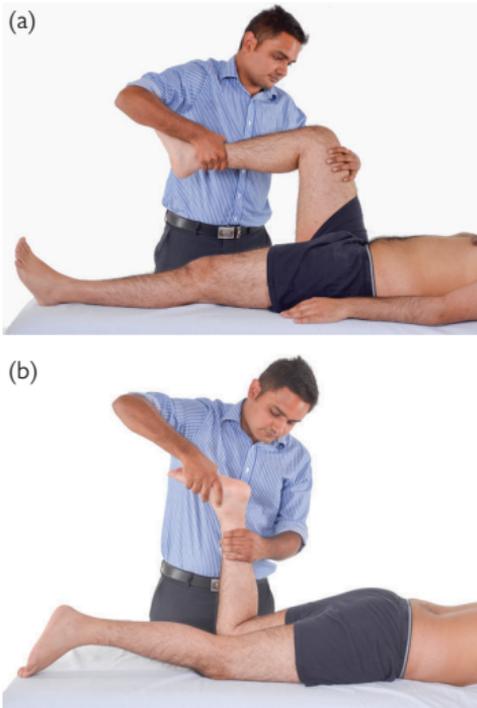
**Fig. 10.9** Testing for meniscal tears. (a) McMurray's test. (b) Apley's grinding test.

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**Skills station 10.3**

*Instruction*

Examine this patient's knees.

*Model technique*
- Clean your hands
- Introduce yourself
- Explain the purpose of examination, obtain informed consent
- Ask for any painful areas you should avoid
- Ask the patient to expose their lower limbs including the ankles and feet, and stand up
- Look for knee alignment and swellings
- Ask the patient to walk. Watch the gait
- Ask the patient to lie on the couch. Inspect the knees again
- Palpate the knees for warmth
- Examine for effusions
- Feel for localized tenderness
- Test active and passive movements
- Test for integrity of the cruciate and collateral ligaments
- To assess function, ask the patient to stand from sitting and ask how they find climbing stairs.

# Ankle and foot

## Look

Expose the lower limbs and make note of any walking or other aids. Take a moment to also examine the footwear for any adaptations, abnormal wear, or stretching.

Examine the feet and ankles with the patient lying on a couch or bed. Look from front, sides, back, and inspect the plantar surface.

Also examine the feet and ankle when the patient is standing up. Look from front, sides, and back. If you think the patient can, ask them to stand on tip-toes and then on their heels while you watch the foot from behind and from the sides.

Finally, watch the patient walk.

Look for:
- Skin or soft-tissue lesions including calluses, swellings, ulcers and scars.
- Muscle wasting at the calf and lower leg.
- Swellings.
- Deformities.
- 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 clubbing of the feet (talipes equinovarus).

### *Swellings*
- *Ankle synovitis:* diffuse anterior swelling ± lateral or medial extension.
- *Tenosynovitis:* tubular swelling oriented longitudinally along a tendon.
  - Anterior: tibialis anterior, extensor hallucis, or extensor digitorum
  - Posterior to medial malleolus: tibialis posterior, flexor digitorum, or flexor hallucis
  - Posterior to lateral malleolus: peroneal tendons.
- *Achilles tendinopathy/tear, retrocalcaneal and retroachilles bursitis:* posterior to calcaneum.

### *Deformities*
Deformities involving the arch and hindfoot are better appreciated with the patient standing. Look for:
- *Hallux valgus:* 'bunion'. Medial deviation of the 1st metatarsal and lateral deviation and/or rotation of the great toe. Commonly bilateral.
- *Hammer toes:* flexion deformity at the PIPJ of the affected toe(s) (commonly the 2nd toe), hyperextension of the MTPJ and DIPJ. Caused by an overpull of the extensor digitorum longus tendon.
- *Claw toes:* Extension contracture with dorsal subluxation of the MTPJ, flexion deformities of the PIPJ and DIPJ.
  - Often idiopathic. Often elderly females with diabetes or RA.
- *Mallet toes:* A flexed DIPJ (commonly the 2nd toe). May exist in conjunction with a claw-toe deformity.
- *Pes planus (flat foot):* a lack of the normal plantar arches.
  - Physiologic pes planus (but not pathologic) may correct when standing on tip-toes.
- *Pes cavus (high-arched foot):* exaggeration of the plantar arches.

## 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 the ankle bony prominences (lateral and medial malleoli, MTPJs, interphalangeal joints, and heel) as well as the metatarsal heads.
- Squeeze across the MTPJs, 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 to their head (normal ~20°).
- *Ankle plantar flexion:* ask the patient to push the toes down towards the floor 'like pushing on a pedal' (normal ~45°).
- *Inversion:* (subtalar joint between the talus and calcaneum). Ask the patient to turn their sole inward (you may have to demonstrate this) (normal ~20°).
- *Eversion:* as inversion but turn the sole outwards, away from the midline (normal ~10°).
- *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.

*Passive movements*

Palpate for crepitus. Whilst checking inversion and eversion passively, grasp the ankle with one hand and with the other, grasp the heel, and turn the sole inwards towards the midline and then outwards.

## 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 plantar-flex. 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 first.

# Important presentations

## Rheumatoid arthritis (RA)

RA is a chronic inflammatory multisystem autoimmune disease mediated by pro-inflammatory cytokines such as tumour necrosis factor alpha (TNF-$\alpha$) and may associate with antibodies such as rheumatoid factor and anti-CCP. However, it is worth remembering that between 30–40% of patients with rheumatoid arthritis are not positive for rheumatoid factor. A smaller proportion of patients (10–20%) does not develop anti-CCP antibodies.

Usually the onset of symptoms in RA is over a few days to a few weeks, and the progression is slow. Additional joints are involved over weeks and months. It can rarely have an acute onset (over a day or two). The course can be episodic with complete resolution between attacks (palindromic). The clinical features of RA can be divided into articular and extra-articular features summarized below.

### Demographics

- Affects around 1–3% of the population.
- Occurs in all races.
- Peak age of onset in the 4th and 5th decades.
- Female:male ratio ~3:1.

### 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 mono- or oligo-arthritis of larger joints such as the knees, wrists, shoulders, or elbows. Common symptoms are joint pain, stiffness, and swelling which are typically worse in the morning and improve as the day progresses.

### Signs of RA

Synovitis involving the wrists (dorsal swelling), metacarpo-phalangeal (filling in of gaps between the MCP heads) and proximal interphalangeal joints (lateral expansion of IPJs), with sparing of the distal interphalangeal and 1st carpometacarpal joints.

With modern and aggressive treatment of synovitis, joint destruction and resulting deformities are not common in patients developing RA in the last decade. However, these deformities may be present in the patients who have had the disease for some years or in some with aggressive uncontrolled RA.

### Inspection:

- Symmetric swelling of proximal interphalangeal (PIP) joints.
- Symmetric swelling of metacarpo-phalangeal (MCP) joints.
  - Ask the patient to make a fist; subtle swelling of MCP joints is seen as filling in of the 'valleys' between metacarpal heads.
- Ulnar deviation at MCP joints.
- Thin skin with scars. (Long-term corticosteroid use.)
- Wasting of intrinsic muscles.

- *Tuck sign:* tubular swelling due to extensor tenosynovitis, seen on the dorsal aspect of wrist and on finger extension.
- Swan-neck deformity.
  - Hyperextension of PIPJ and flexion of DIPJ.
- Boutonnière deformity.
  - Flexion at PIPJ, extension at DIPJ.
- Volar subluxation at MCP and wrist joints.
- Rheumatoid nodules on extensor tendons, joints, sites of mechanical irritation (elbow, toe, and heel).

*Palpation:*
- Warmth and tenderness at DIP, PIP, MCP, and wrist joints (if active).
- 'Doughy' feeling of synovial proliferation at joints.
- *Piano key sign:* up and down movement of the ulnar styloid in response to pressure from examiners' fingers.

### Extra-articular features of RA
Extra-articular features are the systemic manifestations of RA which are unique to, and caused by, the immune-pathological process of RA.

*Common:*
- Rheumatoid nodules: common at sites of pressure (elbows and wrists). Associated with more severe disease and rheumatoid factor positivity.
- Sjögren's syndrome (keratoconjunctivitis sicca).
- Raynaud's phenomenon.
- Interstitial lung disease (pulmonary fibrosis, pulmonary nodules).
- Pleurisy/pleural effusions.
- Episcleritis/scleritis.

*Uncommon:*
- Neurological features:
  - Mononeuritis multiplex
  - Peripheral neuropathy.
- Cardiac features:
  - Pericarditis/pericardial effusions.
- *Systemic features* (fever, malaise, weight loss, and lymphadenopathy).

*Rare:*
- Vasculitis:
  - Nail-fold infarcts
  - Cutaneous ulceration
  - Digital gangrene.
- Skin lesions:
  - Pyoderma gangrenosum.
- Lung features:
  - Caplan's syndrome (massive lung fibrosis in RA patients with pneumoconiosis)
  - Obliterative bronchiolitis
  - Felty's syndrome (RA, splenomegaly, and neutropenia).
- Amyloidosis (proteinuria, hepatosplenomegaly).

*Complications of RA*

Extra-articular features of RA should be distinguished from 'complications'. These are consequences of joint inflammation, systemic inflammation, or drug treatment and include:

- Anaemia (Box 10.15).
- Cataracts (chloroquine, steroids).
- Peripheral nerve entrapment (e.g. carpal tunnel syndrome).
- Cervical myelopathy (atlanto-axial subluxation).
- Palmar erythema, skin thinning, and muscle wasting (synovitis in nearby joints).

Recently, non-Hodgkin's lymphoma (systemic inflammation), ischaemic heart disease (systemic inflammation), osteoporosis, and a propensity to lower respiratory tract infections have been recognized as complications of RA.

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**Box 10.15 Causes of anaemia in RA**

- Anaemia of chronic disease
- GI bleeding
  - Non-steroidal anti-inflammatory drugs (NSAIDs) or corticosteroid use.
- Bone marrow suppression
  - Disease-modifying anti-rheumatic drugs (e.g. methotrexate).
- Megaloblastic anaemia
  - Due to folic acid deficiency or pernicious anaemia.
- Macrocytic anaemia.
  - Methotrexate, azathioprine.

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## 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 has a female preponderance.

Secondary causes of OA include:

- Trauma (fracture, meniscal, or cruciate injury).
- Inflammatory arthritis (e.g. RA).
- Abnormalities in articular contour (hip and acetabular dysplasias) or alignment (varus or valgus knee malalignment).
- Generalized or localized hypermobility (Ehlers–Danlos syndrome, Marfan's syndrome, benign hypermobility syndrome).
- Previous septic arthritis.
- Avascular necrosis.

*Symptoms*

*Common* symptoms include swelling, deformity, stiffness, weakness, and pain which is normally worse after activity and relieved by rest.

*Inspection*

- Posterolateral swelling at the distal interphalangeal (DIP) (Heberden's nodes) and proximal interphalangeal (PIP) (Bouchard's nodes) with characteristic radial or ulnar deviation of the phalanx.
- Squaring of thumb base, wasting of thenar eminence observed on the volar aspect (1st carpometacarpal joint).

*Inspection (knee)*

Patient standing, examine from front. Look for:

- Varus and valgus deformity.
- Suprapatellar and infrapatellar effusion.
- Quadriceps wasting.
- Fixed flexion at knee with the patient lying supine.

*Palpation (hands)*

- Cool bony swelling at IPJs.
- Joint line tenderness at IPJ and 1st CMCJ.

## Paget's disease (osteitis deformans)

A disorder of bone remodelling characterized by ↑ osteoclast and osteoblast activity, leading to accelerated bone resorption and disorganized bone formation.

Paget's disease is more common in males and affects around 1–2% of the Caucasian adults >55 years. It occurs more commonly in the UK than anywhere else in the world. The exact aetiology remains unknown, however a number of factors have been implicated, including a slow viral infection such as paromyxovirus. The axial skeleton is preferentially affected; common sites of involvement include the pelvis, femur, lumbar spine, skull, and tibia in a descending order of frequency.

*Important clinical features and complications*
*Common*

- Pain: bone pain, not joint pain. Pain is present day and night and is not made worse by joint movements.
- Deformity.
  - Enlargement of the skull
  - Exaggerated thoracic kyphosis
  - Anterior bowing of the tibia
  - Lateral bowing of the femur.
- Fractures.
- Hearing loss (ossicle involvement, or VIII nerve compression).

*Less common*

- Spinal stenosis.
- Nerve compression syndromes.

*Rare*

- Hypercalcaemia during immobilization.
- Cardiac failure.
- Sarcomatous change.
- Hydrocephalus.
- Cord compression.

## Crystal arthropathies

*Gout*

A disorder of purine metabolism. Characterized by hyperuricaemia due to either overproduction or underexcretion of uric acid. Prolonged hyperuricaemia (Box 10.16) leads to the deposition of urate crystals in synovium, connective tissues, and the kidney. These crystals are then shed leading to acute gout.

Gout is associated with metabolic syndrome (central obesity, insulin resistance, hypertension, and ischaemic heart disease).

Most patients are middle aged or older with risk factors for gout such as renal failure, excess alcohol intake, and diuretic usage. Causes of premature gout include renal failure, solid organ transplant with immunosuppression with calcineurin inhibitors, haematological malignancy, and inherited errors of metabolism.

*Clinical features of acute gout*
- Sudden onset (hours) of severe pain and swelling classically in the great toe MTPJ, worse at night, and associated with redness.
- Occasionally multiple joints are involved e.g. knees, ankles ± systemic symptoms.
- Some patients (frequently elderly and those on diuretics) present with large-joint (knee, ankle, shoulder, or wrist) involvement or with polyarticular gout.

*Clinical features of chronic (tophaceous) gout*
- Tophi (deposits of urate crystal) occur in:
  - The digits (at IPJs, finger pulp). In the presence of osteoarthritis, gouty tophi preferentially occur at the IPJs affected by Heberden's or Bouchard's nodes.
  - Near the 1st metatarsophalangeal joint
  - In bursae (e.g. olecranon bursa)
  - Near the Achilles tendon
  - In tendon sheaths
  - On the helix (ear). In hands, gouty tophi preferentially occur at the IPJs affected by Heberden's or Bouchard's nodes.

*Calcium pyrophosphate deposition (CPPD)*

CPPD may occur in the cartilage (chondrocalcinosis), joint capsule, and tendons. Established risk factors include ↑ age (>60 years) and osteoarthritis. If CPPD is present in those <55 years or is florid and polyarticular, the patient should be screened for haemochromatosis, hypophosphataemia, hypomagnesaemia, and hyperthyroidism.

Knees, wrists, MCPJs, and hips are the most commonly involved joints. CPPD may present as:
- *Asymptomatic:* chondrocalcinosis.
- *Acute CPP crystal arthritis (formerly 'pseudogout'):* the commonest cause of acute mono-arthritis in the elderly.
- *CPPD and OA:* symptoms of OA with or without superimposed episodes of acute synovitis.
- *Chronic CPP crystal inflammatory arthritis:* uncommon.

**Box 10.16 Causes of hyperuricaemia**

More common in the summer months due to reduced fluid intake and increased fluid loss.

- Drugs: diuretics, ethanol, salicylates, pyrazinamide, ethambutol, nicotinic acid, and ciclosporin
- Chronic renal failure
- Myeloproliferative and lymphoproliferative disorders (↑ purine metabolism)
- Obesity
- Hypertension
- Hypothyroidism
- Hyperthyroidism
- Familial
- Excessive dietary purines.

## 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 clinical features:
- Enthesitis (an enthesis is the insertion of a tendon, ligament, or joint capsule onto a bone).
- 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*
- Back pain (which may be localized to the buttocks) and stiffness which are typically worse in early hours of the night (2am–5am) and on waking up in the morning.
  - Pain recurs after long periods of rest and is relieved by activity. Patients report a dramatic response to NSAIDs, which remains the first line of treatment.
- Chest pain may be present as a result of T-spine involvement as well as enthesitis at the costochondral joints.
- Pain, swelling, and stiffness may be present in peripheral joints affected by inflammatory arthritis e.g. shoulders, hips, knees, and ankles.

*Musculoskeletal features/signs*
- 'Question mark' posture (loss of lumbar lordosis, fixed kyphosis of the T-spine, compensatory hyperextension of the C-spine).
- Protuberant abdomen.
- Schober's test positive.
- Sacroiliac joint tenderness. (SIJ distraction test may be positive.)
- Achilles tendonitis.
- Plantar fasciitis.

*Some extra-skeletal features*
- Anterior uveitis.
- Aortic regurgitation.
- Apical lung fibrosis.
- AV block.
- Amyloidosis (secondary).
- Weight loss.

In some cases, a fracture may occur through the rigid spine and involve the intervertebral discs. A similar lesion may be produced by inflammatory granulation tissue. These are known as disco-vertebral or 'Andersson' lesions.

*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 five main subtypes of psoriatic arthropathy:

- Asymmetrical distal interphalangeal joint arthropathy.
- Asymmetrical large joint mono- or oligo-arthropathy.
- Spondyloarthropathy and sacroiliitis (usually asymmetric).
- Rheumatoid-like hands (clinically identical to RA but seronegative).
- Arthritis mutilans (a destructive form with telescoping of the fingers).

*Associated clinical features*

- Psoriatic plaques (extensor surfaces, scalp, behind the ears, navel and natal clefts).
- Nail involvement (pitting, onycholysis, discoloration, and thickening).
- Dactylitis (sausage-shaped swelling of the digits due to tenosynovitis).
- Enthesitis.

*Reactive arthritis*

- An aseptic arthritis, strongly linked to a recognized episode of infection. Common causes are gut and genitourinary pathogens.
- It mainly affects young adult males and usually presents with an asymmetric oligoarthritis. Symptoms start a few days to a few weeks after the infection.
- Enthesitis and dactylitis are other common features.
- Extra-articular features include urethritis, conjunctivitis, and skin 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, campylobacter, 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 plantar fasciitis.

*Enteropathic arthritis*

Enteropathic arthritis is a peripheral or axial arthritis and is the commonest extra-intestinal manifestation of inflammatory bowel disease. Patients are usually young adults and there is no gender predisposition. The musculoskeletal manifestations include:

- Sacroiliitis (symmetric usually).
- Peripheral arthritis.
- Dactylitis.
- Enthesopathy (Achilles, plantar fascia, costovertebral, costosternal).

Only a minority of patients (7%) are HLA B27 positive. Enteropathic spondyloarthropathy does not typically correlate with the severity of bowel disease. However, in some cases, 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 (Box 10.17). Classification (and treatment) is based on measurement of the bone mineral density (BMD), with comparison to that of a young healthy adult.

The underlying pathology is related to an imbalance between the osteoblasts producing bone and the osteoclasts removing bone which ultimately produces net bone loss. By the World Health Organization definition, patients with a BMD of <2.5 standard deviations below the mean of young adult BMD of the same gender have osteoporosis.

*Primary osteoporosis*

- 95% of osteoporosis in women, 70–80% osteoporosis in men.
- Seen in postmenopausal women and elderly men.
- No single underlying cause of osteoporosis. However, patients may have several risk factors including:
  - Older age (>50 years)
  - Female gender
  - Low dietary calcium and vitamin D intake
  - FHx of osteoporosis
  - Parental history of hip fracture
  - BMI <19 kg/m$^2$
  - Delayed menarche
  - Premature menopause
  - Sedentary lifestyle
  - Excess caffeine intake.

*Secondary osteoporosis*

- Has an identifiable underlying cause of osteoporosis (see Box 10.17).
- In addition, patients may have other identifiable risk factors as for primary osteoporosis.

*Clinical features*

- The process leading to osteoporosis is asymptomatic.
- The condition is diagnosed usually after the patient has a fragility fracture.
  - A fragility fracture is a fracture caused by falling from a standing height or less
  - Common sites of osteoporotic fracture include femoral neck, wrist, and vertebrae
  - Vertebral fracture may be asymptomatic sometimes, being diagnosed only when the patient has a spinal radiograph for kyphosis, loss of height, or for unrelated reasons.

**Box 10.17 Secondary causes of osteoporosis**
- Prolonged immobilization/weightlessness
- Malignancy
- GI diseases: malabsorption syndrome, IBD, liver disease, anorexia nervosa
- Rheumatologic diseases: RA, SLE, AS
- COPD
- Genetic diseases: cystic fibrosis, Ehlers–Danlos syndrome
- Endocrine diseases: diabetes type 1, hyperparathyroid, hyperthyroid, hyperprolactinaemia, Cushing's syndrome, hypogonadism
- Drugs: corticosteroids, phenytoin, long-term heparin
- Alcohol (>recommended daily allowance) and smoking.

**See also:**
More information regarding the presentation and clinical signs of locomotor diseases to aid preparation for OSCE-type examinations and ward rounds can be found in the *Oxford Handbooks Clinical Tutor Study Cards*.

*'Medicine' Study Card set:*
- Rheumatoid arthritis
- Osteoarthritis
- Psoriatic arthritis
- Ankylosing spondylitis
- Paget's disease
- Tophaceous gout
- Marfan's syndrome
- Systemic sclerosis
- Vasculitides
- Systemic lupus erythematosus
- Rickets.

*'Surgery' Study Card set:*
- Dupuytren's contracture
- Hammer toe
- Claw toes
- Mallet toe
- Mallet finger
- Trigger finger
- Olecranon bursitis
- Swellings around the knee
- Osteochondroma
- Pes cavus
- Charcot's joint
- Hallux valgus.

# 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 also to 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. Myriad causes contribute to back pain, but never forget deep-seated infection such as discitis or osteomyelitis which may be a consequence of something as 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 sections. 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:* 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' (Box 10.18), a well-validated test of gait and balance. This assessment should overlap with neurological assessment when appropriate. (See Box 10.19.)

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**Box 10.18 Get up and go**

An easy test to do, and one which gives a wealth of information. Ask the patient to perform the following 3-part task:
- 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.

---

**Box 10.19 A word on labels and respect**

It is a sad fact that we still see patients labelled with awful terms such as 'acopia' or 'social admission' after failing and/or falling at home. They are not to be used as they reflect:
- Your (and your seniors') limited thinking
- Missed diagnoses
  - Such as infections, overmedication, pain, fractures.
- A lack of respect.
  - Older people would almost certainly prefer not to be in hospital and it is extremely rare that the reason for admission is their 'fault'.

▶ Do your best for them with as thorough and detailed an assessment as you would do for any other patient presentation.

▶ Consider using your own 'family and friends test'. Would you be satisfied if these labels were attached to an older person you knew who had been admitted acutely to hospital?

# The ear, nose, and throat

# Introduction

## Ear

The ear is involved in both balance and hearing and is divided into the external, middle, and inner ear.

### The external ear

This is composed of the pinna (auricle), external auditory meatus, and the lateral wall of the tympanic membrane.

The auricle is divided into the antihelix, helix, lobe, tragus, and concha and is composed of fibrocartilage; the ear lobe is adipose only.

The tympanic membrane is a thin, grey, oval, semitransparent membrane at the medial end of the external acoustic meatus ~1cm in diameter. It detects air vibrations (sound waves). These tiny movements are then transmitted to the auditory ossicles.

### The middle ear

This lies in the petrous part of the temporal bone and is connected to the nasopharynx via the Eustachian tube. It connects with the mastoid air cells. The tympanic cavity contains three tiny bones (ossicles: the malleus, incus, and stapes) which transmit vibrations to the cochlea and two small muscles (stapedius and tensor tympani). The chorda tympani branch of the facial nerve passes through here before it exits the skull.

### The inner ear (vestibulocochlear organ)

This is involved in the reception of sound and the maintenance of balance. It consists of a series of interconnecting bony-walled fluid-filled chambers (vestibule, semicircular canals, and cochlea). Within the bony labyrinth is a further series of interconnecting membranous chambers (membranous labyrinth: saccule, utricle, cochlear duct, and semicircular ducts).

The vestibule and semicircular canals contain the peripheral balance organs. These have connections to the cerebellum and are important in the maintenance of posture and fixed gaze. The sensory impulses are conducted by the cochlear and vestibular divisions of cranial nerve VIII.

## Nose and paranasal sinuses

The main functions of the nose and nasal cavities are olfaction, respiration, and air filtration.

The upper 1/3 of the external nose is bony; the rest is cartilaginous. The inferior surface holds the anterior nares (nostrils) which are separated from each other by the bony/cartilaginous nasal septum. The lateral wall of each cavity supports a series of three ridges called turbinates (superior, middle, and inferior).

The paranasal sinuses are air-filled extensions of the nasal cavity. They are named according to the bones in which they are located: frontal, ethmoid, sphenoid, and maxilla (see Fig. 11.1). Their purpose is thought to include protection of intracranial structures and the eyes from trauma, as an aid to vocal resonance, and reduction of skull weight.

#### Mouth and throat

*The oral cavity* comprises the lips, the anterior 2/3 of the tongue, hard palate, teeth, and alveoli of the mandible and maxilla.

The tongue is involved with mastication, taste, swallowing (deglutination), and articulation. Two sets of teeth develop within a lifetime. The first set is deciduous (milk teeth). The incisors are the first to erupt at ~6 months; the rest follow within 3 years. In the permanent set, the first molar or central incisor erupts first (~6 years), the second molar erupts ~11 years of age; the third molar emerges ~18 years (wisdom teeth).

*The pharynx* is divided into three parts: nasopharynx, oropharynx, and hypopharynx. The nasopharynx lies posterior to the nose and superior to the soft palate. The oropharynx lies posterior to the oral cavity, extending from the soft palate to the epiglottis, and contains the tonsils. The hypopharynx lies posterior to the larynx, extending from the epiglottis to the inferior border of the cricoid cartilage, where it is continuous with the oesophagus.

*The larynx* lies at the level of the bodies of C3–C6 vertebrae. It connects the inferior part of the pharynx with the trachea. It functions to prevent food and saliva entering the respiratory tract and as a phonating mechanism for voice production. It is supported by a framework of hyaline cartilage connected by ligaments. The thyroid cartilage is the largest of the laryngeal cartilages and can be seen as the 'Adam's apple'. The nervous supply of the larynx is from cranial nerve X (sensory and motor).

*The epiglottis* is attached to the thyroid cartilage and occludes the laryngeal inlet during swallowing.

#### Salivary glands

The main salivary glands are the parotid, submandibular, and sublingual. The sublingual glands are the smallest and their ducts open on to the floor of the mouth—as do the ducts of the submandibular glands. The parotid glands are the largest. The parotid ducts cross the masseter muscles and open into the oral cavity opposite the upper 2nd molar teeth.
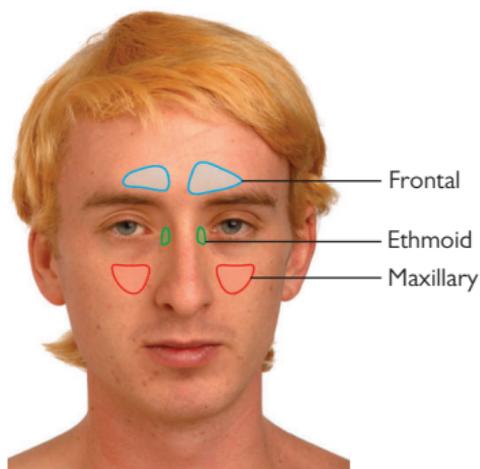


**Fig. 11.1** The surface anatomy of the facial sinuses.

# Symptoms of ear disorders

## Otalgia

You should take a standard 'pain' history as in �'Chapter 2.

Ask about associated discharge, hearing loss, previous ear operations, or ear syringing, use of cotton buds, trauma, swimming, and air travel.

▶ Remember that the ear has a sensory supply from cranial nerves V, IX, X, and the 2nd and 3rd cervical nerves so otalgia may be referred from several other areas (Box 11.1).

---

### Box 11.1  Some causes of otalgia

*Otological*
- Acute otitis externa
- Acute otitis media
- Perichondritis
- Furunculosis
- Trauma
- Neoplasm
- Herpes zoster (Ramsay Hunt syndrome).

*Non-otological (referred)*
- Cervical spine disease
- Tonsillitis
- Dental disease
- Temporo-mandibular joint disease
- Neoplasms of the pharynx or larynx.

---

## Otorrhoea

This is discharge from the external auditory meatus. Ask about other ear symptoms, when the discharge began, and any precipitating or exacerbating factors. Ask especially about the nature of the discharge:
- *Watery:* eczema, CSF.
- *Purulent:* acute otitis externa.
- *Mucoid:* chronic suppurative otitis media with perforation.
- *Mucopurulent/blood-stained:* trauma, acute otitis media, cancer.
- *Foul-smelling:* chronic suppurative otitis media ± cholesteatoma.

## Dizziness

The term 'dizziness' can mean different things to different people and must be distinguished from light-headedness, pre-syncope, and pure unsteadiness. Two features of the dizziness suggest that it arises from the vestibular system:
- Vertigo (a hallucination of movement, most commonly rotational).
- Dizziness related to movement or position change.

Both these symptoms can occur together, separately in time, or alone in different people. Disequilibrium (unsteadiness or veering) may accompany vestibular dizziness.

*Important points from the history*

You should obtain a precise history, aiming to establish whether or not the dizziness is due to vestibular disease (Box 11.2). Ask about:

- The nature and severity of the dizziness.
- Whether it is persistent or in intermittent 'attacks'.
- The duration of attacks (seconds, hours, or days).
- The pattern of events since the onset.
- Relation to movement or position, especially lying down.
- Associated symptoms (e.g. nausea, vomiting, hearing change, tinnitus, headaches).
- DHx including alcohol.
- Other ear problems or previous ear surgery.

*Peripheral vestibular lesions*

Vertigo caused by vestibular problems is most commonly rotational, but may be swaying or tilting. Whether it is movement of the person or surroundings is irrelevant.

Any rapid head movement may provoke the dizziness, but dizziness provoked by lying down, rolling over, or sitting up is specific to benign paroxysmal positional vertigo.

*Central vestibular lesions*

These are not always easy to distinguish on the history but vertigo is not so marked and gait disturbances and other neurological symptoms and signs would suggest this.

---

**Box 11.2 Some causes of dizziness**

*Otological*

- Benign paroxysmal positional vertigo
- Ménière's disease
- Vestibular neuronitis
- Trauma (surgery or temporal bone fracture)
- Perilymph fistula
- Middle ear infection
- Otosclerosis
- Syphilis
- Ototoxic drugs
- Acoustic neuromas.

*Non-otological*

- These are often more disequilibrium than dizziness
- Ageing (poor eyesight and proprioception)
- Cerebrovascular disease
- Parkinson's disease
- Migraine
- Epilepsy
- Demyelinating disorders
- Hyperventilation
- Drugs (e.g. cardiovascular, neuroleptic drugs, and alcohol).

## Hearing loss

Deafness or total hearing loss is unusual. Hearing loss is usually described as being mild, moderate, or profound.

Hearing loss may be conductive, sensorineural, mixed, or non-organic.

Conductive hearing loss may be due to pathology of the ear canal, eardrum, or middle ear. Sensorineural hearing loss is caused by disease in the cochlea or the neural pathway to the brain. (See Box 11.3.)

You should take a full history as in ➲ Chapter 2. In particular, note:

- *PC:* As well as the usual questions, establish:
  - The time and speed of onset
  - Is it partial or complete?
  - Are both ears affected or just one?
  - Is there associated pain, discharge, or vertigo?
- *PMH:* especially tuberculosis and septicaemia.
- *FHx:* hearing loss may be inherited (e.g. otosclerosis).
- *DHx:* certain drugs, particularly those which are toxic to the renal system, affect the ear (e.g. aminoglycosides, some diuretics, cytotoxic agents). Salicylates and quinine show reversible toxicity.
- *SHx:* occupation and leisure activities should not be overlooked. Prolonged exposure to loud noise (e.g. heavy industrial machinery) can lead to sensorineural hearing loss. Levels of 90dB or greater require ear protection.

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### Box 11.3  Some causes of hearing loss

*Conductive*

- Wax
- Otitis externa, if ear is full of debris
- Middle ear effusion
- Trauma to ossicles
- Otosclerosis
- Chronic middle ear infection (current or previous)
- Tumours of the middle ear.

*Sensorineural*

- Presbyacusis
- Vascular ischaemia
- Noise exposure
- Inflammatory/infectious diseases (e.g. measles, mumps, meningitis, syphilis)
- Ototoxicity
- Acoustic tumours (progressive unilateral hearing loss, but may be bilateral).

---

*Non-organic hearing loss*

❶ Only diagnose after fully excluding an organic cause. In such cases, there may be a discrepancy between the history and clinical and audiometric findings.

### Tinnitus

As well as the full standard history, ask the patient about the character of the tinnitus, associated hearing loss, how the tinnitus bothers them (i.e. is sleep or daily living affected) and any previous history of ear disease as well as the full standard history. (See Box 11.4.)

- *Rushing, hissing, or buzzing* tinnitus is the commonest and usually associated with hearing loss. It is caused by pathology in the inner ear, brainstem, or auditory cortex (although it can sometimes appear with conductive hearing loss).
- *Pulsatile* tinnitus is caused by noise transmitted from blood vessels close to the ear. These include the internal carotid artery and internal jugular vein (the latter can be diagnosed by abolition of the noise by pressure on the neck). Occasionally, pulsatile tinnitus can be heard by an observer by using a stethoscope over the ear or neck.
- *Cracking and popping* noises can be associated with dysfunction of the Eustachian tube or rhythmic myoclonus of the muscles in the middle ear or attached to the Eustachian tube.

▶ Remember to distinguish tinnitus from complex noises (e.g. voices, music) which may constitute auditory hallucinations.

---

**Box 11.4 Some causes of tinnitus**

- Presbyacusis
- Noise-induced hearing loss
- Ménière's disease
- Ototoxic drugs, trauma
- Any cause of conductive hearing loss
- Acoustic neuromas.

*Pulsatile tinnitus*
- Arterial aneurysms
- Arteriovenous malformations.

---

### Injury to the ear

- Trauma may be self-inflicted, especially in children, when foreign bodies inserted in the ear can damage the meatal skin or the eardrum.
- Head injuries can cause temporal bone fractures, with bleeding from the ear and may be associated with dislocation of the ossicles, or may involve the labyrinth causing severe vertigo and complete deafness.
- Temporary or permanent facial nerve palsy may also occur.

### Deformity of the ear

- This may be either congenital or acquired (usually traumatic).
- Complete or partial absence of the pinna (anotia or microtia), accessory auricles (anterior to the tragus), or a pre-auricular sinus. Protruding ears may cause social embarrassment and can be surgically corrected.
- Small auricles are seen in Down's syndrome—often with a rudimentary or absent lobule.

# Symptoms of nasal disorders

### Nasal obstruction

As well as the full standard history (⬧ Chapter 2), establish:
- Is the nose blocked constantly or intermittently?
  - Constant: long-standing structural deformity such as deviated septum, nasal polyps, or enlarged turbinates
  - Intermittent: allergic rhinitis or common cold.
- Unilateral or bilateral obstruction?
- Associated nasal discharge.
- Relieving or exacerbating factors.
- Use of nose drops or any other 'per-nasal' substance (e.g. glue-sniffing or drug-snorting).
- ▶ Don't miss a previous history of nasal surgery.

### Nasal discharge

Ask about the specific character of the discharge which is often very helpful in deciding aetiology. See Box 11.5.

🔔 The terms 'catarrh' and 'postnasal drip' should be reserved only for complaints of nasal discharge pouring backwards into the nasopharynx.

### Epistaxis

This is a nasal haemorrhage or 'nose-bleed'. The anterior septum, known as Little's area, is the point of convergence of the anterior ethmoidal artery, the septal branches of the sphenopalatine and superior labial arteries, and the greater palatine artery. A common site of bleeding.

Epistaxis is most commonly due to spontaneous rupture of a blood vessel in the nasal mucous membrane.

Your history should explore the possible causes (see Box 11.6).

Distinguish between anterior bleed (blood running out of the nose, usually one nostril) and posterior bleed (blood running into the throat or from both nostrils).

### Sneezing

Sneezing is a very frequent accompaniment to viral upper respiratory tract infection and allergic rhinitis. It is commonly associated with rhinorrhoea and itching of the nose and eyes.

Ask about exacerbating factors and explore the time-line carefully, looking for precipitants.

### Disorders of smell

Patients may complain of a ↓ sense of smell (hyposmia) or, more rarely, a total loss of smell (anosmia). Ask about the exact timing of the hyposmia and any other associated nasal symptoms.
- *Anosmia:* most commonly caused by nasal polyps but may be caused by head injury disrupting the olfactory fibres emerging through the cribiform plate. It may also complicate a viral upper respiratory tract infection (viral neuropathy).
- *Cacosmia:* the hallucination of an unpleasant smell and may be caused by infection interfering with the olfactory structures.

### Nasal deformity

Nasal deformity may occur as a result of a trauma causing pain ± swelling ± epistaxis ± displacement of nasal bones and septum.

Disruption of the bones and nasal septum may produce a 'saddle' deformity. Other causes of a 'saddle' nose include Wegener's granulomatosis, congenital syphilis, and long-term snorting of cocaine.

Acne rosacea can cause an enlarged, red, and bulbous rhinophyma. Widening of the nose is an early feature of acromegaly.

### Nasal and facial pain

Facial pain is not normally due to local nasal causes. More frequently, it is related to infection within the sinuses, trigeminal neuralgia, dental sepsis, migraine, or mid-facial tension pain.

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#### Box 11.5 Some causes of nasal discharge

*Watery or mucoid*
- Allergic rhinitis
- Infective (viral) rhinitis
- Vasomotor rhinitis
- A unilateral copious watery discharge may be due to CSF rhinorrhoea.

*Purulent*
- Infective rhinosinusitis
- Foreign body (especially if unilateral).

*Blood stained*
- Tumours (with unilateral symptoms)
- Bleeding diathesis
- Trauma.

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#### Box 11.6 Some causes of epistaxis

- Trauma from nose picking, nasal surgery, cocaine use, or infection
- Prolonged bleeding may be caused by hypertension, alcohol, anticoagulants, coagulation defects, Waldenström's macroglobulinaemia, Wegener's granulomatosis, and hereditary telangiectasia
- Neoplasia and angiomas of the postnasal space and nose may present with epistaxis.

# Symptoms of throat disorders

## Oral pain

- The commonest cause of pain in the oral cavity is dental caries and periodontal infection. Periodontal disease can cause pain on tooth-brushing and is associated with halitosis.
- Gum disease is a common cause of oral pain.
- In elderly patients, dentures may cause pain if improperly sized or if they produce an abnormal bite.
- Take a full pain history as in ➲ Chapter 2 and ask about other mouth/throat symptoms.

## Throat pain

- A sore throat is an extremely common symptom. You should clarify the full nature of the pain as discussed in ➲ Chapter 2. It is important to establish exactly where the pain is felt.
  - Throat pain often radiates to the ear because the pharynx and external auditory meatus are innervated by the vagus (X) nerve.
- Most acute sore throats are viral in origin and are associated with rhinorrhoea and a productive cough. Consider infectious mononucleosis in teenagers.
- Acute tonsillitis is associated with systemic symptoms such as malaise, fever, and anorexia.
- You should consider malignancy in all chronically sore throats in adults.
  - Ask about symptoms associated with cancer such as dysphagia, dysphonia, weight loss, and a history of smoking or excessive alcohol.

## Lumps in the mouth

*Lips*

- The lips are a common site for localized malignancy, e.g. BCC, SCC.

*Tongue*

- Lumps here are nearly always neoplastic

*Oral cavity*

- Blockage of a minor salivary gland might give rise to a cystic lesion called a ranula and is usually sited in the floor of the mouth.
  - Most malignant lesions on the floor of the mouth present late: pain, dysphagia, and odynophagia (pain on swallowing) are common symptoms. The buccal lining is also another very common site for cancer.

## Globus pharyngeus

This is the sensation of a lump in the throat (globus pharyngeus or globus syndrome). It is important to ask about symptoms of gastro-oesophageal reflux or postnasal drip.

It is occasionally associated with a malignancy. You should ask about dysphagia, odynophagia, hoarseness, and weight loss.

### Lumps in the neck

Neck lumps are usually secondary to infection but a minority are due to malignant disease. The most common cause of neck swelling is lymph node enlargement. A comprehensive history and examination of the head and neck is important.

▶ In the adult, it is worth remembering that metastatic neck disease may represent spread from structures below the clavicle including lung, breast, stomach, pancreas, kidney, prostate, and uterus. If malignancy is suspected, the history and examination should include a search for symptoms and signs in other systems.

As well as the full standard history, ask especially about:
- The duration of the swelling.
- Progression in size.
- Associated pain or other symptoms in the upper aerodigestive tract:
  - Odynophagia
  - Dysphagia
  - Dysphonia.
- Systemic symptoms (weight loss, night sweats, malaise).
- Smoking and alcohol habits.

### Dysphonia

This is an alteration in the quality of the voice. There are several causes which your history should be aimed at identifying including:
- *Inflammatory:* acute laryngitis, chronic laryngitis (chronic vocal abuse, alcohol, smoke inhalation).
- *Neurological:*
  - Central: pseudobulbar palsy, cerebral palsy, multiple sclerosis, stroke, Guillain–Barré syndrome, head injury
  - Peripheral: lesions affecting X and recurrent laryngeal nerves (e.g. lung cancer, post-thyroidectomy, cardiothoracic, and oesophageal surgery), myasthenia gravis, motor neuron disease.
- *Neoplastic:* laryngeal cancer for example.
- *Systemic:* rheumatoid arthritis, angiogenic oedema, hypothyroidism.
- *Psychogenic:* these are dysphonias in the absence of laryngeal disease and mainly occur due to an underlying anxiety or depression (i.e. musculoskeletal tension disorders, conversion voice disorders). Like all other non-organic disorders, you must rule out organic pathology.

### Halitosis

This is offensive-smelling breath. It is commonly caused by poor dental hygiene or diet. Tonsillar infection, gingivitis, pharyngeal pouch, and chronic sinusitis with purulent postnasal drip can also cause bad breath.

### Stridor

This is a noise from the upper airway (see also ⊋ Chapter 6) and is caused by narrowing of the trachea or larynx.

The main causes of stridor in adults are laryngeal cancer, laryngeal trauma, epiglottitis, and cancer of the trachea or main bronchus.

# Examining the ear

### Inspection and palpation

- Briefly inspect the external structures of the ear, paying particular attention to the pinna, noting its shape, size, and any deformity.
- Carefully inspect for any skin changes suggestive of cancer.
- Don't forget to look behind the ears for any scars or a hearing aid.
- Pull on the pinna and ask the patient if it is painful.
  - Infection of the external auditory meatus.
- Palpate the area in front of the tragus and ask if there is any pain.
  - Temporo-mandibular joint disease.
- Look for any discharge (Fig. 11.2).

### Otoscopy

The otoscope (or auroscope) allows you to examine the external auditory canal, the eardrum, and a few middle ear structures.

*The otoscope*

The otoscope consists of a light source, a removable funnel-shaped speculum, and a viewing window which often slightly magnifies the image (Fig. 11.3).

On many otoscopes, the viewing window can be slid aside to allow insertion of instruments (e.g. scrapers and swabs) down the auditory canal.

*Technique*

The following is the method for examining the patient's right ear. Examination of the left ear should be a mirror-image of this.

- Introduce yourself and clean your hands.
- Explain the procedure to the patient and obtain verbal consent.
- Turn the light source on.
- Place a clean speculum on the end of the scope.
- Gently pull the pinna upwards and backwards with your left hand.
  - This straightens out the cartilaginous part of the canal allowing easier passage of the scope.
- Holding the otoscope in your right hand, place the tip of the speculum in the opening of the external canal. Do this under direct vision before looking through the viewing window.
- Slowly advance the otoscope whilst looking though it.
  - It is often helpful to stabilize the otoscope by extending the little finger of the right hand and placing it on the patient's head.
- Inspect the skin of the auditory canal for signs of infection, wax, and foreign bodies.
  - If wax is causing obstruction, it may be necessary to perform ear syringing before continuing.
- Examine the tympanic membrane (Fig. 11.4).
  - A healthy eardrum should appear greyish and translucent
  - Look for the light reflex. This is the reflection off the surface of the drum visible just below the malleus
  - Notice any white patches (tympanosclerosis) or perforation
  - A reddened, bulging drum is a sign of acute otitis media
  - A dull grey, yellow drum may indicate middle ear fluid.

## Testing auditory and vestibular function

See cranial nerve VIII in ➲ Chapter 8.
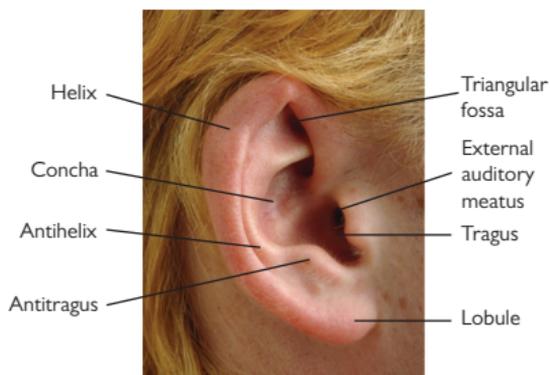


**Fig. 11.2** The surface anatomy of the normal ear.

Helix — Triangular fossa
Concha — External auditory meatus
Antihelix — Tragus
Antitragus — Lobule
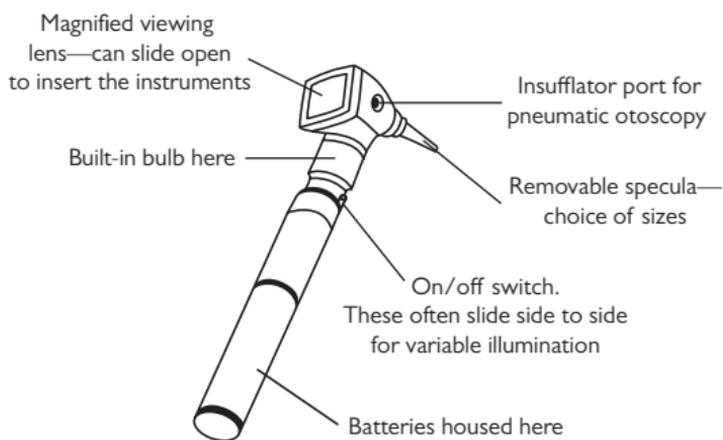


**Fig. 11.3** A standard otoscope.

Magnified viewing lens—can slide open to insert the instruments
Insufflator port for pneumatic otoscopy
Built-in bulb here
Removable specula—choice of sizes
On/off switch. These often slide side to side for variable illumination
Batteries housed here
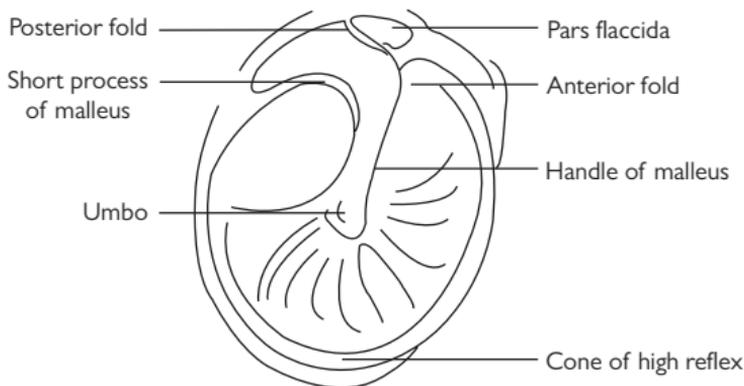


**Fig. 11.4** The appearance of the normal eardrum on otoscopy.

Posterior fold — Pars flaccida
Short process of malleus — Anterior fold
Handle of malleus
Umbo
Cone of high reflex

# Examining the nose

See Box 11.7 for rhinitis.

## External inspection
- Inspect the external surface and appearance of the nose noting any disease or deformity.
- Stand behind the patient and look down, over their head to detect any deviation.

## Palpation
- Gently palpate the nasal bones and ask the patient to alert you to any pain.
- If a visible deformity is present, palpate to determine if it is bony (hard and immobile) or cartilaginous (firm but compressible).
- Feel for facial swelling and tenderness.
  - Tenderness suggests underlying inflammation.

*Nostril patency*

Assess whether air moves through both nostrils effectively.
- Push on one nostril until it is occluded.
- Ask the patient to inhale through their nose.
- Then repeat on the opposite side.
  - Air should move equally well through each nostril.

## Internal examination
The postnasal space (nasopharynx) can be examined using fine-bore endoscopy. This is done by trained professionals; the student or non-specialist should examine the anterior portion of the nose only.
- Ask the patient to tilt their head back.
- Push up slightly on the tip of the nose with the thumb.
  - You should now be able to see just inside the anterior vestibule.
- In adults, you can use a nasal speculum to widen the nares allowing easier inspection.
- Pinch the speculum closed, place the prongs just inside the nostril, and release your grip gently allowing the prongs to spread apart.
- Look at:
  - The colour of the mucosa
  - The presence and colour of any discharge
  - The septum (which should be in the midline)
  - Any obvious bleeding points, clots, crusting, or perforation
  - The middle and inferior turbinates along the lateral wall for evidence of polypoid growth, foreign bodies, and other soft tissue swelling.

## Testing olfaction
This is described under cranial nerve I in ⊃ Chapter 8.

**Box 11.7 Rhinitis**

*Allergic rhinitis*
- Inhaled allergens cause an antigen-antibody type I hypersensitivity reaction
- Common allergens:
  - Pollen (including from grass) and flowering trees: seasonal allergic rhinitis (hayfever)
  - Animal dander*, dust mites**, and feathers: perennial allergic rhinitis
  - Digested allergens such as wheat, eggs, milk, and nuts are also rarely involved.
- The main symptoms include:
  - Bouts of sneezing
  - Profuse rhinorrhoea due to activity of glandular elements
  - Postnasal drip
  - Nasal itching
  - Nasal obstruction due to nasal vasodilatation and oedema.

*Non-allergic (vasomotor) rhinitis*
- This has all the clinical features of allergic rhinitis but the nose is not responding to an antigen-antibody type I reaction
- The reactions tend to be to inhaled chemicals such as deodorants, perfumes, or smoke, although alcohol and sunlight can provoke symptoms
- Allergies can coexist and some people seem to have an instability of the parasympathetic system in the nose with excessive secretion of watery mucus and congestion (vasomotor rhinitis).

\* Cat allergy is actually an allergy to one of the proteins in feline saliva—their fur is covered in it through licking.
\*\* Actually an allergy to dust mite faeces.

# Examining the nasal sinuses

The reader should revisit the anatomy of the sinuses and Fig. 11.1. The frontal and maxillary sinuses are the only two that can be examined, albeit indirectly.

## Palpation
- Palpate and percuss the skin overlying the frontal and maxillary sinuses.
- Tap on the upper teeth (which sit in the floor of the maxillary sinus).
  - In both of the above, pain suggests inflammation (sinusitis).

# Examining the mouth and throat

Ensure that the room is well lit. You should have an adjustable light-source. An otoscope or pen-torch should be adequate for non-specialists. (See Fig. 11.5.)

## Inspection

- *Face:* look at the patient's face for obvious skin disease, scars, lumps, signs of trauma, deformity, or facial asymmetry (including parotid enlargement).
- *Lips, teeth, gums:* inspect the lips at rest first.
  - Ask the patient to open their mouth and take a look at the buccal mucosa, teeth, and gums (see Box 11.8)
  - Note signs of dental decay or gingivitis
  - Ask the patient to evert the lips and look for any inflammation, discoloration, ulceration, nodules, or telangiectasia.
- *Tongue and floor of the mouth:* inspect the tongue inside and outside the mouth. Look for any obvious growths or abnormalities.
  - Included in this should be an assessment of cranial nerve XII
  - Ask the patient to touch the roof of the mouth with their tongue
  - This allows you to look at the underside of the tongue and floor of the mouth.
- *Oropharynx:* to look at the posterior oropharynx, ask the patient to say 'Aaah' (elevates the soft palate).
  - Using tongue depressor may provide a better view
  - Uvula: should hang down from the roof of the mouth, in the midline. With an 'Aaah' the uvula rises up. Deviation to one side may be caused by cranial nerve IX palsy, tumour, or infection.
- *Soft palate:* look for any cleft, structural abnormality, or asymmetry of movement and note any telangiectasia.
- *Tonsils:* inspect the tonsils noting their size, colour, and any discharge.
  - The tonsils lie in an alcove between the posterior and anterior pillars (arches) on either side of the mouth.

## Palpation

This is reserved for any abnormal or painful areas which you have detected on initial inspection.

- Put on a pair of gloves and palpate the area of interest with *both* hands (one hand outside on the patient's cheek or jaw and the other inside the mouth).

## The rest of the neck

Palpate the cervical and supraclavicular lymph nodes (➲ Chapter 3), thyroid (➲ Chapter 3), and look for any additional masses.

Hard palate

Soft palate

Anterior arch (pilla)

Uvula

Posterior arch (pilla)

Oropharynx

Tonsillar fossa
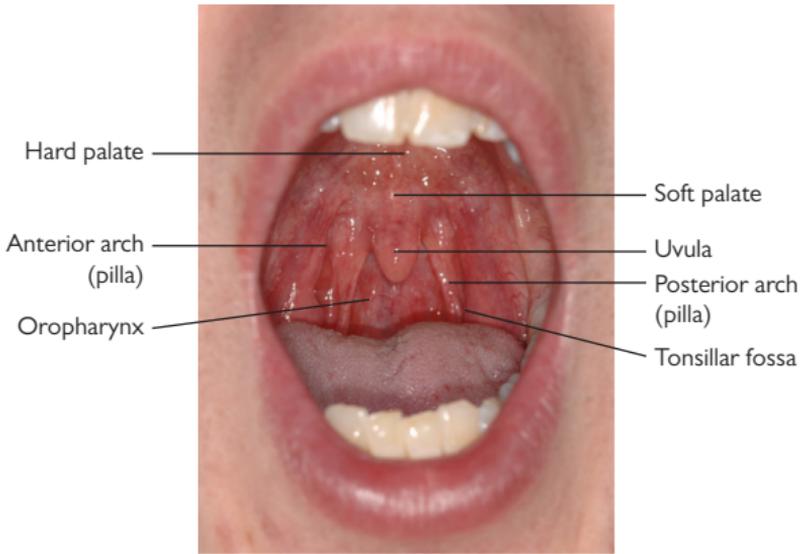
**Fig. 11.5** The normal appearance of the oral cavity.

## Findings

- *Mucosal inflammation:* bacterial, fungal (candidiasis), and viral (e.g. herpes simplex) infections, or after radiotherapy treatment.
- *Oral candidiasis:* think radiotherapy, use of inhaled steroids, and immunodeficiency states (e.g. leukaemia, lymphoma, HIV).
- *Gingivitis:* inflammation of the gums may occur in minor trauma (teeth brushing), vitamin and mineral deficiency, or lichen planus.
- *Tonsillitis:* mucopus on the pharyngeal wall implies bacterial infection. Think of infectious mononucleosis in teenagers, particularly if the tonsils are covered with a white pseudomembranous exudate.
  - Acute tonsillitis is often associated with systemic features of malaise, fever, anorexia, cervical lymphadenopathy, and candidiasis.

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**Box 11.8 Gum changes in systemic conditions**

- Chronic lead poisoning: punctate blue lesions
- Phenytoin treatment: firm and hypertrophic gums
- Scurvy: gums are soft and haemorrhagic
- Cyanotic congenital heart disease: gums are spongy and haemorrhagic.

# Important presentations

### Otitis externa
- Inflammation of the outer ear.
- Commonly caused by bacterial infection of the ear canal (e.g. *Streptococci*, *Staphylococci*, *Pseudomonas*) and fungi.
  • Heat, humidity, swimming, and any irritants causing pruritus can all predispose a patient to otitis externa.
- Often occurs in patients with eczema, seborrhoeic dermatitis, or psoriasis due to scratching.
- Symptoms can vary from irritation to severe pain ± discharge.
- Pressure on the tragus or movement of the auricle may cause pain.
- *'Malignant otitis externa':* very aggressive form caused by a spreading osteomyelitis of the temporal bone (usually *Pseudomonas pyocaneus*).

### Furunculosis
- An infection of hair follicles in the auditory canal.
- Presents with severe throbbing pain exacerbated by jaw movement with pyrexia and often precedes rupture of an abscess.

### Otitis media and glue ear
- Inflammation of the middle ear, usually following an URTI.
- In the early stages, the eardrum becomes retracted as the Eustachian tube is blocked, resulting in an inflammatory middle ear exudate.
- If there is infection, pus builds up causing the middle ear pressure to rise and this is seen on otoscopy as bulging of the eardrum.
- The eardrum may eventually rupture if untreated.

*Complications*
- Include: inflammation in the mastoid air cells (mastoiditis), labyrinthitis, facial nerve palsy, extradural abscess, meningitis, lateral sinus thrombosis, cerebellar and temporal lobe abscess.

*Chronic suppurative otitis media*
- Associated with a central persistent perforation of the pars tensa. The resulting otorrhoea is usually mucoid and profuse in active infection.

*Glue ear*
- (Otitis media with effusion) is the commonest cause of acquired conductive hearing loss in children (peaks between 3–6 years).
- Higher incidence in patients with cleft palate and Down's syndrome.
- The aetiology is usually Eustachian tube dysfunction with thinning of the drum.

### Cholesteatoma
- Destructive disease consisting of overgrowth of stratified squamous epithelial tissue in the middle ear and mastoid causing erosion of local structures and the introduction of infection.
- When infected, there may be a foul-smelling discharge.
- Bone destruction and marked hearing loss can occur.
- May be complicated by meningitis, cerebral abscesses, and VII palsy.

### Ménière's disease
- Also known as endolymphatic hydrops.
- Distension of the membranous labyrinthine spaces.
  - The exact cause is not known.
- Symptoms: attacks of vertigo with prostration, nausea, vomiting, a fluctuating sensorineural hearing loss at the low frequencies, tinnitus, and aural fullness or pressure in the ear.
- Attacks tend to occur in clusters with quiescent periods between.
  - Each attack only lasts a few hours and the patient usually has normal balance between. Over years, the hearing gradually deteriorates in the affected ear.

### Vestibular neuronitis
- Typically associated with sudden vertigo, vomiting, and prostration.
- The symptoms are exacerbated by head movement.
- Often follows a viral illness in the young or a vascular lesion in the elderly.
- No deafness or tinnitus.
- Vertigo lasts for several days, but complete recovery of balance can take months, or may never be achieved.

### Otosclerosis
- A localized disease of bone which affects the capsule of the inner ear.
  - Vascular, spongy bone replaces normal bone around the oval window and may fix the footplate of the stapes
  - Both ears are affected in >50% of patients.
- Otoscopic examination is usually normal.
- There may be progressive conductive deafness manifesting after the second decade, possibly with tinnitus and, rarely, vertigo.
- Pregnancy and lactation aggravate the condition.
- There is often a strong FHx.

### Benign positional vertigo
- Attacks of sudden-onset rotational vertigo provoked by lying flat or turning over in bed.
- Caused by crystalline debris in the posterior semicircular canal.
- Can follow an upper respiratory tract infection or head injury, but often there may be no preceding illness.
- Hallpike's manoeuvre is diagnostic (see ➲ Chapter 8).
- If diagnosed, the person should have an Epley manoeuvre which is often curative.
  - This repositions the debris in the posterior semicircular canal into the utricle.

### Labyrinthitis
- Localized infection of the labyrinth apparatus.
  - Difficult to distinguish clinically from vestibular neuronitis, unless there is hearing loss due to cochlear involvement.

### Acoustic neuromas
- Benign tumours of the vestibular element of cranial nerve VIII.
- Usually present in middle age and occur more frequently in females.
- Bilateral neuromas occur in 5% of patients.
- The early symptoms are unilateral or markedly asymmetric, progressive sensorineural hearing loss and tinnitus.
- Vertigo is rare but patients with large tumours may have ataxia.

### Presbyacusis (senile deafness)
- Progressive loss of hair cells in the cochlea with age, resulting in a loss of acuity for high-frequency sounds.
- It usually becomes clinically noticeable from the age of 60–65 years.
  - The degree of loss and age of onset are variable.
- Hearing is most affected in the presence of background noise.

### Glomus jugulare tumour
- A highly vascular tumour arising from 'glomus jugulare' tissue lying in the bulb of the internal jugular vein or the mucosa of the middle ear.
- Usually presents with a hearing loss or pulsatile tinnitus.
- Examination may show a deep red mass behind the eardrum.
- Occasionally associated with other tumours such as phaeochromocytomas, or carotid body tumours.

### Nasal polyps
- Nasal polyps are pale, greyish, pedunculated, oedematous mucosal tissue which project into the nasal cavity.
  - Most frequently arise from the ethmoid region and prolapse into the nose via the middle meatus
  - Nearly always bilateral
- In the majority of cases, they are associated with non-allergic rhinitis and late-onset asthma.
- Other causes to consider include:
  - Chronic paranasal infection
  - Neoplasia (usually unilateral ± bleeding)
  - Cystic fibrosis
  - Bronchiectasis.
- The main symptoms are watery anterior rhinorrhoea, purulent postnasal drip, progressive nasal obstruction, anosmia, change in voice quality, and taste disturbance.

### Septal perforation
- May be idiopathic or be caused by trauma (especially post-op nasal surgery), infection (e.g. tuberculosis, syphilis), neoplasia (SCC, BCC, malignant granuloma), and inhaling cocaine and toxic gases.
- The main clinical complaints include crusting, recurrent epistaxis, and a whistling respiration.

### Tonsillitis

- Acute tonsillitis is uncommon in adults in comparison to its frequency in children.
- The diagnosis is made from the appearance of the tonsils which are enlarged with surface exudates.
- The patient is usually systemically unwell with pyrexia, cervical lymphadenopathy, dysphagia, halitosis, and abdominal pain in children.
- Complications include peritonsillar abscess (quinsy) and retropharyngeal abscess.

### Laryngitis

- Frequently associated with an URTI and is self-limiting.
- May be associated with secondary infection with *Staph.* and *Strep.*
- Patient typically complains of hoarseness, malaise, and fever.
  - There may also be odynophagia, dysphagia, and throat pain.

### Epiglottitis

- 🚸 This is a medical emergency.
- Caused by group B *Haemophilus influenzae*.
- Characterized by gross swelling of the epiglottis and is primarily seen in 3–7-year-olds, although adults may also be affected.
- Clinical features include pyrexia, stridor, sore throat, and dysphagia.

### Croup (laryngotracheobronchitis)

- The majority of cases are viral (parainfluenza or respiratory syncytial virus). It mainly occurs between the ages of 6 months and 3 years.

### Branchial cyst

- This is an embryological remnant of the branchial complex during development of the neck.
- Located in the anterior triangle just in front of the sternomastoid.
- Presentation is typically at the age of 15–25 years.
- For more, see the *Oxford Handbooks Clinical Tutor: Surgery.*

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**See also:**

More information regarding the presentation and clinical signs of ENT diseases to aid preparation for OSCE-type examinations and ward rounds can be found in the *Oxford Handbooks Clinical Tutor Study Cards*.

*'Surgery' Study Card set:*

- Thyroglossal cyst
- Branchial cyst
- Pharyngeal pouch
- Tumours of the parotid
- Diffuse parotid enlargement
- Submandibular calculi.

# The male reproductive system

Notes

This chapter describes the history and examination related to male sexual function. Urinary and prostatic history and examination can be found in 'the abdomen' ➲ Chapter 7.

# Introduction

## 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 two 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 three 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 to or further away from the body.

### The testes

These are paired, ovoid organs measuring 4 x 3 x 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 76cm in length lying on the posterior surface of the testis. It is a specialized part of the collecting apparatus where spermatozoa are matured before travelling 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 which 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 four 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 is 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 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.

# 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.

### Rashes, warts, ulcers

Treat a genital lesion as you would any other rash (see ⊃ Chapter 4).
Ask also about:

- Similar lesions elsewhere (e.g. mouth, anus).
- Foreign travel.

You should determine the risk of recent exposure to STDs as previously.

### Testicular pain

This is often felt as a deep burning and accompanied by nausea. Treat as pain in any other location (see ⊃ Chapter 2). 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 erectile dysfunction, 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. β-blockers), alcohol, oestrogens, methadone, and 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?

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).

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### Box 12.1 The rest of the history

A full history needs to be taken as described in ⟳ Chapter 2. The following may have particular relevance here:

*PMH*

Ask especially about:

- Sexually transmitted diseases
- Orchitis
- Inguinal, scrotal, and testicular injury/surgery
- Urethral/penile injury.

*Smoking and alcohol*

Detailed histories should be taken as described elsewhere 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 will not 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 Peyronie'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 non-retractile and attempts may cause considerable pain.

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### Box 12.2 Hypospadias

Hypospadias is the abnormal, ventral, positioning of the urethral meatus. It is more common than many realize, 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 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 its contents

See

### The perineum and rectum

Don't overlook the perineum, anal canal, and rectum. In particular, a digital rectal examination should be performed as described in ➲ Chapter 7 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 (Fig. 12.1).
- 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 (see ➲ Chapter 4).



Horizontal group

Vertical group
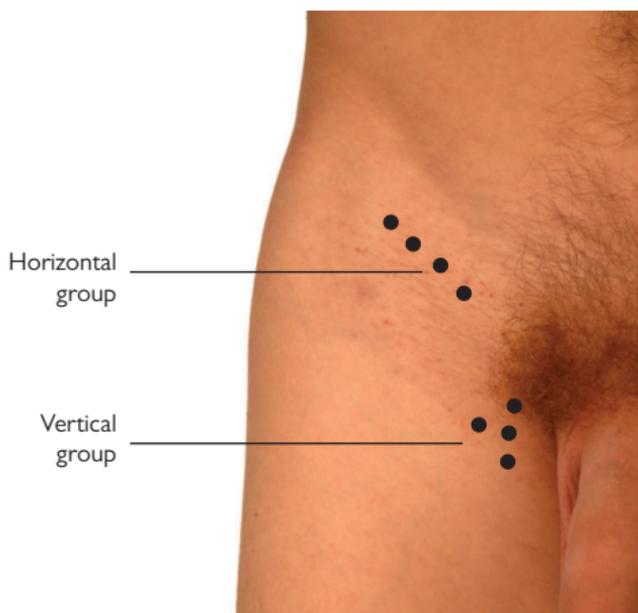
**Fig. 12.1** Diagrammatic representation of the inguinal lymph nodes.

## 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 on 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. 12.2).
- Check that the scrotum contains two 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 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 posterolaterally.
- 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 (see also Boxes 12.3 and 12.4):
- 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 in ➲ Chapter 4.
- Transillumination is often important here. Darken the room and shine a small torch through the posterior part of the swelling (see Fig. 12.3).
  - A solid mass remains dark while a cystic mass or fluid will transilluminate.

(a)



(b)



**Fig. 12.2** (a) Examine the scrotum with the patient standing and use both hands. It is sometimes preferable to ask the patient to hold their penis aside (b).



**Fig. 12.3** Attempt to transilluminate any swelling by shining a small torch through it. NB Unlike the figure above, the room should be darkened.

# Important presentations

### Testicular torsion

This presents in a very similar way to orchitis 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 due to an internal rotation, 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.

Rough salvage rates are:
- <6 hours: 80–100%.
- 6–12 hours: 76%.
- 12–24 hours: 20%.
- >24 hours: 0%.

*Inspection*
- Patient usually exhibits signs of distress or pain.
- Hemi-scrotum may be swollen and erythematous compared to opposite side.

*Palpation*
- Testis will be exquisitely tender and often slightly swollen.
  - May be higher than normal and horizontal in orientation.
- Epididymis may be palpated anteriorly and spermatic cord may be thickened.
- Cremasteric reflex may be absent.

### 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 tumour

This should be at the top of your list of differential diagnoses in the case of an intra-scrotal mass. 90% are germ cell tumours (of which 48% are seminomas and 42% non-seminomatous germ cell tumours e.g. teratomas). Teratomas commonly occur between the ages of 20–30 years while seminomas are more common between 30–40 years.

Look for constitutional symptoms and signs 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.

Symptoms include a gradually enlarging, painless testicular lump. A dull ache or heaviness is not unusual. 10% of patients are asymptomatic. 10% present with symptoms related to metastatic disease. 10% give a history of trauma prior to discovery of a lump. 5% present with acute scrotal pain secondary to intratumoural haemorrhage.

*Inspection*
- Swollen or asymmetrical hemi-scrotum compared to opposite side.
- Patient may appear cachexic.
- There may be gynaecomastia (due to trophoblastic elements secreting human chorionic gonadotropin e.g. Leydig and Sertoli cell tumours).

*Palpation*
- A hard, non-tender, irregular, non-transilluminable mass in the testis or even replacing testis.
  - Should be able to palpate above mass
  - Assess the epididymis, spermatic cord, and scrotal wall.
- 🛈 The lump may be impalpable if there is an associated hydrocoele.

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### Box 12.3 Differential diagnosis of scrotal lumps
- *Indirect inguinal hernia:* has cough impulse, reduces with direct pressure or lying down and examiner cannot 'get above' the swelling
- *Epididymal cyst:* swelling confined to epididymis, should be able to palpate normal testis
- *Testicular tumour:* usually hard, craggy mass in body of testis (but can invade surrounding structures)
- *Varicocoele:* feels like a 'bag of worms', often disappears on lying down
- *Sebaceous cyst:* smooth and fixed to scrotal skin. Able to palpate scrotal contents separately
- *Epididymo-orchitis:* tender, painful swelling of testis and epididymis. May have erythema of scrotal skin
- *Testicular torsion:* sudden onset of severe pain with exquisitely tender testis, thickened spermatic cord, and horizontal testicular lie
- *Gumma of testis:* rare and secondary to syphilis. Feels round, hard, insensitive ('like a billiard ball')
- *Carcinoma of scrotal skin:* irregular and fixed to scrotal skin, may be associated with inguinal lymphadenopathy.

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### Box 12.4 Differential diagnosis of an epididymal lump
- *Epididymo-orchitis:* usually tender, painful swelling of testis and epididymis. May have erythema of scrotal skin
- *Tuberculous epididymo-orchitis:* usually painless and non-tender, epididymis is hard with an irregular surface. The spermatic cord is thickened and the vas deferens feels hard and irregular (like a string of beads)
- *Sperm granuloma:* seen in roughly 1 in 500 men who have undergone vasectomy. Congested sperm and fluid extravasates from the end of the vas deferens, is recognized by the immune system as 'foreign' and initiates an inflammatory reaction giving a palpable 'lump'. These are usually painless and can be felt as separate to the testis.

## Hydrocoele

This is fluid entrapment in the tunica vaginalis causing usually painless swelling of the scrotum—the size of which can be considerable. Hydrocoele 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.

*Inspection*
- Swollen hemi-scrotum compared to opposite side.
- Usually no abnormality of scrotal skin.

*Palpation*
- Hemi-scrotal content will usually be painless and swollen with a tense, smooth surface.
- Unable to palpate testis separately from swelling.
- Superior margin can be palpated (i.e. you can 'get above it').
- Spermatic cord is palpable separately.
- Cough reflex is present.
- Swelling can be transilluminated.

## Epididymal cysts

These are harmless, painless swellings arising behind, and separate from, the testis itself. If aspirated, the fluid may appear opalescent, like lime water, because a few sperm are present. The aetiology is unknown.

Occasionally they occur as a complication of vasectomy, in which case they are full of sperm and are termed 'spermatocoeles'.

*Inspection*
- The hemi-scrotum may be swollen compared to opposite side.

*Palpation*
- No abnormality of scrotal skin.
- Examination of the testis is normal.
- A firm, loculated, painless mass can be felt within the epididymis, the rest of the epididymis may not be palpable.
  - Superior margin can be palpated (i.e. you can 'get above it').
- The mass can be transilluminated.
  - Normally only demonstrable in large cysts
  - No transillumination if cyst contains sperm.

## Varicocoele

A dilatation of the veins of the pampiniform plexus of the spermatic cord. Found in 15% of men in the general population and 40% of men with infertility. Either bilateral or unilateral, the left side is affected in 90%.

Incompetent valves in the internal gonadal veins lead to retrograde blood flow, vessel dilatation, and tortuosity of pampiniform plexus.

*Inspection*
Hemi-scrotum may be swollen compared to opposite side when standing.

*Palpation*
- Usually no abnormality of scrotal skin.

- If moderate or large in size, a mass of dilated and tortuous veins will be felt above testis around the spermatic cord (described as feeling like a 'bag of worms').
  - If no mass is palpated, ask patient to perform Valsalva manoeuvre (strain down). Small varicocoeles will then be palpable
  - Examine the patient lying supine; the mass will decompress and disappear.
- The testis and epididymis are palpable separately.
- The testis may be atrophied.

*Varicocoeles and renal malignancy*

- 5% of renal cell carcinomas present with acute left varicocoele due to obstruction of the testicular vein by tumour in the left renal vein.
- In the presence of a left-sided varicocoele, the abdomen should be examined for a renal mass and the patient should be asked about pain and haematuria.
- Abdominal/renal ultrasound should be considered.

## 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 include congenital, infection, trauma, and 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.

## 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 include *Candida albicans* (especially in patients with diabetes), herpes infection, carcinoma, drug eruptions, and 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.

# 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. (See Box 12.5.)

Retain a 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. transurethral resection of the prostate (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 antidepressants, 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.

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**Box 12.5  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.

# The female reproductive system

**The obstetric assessment**
The obstetric history and examination is covered in
⊃ Chapter 14.

# Introduction

### The pelvis

The bony pelvis is composed of the two 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 three 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 forming 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 open into the peritoneal cavity near the ovaries. The Fallopian tube is divided into four 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 x 2cm which are responsible for the production of the female germ cells, the ova, and the 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 joins in the midline forming the 'body' capped by the sensitive 'glans'.
- *Bulbs of vestibule:* two masses of elongated erectile tissue, ~3cm long, lying along the sides of the vaginal orifice.
- *Greater and lesser vestibular glands.*

## 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 three 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 divided into three phases.

*Phases of the menstrual cycle*

The first day of 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. Gonadotrophin-releasing 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 luteinizing 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-synthesizing cells. High progesterone levels exert negative feedback on GnRH which, in turn, reduces 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 increased 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 decrease. As oestrogen and progesterone levels fall, the endometrium undergoes involution.

If the corpus luteum is not rescued by human chorionic gonadotropin (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, leukocytes and red blood cells are all present in the menstrual flow.

Shedding usually lasts ~4 days.

# The gynaecological history

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. We detail here those parts that may differ from the basic format.

## History of presenting complaint

More detailed questioning will depend on the nature of the presenting complaint. Ascertain:

- The exact nature of the symptom.
- The onset.
  - When and how it began (e.g. suddenly, gradually—over how long?)
  - If long-standing, 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.
- 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

- 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 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.

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

Pay particular attention to any history of chronic lung or heart disease and make note of all previous surgical procedures.

### Drug history

Ask about all medication/drugs taken (prescribed, over-the-counter, and illicit drugs). Record dose and 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.

### 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

## 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. See Box 13.1.

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 two 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. See Box 13.2.

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, a detailed menstrual history, 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. See Box 13.3 for causes.

As for all these symptoms, a full standard battery of questions should be asked, a full menstrual history, past medical and gynaecological histories, and sexual history.

Ask also about the association of the bleeding with hormonal therapy, contraceptive use, and previous cervical smears.

## Post-coital 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 always.

See Box 13.4 for causes.

**Box 13.1 Some causes of menorrhagia**
- Hypothyroidism
- Intra-uterine contraceptive device (IUCD)
- Fibroids
- Endometriosis
- Polyps—cervix, uterus
- Uterine cancer
- Infection (STDs)
- Previous sterilization
- Warfarin therapy
- Aspirin
- Non-steroidal anti-inflammatory drugs (NSAIDs)
- Clotting disorders (e.g. von-Willebrand's disease).

**Box 13.2 Some causes of dysmenorrhoea**
- Pelvic inflammatory disease
- Endometriosis
- Uterine adenomyosis
- Fibroids
- Endometrial polyps
- Premenstrual syndrome
- Cessation of oral contraceptive.

**Box 13.3 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 13.4 Some causes of post-coital bleeding**

Similar to intermenstrual bleeding, as well as:
- Vaginal infection:
  - Chlamydia
  - Gonorrhoea
  - Trichomaniasis
  - Yeast.
- Cervicitis.

## Amenorrhoea

This is the absence of periods and may be 'primary' or 'secondary'. See Box 13.5 for causes.

- *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. Ask especially about:

- Childhood growth and development.
- If secondary amenorrhoea:
  - Age of menarche
  - Cycle days
  - Day and date of LMP
  - Presence or absence of breast soreness
  - Mood change immediately before menses.
- Chronic illnesses.
- Previous surgery (including cervical surgery which 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.
- Social history 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. increased 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. See Box 13.6 for some causes.

As well as all the points outlined under 'amenorrhoea', ask about:

- Local symptoms of oestrogen deficiency such as vaginal dryness, soreness, and superficial dyspareunia.
- 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 blood-stained offensive discharge.

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**Box 13.5 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:* Müllerian 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.

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**Box 13.6 Some causes of postmenopausal bleeding**

- Cervical carcinoma
- Uterine sarcoma
- Vaginal carcinoma
- Endometrial hyperplasia/carcinoma/polyps
- Cervical polyps
- Trauma
- Hormone replacement therapy
- Bleeding disorder
- Vaginal atrophy.

# 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.

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**Box 13.7 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.

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### History taking

When taking a history of pelvic pain or dyspareunia, you should obtain a detailed history as for any type of pain (⮕ Chapter 2). Carefully differentiate from gastrointestinal pain (Box 13.7). Some causes of dyspareunia are shown in Box 13.8.

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.
- A detailed sexual history (⮕ Chapter 2) should also include contraceptive use and the degree of impact the symptoms have on the patient's normal life, and psychological health.

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**Box 13.8 Some causes of dyspareunia**
- Scars from episiotomy
- Vaginal atrophy
- Vulvitis, vulvar vestibulitis
- Pelvic inflammatory disease
- Ovarian cysts
- Endometriosis
- Varicose veins in pelvis
- Ectopic pregnancy
- Infections (STIs)
- Bladder or urinary tract disorder
- Cancer in the reproductive organs or pelvic region.

# Vaginal discharge

Vaginal discharge is a common complaint during the child-bearing years. As well as the standard questions, 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 (➲ Chapter 2). A full gynaecological history should include history of cervical smear testing, use of ring pessaries, and recent history of surgery (increased 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 increases 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.

# Vulval symptoms

The main symptom to be aware of is itching or irritation of the vulva (pruritus vulvae). It can be debilitating and socially embarrassing. Embarrassment often delays the woman seeking advice. See Box 13.10 for some other vulval conditions.

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.
- 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 *detrusor 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. 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 (increased risk with increased parity).
- Genital prolapse.
- Previous pelvic floor surgery.

*Detrusor over-activity*

Urge incontinence, urgency, frequency, and nocturia. Ask about:
- History of nocturnal enuresis.
- Previous neurological problems.
- Previous incontinence surgery.
- Incontinence during sexual intercourse.
- DHx (see note under 'the elderly patient').

*Overflow incontinence*

Voiding disorders can result in chronic retention leading to overflow incontinence and increased 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 causes are outlined in 13.9. 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).

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**Box 13.9  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.

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**Box 13.10  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

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, preferably female.

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 in Box 13.11. 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.

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**Box 13.11 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).

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### 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.

### Abdominal examination

A full abdominal examination should be performed (see ⊃ Chapter 7). 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.
  - Bartholin'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, to visualize the cervix, or take a cervical smear or swabs.

There are different types of vaginal specula (see Fig. 13.1) but the commonest is the Cusco's or bivalve speculum. See Box 13.12.

### 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. 13.2) 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.

(a)

(b)

Upper blade

Lower blade

Thumbscrew
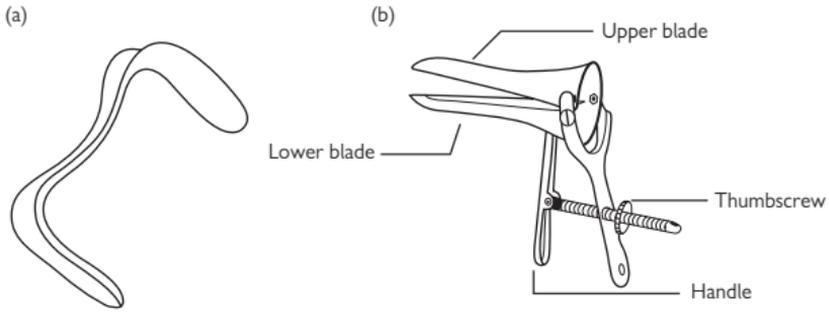
Handle

**Fig. 13.1** (a) Sim's speculum—used mainly in the examination of women with vaginal prolapse. (b) Cusco's speculum.
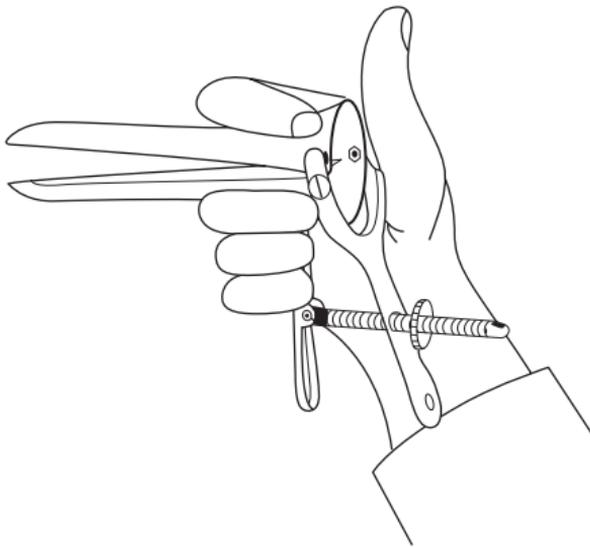
**Fig. 13.2** Hold the speculum in the right hand such that the handles lie in the palm and the blades project between the index and middle fingers.

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**Box 13.12 A word about specula**

Many departments and clinical areas now use plastic/disposable specula. These do not have a thumbscrew 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 previously.
- 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. 13.3).

## 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 two hands.
- Note the 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.
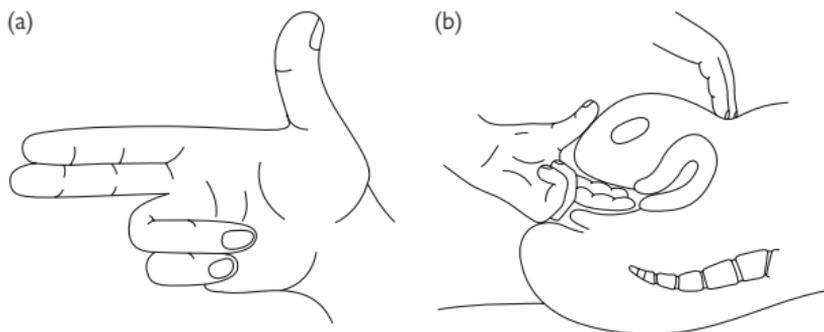


**Fig. 13.3** (a) Correct position of the fingers of the right hand for *per vaginum* examination. (b) Bimanual examination of the uterus.

# Taking a cervical smear

We describe the technique for obtaining a sample for 'liquid-based cytology' (LBC), now used by the majority of units in the UK.

## Equipment

- Cusco's specula of different sizes.
- Disposable gloves.
- Request form.
- Sampling device: plastic broom (Cervex-Brush®).
- Liquid-based cytology vial: preservative for sample.
- Patient information leaflet.

## Procedure

- Introduce yourself, confirm the patient's identity, ensure patient understands the purpose of the procedure and has been given patient information leaflet.
- Explain the procedure and obtain informed consent.
- Ensure a chaperone is available during the examination.
- Write the patient's identification details on LBC vial.
- 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.
- 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 with the main body of the speculum in the palm 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.
- Ensure entire cervix is clearly visualized and note any obvious abnormalities or irregularity.
- Once in optimum position, tighten the thumbscrew.
- Insert the plastic broom so that the central bristles of the brush are in the endocervical canal, the outer bristles in contact with the ectocervix.
- Using pencil pressure, rotate the brush five times clockwise (Fig. 13.4).
  - 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®).
- 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.
- Allow the patient to re-dress in privacy.
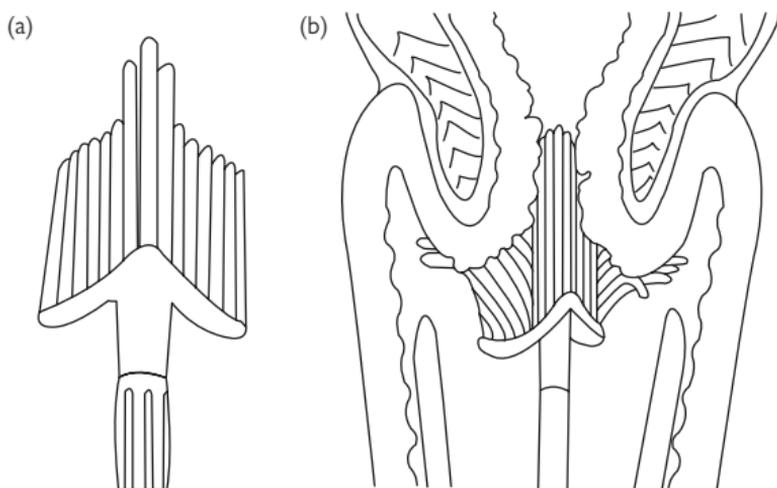


**Fig. 13.4** (a) The end of a typical cervix-brush. (b) Representation of how to use a Cervex-Brush®. Note that the longer, central bristles are within the cervical canal whilst the outer bristles are in contact with the ectocervix.

### Documentation
- Date, time, indication, informed consent obtained.
- Those present, including chaperone.
- Date of last menstrual period and use of hormonal treatments.
- Date of last smear and any abnormal results.
- Any abnormalities identified.
- Any immediate complications.
- Signature, printed name, and contact details.

### Procedure tips
- Cervical smears should not be performed during pregnancy.
  - The increase in cervical mucus (and resultant decrease in the number of cells obtained) usually renders the sample inadequate and the results unreliable.
- Neither abnormal vaginal bleeding or discharge or a visible or palpable cervical lesion is an indication for a cervical smear per se as it is a test for cervical atypia which is asymptomatic. However, a speculum examination should be performed to inspect the cervix and infection screening offered.
  - A cervical smear can be offered to women who have not had a normal test within the usual screening period.
- Ensure the patient knows when and how she will receive the results of the test and who to contact in case of problems.

# 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. 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 postmenopausal changes—such as vaginal atrophy (Box 13.13) 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.
- *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 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?

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**Box 13.13 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 increased vaginal pH, and thinned endometrium lead to both genital and urinary symptoms and signs. A decrease in vaginal lubrication presents with dryness, pruritus, and discharges, accompanied by an increase 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.

# The obstetric assessment

# 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.

## Demographic details
- Name, age, and date of birth.
- Gravidity and parity—see Boxes 14.2 and 14.3.

## 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 (see Box 14.1).

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**Box 14.1 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.

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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 three months prior to conception? If so, calculations based on her LMP are unreliable.

## Current pregnancy

Ask about the patient's general health and that of her fetus. If there is a presenting complaint, the details should be documented in full. 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).

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* Named after the German obstetrician, 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.

#### Box 14.2 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 two 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.

#### Box 14.3 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!

### Past obstetric history

Ask about all of the patient's 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 puer-perium 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

Note especially those conditions which may have an impact on the preg-nancy including:
- Diabetes.
- Endocrine disorders such as thyroid disorders or Addison's disease.
- Asthma.
- Epilepsy.
- Hypertension and 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' postnatal depression.

### Drug history

- Take a full drug history 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 400 micrograms of folic acid daily until 12 weeks' gestation to reduce the incidence of spina bifida.

### Family history

- 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

As well as the full standard social history, 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.

# 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 (Boxes 14.6 and 14.7).

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.

## After pregnancy

This is called 'post-partum haemorrhage' or PPH (Boxes 14.4 and 14.5).

- *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. Ask also about symptoms of infection—an important cause of secondary PPH.

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**Box 14.4  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.

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**Box 14.5  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.

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**Box 14.6 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.

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**Box 14.7 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, reduced or absent fetal movements, fetal distress, or absent fetal heart sounds.

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[1] Collins et al. (2013). *Oxford Handbook of Obstetrics and Gynaecology*. OUP, Oxford.

# Abdominal pain

A full pain history should be taken as in ⊃ Chapter 2 including site, radiation, character, severity, mode and rate of onset, duration, frequency, exacerbating factors, relieving factors, and associated symptoms.

## History taking

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, and cholecystectomy.

## Causes

⚠ 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.

# 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.
- Proteinuria 2+.

### Severe proteinuric pregnancy-induced hypertension

- Diastolic blood pressure 110 or greater after 20 weeks' gestation.
- 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.

# Minor symptoms of pregnancy

These so-called 'minor' symptoms of pregnancy are often experienced by a number of woman as normal 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
  - Hepatitis.

### Heartburn/gastro-oesophageal reflux
- Heartburn is a frequent complaint during pregnancy due partially to compression of the stomach by the gravid uterus.

### Constipation
- Often secondary to ↑ progesterone.
- Improves with gestation.

### 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 pulmonary embolus) need to be considered.

### 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.

### 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 on ➲ p. 475. 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 also establishes a 'physical rapport' before you examine more delicate or embarrassing areas (Box 14.8).

## General inspection

Always begin with a general examination of the patient (as in ➲ Chapter 3) including:

- Temperature.
- Hydration.
- Coloration.
- Nutritional status.
- Lymph nodes.
- 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.
- ▶ Blood pressure should be measured in the left lateral position at 45° to avoid compression of the inferior vena cava by the gravid uterus.
- 🔵 Anaemia is a common complication of pregnancy so examine the mucosal surfaces and conjunctivae carefully.

## Examining other systems

- Examine the cardiovascular and respiratory systems in turn (see ➲ Chapters 5 and 6).
  - 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.

**Box 14.8 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.

# Abdominal 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 increased 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 7) before proceeding to more specific manoeuvres in an obstetric examination.

## Uterine size

▶ The symphysial–fundal height (cm) = weeks of gestation.

The distance from the symphysis pubis to the upper edge of the uterus provides an estimation of gestational age and is objectively measured and expressed in centimetres as the *symphysial–fundal* height (Box 14.9 and Fig. 14.1).

Between 16–36 weeks, there is a margin of error of ±2cm, ±3cm at 36–40 weeks, and ±4cm at 40 weeks onwards.

*Technique*

- 🟡 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.

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**Box 14.9  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.

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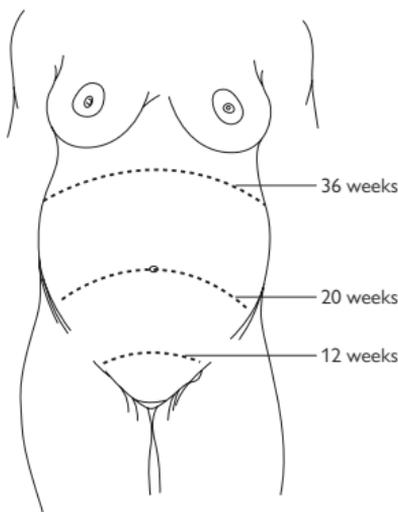


**Fig. 14.1** A guide to the surface landmarks for uterine size.

### 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 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:* cephalic 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'.

*Paulik's grip*

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.

**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 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 fewer 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 reduced or increased amniotic fluid volume.

- Increased volume will give a large-for-dates uterus that is smooth and rounded. The fetal parts may be almost impossible to palpate.
- Reduced 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 unless you suspect polyhydramnios in which case, you may wish to attempt to elicit a fluid thrill.

# 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.

- Place the bell of the instrument over the anterior fetal shoulder.
- 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.

# 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 in ⊃ Chapter 13. 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 practise 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.

# The breast

# Introduction

### Anatomy of the breast

The two mammary glands are highly developed apocrine sweat glands. They develop embryologically along two lines extending from the axillae to the groin—the milk lines (see Fig. 15.1). 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 four 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 (Fig. 15.2).

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 five groups (see Fig. 15.7).

### 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 is produced. Suckling stimulates secretion of prolactin as well as releasing oxytocin which stimulates myoepithelial cells to contract.
- *Menopause:* the breasts become softer, more homogeneous, and undergo involutional changes including a decrease in size, atrophy of the secretory portions, and some atrophy of the ducts.

**Fig. 15.1** Illustration of the two milk lines along which the nipples form—occasionally extra nipples can be found.



Tail of
Spence

Upper
inner

Upper
outer

Lower
inner

Lower
outer
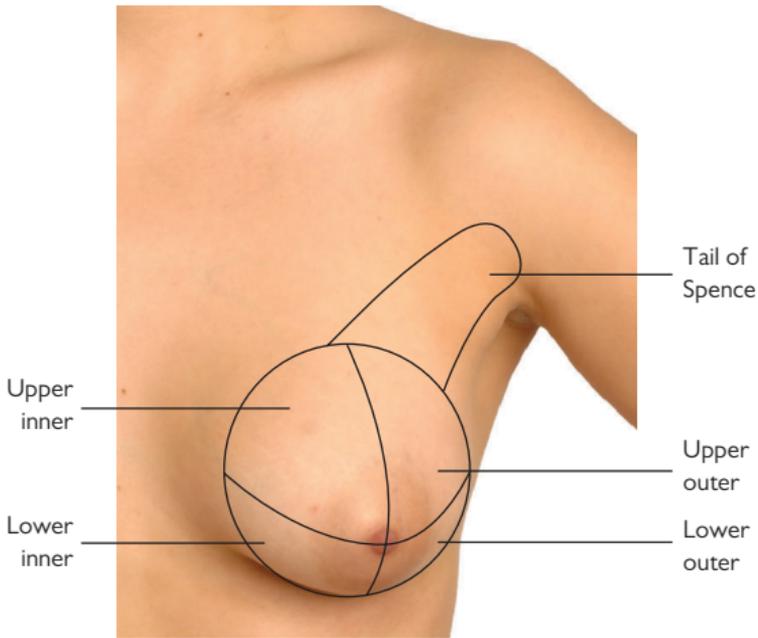
**Fig. 15.2** Illustration showing the four quadrants of the breast with the axillary tail of Spence.

# Important symptoms

### First steps

You should begin by establishing a menstrual history (see ➲ Chapter 13). 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 hormone-dependent change. Other benign causes include mastitis and abscesses. 1 in 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? (See Box 15.1.)
- 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 suppurativa. Ask about abscesses elsewhere, e.g. axilla and groins. The symptoms are often recurrent.

---

**Box 15.1 Galactorrhoea**

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
- Any other neurological symptoms.

---

**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.

**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 oestrogen 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.

# 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.

### 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 in ⟳ Chapter 4).
- 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. 15.3):
- Lean forward whilst sitting.
- Rest her hands on her hips.
- Press her hands against her hips ('pectoral contraction manoeuvre').

**Fig. 15.3** Manoeuvres for breast inspection. (a) Anatomical position. (b) Hands on hips. (c) Arms crossed above the head.



**Fig. 15.4** Correct position of the patient for palpation of the breast.

# Palpation of the breast

### Before you start

Palpation of the breast should be performed with the patient lying at about 45 degrees 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. 15.4).

### 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.

*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 two regularly used methods (see Fig. 15.5) 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 thoroughly 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.

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. 15.3c.

*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 is 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 two 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 the 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) or may be a lump arising from the chest wall.

*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).

(a)  (b)
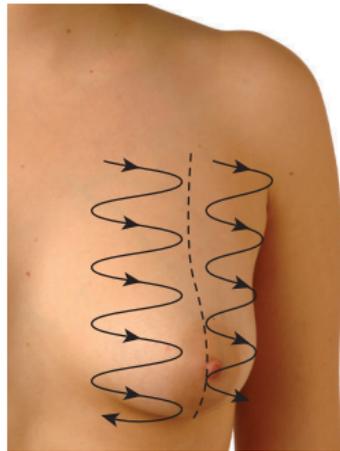


**Fig. 15.5** Two methods for the systematic palpation of the breast. (a) Work circumferentially from the areola. (b) Examine each half at a time, working from top to bottom.

# Examining beyond the breast

### Lymph nodes

The technique is described in detail in ➲ Chapter 3.

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 (Fig. 15.6).
- 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, it 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.

---

**Skills station 15.1**

*Instruction*

Examine this patient's breasts.

*Model technique*
- Clean your hands
- Introduce yourself
- Explain the purpose of examination, obtain informed consent
- Ask for any painful areas you should avoid
- Ask the patient to undress to the waist and to sit upright facing you
- Look for asymmetry, swellings, ulceration, skin changes, scars
- Repeat the inspection with the patient's arms crossed over her head and tensed at her hips (to tense the pectoral muscles)
- Ask the patient to lie back on the couch as in Fig. 15.4
- Using the palmar surface of your first three fingers, gently palpate the entire breast, remembering the axillary tail
  - Remember to elevate the breast and inspect and palpate below
- Palpate the nipple between index finger and thumb. Massage to express any discharge and carefully collect in a universal container
- Palpate the axillary lymph nodes
- Palpate the supraclavicular and cervical lymph nodes
- Examine the opposite side
- Thank the patient and ask them to re-dress.
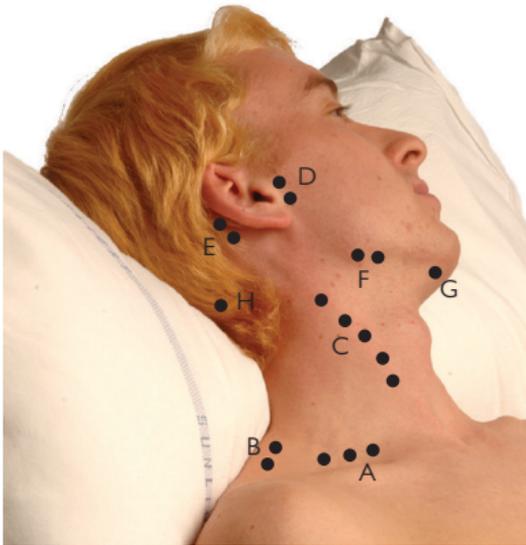
A = Supraclavicular
B = Posterior triangle
C = Jugular chain
D = Preauricular
E = Postauricular
F = Submandibular
G = Submental
H = Occipital

**Fig. 15.6** Cervical and supraclavicular lymph nodes.
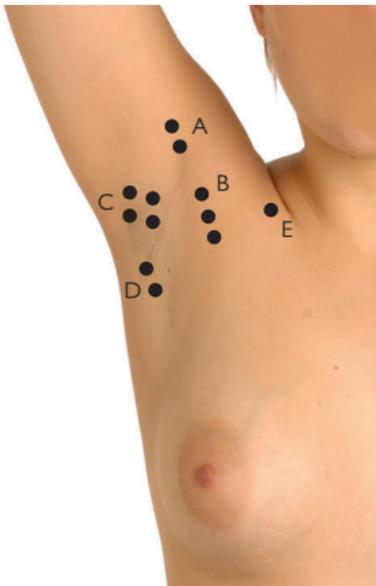


A = Lateral
B = Pectoral
C = Central
D = Subscapular
E = Infraclavicular

**Fig. 15.7** Axillary lymph nodes.

# Breast cancer

## Background
- 1 in 9 women will develop breast cancer in their lifetime (most >50).
- Breast cancer is the most common cancer in women worldwide. It accounts for about 25% of all female malignancies, with a higher proportion in developed countries (see Box 15.2).
  - Male:female ratio is 1:100. Male patients present with the same physical signs and have the same prognosis as female patients.
- Over 1,000,000 new cases occur each year worldwide.

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**Box 15.2 Breast cancer risk factors**
- Female
- Increasing age (80% of cases occur in postmenopausal women)
- Previous history of breast cancer, previous benign breast disease
- Not breastfeeding long term
- Use of hormone replacement therapy or oral contraceptives
- Family history of breast cancer
- No children or few children
- Having children late (especially over 30)
- Early puberty; late menopause
- Obesity (for postmenopausal women only)
- High consumption of alcohol
- Geographical (e.g. higher in Northern Europe, USA).

---

## Symptoms
- 75% symptomatic, 25% present through screening.
  - Patients may present reporting a breast lump, nipple changes, skin changes, or symptoms of metastases
  - 1% of patients present with pain as the only symptom.

## Triple assessment
All suspected breast cancer cases should have 'triple assessment':
- Clinical history and examination.
- Radiological examination (e.g. mammography, ultrasound).
- Pathological examination (e.g. fine needle aspiration/biopsy).

## Inflammatory breast cancer
- Presents with oedematous, indurated, and inflamed skin. Skin may be red, hot, and itchy (easily misdiagnosed as mastitis).
- Accounts for 1–5% of all breast cancers.
- Prognosis is very poor (5-year survival 25–50%).
- Not usually associated with a lump and may be difficult to diagnose by mammography or ultrasound. MRI may be useful.

### Examination findings

*Inspection*
- There may be no features visible on inspection.
  - Mass or dimpling
  - When there is lymphatic invasion the overlying skin has an oedematous look or *peau d'orange* (orange peel)
  - In late disease ulceration may be present
  - Nipples may be normal or show inversion, destruction, deviation, or be associated with a bloody discharge (see Box 15.3)
  - Paget's disease of the nipple/areola looks like eczema.

*Palpation*
- Hard, non-tender lump (may be impalpable).
  - 50% occur in the upper-outer quadrant.
- Indistinct surface with exact shape often difficult to define.
- The lump can be tethered or fixed to the skin, surrounding breast tissue, or chest wall.
- Look for axillary or supraclavicular lymphadenopathy.
  - May present with lymphoedema of the affected arm.

*Further examination*
- A full systems examination should be conducted, searching for evidence of metastasis.

---

**Box 15.3 Nipple discharge**
- 10% due to neoplasia (papilloma or cancer)
- ► Commonest symptom of cancer after 'lump'
- ❶ Beware of neoplasia if discharge is blood-stained, persistent, and from a single duct
- Multiple duct creamy discharge is often due to duct ectasia
- Bilateral galactorrhoea is usually medication-induced in the absence of pregnancy or beyond six months post-partum. The most common pathological cause is pituitary tumour.

# **Other important presentations**

### **Fibroadenoma**

*Background and epidemiology*

- Benign tumours that represent a hyperplastic or proliferative process in a single terminal ductal unit. The cause is unknown.
- Reducing incidence with increasing age; majority occur before the age of 30. Higher incidence in those taking the oral contraceptive pill.
  - May involute in postmenopausal women. May grow rapidly during pregnancy, hormone replacement therapy, or immunosuppression.
- Most stop growing after they reach 2–3cm.
  - Rule of thirds: 1/3 enlarge, 1/3 stay the same size, 1/3 get smaller.

*Symptoms*

- Often asymptomatic.

*Examination findings*

- Typically a smooth, mobile palpable lump.
- No fixation to skin or deep tissues.
- May occur in any area of breast.
  - Like other lumps, occurs especially in the upper-outer quadrant.
- Non-tender.
- Normally solitary but may be multiple.
- No lymphadenopathy.

### **Cysts**

*Background and epidemiology*

- Can appear suddenly and cause pain.
- Commonest palpable lump in women aged 30–50 years.
  - Subareolar main duct cysts may occur in those aged 10–20 years.
- Related to oestrogen metabolism.
  - Can be perpetuated by hormone replacement therapy in women >50.
- Can coexist with cancer.

*Symptoms*

- Often asymptomatic and incidentally picked up on imaging.
- Patient may complain of a palpable, visible, or painful lump.

*Examination findings*

- Round, smooth, symmetrical, discrete lump.
- May be mobile or tender.
- May range from soft to hard.
- It is rare to be able to elicit fluctuance, fluid thrill, or transillumination.

### **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 breastfeeding.

Present with a painful, spherical lump with surrounding oedema. They often show additional signs of inflammation (hot, red). The patient may have constitutional symptoms such as 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 lead 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 intra-ductal 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.

### Mastitis

*Puerperal mastitis*
- Most commonly seen in the first 6 weeks of breastfeeding.
- Caused by staphylococcal infection of ducts.

*Periductal mastitis*
- Mean age is 32 years and there is an increased incidence in smokers.
- Recurs in up to 50% due to persistence of underlying diseased duct.
- Mammary duct fistula:
  - Communication between the skin and a major subareolar breast duct
  - Develops in 1/3 of non-lactating periareolar abscesses.

*Non-puerperal mastitis*
- In many cases, starts as non-bacterial inflammation. Risk of recurrence, secondary infection, and abscess formation is high.
- Risk factors: smoking, diabetes, trauma, hyperprolactinaemia.

*Symptoms*
- Pain, tenderness, swelling (80%).
- Redness (80% cases).
- Lump or diffuse swelling of the breast.
- Systemic features of infection.

*Examination findings*
- Skin of affected area is red, hot, and tender.
- A cracked nipple may be evident.
- There may be a discrete tender lump or diffuse swelling.
- There may be ipsilateral tender axillary lymphadenopathy.
- If there is abscess formation, this may be evident as a firm, tender lump initially which may then develop into a fluctuant swelling.

# 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 is the art of active listening 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 increase 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 in Box 16.1. However, don't overlook physical examination as this is often an important part of the assessment. Physical examination is described elsewhere in this book.

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#### Box 16.1 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.

---

# The history (part 1)

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 Boxes 16.2 and 16.3).

## Demographics

Start by making a note of the patient's name, age, marital status, occupation, ethnic 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—for example, if the patient admits to some depressive symptoms, ask about other symptoms of depression.

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**Box 16.2 (S)OC(R)ATES**

Exactly as in ➲ Chapter 2, you should establish the factors relating to a psychiatric symptom just as you would a physical symptom (see Box 2.5). Obviously, 'site' and 'radiation' do not quite translate to a psychiatric history.

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 long-standing, 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 16.3 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.

# The history (part 2)

### 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 (➔ Chapter 2).

### 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*

- 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.
- Record any adverse experiences (physical or emotional abuse).
- Note any significant life events such as separations and bereavement.

*School*

- Explore the school experiences: 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 volunteer 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 non-threatening 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).

# Risk assessment

This includes not only an assessment of self-harm but also of the possibility of harm to others (Boxes 16.4–16.6). 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 led 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 and/or 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.)

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**Box 16.4 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 16.5 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 16.6 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 means 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/help

# 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 (Box 16.7).

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**Box 16.7 Framework for the mental state examination**

- Appearance and behaviour
- Speech
- Mood and affect
- Thought content
  - Preoccupations
  - Abnormal beliefs
- Perception
  - Disorders of self-awareness
  - Illusions
  - Hallucinations
  - Sensory distortions
- Cognitive function
- Insight
- Summary.
  - Include a statement of diagnosis or differential diagnosis, aetiological factors, and a plan for further investigations and management.

---

### Appearance and behaviour

This involves a brief descriptive note of your observations, both at first contact and through the interview process. It should include:

- Dress and grooming.
  - Evidence of self-neglect? (e.g. seen in depression or drug abuse)
  - Flamboyant clothes with clashing colours? (e.g. in mania)
  - Loose-fitting clothes (may indicate an underlying anorexia or other eating disorder).
- Facial appearance including 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.

### Speech

Describe in terms of:
- Rate.
- Quantity.
  - Increased = 'pressure of speech' and is often associated with flight of ideas (see Box 16.8)
  - Decreased = 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 (Box 16.8).

---

**Box 16.8 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 off 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. The invention of new words with no meaning. Sometimes this term is also used for an abnormal usage of an existing word, sometimes called a 'semantic extension'
- *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.

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 often 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 pre-occupy 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 (Box 16.9). 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 discernible 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 16.9 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:* an exaggerated perception of 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
  - *Thought echo:* the belief that one can hear their thoughts being spoken aloud
  - *Thought blocking* is the experience of having your train of thought suddenly halted.
- *Passivity feelings:* these are 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, often misinterpreting innocent glances
- *The Capgras delusion:* 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 a duplicated world. 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.

## 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, derealization).

*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 and 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*

A 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 (Box 16.10).

They may occur in any sensory modality, although visual and auditory hallucinations are the commonest.

Importantly, hallucinations do not necessarily 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 16.11).

*Sensory distortions*

This includes heightened perception with especially vivid sensations (e.g. hyperacusis), dulled perception, and 'changed perception'. For example, patients may experience objects as having a changed shape, size, or colour.

**Box 16.10 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
- *Gustatile 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 kinaesthetic (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 16.11 Charles Bonnet syndrome**

We highlight this particular syndrome as it 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 realizes 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 realize. 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.

*Notes on conducting the Mini-Mental State Examination (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 (Box 16.12).

- *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 objects 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. Speak 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 lines 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.

Scores of <23 are taken to indicate mild, <17 moderate and <10 severe cognitive impairment. This is a non-linear scale, however.

## 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?

**Box 16.12 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 on p. 512.

*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.
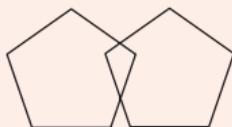


Maximum total score = 30

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.

# Important presentations

## 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 behavioural 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 approximately 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 (Box 16.13).

### 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 two 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.

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* Schizophrenia comes through Latin from the Greek *skhizein* 'to split' and *phren* '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.

*Schneider's first-rank symptoms*

Kurt Schneider[2] listed his 'first-rank' symptoms of schizophrenia in 1959. 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 16.13 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.

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## 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.

[1] Crow TJ (1980). Molecular pathology of schizophrenia: More than one disease process? *British Medical Journal*, 280: 66–8.
[2] Schneider K (1959). *Clinical Psychopathology*. New York: Grune and Stratton.

## 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 three 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 four or more of tachycardia, sweating, trembling or shaking, shortness of breath, a feeling of choking, chest pain, dizziness, light-headedness 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.

*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).

* The word actually means 'fear of the market place'.

## Affective disorders

### Bipolar disorder

It is important to note that patients presenting only with mania, and no evident depression, are also said to have bipolar disorder. There are three 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, self-worth, and glory. The key feature is 'disinhibition'.

There may be several of the following:

- Inflated self-esteem or grandiosity.
- Reduced need for sleep.
- Racing thoughts, 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).

### 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 mood-congruent) in the case of severe depression.

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 suicide plans.

### 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.

## Dementia

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.
- Raised intracranial pressure: neoplasia, hydrocephalus.
- Toxins: heavy metals, organic chemicals, chronic substance abuse.
- Vitamin deficiencies: B12, 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.
- Apraxia.
- Agnosia.
- 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 than 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 extra-pyramidal signs.

*Fronto-temporal dementia*

This accounts for 5% of all dementia. Pick's disease is a form of fronto-temporal dementia characterized by the presence of neuronal 'Pick's bodies' (masses of cytoskeletal elements). The predominance of frontal lobe involvement is evinced by profound personality changes, social impairment, and stereotyped behaviour. However, visuospatial skills are usually preserved. The patient may also show 'primitive reflexes'.

*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. 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 fourth 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 spongiform encephalopathy (BSE). The average age of onset is 27 years, presenting initially with behavioural symptoms.

## 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.

*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.

# Medical conditions with psychiatric symptoms and signs

There are many medical conditions that can give psychiatric clinical features. This can sometimes lead to failure to recognize and treat the underlying medical condition 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
  - Automatisms 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 agnosia, 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:* acute changes of mental state, confusion..
- *Uraemia encephalopathy:* memory impairment, depression, apathy, social withdrawal.

### Vitamin deficiencies

- $B_1$ *(thiamine):* asthenia, fatigue, weakness, depression.
- $B_{12}$ *(cyanocobalamin):* impaired cognitive function.

# The paediatric assessment

# History taking

## Children and doctors

The specialty 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.
- ▶ Let the child know what you are going to do. They may be dreading a needle when you have no intention of using one!

## 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 list in Box 17.1 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; parents are astute observers.
- 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 17.1 Outline 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 siblings' ages, including deaths, miscarriages, and stillbirths
  - Any consanguinity? ('Are you related to your husband by blood?')
- 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.

# Approaching the examination

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

Unless your patient is a neonate, 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?

▶ The vast majority of the examination can be done by inspection so spend most of your time doing this. A common error in formal examinations is that students rush to touch the child and don't spend enough time observing at the start of the examination.

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. See Boxes 17.2, 17.3, and 17.4 for other examination tips.

## 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 saying that 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.

**Box 17.2 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
- Constant chatter from yourself.

**Box 17.3 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 17.4 '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.

# 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? (See Box 17.5.)
- 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*

See Box 17.6. 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 (see Table 17.1).
- Listen for wheeze or stridor (a harsh inspiratory sound caused by upper airways obstruction).
- What type of cough does the child have?
  - If they don't cough spontaneously, ask them to cough for you.
- 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 paradoxus is an important feature of acute severe asthma in children.

*Face*

- Check the conjunctiva for anaemia and the tongue for central cyanosis.
- 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 with splaying of the costal margin in chronic respiratory disease
  - Barrel chest (hyperventilation): air trapping in poorly controlled asthma
  - Pectus carinatum: 'pigeon chest' seen in long-standing asthma
  - Pectus excavatum: normal (and common) variant.

Table 17.1 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 |

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**Box 17.5 Some childhood coughs**

These 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.

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**Box 17.6 The importance of inspection**

▶ ↑ 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.

*Palpation*
- Feel the neck for enlarged cervical lymph nodes.
- Palpate the trachea to ensure that it is central.
- Then move on to the chest:
  - Feel for the apex beat. This may be displaced in effusion, collapse, or tension pneumothorax. The apex may be on the right in dextrocardia with primary ciliary dyskinesia
  - Assess expansion, commenting on extent and symmetry
  - In young children, you may be able to feel crackles.

*Percussion*
Percussion is rarely useful in infants but is in children 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 ➲ Chapter 6 for more details.
- Absent breath sounds in one area suggests a pleural effusion, pneumothorax, or dense consolidation.

## Putting it all together
- See Table 17.2 for some common respiratory conditions.

Table 17.2 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-tracheobron-chitis) | 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 |

# 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 17.7).
- 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 (➲ Chapter 11). See Table 17.3.

*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 parent's 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 17.7 The importance of inspection**

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 17.3 Some common findings when examining the eardrums

| 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 or sudden death?

## 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.

*Blood pressure*

Blood pressure recordings in children are not easy, but they are important, so remember to 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: chest and abdomen*

*Apex beat*

- 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 intercostal 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.

*Four valve areas*

- Palpate in the aortic, pulmonary, tricuspid, and mitral areas for thrills.

*Abdomen*

- Palpate 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.

*Palpation: 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 practise feeling lots of pulses to appreciate the difference.) A thready, weak, or small volume pulse is indicative of hypovolaemia. Look for pulsus paradoxus (➔ Chapter 5).
- 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.

*Auscultation*

Listen to the four valve areas with the diaphragm and bell of the stethoscope (preferably paediatric size) for:

- Heart sounds.
- Added sounds.
- Murmurs (see Box 17.8 and Table 17.4).

*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.

*Fourth heart sound*

Due to forceful atrial contraction.

- Causes include hypertrophic cardiomyopathy and severe hypertension.

### Box 17.8 Murmurs

Auscultate for murmurs over the four 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 2nd–4th left intercostal 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 2nd 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 Chapter 5.

Table 17.4 Quick-spot guide to common paediatric murmurs

| Signs | 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 external genitalia

## 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.

- 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 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:

- Does the abdomen move with respiration?
- Is the patient in pain?
- Jaundice.
- Observe for signs of liver disease (see ⊃ Chapter 7), including spider naevi, xanthomata, and purpura.
- Oedema over the tibia and sacrum.
- Is the child 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, visible liver edge, or spleen (see Box 17.9).
- Peristalsis (important in diagnosing pyloric stenosis during a test feed).
- Gross ascites may be evident—the umbilicus everted.
- Caput medusae (cutaneous collateral veins with blood flowing away from the umbilicus due to ↑ portal venous pressure).
- Check for peritonism as in Box 17.10.

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**Box 17.9 Causes of abdominal distension in a child**

- Fat
- Fluid
- Flatus
- Faeces
- Organomegaly
- Muscle hypotonia.

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**Box 17.10 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'. In the case of peritonitis, any of these manoeuvres may result in pain.

Also ask them to hop on each leg. If they can do this, they do not have peritonitis.

### 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)
  - ▶ ALWAYS watch the child's face (rather than your hand) to see if they are in pain whilst you palpate.
- 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. See Box 17.11.
  - 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. Percuss from the midline to the flanks. If you suspect ascites (dullness in the flanks), test for shifting dullness (➲ Chapter 7).
- 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.

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**Box 17.11  Confirming hepatomegaly**

If in doubt, confirmation of liver enlargement can be made by:
- Placing the stethoscope over the xiphisternum
- Gently scratching the abdomen, progressing upwards from the right iliac fossa.
  - When the scratching hand is over the liver, the sound will be heard through the stethoscope.

# Examining the external 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 epispadias (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 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.

# The nervous system

## Key points from the history

- Detailed birth and perinatal history including:
  - Maternal drugs/illness
  - Presence of polyhydramnios (neuromuscular disease)
  - Whether resuscitation was needed at delivery
  - Neonatal infections.
- Careful history of the developmental milestones (see Table 17.5).
  - At exactly what ages were they attained?
  - Is there any delay (common) or regression (rare)?
- 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?
  - Are there increased falls or trips compared with other children?
  - Is there a change in coordination?
- Any evidence of weakness. Does the child have difficulty with:
  - Climbing stairs or brushing their hair (proximal weakness)?
  - Opening jars or writing (distal weakness)?
- 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

Neurological examination of children and infants can be made largely by observation of their play and gait. Doing this provides a useful screening test, after which a more formal neurological assessment can be done to more specifically define an abnormality.

Neurological examination of children and infants is often dreaded by students and junior doctors alike, but if you learn a stereotyped examination sequence, it becomes easy.

The examinations of younger and older children are described separately.

Table 17.5 Developmental milestones

| Age | Gross motor | Fine motor | Language | Social |
|-----|-------------|------------|----------|--------|
| 3 months | Head control, pushes up with arms | Opens hand | Laughs | Smiles (6 weeks) |
| 6 months | Sits | Palmar grasp, reaches, transfers | Babbles (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 block tower | 2-word phrases | Mimics |
| 2 years | Kicks, runs | Draws straight line, 6–8 block tower | 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 |

### Neurological examination: infant or small toddler

*Start with the infant sitting on the mother's lap:*
- Note how alert they are.
  - Do they interact with their mother and with you?
  - Any spontaneous vocalization or language?
- Observe range and symmetry of eye movements whilst tracking an interesting object (a ball, a torch, or your face).
  - If the baby can re-fix on an object moved from the central to peripheral vision, you can assume that their visual fields are intact
  - Note their facial symmetry when smiling (or crying)
  - You have now made a basic assessment of their cranial nerves!
- Opportunistically note the baby's arm and hand movements.
  - Do they grasp objects with each hand?
  - Can they transfer objects between each hand?
  - Do they mouth objects?
  - Can they scribble on paper?
  - Observe the upper-limb coordination, dexterity, and distal power.
- If the toddler can walk, watch them wander about the room.
  - What is the gait like?
  - Are they able to squat to pick objects up from the floor and then stand again (this requires good proximal muscle power)?

*On the examination couch:*
- Start with the baby lying supine, and note attempts to roll over or sit up (this will help you assess truncal and limb power).
- Also note their limb posture as an indication of tone:
  - Are they lying in a 'frogs-legs' position (hypotonia)?
  - Are their upper limbs held in flexion or extension (hypertonia)?
  - What are the spontaneous movements like?
- Using the baby's arms, pull them to sit.
  - Note the degree of head control.
- From sitting, note the need for support, and how curved the back is.
  - If the baby is sitting unsupported, gently tip to one side and note the righting reflex and its symmetry (if tipped to the left, the left arm should extend to prevent the baby from falling)
  - This will help you assess tone, power, and movement of the arms.
- If the baby is over about 6 months of age, lift to stand.
  - See if the legs 'scissor' (adduction is a sign of hypertonia in the lower limbs)
  - Does the baby seem to 'move as one piece' (hypertonia)?
- Now lift the baby onto their front whilst watching for parachute reflex (both arms should extend forwards. Is this equal?).
  - Observe their head control and upper limb strength when prone
  - Do they try to lift themselves up like doing a press-up?

*Lift the baby back onto the mother's knee*
- Fundoscopy. You will need an assistant to get the baby's attention behind you and plenty of patience!
- Examine the deep tendon reflexes.
- Take the head circumference and plot it on a centile chart.

*Primitive reflexes*
- These are reflexes (Box 17.12) seen in young babies which become 'extinct' at certain predictable ages (see Table 17.6).
- Preservation of primitive reflexes (or redevelopment) may be an indicator of neurological disease.

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**Box 17.12 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 5th and 6th cervical roots producing Erb's palsy.

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Table 17.6 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 |

### Neurological examination: older child

- Whilst taking your history and performing the examination, you can make an informal assessment of the child's language and general understanding and participation in the consultation—is it age appropriate?
- Ask the child to stand up with their feet together, arms outstretched with palms facing upwards and eyes closed for 30 seconds. Watch for:
  - Pronator drift: a sensitive sign of UMN weakness of the upper limbs. The affected arm drifts downwards and pronates
  - Romberg's sign: standing with eyes closed is difficult due to impaired proprioception
  - Is the child able to stand straight? Hip or knee flexion causes a crouching stand.
- Ask the child to walk and then run over a good distance. Is the gait normal or abnormal? Some signs to watch to test for include:
  - Spastic hemiparesis: foot is kept in plantar flexion and catches the floor. There may be a swing of that leg, and the ipsilateral arm may be held flexed. Look for asymmetric toe wear on the soles of the shoes
  - Spastic diplegia: the child walks on their toes with both feet and has a crouched stance
  - Cerebellar ataxia: wide-based, staggering irregularly. Walking on a pretend tight-rope is very difficult
  - Proximal weakness: waddling gait. Throwing of hips to each side with each step. Patients often also have a lumbar lordosis.
- Move to the couch.
- Inspect the symmetry of the child's muscle bulk, and look at the soles of their shoes for unequal or abnormal wear.
- Tone. This may be easier to comment on through inspection of posture—children find it difficult to relax. Increased tone can be:
  - Spastic: feet held in equinus, legs extended and adducted (scissoring), arms either flexed or extended with wrists pronated
  - Dystonic: unusual postures, sometimes brought about by movement.
- Power. Demonstrate to the child what it is that you want them to do, and make it fun for them. Follow the adult sequence (➲ Chapter 8).
  - During your examination, try to distinguish between proximal and distal muscle weakness.
- Reflexes. Hold your thumb over the tendon and hit this with the hammer—this is less threatening to children.
  - Plantar reflex: run your thumb nail along the lateral aspect of their sole and then medial aspect of the ball of their foot (rather than the sharp end of a tendon hammer)
  - Look for the first movement of the big toe
  - The plantar reflex can be extensor until one year of age.
- Sensation: Generally only test if there is a specific indication to do so.
  - Isolated sensory losses without accompanying motor signs are extremely rare in childhood, so motor examination is generally enough unless there is a specific concern about sensation.

- • Distinguish spinothalamic sensation (pain, temperature) from dorsal column sensation (light touch, two-point discrimination, proprioception).
- • Coordination.
  - • Lower limbs: you have already assessed this by getting the child to walk
  - • Upper limbs: finger–nose test. Ask the child to move their finger from their nose to your finger and back again as accurately as possible. Do this with both hands. Are they able to reach their target without missing?

For examination of the cranial nerves of an older child, follow the same sequence as for an adult (🔗 Chapter 8).

### Additional optional tests

*Gower's sign:*
- • A test for proximal muscle weakness.
  - • Ask the child to lie supine on a mat, and then get them to stand
  - • A child with a positive Gower's sign will turn prone, then use their hand to 'climb up' their legs to stand.

*Fog's test*
- • Associated movement in the upper limbs (e.g. flexion due to spasticity) when the child is asked to walk on their heels or tip-toes.

# 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.

# Developmental assessment

Development is a continuous process, the rate of which varies considerably between normal children. 'Development' describes the acquisition of learned skills and occurs in a cephalo-caudal direction (head to toe)—a child cannot sit before they develop head control, otherwise they would not be able to look around.

Development is divided into four areas (see also skills station 17.1):
- Gross motor.
- Fine motor and vision.
- Speech and hearing.
- Social.

Delay in all four 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 to them and they do not bother to learn this until later.

## 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.
- You will have to be opportunistic, and record (or present) your findings as you go along.
- Be systematic and evaluate each of the four 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.

## Developmental milestones
These are detailed in Table 17.5 and warning signs in Table 17.7.

## Equipment for developmental assessment
- Wooden blocks.
- 'Hundreds and thousands'.
- Pencil and paper: for assessing fine motor skills.
- Different coloured card/colourful books.

| Table 17.7 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 |

**Skills station 17.1 Developmental assessment**

*Gross motor*
- Say what you see!
  - Are they sitting up? Cruising? Walking? Are they crouching down and standing again?
  - Roll a ball to them. Can they kick it? Can they catch a ball?

*Fine motor*
- Hand them a block.
  - Do they take it with a palmar grasp?
  - Do they transfer the block to the other hand?
  - Do they put it in their mouth?
  - How many blocks can they put on top of each other to make a tower?
  - Can they pick up a 'hundreds and thousands' from your hand?
  - How accurate is their pincer grip?
  - The child needs good vision to see the 'hundreds and thousands', so you are also making a crude assessment of their sight.
- Give them a pencil and paper.
  - Do they scribble?
  - Is it linear or circular?
  - Can they copy a line/circle/cross/square?

*Speech*
- Chat to them during the examination.
  - Are they babbling?
  - Do they say any words?
  - Are they putting one or more words together?
  - Can they name body parts or colours?

*Social*
- Examine the child's interactions with others.
  - Does the child smile and laugh?
  - Are they anxious about strangers (you!)?
  - Do they play peek-a-boo with you?
  - Play with them—what do they like to do?
  - Do they play symbolically?
- Do they have make-believe play?

# 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 (Table 17.8) is used to gauge the need for resuscitation.

Table 17.8 APGAR score

| Sign | 0 | 1 | 2 |
| --- | --- | --- | --- |
| Appearance | White/blue | Blue extremities, pink trunk | 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 postnatal 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.

*Hand 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 (preferably sleeping).
- *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 pulse 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 an 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 4th or 5th 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 to check for the common condition of hydrocoele.
- *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 syndactly (fused digits) or polydactyly (extra digits—surprisingly easy to miss!).

*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 two 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 fingertips 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 semi-wakefulness 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 investigation.

*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 Table 17.6)*
- 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 birth-weight on a centile chart is of utmost importance.

# 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 should only be performed once you have been trained specifically in the correct technique by a more senior colleague.

## 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.

# 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 pull back

- Whenever you inject anything, you should advance the needle and attempt to pull back 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 = 10mg/ml) 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.

## Other notes on local anaesthetics

- The maximum does of lidocaine is 3mg/kg in an adult.
  - This can be increased to 7mg/kg if mixed with adrenaline (although never to be used in this way at end-arteries).
- Lidocaine is a weak base and only works in its non-ionized form. It is, therefore, relatively ineffectual in infected (acidic) tissue.
- Lidocaine and other local anaesthetics sting on initial infiltration so warn the patient.

# Hand hygiene

### When?

The WHO World Alliance for Patient Safety, in 2006, identified 'five moments' for hand hygiene. These are:

- Before patient contact.
- Before an aseptic task.
- After body fluid exposure risk.
- After patient contact.
- After contact with a patient's surroundings.

### Soap or alcohol gel?

Repeated washing with soap and water can cause skin dryness and can be time consuming. For these reasons, alcohol gel has become commonplace in clinical settings. There are no hard and fast rules but:

- Alcohol gel should not substitute soap and water if your hands are visibly soiled or if you are undertaking an aseptic procedure.
- Remember that alcohol gel is not effective against *Clostridium difficile*.

### Soap and water technique

- Adhere to the 'bare below elbow' rule.
- Wet hands with water.
- Apply soap (from a dispenser) to cover all hand surfaces.
- Ensure all seven parts of the hands are thoroughly cleaned:
  - Rub hands palm-to-palm
  - Rub back of each hand with the palm of the other, fingers interlaced
  - Rub hands palm-to-palm with fingers interlaced
  - Lock hands together and rub backs of fingers against opposite palm
  - Rub thumbs in rotational movement with opposite hand
  - Rub tips of fingers into opposite palms
  - Rub each wrist with opposite hand.
- Hold hands under running water, rub vigorously to remove all suds.
- Turn off taps using elbows.
- Dry thoroughly with paper towel.
- Dispose of paper towels in appropriate clinical bin (using foot pedal).
- DO NOT TOUCH any other objects until task is undertaken and completed.

### Alcohol gel technique

Essentially the same technique as above but no need to rinse or dry with a paper towel.

- Squirt small amount of gel onto centre of palm.
- Ensure all seven parts of the hands are thoroughly cleaned as above.
- Allow 20–30 seconds for hands to dry, holding hands up.
- Following disinfection, DO NOT TOUCH any other objects prior to commencing procedure.

# Consent

See the latest guidance at ✆ http://www.gmc-uk.org

## Introduction

Consent is permission granted by a person allowing you to subject them to something; anything from physical examination to surgical procedures. Performing an act on a competent adult without their consent constitutes a criminal offence. (See also Boxes 18.1 and 18.2.)

## Capacity

The patient must be able to understand what a procedure involves, the possible consequences of a decision or of failure to make a decision. All adults are assumed to have capacity unless demonstrated otherwise.

*Assessing capacity*

The patient must be able to:
- Understand the information including any consequences.
- Retain the information.
- Weigh the information as part of decision making.
- Communicate their decision.

*A patient lacking capacity*
- Your reasons for believing a patient lacks capacity to make a certain decision should be clearly documented.
  - A patient may become temporarily incapacitated by, for example, acute confusion. Treatment may only be carried out in these circumstances if it cannot reasonably be delayed until incapacity is resolved. If this is the case, treatment must be decided according to the best interests of the patient.

## Voluntary consent
- Consent is only valid if given voluntarily, without pressure from relatives, friends, or medical professionals.

## Information

Patients must be provided with sufficient information to enable them to make an informed decision. Information must include:
- What the procedure entails and the rationale for doing it.
- Any alternatives available.
- Significant risks.
  - This includes any 'significant risk which would affect the judgement of a reasonable patient', not just those risks deemed significant by a responsible body of medical opinion (the Bolam test). Failure to disclose such risks may render you guilty of negligence.
- Additional procedures that may be necessary under the same anaesthetic should be discussed during initial consent.
- If patients refuse information about a procedure, this should be clearly documented and the patient provided with the opportunity to discuss later.

## Consent forms
- Written consent is evidence that consent has been sought but does not confirm its validity.
- If consent is not voluntary, information is lacking, or the patient lacks capacity, then consent is not valid regardless of the presence of a consent form.
  - Certain procedures (included in the Mental Health Act and Human Fertilisation and Embryology Act) require written consent.
- ▶ Consent that is oral or non-verbal may also be valid.

## Who should seek consent?
- Ideally, the professional providing the treatment or investigation in question, though this is not always possible.
- The professional seeking consent should at least have sufficient knowledge to understand and explain the procedure, its indication, and any risks involved.
  - If you are asked to seek consent for a procedure but lack this knowledge, it is your responsibility to seek advice from colleagues; failure to do so may result in invalid consent.

## Refusal to consent
- If an adult with capacity refuses to give consent for a procedure, this **must** be respected (except in specific circumstances outlined in the Mental Health Act), even if refusal will lead to death of the patient or their unborn child.
- In these circumstances, rigorous examination of a patient's competence is necessary.
  - The same is true if a patient withdraws consent at any time, if they still have capacity.

## Advanced refusal
- Advance refusal is valid if made at a time when a patient is competent and appropriately informed.
- Applicable when patient lacks capacity.
- Failure to respect the refusal may result in legal action.
- If doubt exists as to validity, the courts must be consulted.

## Adults lacking capacity
- May be temporary, permanent, or fluctuating.
- ▶ No-one may give consent on behalf of an incompetent adult, unless a valid Lasting Power of Attorney exists.
- ▶ Patients must be treated in their best interests (not just medical interests) taking into account psychological, religious/spiritual, and financial well-being.
- Those close to the patient should be involved unless the patient has previously made clear that they should not be; independent patient advocacy services exist for consultation when the patient does not have anyone close.
- Where there is doubt as to best interests or capacity, the High Court may give a ruling.

## Lasting powers of attorney (Mental Capacity Act 2005)

- A document created by someone (the 'donor') to confer authority to give consent for investigation or treatment (as well as other issues) to a named individual(s) ('donees').
- Must be registered.
- Only valid when the patient lacks capacity.
- Must specifically authorize the donee to make decisions regarding welfare or medical treatment.
  - ▶ Unless specifically stated, do not extend to decisions about life-sustaining treatment.

## Patients under 18 years of age

*16–17 years*

- If competent, may consent to or refuse an intervention.
- If incompetent, an individual with parental responsibility may provide consent.

*Under 16: Gillick competence*

- A child under 16 may consent to treatment if they are able to fully understand what is involved in an intervention.
  - This may apply to some interventions and not others.
- If a child is Gillick competent, parental consent is not required, though it is good practice to encourage a child to inform their parents unless this is not in their best interests.

*Overriding decisions: under 18 years*

- Refusal may be overridden by an individual with parental responsibility or the courts.
- Should consider the person's welfare as a whole. May involve sharing information that the child does not wish divulged; necessary if refusal puts the child at serious risk.
- ▶ In dire emergency, where a person with parental responsibility is unreachable or refuses consent for life-saving treatment that appears to be in the best interests of the child, it is acceptable to preserve life.

**Box 18.1 Pre-procedure ABCDE**

Questions to ask yourself before any procedure:
- **A** = Allergies
- **B** = Bloods
- **C** = Consent
- **D** = Drug history
- **E** = Emergency cover in case of complication or failure of the procedure.

**Box 18.2 WHO checklist**
- The WHO pre-procedure checklist is a series of questions to ask of the patient and the person performing the procedure
- This is usually reserved for complex interventional procedures and surgery
  - Check your local guidance
- Questions cover introductions, patient details, allergies, details of the procedure and any other pre-procedure checks
  - You should familiarize yourself with the questions and perform these checks yourself before performing any procedure (even if not on a formal WHO checklist form)
- For more information, go to ℘ http://www.who.int

# Aseptic technique

▶ You should always consider the sterility of the items to be touched before you begin each procedure. If some or all items need to remain sterile, an aseptic technique should be used.

## Aseptic non-touch technique

The highest level of asepsis, designed to minimize or completely remove the chance of contamination, is known as 'aseptic non-touch technique' (ANTT) (Boxes 18.3 and 18.4).

*Before*
- Wash hands with soap and water or alcohol gel.
- Put on disposable apron and any other protective items.
- Clean trolley/tray with wipes and dry with a paper towel.
- Gather equipment and put on the lower shelf of the trolley.
- Take trolley/tray to the patient.

*During*
- Wash hands with alcohol gel.
- Remove sterile pack outer packaging and slide the contents on to the top shelf of the trolley or onto the tray, taking care not to touch the sterile pack.
- Open the dressing pack using only the corners of the paper, taking care not to touch any of the sterile equipment.
- Place any other required items on the sterile field ensuring the outer packaging does not come into contact with the sterile field.
- Put a pair of non-sterile gloves on to remove any dressings on the patient and ensure that they are positioned appropriately.
- Discard gloves and wash hands.
- Put sterile gloves on.

*After*
- Dispose of contaminated equipment in the rubbish bag from the dressing pack. Dispose of all packaging.
- Dispose of aprons and gloves in the appropriate waste as per local policy.
- Wash hands.
- Clean the trolley with detergent wipes and dry with a paper towel.

## Two-person technique

- An assistant can be very helpful in maintaining the position of the patient, opening packs, and decanting solutions for the person performing the procedure.
- The 'clean practitioner' must wear the sterile gloves and open the first pack to establish a sterile field.
- The second ('dirty') practitioner can then open all the other equipment and drop onto the sterile field.

**Clean technique**
- This is a modified aseptic technique, aiming to prevent the introduction or spread of micro-organisms and to prevent cross-infection to patients and staff. This is used when true asepsis is not required (e.g. when dealing with contaminated sites or when *removing* drains and catheters).
- Sterile equipment is not always used.
- 'Clean technique' allows the use of tap water, non-sterile gloves, multi-pack dressings, and multi-use containers of creams and ointments.

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**Box 18.3 ANTT or 'clean' technique?**

*When to use ANTT*
- Insertion, repositioning, or dressing invasive devices such as catheters, drains, and intravenous lines
- Dressing wounds healing by primary intention
- Suturing
- When sterile body areas are to be entered
- If there is tracking to deeper areas or the patient is immunocompromised.

*When to use clean technique*
- Removing sutures, drains, urethral catheters
- Endotracheal suction, management of tracheostomy site
- Management of enteral feeding lines
- Care of stomas
- Instillation of eye drops.

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**Box 18.4 Interruptions**
- If the sterile procedure is interrupted for more than 30 minutes, new sterile packs should be opened and the sterility process started from scratch.

# Subcutaneous and intramuscular injections

Usual sites for subcutaneous injections are upper arms and the abdomen, particularly the periumbilical region.

Intramuscular injections can be administered at any site with adequate muscle mass. Usual sites are deltoids and the gluteal region (upper, outer quadrant of buttock).

## Contraindications
- Contraindications regarding the drugs being injected will vary dependent upon the drugs being administered.
- Infection at the injection site.
- Oedema or lymphoedema at the injection site.

## Risks
- Incorrect drug and/or dosage administered.
- Allergy to drug(s).
- Haemorrhage, haematoma.
- Infection.
- Injection into a blood vessel.
- Injection into a nerve.

## Equipment
- Appropriate syringe.
- 25G (orange) needle (usually).
- Prescribed drug.
- Prescription chart.
- Antiseptic swab.
- Plaster.

## Before you start
- Assess patient for drugs required (i.e. for pain relief, vomiting, etc.).
- Refer to prescription chart, double-checking the appropriate drugs and dosage to be given.
  - ► Always ensure you are fully aware of any possible side effects of any drugs you are due to administer.
- Double-check the prescription chart for date and appropriate route for administration.
- Check administration of previous dose—not too soon after last dose?
- Ensure that the drug to be given is within its use-by date.
- Check patient and chart for any evidence of allergies, or reactions.
- Once all above completed as per hospital policy, draw-up required drug and check appropriate needle size.
- Complete appropriate documentation.
- Once checked by suitably qualified staff, take drug and prescription chart to the patient.

### Subcutaneous procedure

- Introduce yourself, confirm the patient's identity, explain the procedure and obtain informed consent.
- Check with patient: name and date of birth (if capable).
  - If incapable, check name band with another healthcare professional.
- Select appropriate site, and cleanse with the antiseptic wipe.
- Grasp skin firmly between thumb and forefinger of your left hand.
- Insert needle at 45° angle into the pinched skin, then release skin from your grip.
- Draw syringe plunger back, checking for any blood. If none, inject drug slowly.
  - ▶ If any blood is noted on pulling the plunger back, withdraw and stop procedure—provide reassurance and explanation to the patient.
- Once the procedure is completed without complication, withdraw needle and discard into a sharps bin.
- Monitor patient for any negative effects of the drug.

### Intramuscular procedure

- Introduce yourself, confirm the patient's identity, explain the procedure, and obtain informed consent.
- Check with patient: name and date of birth (if capable).
  - If incapable, check name band with another healthcare professional
- Select appropriate site, and cleanse with the antiseptic wipe.
  - 🚸 If injecting into the buttock, mark a spot at the upper, outer quadrant to avoid the sciatic nerve
  - 🚸 If using the deltoid muscle, feel the muscle mass and ensure there is enough muscle to take the needle.
- Insert needle at 90° angle into the skin.
- Draw syringe plunger back, checking for any blood. If none, inject drug slowly.
  - ▶ If any blood is noted on pulling the plunger back, withdraw and stop procedure—provide reassurance and explanation to the patient.
- Once the procedure is completed without complication, withdraw needle and discard into a sharps bin.
- Monitor patient for any negative effects of the drug.

### Documentation

- Drugs should always be signed for as per local policy.
- Signature and time should be clearly recorded.
- Site drug administered.
- Reason for drug administration, time given and any impact on the patient should be recorded.
- Immediate vital signs should be recorded in notes.
- Any causes for concern arising from administration of drugs should be clearly documented in the medical notes.
- Signature, printed name, contact details.

# Intravenous injections

Intravenous injections can be administered by puncturing the vein with a needle and syringe and injecting directly. The procedure below describes injecting via an intravenous cannula. If no cannula is in place, cannulate first.

▶ Ensure that you comply with the local policy regarding drug administration. In hospital, two healthcare professionals should usually check and administer medication.

### Contraindications

- Contraindications regarding the drugs being injected will vary dependent upon the drugs being administered.
- Infection at the cannula insertion site.
- Thrombosis within the vein to be injected.

### Risks

- Incorrect drug and/or dosage administered.
- Allergy to drug(s).
- Injection of air embolus.

### Equipment

- Appropriate syringe (dependent upon quantity of drug to be administered).
- Prescribed drug.
- Saline flush (10ml syringe with sterile saline).
- Prescription chart.
- Antiseptic swab.

### Before you start

- Assess patient for drugs required (i.e. for pain relief, vomiting, etc.).
- ▶ Refer to prescription chart, double-checking the appropriate drugs and dosage to be given.
  - ▶ Always ensure you are fully aware of any possible side effects of any drugs you are due to administer.
- ▶ Double-check the prescription chart for date and appropriate route for administration.
- ▶ Check administration of previous dose—not too soon after last dose?
- ▶ Ensure that the drug to be given is within its use-by date.
- ▶ Check patient and chart for any evidence of allergies, or relevant drug reactions.
- Always comply with the local hand hygiene practices.
- Once all above completed as per hospital policy, draw-up required drug and check appropriate needle size.
- Complete appropriate documentation.
- Once checked by suitably qualified staff take drug and prescription chart to the patient.

**Procedure**

- Introduce yourself, confirm the patient's identity, explain the procedure and obtain informed consent.
- Check with patient: name and date of birth (if capable).
  - If incapable, check name band with another healthcare professional
  - ⚠ The patient may need to be assisted to change position, if unable to move themselves, and to enable access to an appropriate site.
- Cleanse the cannula port with the antiseptic wipe.
- Attach the saline flush to the syringe port and inject a few ml to check patency of the cannula.
  - ► Watch for a bleb forming as consequence of extravasation.
- If no problems are encountered, swap the flush for the drug-containing syringe and inject drug slowly.
- To finish, inject a few more ml of saline into the cannula port and re-attach the bung.
- Once the procedure is completed without complication, withdraw needle and discard into a sharps bin.
- Monitor patient for any negative effects of the drug.

**Documentation**

- Drugs should always be signed for as per local policy.
- Signature and time should be clearly recorded.
- Site drug administered.
- Reason for drug administration, time given, and any impact on the patient should be recorded in the notes.
- Immediate vital signs should be recorded in notes.
- Any causes for concern arising from administration of drugs should be clearly documented in the medical notes.
- Signature, printed name, contact details.

# Venepuncture

### Risks
- Bleeding, haematoma.
- Infection.
- Accidental arterial puncture.

### Inappropriate sites
- Oedematous areas.
- Cellulitis.
- Haematomas.
- Phlebitis or thrombophlebitis.
- Scarred areas.
- Limb in which there is an infusion.
- Upper limb on the side of a previous mastectomy and axillary clearance.
- Limbs with arteriovenous (AV) fistulae or vascular grafts.

### Equipment
- Gloves.
- Sterile wipe (e.g. chlorhexidine or isopropyl alcohol).
- Cotton wool balls or gauze.
- Tape.
- Tourniquet.
- Needle (try 12G first).
- Syringe (size depends on amount of blood required).
- Collection bottles.

### Procedure: needle and syringe
- Introduce yourself, confirm the patient's identity, explain the procedure, and obtain verbal consent.
- Position the patient appropriately: sat comfortably with arm placed on a pillow.
- Wash hands, put on your gloves and apply the tourniquet proximally.
- Identify the vein; the best location is often at the antecubital fossa.
  - Palpable (not necessarily visible) veins are ideal.
- Clean the site with the wipe, beginning centrally and moving outwards in concentric circles/swirls.
- Whilst the sterilizing solution dries, remove the needle and syringe from packaging and connect together.
- Unsheathe the needle.
- Using your non-dominant thumb, pull the skin taut over the vein in order to anchor it.
- Warn the patient to expect a 'sharp scratch'.
- Insert the needle, bevel up, at an angle of 30 until a flashback is seen within the hub of the needle.
  - With experience you will feel a 'give' as the vein is entered.
- Hold the syringe steady and withdraw the plunger slowly until the required amount of blood is obtained.
- Release the tourniquet.

- Remove the needle, holding cotton wool or gauze to the puncture site.
- Secure the cotton wool or gauze in place or replace with a plaster.
- Vacuum collection bottles are filled by puncturing the rubber top with the needle and allowing the blood to enter the tube.
- Label the tubes at the patient's bedside and dispose of the sharps in a sharps bin.

### Procedure: vacuum device

The procedure is much the same as with a syringe but:
- Vacutainer needles are double-ended, with one end a standard needle, the other covered by a rubber sheath. This end inserts into the holder and is screwed in place.
- On penetrating the vein no flashback is seen.
- Once the needle is in place, vacuum collection bottles are inserted into the holder over the sheathed needle in turn—the holder must be held firmly in place.
- Bottles are self-filling; some require filling to a pre-defined level or tests will be invalidated.
- Remove the tourniquet before removing the last bottle, then remove the needle from the skin.

### Procedure tips

- If no veins are visible or palpable, don't limit yourself to the upper limb: any peripheral vein will suffice.
  - If veins are still not visible, try warming the limb.
- If several attempts have failed, seek help from a colleague.
- If the vacuum collection system is proving difficult, try using needle and syringe:
  - A 'flashback' will be seen on entering the vein
  - The flow of blood may be controlled
  - If this also proves unsuccessful, try using a butterfly needle (Box 18.5).

### Documentation

- Detailed documentation of the procedure is usually not required—but you should record that blood was taken and what tests it has been sent for.
- Record any adverse incidents during the procedure or if multiple attempts were performed.
  - If a particularly good vein was found, you may wish to record this for the benefit of the person taking blood next time.
- Signature, printed name, contact details.

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**Box 18.5 Butterfly needles**

A butterfly is a short needle with flexible 'wings' on either side, and a length of flexible tubing to connect to the syringe. It is easy to manoeuvre once the skin is penetrated, and can be easily fixed in place by the wing, pressed down by the non-dominant thumb. It carries a greater risk of needle-stick injury.

# Sampling from a central venous catheter

▶ Central venous lines should only be used for blood sampling 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.

The following describes venous blood sampling from a line in the internal jugular vein. The principles are the same for a line at any site.

### Risks
- Clot or infection in the line.
- Air embolus.
- Physical damage to the line: burst or torn port.

### Equipment
- 3 x 10ml syringes.
- 0.9% isotonic or heparinized saline.
- Chlorhexidine spray or iodine solution.
- Sterile gauze.
- Sterile gloves and apron.
- Sterile drape.

### Procedure
- Introduce yourself, confirm the identity of the patient, explain the procedure, and obtain verbal consent.
- Stop any infusions (if possible) 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 and put on a pair of sterile gloves and apron.
- Spray the line end with the chlorhexidine solution or wipe with gauze dipped in iodine.
- Clamp the line port and remove the cap, if present.
- Connect a 10ml syringe to the port and then unclamp.
- Withdraw 5–10ml of blood, clamp the line, and remove the syringe.
- Discard the blood.
- Repeat the procedure with a new syringe, withdrawing 10ml.
- Clamp the line, disconnect the syringe.
  - Keep this sample.
- Fill the final syringe with saline and attach it to the port.
- Unclamp the port and instil the saline.
- Clamp the port again before disconnecting the syringe.
- Replace the port cap.

**Procedure tips**

- Always be sure to clamp the port *before* removing the syringe and unclamp before withdrawing blood or instilling the saline.
- Most central lines have several ports: which should I use?
  - Blood should ideally be sampled from the port with its hole at the tip of the line—this is often the brown port
  - Check the ports: most will have the gauge printed on them, choose the largest gauge port available.
- Be sure to remove any bubbles from the saline before instilling.
- Infusions must be stopped: otherwise a significant portion of the sample obtained may be the solution that is entering via the other port giving inaccurate results at analysis!

# Arterial blood gas sampling

### Contraindications
- Negative modified Allen's test.
- Cutaneous or subcutaneous lesion at the puncture site (Box 18.6).
- Surgical shunt (e.g. in a dialysis patient) in the limb.
- Infection or known peripheral vascular disease at the puncture site.
- Coagulopathy.

### Risks
- Bleeding.
- Haematoma.
- Arteriospasm.
- Infection.
- False aneurysm formation.
- Arterial occlusion.

### Equipment
- Gloves.
- Sterile wipe (e.g. isopropyl alcohol).
- Cotton wool balls.
- Tape.
- Gauze.
- Heparinized self-filling syringe and needle.

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#### Box 18.6 Choosing a site

The radial artery at the level of the radial styloid is the usual site of choice as it is both superficial and easily accessible.

If the vessel is not obviously palpable, it is also possible to sample arterial blood at the brachial artery in the antecubital fossa or femoral artery just distal to the inguinal ligament.

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### Procedure: radial artery
- Introduce yourself, confirm the patient's identity, explain the procedure, and obtain informed consent.
- Position the patient appropriately: sitting comfortably with arm placed on a pillow, forearm supinated, wrist passively dorsiflexed.
- ▶ Confirm ulnar arterial supply to the hand before starting (modified Allen's test):
  - Compress the radial and ulnar arteries with your thumbs
  - Ask the patient to make a fist and open it
  - The hand should appear blanched
  - Release pressure from the ulnar artery and watch the palm
  - The palm should flush to its normal colour
  - ▶ If not, there may be inadequate ulnar arterial supply and damage to the radial artery during blood taking may result in critical ischaemia.
- Put on your gloves.
- Identify the radial artery with index and middle fingers of your non-dominant hand.

- Clean the site, beginning centrally and spiralling outwards.
- Whilst the sterilizing solution dries, remove the needle and syringe from packaging and attach the needle to the end of the syringe.
- Eject excess heparin from the syringe through the needle.
  - ⚠ Check local equipment. Some heparinized syringes contain a heparinized sponge and excess heparin/air should not be expelled
- Warn the patient to expect a 'sharp scratch'.
- Whilst palpating the artery (but not obliterating the pulsation), insert the needle just distal to your fingertips, bevel facing proximally, at an angle of 45–60° until a flashback is seen within the needle chamber.
- Hold the syringe steady and allow it to fill itself with 1–2ml blood.
- As you withdraw the needle, apply the gauze swab to the site, maintaining firm manual pressure over for at least 2 minutes
- Dispose of the needle and apply a vented cap, expelling any excess air.
  - (This may not be necessary depending on your equipment.)

### Procedure: brachial artery

- Position the elbow in extension. Angle the needle 60°.

### Procedure: femoral artery

- Position the patient with hip extended.
- The pulse is felt 2cm below the midpoint between pubic tubercle and anterior superior iliac spine.
- Angle the needle at 90° to the skin.
- Pressure must be applied for at least 5 minutes.

### Procedure tips

- Before you start: know where the analyser is and how to use it!
- The key is carefully palpating the artery and lining the needle up to puncture it. Take your time!
- The majority of the pain comes from puncturing the skin. If no flashback is seen immediately, try repositioning the needle by withdrawing slightly without removing it from the skin.
- If there will be some delay in analysing the sample, store the blood-filled syringe on ice.
- Errors occur: if there is air in the syringe, if the sample is delayed in reaching the analyser (if this is anticipated, put the sample on ice), or if a venous sample is accidentally obtained.

### Documentation

- Date, time, indication, consent obtained.
- Record how much (if any) supplemental oxygen the patient is on.
- Artery punctured.
- Modified Allen's test?
- How many passes?
- Any immediate complications.
- Signature, printed name, contact details.

# Peripheral venous cannulation

### Contraindications
- Cannulae should not be placed unless intravenous access is required.
- Caution in patients with a bleeding diathesis.

### Risks
- Infection, which could be local or systemic.

### Before you start
- Can the drug be given by another route?
- What is the smallest appropriate cannula? (Table 18.1)
- What is the most appropriate location for the cannula? (Box 18.7)

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**Box 18.7  Choosing a vein**
- Avoid areas of skin damage, erythema, or an arm with an AV fistula
- Excessive hair should be cut with scissors before cleaning the skin
- It is best to avoid joint areas such as the antecubital fossa
  - This can cause kinking of the cannula and discomfort
  - A straight vein, in an area such as the forearm or dorsum of the hand where long bones are available to splint the cannula are usually best.
- Wide-bore access requires siting in large veins and often this is only practicable in the antecubital fossa
- In practice, especially in patients who have been cannulated many times before, it is often necessary to go wherever you find a vein.

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### Sizing cannulae
- Cannulae are colour-coded according to size. The 'gauge' is inversely proportional to the external diameter.
- The standard size cannula 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.

Table 18.1  Cannula sizes

| 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   |

**Equipment**
- Gloves.
- Sterile wipe (e.g. chlorhexidine).
- Cannula of appropriate gauge.
- Sterile saline for injection ('flush') and a 5ml syringe.
- Cannula dressing.
- Cotton wool balls/gauze.
- Tourniquet.

**Procedure**
- Introduce yourself, confirm the patient's identity, explain the procedure, and obtain informed consent
- Put the gloves on.
- Apply the tourniquet proximally on the limb.
- Once the veins are distended, select an appropriate vein: it should be straight for the length of the cannula.
- Wipe with sterile wipe, beginning where you intend to insert the cannula and moving outwards in circles.
- Fill the syringe with saline and eject any air bubbles.
- Remove the dressing from its packaging.
- Unwrap the cannula and check that all parts disengage easily. Fold the wings down so that they will lie flat on the skin after insertion.
- Using your non-dominant hand, pull the skin taut over the vein in order to anchor it in place.
- Hold the cannula with index and middle fingers in front of the cannula wings, thumb behind the cap.
- Warn the patient to expect a 'sharp scratch'.
- Insert the needle, bevel up, at an angle of 30° to the skin, until a flashback of blood is visible within the chamber of the cannula.
- Advance the needle a small amount further, then advance the cannula into the vein over the needle, whilst keeping the needle stationary.
- Release the tourniquet.
- Place your non-dominant thumb over the tip of the cannula, compressing the vein.
- Flush the cannula with a little saline from the end and replace the cap.
- Write the date on the cannula dressing and secure in place.

**Procedure tips**
- Local anaesthetic cream may be of benefit if you have time.
- Reliable veins are located on the radial aspect of the wrist (cephalic vein), antecubital fossa, and anterior to the medial malleolus (long saphenous).
- If you fail initially with a large-bore cannula, try a smaller gauge.
- If no veins are visible/palpable at first, try warming the limb in warm water for a couple of minutes.
- It may be useful to get assistance to hold the patient's arm still if they are likely to move it during the procedure.
- If you are unable to cannulate after several attempts, try asking someone else. A pair of fresh eyes make a lot of difference!

# Femoral venous catheter insertion

### Contraindications
- Fem-fem bypass surgery, IVC filter, infected site, thrombosed vein.

### Risks
- Arterial puncture, infection, haematoma, thrombosis, air embolism, arteriovenous fistula, peritoneal puncture.

### Equipment
- Central line catheter pack.
  - Containing: central line (16–20cm length, multi-lumen if required), introducer needle, 10ml syringe, guidewire, dilator, blade.
- Large dressing pack including a large sterile drape and gauze.
- Normal saline.
- Local anaesthetic for skin (1% lidocaine).
- Sterile preparation solution (2% chlorhexidine).
- Securing device or stitch.
- Sterile gloves, sterile gown, surgical hat and mask.
- Suitable dressing.

### Procedure
- Introduce yourself, confirm the patient's identity, explain the procedure, and obtain written consent if possible.
- Position the patient supine (1 pillow), abduct the leg and place a spill sheet under the patient's leg.
- Identify the entry point: 1–2 cm below the mid-inguinal point and 1cm medial to femoral artery.
- Wearing a surgical hat and mask, wash hands using a surgical scrub technique and put on the sterile gown and gloves.
- Set up a trolley using an aseptic technique:
  - Open the dressing pack onto the trolley creating a sterile field
  - Open the central line catheter pack and place onto the sterile field
  - Flush all lumens of the catheter with saline and clamp the ends
  - Ensure the guidewire is ready for insertion
  - Attach the introducer needle to a 10ml syringe.
- Clean the area with sterile solution and surround with a large drape.
- Inject local anaesthetic into the skin over the entry point.
- Identify the femoral artery with your non-dominant hand.
- Pierce the skin through the entry point with the introducer needle.
- Direct the needle at a 30–45° angle to the skin and aim for the ipsilateral nipple, aspirating as you advance the needle.
- ▶ On hitting the vein the syringe will fill with blood.
- Keeping the needle still, carefully remove the syringe—blood should ooze (and not pulsate) out through the hub of the needle.
- Insert the guidewire part-way through the hub of the needle.
  - Guidewires are over 50cm in length; do not insert more than 20cm.
- Remove the needle over the guidewire ensuring one hand is always holding either the proximal or distal end of the wire.

- Thread the dilator over the wire, firmly pushing it through the skin.
  - This may require a small stab incision in the skin with a blade
  - Aim to get 2–3cm of dilator into the vein, not its full length.
- Check the guidewire has not been kinked by ensuring it moves freely through the dilator.
- Remove the dilator and apply pressure over with gauze to stop oozing.
- Thread the catheter over the guidewire until it emerges through the end of the distal port (unclamp this lumen!).
- Holding the guidewire at its port exit site with one hand, push the catheter through the skin with the other.
- Remove the guidewire.
- Blood should flow out of the end of the catheter.
- Aspirate and flush all ports.
- Fix catheter to skin using either a securing device or stitches.
- Cover with transparent dressing.

**Procedure tips**

- Placing a sandbag underneath the patient's buttock may improve positioning (if a sandbag is not available, roll up a towel or wrap a 1-litre bag of fluid in a sheet as an alternative).
- Do not force the guidewire. If there is resistance to insertion:
  - Reduce the angle of the needle, attempt a shallower insertion
  - Check you are still within the vein by aspirating with a syringe
  - Rotate the needle: this moves the bevel away from any obstruction.
- ▶ Losing the guidewire can be disastrous—always have one hand holding either the proximal or distal end of it.
- Always consider the possibility of an inadvertent arterial puncture:
  - Signs include pulsatile blood flow, high-pressure blood flow or blood bright red in colour (in the absence of hypotension or hypoxaemia)
  - Do not dilate if in any doubt
  - The use of saline in the aspirating syringe may make flushing the needle easier but also makes it more difficult to differentiate between venous and arterial blood.

**Documentation**

- Time, date, indication, informed consent obtained.
- Site and side of successful insertion.
- Site, side, and complications of unsuccessful attempt(s).
- Aseptic technique: gloves, gown, hat, mask, sterile solution.
- Local anaesthetic: type and amount infiltrated.
- Technique used: e.g. landmark, ultrasound guidance.
- Catheter used: e.g. triple lumen.
- Length of catheter *in situ* (length at skin).
- Signature, printed name, and contact details.

# Central venous access: internal jugular vein

🛈 This is the 'landmark' technique for the internal jugular vein.

## Contraindications
- Infected insertion site.
- Thrombosed vein.
- Coagulopathy.

## Risks
- Pneumothorax.
- Arterial puncture.
- Haematoma.
- Air embolism.
- Arrhythmias.
- Thrombosis.
- Arteriovenous fistula.
- Infection.
- Malposition.

## Equipment
- Central line catheter pack:
  - Central line (16cm length for right side, 20cm for left side), introducer needle and 10ml syringe, guidewire, dilator, blade.
- Large dressing pack including a large sterile drape and gauze.
- Normal saline.
- Local anaesthetic for skin (1% lidocaine) with suitable (22G) needle and syringe.
- Sterile preparation solution (2% chlorhexidine).
- Sterile gloves, sterile gown, surgical hat and mask.
- Trolley and ECG monitoring.

## Procedure
- Introduce yourself, confirm the patient's identity, explain the procedure, and obtain written consent if possible.
- Position the patient supine (1 pillow), tilt the bed head down and place a spill sheet under the patient's head.
- Attach ECG monitoring to the patient.
- Turn the patient's head away from the side of insertion.
- Identify triangle formed by the sternal and clavicular heads of the sternocleidomastoid muscle and the clavicle.
- Identify the entry point at the apex of the triangle.
- Wash hands using a surgical scrub technique and put on the sterile gown and gloves.
- With assistance, set up a trolley using an aseptic technique:
  - Open the dressing pack onto the trolley creating a sterile field
  - Open the central line catheter pack and place onto the sterile field
  - Flush all lumens of the catheter with saline and clamp the ends
  - Attach the introducer needle to a 10ml syringe.

- Clean the area with sterile preparation solution and place a large drape around it.
- Inject local anaesthetic into the skin over the entry point.
- Identify the carotid artery with your non-dominant hand. Pierce the skin through the entry point with the introducer needle ensuring the needle is lateral to the artery.
- Direct the needle at a 30° angle to the skin and advance using continuous aspiration, aiming for the *ipsilateral* nipple.
- On hitting the vein, the syringe will fill with blood.
- Keeping the needle still, carefully remove the syringe.
  • Blood should ooze (not pulsate) through the hub of the needle.
- Insert the guidewire through the needle and watch the ECG.
  • ⚠ Guidewires tend to be over 50cm in length but do not introduce more than 20cm as this may lead to arrhythmias.
- Remove the needle over the guidewire ensuring one hand is always holding either the proximal or distal end of the wire.
- Thread the dilator over the wire, firmly pushing it through the skin.
  • This may require a small stab incision in the skin with a blade
  • Aim to get 2–3cm of dilator into the vein, not its full length
- Check the guidewire has not been kinked by ensuring it moves freely through the dilator.
- Remove the dilator over the guidewire and apply pressure over the site with gauze.
- Thread the catheter over the guidewire until it emerges through the end of the distal port (unclamp this lumen!).
  • This may require withdrawing some of the guidewire.
- Holding the guidewire at its port exit site with one hand, push the catheter through the skin with the other.
- ▶ Avoid handling the catheter, in particular its tip.
  • Insert 16cm for a right-sided line and 20cm for a left-sided line.
- Remove the guidewire.
  • Blood should flow out through the end of the catheter.
- Aspirate and flush all ports with normal saline.
- Fix catheter to skin with a fixing device or sutures.
- Cover with a transparent dressing.
- Request a chest radiograph to confirm position.

### Documentation

- Time, date, indication, and informed consent obtained.
- Site and side of successful insertion.
- Site, side, and complications of unsuccessful attempt(s).
- Aseptic technique: gloves, gown, hat, mask, type of sterile solution.
- Local anaesthetic: type and amount infiltrated.
- Technique used: e.g. landmark, ultrasound guidance.
- Catheter used: length and number of lumens.
- Aspirated and flushed.
- Length of catheter *in situ* (length at skin).
- Chest radiograph: site of tip, absence/presence of pneumothorax.
- Signature, printed name, and contact details.

**Procedure tips**
- The right internal jugular vein is usually favoured due to its relatively straight course and the absence of the thoracic duct on this side (Fig. 18.1).
- Tilting the bed head down will minimize the risk of air embolism and help distend the veins of the neck.

*Getting started*
- Asking the patient to sniff or lift their head off the bed will help identify the sternocleidomastoid muscle.
- Asking the patient to perform the Valsalva manoeuvre will distend the veins of the neck and help identify the internal jugular vein.
- For added safety, you may wish to start by using a 21G ('green') hypodermic needle instead of the introducer needle to 'seek' out the vessel using the same technique.
- Check clotting prior to insertion. Aim for INR <1.5 and platelets >50x10$^9$/L.
- Minimize spillage.

*During the procedure*
- ▶ The internal jugular vein is relatively superficial and should be encountered within 2–3cm. Do not continue advancing the needle if the vein has not been hit by this point.
- ▶ Do not force the guidewire in. If there is resistance to guidewire insertion:
  - Try lowering the angle of the needle making it more in line with the long axis of the vessel
  - Check you are still within the vein by aspirating with a syringe
  - Try rotating the needle thereby moving the bevel away from any obstruction.
- ▶ Losing the guidewire can be disastrous. Always have one hand holding either the proximal or distal end of it.
- The use of saline in the aspirating syringe may make flushing the needle easier but also makes it more difficult to differentiate between venous and arterial blood.
- 🚺 Always consider the possibility of an inadvertent arterial puncture (Box 18.8):
  - Signs include pulsatile blood flow, high-pressure blood flow, or blood bright red in colour (in the absence of hypotension or hypoxaemia)
  - Do not dilate if in any doubt
  - Consider sending blood for a blood gas to confirm venous placement.

*Finishing off*
- There is an increased incidence of vascular injuries and thrombosis with left-sided catheters mainly because of insufficient catheter depth leading to the tip abutting the lateral wall of the upper SVC. You must ensure left-sided lines are long enough so that their tip lies within the lower part of the SVC.

- On the chest radiograph, confirm catheter position and the absence of a pneumothorax.
- The tip of the catheter should lie at the junction of the superior vena cava and right atrium which is approximately at the level of the carina.

*Alternative approaches*
- Anterior approach: midpoint of sternal head of sternocleidomastoid aiming towards ipsilateral nipple.
- Posterior approach: posterior border sternocleidomastoid at the crossing of the external jugular vein aiming for the sternal notch.
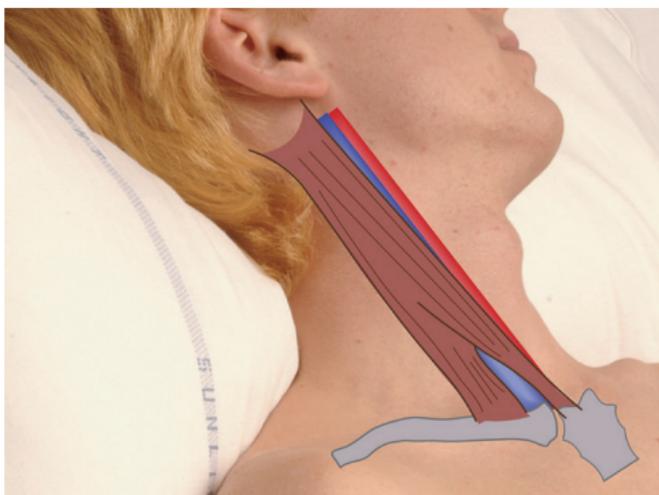


**Fig. 18.1** Surface anatomy of the internal jugular vein.

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**Box 18.8 Structures your needle may hit**

*In front of the vein*
- Internal carotid artery

*Behind the vein*
- Transverse process of the cervical vertebrae
- Sympathetic chain
- Phrenic nerve
- Dome of pleura
- Thoracic duct on left-hand side.

*Medial to vein*
- Internal carotid artery
- Cranial nerves IX–XII
- Common carotid and vagus nerve.

# Central venous access: subclavian vein

🛈 This is the 'landmark' technique for the right subclavian vein (Fig. 18.2).

## Contraindications
- Hyperinflated lungs (e.g. COPD patients).
- Coagulopathy.
- Infected insertion site.
- Thrombosed vein.

## Risks
- Pneumothorax.
- Haemorrhage.
- Arterial puncture.
- Air embolism.
- Arrhythmias.
- Thrombosis.
- Arteriovenous fistula.
- Infection.
- Malposition.

## Equipment
- Central line catheter pack:
  - Central line (16cm length for right side, 20cm for left side), introducer needle and 10ml syringe, guidewire, dilator, blade.
- Large dressing pack including a large sterile drape and gauze.
- Normal saline.
- Local anaesthetic for skin (1% lidocaine) with (22G) needle and syringe.
- Sterile preparation solution (2% chlorhexidine).
- Sterile gloves, sterile gown, surgical hat and mask.
- Trolley and ECG monitoring.

## Procedure
- Introduce yourself, confirm the patient's identity, explain the procedure, and obtain written consent if possible.
- Position the patient supine (1 pillow), place a sandbag between shoulder blades and tilt the bed head down.
- Attach ECG leads onto the patient making sure they are not in the surgical field.
- Turn the patient's head away from the side of insertion.
- Identify the entry point, just inferior to the midpoint of the clavicle.
- Wash hands using a surgical scrub technique and put on the sterile gown and gloves.
- With assistance, set up a trolley using an aseptic technique:
  - Open the dressing pack onto the trolley creating a sterile field
  - Open the central line catheter pack and place onto the sterile field
  - Flush all lumens of the catheter with saline and clamp the ends
  - Ensure the guidewire is ready for insertion
  - Attach the introducer needle to a 10ml syringe.

- Clean the area with sterile preparation solution and place a large drape around it.
- Inject local anaesthetic into the skin over the entry point.
- Insert the introducer needle under the clavicle at a very shallow angle almost parallel to the floor.
- Advance the needle towards the sternal notch, aspirating as you advance.
- On hitting the vein the syringe will fill with blood.
- Keeping the needle still, carefully remove the syringe.
  • Blood should ooze (not pulsate) through the hub of the needle.
- Insert the guidewire through the needle and watch the ECG.
  • 🛇 Guidewires tend to be over 50cm in length but do not introduce more than 20cm as this may lead to arrhythmias.
- Remove the needle over the guidewire ensuring one hand is always holding either the proximal or distal end of the wire.
- Thread the dilator over the wire, firmly pushing it through the skin.
  • This may require a small stab incision in the skin with a blade
  • Aim to get 2–3cm of dilator into the vein, not its full length.
- Check the guidewire has not been kinked by ensuring it moves freely through the dilator.
- Remove the dilator over the guidewire and apply gauze to the site to mop up any spills.
- Thread the catheter over the guidewire until it emerges through the end of the distal port (unclamp this lumen!).
  • This may require withdrawing some of the guidewire.
- Holding the guidewire at its port exit site with one hand, push the catheter through the skin with the other.
- ▶ Avoid handling the catheter, in particular its tip.
  • Insert 16cm for a right-sided line and 20cm for a left-sided line
- Remove the guidewire.
  • Blood should flow out through the end of the catheter.
- Aspirate and flush all ports with normal saline.
- Fix catheter to skin with a fixing device or sutures.
- Cover with a transparent dressing.
- Request a chest radiograph to confirm catheter position and the absence of a pneumothorax.

### Documentation

- Time, date, indication, and informed consent obtained.
- Site and side of successful insertion.
- Site, side, and complications of unsuccessful attempt(s).
- Aseptic technique: gloves, gown, hat, mask, type of sterile solution.
- Local anaesthetic: type and amount infiltrated.
- Technique used: e.g. landmark, ultrasound guidance.
- Catheter used: length and number of lumens.
- Aspirated and flushed.
- Length of catheter *in situ* (length at skin).
- CXR: site of tip, absence/presence of a pneumothorax.
- Signature, printed name, and contact details.

**Procedure tips**

*Getting started*
- Check clotting prior to insertion. Aim for INR <1.5, platelets >50x10⁹/L.
- 🛈 Direct pressure cannot be applied on the subclavian vessels so this route should be avoided in patients with a coagulopathy
- 🛈 There is a greater risk of pneumothorax than with internal jugular cannulation. A subclavian approach should, therefore, be avoided in patients with hyperinflated lungs.
- Minimize spillage.
- The underside of the clavicle can be reached by first directing the needle onto the clavicle and then carefully walking off it. The angle of the needle should however remain parallel to the floor.
- Asking an assistant to pull the ipsilateral arm caudally can improve access.
- If a sandbag is not available, roll up a towel or wrap a 1-litre bag of fluid in a spill sheet as an alternative.

*During the procedure*
- ▶ The subclavian vein should be encountered within 3–4cm. Do not continue advancing the needle if the vein has not been hit by this point.
- ▶ Do not force the guidewire in. If there is resistance to guidewire insertion:
  - Try lowering the angle of the needle making it more in line with the length of the vessel
  - Check you are still within the vein by aspirating with a syringe
  - Try rotating the needle thereby moving the bevel away from any obstruction.
- Catheter malposition, particularly into the ipsilateral internal jugular vein, is more common using the subclavian vein approach.
  - Many guidewires have a 'J' tip. Directing the 'J' tip caudally may help correct placement.
- ▶ Losing the guidewire can be disastrous. Always have one hand holding either the proximal or distal end of it.
- 🛈 Always consider the possibility of an inadvertent arterial puncture (Box 18.9):
  - Signs include pulsatile blood flow, high pressure blood flow or blood bright red in colour (in the absence of hypotension or hypoxaemia)
  - Do not dilate if in any doubt.
- The use of saline in the aspirating syringe may make flushing the needle easier but also makes it more difficult to differentiate between venous and arterial blood.

*Finishing off*
- The incidence of vascular injuries and thrombosis is increased with left-sided catheters mainly due to insufficient catheter depth leading to the tip abutting the lateral wall of the upper SVC.
  - You must ensure left-sided lines are long enough so that their tip lies within the lower part of the SVC.

- On the chest radiograph, confirm catheter position and the absence of a pneumothorax.
  - The tip of the catheter should lie at the junction of the superior vena cava and right atrium which is approximately at the level of the carina.

*Alternative approaches*
- Medial approach: junction of medial and middle thirds of the clavicle.
- Lateral approach: lateral to the mid-clavicular point. Often used with ultrasound guidance.
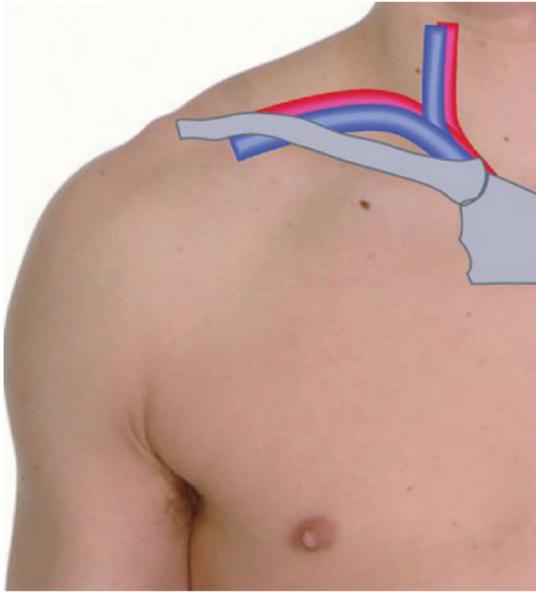


**Fig. 18.2** Surface anatomy of the right subclavian vein.

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**Box 18.9 Structures your needle may hit**

*In front of the vein*
- Clavicle
- Subclavius muscle.

*Behind the vein*
- Phrenic nerve
- Anterior scalene muscle
- Subclavian artery.

*Below vein*
- First rib
- Pleura.

# Central venous access: ultrasound guidance

▶ Current recommendations in the UK are that ultrasound guidance should be considered when inserting any central venous catheter (NICE guidelines 2002).

## Ultrasound basics

- 'Ultrasound' refers to sound waves of such a high frequency as to be inaudible to the human ear (>20 kHz).
- Medical ultrasound uses frequencies between 2 and 14 MHz.
- The 'linear' (straight) transducer is the probe of choice for imaging the vessels and other superficial structures.
- The frequency of the probe should be between 7.5–10 MHz for central venous access.

*Basic controls*
- Frequency.
  - Higher frequency may result in a better resolution but will not penetrate the tissues as deeply.
- Gain.
  - The gain control alters the amplification of the returned signals
  - This changes the grey scale of the image (can be thought of as increasing the brightness) but may not improve its quality.
- Depth.
  - The depth of the image on screen can be manually adjusted
  - It is wise to see the structures deep to the vessel to be cannulated.
- Focal length.
  - The focal point is usually displayed as an arrow at the side of the image
  - At this point, the image will be sharpest but resolution of the deeper structures will suffer
  - The focal point should be positioned in line with the vein to be cannulated.

*Orientation*
- By convention, the left of the screen should be that part of the patient to your left (i.e. the patient's right if you are facing the patient, the patient's left if you are scanning from behind them).
  - Touch edge of the probe and watch for the movement on screen to be sure you have the transducer the right way round.

**Procedure: internal jugular vein catheterization**

- With the patient positioned, squeeze sterile gel onto the patient's neck.
- Hold the probe cover open like a sock. Ask an assistant to squeeze ultrasound gel into the base and *carefully* lower the probe in after it. You can then unfurl the probe cover along the length of the wire using aseptic technique.
- Place probe over the surface markings of the vein (short axis of vessel).
- On the screen, look for two black circles side by side. These represent the vein and the artery.
- Identify the vessels by pressing down with the probe.
  - The vein will be compressible and the artery will not
  - The artery will be pulsatile. 🛈 Note that the IJV may also be pulsatile with the patient head down (the JVP)
  - The artery is often circular in cross-section, the vein may be oval or a more complex ovoid shape.
- Follow the course of the vein up the patient's neck and identify a site where the artery sits relatively medial to the vein. At this point, centre the vein on to the screen holding the probe still with your non-dominant hand.
- 🛈 Don't press too hard with the probe—you may compress the vein.
- Inject local anaesthetic into the skin around the midpoint of the probe using your dominant hand.
- Insert the introducer needle through the skin at the midpoint of the probe.
- Gently move the needle in and out to help locate the tip and its course on the screen.
  - 🛈 The tip of the needle will only be visualized if it is advancing in the same plane as the ultrasound beam.
- Advance the needle (with continuous aspiration) towards the vein ensuring the tip is always in view.
- On hitting the vein, blood will be aspirated into the syringe. Flatten the needle ensuring blood can still be aspirated. At this point, the probe can be removed and the vein can be catheterized using the Seldinger technique (see previous pages).
- The ultrasound can be used later in the procedure to ensure that the guidewire lies within the vein, if necessary.

# Intravenous infusions

### Equipment
- Gloves.
- An appropriate fluid bag.
- Giving set.
- Drip stand.
- 10ml syringe with saline flush.

### Procedure
- 🕛 Intravenous infusions require intravenous access.
- 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.
- 🕛 Flush the patient's cannula with a few millilitres of saline to ensure there is no obstruction. If there is evidence of a blockage, swelling at the cannula site, or if the patient experiences pain, you may need to replace the cannula.
- 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.
- Using quite a lot of force, push the giving set end into the bag outlet.
- Invert the bag and hang on a suitable drip-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 (Box 18.10).

### Documentation
- Ensure fluid and/or the drug is clearly timed and signed for as per local policy.
- Nursing and/or medical notes should be completed to include the reason for the infusion.
- Medical notes should be used to record any causes for concern arising from administration of the infusion.
- Cannula site (and cannula documentation) should be dated and signed on insertion.
- Ensure any fluid-monitoring chart is complete and updated as appropriate.
- Ensure that all entries in notes finish with your signature, printed name, and contact details.

**Box 18.10 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 healthcare 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 in Table 18.2.

Table **18.2** Drip rate

| 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 | 250 | 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 |

# Arterial line insertion

The following is the procedure for cannulating the radial artery.

## Contraindications
- Infection at insertion site.
- Working arterio-venous fistula in the same limb.
- Traumatic injury proximal to the insertion site.
- Vascular insufficiency in the distribution of the artery to be cannulated.
- Significant clotting abnormalities.

## Risks
- *Non-vascular:* superficial bleeding, infection, inadvertent arterial injection.
- *Vascular:* vasospasm, thrombosis, thromboembolism, air embolism, blood vessel injury, distal ischaemia.

## Equipment
- Arterial catheter set:
  - Arterial catheter (20G), needle, guidewire
- Sterile gloves, sterile gown (+/− surgical hat and mask).
- Dressing pack including a sterile drape.
- Sterile preparation solution (e.g. 2% chlorhexidine).
- Local anaesthetic (e.g. 1% lidocaine), 22G needle, and 5ml syringe.
- (Optional) A three-way tap with a short extension (flushed with normal saline) connected to a 10ml syringe containing normal saline.
- Suture.
- Transducer set with pressurized bag of heparinized saline.

## Procedure (modified Seldinger technique)
- Introduce yourself, confirm the patient's identity, explain the procedure, and obtain informed consent.
- Choose a site for arterial line insertion.
- Position the forearm so that it is supported from underneath and hyperextend the wrist.
- Set up a trolley keeping everything sterile:
  - Open the dressing pack onto the trolley creating a sterile field
  - Open the arterial catheter set and place onto the sterile field.
- Wash hands using a surgical scrub technique and put on the sterile gown and gloves.
- Clean the wrist, hand, and forearm with a sterile preparation solution and create a sterile field with the drape.
- Palpate the radial artery with your non-dominant hand and infiltrate the skin overlying the pulsation with some local anaesthetic.
- Insert the arterial needle, directing it towards the radial pulsation at a 30–45° angle. (Do not attach to a syringe.)
  - You can also use a syringe with the plunger removed. This allows identification of the arterial pulsation without excess spillage.
- On hitting the artery, blood will spurt out of the hub of the needle.

- Keeping the needle still, insert the guidewire through the hub of the needle. 🟢 Don't force the guidewire.
- Remove the needle leaving the guidewire in place.
- Thread the arterial catheter over the guidewire making sure that the guidewire is seen at all times through the distal end of the catheter.
- Holding the distal end of the guidewire with one hand, push the arterial catheter through the skin with the other.
- Remove the guidewire.
  - Blood should spill out of the end of the catheter if it is within the artery.
- Connect to the short extension of the three-way tap, aspirate and flush with normal saline, and close off the tap.
  - Alternatively, connect immediately to a pressurized transducer set, aspirate and flush
  - 🟢 Do not delay connection to transducer and flush-bag
  - 🟢 Take extreme care not to allow any air bubbles to flush into the artery (risk of distal embolization).
- Suture in place.
- Label catheter as arterial and inform relevant staff.

## Documentation

- Time, date, indication, and informed consent obtained.
- Site and side of successful insertion.
- Site, side, and complications of unsuccessful attempt(s).
- Aseptic technique: gloves, gown, hat, mask, sterile solution.
- Local anaesthetic: type and amount infiltrated.
- Technique used: modified Seldinger, cannula over needle.
- Catheter size used: 20G.
- Aspirated and flushed.
- Signature, printed name, and contact details.

## Procedure tips

- 🟢 Do not force the guidewire. If there is resistance, try lowering the needle to a shallower angle without removing it from the artery.
- 🟢 Cover the floor with spill sheets as the procedure can be messy!
- ▶ The modified Allen's test should be used for assessment of the collateral supply to the hand before the radial artery is punctured but may not be completely reliable in predicting ischaemic injury.

*Modified Allen's test*

- Compress the radial and ulnar arteries at the wrist and ask the patient to clench their fist.
- Ask the patient to open the hand.
- Release pressure over the ulnar artery.
- Watch the palm for return of colour.
  - Return of colour should normally occur in 5–10 seconds.
- ▶ Return of colour taking over 15 seconds suggests an inadequate collateral supply by the ulnar artery and radial artery cannulation should not be performed.

# Fine needle aspiration (FNA)

A method for obtaining a cytological sample of a mass lesion. This procedure should only be performed by, or under strict supervision of, an experienced practitioner.

▶ Fine needle aspiration usually takes place in the radiology department and is performed by an experienced radiologist under ultrasound or CT guidance. The following describes the older, 'blind' technique.

## Contraindications
- Bleeding diathesis.
- Overlying infection.
- 🛈 Adjacent vital structures.
  - Image-guidance should always be used if available.

## Risks
- Bleeding.
- Local infection.
- Damage to surrounding structures depending on site e.g. blood vessels, nerves.

## Equipment
- Local anaesthetic (e.g. 1% lidocaine).
- Small-gauge (blue) needle and 10ml syringe.
- Sterile pack.
- Cleaning solution (e.g. chlorhexidine).
- Medium-gauge (green) needle.
- 10 or 20ml syringe for aspiration.
- Sterile gloves.

## Procedure
- Introduce yourself, confirm the patient's identity, explain the procedure, and obtain informed consent.
- Position the patient according to the biopsy site, allowing easy palpation of the mass.
- Expose appropriately.
- Wash your hands and put on sterile gloves.
- Clean the area with the cleaning solution and apply drapes.
- Instil local anaesthetic to the skin and subcutaneous tissues, withdrawing the plunger prior to each injection to avoid intravenous injection and warning the patient to expect a 'sharp scratch'.
- Immobilize the mass with your non-dominant hand.
- Using your dominant hand, insert the needle through the skin into the lump, maintaining negative pressure on the plunger as you go.
- Once in the lump, the needle may be moved gently back and forth to obtain a greater volume of cells.
- It may be necessary to insert the needle several times to obtain a sufficient sample.
- Do not expect a large amount of material within the syringe! A tiny sample within the needle will usually suffice.

- Remove the needle and send the sample for cytology (you will need to gently expel the sample from the needle into a suitable container).
- Apply a sterile dressing to the site.

### Alternative method

- There are two schools of thought in fine needle aspiration.
- Some practitioners use a small (blue) needle without a syringe attached.
  - This is moved in and out very quickly within the mass whilst also applying rotation
  - Capillary action deposits a cellular sample within the needle which can then be gently expelled using an empty syringe.
- This capillary action technique may result in a larger number of intact cells in the resultant sample as the negative pressure created when using a syringe can disrupt cell membranes.

### Documentation

- Date and time.
- Indication, informed consent obtained.
- Type and amount of local anaesthetic used.
- Site of puncture.
- Aseptic technique used?
- How many passes?
- Volume and colour of sample obtained.
- Any immediate complications.
- Tests requested on resultant sample.
- Signature, printed name, and contact details.

### Procedure tips

- ⚠ Radiological guidance should always be used if available.
- Contact the histopathology department in advance to ensure appropriate transport medium is used.
  - It may be possible to arrange immediate analysis, allowing diagnosis and repeat FNA if insufficient cells are obtained.

# Lumbar puncture

### Contraindications
- Infected skin or subcutis at the site of puncture.
- Coagulopathy or thrombocytopenia.
- Raised intracranial pressure with a differential pressure between the supra- and infra-tentorial compartments such as seen in space-occupying lesions. If in doubt, image first!

### Risks
- Post-procedure headache.
- Infection.
- Haemorrhage (epidural, subdural, subarachnoid).
- Dysaesthesia of the lower limbs.
- Cerebral herniation (always check local procedures regarding contraindication to LP and whether to perform CT head first).

### Equipment
- Sterile gloves.
- Sterile pack (containing drape, cotton balls, small bowl).
- Antiseptic solution (e.g. iodine).
- Sterile gauze dressing.
- 1 x 25G (orange) needle.
- 1 x 21G (green) needle.
- Spinal needle (usually 22G).
- Lumbar-puncture manometer.
- 3-way tap (may be included in a lumbar puncture 'kit').
- 5–10ml 1% lidocaine.
- 2 x 10ml syringes.
- 3 x sterile collection tubes and one biochemistry tube for glucose measurement.

### 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.
  - Ensure that the patient can hold this position comfortably.
- Place a pillow between the patient's knees to prevent the pelvis tilting.
- Label the collection tubes '1', '2', and '3'.
- 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 at this point with a pen.
- Wash hands and put on the sterile gloves.
- Unwrap all equipment and ensure it fits together correctly.
  - It is usually useful to give the 3-way tap a few twists as it can stick.
- 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.
- Wait for ~1 minute for the anaesthetic to take effect.
- Introduce the spinal needle through the marked site at about 90° to the skin, heading slightly toward the umbilicus.
  - Keep the bevel facing cranially.
- Gently advance the needle to ~5 cm depth.
- A further slight push of the needle should produce a 'give' as the needle enters the subarachnoid space (this takes a little practice to feel).
- 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).
- Turn the tap such that the CSF within the manometer pours out in a controlled manner and further CSF can drip freely.
- Collect about 5 or 6 drops into each collection tube *in the order in which they have been labelled*.
- Collect a few more drops into the biochemistry tube for glucose measurement.
- Close the tap so that the manometer will measure the pressure at the end of the collection ('closing pressure').
- Remove the needle, tap, and manometer in one action.
- 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 (biochemistry tube), protein (bottle 2).
- Advise the patient to lie flat for ~1 hour and ask nursing staff to check CNS observations (see local guidelines).

## Documentation
- Date, time, indication, and informed consent obtained.
- Vertebral level needle inserted.
- Number of passes before CSF obtained.
- Initial ('opening') pressure and final ('closing') pressure.
- Amount and appearance of CSF.
- Tests samples sent for.
- Any immediate complications.
- Signature, printed name, and contact details.

## Procedure tips
- Always use the smallest gauge spinal needle available.
  - In some centres, 'pencil-point' needles are used which are associated with a much reduced incidence of post-procedure headache.
- If the needle strikes bone and cannot be advanced, withdraw slightly, re-angle, and advance in a stepwise fashion until the gap is found.
- Lumbar puncture can be performed with the patient sitting, leaning forwards. This is particularly useful if the patient is obese. However, pressure measurements will be erroneous if taken in this position.

# Male urethral catheterization

### Contraindications
- Urethral/prostatic injury.

### Risks
- Urinary tract infection.
- Septicaemia.
- Pain.
- Haematuria.
- Creation of a 'false passage' through prostate.
- Urethral trauma.
- 🛑 Beware latex allergy.

### Equipment
- Foley catheter (male) of appropriate French, usually 12–14 gauge.
- 10 ml syringe of sterile water.
- Syringe of lidocaine gel 1% (e.g. Instilligel®).
- Catheter bag.
- Sterile gloves.
- Catheter pack containing drape, kidney dish, swabs/cotton balls, and a small dish.
- Sterile water/chlorhexidine sachet.

### Procedure
- Introduce yourself, confirm the patient's identity, explain the procedure, and obtain informed consent.
- Position the patient lying supine with the external genitalia uncovered.
  - Uncover from umbilicus to knees.
- Using aseptic technique, unwrap the equipment and pour the chlorhexidine or sterile water into the dish.
- Wash your hands and put on the sterile gloves.
- Tear a hole in the middle of the drape and place it over the genitals so as to allow access to the penis.
- Use your non-dominant hand to hold the penis upright.
- Withdraw the foreskin and clean around the urethral meatus using the water/chlorhexidine and a swab, moving from the centre outwards.
- Instil local anaesthetic via the urethral meatus, with the penis held vertically.
- Wait at least one minute for the anaesthetic to act.
- Place the kidney bowl between the patient's thighs.
- Remove the tip of the plastic sheath containing the catheter, being careful not to touch the catheter itself.
- Insert catheter into urethra, feeding it out of the plastic wrapper as it is advanced.
- Insert the catheter to the 'hilt'.
  - If the catheter will not advance fully, don't force it. Withdraw a little, extend the penis fully, and carefully try again.

- At this point, urine may begin to drain.
  - Let the hub end of the catheter rest in the kidney bowl to catch the inevitable spills.
- Inflate the balloon using sterile water inserted into the catheter side-arm according to the balloon's capacity (written on the cuff of the balloon lumen).
  - ▶ Watch the patient's face and ask them to warn you if they feel pain.
- Once the balloon is inflated, remove the syringe and attach the catheter bag.
- Gently pull the catheter until you feel resistance as the balloon rests against the bladder neck.
- Replace the foreskin (this is essential to prevent paraphimosis).
- Re-dress the patient appropriately.

## Documentation

- Date and time.
- Indication, informed consent obtained.
- Size of catheter inserted.
- Aseptic technique used?
- Volume of water used to inflate the balloon.
- Residual volume of urine obtained.
- Foreskin replaced?
- Any immediate complications.
- Signature, printed name, and contact details.

## Procedure tips

- Difficulty passing an enlarged prostate is a common problem. Tricks to try to ease the catheter past include:
  - Ensure the catheter is adequately lubricated
  - Try moving the penis to a horizontal position between the patient's legs as prostatic resistance is reached
  - Ask the patient to wiggle his toes
  - Rotate the catheter back and forth as it advances
  - If catheter fails to pass, consider using larger bore catheter (e.g. 16F instead of 14F) as this may prevent coiling in the urethra.
- If urine fails to drain despite the catheter being fully advanced:
  - Palpate the bladder: if palpable, the catheter is inappropriately placed
  - Manual pressure on the bladder may express enough urine from a near-empty bladder to show itself
  - Aspirate with a bladder syringe, or flush with a little sterile saline.
- ▶ If it is impossible to pass the catheter, ask for help.
  - If all else fails, it may be necessary to proceed to suprapubic catheterization.

# Female urethral catheterization

### Contraindications
- Urethral injury.

### Risks
- Urinary tract infection.
- Septicaemia.
- Pain.
- Haematuria.
- Urethral trauma.
- 🚯 Beware latex allergy.

### Equipment
- Foley catheter (female) of appropriate French, usually 12–14 gauge.
- 10 ml syringe of sterile water.
- Syringe of lidocaine gel 1% (e.g. Instilligel®).
- Catheter bag.
- Sterile gloves.
- Catheter pack: drape, kidney dish, swabs/cotton balls, and a small dish.
- Sterile water/chlorhexidine sachet.

### Procedure
- Introduce yourself, confirm the patient's identity, explain the procedure, and obtain informed consent.
- Position the patient with hips externally rotated and knees flexed. Uncover from waist down.
- Using aseptic technique, unwrap the equipment and pour the chlorhexidine or sterile water into the dish.
- Wash your hands and put on the sterile gloves.
- Tear a hole in the middle of the drape and place it over the genitals so as to allow access.
- Use your non-dominant hand to part the labia.
- Clean around the urethral meatus using the water/chlorhexidine and a swab, moving from the centre outwards.
- Instil local anaesthetic via urethral meatus.
  - Wait at least one minute for the anaesthetic to act.
- Place the kidney bowl between the patient's thighs.
- Remove the tip of the plastic sheath containing the catheter, being careful not to touch the catheter itself.
- Insert catheter into urethra, feeding it out of the plastic wrapper as it is advanced.
- Insert the catheter to the 'hilt'.
- At this point, urine may begin to drain. Let the end of the catheter rest in the kidney bowl to catch any spills.
- Inflate the balloon using sterile water inserted into the catheter side-arm according to the balloon's capacity (written on the cuff of the balloon lumen).
- ▶ Watch the patient's face and ask them to warn you if they feel pain.

- Once the balloon is inflated, remove the syringe and attach the catheter bag.
- Gently pull the catheter until you feel resistance as the balloon rests against the bladder neck.
- Re-dress the patient appropriately.

## Documentation

- Date and time.
- Indication, informed consent obtained.
- Size of catheter inserted.
- Aseptic technique used?
- Volume of water used to inflate the balloon.
- Residual volume of urine obtained.
- Any immediate complications.
- Signature, printed name, and contact details.

## Procedure tips

- Difficulty passing the catheter may be alleviated by slowly rotating the catheter whilst inserting.
- Difficulty seeing the urethral meatus may be overcome by asking the patient to 'bear down'.
- If urine fails to drain despite the catheter being fully advanced:
  • Palpate the bladder: if palpable, the catheter is inappropriately placed
  • Manual pressure on the bladder may express enough urine from a near-empty bladder to show itself
  • Aspirate with a bladder syringe, or flush with a little sterile saline.
- ▶ If it is impossible to pass the catheter, ask for help
  • If all else fails, it may be necessary to proceed to suprapubic catheterization.

# Basic airway management

### Airway manoeuvres

The following manoeuvres are performed with the patient lying supine and the attender positioned above the head. The aim is to prevent the flaccid tongue from falling back and causing the epiglottis or tongue itself from occluding the airway (Box 18.11 and Fig.18.3). These can be performed with no equipment.

*Before you start*
- Get help!
  - A patient with an obstructed airway can rarely be adequately treated by one individual, even if appropriate kit is within reach.

*Head tilt*
- Place your hands on the forehead and tilt the head backwards, extending the neck.

*Chin lift*
- Place two fingertips below the mental protuberance of the mandible, with thumb in front.
- Draw the mandible anteriorly.

---

**Box 18.11  The head tilt/chin lift**
- Head tilt and chin lift are usually performed together
- ▶ Head tilt and chin lift are not suitable if there is any suspicion of cervical spinal injury
- Jaw thrust alone should be used in this situation.

---

*Jaw thrust*
- Place your fingertips behind the angle of the mandible.
- The base of the thenar eminence of each hand should be rested on the cheek bones.
- Use your fingers to pull the mandible anteriorly, whilst using your thumbs to open the mouth.
- If performed with a mask, the thenar eminence may be used to maintain a good seal.

### Procedure tips
- After each manoeuvre, check for success.
- It is worth-while practising these skills on resuscitation dummies prior to having to do them in real life!
- Use the above manoeuvres in conjunction with face masks or bag–valve mask ventilation.

(a)

(b)

(c)
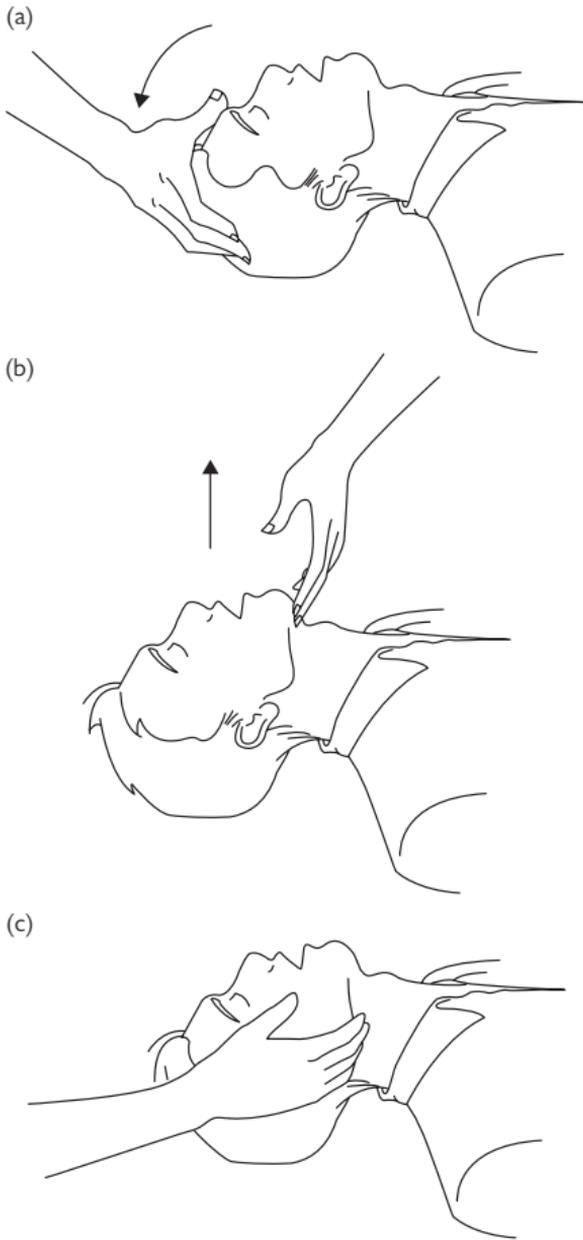


**Fig. 18.3** Airway manoeuvres. (a) Head tilt. (b) Chin lift. (c) Jaw thrust.

## Oropharyngeal (Guedel) airway

- A stiff tube with a fixed curvature is inserted through the mouth. A flange limits the depth of insertion.
- 🛈 Use when the patient is semi-conscious.

*Indications*

- Airway compromise in the patient with reduced conscious level.

*Contraindications*

- Active gag reflex.
- Conscious patient.

*Procedure*

- Insert the airway initially with the curvature upwards.
- Once inside the mouth, rotate 180°.
- Continue to insert, following the curvature of the tongue until the flange rests against the teeth or gums.
- Ensure there is no gagging, snoring, or vomiting and that air can move in and out freely.

*Procedure tips*

- May be used for suction (size 10, 12, or 14 catheters).
- Insertion can be guided with a tongue depressor.

*Airway sizes*

- Oropharyngeal airways come in many sizes and are colour-coded for convenience.
- ▶ Select the correct size of airway for the patient by measuring it against the side of the patient's face. The flange should sit at the corner of the patient's mouth and the tip at the angle of the jaw (Fig. 18.4).
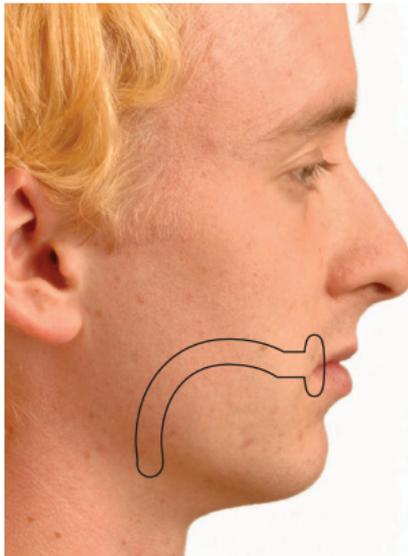


**Fig. 18.4** Choose the correct size of oropharyngeal airway by measuring from the corner of the patient's mouth to the angle of the jaw.

## Nasopharyngeal airway

- Tolerated better than a Guedel airway in semi-conscious patients.
- Consists of a soft plastic tube with flanged end.
- The pharyngeal end has a bevel and the body is curved to facilitate insertion.
  - Some designs have a small flange and a safety pin is often used to ensure the device does not migrate fully into the patient's nose.

*Indications*

- Patients with reduced conscious level and/or airway compromise who will not tolerate an oropharyngeal airway (intact gag reflex).

*Contraindications*

- Known basal skull fracture (relative contraindication).

*Procedure*

- Lubricate the device.
- Insert bevelled end into the wider nostril.
- Pass the tube along the floor of the nasal airway.
- Aim no higher than the back of the opposite eyeball.
- Use size 10 or 12 catheter for suction if required.
- Advance until the flange is flush against the nostril.

*Procedure tips*

- If insertion proves difficult, try the opposite nostril.

*Airway sizes*

- Nasopharyngeal airways come in several sizes, the size is usually stamped on the side.
- Determine the correct size by comparing those available with the pateint's little finger and the distance between the nostril and the tragus (Fig. 18.5).
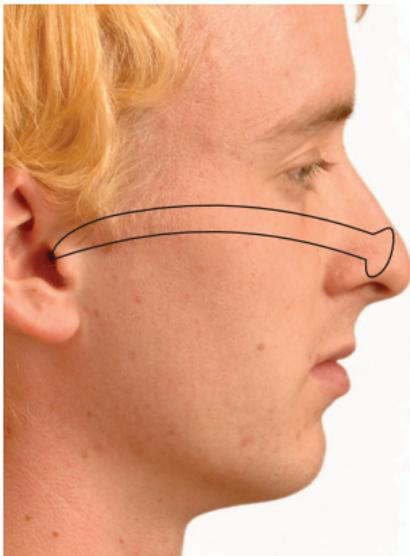


**Fig. 18.5** Choose the correct size of nasopharyngeal airway by measuring from the nostril to the tragus.

### Laryngeal mask airway (LMA)

- A tube with an inflatable cuff ('mask') around its base to create a seal around the laryngeal inlet.
- ▶ This does not prevent aspiration of stomach contents.

*Indications*

- Unconscious patient requiring ventilation.

*Contraindications*

- Conscious patient (absolute).
- Maxillofacial trauma.
- Risk of aspiration.
- >16 weeks' pregnant.

*Procedure*

- Ensure that the cuff inflates and deflates satisfactorily.
- For insertion, the mask should be completely deflated.
- Deflate the cuff with a 20ml syringe. Lubricate the outer cuff with aqueous gel.
- Gently extend the head and flex the neck (except in possible cervical trauma).
- Hold the LMA tubing near the cuff, like a pen.
- With the mask facing down, pass along the under-surface of the palate until it reaches the posterior pharynx.
- Guide the tube backwards and downwards (using an index finger if necessary) until resistance is felt.
- Remove your hand and fill the mask with the required amount of air (usually 20–30ml).
  - The tube should lift out of the mouth slightly and the larynx is pushed forward if it is in the correct position.
- Connect the bag-valve mask and ventilate.
- Auscultate in both axillary regions to confirm ventilation.
- Insert a bite block/Guedel airway next to the tube in case the patient bites down.
- Secure in place with tape/ribbon.

*Procedure tips*

- If inadequately deflated, lubricated, or not pressed against the hard palate on insertion, the LMA may fold back on itself making insertion difficult or preventing appropriate positioning of the mask (Fig. 18.6).
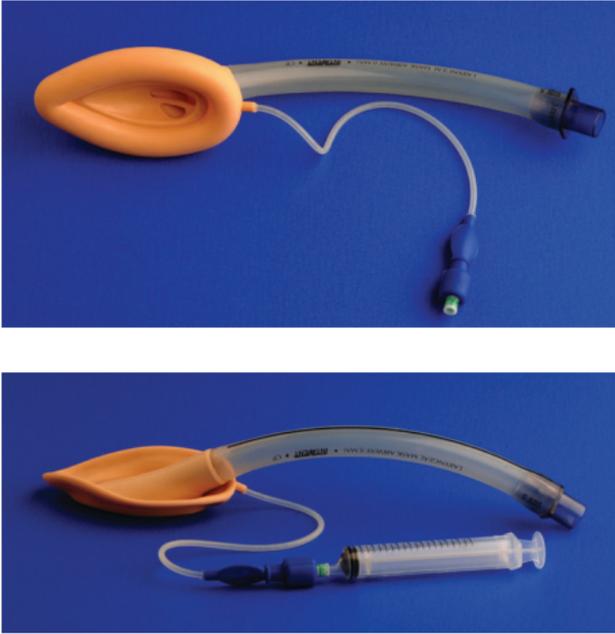
**Fig. 18.6** Laryngeal mask airways. (a) Inflated. (b) Deflated.

# Oxygen administration

▶ Oxygen is a drug with a correct dosage and side effects which when administered correctly may be life saving.

## Oxygen prescribing

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.

## Procedure

- Explain what is happening to the patient and ask their permission.
- Choose an appropriate oxygen delivery device.
- Choose an initial dose:
  - Cardiac or respiratory arrest: 100%
  - Hypoxaemia with $PaCO_2$ < 5.3kPa: 40–60%
  - Hypoxaemia with $PaCO_2$ > 5.3kPa: 24% initially.
- If possible, try to measure a $PaO_2$ in room air prior to giving supplementary oxygen.
- Apply the oxygen and monitor via oximetry ($SaO_2$) and/or repeat ABGs ($PaO_2$) in 30 minutes.
- If hypoxaemia continues, the patient may require respiratory support.

## 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. (Fig.18.7).
- Each oxygen delivery device comprises an oxygen supply, flow rate, tubing, interface ± humidification.

*Nasal cannulae*
- These direct oxygen via two short prongs up the nasal passage.
  - 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*
- Deliver oxygen concentrations that vary depending on the patient's minute volume. At low flow rates there may be some rebreathing of exhaled gases (they are not sufficiently expelled from the mask).

*Fixed performance masks*
- A constant $O_2$ concentration independent of the 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.
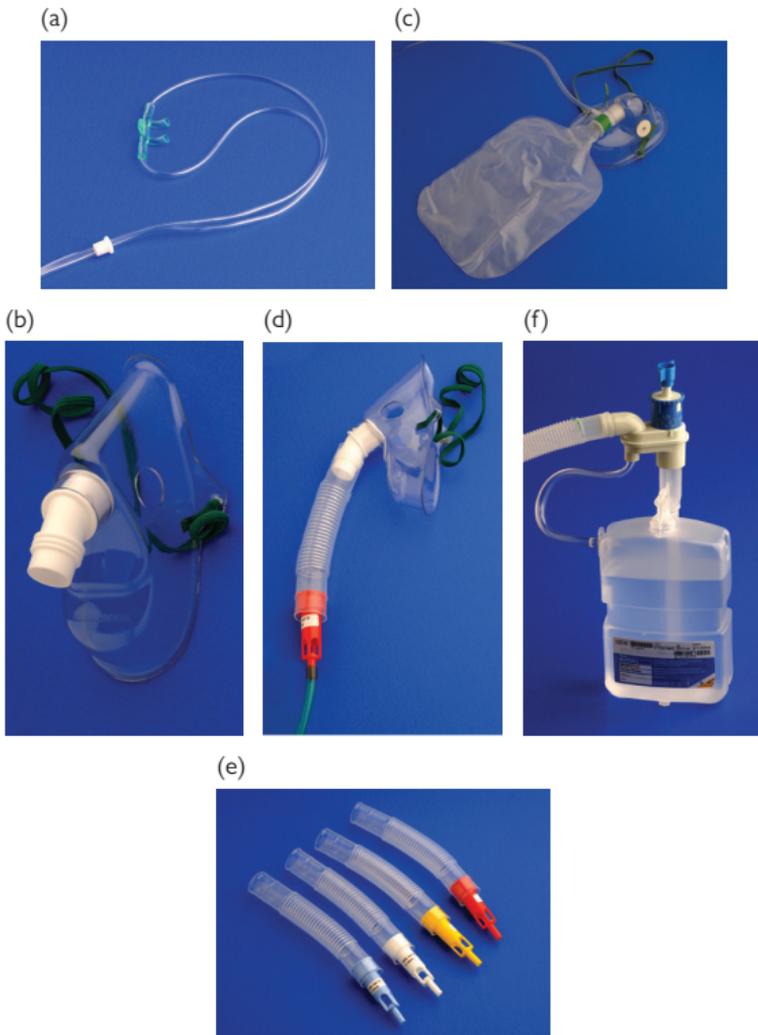


**Fig. 18.7** (a) Nasal cannulae. (b) Low flow/variable concentration mask. (c) Non-rebreathe mask. (d) Mask with Venturi valve attached. (e) Selection of Venturi valves. (f) Humidification circuit.

# Peak expiratory flow rate (PEFR) measurement

### Background
- Normal values vary according to height, age, and gender (Fig.18.8).
- The value obtained may be compared against this and/or the patient's previous best PEFR.

### Indications
- Asthma. Either in an acute attack to assess severity, or during the chronic phase to determine reversibility in response to treatment (>60L/min change defined as reversible).
  - PEFR may also aid in the diagnosis of asthma by examining the greatest variation over two weeks.
- PEFR may also be useful in assessment of COPD, particularly the degree of reversibility in response to inhaled bronchodilator.

### Contraindications
- Any features of life-threatening asthma or severe respiratory distress.

### Equipment
- A peak flow meter.
- A clean disposable mouthpiece.

### Procedure
- Introduce yourself, confirm the patient's identity, 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.
  - ▶ Ensure that the patient holds the device at the sides, avoiding obstructing the marker with a finger.
- The patient should blow out as *hard* and as *fast* as possible.
  - Patients sometimes have difficulty with this and a quick demonstration or advice to 'imagine blowing out a candle at the other end of the room' can help.
- Make a note of the reading achieved.
- Repeat the procedure and record the best of three efforts.
- If the patient is to keep a record, be sure to explain how to record the readings appropriately. (Sometimes a two-week diary is kept by the patient to assess for diurnal variation.)

### Procedure tips
- If the patient is having difficulty performing correctly, a brief demonstration often proves very useful.
- If the highest two values are not within 40L/min, further values should be obtained.
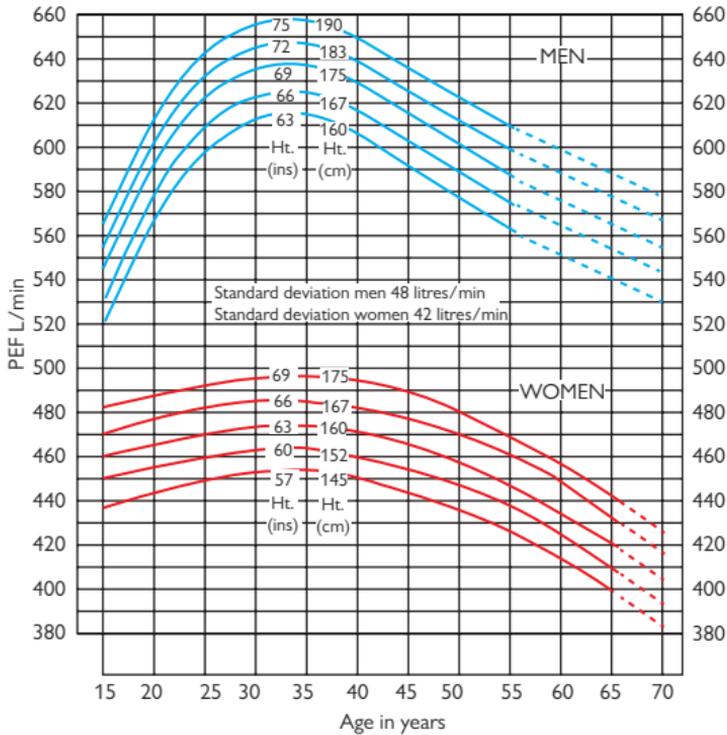
**Fig. 18.8** Normal PEFR by age and gender. From BMJ 1989; 298: 1068–70.

### Documentation

- Record the highest PEFR in L/min and as a percentage of the patient's best previous or predicted PEFR.
- Make a note of the time and whether the measurement was made before or after therapy.

# Inhaler technique

### Metered dose inhaler

- Requires coordination to use effectively and lacks a dose counter.
- May be unsuitable for the very young, elderly, or those with arthritis affecting the hands. (Fig.18.9.)

*How to use*

- Take only one dose at a time.
- Remove the cap and shake the inhaler several times.
- Sit upright, breathe out completely.
- Insert mouthpiece in mouth, sealing with lips.
- Take a deep breath in. Just after you begin to breathe in depress the canister whilst continuing to inhale.
  - The canister should be pressed *just after* the start of inhalation, not before.
- Inhale slowly and deeply.
- Remove inhaler and hold your breath for 10 seconds or as long as is comfortable.
- Recover before taking the next dose and repeat above as necessary.
- Replace cap.



**Fig. 18.9** A metered dose inhaler (MDI). A salbutamol inhaler is pictured.

## Autohaler

- This is a 'breath-actuated' inhaler, releasing a dose automatically as a breath is taken (Fig.18.10).
- No hand coordination is required.
- The priming lever, however, can prove difficult to use and requires priming before each dose.

*How to use*

- Remove cap and shake inhaler several times.
- Prime by pushing the lever into the vertical position whilst keeping the inhaler upright.
- Sit upright, breathe out completely, and insert mouthpiece, sealing with lips.
- Inhale slowly and deeply.
  - Don't stop when the inhaler clicks.
- Remove inhaler and hold your breath for 10 seconds or as long as is comfortable.
- Push lever down and allow time to recover before taking the next dose.
- Once doses are taken, replace cap.



**Fig. 18.10** A typical Autohaler.

*Procedure tips*

- Patients unable to operate the lever by hand may be able to use a hard surface such as the edge of a table for assistance.
- Use inhaler only for the number of doses written on the label.
- Patients should inhale slowly and steadily rather than hard and fast.

### Easi-breathe

- Breath-actuated inhaler, as autohaler only primed by opening the cap hence this must be closed and opened again between successive doses (Fig.18.11).

*How to use*

- Shake the inhaler several times.
- Hold upright and prime by opening the cap.
- Sit upright, breathe out completely, and insert mouthpiece, sealing with lips.
  - Make sure that your fingers are not covering the air holes at the top.
- Inhale slowly and deeply.
  - ❗ Don't stop when the inhaler puffs.
- Remove inhaler and hold your breath for 10 seconds or as long as is comfortable.
- Close the cap, with the inhaler upright.
- Recover before taking the next dose.



**Fig. 18.11** A typical Easi-breathe inhaler.

*Procedure tips*

- It is essential to close and then open the cap between successive doses. This primes the inhaler.
- Advise the patient not to dismantle the inhaler. Patients used to using MDIs may be tempted to take the top off and attempt to depress the canister manually.

## Accuhaler

- Dry powder device, superseding the Diskhaler and Rotahaler (Fig.18.12).
- Has a dose counter.
- The several step priming mechanism may be difficult for some to manage.

*How to use*

- Hold the outer casing in one hand whilst pushing the thumb grip away, exposing the mouthpiece, until you hear a click.
- With the mouthpiece towards you, slide the lever away from you until it clicks. The device is now primed.
- Sit upright, breathe out completely, and insert mouthpiece, sealing with lips.
- Inhale quickly and deeply.
  - (In contrast to breath-actuated devices).
- Remove inhaler and hold your breath for 10 seconds or as long as is comfortable.
- To close, pull the thumb grip towards you, hiding the mouthpiece in the cover, until you hear a click.
- Recover before taking the next dose.



**Fig. 18.12** A typical Accuhaler.

*Procedure tips*

- The Accuhaler must be closed and re-primed between successive doses.
- The dose counter indicates how many doses are left.

## Turbohaler

- Dry-powder device with preloaded tasteless drug (Fig.18.13).
- 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.
- Those with impaired dexterity may find the inhaler difficult to use.

*How to use*
- Unscrew and remove the white cover.
- Hold the inhaler upright and prime the device by twisting the grip clockwise and anticlockwise as far as it will go (until you hear a click).
- Sit upright, breathe out completely, and insert mouthpiece, sealing with lips.
- Inhale slowly and deeply.
- Remove inhaler and hold your breath for 10 seconds or as long as is comfortable.
- Recover before taking the next dose.
- The device must be primed again between successive doses.



**Fig. 18.13** A typical Turbohaler.

*Procedure tips*
- Advise the patient that they will not feel the dose hit the back of their throat.
- Patients used to an MDI may find this off-putting.

**Clickhaler**
- Disposable dry-powder inhaler with dose meter which turns red when only 10 doses are left to use.
- The inhaler locks when empty so patients can be sure that they have taken a dose.

*How to use*
- Take only one dose at a time.
- Remove the cap and shake.
- Whilst holding inhaler upright, depress the button firmly and release until you hear a click.
- Sit upright, breathe out completely, and insert mouthpiece, sealing with lips.
- Inhale deeply.
- Remove inhaler and hold your breath for 10 seconds or as long as is comfortable.
- Recover before taking the next dose and repeat above as necessary.
- Replace cap.

**Handihaler**
- A dry-powder device with an integrated cap.
- This requires a lower inspiratory flow rate than other devices.
- A dose needs to be inserted via a capsule at each use requiring some dexterity.
- Patients may also find the cap rather hard to open as it requires a moderate amount of strength.

*How to use*
- Open cap by pulling upwards exposing mouthpiece.
- Open the mouthpiece by pulling upwards exposing the dose chamber.
- Take a capsule from the blister-pack and insert it into the chamber.
- Replace the mouthpiece (it should click shut).
- 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.
- Remove inhaler and hold breath for as long as is comfortable.
- Remove the used capsule and replace the cap.

# Non-invasive ventilation

▶ Non-invasive ventilation should only be set up by experienced operators. The following is a guide only.

## Background
- CPAP = continuous positive airways pressure.
  - CPAP traditionally has its own equipment and 'set-up'
  - Recently more clinicians are delivering CPAP through the BiPAP Vision®. There is also a 'low flow' version used mainly for transport of CPAP dependent patients.
- BiPAP = bilevel positive airways pressure.

## Contraindications/cautions
- ▶ Undrained pneumothorax. (Absolute contraindication).
- Facial fractures.
- Life-threatening epistaxis.
- Bullous pulmonary disease.
- Proximal lung tumours (air trapping).
- Active TB (spread).
- Acute head injury.
- Low blood pressure.
- Uncontrolled cardiac arrhythmias.
- Sinus/middle ear infection.

## Risks
- Abdominal distension (secondary to 'swallowing' air).
- Decreased cardiac output (drop in BP).
- Pressure sores from mask.
- Aspiration of vomit.
- $CO_2$ retention if patient breathing small tidal volume against high PEEP.

## Documentation
- Oxygen prescription charts.
- Ventilation prescription charts.
- Clear record of ABGs with evidence of time, inspired oxygen, and ventilation levels.
- Good practice to document the 'ceiling' of pressures and $FiO_2$ for the clinical environment.

## CPAP equipment
- Mask (+/− T-piece), hood.
- Head strap (mask), shoulder straps (hood).
- Oxygen circuit and humidification.
- High flow generator (e.g. Whisper Flow®, Vital Signs®).
- PEEP valves (usually 5, 7.5, or 10cmH$_2$O).
- 'Blow off' safety valve (10cmH$_2$O above the PEEP used).

### CPAP procedure

- Use available templates to assess appropriate sized interface and minimize air leaks (if using the BiPAP Vision®).
- Decide on level of PEEP to apply.
- Attach PEEP valve to mask (if using traditional set-up, may need T-piece).
- Attach oxygen circuit with humidification including 'blow-off' valve (for safety).
- Set inspired oxygen level.
- Set flow rate to ensure the PEEP valve opens a small distance and never closes.
- Titrate oxygen and PEEP in response to the patient's work of breathing, saturations, pH, $PaO_2$, and $PaCO_2$.
- If appropriate, set alarms on ventilator (if using BiPAP Vision®).
- Write a prescription chart of PEEP or ventilation settings and acceptable saturations, $PaO_2$, and $PaCO_2$, continuous or intermittent.

### BiPAP equipment

- Interface (face mask, nasal pillows, nasal mask, etc.).
- Head straps.
- Ventilation circuit (exhalation port unless on mask).
- Humidification (if required).
- Ventilator (NIPPY 1/2/3/3+, BiPAP Vision®, etc.).
- Entrained oxygen (unless with ventilator, e.g. BiPAP Vision®).

### BiPAP procedure

- Decide on which interface to use.
- Use available templates to assess appropriate sized interface and minimize air leaks.
- Start with low pressures (EPAP 4cmH$_2$O, IPAP 12cmH$_2$O).
  - Slowly increase pressures to levels agreed by MDT, for patient comfort and in response to pH, $PaO_2$, and $PaCO_2$
  - The aim being to reduce RR and work of breathing, normalize ABGs (for the individual) using the minimal pressures possible.
- Set inspiratory and expiratory times to those of the patient.
- Continually reassess RR as this will change and therefore set times will have to change.
- Titrate oxygen and pressures in response to the patient's saturations, pH, $PaO_2$, and $PaCO_2$.
- If appropriate set alarms on ventilator.
- Write a prescription chart of ventilation settings and acceptable saturations, $PaO_2$, and $PaCO_2$.

# Pleural fluid aspiration

This describes the procedure for aspirating as much pleural fluid as possible. If only a small sample is required for diagnostic purposes, use a green needle and 20ml syringe and follow a similar method to that described under 'ascitic fluid sampling'. (See Box 18.12 for alternative method.)

▶ Fluid should be aspirated from a position 1–2 intercostal spaces below the highest level at which dullness is percussed.

## Contraindications

- Recurrent effusion (chest drain or pleurodesis should be considered).
- Empyema (requires intercostal drainage).
- Mesothelioma (tumour may spread down needle track).
- Bleeding diathesis.

## Risks

- Pain.
- Cough.
- Failure to resolve.
- Re-expansion pulmonary oedema.
- Pneumothorax.

## Equipment

- Sterile pack.
- Sterile gloves.
- Cleaning solution (e.g. chlorhexidine).
- Large-bore (green) cannula.
- 3-way tap.
- 50ml syringe.
- 5ml 1% lidocaine.
- 23G (blue) needle.
- 2 x 10ml syringe.
- Dressing/gauze.
- Selection of sterile containers and blood bottles.
- Heparinized (ABG) syringe.

## Procedure

- Introduce yourself, confirm the patient's identity, explain the procedure, and obtain informed consent.
- Position the patient leaning forward with arms rested on a table or over the back of a chair.
- Percuss the effusion and choose a suitable spot for needle insertion.
- Clean the area with chlorhexidine.
- Using the blue needle and syringe, infiltrate local anaesthetic down to the pleura.
  - 🛈 Insert needle *just above* a rib to avoid the neurovascular bundle
  - Be sure to pull back on the syringe each time before injecting to ensure you are not in a blood vessel
  - Once fluid is withdrawn, you have reached the pleura.

- Insert the cannula perpendicular to the chest wall, aspirating with another syringe as you advance until resistance reduces and pleural fluid is aspirated.
- Remove the needle and attach the 3-way tap.
- You may now aspirate fluid using the 50ml syringe.
- Once the syringe is full, close the tap, disconnect the syringe, and empty into a container. Re-attach the syringe, open the tap, and repeat.
  - ❗ The pleural space should never be in continuity with the environment or pneumothorax will occur.
- Do not drain more than 2.5L at one time.
- Remove the cannula and apply the dressing.
- Send samples for:
  - Microbiology: microscopy, culture, Auramine stain, TB culture
  - Chemistry: protein, LDH, pH, glucose, amylase
  - Cytology
  - Immunology: ANA, rheumatoid factor, complement.
- Take simultaneous venous blood for glucose, protein, LDH.
- Request chest radiograph to confirm success and look for iatrogenic pneumothorax.

**Procedure tips**
- If unsuccessful, aspiration may be performed under ultrasound guidance: discuss with your radiology or respiratory department, depending on local policy.
- Passing a small fluid sample through a blood gas analyser may yield a rapid pH but should be avoided if the sample is purulent.

**Documentation**
- Date, time, indication, informed consent obtained.
- Aseptic technique used?
- Local anaesthetic used.
- Site needle inserted.
- Colour, consistency, and volume of fluid aspirated.
- Any immediate complications.
- Investigations requested.
- Signature, printed name, and contact details.

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**Box 18.12 An alternative method**
- An alternative method is to attach a fluid-giving set to one port of the 3-way tap and the 50ml syringe to the other
- With this set-up, you can aspirate 50ml into the syringe, turn the tap and empty it down the tubing into a container before turning the tap back to the syringe port
- The syringe, therefore, never needs to be disconnected and the risk of pneumothorax or other complication is reduced.

# Pneumothorax aspiration

### Simple vs secondary pneumothorax

*Simple pneumothorax*
- Aspiration is indicated if the rim of pleural air visible on chest radiograph is larger than 2cm or the patient is breathless.
- If initial aspiration is unsuccessful, repeat aspiration may be successful in >30% of cases and may avoid intercostal drain insertion.
- The total volume aspirated should not exceed 2.5L.

*Secondary pneumothorax*
- That is, a pneumothorax in the presence of underlying lung disease.
- Aspiration is only indicated in minimally symptomatic patients with small pneumothoraces (<2cm) aged <50.

### Contraindications
- Previous failed attempts at aspiration.
- Significant secondary pneumothorax.
- Traumatic pneumothorax.

### Risks
- Pain.
- Cough.
- Failure to resolve/recurrence.
- Re-expansion pulmonary oedema may theoretically occur if large volumes (>2.5L) are aspirated.

### Equipment
- Sterile pack.
- Sterile gloves.
- Cleaning solution (e.g. chlorhexidine).
- Large-bore (green) cannula.
- 3-way tap.
- 50ml syringe.
- 5ml 1% lidocaine.
- 23G (blue) needle.
- 2 x 10mL syringe.
- Dressing/gauze.

### Procedure
▶ Pneumothorax is usually aspirated from either 2nd intercostal space at the midclavicular line or the 4th–6th intercostal spaces at the midaxillary line.
- Introduce yourself, confirm the patient's identity, explain the procedure, and obtain informed consent.
- Position the patient leaning back comfortably at about 45°.
- Identify the site for needle insertion and double-check the radiograph to be certain you have the correct side. Confirm with clinical examination.
- Clean the area with the chlorhexidine.

- Infiltrate local anaesthetic down to the pleura using the blue needle and a 10ml syringe.
- Attach the other 10ml syringe to the cannula and insert the cannula perpendicular to the chest wall, aspirating as you advance until resistance reduces.
  - ▶ Insert the cannula *just above* a rib to avoid the neurovascular bundle.
- Remove the needle and quickly attach the 3-way tap and 50ml syringe.
- Aspirate with the syringe; close the 3-way tap when the syringe is full, remove the syringe and eject the air; reattach and open the 3-way tap to continue aspiration.
  - � The pleural space should never be in continuity with the environment (i.e. tap open with syringe detached) or pneumothorax will reaccumulate.
- Aspirate until resistance is felt, or up to a maximum of 2.5L.
- Remove the cannula and apply the dressing.
- Request chest radiograph to re-assess.

## Documentation
- Date, time, indication, informed consent obtained.
- Aseptic technique used?
- Local anaesthetic used.
- Site needle inserted.
- Volume of air aspirated.
- Any immediate complications.
- Investigations requested.
- Signature, printed name, and contact details.

## Tension pneumothorax
In the case of tension pneumothorax, a wide-bore cannula should be inserted into the 2nd intercostal space, midclavicular line, *without delay* and left open to convert the tension pneumothorax to a simple pneumothorax.

# Chest drain insertion (Seldinger)

- This describes the procedure for a Seldinger-type drain. Other drains are available.
- More and more trusts now recommend chest drain insertion under ultrasound guidance. Check your local policy and discuss with your radiology or respiratory departments as appropriate.

## Contraindications

- ▶ The need for an emergency thoracotomy. This should not be delayed for the insertion of a chest drain.
- Coagulopathy.
- Large bullae.
- Thoracic/pleural adhesions.
- Skin infection over the insertion site.

## Risks

- Inadequate placement.
- Bleeding (local or haemothorax).
- Liver or spleen injury +/− haemoperitoneum.
- Organ penetration (lung, liver, spleen, stomach, colon, heart).
- Infection.
- Iatrogenic pneumothorax.

## Equipment

- 10ml 1% lidocaine.
- 10ml syringe.
- 25G (orange) needle.
- 21G (green) needle.
- Sterile gloves.
- Sterile pack (containing cotton balls, drape, container).
- Seldinger chest drain kit.
  - Chest drain, introducer, needle, syringe, scalpel, 3-way tap, wire.
- Suture (e.g. 1.0 Mersilk).
- Cleaning solution (e.g. chlorhexidine or iodine).
- Chest drain tubing and drainage bottle.
- 500ml sterile water.
- Suitable dressing (e.g. Hypofix® or drainfix®).

## Procedure

- Introduce yourself, confirm the identity of the patient, explain the procedure, and obtain informed consent.
- ▶ Double-check radiograph and perform clinical examination to be sure of which side needs the drain.
- Position the patient sitting on a chair or the edge of their bed, arms raised and resting on bedside table with a pillow.
- 🛈 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 ('triangle of safety').
- Mark your spot (just *above* a rib to avoid the neurovascular bundle).

- Wash hands and put on sterile gloves.
- Clean the area with antiseptic solution on cotton wool balls working in a spiral pattern outwards.
- Using the 10ml syringe and orange needle, anaesthetize the skin forming a subcutaneous bleb.
- With the green needle anaesthetize down to the pleura, withdrawing the plunger before injecting each time.
- Use the scalpel to make a small cut in the skin.
- 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 area until you aspirate either air or fluid.
- Remove the syringe and hold the needle steady.
- Thread the guidewire through the needle into the chest.
  • Once the wire is half in the chest, discard the covering.
- Withdraw the needle from the chest but be sure to not remove the guidewire, keeping hold of it at all times, and thread the needle right off the end of the guidewire.
- Thread the introducer over the guidewire and into the chest, twisting back and forth as you go to open up a tract for the drain's passage. Then slide the introducer back off the wire, being careful not to pull the wire out of the chest.
- With the central stiffener in place, thread the drain over the wire and into the chest, curving downwards.
  • Keep hold of the guidewire at all times and do NOT push it into the chest cavity!
- Once the drain is in place, remove the wire and stiffener.
- Attach the 3-way tap, making sure all the ports are closed.
- Stitch the drain in place (unless using a drainfix®).
- Apply a drainfix® or other suitable dressing.
- Attach the drain to the tubing and the tubing to the 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 breaths and watch the water level in the tubing to see it rising and falling ('swinging').
- Request a post-insertion chest radiograph.

**Documentation**

- Date, time, indication, informed consent obtained.
- Aseptic technique used?
- Local anaesthetic used.
- Site drain inserted.
- Any immediate complications.
- Colour and consistency of fluid obtained.
- Investigations requested.
- Signature, printed name, and contact details.

# Recording a 12-lead ECG

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).
  - These should be attached to the 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.
- Position the stickers on the patient's body (Fig.18.14).
- The chest leads:
  - V1: 4$^{th}$ intercostal space at the right sternal border
  - V2: 4$^{th}$ intercostal space at the left sternal border
  - V3: midway between V2 and V4
  - V4: 5$^{th}$ intercostal space in the midclavicular line on the left
  - V5: left anterior axillary line, level with V4
  - V6: left mid-axillary line, level with V4.
- The limb leads are often colour-coded:
  - Red: Right arm (**R**ed: **R**ight)
  - Yellow: Left arm (Ye**LL**ow: **L**eft)
  - Green: right leg
  - Black: left leg.
- Attach the leads to the appropriate stickers.
- Turn on the ECG machine.
- Ask the patient to lie still and not speak for approximately 10 seconds whilst the machine records.
- Press the button to record, usually 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, date of birth as well as the date and time of the recording are clearly recorded on the trace.
- Remove the leads, discard the sticky electrode pads.
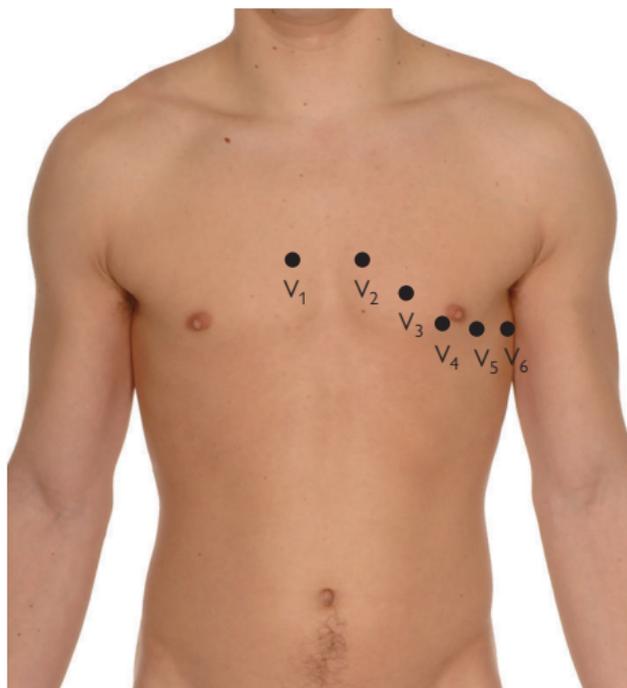
**Fig. 18.14** Correct positioning of the chest electrodes for a standard 12-lead ECG.

### Procedure tips
- Encourage the patient to relax otherwise muscle contraction will cause interference.
  - If unable to relax, or access to the peripheries is difficult, the 'arm' leads can be placed at the shoulders and the 'leg' leads at the groins.
- Breathing may cause a wandering baseline; breath holding for 6 seconds whilst recording may alleviate this.
- Ensure that you cleanse the area gently with an alcohol swab before attaching an electrode to ensure a good connection.
  - It may be necessary to cut chest hair to allow good contact and adhesion with the chest leads.
- The AC mains electricity may cause interference. If this is the case, try turning off nearby fluorescent lights.

# Carotid sinus massage

## Background

*Anatomy and physiology*

- The carotid sinus is located at the bifurcation of the common carotid artery.
  - It lies just under the angle of the jaw at the level of the thyroid cartilage.
- The carotid sinus contains numerous baroreceptors which coordinate homeostatic mechanisms responsible for maintaining blood pressure.
- These baroreceptors are innervated by a branch of the glossopharyngeal nerve (cranial nerve IX), which relays back to the medulla and modulates autonomic control of the heart and blood vessels.

*Carotid sinus hypersensitivity*

- The carotid sinus can be oversensitive to manual stimulation, a condition known as carotid sinus hypersensitivity (also 'carotid sinus syndrome' or 'carotid sinus syncope').
- In this condition, manual stimulation of the carotid sinus provokes significant changes in heart rate and/or blood pressure due to an exaggerated response to carotid sinus baroreceptor stimulation.
- This may result in marked bradycardia, vasodilation, and subsequent hypotension.
- The patient may complain of episodes of dizziness or syncope related to pressure on the neck (e.g. wearing a tight collar or turning the head quickly).
- The underlying mechanism behind this exaggerated response is not fully understood.

*Carotid sinus massage*

- Carotid sinus massage is a diagnostic technique used to confirm carotid sinus hypersensitivity and is sometimes useful for determining the underlying rhythm disturbance in supraventricular tachycardia (SVT).
- The procedure acts in a similar way to the Valsalva manoeuvre, increasing vagal tone and, therefore, reducing the heart rate.
- Carotid massage is less effective than pharmaceutical management of SVT (verapamil or adenosine) though is still the preferable choice in the young haemodynamically stable patient.
- ❗ This procedure should be performed with caution in the elderly as it may cause disruption of atheromatous plaque disease in the carotid artery and result in stroke.

**Before you start**
- Explain the procedure in full to the patient and obtain written consent.
  - If the test is to confirm carotid sinus hypersensitivity, then warn the patient that they may feel like they are going to faint but reassure them it is a controlled procedure
  - If the test is to determine the underlying rhythm in SVT, explain that they may feel a bit peculiar as the heart rate slows down transiently.
- Auscultate over the carotids for any bruits.
  - ⚠ If present the procedure will have to be abandoned as the risk of stroke is significant
- ► Document discussion of risks including failure, arrhythmias, stroke, faint, cardiac arrest.
- ► Secure intravenous access.
- ► Ensure that you have ECG monitoring with a recordable rhythm strip.
- ► Ensure access to full resuscitation equipment, including emergency drugs such as atropine and adrenaline.

**Procedure**
- Position the patient supine on a bed with the neck extended and head turned away from the side to be massaged.
- Whilst watching the ECG monitor (recording on a rhythm strip) gently massage the carotid sinus for 10 to 15 seconds using circular motions of your hand.
- If there is no response, switch to the opposite side.
- If successful (or 'positive' in the case of sinus hypersensitivity), the heart rate will slow.
  - This may allow you to determine the underlying rhythm in SVT.
- Ensure that the patient feels back to normal afterwards.

**Documentation**
- Date, time, indication, informed consent obtained.
- Intravenous access secured.
- ECG recording equipment operational.
- Emergency drugs on stand-by.
- Insert the rhythm strip into the patient's notes.
- Record details of what was seen on massage.
- Which carotid was used?
- Did the patient feel back to normal afterwards?
- Signature, printed name, and contact details.

# Vagal manoeuvres

## Background

*The purpose*

- Vagal manoeuvres can be used to determine the underlying rhythm or terminate supraventricular tachycardia (SVT) in haemodynamically stable patients.
- If the underlying rhythm is atrial flutter, slowing of the ventricular response by increasing vagal tone will reveal flutter waves.
- Vagal manoeuvres are part of the adult peri-arrest algorithm for management of narrow complex tachycardia. They can be performed in a controlled clinical situation (i.e. attached to an ECG machine), or taught to the patient to perform at home if the sensation of the arrhythmia recurs.

*Physiology*

- Vagal manoeuvres increase vagal tone by activation of the parasympathetic nervous system, conducted to the heart by the vagus nerve.
- Increasing vagal tone impedes the AV node and so slows transmission of the electrical impulse from the atria to the ventricle. In this way, any supraventricular tachycardia that relies upon the AV node will be modified by an increase in vagal tone.

## The Valsalva manoeuvre

- This is forced expiration against a closed glottis. Increasing intra-thoracic pressure stimulates baroreceptors in the aortic arch and results in increased vagal stimulation.
- This can be successful in 25–50% of cases.

*Procedure*

- Ask the patient to take a deep breath in and then 'bear down' as if they are trying to open their bowels (or for women—as if they are in labour).

Some patients may struggle with this concept and so alternatively:

- Give them a 10ml syringe and ask them to blow into the tip, in an attempt to expel the plunger.

## The diving reflex

- This involves either submerging the face in ice cold water (not very practical) or covering the face with a towel soaked in ice cold water.

## Carotid sinus massage

- This is described separately (see previously).

## Eyeball pressure

- 🛑 This is not recommended as a clinical procedure as it can be both painful and damaging. Do NOT perform.

# Temporary external pacing

🛈 This describes temporary transcutaneous pacing as an emergency.

### Before you start

- ▶ External pacing is usually performed in an emergency resuscitation situation following failure of response to initial management as per the bradycardia algorithm (see bradycardia algorithm from Resuscitation Council at ✇ www.resus.org.uk).
- ▶ A senior doctor should be present and make the decision to proceed with external transcutaneous pacing.
- ▶ There should be a plan in place for an experienced clinician to insert a temporary pacing wire within the next few hours. External pacing should only be a short-term management of decompensated bradycardia.
- ▶ There should also be a bed available for the patient on a high-dependency unit or coronary care unit so that they can be closely monitored by experienced nursing staff whilst waiting for a temporary pacing wire. 🛈 The patient should not be left on a general hospital ward.

### Indications

- Symptomatic bradycardia unresponsive to treatment (see bradycardia algorithm from Resuscitation Council at end of this topic).
- Mobitz type II block.
- Complete heart block.
- Heart block secondary to myocardial infarction.
- Profound bradycardia secondary to drug overdose e.g. beta blockers, digoxin.
- Asystole or ventricular standstill.

*Overdrive pacing*

- External pacing can be used to terminate certain tachyarrhythmias that are unresponsive to initial treatment e.g. polymorphic ventricular tachycardia (torsades de pointes) or refractory ventricular tachycardia.

### Risks

- Failure and progression to temporary pacing wire insertion.

### Equipment

- Full resuscitation equipment: defibrillator with pacing setting.
- Defibrillator pads.
- Oxygen.
- ECG monitoring.
- Emergency drugs (including atropine and adrenaline).
- Intravenous fluids.
- Sedative drugs (e.g. midazolam or diazepam).
- Analgesia (e.g. morphine).
- Intubation equipment (in case indicated).
- Senior support.

**Procedure**
- The patient should already have:
  - Large-bore intravenous access
  - Intravenous fluids running (unless in heart failure)
  - Oxygen via a non-rebreathe mask at 15L/min
  - ECG monitor connected and running
  - Interval BP monitoring.
- Place the pacing pads from the defibrillation kit on the patient's chest: one anteriorly in the V3 position and one posteriorly below the left scapula.
- Sedation and analgesia may be required.
- Attach the leads from the defibrillator to the pads.
- Switch the defibrillator to its pacing mode.

**Documentation**
Temporary external pacing is usually an emergency procedure so documentation may be delayed until the patient is stable. It should outline the resuscitation and external pacing simultaneously:
- Date and time.
- Name and grade of persons present.
- Events leading up to the need for external pacing.
- Any drugs used e.g. atropine or adrenaline, volume/dose, and response.
- Indication for external pacing.
- If patient was conscious, document consent (usually verbal consent only).
- Any sedation used.
- When external pacing commenced.
- Details of plans for temporary pacing wire insertion.
- Sign and bleep/contact details.

# DC cardioversion

### Indications
- Elective cardioversion of atrial fibrillation.
- Emergency cardioversion in a peri-arrest situation where a tachyarrhythmia is associated with adverse signs.

### Equipment
The 'crash trolley' should contain all the equipment required:
- Gloves, aprons, defibrillator, pads, leads, ECG electrodes.
- Oxygen, reservoir bag and mask with tubing, airways.
- Intubation equipment.
- Intravenous fluids, giving sets, selection of syringes, needles, intravenous cannulae, and fixation dressings.
- Access to emergency drugs (atropine, adrenaline, amiodarone).

### Contraindications
- Elective: patients unsuitable for general anaesthetic, not anticoagulated or who have not signed a consent form.
- Emergency: only performed when a tachyarrhythmia is associated with adverse events in the presence of a pulse (pulseless rhythms require management as per the resuscitation guidelines).

### Risks
- General anaesthetic risk, if performed electively.
- Embolic phenomenon, stroke, myocardial infarction.

### Before you start

*Elective procedure*
- Obtain informed consent and save a copy of signed form.
- Ensure patient fasted >6hrs.
- Check serum potassium (>4.0mmol/L gives greater success).
- Confirm patient anticoagulated for previous 4 weeks (INR >2).
  - Warfarin is continued for 3 months post-procedure if successful.
- The procedure should be performed in an anaesthetic room, following short-acting induction by an anaesthetist.

*Emergency procedure*
- Ensure a senior doctor is involved in the decision.
- Ensure all other options have been tried or considered.
- If possible discuss with the patient or next of kin.

### Energy selection
DC cardioversion usually uses biphasic energies. A reasonable guide is:
- 50 Joules synchronized shock. If fails ...
- 100 Joules synchronized shock. If fails ...
- 150 Joules synchronized shock. If fails ...
- 150 Joules synchronized anteroposterior shock. If fails ...
- ⚠ Abandon procedure if elective, consult seniors if emergency (may need ICU input).

## Procedure

- Ensure skin is dry, free of excess hair, jewellery is removed.
- Attach the ECG electrodes; red under right clavicle, yellow under left clavicle, green at the umbilicus.
- Switch on defibrillator and confirm the ECG rhythm.
- Place the defibrillator gel pads on the patient's chest; one under the right clavicle and the other inferolateral to the cardiac apex.
- ▶ Select the 'synchronous mode' on the defibrillator.
- ▶ Select the Joules required (see ➲ p.634).
- Place the paddles firmly on the chest on the gel pads.
- Press the charge button on the paddles to charge the defibrillator and shout 'Stand clear! Charging!'
- ▶ Check all persons are standing well clear of the patient and bed (including yourself) and that no-one is touching the patient or bed (including yourself).
- ▶ Ensure the oxygen has been disconnected and removed.
- ▶ Check the monitor again to ensure a shockable rhythm.
- Shout 'Stand clear! Shocking!'.
- Press both discharge buttons on the paddles to discharge the shock.
- Return the paddles to the defibrillator or keep them on the chest if another shock is required.

## Documentation

*General*

- Date, time, and place. Name and grade of persons present.
- ECG rhythm, intravenous access secured.
- Number, volume, dose of any drugs used, and any response noted.
- Type of defibrillator machine used.
- Method of sedation/anaesthetic.
- Asynchronous or synchronous mode. Specify Joules of each shock.
- Confirm rhythm at end and 12-lead ECG findings.
- Sign and bleep/contact details.

*Elective*

- Indication for DC cardioversion.
- Informed consent obtained (retain copy of signed form).
- State time fasted from.
- Document anticoagulation type and duration.
- Serum potassium level.
- Any drug allergies.
- Name and grade of anaesthetist, type of anaesthetic used.

*Emergency*

- Events leading up to the peri-arrest situation.
- HR, BP, GCS on arrival and any deterioration.
- Time of decision to shock, name and grade of decision-maker.
- Verbal consent obtained? Type of sedation used.
- Next of kin have been informed or if they are present or en route?

# Pericardiocentesis

### Contraindications
- Cardiac tamponade secondary to cardiac trauma or aortic dissection (surgical intervention is preferable).
- Recurrent pericardial effusions (surgical pericardial window indicated).

### Risks
- Pneumothorax.
- Myocardial perforation.
- Cardiac tamponade.
- Coronary artery laceration.
- Cardiac arrhythmias.
- Intra-abdominal trauma (especially to liver).
- Haemorrhage.
- Infection.
- Acute pulmonary oedema.
- Failure of procedure.
- Death.

### Equipment
- Echocardiogram machine and sterile probe cover.
- Pericardial drain kit (14 gauge needle, syringe, guidewire, pigtail catheter, and drain).
- Sterile drape and towels.
- Iodine solution.
- Sterile gloves and gown.
- Local anaesthetic (1% lidocaine).
- 2 x 10ml syringe.
- Orange/blue/green needles.
- Sterile gauze.
- 50ml syringe.
- Three-way tap.
- Suture, scissors, sticky dressing (e.g. Tegaderm®).

*You will also need:*
- Intravenous access.
- ECG monitoring.
- Access to 'crash' trolley (defibrillator and emergency drugs).

### Procedure
- Introduce yourself, explain procedure, and obtain informed written consent.
- Ensure IV access, ECG monitoring, normal clotting, and access to resuscitation equipment.
- (Consider light sedation).
- Position patient supine with 20–30° head tilt.
- Ensure all equipment is sterile and laid out on sterile trolley.
- Wash hands using surgical scrub technique and put on the sterile gown and gloves.

- Clean and drape site at the inferior border of the sternum.
  - Insertion point is below and to the left of the xiphisternum
- ► Confirm location of effusion using echocardiogram machine with sterile probe cover.
- Infiltrate overlying skin and subcutaneous tissue with 1% lidocaine. (Always aspirate before each injection).
- Attach the 10ml syringe attached to the 14G needle.
- Insert the needle between the xiphisternum and left costal margin advancing slowly at 35° to the patient and aiming towards the patient's left shoulder. Aspirate continuously as the needle advances.
  - Pericardial fluid is usually aspirated at about 6–8cm depth
  - Depending on the size of the pericardial effusion and indication for the procedure, you may wish to attach the 50ml syringe and aspirate fluid to send for diagnostic purposes.
- A modified Seldinger technique should be used to insert the drain.
- Once pericardial fluid is aspirated, hold the needle in position, remove the syringe, and insert the guidewire slowly through the needle into the pericardial space.
- Remove the needle, holding the wire in place at all times.
- Pass the catheter over the wire into the pericardial space.
- Once the catheter position is confirmed on echo, remove the wire and attach the three-way tap and drain bag.
- Suture the drain in place and dress to maintain sterility.
- Request a chest radiograph to exclude iatrogenic pneumothorax.

## Procedure tips

- ► Pericardiocentesis should be performed by a trained doctor (either cardiologist or thoracic surgeon usually) preferably in a sterile environment (theatre or the cardiac catheterization lab) and under echocardiographic guidance, with access to full resuscitation equipment.
  - ► The only exception is during cardiopulmonary resuscitation when pericardiocentesis is performed as an emergency to exclude cardiac tamponade as a reversible cause of cardiac arrest.
- ► Always check the patient's clotting beforehand.
- The clinician who performed the procedure should confirm the position of the drain using echo.
- Always request a post-procedure chest radiograph to exclude iatrogenic pneumothorax.

## Documentation

- Date, time, and place.
- Name and grade of person who performed the procedure (and anyone who supervised).
- Consent obtained (enclose copy of consent form).
- Aseptic technique used and volume of anaesthetic used.
- Approach taken and anatomy confirmed by echocardiogram.
- Any difficulties i.e. 'first pass' or 'second attempt', etc.
- Appearance of pericardial fluid aspirated.
- Volume of pericardial fluid aspirated.

# Nasogastric tube insertion

### Indications
- Feeding in patients with poor swallow (e.g. post-cerebrovascular accident).
- Lavage of gastric contents in poisoning.
- Post-operative for stomach decompression.
- Bowel obstruction.

### Contraindications
- Oesophageal stricture, obstructing tumour.
- Tracheo-oesophageal fistula.
- Achalasia cardia.
- Deviated nasal septum.
- Fractured base of skull.

### Risks
- Malpositioning in a lung.
- Trauma to the nasal and/or pharyngeal cavities.
- Perforation of oesophagus.

### Equipment
- Lubricant (e.g. Aquagel®).
- pH-testing strips.
- 50 ml syringe.
- Gallipots.
- Dressing pack.
- Nasogastric tube (12–18 French size).
- Hypoallergenic tape.
- Sterile gauze.
- Gloves.
- Disposable bowl.

### Procedure
- Introduce yourself, confirm the patient's identity.
- Explain the procedure to the patient, stating that it may be uncomfortable and can cause gagging, which is transient.
- Make sure that the patient understands the procedure and agree a signal to be made if patient wants you to stop (e.g. raising hand).
- To estimate the length of the tube required, measure the distance from the bridge of the nose to the tip of the earlobe and then to the xiphoid process.
- Position the patient semi-upright.
  - If unconscious, place the patient on their side.
- Check the patency of the nostrils and select a suitable side.
- Wash hands and put on gloves.
- Unwrap the tube and lubricate the tip by wiping it through a blob of lubricating gel.
- Insert the tip of the tube in the nostril and advance the tube horizontally along the floor of the nasal cavity backward and downwards.

- As the tube passes into the nasopharynx, ask the patient to swallow if they are able to do so.
  - Using a cup of water and straw often helps here.
- If there is any obstruction felt during advancement, withdraw and try in the other nostril.
- 🛑 Watch for any signs of distress; namely cough or cyanosis and remove the tube immediately if any of the above occurs.
- Once the tube has reached the measured distance, secure it in place with the tape.
  - The GOJ is generally 38–42cm from the nostril so advancement of the tube 55–60cm from the nostril usually positions the NG tube tip within the stomach.
- Aspirate a sample of fluid using a syringe.
- Place the aspirate on a pH-testing strip.
  - A pH of 5.5 or less suggests that the tube is in the stomach.
- If no aspirate obtained, change position and try again. If still unsuccessful, perform chest radiography to confirm position.
  - Be sure to leave the internal wire in the tube if you are sending the patient to x-ray. The tube itself is not radio-opaque and will be invisible on the resultant image.
- Once satisfied that the tube lies within the stomach, remove the inner wire and secure the tube to the tip of the nose.
  - It is sometimes helpful to curve the remainder of the tube towards the ear and secure to the cheek also.

**Procedure tips**

- 🛑 Medications such as proton pump inhibitors and acid-suppressing drugs may elevate the pH of the aspirate giving a 'false-negative' result. If in doubt, request a chest radiograph before using.
- 🛑 Low-pH fluid may also be aspirated from the lung in cases of aspirated stomach contents. If in doubt, request a chest radiograph before using.
- Chest radiography should be performed routinely in high-risk patients (those that are unconscious, intubated, or have poor swallow).
- The absence of cough reflex does not rule out misplacement of the tube in the airways.
- Auscultation for gurgling in the stomach is not a recommended method for confirming position.

**Documentation**

- Date, time, indication, informed consent obtained.
- Size of tube inserted.
- Length of tube internally (there are markings on the tube).
- This is important to allow other staff to assess whether the tube has moved in or out since insertion.
- Method by which correct placement was confirmed.
- Any immediate complications.
- Signature, printed name, and contact details.

# Ascitic fluid sampling (ascitic tap)

### Indications
- Diagnosing nature of new-onset ascites (i.e. exudate or transudate).
- Diagnosis of spontaneous bacterial peritonitis (SBP).
- Cytology to diagnose malignant ascites.

### Contraindications
- Acute abdomen that requires surgery.
- Pregnancy.
- Intestinal obstruction.
- Grossly distended urinary bladder.
- Superficial infection (cellulitis) at the potential puncture site.
- Hernia at the potential puncture site.

### Risks
- Persistent leak of ascitic fluid.
  - This is more likely if there is a large amount of fluid under tension
- Perforation of hollow viscera (e.g. bowel and bladder). This is very rare.
- Peritonitis.
- Abdominal wall haematoma.
- Bleeding is very rare but may occur if there is injury to inferior epigastric artery (be careful to tap lateral abdominal wall as described).

### Equipment
- Sterile gloves.
- Dressing pack.
- Antiseptic solution (e.g. iodine).
- 1% or 2% lidocaine.
- 1 x 20ml syringe.
- 2 x 5ml syringes.
- 21G (green) and 25G (orange) needles.
- Sterile containers.
- Culture bottles.
- Sterile dressing.

### Procedure
- Introduce yourself, confirm the patient's identity, explain the procedure, and obtain informed consent.
- Examine the abdomen and select a site for aspiration, three finger-breadths cranial to the anterior superior iliac spine.
  - ► Beware of positioning too medial as this risks hitting the inferior epigastric vessels
  - ► Be sure to identify and avoid any organomegaly which might interfere with procedure (in patients with massive splenomegaly, for example, avoid left iliac fossa).
- Clean the area with disinfectant and apply sterile drape.
- Using the 25 gauge (orange) needle and the 5ml syringe, administer local anaesthetic to the skin and subcutis, raising a wheal.

- Using the 21 gauge (green) needle, infiltrate deeper tissues, intermittently applying suction until the peritoneal cavity is reached, confirmed by flow of ascitic fluid into the syringe.
- Note the depth needed to enter the peritoneal cavity.
- Discard the used needles and attach a clean 21G needle to the 20ml syringe.
- With the green needle perpendicular to the skin, insert carefully, aspirating continuously until you feel resistance give way.
- Aspirate as much fluid as needed (usually 20ml is plenty).
- Withdraw needle and syringe and apply dressing.
- Send sample for Gram stain and culture (in blood culture bottles), white cell count/neutrophils, biochemistry, cytology (if malignancy suspected).
  - White cell count can be calculated in haematology lab; send fluid in EDTA-containing bottle
  - ► Total white cell count >500/mm$^3$ or neutrophils >250/mm$^3$ suggests spontaneous bacterial peritonitis—SBP
  - Neutrophil count is usually a manual procedure via microbiology and may take longer
  - If malignancy is suspected, a large volume of ascites (e.g. 500ml) should be sent to cytology.

## Procedure tips

- Check the patient's clotting and platelet count before the procedure and proceed with caution and senior advice if abnormal (correct if platelets <20x10$^9$/L, INR ≥2.5).
- Inform the laboratory especially during out of hours if cultures needed urgently and if SBP is suspected.
- ► If unable to obtain fluid despite correct technique, do not persist! Stop and seek senior advice.

## Documentation

- Date, time, indication, informed consent obtained.
- Type and amount of local anaesthetic used.
- Site aspirated.
- Aseptic technique used?
- How many passes?
- Volume and colour of aspirate obtained.
- Tests requested on samples.
- Any immediate complications.
- Signature, printed name, and contact details.

# Abdominal paracentesis (drainage)

The procedure below relates to a 'RocketMedical' non-locking drainage kit—the essence is the same for other catheter kits although minor details may differ. You should refer to the kit's instructions.

## Contraindications
- Acute abdomen that requires surgery.
- Pregnancy.
- Intestinal obstruction.
- Grossly distended urinary bladder.
- Superficial infection (cellulitis) at the potential puncture site.
- Hernia at the potential puncture site.
- Caution is needed in the presence of omental or peritoneal metastatic disease. In these cases, drainage is often performed under imaging guidance by a radiologist.

## Risks
- Haemodynamic instability, especially in cirrhotic patients; avoided by albumin replacement. (Usually 100ml 20% human albumin solution IV for every 2.5 litres fluid drained—check local protocols with the gastroenterology department).
- Renal dysfunction (in those with abnormal baseline renal function. May need to withhold diuretics and limit drain volume to 5L).
- Wound infection.
- Bleeding.
- Perforation of bowel and bladder.
- Abdominal wall haematoma.

## Equipment
- Rocket abdominal catheter pack (catheter sleeve, puncture needle, and adaptor clamp).
- Catheter bag and stand.
- 1 x 25G (orange) needle.
- 1 x 21G (green) needle.
- 3 x 10ml syringes.
- 5ml 1% lidocaine.
- Iodine or antiseptic solution.
- Sterile pack (including gloves, cotton wool balls, and bowl).
- Suitable adhesive dressing.
- Scalpel/blade.

## Procedure
- Introduce yourself, confirm the identity of the patient, explain the procedure, and obtain informed consent.
- Ensure that the patient has emptied their bladder.
- Position the patient lying supine or semi-recumbent.
- Percuss the extent of the ascitic dullness.
- Mark your spot in the left iliac fossa within the area of dullness.
  - Double-check clinical examination and imaging, if available. If splenomegaly is present, right-sided drainage is recommended.

- Wash hands and put the sterile gloves on.
- Clean the area thoroughly with antiseptic.
- Infiltrate the skin and subcutaneous tissues with lidocaine via the orange needle and 10ml syringe.
- Attach the green needle to another 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 curled catheter 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.
- Remove the plastic covering sheath.
- Attach a 10ml syringe to the end of the catheter.
- Make a small incision in the skin using the scalpel.
- Grasp the catheter needle ~10cm above the distal end and, with firm but controlled pressure, push the needle through the abdominal wall to ~3.5–4cm deep, aspirating with the syringe.
- Disengage needle from the catheter hub and advance catheter until the suture disc is flat against the skin, then withdraw the needle.
- Connect adaptor-clamp to the catheter hub and securely attach the rubber portion of the clamp into a standard drainage catheter bag.
- Secure the catheter to the abdomen using a suitable adhesive dressing.
- Ensure the clamp is open to allow fluid to drain.

### Procedure tips

- Avoid any scars or engorged veins to minimize complications
- Low-grade coagulopathy is common in cirrhotic patients and fresh frozen plasma and platelets is not routinely recommended; seek advice.
- Fluid leak can be minimized by the z track technique, moving the skin and subcutaneous tissue during insertion of drain, creating a zigzag path.
- If no aspirate is obtained despite multiple attempts, liaise with radiology and request an ultrasound and marking of a suitable site for aspiration. Alternatively, ask the radiology department to insert the drain under ultrasound guidance.

### Documentation

- Date, time, indication, informed consent obtained.
- Type and amount of local anaesthetic used.
- Site of drain.
- Aseptic technique used?
- How many passes?
- Volume and colour of fluid obtained.
- Any immediate complications.
- Document the required albumin replacement (if appropriate) and when the catheter should be clamped.
- Signature, printed name, and contact details.

# Sengstaken–Blakemore tube insertion

🛈 This should be performed only by senior medical staff in close liaison with an anaesthetist and, ideally, with endotracheal intubation especially in agitated patients and those with hepatic encephalopathy.

▶ The threshold to perform endotracheal intubation should be low, as the risk of regurgitation and aspiration is extremely high. Perform nasogastric lavage and stomach evacuation prior to procedure.

## Indications
- Life-threatening variceal bleeding where facilities for endoscopy are not available or pending endoscopic therapy.
- Life-threatening variceal bleeding where other modalities to control bleeding have failed.

## Contraindications
- Variceal bleeding has ceased or significantly slowed.
- Recent surgery to the gastro-oesophageal junction.
- Known oesophageal stricture(s).

## Risks
- Mucosal necrosis due to inadvertent traction.
- Oesophageal perforation. This may be due to a gastric balloon being inflated within the oesophagus or can occur secondary to over- or prolonged-inflation of the oesophageal balloon.
- Aspiration of fluid into the respiratory tract.
- Asphyxiation due to superior migration of the tube and balloons. See last 'procedure tip' below.

## Equipment
- Gloves, gown, and goggles.
- Saline flush.
- 2 x 50ml syringe.
- Local anaesthetic spray.
- Sengstaken–Blakemore tube (usually kept in refrigerator to increase its stiffness).
- Lubricant jelly (e.g. Aquagel®).
- Basin with sterile water.
- Suction equipment.
- Sphygmomanometer for pressure monitoring.

## Procedure
- Introduce yourself, confirm the patient's identity, explain the procedure to the patient, and obtain informed consent.
- Position the patient at 45 degrees.
- Administer anaesthetic throat spray to the oropharynx.
- Check the balloons in the tube for air leak by inflating them with an air-filled syringe and immersing in a basin of water. Air leak is indicated by air bubbles appearing.
- Deflate the balloons.

- Apply lubricant over the tip of the tube and advance it through the oral cavity slowly until it crosses the gastro-oesophageal junction.
  - The GOJ is generally 38–42cm from the nostril so advancement of the tube 55–60cm usually positions the tip within the stomach.
- Withdraw if the patient becomes breathless.
- Inflate the gastric (**not** oesophageal) balloon with 50ml air.
- At this stage an abdominal radiograph may be performed to confirm the position of the tube in the stomach.
- Once position is confirmed, inflate the gastric balloon to a total volume of 250ml air.
- Pull gently on the tube until resistance is felt.
- Secure with tape near the mouth with gauze pads, maintaining traction and tie the tube to a 500ml bag of saline. A pulley (e.g. a drip stand) is helpful in maintaining traction.
- Mark the tube near the mouth which will serve as an indicator to whether the tube has migrated later.
- Flush the gastric port with normal saline and aspirate at frequent intervals until it is clear, which indicates that bleeding has ceased.
- 🛈 If bleeding continues, inflate the oesophageal balloon with 40ml air and monitor the pressures using the sphygmomanometer frequently.
- ▶ After 12 hours' traction, relax the tension and push the tube into the stomach. If there is evidence of further bleeding, the gastric balloon can be re-inflated and traction re-applied with a view to repeat therapeutic endoscopy.
- ▶ During extubation (usually after 10–12 hours depending on clinical condition), deflate the gastric balloon first then the oesophageal balloon and withdraw the tube slowly.

## Procedure tips

- The tube can be used as a measure to control bleeding for about 12–18 hours. It should not be left in place for more than 24 hours.
- Frequent aspirations from the gastric port are needed to assess the status of bleeding.
- The tube has to remain in traction at the gastric balloon which will decompress the varices. However, direct pressure from the tube can cause mucosal ulceration. Examine frequently to ensure that excessive force is not being exerted.
- If the balloons migrate superiorly, airway obstruction may occur. In this instance, as an emergency measure, the tube can be quickly cut with a pair of scissors and removed. Keep a pair of scissors handy.

## Documentation

- Date, time, indication, informed consent obtained.
- Those present, including anaesthetic support.
- How many passes?
- Volume balloon inflated to and level of tube insertion.
- Any immediate complications.
- Signature, printed name, and contact details.

# Basic interrupted suturing

Many suturing techniques exist. The following is the most commonly used 'interrupted suture'.

### Contraindications
- Bites.
- Contaminated wounds.

### Risks
- Infection, bleeding, scar (including keloid scars).

### Equipment
- Suture (use cutting 3/8 or 1/2 circle needle for skin).
- Needle holder.
- Forceps.
  - Toothed for handling skin; non-toothed for other tissues.
- Scissors.
- Antiseptic solutions, drapes, sterile gloves.
- Dressing.

### Procedure: placing the suture
- Introduce yourself, confirm the identity of the patient, explain the procedure, and obtain verbal consent.
- Position the patient comfortably such that the wound is exposed. Clean and drape the area to be sutured.
- Mount the needle in the needle holder approximately 3/4 of the way from the point.
- Start suturing in the middle of the wound to ensure skin edges match up.
- Grasp the skin edge and support it with the forceps.
- Pass the suture through the skin at a 90° vertical angle and approximately 0.5cm from the skin edge.
- Rotate your wrist and follow the contour of the needle until the needle point is visible in the wound.
- Support the needle tip with the forceps and withdraw it from the wound.
- Remount the needle in the needle holder.
- Support the other edge of the wound with the forceps.
- Pass the needle horizontally into the skin edge. Aim to insert the needle at the same depth from the skin's surface as the needle emerged on the other side.
- Rotate your wrist until the needle is seen at the skin surface. Aim to pass the suture 0.5cm from the wound edge.
  - Ensure the entry and exit points are directly opposite each other to prevent distortion of the wound when the suture is tied.
- Support the needle with the forceps and withdraw it through the skin.
- Tie the suture (instructions follow).
- Cut suture ends with scissors leaving 0.5cm behind.
  - This allows it to be grasped when removing.

- Repeat the process proximal and distal to the first suture until the wound is closed.
- Cover with absorbable dressing.
- Give advice on signs of infection, wound care, and when sutures should be removed.

## Procedure: tying the suture (instrument tie)

- Pull the suture through until a 2–3cm 'tail' remains.
- Place the needle down at a safe site.
- Grasp the exiting suture (attached to the needle) with your non-dominant hand.
- Hold the needle holders (closed) in your dominant hand.
- Loop the suture twice around the needle holder.
- Without letting the loops slip, open the needle holder and use the tip to grasp the end of the suture 'tail'.
- Move your hands in opposite directions such that the loops slip off the jaws and around the suture.
- Snug the knot down and tighten it.
- Repeat the knot but wrap a single loop around the jaws of the needle holder in the opposite direction to previously.
- Tighten the suture.
- Pull the suture through the wound so the knot lies to one side of wound.
- Repeat until 3 knots are tied.

## Documentation

- Date, time, indication, informed consent obtained.
- Anaesthetic used?
- Suture used.
- Number of sutures.
- Dressing.
- Advice given on wound care and follow-up to patient.
- Signature, printed name, and contact details.

# Cleaning an open wound

The treatment of open wounds depends on:
- Depth and area.
- Contamination.
- Tissue loss (e.g. vascular, tendon, or nerve damage).
- Other (open fractures or joints, compartment syndrome).

## Contraindications
- Major injuries: vascular compromise, tendon rupture, nerve injury, open factures, or joints. These require senior and/or specialist advice.

## Risks
- Infection, failure to decontaminate wound.
- Haemorrhage, scar, further surgery.

## Equipment
- Anaesthetic (local or general).
- Gloves, mask and eye protection.
- 2 x kidney dish (1 for cleaning solutions, 1 to collect used wash).
- 50ml syringe.
- Swabs.
- Forceps, scalpel, scissors.
- Normal saline or antiseptic solution.
- Sterile drapes.

## Procedure

*Wound cleaning*
- Swab for microbiology if visibly contaminated or history suggestive.
- Clean wound with copious amounts of normal saline and/or water-based antiseptics using syringe.
- Clean wound with swabs from the centre outwards.
  - ► Do not use high-pressure irrigation (can push debris deeper).

*Inspection and removal of gross contamination*
- Photograph wound with adjacent ruler to document size.
- Look for gross contamination and remove with forceps.

*Deep palpation*
- Methodically check each area visually and with deep palpation to avoid missing contaminants and tissue injuries.
- Use forceps and wound retraction to examine all areas.
- Look for any damage to blood vessel, nerves, and tendons.
- Move the joints above and below the injury whilst looking at the tendon as it moves. Tendon injuries are easily missed if the wound was incurred in a different position to the resting state (e.g. clenched fist).
- ► If deep tracts are palpated, the wound may need to be extended into the skin above it to allow adequate drainage.

*Excision of dead tissue*
- Cut away any dead tissue until healthy tissue is visible.

*Maintaining drainage*
- Any cavity must be adequately drained.
- Siting a drain:
  - Identify the most dependent part of the cavity
  - Use artery forceps to identify the depth of the tract
  - With scissors, taper and cut a corrugated drain to fit into the tract
  - Pass the tip of the forceps from the tract base so they can be seen at the skin surface
  - Make an incision over the forceps to allow the drain to be sited
  - Grasp the tip of the drain with the forceps and ease into the wound
  - To stop the drain dislodging, a loose suture can be placed into the skin and either around the drain or sutured through one of its corrugations. (This depends on the type of drain used.)
- Finally, wash the wound with antiseptic solution.
- A pack can be used to keep small tracts open and allow drainage.
- A loose suture can be placed to keep the pack in place.
- ▶ Contaminated wounds and bites should not be sutured closed.

*Dressing*
- A non-stick dressing should be placed over the wound and edges, followed by gauze and bandage or tape.
- Further wound inspection and debridement is required at 48–96 hrs.
  - Examine sooner in heavily contaminated wounds.

## Procedure tips
- Instead of a syringe, a normal saline bag and giving set can be used.
- For finger lacerations, a digital nerve block provides good analgesia.
  - 🛑 Don't use adrenaline as this can infarct the digit!
- In an ATLS scenario, open wounds should be photographed and covered with an antiseptic-soaked dressing and bandage. The photograph will allow wound inspection by others, without the need to remove bandages and contaminate the wound further.
- ▶ Always x-ray glass and metal wounds.
- Small superficial wounds with no evidence of contamination on inspection can be closed with interrupted non-absorbable sutures.
  - Patients need to be given information on wound care, signs of infection and when the sutures should be removed.
- Superficial face and head wounds can be closed with skin glue.
- ▶ In some centres, facial wounds are only sutured by maxillo-facial specialists to improve cosmetic results. Check your local policy.

## Documentation
- Time, date, mechanism of injury.
- Vaccination status.
- Sensation and pulses.
- Analgesia.
- Draw diagram of wound site and inspection findings.
- How much wash was used?
- If sutured, which suture and when should it be taken out?
- Printed name, signature, and contact details.

# Applying a backslab

Plaster backslabs are used as immediate splints for fractures until definitive treatment is performed and are also used to protect the fracture fixation post-surgery.

## Equipment
- Stockinette.
- Padding (10cm × 1 roll = above or below elbow backslab, 15cm x 2 rolls = below knee backslab).
- Plaster of Paris bandages.
- Bowl or bucket of water (lukewarm, 25–35°C).
- Crêpe bandage.
- Scissors.

## Risks
- Circulatory and nerve impairment, compartment syndrome, pressure sores, joint stiffness.

## Procedure tips
- Backslab application is a 2-person procedure.
- Ensure the plaster fits well. A loosely applied cast will not provide adequate splintage and can rub, causing soreness.
- Ensure the plaster does not cause constriction. In the early stages following fractures, the limb may swell, further restricting blood and nervous supply to the limb.
- Ensure bony prominences are adequately padded.

## Documentation
- Date, time, indication, informed consent obtained.
- Neurovascular status of limb.
- Procedure performed.
- Plan of further management.
- Patient given instructions to contact staff if develops increasing pain, if extremities change colour (e.g. become blue), or develops 'pins and needles' or numbness.
- Signature, printed name, and contact details.

**Procedure: below knee backslab**

Used for fractures/dislocations at the ankle and fractures of the foot.

- Use a padded knee rest if available to hold the knee at an angle of 10–15°.
- Hold the ankle at 90° with the foot in a neutral position.
- Cut a length of the stockinette from just below the knee to the toes and apply onto the patient.
- Apply a layer of padding over the stockinette.
  - The padding should extend from just below the knee to the toes
  - Start the padding from one end, rolling it around the limb evenly, overlapping half of the previous turn each time.
- Measure a slab of 10 layers of 15cm plaster of Paris from just below the back of the knee down to the base of the toes.
- Fold the plaster slab and dip it into the water holding the ends.
- Remove the plaster from the water, squeeze gently, and straighten it out.
- Fan out the upper end of the slab to fit the calf area.
- Place from just below the knee along the posterior surface of the lower leg, underneath the heel, and down to the base of the toes.
- Mould and smooth the plaster to fit the contours of the leg with the palms of your hands.
- Cut two side slabs 10x20cm long (length dependent on size of patient) made from 6 layers of plaster.
- Dip these in water and apply either side of the ankle joint.
  - A U-slab may be used instead of the side slabs. A 10cm wide U-slab (made of 6 layers of plaster) should be applied down one side of the leg under the heel of the foot and up the other side. Great care must be taken not to let the slabs overlap anteriorly.
- Finally, turn the stockinette back over the top and bottom edges of the plaster.

## Procedure: below elbow backslab

Used for fractures/dislocations at the forearm (including Colles-type injuries) and fractures of the hand.

- Cut a length of the stockinette from just below the elbow to the knuckles, cut a small hole for the thumb.
- Apply the stockinette to the patient.
- Apply a layer of padding over the stockinette.
  - The padding should extend from the elbow to the knuckles of the back of the hand and showing the palmar crease, allowing flexion of the fingers
  - The thumb should be completely free
  - Start the padding from one end, rolling it around the limb evenly and overlapping half of the previous turn each time.
- Cut a length of plaster from below the elbow to the knuckles from a plaster of Paris slab dispenser 15 or 20cm wide (dependent on size of patient), or by forming a slab from 15 or 20cm plaster of Paris bandage using 5 layers.
- Fold the plaster and dip it into the water holding the ends.
- Remove the plaster from the water, squeeze gently, and straighten it out.
- Carefully position the slab on the limb over the padding from just below the elbow, down the dorsal surface of the limb to the knuckles.
- Mould and smooth the plaster to fit the contours of the forearm with the palms of your hands.
- Turn the stockinette back over the edge of the plaster cast at either end.
- Finally, apply the roll of crêpe bandage over the plaster and the overturned stockinette to hold the plaster in place as it sets.

**Procedure: above elbow backslab**

Used for fractures/dislocations at the forearm and elbow, also supracondy-lar fractures of the humerus.

- Place the limb in a position of 90° flexion at the elbow.
- Cut a length of the stockinette from the axilla to the knuckles of the hand, cut a small hole for the thumb.
- Apply the stockinette to the patient.
- Apply a layer of padding over the stockinette.
  - The padding should extend from the axilla to the knuckles of the back of the hand and showing the palmar crease, allowing finger flexion
  - The thumb should be completely free
  - Start the padding from one end, rolling it around the limb evenly and overlapping half of the previous turn each time.
- Prepare a 10 or 15cm plaster of Paris slab (dependent on patient size), using 5 layers. The slab should be long enough to extend from the axilla to the knuckles of the hand.
- Fold the plaster and dip it into the water holding the ends.
- Remove the plaster from the water, squeeze gently, and straighten it out.
- Carefully position the slab on the limb over the padding running down the posterior surface of the limb over the back of the elbow.
- Mould and smooth the plaster to fit the contours of the forearm with the palms of your hands.
- Prepare two 10cm-wide slabs of five layers of 25cm length (adjust length according to size of patient). Place these on each side of the elbow joint to reinforce it.
- Turn the stockinette back over the edge of the plaster cast at either end.
- Finally, apply the roll of crêpe bandage over the plaster and the overturned stockinette to hold the plaster in place as it sets.

# Manual handling

### Assisting a patient to stand

Moderate assistance is required from the patient.

*Procedure*
- ▶ Before beginning the procedure, ensure the patient has been assessed as able to weight-bear.
- ▶ Ensure the immediate area is clutter free.
- Ensure the patient has full understanding of the manoeuvre, and what is expected of them.
- Encourage the patient to move forward in the chair.
- Stand at the side of the chair, slightly behind the patient.
- Ensure the patient, and any other staff, are aware of which command to respond to, e.g. 'ready, steady, stand'.
- With one hand, place your arm nearest the patient around the patient's lower back, reaching as long and as low as is comfortable.
- Place the other hand at the front of the patient's shoulder.
- On the 'stand' command, as the patient rises from the chair, move your position forward such that you are standing next to the patient when upright, to aid their balance.
- ▶ Get the patient to help as much as possible during the manoeuvre e.g. pushing down on the arms of the chair if available.
- ⚠ If the patient is unsteady and unable to complete the manoeuvre, gently lower the patient back into the chair and re-assess the situation.

*Procedure tips*
- ▶ This procedure is only possible with cooperative patients who are able to weight-bear, and are able to understand basic commands.
- This can be carried out with 1 or 2 people, dependent on the patient.
- Allow sufficient time, so that the patient understands the process.
- It is important to encourage the patient's independence; ask them how they would carry out this manoeuvre at home.
- Include the patient in all decision making about the procedure e.g. they may feel comfortable using a Zimmer frame or similar walking aid.
- Check bed area for any furniture/equipment that could be moved to allow more space to complete the manoeuvre.
- ▶ Always check that intravenous fluids, catheters, drains, and other devices are safe and not likely to be pulled out during the procedure.
- Check with staff whether the patient has any history of cognitive problems, violence, or aggression or has any health problems which may prevent or impact upon the manoeuvre.

*Documentation*
- All patients should have had a moving/handling assessment completed by a physiotherapist in the first 24 hours after admission.
- Any issues raised following the move should be documented in notes.
- Full assessment should be completed prior to each move if the patient's condition has changed.

### Assisting a patient to roll whilst lying

*Equipment*
- 1 (or 2) members of staff.

*Procedure*
- Ensure the bed/trolley is at waist height and that the brakes are on, to avoid staff injuries.
- ▶ If the manoeuvre is being carried out with 1 member of staff, always roll the patient towards you.
  - ▶ If 2 members of staff are available, they should stand either side of the bed/trolley.
- Ensure adequate explanation is given to the patient.
- Ensure the patient's head is facing the way the patient will be moving.
- Place the patient's distant arm across their chest, and flex their distant hip and knee.
- Place an open-palmed hand on the patient's shoulder, and your other hand on the patient's hip or knee.
  - Staff may find it more comfortable to put one of their knees on the bed, to avoid stretching or bending.
- On the command 'ready, steady, roll', move back slightly, aiding the patient to roll towards you.
- Once the patient is on their side, they can be made comfortable with pillows.
- ⚠ It is also important to ensure the patient is secure, by making use of bedrails.

*Procedure tips*
- ⚠ Before carrying out the procedure ensure the area around the bed/trolley is clear of any obstacles.
- ⚠ Ensure there is adequate space on the bed/trolley for the patient to roll onto.
- It is important to have the correct number of staff available to carry out the manoeuvre.
- Do not rush and leave enough time to explain the procedure to the patient and other members of staff involved.
- It is important to have assessed the patient prior to carrying out this technique, to discover any contraindications to the patient lying on their side (e.g. problems with the patient's head and neck control, or any potential difficulties such as the patient's size).

*Documentation*
- All patients should have assessments carried out within 24 hours of admission. Care plan to be maintained/consulted as appropriate.
- Any issues or problems with manoeuvre should be documented in the notes.

## Assisting a patient to change position in bed (using a glide sheet)

*Equipment*
- Single-patient use multi-directional slide sheet/glide sheet.
- Minimum of two staff.

*Procedure*
- Ensure patient is aware of the procedure and has given consent, if able.
- Patient should be lying flat in bed.
- 🛈 Discuss desired end position of patient with the other handler(s).
- Move the bed to waist height to prevent staff injuries.
- Ensure the brakes on bed are secure.
- Staff should stand either side of the bed facing each other.
- To place glide sheet under patient, roll patient on bed sheet over to one side of the bed. Either:
  - One staff member leans over patient and pulls the bottom sheet to roll patient onto one side
  - Or, if possible, encourage the patient to roll themselves onto one side.
- The handler nearest the patient should hold sheet (and patient on their side) whilst the glide sheet is inserted by the other handler.
- Place the glide sheet between mattress and bottom sheet.
- The second handler should hold the glide sheet and push as far as possible under bottom sheet and the patient rolls back onto their back.
- Repeat from the other side until glide sheet is fully under the patient.
- Once the sheet is in place, agree which handler will give commands.
- Both handlers should grip the *bed* sheet, with both hands, as close to patient as possible. Place both feet firmly on the floor.
- On command of 'ready, steady, move', both handlers grip bottom sheet and gently move patient to previously agreed position.
- Place pillows appropriately for the patient's revised position.
- Reverse patient movement procedure to remove glide sheet.

*Procedure tips*
- Do not rush. Ensure sufficient time available to explain the manoeuvre to the patient and safely complete the manoeuvre.
- Check bed area for any furniture/equipment that could be moved to allow more space to complete the manoeuvre.
- ▶ Always check that intravenous fluids, catheters, drains, and other devices are safe and not likely to be pulled out during procedure.
- Check with staff whether the patient has any history of cognitive problems, violence, or aggression or has any health problems which may prevent or impact upon the manoeuvre.
- Ensure bedrails are put back into place following procedure.

*Documentation*
- All patients should have assessments carried out within 24 hours of admission and placed in their file.
- Any issues or concerns should be documented in the patient's notes to ensure other ward staff are aware of problems.

### Transferring a patient laterally using a transfer board

Use to transfer patients who are unable to move themselves.

*Equipment*
- Patient transfer board or 'Patslide®'.

*Procedure*
- There should be at least three handlers.
- Open transfer board (if folded) and place on bed/trolley you plan to transfer patient to.
- Explain the manoeuvre to the patient.
- Place destination bed/trolley alongside origin bed/trolley.
  - Ensure there is only a minimal gap between the bed/trolley.
- Check bed is at waist height to prevent staff injuries.
- Staff stand either side of bed/trolley facing each other, two people on the 'destination' side and one on the other.
- 🔔 Check brakes on bed and trolley secure.
- Staff at the patient's bedside to lean over patient and grip bed sheet as close to the patient's body as possible in both hands and roll the patient towards them.
- Staff at the bed/trolley onto which patient is to be transferred, put transfer board onto patient's bed/trolley.
- Staff at bedside allow patient to roll back onto board (which should be under the bed sheet).
- On command of 'ready, steady, move'....
- Handlers push and pull patient gently across on transfer board, dependent upon their position.
  - 🔔 Staff should ensure their arms remain straight and they do not lean forward, bending at the waist.
- Once patient is transferred, ensure sheets/blankets are replaced.
- Bedrails should be put into place as appropriate.

*Procedure tips*
- Ensure time is available to safely complete the manoeuvre.
- Check bed area for any furniture/equipment that could be moved.
- Always check IV fluids, catheters, drains, etc. are safe and unlikely to be caught or pulled out during procedure.
- Move any attachments onto transferring bed/trolley prior to the move.
- Check with qualified staff/physiotherapists regarding any changes in the patient's condition prior to manoeuvre.
- Staff should wear suitable footwear and non-restrictive clothing.
- Check with ward staff that patient can be laid flat.
- If NG-fed, ensure it is switched off to prevent patient aspirating.
- 🔔 Do not climb onto the bed/trolley.
- 🔔 Ensure both surfaces are the same height, making the manoeuvre both easier and more comfortable for the patient.

*Documentation*
- Any issues or problems with equipment or manoeuvre should be conveyed to the nurse in charge, documented in the notes, and an appropriate incident form completed.

### Transferring a patient using a hoist

Limited input from patient. Use this technique to transfer patients who are unable to weight-bear, sit patients up in the bed, or use a bedpan.

*Equipment*
- Hoist.
- Sling: single patient use (disposable).

*Procedure*
- ▶ There should be at least two handlers.
  - Check care plan regarding patient's suitability for hoist usage
  - Before getting equipment, ensure manoeuvre is explained to patient.
- Select appropriate sling: small, medium, or large.
- Ensure hoist and sling are compatible.
- Check hoist is able to take patient's weight: most are able to take up to 25 stones (170kg).
- Check bed is at waist height to prevent staff injuries.
- Staff stand either side of bed facing each other.
- ❗ Check brakes on bed secure.
- Patient should be rolled to one side of bed.
- Lay the hoist sling on the bed.
- Roll the patient to other side of the bed.
  - Sling should now be in a position from patient's head to thigh.
- Place the loops at shoulder end of sling on arm of hoist.
- Pass the thigh-end loops through each other, then place on hoist.
  - ▶ Ensure the loops are correctly positioned before moving.
- One handler should now manage the controls of the hoist.
- Second handler lowers patient's bed, then moves behind the patient/hoist, ready to guide them into the chair.
- Move patient back with hoist.
- Second handler gently guides patient into the chair.
- Once patient is in chair, disconnect loops from hoist.
- Remove sling from beneath lower legs of patient.

*Procedure tips*
- Ensure sufficient time available to safely complete the manoeuvre.
- Check bed area for any furniture/equipment that could be moved.
- Always check items such as IV fluids, catheters, and drains are safe and unlikely to be caught in hoist or pulled out during procedure.
- Check with qualified staff/physiotherapists regarding any changes in the patient's condition prior to manoeuvre. Transfer may be inadvisable.
- Staff should wear suitable footwear and non-restrictive clothing.
- Hoist should only be used to transfer patients short distances.
- Ensure hoist is fully charged before commencing manoeuvre.
- ❗ Ensure the brakes of the hoist are 'off'. This will allow the hoist to find its own centre of gravity.

*Documentation*
- Any issues with equipment or manoeuvre—advise nurse in charge and document in notes and complete an appropriate incident form.

### Transferring a patient using a log roll

Use this technique to transfer patients in whom a cervical spine injury is suspected or confirmed. The following assumes that the patient's neck is immobilized in a brace or blocks.

*Equipment*
- ▶ Minimum five members of staff.
- Patient transfer board or 'Patslide®'.

*Procedure*
- ▶ The most senior member of the team should take charge of the patient's head and neck and initiate commands.
- Ensure adequate explanation is given to the patient, and to all members of staff involved.
- Place destination bed alongside origin bed at waist height.
- One member of staff should position themselves at the head end of the patient, the other three should be spread alongside the patient, at the origin side. The final member of staff should be at the destination.
- 🌓 Check brakes on bed secure.
- ▶ The person responsible for the patient's head should have one hand either side of the patient's head, supporting the patient's shoulders.
- ▶ The person responsible for the patient's upper body should have one hand on the patient's distant shoulder, and the other on the lateral aspect of the patient's chest.
- ▶ The person responsible for the patient's pelvis should have one hand on the lateral aspect of the pelvis and the other under the thigh.
- ▶ The person responsible for the patient's lower legs should have both hands under the calves.
- On the command 'ready, steady, roll' the three members of staff at the side of the patient will slowly move backwards with straight arms, rolling the patient towards them.
- Staff at the bed/trolley onto which patient is to be transferred, put transfer board onto patient's bed/trolley.
- On the command 'ready, steady, roll' the four members of staff at the side of the patient roll the patient back flat, keeping the neck straight.
- One member of staff should now move around the bed such that there are two on each side and one at the head.
- On the command of 'ready, steady, move', handlers move the patient gently across keeping the head and neck immobilized.

*Procedure tips*
- Ensure sufficient time available to safely complete the manoeuvre.
- Check bed area for any furniture/equipment that could be moved.
- Staff should wear suitable footwear and non-restrictive clothing.
- ▶ It is essential that the patient's body be kept in alignment, and the manoeuvre is carried out in one smooth and controlled movement.

*Documentation*
- Any issues with equipment or manoeuvre, advise nurse in charge and document in notes and complete an appropriate incident form.

### Aiding a falling patient

- ▶ It is essential that if a patient falls, the member of staff **must not** try to catch the patient, but must allow them to fall, as there is no safe method for this situation.
- ▶ Allowing the patient to fall may feel contrary to the staff's natural instincts to help but trying to catch a patient will only result in injury to staff.
- ❶ Instead, every attempt must be made to reduce injury to the patient (e.g. moving objects out of the patient's way if possible).

*Falling in a forward direction*
- If a member of staff is walking with a patient as they fall in a forward direction, the member of staff must allow the patient to fall.

*Falling towards a member of staff*
- If the fall is towards the member of staff, it may be possible to control the patient's movements safely to minimize injury to them.
- The member of staff should move close to the patient, standing directly behind them with their leg closest to the patient flexed. Then they should gently guide the patient's body down their flexed leg to the floor.

*Procedure tips*
- The risk of falling should be minimized by only performing tasks appropriate to the patient's ability (e.g. only allow patients to walk if they are fully mobile).
- Use equipment to reduce the risk of falls i.e. Zimmer frames or walking sticks.
- ❶ A patient falling is an unpredictable and sudden event. However, the member of staff should take every care to maintain a good posture at all times, avoiding twisting or stretching.
- ▶ If present when a patient falls, the member of staff should immediately call for assistance, to ensure an adequate number of staff are present if the situation turns into an emergency.

*Documentation*
- All patients should have assessments carried out within 24 hours of admission and placed in their file.
- Any fall or issues should be documented in the patient's notes to ensure other ward staff are aware of problems.

### Aiding a fallen patient

- ▶ It is important to assess the fallen patient immediately, to establish the cause for the fall and any immediate consequences (e.g. fainting, fractures, or cardiac arrest) so that staff can respond to the situation accordingly.

*Equipment*

- Minimum of two members of staff.
- Other equipment dependent on circumstances:
  - Two chairs, trolley, slide sheets, hoist with appropriate sling.

*Procedure: if patient is cooperative*

- Instructions may be given to help the patient up from the floor. Ask the patient to follow this routine:
- Roll onto their side ...
- Push up on their hands until they are in a sitting position ...
- Bend their knees up and move onto all fours ...
- Place their hands onto the seat of a chair for balance ...
- Move one leg forward, so they are in a half-kneeling position ...
- At this point, the patient should be able to push with their hands to stand up, and sit on a chair placed behind them.
- If needed, the patient can now be hoisted onto a trolley for further assessment.

*Procedure: if patient is uncooperative*

- A hoist should be used.

*Procedure: if fallen in a confined space*

- Place a slide sheet under their body.
- With a minimum of two members of staff, the patient can then be slid on the floor a short distance to allow better access to assist the patient.
  - It is essential that the members of staff maintain a good posture at all times during this procedure.

*Procedure tips*

- ▶ It is essential to establish the cause of the fall and act accordingly.
- ▶ It is important that, as the patient is moving up from the floor, their condition is continuously monitored.
  - 🛈 If the patient has fainted, they may be at risk of falling again.
- It is important to allow the patient time to carry out the manoeuvre, as this will reduce the amount of manual assistance required from staff.
- ▶ It is extremely important that the patient is NEVER LIFTED.
  - Lifting a patient is hazardous and may result in staff injury.

# Clinical data interpretation

# Electrocardiography (ECG)

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 myocytes 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. 19.1).

### 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 down the rapidly conducting Purkinje fibres—bundle of His, then down left and right bundle branches to depolarize both ventricles (see Fig. 19.2). 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)).
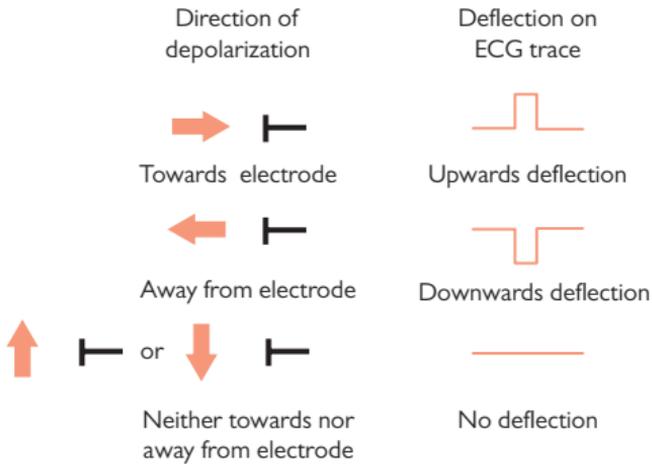
**Fig. 19.1** Diagrammatic representation of how waves of depolarization are translated onto the ECG trace depending on the relationship to the electrodes.
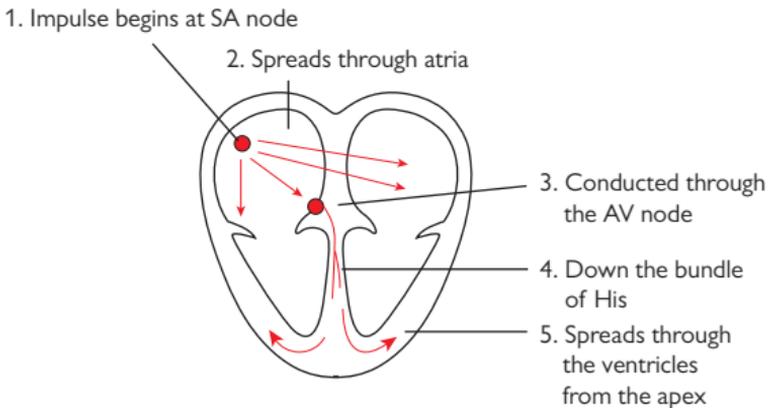


**Fig. 19.2** Diagrammatic representation of the electrical conduction pathway in the normal heart.

### 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. 19.3). 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.

*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. 19.4).
- aVR looking at the right atrium (all the vectors will be negative for this lead in the normal ECG).
- aVF, II, and III viewing 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 the sake of simplicity when considering the standard ECG trace, we can describe the basic shape common to all leads (see Fig. 19.5).
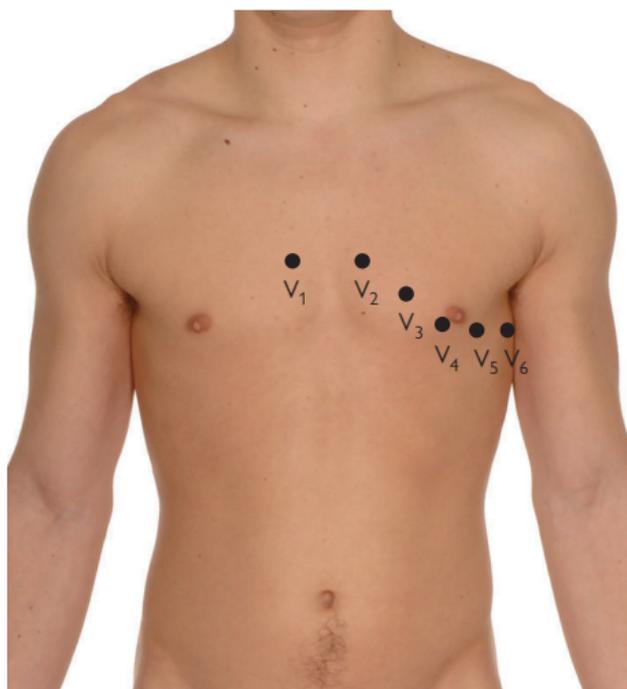
**Fig. 19.3** Correct placement of the 6 chest leads.
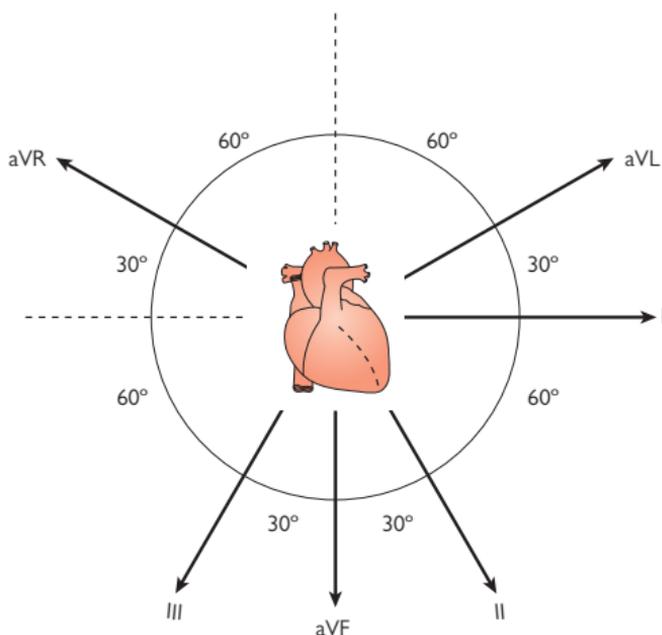


**Fig. 19.4** The respective 'views' of the heart of the 6 limb leads. Note the angles between the direction of the limb leads – these become important when calculating the cardiac axis.

## 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 myocardial infarction
  - 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.

*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.
- 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)
  - If irregular but no pattern, then it is said to be 'irregularly irregular' (e.g. atrial fibrillation).
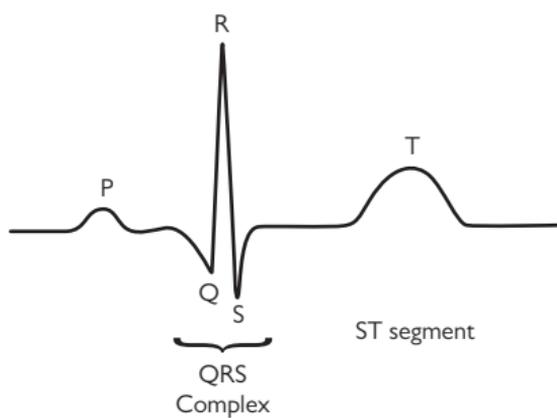
**Fig. 19.5** The basic shape of a typical ECG trace.

## 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 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. 19.6). An axis outside of this range may suggest pathology, either congenital or acquired.

Note, however, that 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 (Box 19.1).

*Calculating the axis*

Look at Fig. 19.7. 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 in Fig.19.7. There are less reliable short cuts, however.

- Draw a diagram like Fig.19.6 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. 19.6).
- 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.

An even easier method is to look at the print-out. Most computerized machines will now tell you the ECG axis (but you should still have an understanding of the theory behind it).
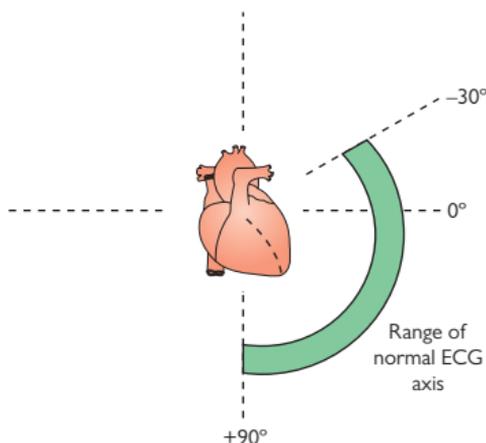
**Fig. 19.6** The normal ECG axis.
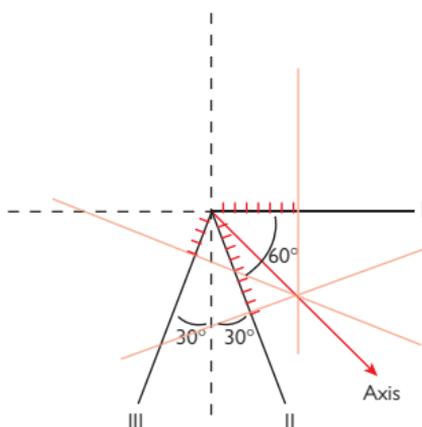


**Fig. 19.7** Calculating the ECG axis using leads I, II, and III. See text.

---

**Box 19.1 Some 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 myocardial infarction
- Cardiomyopathies
- Tricuspid atresia.

*Right axis deviation (>+90 degrees)*
- Right ventricular hypertrophy
- Right bundle branch block (RBBB)
- Anterolateral myocardial infarction
- Right ventricular strain (e.g. pulmonary embolism)
- Cor pulmonale
- Fallot's tetralogy (pulmonary stenosis).

### 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' (Fig.19.8).

Causes of heart block include ischaemic heart disease, idiopathic fibrosis of the conduction system, cardiomyopathies, inferior and anterior MI, drugs (digoxin, β-blockers, verapamil), and physiological (1st 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. 19.8).

*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. 19.8). 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. 19.8.

*Third degree heart block*

Also called complete heart block. See rhythm strip 4 (Fig. 19.8). 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.
- The QRS complex is an abnormal shape as the electrical impulse does not travel through the ventricles via the normal routes (see ventricular escape).
- P waves may be seen 'merging' with QRS complexes if they coincide.

*Notes*
- 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 $V_1$.
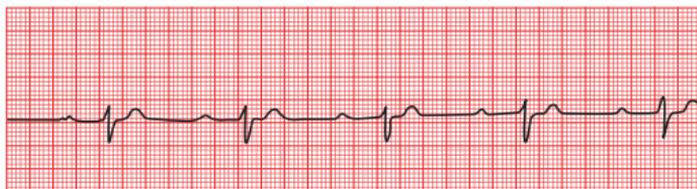
Rhythm strip 1—first degree heart block.
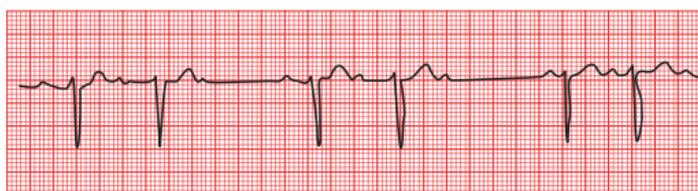


Rhythm strip 2—second degree heart block Möbitz type I.



Rhythm strip 3—second degree heart block Möbitz type II.



Rhythm strip 4—third degree (complete) heart block.

**Fig. 19.8** 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. 19.9). 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' (Fig.19.9).

*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 (Fig.19.10).

*ECG changes*

(See Box 19.2 for bundle branch block mnemonic.)
- '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 flattening or inversion 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 (Fig.19.11).

LBBB should always be considered pathological.

*ECG changes*

- 'M' pattern seen in $V_6$.
- T wave flattening or inversion in lateral chest leads ($V_5$–$V_6$).

*Some causes of LBBB*

- Hypertension.
- Ischaemic heart disease.
- Acute myocardial infarction.
- 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.
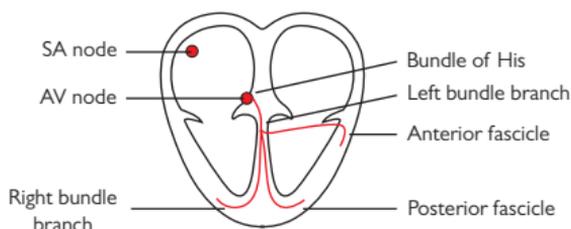
**Fig. 19.9** Diagrammatic representation of the conducting system of the heart.
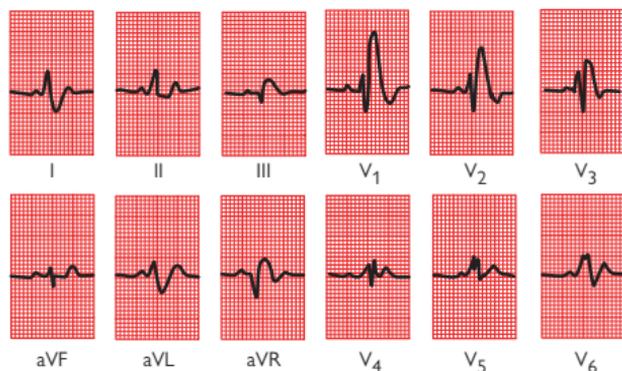


**Fig. 19.10** Typical 12-lead ECG showing RBBB.
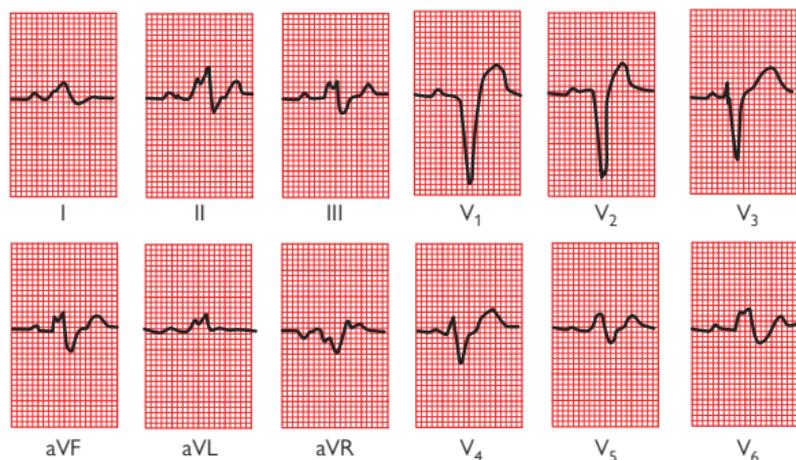


**Fig. 19.11** Typical 12-lead ECG showing LBBB.

---

**Box 19.2 Bundle branch block mnemonic**
- 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 an '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. (Rhythm strip 1, Fig. 19.12.)

*Some causes of sinus bradycardia*

- Drugs (β-blockers, verapamil, amiodarone, digoxin).
- Sick sinus syndrome.
- Hypothyroidism.
- Inferior MI.
- Hypothermia.
- Raised 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 2, Fig. 19.12.)

*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.
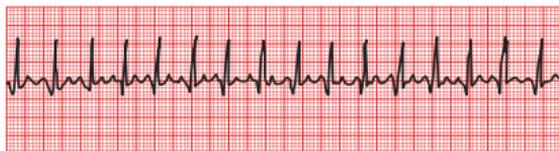


Rhythm strip 2—sinus tachycardia.

**Fig. 19.12** 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 (Fig.19.13).

There are four main causes of a supraventricular tachycardia that you should be aware of: atrial fibrillation, atrial flutter, junctional tachycardia, and re-entry tachycardia.

*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 discernible pattern.

*ECG features*
- No P waves. Rhythm is described as *irregularly irregular.*
- Irregular QRS complexes.
- Normal appearance of QRS.
- Ventricular rate may be increased ('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 the 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.



Rhythm strip 1—atrial fibrillation.



Rhythm strip 2—atrial flutter with 2:1 block.



Rhythm strip 3—atrial flutter with 4:1 block.

**Fig. 19.13** 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.
- Ischaemia 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'.

This is an example of a 'fusion beat' in which normal and abnormal ventricular depolarization combine to give a distortion of the QRS complex (Fig. 19.14 and Box 19.3).

*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' (Figs 19.15 and 19.16).

**Fig. 19.14** Rhythm strip showing Wolff–Parkinson–White syndrome.



Conduction at the AV node

Accessory conduction pathway
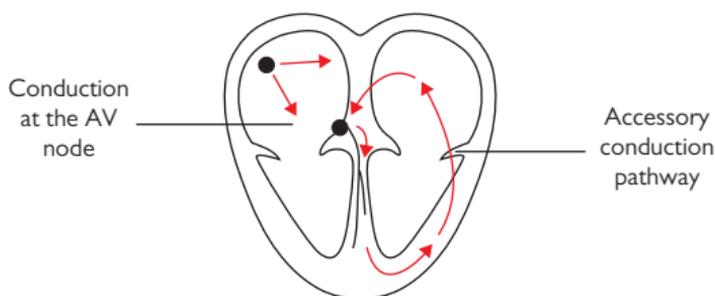
**Fig. 19.15** Diagrammatic representation of re-entry tachycardia.
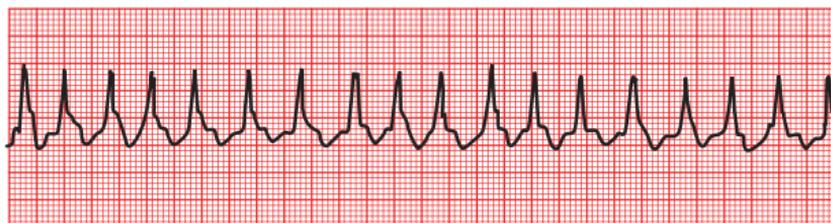


**Fig. 19.16** Rhythm strip showing a re-entry tachycardia.

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**Box 19.3 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 two 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.

### 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 (Fig.19.17).

*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 (see also Box 19.5).

*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*

- Ischaemia (acute including MI or chronic).
- Electrolyte abnormalities (reduced $K^+$, reduced $Mg^{2+}$).
- 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 (Box 19.4).

*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.
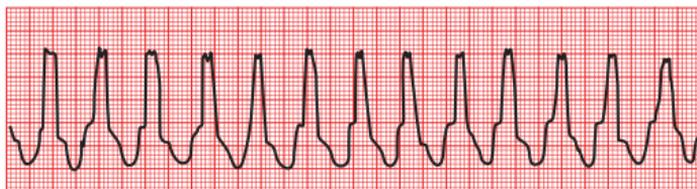
Rhythm strip 1—monomorphic ventricular tachycardia (VT).



Rhythm strip 2—ventricular fibrillation (VF).



Rhythm strip 3—'fine' ventricular fibrillation.

**Fig. 19.17** Rhythm strips showing ventricular rhythms.

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**Box 19.4 Fine VF**

This is VF with a small amplitude waveform. It may resemble asystole on the ECG monitor (see Fig. 19.19), 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 management 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 (Fig.19.19).

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).

The ventricular beats will 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.

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 recognizable activity is lost (asystole).

---

#### Box 19.5 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 (Fig.19.18).

Torsades results from a prolonged QT interval. Causes include congenital long-QT syndromes and drugs (e.g. anti-arrhythmics). Patients may also have reduced $K^+$ and $Mg^{2+}$.

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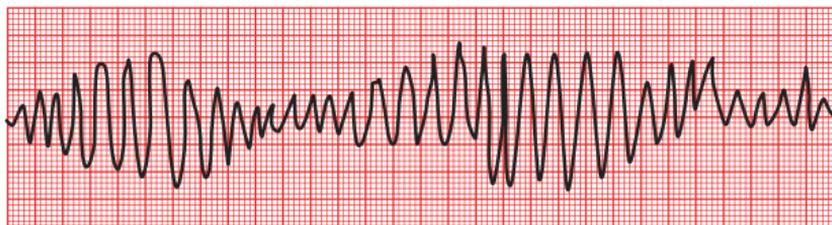


**Fig. 19.18** Rhythm strip showing torsades.

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.
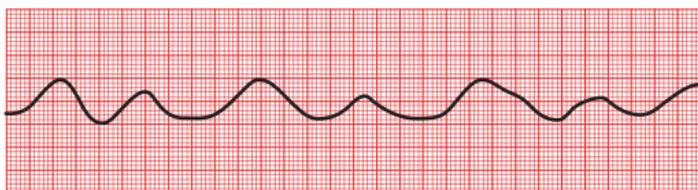


Rhythm strip 4—agonal rhythm.
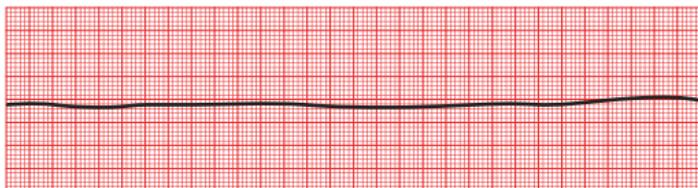


Rhythm strip 5—asystole.

**Fig. 19.19** Rhythm strips showing 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'.
  - 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 $V_1$–$V_3$ 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 (Fig.19.20).
- 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.
- Electrolyte imbalances cause a number of T wave changes:
  - Raised $K^+$ can cause tall tented T waves
  - Low $K^+$ can cause small T waves and U waves (broad, flat waves occurring after the T waves)
  - Low $Ca^{2+}$ can cause small T waves with a prolongation of the QT interval. (Raised $Ca^{2+}$ 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—hyperkalaemia with small T waves and U waves.

**Fig. 19.20** 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. Changes in the ST segment can represent myocardial ischaemia and, most importantly, acute MI (Fig.19.21).

*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.

*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.

## 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 $V_1$. 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 $V_1R$, $V_2R$, $V_3R$, and so on.
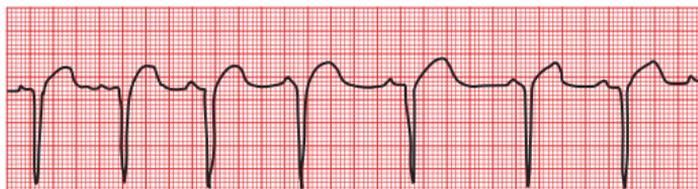
Rhythm strip 1—lead $V_2$ showing acute myocardial infarction.



Rhythm strip 2—pericarditis. The ST elevation is usually described as 'saddle-shaped'.



Rhythm strip 3—ischaemia.



Rhythm strip 4—digoxin use showing the 'reverse tick'.

**Fig. 19.21** 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 increase its muscle mass in response. This increased 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 include:
  - 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 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 (Fig.19.22).

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 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.
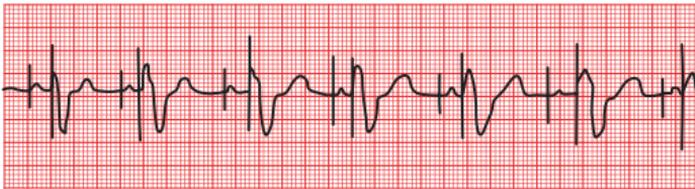


**Fig. 19.22** Rhythm strip showing dual chamber pacing.

# Peak expiratory flow rate (PEFR)

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 (Fig.19.23).

See ➲ Chapter 18 for how to perform this test.

See Boxes 19.6 and 19.7 for other tests.

*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.

*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.
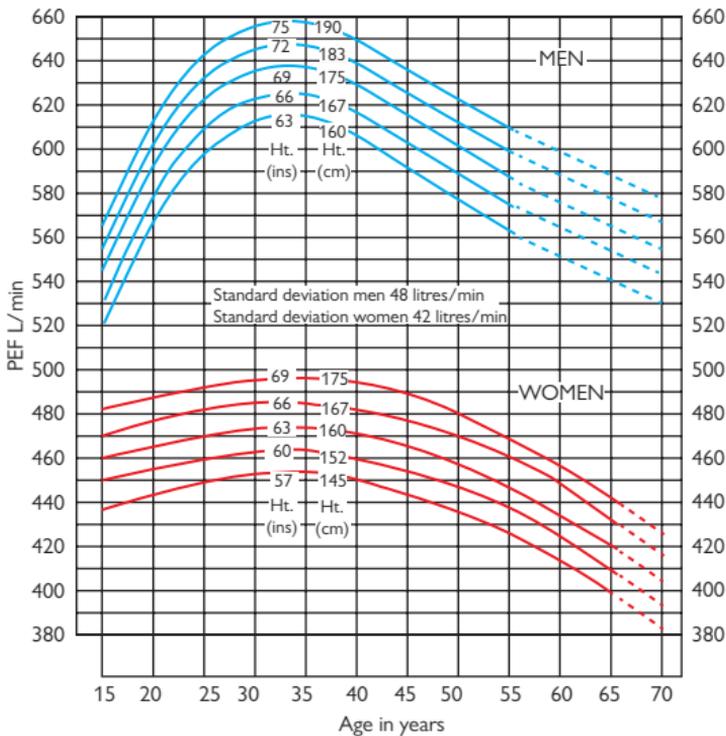


**Fig. 19.23** Normal PEFR by age and gender. Image reproduced from the *Oxford Handbook of Clinical Medicine*, with permission.

**Box 19.6 Gas transfer**

- This test measures the capacity of a gas to diffuse across the alveolar–capillary membranes. This not only adds further clues to the nature of the lung disease but is also a measure of function which can give important prognostic information and help guide treatment
- DLCO (carbon monoxide diffusion capacity) measures the uptake from a single breath of 0.3% CO
- DLCO is reduced in interstitial lung disease (the fibrotic insterstitium limits gas diffusion) and emphysema (the total surface area available for gas transfer is reduced).

**Box 19.7 Other lung function tests**

Specialized lung function centres can calculate static lung volumes with a body plethysmograph or using helium rebreathe 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.

# Basic spirometry

Spirometry measures airflow and functional lung volumes; this can aid diagnosis of a number of conditions, but is primarily used to distinguish between restrictive and obstructive lung diseases.

Patients are asked to blow, as fast as possible, into a mouthpiece attached to a spirometer. This records the rate and volume of airflow.

Most spirometers are now hand-held computerized devices which will print a spirometry report for you and calculate normal values.

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.

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 as well as to assess for small airways obstruction (Figs 19.24 and 19.25).



IC = Inspiratory capacity
TLC = Total lung capacity
RV = Residual volume
FRC = Functional residual capacity
TV = Tidal volume
FVC = Forced vital capacity
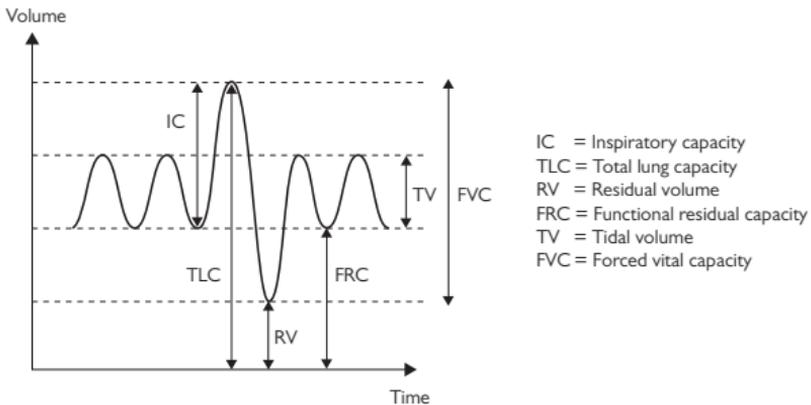
**Fig. 19.24** Normal pattern of lung volumes.
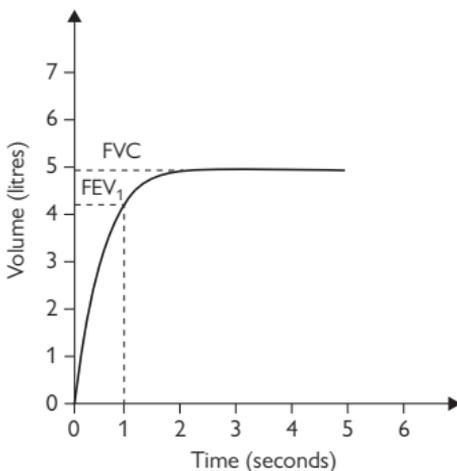


**Fig. 19.25** Spirogram showing normal volume–time graph.

### Common patterns of abnormality

*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 include COPD, asthma, and bronchiectasis as well as 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 include 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, and pleural thickening (Table 19.1 and Fig. 19.26).

Table 19.1 Obstructive vs restrictive spirometry results

| Pattern | $FEV_1$ | FVC | $FEV_1$/FVC ratio | TLC | RV |
|---|---|---|---|---|---|
| Obstructive | ↓ | ↔/↓ | <75% | ↑ (or ↔) | ↑ |
| Restrictive | ↓ | ↓ | >75% | ↓ | ↓ |
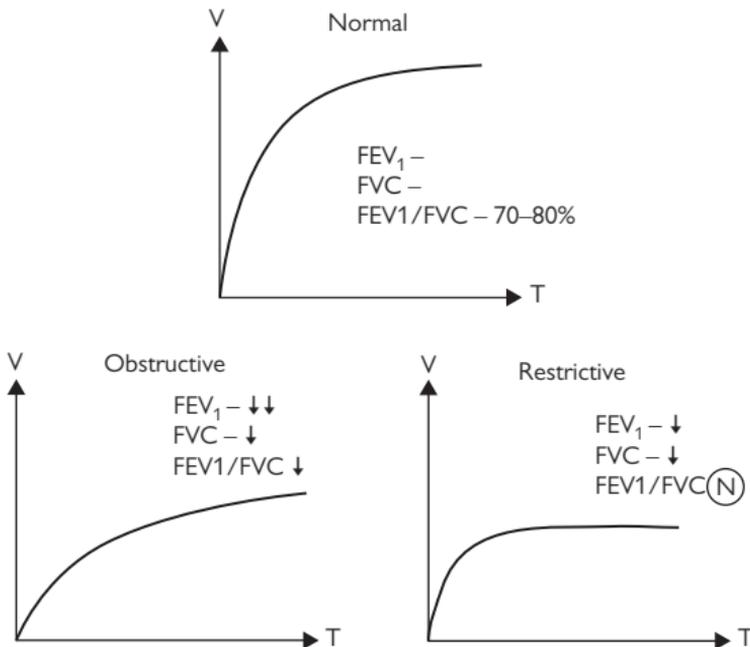


**Fig. 19.26** Spirograms showing obstructive and restrictive volume/time curves.

# Arterial blood gas analysis

### 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 (Box 19.8):

*pH*
- Is it low (acidosis) or high (alkalosis)?

*$PaCO_2$*
- 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.
- If $PaCO_2$ is low and there is acidosis then the respiratory system will not be to blame and there is a metabolic acidosis.
  - Confirm this by looking at the $HCO_3^-$, it should be low.
- If $PaCO_2$ is high or normal and there is alkalosis, there must 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 a 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.

▶ A compensated picture suggests chronic disease.

**Box 19.8 Reference ranges**
- pH 7.35–7.45
- $PaCO_2$ 4.7–6.0kPa
- $PaO_2$ 10–13kPa
- $HCO_3^-$ 22–26mmol/L
- Base excess –2 to +2.

Table 19.2 Obstructive vs restrictive spirometry results

| Pattern | pH | TLC | RV |
|---|---|---|---|
| Respiratory acidosis | ↓ | ↑ | ↔ (↑ if compensated) |
| Metabolic acidosis | ↓ | ↔ (↓ if compensated) | ↓ |
| Respiratory alkalosis | ↑ | ↓ | ↔ (↓ if compensated) |
| Metabolic alkalosis | ↑ | ↔ (↑ if compensated) | ↑ |

**Box 19.9 Anion gap**
- $(Na^+ + K^+) - (HCO_3^- + Cl^-)$
- Normal range = 10–18 mmol/L.

### Acidosis

A relative excess of cations (e.g. $H^+$), unless adequately compensated, will result in acidosis (more correctly acidaemia) (Table 19.2).

*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.

*Metabolic acidosis*
- pH ↓.
- $HCO_3^-$ ↓.
- $PaCO_2$ may be ↓ if compensated.

It is useful to calculate the anion gap to help distinguish causes of metabolic acidosis (Box 19.9).

An increased anion gap points to increased production of immeasurable anions.

*Conditions which can lead to metabolic acidosis:*
- Raised 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 $HCO_3^-$).

## Alkalosis

A relative excess of anions (e.g. $HCO_3^-$), unless adequately compensated, will result in alkalosis (more correctly alkalaemia). (See Box 19.10.)

*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.

*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.

---

**Box 19.10 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 low $PaO_2$, and $O_2$ depleted cells then produce lactic acid.

---

# 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 (see Table 19.3).

## Normal adult CSF

- Pressure 6–20cm $H_2O$.
- Red cells nil.
- Lymphocytes ≤5 x $10^6$/L.
- Neutrophils nil.
- Protein <450 mg/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.

❶ Premature babies, newborns, children, and adolescents have different normal ranges.

**Table 19.3** 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 |

# 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.

## Dipstick

Dipstick testing gives semi-quantitative analysis of:
- Protein (normally negative).
- Glucose (normally negative).
- Ketones (normally negative).
- Nitrites (normally negative).
- Blood (normally negative).
- Leukocytes (normally negative).
- Bilirubin (normally negative).
- pH (normally acidic with range 4.5–8.0).
- Specific gravity (normal range 1.000–1.030).

*Notes on dipstick testing*
- ▶ Test the urine within 15 minutes of obtaining the sample.
- ▶ 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, leukocytes, and red blood cells). Organism growth and antibiotic sensitivities and can also be determined.

▶ Asymptomatic bacteriuria is more common in pregnancy (up to 7%) and can lead to pyelonephritis and potential fetal complications.

*Characteristic urinalysis findings*
- UTIs: nitrites, leukocytes.
- 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: leukocytes, no organisms grown (sterile pyuria).
- Sexually transmitted diseases (chlamydia, gonorrhoea): sterile pyuria.

# Pleural fluid

Fluid in the pleural space can be classified as:
- Exudate (protein content >30g/L).
- 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 ⟳ Chapter 18.

See Box 19.11 for other tests.

*Transudate causes*

Transudates are largely cause by increased venous or reduced oncotic pressure.
- Heart failure.
- Hypoproteinaemia (liver failure, malabsorption, nephrotic syndrome).
- Hypothyroidism.
- Constrictive pericarditis.
- Meig's syndrome (ovarian fibroma and pleural effusion).

*Exudate causes*

Exudates are largely caused by increased capillary permeability.
- Pneumonia.
- Empyema.
- Malignancy (lung, pleura, lymph).
- Pulmonary infarction.
- TB.
- Systemic lupus erythematosus (SLE).
- Rheumatoid arthritis.
- Dressler's syndrome (post MI).

---

**Box 19.11 Other pleural fluid tests**
- Microscopy, culture (conventional and TB culture), and sensitivity (Gram stain, Ziehl–Nielsen stain)
- Cytology (malignant cells)
- Biochemistry.
  - Protein
  - Glucose (reduced if rheumatoid or pneumonia related)
  - Amylase (increased in pancreatitis)
  - LDH (lactate dehydrogenase—increased in empyema, malignancy, rheumatoid disease).

---

# Ascitic fluid

Fluid in the peritoneal cavity can result in abdominal distension and breath-lessness. As with pleural fluid, analysis of an aspirated sample can aid diagnosis. See ➜ Chapter 18 for ascitic tap guidance. See Box 19.12 for other tests.

## Common causes of ascites

- 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)

- SAAG = [serum albumin] − [ascitic fluid albumin].

*SAAG >11g/L*

- Portal hypertension.
  - Cirrhosis
  - Alcoholic hepatitis
  - Cardiac ascites
  - Budd–Chiari syndrome
  - Portal vein thrombosis
  - Massive liver metastases
  - Acute fatty liver of pregnancy.

*SAAG <11g/L*

- Infection.
- Malignancy.
- Nephrotic syndrome.
- Pancreatitis.
- Biliary ascites.
- Serositis in connective tissue disease.
- Bowel perforation or infarction.

---

**Box 19.12 Other ascitic fluid tests**

- MCS (bacterial peritonitis, TB)
  - Spontaneous bacterial peritonitis = neutrophils >250/mm$^3$.
- Cytology (malignant cells, macrophages in inflammatory diseases)
- Biochemistry (protein, glucose, amylase).

Further tests you may consider for a patient with ascites include: liver function tests, clotting, urea and electrolytes (U&Es), hepatitis serology, auto-antibodies, ultrasound scan of liver/pelvis, OGD (varices).

---

# Other investigations

**Notes**

The procedures detailed in this chapter are for information
only—to enable the reader to discuss it with their patients, to
prepare the patients correctly, and to identify those patients
who may or may not be suitable.
▶ The reader is not expected to perform any of these
investigations themselves and this chapter is not intended as a
resource for those learning how to perform the investigations.

# Computed tomography (CT)

### Indications
- Indications are manifold and too numerous to list. See 'making best use of a department of clinical radiology' via ℞ http://www.rcr.ac.uk

### Contraindications
- The standard radiation protection precautions apply.
- The patient must be able to lie flat and still.
- Examinations of the chest usually require the patient to hold their breath.

### Technology
- The CT scanner (Fig. 20.1) houses an x-ray tube and rows of detectors which spin at 2–3 revolutions per second, creating a force of up to 25g.
- As the patient is moved slowly through the machine, spiral data is acquired which is then converted to 'slices' by the CT software and sent to PACS or a connected workstation for viewing.

### Procedure
This depends on the part of the body examined and the indications for the examination.
- If indicated, the patient may be given oral contrast an hour or more before the examination.
- The patient lies (usually supine) on the scanner table.
  - Head-first for head and neck; feet-first for almost everything else.
- 'Scout' views are acquired which are brief swipes across the area of interest. The resultant images are then used by the radiographer to set the parameters for the scan.
- Most examinations involve intravenous iodinated contrast being given. Note that the contrast is not radioactive.
  - This is usually delivered via an intravenous cannula by an automatic pump-injection device, controlled remotely by the radiographer
  - Contrast may be hand-injected immediately before some scans.
- Depending on the part of the body examined, the patient may be asked to hold their breath via speakers in the machine. Microphones within the scanner allow the staff in the control room to hear the patient.
- The scan itself lasts no more than a couple of minutes.
- Time taken to transfer the patient onto the scanner and set up the intravenous injections will vary.

### Risks
- Intravenous contrast reactions include anaphylaxis and nephrotoxicity.
  - Intravenous contrast should not be given to patients with renal impairment unless in special circumstances. Check local guidance.
- Extravasation of intravenous contrast (pain, swelling, erythema).

**Fig. 20.1**  A typical CT scanner. Note the presence of metal in the room (oxygen cylinder, etc.) indicating this is not an MRI scanner and the lead apron indicating that x-rays are being used. The CT scanner has a laser marker (shown) to help with patient positioning, an MRI scanner does not.

## Patient preparation

- Fasting: not required for most examinations.

# Magnetic resonance imaging (MRI)

## Indications

- Indications are manifold and too numerous to list. See 'making best use of a department of clinical radiology' via ✋ http://www.rcr.ac.uk

## Contraindications

- As there is no ionizing radiation, radiation precautions do not apply.
- ▶ All ferromagnetic materials will be strongly attracted to the scanner creating missiles which may prove extremely dangerous. MRI-safe trolleys, resuscitation equipment and wheelchairs must be employed.
- ▶ Implanted ferromagnetic devices, aneurysm clips, and retained foreign bodies (e.g. shrapnel or metallic fragments in the eyes) will also move towards the scanner potentially causing major injury.
- ▶ Although electronic pacemakers are not made of ferromagnetic material, they may be 'reset' or stop altogether. The next generation of very new pacemakers is 'MRI safe' – check with the manufacturer.
- A strict questionnaire is employed before anyone (staff or patient) is allowed near the magnet. If in doubt, access is denied.
- Magnetic tape and credit cards may be 'wiped' by the magnet.
- ▶ Many brands of mascara contain ferromagnetic filaments which may heat and cause burns to the eyelids.
- ▶ Caution should also be taken with tattoos; some contain iron.
- The patient must be able to lie flat and still for the duration of the scan.
- Most scanners are relatively tight; larger patients may not fit—check the size and weight limits with your local department.

## Technology

- The MRI scanner (Fig. 20.2) houses a very large electromagnet which is always on.
- Radiowaves are produced by the machine which interact with hydrogen atoms in the patient. Radiowaves are, in turn, produced by the interaction with the hydrogen atoms and are detected by the machine which converts the data into images. The scanner has no internal moving parts.

## Procedure

- This depends on indications and the part of the body examined.
- The patient lies on the scanner table. 'Coils' may be placed over the body part of interest.
- Most examinations do not involve intravenous contrast being given. If this is given, contrast containing gadolinium (Gd) is usually used.
  - This is usually hand-injected immediately before the scan.
- Depending on the part of the body examined, the patient may be asked to hold their breath via speakers in the machine.
- The scan itself can last up to 40–50 minutes for some body parts.

### Risks

- ▶ *Nephrogenic systemic fibrosis (NSF):* linked to gadolinium exposure in 2006. Symptoms may begin up to 3 months from exposure and may include pain, swelling, erythema, fibrosis of internal organs, and death. Patients with renal impairment are at greatest risk (no cases recorded in those with GFR >60) and at least 9 hours of haemodialysis is required to remove it from the bloodstream. See latest guidance at ✆ http://www.rcr.ac.uk.
- *Metallic artefacts:* twisting or movement of artefacts within the body.
- *Biological effects:* the magnetic fields employed may induce voltages within the body. The most common effect is 'magnetophosphenes' or visual flashes seen by the patient as the optic nerve is stimulated. Stimulation of other nerves and muscles may occur.
- *Tissue burns:* may occur if conducting loops (e.g. ECG leads) are in contact with skin.
- *Temperature:* the oscillating voltages create tissue heating. Overall body temperature may rise by 0.3°C.
- *Noise:* may reach up to 95dB. Headphones or earplugs are usually worn.
- *Claustrophobia:* experienced by up to 10% of patients.



**Fig. 20.2** A typical MRI scanner. Note the absence of metal in the room (oxygen cylinder, etc.). A 'coil' is shown within the scanner.

# Barium swallow and meal

Barium swallows examine the oropharynx, oesophagus, and gastro-oesophageal junction; barium meals examine the stomach and first part of the duodenum. Swallows and meals are usually performed together as described here.

## Indications

- Investigation of oesophageal and gastric pathology. Indications include dysphagia, odynophagia, dyspepsia, weight loss, anaemia, epigastric mass, partial obstruction.
- ▶ Always consider alternatives (e.g. OGD, MRI).

## Contraindicitions

- *Absolute:* lack of informed consent, complete bowel obstruction, suspected perforation (a water-soluble contrast may be used instead).
- *Relative:* a large degree of patient cooperation is required so those unable to understand or follow instructions are unsuitable. Also, the patient must be able to stand for the duration of the examination and to lie supine if necessary.

## Procedure

The patient drinks barium whilst the oesophagus and stomach are imaged fluoroscopically. Usually performed by a radiologist.

- The patient stands in the fluoroscopy machine (Fig. 20.3).
- A gas-producing agent is ingested (e.g. Carbex®) and the patient is asked not to belch.
- Images are taken as the patient swallows mouthfuls of barium. The patient must be able to hold the liquid in their mouth and swallow on command.
- Once views of the oesophagus have been obtained, the machine is tilted so the patient is supine. The patient is instructed to roll and tilt as images of the stomach are obtained from several angles.
  - ⚠ This requires a certain degree of patient fitness.
- The time taken depends to a degree on how easily the patient follows the commands, although usually lasts 15–20 minutes.
- After the procedure, the patient may eat and drink as usual but is advised to open their bowel regularly to avoid barium impaction.

## Risks

- Leakage of barium through an unsuspected perforation.
  - Intraperitoneal and intramediastinal barium has a significant mortality rate.
- Barium impaction (causing large bowel obstruction) or barium appendicitis.

## Other information

- ▶ A barium study will prevent a CT examination of the same area for a period of time as intestinal barium creates dense streak artefact.

**Fig. 20.3** A typical fluoroscopy room set up for an upper gastrointestinal barium examination.

## Patient preparation

- *Fasting:* nil by mouth for 6 hours before the examination.
- *Bowel preparation:* none required.
- *Smoking:* patients are asked not to smoke for 6 hours before the procedure as this increases gastric motility.

## Water-soluble contrast examinations

- In the case of recent surgery, suspected perforation, or investigation of a leak, water-based iodinated contrast is used instead of barium. Examples include Gastrograffin, Urograffin, Niopam, and Omnipaque.
- A single-contrast examination is performed (i.e. the gas-producing agent is not given) and many of the 'standard' views are not included.
- In contrast to the barium examinations, these studies can be carried out on patients who are frail and/or have recently had surgery.
- Intraperitoneal or intramediastinal water-soluble contrast does not carry the risks of barium but aspiration of the contrast can result in pulmonary oedema and lung fibrosis. Hypersensitivity is also a risk.

# Barium follow-through

### Indications

- Investigation of small bowel pathology, particularly suspected Crohn's disease and strictures. Indications include pain, diarrhoea, malabsorption, partial obstruction, and anaemia.
- ▶ Always consider alternatives (e.g. MRI, small bowel enema).

### Contraindications

- Absolute: lack of informed consent, complete small bowel obstruction, suspected perforation (a water-soluble contrast may be used instead).

### Procedure

The patient drinks barium and the small bowel is intermittently imaged until the barium has reached the caecum. Usually performed by a radiologist or senior radiographer.

- The patient is given a mixture of barium to drink.
- The exact mixture given to the patient varies between centres and between radiologists. Some add Gastrograffin to the barium, which has been shown to reduce transit time. Many add 20mg of metoclopramide to the mixture which enhances gastric emptying.
- Once the barium has been consumed, the patient is asked into the fluoroscopy room and images are taken of the small bowel with the patient lying supine (Fig. 20.4).
- Real-time fluoroscopy is employed to assess small bowel motility.
- Images are taken every 20–30 minutes until the barium has reached the colon.
- The radiologist may use a plastic 'spoon' or similar radio-lucent device to press on the patient's abdomen to separate loops of bowel.
- Additional images of the terminal ileum are usually obtained, often with the patient supine, and many radiologists also acquire an 'overcouch' plain abdominal radiograph with compression applied to the lower abdomen.
- The time taken depends on the small bowel transit time and, although usually an hour, patients are advised to allow up to 3 hours for the appointment.
- After the procedure, the patient may eat and drink as usual but is advised to keep their bowel moving to avoid barium impaction.

### Risks

- Leakage of barium through an unsuspected perforation.
  - Intraperitoneal barium causes hypovolaemic shock and has a 50% mortality rate. Of those that survive, 30% have adhesions.
- Barium impaction (causing large bowel obstruction) or barium appendicitis.
- Medication effects (see 'other information').

**Fig. 20.4** A typical fluoroscopy room set up for an upper gastrointestinal barium examination.

## Patient preparation
- Fasting: nil by mouth for 12 hours before the examination.
- Bowel preparation: laxative (usually Picolax® or similar) taken 12 hours before.

## Other information
- Metoclopramide aids gastric emptying. Extra-pyramidal side effects may occur, especially in young women, and there is a risk of acute dystonic reactions such as an oculogyric crisis. Contraindicated in patients with Parkinsonism/Parkinson's disease.
- ▶ A barium study will prevent a CT examination of the same area for a period of time as intestinal barium creates dense streak artefact.

# Barium enema

▶ The following refers to the standard 'double contrast' barium enema.

## Indications

- Investigation of colonic pathology. Indications include pain, melaena, anaemia, palpable mass, change in bowel habit, failed colonoscopy, and investigation of remaining colon in the case of a known colonic tumour.
- ▶ Always consider alternatives (e.g. colonoscopy, CT colonography).

## Contraindications

- Absolute: lack of informed consent, possible perforation, pseudomembranous colitis, toxic megacolon, biopsy via rigid sigmoidoscope within 5 days, biopsy via flexible endoscope within 1 day.
- Relative: barium meal within 7–14 days, patient frailty or immobility.
  - ▶ The procedure requires a large amount of patient cooperation. The patient must be able to lie flat and to turn over easily
  - ▶ The patient must be able to retain rectal barium and air.

## Procedure

The colon is coated with barium, then inflated with air and images are taken from several different angles. Performed by a radiographer or radiologist.

- The patient lies in the left lateral position on the fluoroscopy table (Fig. 20.5).
- The operator may perform a digital rectal examination before starting.
- A rectal tube is placed, attached to a bag of barium sulphate. The barium is run into the colon under x-ray guidance until it reaches the right colon.
- The barium is drained.
- Intravenous buscopan or, if contraindicated, glucagon is given.
- The colon is inflated with air (or with $CO_2$ in some centres).
- The patient is instructed to roll and is tilted as images are acquired.
- Once the images are obtained, the colon is deflated and the patient can go to the bathroom to empty their bowel and shower if necessary.
- The examination may last 15–30 minutes.
- The patient should be kept in the department until any medication side effects (e.g. blurred vision) have worn off.

## Risks

- Perforation (increased risk in elderly, ulcerating lesions, systemic steroids, hypothyroidism, large bowel obstruction).
  - Intraperitoneal barium causes hypovolaemic shock and has a 50% mortality rate. Of those that survive, 30% have adhesions.
- Cardiac arrhythmia (secondary to the large bowel distension).
- Medication effects (see 'other information').

**Fig. 20.5** A typical fluoroscopy room set up for a barium enema examination.

## Patient preparation
- *Iron tablets:* stop 5 days before.
- *Constipating agents:* stop 2 days before.
- *Fasting:* low residue diet 2 days before, fluids only on the day before.
- *Bowel preparation:* laxative (usually Picolax®) taken at 08:00 and 18:00 on the day before.

## Other information
- Buscopan is given to inhibit intestinal motility. Side effects include blurred vision, dry mouth, and tachycardia.
  - Contraindicated in angina, untreated closed angle glaucoma, prostatic hypertrophy, myasthenia gravis, paralytic ileus, pyloric stenosis
  - Glucagon is given if buscopan cannot be given. Risk of hypersensitivity and is contraindicated in phaeochromocytoma, insulinoma and glucagonoma.
- After the procedure, the patient may eat and drink as usual but is advised to keep their bowel moving to avoid barium impaction.
- ► A barium study will prevent a CT examination of the same area for a period of time as intestinal barium creates dense streak artefact.

## Water-soluble contrast examinations
- In the case of recent surgery, suspected perforation, or investigation of a leak, water-based iodinated contrast is used instead of barium. Examples include Gastrograffin, Urograffin, Niopam, and Omnipaque.
- A single-contrast examination is performed (i.e. the colon is not inflated with air) and many of the 'standard' views are not included.
- No bowel preparation or fasting is needed.

# Endoscopic retrograde cholangiopancreatography (ERCP)

## Indications

- *Diagnostic:* largely superseded by safer modalities such as endoscopic ultrasound and MRI/MRCP. Diagnostic indications include sphincter of Oddi dysfunction and primary sclerosing cholangitis.
- *Therapeutic:* endoscopic sphincterotomy (biliary and pancreatic), removal of stones, dilation of strictures (e.g. PSC), stent placement.

## Contraindications

- Lack of informed consent, uncooperative patient, recent attack of pancreatitis, recent MI, history of contrast dye anaphylaxis, severe cardiopulmonary disease, futility (anticipated short-term survival with no features of sepsis).

## Procedure

An ERCP involves the passage of an endoscope into the duodenum. The endoscopist injects contrast medium through the ampulla of Vater via a catheter. Real-time fluoroscopy is used to visualize the pancreas and biliary tree. Selected images are taken.

- Dentures (if present) are removed.
- Patient is given anaesthetic throat spray (lidocaine) and sometimes intravenous sedation/analgesia (e.g. midazolam, pethidine).
- Patient lies on the couch in a modified left lateral ('swimmers') position with the left arm adducted and the right abducted. The endoscope is inserted as for OGD.
- Under x-ray guidance, a polyethylene catheter is inserted into the biliary tree and contrast instilled to outline the pancreatic duct as well as the common bile duct and its tributaries.
- Procedure time varies from 30–90 minutes.

## Risks

- Pancreatitis (2–9% of procedures of which 10% of cases are mild–moderate). Serum amylase is temporarily raised in 70%.
- Infection (ascending cholangitis, acute cholecystitis, infected pancreatic pseudocyst, liver abscess, endocarditis).
- Bleeding, perforation of the oesophagus, duodenum, bile ducts.
- Failure of gallstone retrieval.
- Prolonged pancreatic stenting associated with stent occlusion, pancreatic duct obstruction, pseudocyst formation.
- Basket impaction around a large gallstone (may require surgery).

## Patient preparation

- *Blood tests:* Liver enzymes, platelets, and clotting are checked prior to the procedure.
- *Fasting:* 4 hours except in the case of an emergency.
- *Antibiotic prophylaxis:* recommended for:
  - Patients in whom biliary decompression is unlikely to be achieved at a single procedure (e.g. dilatation of dominant stricture in multifocal sclerosing cholangitis or hilar cholangiocarcinoma)
  - Consider also in patients with severe neutropenia ($<0.5 \times 10^9/L$) and/or profound immunocompromise.

## Other information

- ⚠ Intravenous sedation and analgesia is usually administered and the back of the throat is sprayed with local anaesthetic.
- Hilar biliary obstruction demonstrated on MR or CT imaging may be more successfully stented using percutaneous transhepatic cholangiography (PTC) than ERCP.
- Equipment allowing direct cholangioscopy (with the potential for sampling lesions) is becoming more widely available.

# Ultrasound

### Indications
- Indications are manifold and too numerous to list. See 'making best use of a department of clinical radiology' via ☞ http://www.rcr.ac.uk

### Contraindications
- For some examinations, the patient must be able to cooperate with the operator and a degree of mobility is often required.
- Ultrasound becomes increasingly less diagnostic at greater depths. Images of deeper structures in large individuals are often unobtainable and this should be borne in mind when considering who to refer.

### Technology
- The ultrasound probe houses a piezoelectric crystal which both projects and receives high-frequency sound waves. Much like radar, the 'echoes' are converted to images by the machine's software.
- Ultrasound cannot image through gas and requires a semi-liquid 'gel' between the probe and skin surface for optimum imaging.
- A typical ultrasound machine is shown in Fig. 20.6.

### Procedure
- This depends on the part of the body examined and the indications.
- Time taken will vary depending on part of body examined, patient cooperation, and complexity of the findings. Most examinations last between 5–20 minutes.

### Risks
- ▶ There is no published evidence that ultrasound has ever directly caused harm to a patient.
  - The acoustic output of modern machines, however, is much greater than previously used.
- *Heating:* some equipment can produce temperature rises of 4°C in bone. Most equipment in clinical use is unlikely to increase tissue temperature more than the 1.5°C which is considered 'safe'.
- *Non-thermal hazard:* ultrasound has been demonstrated to produce tiny gas pockets and bubbles in animal models. Neonatal lung is considered vulnerable to this but there is no evidence that diagnostic ultrasound can cause harm to other tissues.
  - Machines have a 'mechanical index' (MI) displayed on screen which acts as a guide to the operator.

### Patient preparation
Depends on the indication and body part being examined.
- *Abdomen:* patients are usually asked to fast for 6 hours prior to the examination. This ensures distension of the gallbladder and prevents the epigastric structures being obscured by overlying bowel gas.
- *Renal tract/pelvis:* a full bladder is usually required. A full bladder creates an 'acoustic window', effectively pushing small bowel aside so that deeper structures (e.g. ovaries) may be seen.

**Fig. 20.6** A typical ultrasound room.

# Oesophagogastroduodenoscopy (OGD)

### Indications

- *Diagnostic:* haematemesis, dyspepsia (>55 years old), oesophageal and gastric biopsies (malignancy?), duodenal biopsies (coeliac?), surveillance (e.g. Barrett's oesophagus), persistent nausea and vomiting, iron-deficiency anaemia, dysphagia.
- *Therapeutic:* treatment of bleeding lesions, variceal banding and sclerotherapy, stricture dilatation, polypectomy, EMR, palliative intent (e.g. stent insertion, laser therapy), argon plasma coagulation for suspected vascular lesions.

### Contraindications

- *Absolute:* lack of informed consent, possible perforation, haemodynamic instability, hypoxaemia with respiratory distress, uncooperative patient.
- *Relative:* pharyngeal diverticulum, recent myocardial infarction, or pulmonary embolus.

### Procedure

- Endoscopic examination of the mucosa of the oesophagus, stomach, and proximal duodenum. Allows direct visualization, mucosal biopsies, and other therapeutic procedures.
- Dentures (if present) are removed.
- Patient is given anaesthetic throat spray (lidocaine) +/− intravenous sedation (e.g. midazolam).
- Patient lies on the couch in the left lateral position.
- Hollow mouthpiece is inserted to protect the patient's teeth and facilitate instrument passage.
- Endoscope (9.5–12.5mm diameter, max 120cm long) is slowly advanced and 'swallowed' by the patient (Fig. 20.7 shows a typical scope).
- Scope advanced and manipulated by the endoscopist to allow visualization of the target structures.
- Procedure time varies but averages 5–15 minutes.

### Risks

- Minor throat and abdominal discomfort.
- Cardiorespiratory: arrhythmias, MI, respiratory arrest, shock, death.
- Infection (uncommon, e.g. aspiration pneumonia).
- Perforation (around 0.03% with a mortality of 0.001% during diagnostic procedures, higher with therapeutic procedures).
  - Overall 2–3% perforation with oesophageal dilatation; mortality 1%.
- Bleeding (caution with low platelet counts and high INR).
- Medication effects including anaphylactic reactions and over-sedation.
- Dental trauma.

**Fig. 20.7** A typical gastroscope.

## Patient preparation

- *Fasting:* 4 hours prior to the procedure unless in an emergency situation.
- *Antibiotic prophylaxis:* none for OGD. See other topics for comparison.

## Other information

- Dosages of benzodiazepines and opiates should be kept to a minimum to achieve sedation, with lower doses being prescribed in elderly patients.
- The pharynx is sprayed with local anaesthetic spray. There is some evidence that the combination use of local anaesthetic spray and intravenous sedation increases the risk of aspiration pneumonia.
- 🛈 Patients who have had intravenous sedation should not drive, operate heavy machinery, or drink alcohol for 24 hours afterwards.

# Colonoscopy

### Indications

- *Diagnostic:* gastrointestinal bleeding, iron-deficiency anaemia, chronic diarrhoea, lower abdominal symptoms (chronic constipation, lower abdominal pain, bloating), evaluation of known IBD, surveillance for cancer (in IBD patients/after colonic polypectomy/after curative intent resection of colorectal cancer), screening for colorectal cancer.
- *Therapeutic:* polypectomy (including endoscopic mucosal resection techniques: EMR), angiodysplasia treated with argon plasma coagulation (APC), decompression of volvulus or pseudo-obstruction, dilatation or stenting of strictures or malignant colonic obstruction.

### Contraindications

- *Absolute:* lack of informed consent, toxic megacolon, fulminant colitis, colonic perforation.
- *Relative:* acute diverticulitis, symptomatic large abdominal aortic aneurysm, immediately post-op, recent myocardial infarction or pulmonary embolus, severe coagulopathies.
  - Colonoscopy can be performed safely in pregnancy but should be deferred in most instances unless requiring immediate resolution.

### Procedure

Colonoscopy is an endoscopic examination of the mucosal surface from the anal canal to the terminal ileum.

- Patient lies on the couch in the left lateral position with knees bent.
- Endoscopist first performs a digital rectal examination.
- Sedation (e.g. midazolam) may be given with monitoring of oxygen saturation. Intravenous analgesia (e.g. pethidine) is also given.
  - Increasing use of either no sedation (with improved techniques such as 'Scopeguide®') or inhaled nitric oxide.
- Lubricated colonoscope (about 12mm wide and 185cm long) is passed rectally. Air is insufflated. Water-jet may also be used via the scope.
  - Figure 20.8 shows a typical scope.
- Aim is to pass to the terminal ileum.
- Duration varies but averages at about 20 minutes.

### Risks

- Perforation (0.2–0.4% diagnostic; higher with therapeutic procedures).
- Bleeding (1 in 1000).
- Abdominal distension, medication effects (allergic reactions, nausea, vomiting hypotension, respiratory depression).
- Rarities: infection, post-polypectomy coagulation syndrome: pain, peritoneal irritation, leukocytosis and fever, splenic rupture, small bowel obstruction.

**Fig. 20.8** A typical colonoscope.

## Patient preparation

- *Iron and constipating agents:* discontinue iron tablets 7 days and constipating agents 4 days prior to the procedure.
- *Anticoagulant and antiplatelet therapy:* in the case of a planned polypectomy or other therapeutic procedure, refer to BSG guidelines on the management of anticoagulant and antiplatelet therapy: ℜ http://www.bsg.org.uk.
- *Antibiotic prophylaxis:* none for colonoscopy. See other topics for comparison.
- *Bowel preparation:* the colon must be empty. Protocols vary but usually include prescribing 1 sachet of sodium picosulphate (Picolax®) for the morning and afternoon of the day before procedure.

## Other information

- The introduction of the bowel cancer screening programme has meant that endoscopists need to pass a 'driving test' to demonstrate high-level competency to perform safe screening colonoscopy.
- Endoscopic mucosal resection (EMR) is used for larger or difficult flat polyps. The lesion is lifted by submucosal injection of gelofusin, adrenaline, and dye followed by snare resection. Polyps can then be retrieved by 'Roth' baskets for histological assessment.

# Capsule endoscopy

## Indications
- Obscure gastrointestinal bleeding (in patients with negative gastroscopy and ileocolonoscopy), known or suspected small bowel Crohn's disease, assessment of coeliac disease, screening and surveillance for polyps in familial polyposis syndromes.

## Contraindications
- Lack of informed consent, intestinal strictures, adhesions, obstruction.
- Diverticula or fistulae that may block the passage of capsule endoscope.
- Cardiac pacemakers or other implanted electronic devices.
- Difficulty in swallowing tablets or known swallowing disorders.
- Pregnancy (lack of available safety data).
- ▶ Patients with obstructive symptoms or known or suspected inflammatory bowel disease should have either a small bowel follow through or a patency capsule (dissolves after 36 hours), with an abdominal radiograph taken 24 hours post ingestion to identify whether capsule is retained within small bowel.
  - If retained, capsule endoscopy is not appropriate
  - ▶ Capsule retention can occur even in the absence of strictures on barium or MR-enteroclysis study.

## Procedure
- The capsule (Fig. 20.9) consists of a disposable, wireless, miniature video camera which can be swallowed and passes through the intestine by peristalsis.
- Images taken by the capsule are transmitted, via sensors secured to the abdominal wall, to a battery-powered data recorder worn on a belt.
- The capsule leaves the stomach within 30 mins and the patient is allowed to drink after 2 hours and eat after 4 hours.
- The external equipment (Fig. 20.10) is removed after 8 hours (approximate battery life) by which time the capsule has reached the caecum in 85% of patients.
- The capsule is expelled naturally after 24–48 hours in the patient's stool and does not need to be collected.
- Data from the recorder is downloaded onto a computer workstation which allows approximately 50,000 images to be viewed as a video.

## Risks
- Capsule retention (may cause partial or complete intestinal obstruction; highest risk in patients with extensive small bowel Crohn's disease, chronic usage of NSAIDs, abdominal radiation injury, previous major abdominal surgery, or small bowel resection).
- Capsule endoscopy may also fail in patients with dysphagia, gastroparesis, and anatomical abnormalities of the gastrointestinal tract.
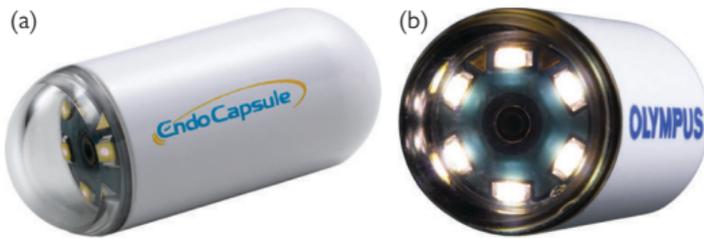
**Fig. 20.9** Examples of a typical capsule endoscope. It is shaped to be easy to swallow and has its own light-source.



**Fig. 20.10** The external equipment which the patient will wear, consisting of a data-recorder and electrodes.

## Patient preparation

- *Iron supplements:* stop taking 1 week prior to procedure.
- *Constipating agents:* stop 4 days before the procedure.
- *Fasting:* patients are fasted for 8–12 hours prior to the procedure and may receive bowel prep (taken day before procedure).

## Other information

- Incomplete examination in 10–25% of cases.
  - Presence of dark intestinal contents in distal small bowel may impair visualization of mucosa
  - Delayed gastric emptying and small bowel transit can lead to exhaustion of battery life before capsule reaches ileocaecal valve.
- Capsules are being developed to screen for oesophageal varices and may be more 'guided' in future as the technology develops.
- Positive findings on capsule endoscopy may be reachable using either single- or double-balloon enteroscopy or spiral enteroscopy.

# Exercise tolerance test (ETT)

## Indications
- Assessment of chest pain in those with known coronary artery disease (there is no longer a role for ETT in patients presenting with chest pain who do not have a history of coronary artery disease).
- Assessment of haemodynamic response in those with known valvular disease who are asymptomatic.
- Diagnosis of exertionally induced arrhythmias or syncope.

## Contraindications
- Any undiagnosed or previously unknown murmur (patient should undergo echocardiogram first).
- Severe aortic stenosis (risk of syncope).
- Hypertrophic cardiomyopathy with significant outflow obstruction (risk of syncope).
- Severe hyper- or hypo-tension.
- Unstable angina (should undergo coronary angiography).
- Known severe left main stem disease.
- Untreated congestive cardiac failure.
- Complete heart block.
- Aortic aneurysm.
- Acute myocarditis or pericarditis.
- Any recent pyrexial or 'flu-like' illness.

## Procedure
- ECG electrodes are put on the patient's chest and a sphygmomanometer cuff on an arm.
- The patient is asked to walk on a treadmill (see Fig. 20.11) connected to the computer whilst their ECG, BP, and heart rate are monitored. The speed and incline of the treadmill increase according to set protocols:
  - Bruce protocol: for assessment of physically fit and stable patients with suspected coronary artery disease. Seven stages starting at a 10% gradient at 1.7mph and increasing to 22% gradient and 6mph
  - Modified Bruce protocol: used in elderly patients or those who have been stabilized after a suspected episode of unstable angina. Starts at 1.7mph and 0% gradient and increases the gradient slowly to 10%.
- Termination of the test depends on the results seen.

## Risks
- Risks are those associated with exercise and include:
  - Arrhythmia, cardiac ischaemia, myocardial infarction, syncope.

## Patient preparation
- No specific preparation is required.
- Patients are asked not to eat or drink for 3 hours prior to the test.
- Comfortable clothing and shoes should be worn.

**Fig. 20.11** A typical ETT room.

## Indications for termination of procedure

- Patient requests to stop.
- *Symptoms:* fatigue, angina, dizziness, significant breathlessness.
- *Signs:* drop in oxygen saturations <94%, target heart rate achieved, hypotension during exercise (e.g. BP <100mmHg), significant hypertension (e.g. BP >200mmHg).
- *ECG:* any atrial or ventricular arrhythmia, frequent ventricular ectopics, new AV or bundle branch block, ST segment shift >1mm.

## Causes of false positive results or low specificity

- Often due to difficulty interpreting results as result of resting ST segment abnormalities:
  - Wolff–Parkinson–White syndrome, LBBB, atrial fibrillation, left ventricular hypertrophy, digoxin therapy, hyperventilation, biochemical electrolyte abnormalities (e.g. hypo- or hyperkalaemia), cardiomyopathies, LV outflow obstruction.
- Beta-blocker therapy prevents the appropriate heart rate/blood pressure response during testing.

# Echocardiography

## Indications

- Myocardial infarction: assess wall motion and left ventricular function.
- Valvular heart disease: assess competency and examine prostheses.
- Embolic stroke: to exclude a cardiac embolic source.
- Infective endocarditis: look for valvular vegetations.
- Cardiomyopathy: assess ventricular dilatation/hypertrophy and function.
- Congenital heart disease.
- Pericardial disease.
- Pericardial effusion: distribution of fluid and suitability for drainage.
- Aortic disease: severity and site of aneurysm, dissection, or coarctation.

## Contraindications

- The only contraindication is lack of patient consent or if the patient is unable to cooperate.

## Technology

- Echocardiography is an ultrasound examination and uses the same technology (and machines) as general ultrasound (Fig. 20.12).
- Ultrasound becomes increasingly less diagnostic at greater depths and cannot see through lung. Images in large individuals are often suboptimal and the heart may not be seen at all in patients with hyperinflated lungs.
- See Box 20.1 for other types.

## Procedure

- Time taken will vary depending on examinations performed and complexity of the findings.
- Most examinations last between 20–25 minutes.
- With the patient lying on their left side, the operator uses a hand-held probe coated with gel to examine the heart usually via the anterior chest and epigastrium.

## Risks

- ▶ There is no published evidence that ultrasound has ever directly caused any harm to a patient.
- *Heating:* some equipment can produce temperature rises of 4°C in bone. Most equipment in clinical use is unlikely to increase tissue temperature more than the 1.5°C which is considered 'safe'.
- *Non-thermal hazard:* ultrasound has been demonstrated to produce tiny gas pockets and bubbles in animal models but there is no evidence that diagnostic ultrasound can cause harm to tissues other than neonatal lung.

## Patient preparation

- No preparation is required.

**Fig. 20.12** A typical echocardiography room.

**Box 20.1  Other types of echocardiography**

Along with 2-dimensional trans-thoracic echocardiography, the following methods exist:

- *3D:* uses computer software to produce a 3-dimensional image. Useful in left-ventricular functional assessment especially post-infarction
- *4D:* 3D imaging with real-time movement captured
- *TOE:* trans-oesophageal echo is an invasive procedure. It requires written consent and is performed under sedation with local anaesthetic spray to the upper pharynx. The probe is covered, lubricated, and passed into the oesophagus behind the heart. It is used to visualize the posterior cardiac structures. The investigation of choice for infective endocarditis
- *Stress echo:* Used to assess myocardial ischaemia at 'rest' and during 'stress'. Stress is induced by exercise or (more commonly) by an intravenous infusion of dobutamine in a controlled environment
- *Bubble studies:* Used to assess for intra-cardiac shunts such as atrial or ventricular septal defects or patent foramen ovale. Air bubbles are agitated in a syringe and injected into a peripheral vein. The Valsalva manoeuvre is performed and, if a shunt exists, bubbles will be seen moving from the right side of the heart to the left.

# Coronary angiography and angioplasty

### Indications
- *Diagnostic:* unstable or refractory angina, acute coronary syndrome, positive or inconclusive stress testing.
- *Emergency therapeutic:* where possible, patients presenting with acute ST-elevation myocardial infarction should have primary coronary intervention rather than thrombolysis.
- *Elective therapeutic:* suitable 'target lesion' identified on diagnostic coronary angiogram.

### Contraindications
- *Absolute:* refusal of patient consent.
- *Relative:* acute renal failure, pulmonary oedema, known radiographic contrast allergy, uncontrolled hypertension, active GI haemorrhage, acute stroke, and untreated coagulopathy.

### Procedure
- A typical cardiac interventional suite is shown in Fig. 20.13.
- Percutaneous access via a guide needle into a peripheral artery (most commonly the radial artery).
- Guide catheter is introduced, the tip is placed at the coronary ostium, radio-opaque contrast is injected, and real-time x-ray is used to visualize the blood flow through the coronary arteries.
- The coronary guidewire is inserted through the catheter into the coronary artery using x-ray guidance.
- The guidewire tip is passed across the site of stenosis.
- The balloon catheter is passed over the guidewire until the deflated balloon lies across the target lesion.
- The balloon is then inflated and compresses the plaque and stretches the artery wall. A stent (wire mesh tube) can be inserted using a similar technique and be left in place maintaining the arterial lumen.
- The guidewire, catheter, and sheath are carefully removed.
- The patient should remain supine for 4 hours following the procedure unless an arterial closure device has been used.

### Risks
- *Minor:* contrast allergy, vasovagal reaction, haemorrhage and haematoma at puncture site, thrombosis formation, false aneurysm, arteriovenous fistulation, pulmonary oedema, and renal failure due to contrast nephropathy.
- *Major:* limb ischaemia, coronary artery dissection, aortic dissection, ventricular perforation, air or atheroma embolism, ventricular arrhythmias, failure of procedure and need to proceed to CABG.
- Death (<1 in 1000).

**Fig. 20.13** A typical cardiac interventional suite.

## Patient preparation

- Pre-procedure checklist: written consent, group and save, ECG, check FBC/clotting/urea and electrolytes.

## Other information

- Coronary angioplasty is associated with increased thrombus formation (balloon inflation disrupts the intima, revealing pro-thrombotic cores of plaques), therefore antiplatelet therapy is necessary.
- Patients will need to have long-term antiplatelet therapy; usually lifelong aspirin 75mg od, but they will also need clopidogrel 75mg od (see local guidelines: usually 3 months for bare metal stents and 12 months for drug-eluting stents or angioplasty after acute coronary syndrome).
- Patients with renal failure should be carefully considered. Iodinated contrast can be nephrotoxic and renal decompensation may occur following coronary angiography/plasty. The risk can be minimized by hydration before and after the procedure. Renal function should be carefully monitored. Check local guidelines.

# Bronchoscopy

### Indications

- *Diagnostic:* histology/cytology in suspected lung malignancy, sample mediastinal lymphadenopathy, alveolar lavage (e.g. tuberculosis), transbronchial biopsy (e.g. diffuse lung disease).
- *Therapeutic:* placement of guidewire for local radiotherapy, direct treatment (e.g. diathermy to strictures).
  - Placement of endobronchial stents and the removal of foreign bodies are usually accomplished at rigid bronchoscopy under GA.

### Contraindications

- *Absolute:* cardiovascular instability, life-threatening arrhythmia, severe hypoxaemia, respiratory failure with hypercapnia (unless intubated/ ventilated).
  - *Rigid bronchoscopy contraindications:* unstable neck, severely ankylosed cervical spine, severely restricted temporomandibular joints.
- *Relative:* uncooperative patient, recent myocardial infarction, tracheal obstruction, un-correctable coagulopathy.
  - Transbronchial biopsy with caution in uraemia, SVCO, pulmonary hypertension (risk of bleeding).

### Procedure

Bronchoscopy is an endoscopic examination of the bronchial tree.

- Patient sits on the couch, leaning back comfortably.
- Sedation (e.g. midazolam) may be given with monitoring of oxygen saturation. Atropine may also be given to decrease secretions.
- Pharynx is anaesthetized with aerosolized lidocaine.
- Lubricated bronchoscope (about 6mm wide and 60cm long) is passed nasally or orally with use of a bite-block (Fig. 20.14).
- Brushings, biopsy, or lavage (50–100ml saline) may be performed.
- Duration varies but averages about 20–30 minutes.

### Risks

- Bleeding from a biopsy site and transient fever (10–15%).
- Medication effects: respiratory depression, hypotension, arrhythmias.
- Topical anaesthesia: laryngospasm, bronchospasm, seizures, arrhythmias.
- Minor laryngeal oedema or injury with hoarseness, hypoxaemia in patients with compromised gas exchange (1–10%).
- Mortality is 1–4 in 10,000 patients.
- Transbronchial biopsy: pneumothorax (2–5%), significant haemorrhage (1%); death (12 in 10,000).

**Fig. 20.14** A typical bronchoscope.

## Patient preparation

- *Anticoagulant and antiplatelet therapy:* stop for 3 days. Clopidogrel should be stopped for 5 days.
- *Blood tests:* check clotting and full blood count.
- *Spirometry:* perform if underlying lung disease.
- *Fasting:* nil by mouth 2 hours before the procedure, no solids 4–6 hours before procedure.

## Post-procedure

- *Oxygen:* supplemental oxygen for up to 1 hour.
- *Eating/drinking:* drink after 1 hour. If no problems, can eat.
- *Chest radiography:* only if dyspnoea or chest pain following biopsy (10% risk of pneumothorax).
- *Driving:* if had midazolam or similar, not to drive or operate heavy machinery for the rest of the day.

# Index