Hands and skin

- Look for signs of tobacco staining (Fig. 7.8).
- Look for peripheral cyanosis.
- Feel the temperature.
- Check for clubbing (p. 49).
- Look at the nails for splinter haemorrhages (linear, reddish-brown marks along the axis of the finger and toenails, thought to be due to circulating immune complexes.
- Look at the palmar aspect of the hands for:
  - Janeway lesions
- Janeway lesions – painless red spots, which blanch on pressure, on the thenar/hypothenar eminences of the palms, and soles of the feet.
- Osler’s nodes – painful raised erythematous lesions which are rare but found most often on the pads of the fingers and toes.

- Look at the palmar and extensor surfaces of the hands for xanthomata (yellow skin or tendon nodules from lipid deposits)
- Look at the entire skin surface for petechiae
- Normal findings
- The hands usually feel dry at ambient temperature. Peripheral cyanosis (p. 45) is common in healthy patients when the hands are cold. One or two isolated splinter haemorrhages are common in healthy individuals from trauma.
Abnormal findings

- Fever is a feature of infective endocarditis and pericarditis and may occur after myocardial infarction. With autonomic stimulation the hands may feel warm and sweaty; with hypotension and shock they may be cold and clammy.

- Splinter haemorrhages are found in infective endocarditis and some vasculitic disorders.

- A petechial rash (caused by vasculitis), most often present on the legs and conjunctivae (Fig. 6.6), is a transient finding in endocarditis and can be confused with the rash of meningococcal disease (Fig. 17.2A). Janeway lesions, Osler’s nodes, nail fold infarcts and finger clubbing are uncommon features of endocarditis (Ch. 3 and Fig. 6.6).
face

- Look:
  - in the mouth for central cyanosis.
  - at the eyelids for xanthelasmata (soft yellowish plaques periorbitally and on the medial aspect of the eyelids associated with hyperlipidaemia).
  - at the iris for a corneal arcus.
  - at the conjunctivae for petechiae.
- Examine the fundi (p. 291) for features of hypertension (Fig. 6.16), diabetes and Roth’s spots (flame-shaped retinal haemorrhages with a ‘cotton-wool’ centre).
Central cyanosis : HF, CHD

Xanthelasmata

(also check the patellar and Achilles tendons for xanthomata (Fig. 6.8B).)

Corneal arcus is a creamy yellow discoloration at the boundary of the iris and cornea caused by cholesterol deposition
Both xanthelasmata and corneal arcus can, however, occur in normolipidaemic patients (Box 6.12).

Roth’s spots (Fig. 6.6) are caused by a similar mechanism to splinter haemorrhages and can also occur in anaemia or leukaemia.
The pulse waveform depends upon heart rate, stroke volume, left ventricular outflow obstruction, arterial elasticity and peripheral resistance.

Use the larger (brachial, carotid or femoral) pulses to assess the pulse volume and character (Box 6.13). When taking a pulse, assess:

- Rate
- Rhythm
- Volume
- Character
Record individual pulses as:

- Normal +
- Reduced ±
- Absent −
- Aneurysmal + +

If you are in any doubt about whose pulse you are feeling, palpate your own pulse at the same time. If it is not synchronous with yours, it is the patient’s.
Radial pulse

- Place the pads of your index and middle fingers over the right radial artery.
- Assess rate, and rhythm
- Count the pulse rate over 30 seconds; multiply by 2 to obtain the beats per minute (bpm).
- To detect a collapsing pulse: first, check that the patient has no shoulder or arm pain or restriction on movement. Feel the pulse with the base of your fingers, then raise the patient’s arm vertically above the patient’s head (Fig. 6.10B).
- Palpate both radial pulses simultaneously, assessing any delay between the two, and any difference in pulse volume.
- Palpate the radial and femoral pulses simultaneously, again noting any timing and volume differences.
Brachial pulse

- Use your index and middle fingers to palpate this over the lower end of the humerus just above the elbow joint. Assess the character and volume.
Carotid pulse

- Some clinicians consider routine examination of the carotid pulse is inappropriate because it may cause distal vascular events, e.g. transient ischaemic attack, or induce reflex, vagally mediated bradycardia. In assessing a patient who may have had a cardiac arrest, however, it is the pulse of choice.

- If you do examine the carotid pulse do this gently and never assess both carotids simultaneously.
Examination of the carotid pulse

- Explain what you are going to do.
- Lie the patient semirecumbent in case you induce a reflex bradycardia.
- Gently place the tips of your fingers between the larynx and the anterior border of the sternocleidomastoid muscle and feel the pulse (Fig 6.10D).
- Listen for bruits over both carotid arteries, using the diaphragm of your stethoscope during held inspiration.
Femoral pulse

- Lie the patient supine if possible and explain what you are going to do.
- Place your index and middle fingers over the femoral artery, which is just inferior to the midpoint between the anterior superior iliac spine and the pubis (Fig. 6.10E).
- Check for radiofemoral delay (coarctation of the aorta, Fig. 6.11) by simultaneously feeling the radial pulse.
- Listen for bruits over both femoral arteries, using the diaphragm of the stethoscope.
Normal!

- Rate: Assess the pulse rate in the clinical context. A pulse rate of 40 bpm can be normal in a fit young adult, whereas a pulse rate of 65 bpm may be abnormally low in acute heart failure. Resting heart rate is normally 60–90 bpm.
- Bradycardia
- Tachycardia
Rhythm Sinus rhythm originates from the sinoatrial node and produces a regular rhythm (Fig. 6.12A). It varies slightly with the respiratory cycle, mediated by the vagus nerve, and is most pronounced in children, young adults or athletes (sinus arrhythmia). During inspiration, parasympathetic tone falls and the heart rate increases; on expiration, the heart rate decreases (Box 6.14)

- Volume refers to the perceived degree of pulsation and reflects the pulse pressure.
- Character Character refers to the waveform or shape of the arterial pulse.
Abnormal!

- The most common causes of bradycardia are medication, athletic conditioning, and sinoatrial or atrioventricular node dysfunction. The most common cause of tachycardia is sinus tachycardia.
<table>
<thead>
<tr>
<th>Artery</th>
<th>Surface marking</th>
</tr>
</thead>
<tbody>
<tr>
<td>Radial</td>
<td>At the wrist, lateral to the flexor carpi radialis tendon</td>
</tr>
<tr>
<td>Brachial</td>
<td>In the antecubital fossa, medial to the biceps tendon</td>
</tr>
<tr>
<td>Carotid</td>
<td>At the angle of the jaw, anterior to the sternocleidomastoid muscle</td>
</tr>
<tr>
<td>Femoral</td>
<td>Just below the inguinal ligament, midway between the anterior superior iliac spine and the pubic symphysis (the mid inguinal point). It is immediately lateral to the femoral vein and medial to the femoral nerve</td>
</tr>
<tr>
<td>Popliteal</td>
<td>Lies posteriorly in relation to the knee joint, at the level of the knee crease, deep in the popliteal fossa</td>
</tr>
<tr>
<td>Posterior tibial</td>
<td>Located 2 cm below and posterior to the medial malleolus, where it passes beneath the flexor retinaculum between flexor digitorum longus and flexor hallucis longus</td>
</tr>
<tr>
<td>Dorsalis pedis</td>
<td>Passes lateral to the tendon of extensor hallucis longus and is best felt at the proximal extent of the groove between the first and second metatarsals. It may be absent or abnormally sited in 10% of normal subjects, sometimes being “replaced” by a palpable perforating peroneal artery</td>
</tr>
</tbody>
</table>
Rhythm The pulse may be regular or irregular. If irregular, it may be regularly irregular, due to an ectopic beat occurring at a regular interval or to second-degree atrioventricular block. Atrial fibrillation is the most common cause of an irregularly irregular pulse. The rate in atrial fibrillation depends on the number of beats conducted by the atrioventricular node. Untreated, the ventricular rate may be very fast (up to 200 bpm). The variability of the pulse rate (and therefore ventricular filling) explains why the pulse volume varies and there may be a pulse deficit, with some cycles not felt at the radial artery. Calculate the pulse deficit by counting the radial pulse rate and subtracting this from the apical heart rate assessed by auscultation.
Volume The ventricles fill during diastole. Longer diastolic intervals are associated with increased stroke volume, which is reflected by increased pulse volume on examination. This is why pulse volume and BP vary widely during atrial fibrillation, and why the ‘compensatory pause’ following a premature ectopic beat is sometimes felt by the patient.
A large pulse volume is a reflection of a large pulse pressure, which can be physiological or pathological (Box 6.18). The most common cause of a large pulse pressure is arteriosclerosis, which is seen in patients with widespread vascular disease, hypertension, and advanced age.
A low pulse volume may be due to reduced stroke volume and occurs in left ventricular failure, hypovolaemia or peripheral arterial disease.
The ventricles fill during diastole. Longer diastolic intervals are associated with increased stroke volume, which is reflected by increased pulse volume on examination. This is why pulse volume and BP vary widely during atrial fibrillation, and why the 'compensatory pause' following a premature ectopic beat is sometimes felt by the patient.

### 6.18 Causes of increased pulse volume

<table>
<thead>
<tr>
<th>Physiological</th>
<th>Pathological</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Exercise</td>
<td>• Increased environmental temperature</td>
</tr>
<tr>
<td>• Pregnancy</td>
<td></td>
</tr>
<tr>
<td>• Advanced age</td>
<td>• Aortic regurgitation</td>
</tr>
<tr>
<td></td>
<td>• Paget’s disease of bone</td>
</tr>
<tr>
<td></td>
<td>• Peripheral atrioventricular shunt</td>
</tr>
<tr>
<td>• Hypertension</td>
<td></td>
</tr>
<tr>
<td>• Fever</td>
<td></td>
</tr>
<tr>
<td>• Thyrotoxicosis</td>
<td></td>
</tr>
<tr>
<td>• Anaemia</td>
<td></td>
</tr>
</tbody>
</table>
Coarctation is a congenital narrowing of the aorta, usually distal to the left subclavian artery. The clinical signs depend on the location and severity of the narrowing and the patient’s age. In children, the upper limb pulses are usually normal with reduced volume lower limb pulses, which are delayed relative to the upper limb pulses (radio-femoral delay) (Fig. 6.11). In adults, coarctation usually presents with hypertension and heart failure.
Character

- A collapsing pulse is when the peak of the pulse wave arrives early and is followed by a rapid descent. This rapid fall imparts the ‘collapsing’ sensation. This is exaggerated by raising the patient’s arm above the level of the heart (Fig. 6.10B). It occurs in severe aortic regurgitation and is associated with wide pulse pressure (systolic BP – diastolic BP > 80 mmHg).

- A slow-rising pulse has a gradual upstroke with a reduced peak occurring late in systole, and

- is a feature of severe A.S
Pulsus bisferiens is an increased pulse with a double systolic peak separated by a distinct mid-systolic dip. Causes include aortic regurgitation, and concomitant aortic stenosis and regurgitation.
Pulsus alternans is a beat-to-beat variation in pulse volume with a normal rhythm. It is rare and occurs in advanced heart failure.

Pulsus paradoxus is an exaggeration of the normal variability of pulse volume with breathing. Pulse volume normally increases in expiration and decreases during inspiration due to intrathoracic pressure changes affecting venous return to the heart. This variability in exaggerated diastolic filling of both ventricles is impeded by increased intrapericardial pressure. This occurs in cardiac tamponade because of accumulation of pericardial fluid and in constrictive pericarditis.
**Fig. 6.18** Jugular venous pressure in a healthy subject. (A) Supine: jugular transition between distended and collapsed vein can usually be seen to pulsate. Transition point obscured.

### 6.23 Differences between carotid artery and jugular venous pulsation

<table>
<thead>
<tr>
<th>Carotid</th>
<th>Jugular</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rapid outward movement</td>
<td>Rapid inward movement</td>
</tr>
<tr>
<td>One peak per heart beat</td>
<td>Two peaks per heart beat (in sinus rhythm)</td>
</tr>
<tr>
<td>Palpable</td>
<td>Impalpable</td>
</tr>
<tr>
<td>Pulsation unaffected by pressure at the root of the neck</td>
<td>Pulsation diminished by pressure at the root of the neck</td>
</tr>
<tr>
<td>Independent of respiration</td>
<td>Height of pulsation varies with respiration</td>
</tr>
<tr>
<td>Independent of position of patient</td>
<td>Varies with position of patient</td>
</tr>
<tr>
<td>Independent of abdominal pressure</td>
<td>Rises with abdominal pressure</td>
</tr>
</tbody>
</table>
### 6.15 Causes of a fast or slow pulse

<table>
<thead>
<tr>
<th>Heart rate</th>
<th>Sinus rhythm</th>
<th>Arrhythmia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fast (tachycardia, &gt;100 bpm)</td>
<td>Exercise</td>
<td>Atrial fibrillation</td>
</tr>
<tr>
<td></td>
<td>Pain</td>
<td>Atrial flutter</td>
</tr>
<tr>
<td></td>
<td>Excitement/anxiety</td>
<td>Supraventricular tachycardia</td>
</tr>
<tr>
<td></td>
<td>Fever</td>
<td>Ventricular tachycardia</td>
</tr>
<tr>
<td></td>
<td>Hyperthyroidism</td>
<td></td>
</tr>
<tr>
<td>Medication:</td>
<td>Sympathomimetics, e.g. salbutamol</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Vasodilators</td>
<td></td>
</tr>
<tr>
<td>Slow (bradycardia, &lt;60 bpm)</td>
<td>Sleep</td>
<td>Carotid sinus</td>
</tr>
<tr>
<td></td>
<td>Athletic training</td>
<td>hypersensitivity</td>
</tr>
<tr>
<td></td>
<td>Hypothyroidism</td>
<td>Sick sinus syndrome</td>
</tr>
<tr>
<td></td>
<td>Medication:</td>
<td>Second-degree heart block</td>
</tr>
<tr>
<td></td>
<td>Beta-blockers</td>
<td>Complete heart block</td>
</tr>
<tr>
<td></td>
<td>Digoxin</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Verapamil, diltiazem</td>
<td></td>
</tr>
</tbody>
</table>

### 6.16 Causes of an irregular pulse

- Sinus arrhythmia
- Atrial extrasystoles
- Ventricular extrasystoles
- Atrial fibrillation
- Atrial flutter with variable response
- Second-degree heart block with variable response

### 6.17 Common causes of atrial fibrillation

- Hypertension
- Heart failure
- Myocardial infarction
- Thyrotoxicosis
- Alcohol-related heart disease
- Mitral valve disease
- Infection, e.g. respiratory, urinary
- Following surgery, especially cardiothoracic surgery
Blood pressure

- BP is an important guide to cardiovascular risk and provides vital information on the haemodynamic condition of acutely ill or injured patients. BP constantly varies and rises with stress, excitement and environment. ‘White-coat hypertension’ occurs in patients only when a patient is seeing a healthcare worker. Ambulatory BP measurement, using a portable device at intervals during normal daytime activity and at night, is better at determining cardiovascular risk.

- BP is measured in mmHg and recorded as systolic pressure/diastolic pressure, together with where, and how, the reading was taken, e.g. BP: 146/92 mmHg, right arm, supine.
- Rest the patient for 5 minutes.
- Always measure BP in both arms (brachial arteries); the higher of the two is closest to central aortic pressure and should be used to determine treatment.
- With the patient seated or lying down, support the patient’s arm comfortably at about heart level, with no tight clothing constricting the upper arm. You can measure over thin clothing, as it makes no difference to the result.
- The usual sphygmomanometer cuff has a bladder width of 12.5 cm and length of 30–35 cm. Apply the cuff to the upper arm, with the centre of the bladder over the brachial artery.
- Palpate the brachial pulse. and
- Inflate the cuff until the pulse is impalpable. Note the pressure on the manometer; this is a rough estimate of systolic pressure.
- Inflate the cuff another 30 mmHg and listen through the diaphragm of the stethoscope placed over the brachial artery.
- Deflate the cuff slowly (2–3 mmHg/s) until you hear a regular tapping sound (phase 1 Korotkoff sounds). Record the reading to the nearest 2 mmHg. This is the systolic pressure.
- Continue to deflate the cuff slowly until the sounds disappear.
- Record the pressure at which the sounds completely disappear as the diastolic pressure (phase 5).
If muffled sounds persist (phase 4) and do not disappear, use the point of muffling as the diastolic pressure.
Common problems

- BP is different in each arm: a difference >10 mmHg suggests the presence of aortic or subclavian artery disease. Unequal brachial BP is a marker of increased cardiovascular morbidity and mortality (Box 6.21). Record the highest pressure and use this to guide management.

- Wrong cuff size
- In obese patients a standard adult cuff will overestimate BP, so use a large cuff

- Auscultatory gap: up to 20% of elderly hypertensive patients have Korotkoff sounds which appear at systolic pressure and disappear for an interval between systolic and diastolic pressure. If the first appearance of the sound is missed, the systolic pressure will be recorded at a falsely low level. Avoid this by palpating the systolic pressure first

- Patient’s arm at the wrong level: the patient’s elbow should be level with the heart. Hydrostatic pressure causes \( \approx 5 \) mmHg change in recorded systolic and diastolic BP for a 7 cm change in arm elevation

- Terminal digit preference: record the true reading rather than rounding values to the nearest 0 or 5
JVP

- The internal jugular vein enters the neck behind the mastoid process. It runs deep to the sternocleidomastoid muscle before entering the thorax between the sternal and clavicular heads and should be examined with the neck muscles relaxed. A pulsation is visible when the pressure in the internal jugular vein is elevated.

- The external jugular vein is more superficial, prominent and easier to see. It can be kinked or obstructed as it traverses the deep fascia of the neck but, when visible, pulsatile and not obstructed, it can be used to estimate the JVP in difficult cases.
Estimate the JVP by observing the level of pulsation in the internal jugular vein. The normal waveform has two main peaks per cycle, which helps to distinguish it from the carotid arterial pulse (Box 6.23). The JVP level reflects right atrial pressure (normally <7 mmHg/9 cmH₂O). The sternal angle is approximately 5 cm above the right atrium, so the JVP in health should be ≤4 cm above this angle when the patient lies at 45°. If right atrial pressure is low, the patient may have to lie flat for the JVP to be seen; if high, the patient may need to sit upright (Fig. 6.18).
Fig. 6.18 Jugular venous pressure in a healthy subject. (A) Supine: jugular vein distended, pulsation not visible. (B) Reclining at 45°: point of transition between distended and collapsed vein can usually be seen to pulsate just above the clavicle. (C) Upright: upper part of vein collapsed and transition point obscured.
- The JVP is best seen on the patient’s right side.
- Position the patient supine, reclined at 45°, with the head on a pillow to relax the sternocleidomastoid muscles.
- Look across the patient’s neck from the right side (Fig. 6.19A). Use oblique lighting if the JVP is difficult to see.
Identify the jugular vein pulsation in the suprasternal notch or behind the sternocleidomastoid muscle.

Use the abdomino-jugular test or occlusion to confirm it is the JVP.

The JVP is the vertical height in centimetres between the upper limit of the venous pulsation and the sternal angle (junction of the manubrium and sternum at the level of the second costal cartilages) (Fig. 6.19B).

Identify the timing & waveform of the pulsation and note any abnormality.
Normal findings

- Aids to differentiate the jugular venous waveform from arterial pulsation:

- Abdomino-jugular test: firmly press over the abdomen. This increases venous return to the right side of the heart temporarily and the JVP normally rises.

- Changes with respiration: the JVP normally falls with inspiration due to decreased intrathoracic pressure.

- Waveform (Fig. 6.19C): the normal JVP waveform has two distinct peaks per cardiac cycle:
  - ‘a’ wave corresponds to right atrial contraction and occurs just before the first heart sound. In atrial fibrillation the ‘a’ wave is absent.
  - ‘v’ wave is caused by atrial filling during ventricular systole when the tricuspid valve is closed.
  - Rarely, a third peak (‘c’ wave) may be seen due to closure of the tricuspid valve.

- Occlusion: the JVP waveform is obliterated by gently occluding the vein at the base of the neck with your finger.
Abnormal findings

- The JVP is primarily a sign of right ventricular function. It is elevated in states of fluid overload, notably in heart failure and in conditions with right heart dilatation, e.g. acute pulmonary embolism and chronic obstructive pulmonary disease (when it is called cor pulmonale). Mechanical obstruction of the superior vena cava (most often caused by lung cancer) may cause extreme, non-pulsatile elevation of the JVP. Here the JVP no longer reflects right atrial pressure and the abdominojugular test will be negative.
Kussmaul’s sign: a paradoxical rise of JVP on inspiration seen in pericardial constriction or tamponade, severe right ventricular failure and restrictive cardiomyopathy.

Prominent ‘a’ wave: caused by delayed or restricted right ventricular filling, e.g. pulmonary hypertension or tricuspid stenosis.

Cannon waves: giant ‘a’ waves occur when the right atrium contracts against a closed tricuspid valve. Irregular cannon waves are seen in complete heart block and are due to atrio-ventricular dissociation. Regular cannon waves occur during junctional rhythm and with some ventricular and supraventricular tachycardias.

‘cv’ wave: a fusion of the ‘c’ and ‘v’ waves resulting in a large systolic wave and associated with a pulsatile liver is seen in tricuspid regurgitation.
Thank you