Anxiety (angor)	Mild or no anxiety
No relief with nitrates	Rapid relief with nitrates
Associated symptoms (nausea and vomiting)	Associated symptoms absent
Favours myocardial infarction	**Favours aortic dissection**
Central chest pain	Radiates to back
Subacute onset (minutes)	Instantaneous onset
May be severe	Very severe
Favours myocardial ischaemia	**Favours chest wall pain**
Exertional	Positional
Occurs with exertion	Often worse at rest
Brief episodes	Prolonged
Diffuse	Localised
No chest wall tenderness (only discriminates between infarction and chest wall pain)	Chest wall tenderness

volume change in the lungs, because of a reduction in compliance of the lungs or increased resistance to air flow. Cardiac dyspnoea is typically chronic and occurs with exertion because of failure of the left ventricular output to rise with exercise; this in turn leads to an acute rise in left ventricular end-diastolic pressure, raised pulmonary venous pressure, interstitial fluid leakage and thus reduced lung compliance. However, the dyspnoea of chronic cardiac failure does not correlate well with measurements of pulmonary artery pressures, and clearly the origin of the symptom of cardiac dyspnoea is complicated.[5] Left ventricular function may be impaired because of ischaemia (temporary or permanent reduction in myocardial blood supply), previous infarction (damage) or hypertrophy (often related to hypertension). As it becomes more severe, cardiac dyspnoea

(often related to hypertension). As it becomes more severe, cardiac dyspnoea occurs at rest.

Orthopnoea (from the Greek *ortho* 'straight'; see Table 4.5), or dyspnoea that develops when a patient is supine, occurs because in an upright position the patient's interstitial oedema is redistributed; the lower zones of the lungs become worse and the upper zones better. This allows improved overall blood oxygenation. Patients with severe orthopnoea spend the night sitting up in a chair or propped up on numerous pillows in bed. The absence of orthopnoea suggests that left ventricular failure is unlikely to be the cause of a patient's dyspnoea (negative likelihood ratio [LR] = 0.04[6]).

TABLE 4.5 Causes of orthopnoea

Cardiac failure
Uncommon causes
Massive ascites
Pregnancy
Bilateral diaphragmatic paralysis
Large pleural effusion
Severe pneumonia

Paroxysmal[b] *nocturnal dyspnoea* (PND) is severe dyspnoea that wakes the patient from sleep so that he or she is forced to get up gasping for breath. This occurs because of a sudden failure of left ventricular output with an acute rise in pulmonary venous and capillary pressures; this leads to transudation of fluid into the interstitial tissues, which increases the work of breathing. The sequence may be precipitated by resorption of peripheral oedema at night while supine. Acute cardiac dyspnoea may also occur with acute pulmonary oedema or a pulmonary embolus.

Cardiac dyspnoea can be difficult to distinguish from that due to lung disease or other causes (page 109)[7]. One should inquire particularly about a history of any cardiac disease that could be responsible for the onset of cardiac failure. For example, a patient with a number of known previous

cardiac failure. For example, a patient with a number of known previous myocardial infarctions who develops dyspnoea is more likely to have decreased left ventricular contractility. A patient with a history of hypertension or a very heavy alcohol intake may have hypertensive heart disease or an alcoholic cardiomyopathy. The presence of orthopnoea or paroxysmal nocturnal dyspnoea is more suggestive of cardiac failure than of lung disease.

Dyspnoea is also a common symptom of anxiety. These patients often describe an inability to take a big enough breath to fill the lungs in a satisfying way. Their breathing may be deep and punctuated with sighs.

Ankle swelling

Some patients present with bilateral ankle swelling due to *oedema* from cardiac failure. Patients with the recent onset of oedema and who take a serious interest in their weight may have noticed a gain in weight of 3 kg or more. Ankle oedema of cardiac origin is usually symmetrical and worst in the evenings, with improvement during the night. It may be a symptom of biventricular failure or right ventricular failure secondary to a number of possible underlying aetiologies. As failure progresses, oedema ascends to involve the legs, thighs, genitalia and abdomen. There are usually other symptoms or signs of heart disease.

It is important to find out whether the patient is taking a vasodilating drug (e.g. a calcium channel blocker), which can cause peripheral oedema. There are other (more) common causes of ankle oedema than heart failure that also need to be considered (page 71). Oedema that affects the face is more likely to be related to nephrotic syndrome (page 213).

Palpitations

This is not a very precise term. It is usually taken to mean an unexpected awareness of the heartbeat.[8] Ask the patient to describe exactly what he or she notices and whether the palpitations are slow or fast, regular or irregular, and how long they last (*Questions box 4.2*).

Questions box 4.2

Questions to ask the patient with palpitations

! denotes symptoms for the possible diagnosis of an urgent or dangerous problem.

1. Is the sensation one of the heart beating abnormally, or something else?

2. Does the heart seem fast or slow? Have you counted how fast? Is it faster than it ever goes at any other time, e.g. with exercise?

3. Does the heart seem regular or irregular—stopping and starting? If it is irregular, is this the feeling of normal heart beats interrupted by missed or strong beats—ectopic beats; or is it completely irregular?—Atrial fibrillation

4. How long do the episodes last?

5. Do the episodes start and stop very suddenly?—Supraventricular tachycardia (SVT)

6. Can you terminate the episodes by deep breathing or holding your breath?—SVT

7. Is there a sensation of pounding in the neck?—some types of SVT[9]

8. Has an episode ever been recorded on an ECG?

9. Have you lost consciousness during an episode?—Ventricular arrhythmias

10. ! Have you had other heart problems such as heart failure or a heart attack in the past?—Ventricular arrhythmias?

11. Is there heart trouble of this sort in the family?

There may be the sensation of a missed beat followed by a particularly heavy beat; this can be due to an *atrial* or *ventricular ectopic beat* (which produces little cardiac output) followed by a compensating pause and then a normally conducted beat (which is more forceful than usual because there has been a longer diastolic filling period for the ventricle).

If the patient complains of a rapid heartbeat, it is important to find out whether the palpitations are of sudden or gradual onset and offset. Cardiac arrhythmias are usually instantaneous in onset and offset, whereas the onset and offset of *sinus tachycardia* is more gradual. A completely irregular rhythm is suggestive of *atrial fibrillation*, particularly if it is rapid.

It may be helpful to ask the patient to tap the rate and rhythm of the palpitations with his or her finger. Associated features including pain, dyspnoea or faintness must be inquired about. The awareness of rapid palpitations followed by syncope suggests *ventricular tachycardia*. These patients usually have a past history of significant heart disease. Any rapid rhythm may precipitate angina in a patient with ischaemic heart disease.

Table 4.6 Causes (differential diagnosis) of dyspnoea, palpitations and oedema

Favours heart failure		**Favours lung disease**
History of myocardial infarction		History of smoking
		Onset after some exertion (asthma)
No wheeze		Wheezing
PND		PND absent
Orthopnoea		Orthopnoea absent
Abnormal apex beat		
Third heart sound (S3)		
Mitral regurgitant murmur		
		Overexpanded chest
		Pursed-lips breathing
Early and mid-inspiratory crackles		Fine end-inspiratory crackles
Cough only on lying down		Productive cough

lying down		
Palpitations differential diagnosis		**Ankle oedema differential diagnosis**
Feature	**Suggests**	**Favours heart failure**
Heart misses and thumps	Ectopic beats	History of cardiac failure
Worse at rest	Ectopic beats	Other symptoms of heart failure
Very fast, regular	SVT (VT)	Jugular venous pressure elevated (+ve LR 9.0[*])
Instantaneous onset	SVT (VT)	**Favours hypoproteinaemia**
Offset with vagal manoeuvres	SVT	Jugular venous pressure normal
Fast and irregular	AF	Oedema pits and refills rapidly, 2–3s[†]
Forceful and regular—not fast	Awareness of sinus rhythm (anxiety)	**Favours deep venous thrombosis or cellulitis**
		Unilateral
		Skin erythema
		Calf tenderness
Severe dizziness or syncope	VT	
Pre-existing heart failure	VT	
		Favours drug-induced oedema
		Patient takes calcium channel blocker
		Favours lymphoedema
		Not worse at end of day

		Not pitting when chronic
		Favours lipoedema
		Not pitting
		Spares foot
		Obese woman

PND = paroxysmal nocturnal dyspnoea.

SVT = supraventricular tachycardia.

VT = ventricular tachycardia.

AF = atrial fibrillation.

* McGee S, *Evidence-based clinical diagnosis*, 2nd edn. St Louis: Saunders, 2007.

† Khan NA, Rahim SA, Avand SS et al. Does the clinical examination predict lower extremity peripheral arterial disease? *JAMA* 2006 Feb 1; 295(5):536–546.

Table 4.7 Differential diagnosis of syncope and dizziness

Favours vasovagal syncope (most common cause)
Onset in teens or 20s
Occurs in response to emotional distress, e.g. sight of blood
Associated with nausea and clamminess
Injury uncommon
Unconsciousness brief, no neurological signs on waking
Favours orthostatic hypotension
Onset when getting up quickly
Brief duration
Injury uncommon
More common when fasted or dehydrated
Known low systolic blood pressure
Use of antihypertensive medications
Favours 'situational syncope'
Occurs during micturition
Occurs with prolonged coughing
Favours syncope due to left ventricular outflow obstruction (AS, HCM)
Occurs during exertion

Favours cardiac arrhythmia
Family history of sudden death (Brugada or long QT syndrome)
Anti-arrhythmic medication (prolonged QT)
History of cardiac disease (ventricular arrhythmias)
History of rapid palpitations
No warning (heart block—Stokes-Adams attack)
Favours vertigo
No loss of consciousness
Worse when turning head
Head or room seems to spin
Favours seizure
Prodrome—aura
Tongue bitten
Jerking movements during episode
Sleepiness afterwards
Head turns during episode
Follows emotional stress
Cyanosis
Muscle pain afterwards
Favours metabolic cause of syncope (coma)
Hypoglycaemic agents, low blood sugar

AS = aortic stenosis.

HCM = hypertrophic cardiomyopathy.

Patients may have learned manoeuvres that will return the rhythm to normal. Attacks of *supraventricular tachycardia* (SVT) may be suddenly terminated by increasing vagal tone with the Valsalva manoeuvre (page 70), carotid massage, by coughing, or by swallowing cold water or ice cubes.

Syncope, presyncope and dizziness

Syncope is a transient loss of consciousness resulting from cerebral anoxia, usually due to inadequate blood flow. Presyncope is a transient sensation of weakness without loss of consciousness. (See *Questions box 11.4*, page 326.)

Syncope may represent a simple faint or be a symptom of cardiac or neurological disease. One must establish whether the patient actually loses consciousness and under what circumstances the syncope occurs—e.g. on standing for prolonged

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Questions box 4.3

Questions to ask the patient with suspected peripheral vascular disease

! denotes symptoms for the possible diagnosis of an urgent or dangerous problem.

1. Have you had problems with walking because of pains in the legs?
2. Where do you feel the pain?
3. How far can you walk before it occurs?
4. Does it make you stop?
5. Does it go away when you stop walking?
6. Does the pain ever occur at rest?—Severe ischaemia may threaten the limb
7. Have there been changes in the colour of the skin over your feet or ankles?
8. Have you had any sores or ulcers on your feet or legs that have not healed?
9. Have you needed treatment of the arteries of your legs in the past?
10. Have you had diabetes, high blood pressure, or problems with strokes or heart attacks in the past?
11. Have you been a smoker?

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periods or standing up suddenly (*postural syncope*), while passing urine (*micturition syncope*), on coughing (*tussive syncope*), or with sudden

(*micturition syncope*), on coughing (*tussive syncope*), or with sudden emotional stress (*vasovagal syncope*). Find out whether there is any warning, such as dizziness or palpitations, and how long the episodes last. Recovery may be spontaneous or the patient may require attention from bystanders.

If the patient's symptoms appear to be postural, inquire about the use of anti-hypertensive or anti-anginal drugs and other medications that may induce postural hypotension. If the episode is vasovagal, it may be precipitated by something unpleasant like the sight of blood, or occur in a crowded, hot room; patients often sigh and yawn and feel nauseated and sweaty before fainting and may have previously had similar episodes, especially during adolescence and young adulthood.

If syncope is due to an *arrhythmia*, there is a sudden loss of consciousness regardless of the patient's posture; chest pain may also occur if the patient has ischaemic heart disease or aortic stenosis.[10] Recovery is equally quick. Exertional syncope may occur with obstruction to left ventricular outflow by *aortic stenosis* or *hypertrophic cardiomyopathy*. Profound and sudden bradycardia, usually a result of complete heart block, causes sudden and recurrent syncope (Stokes-Adams[c] attacks[d]). These patients may have a history of atrial fibrillation. Typically they have periods of tachycardia (fast heart rate) as well as periods of bradycardia (slow heart rate). This condition is called the *sick sinus syndrome*. The patient must be asked about drug treatment that could cause bradycardia, e.g. beta-blockers, digoxin, calcium channel blockers.

It is important to ask about a family history of sudden death. An increasing number of *ion channelopathies* are being identified as a cause of syncope and sudden death. These inherited conditions include the long QT syndrome and the Brugada syndrome. They are often diagnosed from typical ECG changes. In addition, certain drugs can cause the acquired long QT syndrome (Table 4.8).

TABLE 4.8 Drugs and syncope

Associated with QT interval prolongation and ventricular arrhythmias
Anti-arrhythmics; flecainide, quinidine, sotalol, procainamide, amiodarone
Gastric motility promoter; cisapride
Antibiotics; clarithromycin, erythromycin
Antipsychotics; chlorpromazine, haloperidol
Associated with bradycardia
Beta-blockers
Some calcium channel blockers (verapamil, diltiazem)
Digoxin
Associated with postural hypotension
Most antihypertensive drugs, but especially prazosin and calcium channel blockers
Anti-Parkinsonian drugs

Neurological causes of syncope are associated with a slow recovery and often residual neurological symptoms or signs. Bystanders may also have noticed abnormal movements if the patient has epilepsy. Dizziness that occurs even when the patient is lying down or which is made worse by movements of the head is more likely to be of neurological origin, although recurrent tachyarrhythmias may occasionally cause dizziness in any position. One should attempt to decide whether the dizziness is really *vertiginous* (where the world seems to be turning around), or whether it is a presyncopal feeling.

Intermittent claudication and peripheral vascular disease

The word claudication[e] comes from the Latin meaning to limp. Patients with claudication notice pain in one or both calves, thighs or buttocks when they walk more than a certain distance. This distance is called the 'claudication distance'. The claudication distance may be shorter when patients walk up hills. A history of claudication suggests peripheral vascular disease with a poor blood supply to the affected muscles. The most important risk factors are smoking, diabetes, hypertension and a history of vascular disease

elsewhere in the body, including cerebrovascular disease and ischaemic heart disease. More severe disease causes the feet or legs to feel cold, numb and finally painful at rest. Rest pain is a symptom of severely compromised arterial supply. Remember the six P's of peripheral vascular disease:

Pain

Pallor

Pulselessness

Paraesthesiae

Perishingly cold

Paralysed.

Popliteal artery entrapment can occur, especially in young men with intermittent claudication on walking but *not* running. Also, *lumbar spinal stenosis* causes pseudo-claudication: unlike vascular claudication, the pain in the calves is not relieved by standing still, but is relieved by sitting (flexing the spine) and may be exacerbated by extending the spine (e.g. walking downhill).

Fatigue

Fatigue is a common symptom of cardiac failure. It may be associated with a reduced cardiac output and poor blood supply to the skeletal muscles. There are many other causes of fatigue, including lack of sleep, anaemia and depression.

Risk factors for coronary artery disease

An essential part of the cardiac history involves obtaining detailed information about a patient's risk factors—the patient's **cardiovascular risk factor profile** (*Questions box 4.4*).

Questions box 4.4

Questions to ask about possible cardiovascular risk factors

1. Have you had angina or a heart attack in the past?
2. Do you know what your cholesterol level is? Before or after treatment?
3. Are you a diabetic?
4. Have you had high blood pressure and has it been treated?
5. Are you now or have you been a smoker? How long since you stopped?
6. Has anyone in the family had angina or heart attacks? Who? How old were they?
7. Have you had kidney problems?

Previous ischaemic heart disease is the most important risk factor for further ischaemia. The patient may know of previous infarcts or have had a diagnosis of angina in the past.

Hypercholesterolaemia is the next most important risk factor for ischaemic heart disease. Many patients now know their serum cholesterol levels because widespread testing has become fashionable. The total serum cholesterol is a useful screening test, and levels above 5.2 mmol/L are considered undesirable. Cholesterol measurements (unlike triglyceride measurements) are accurate even when a patient has not been fasting. Patients with established coronary artery disease benefit from lowering of total cholesterol to below 4 mmol/L. An elevated total cholesterol level is even more significant if the high-density lipoprotein (HDL) level is low (less than 1.0 mmol/L). Significant elevation of the triglyceride level is a coronary risk factor in its own right and also adds further to the risk if the total cholesterol is high. If a patient already has coronary disease, *hyperlipidaemia* is even more important. Control of risk factors for these patients is called 'secondary prevention'. Patients who have multiple risk factors for ischaemic heart disease (e.g. diabetes and hypertension) should have their cholesterol controlled aggressively. If the patient's cholesterol is known to be high, it is worth obtaining a dietary history. This can be very trying. It is important to remember that not only foods containing cholesterol but those containing saturated fats contribute to the serum cholesterol level. High alcohol consumption and obesity are associated with hypertriglyceridaemia.

Smoking is probably the next most important risk factor for cardiovascular disease and peripheral vascular disease. Some patients describe themselves as non-smokers even though they stopped smoking only a few hours before. The number of years the patient has smoked and the number of cigarettes smoked per day are both very important (and are recorded as packet-years, page 6). The significance of a history of smoking

for a patient who has not smoked for many years is controversial. The risk of symptomatic ischaemic heart disease falls gradually over the years after smoking has been stopped. After about 2 years the risk of myocardial infarction falls to the same level as for those who have never smoked. After 10 years the risk of developing angina falls close to that of non-smokers.

Hypertension is another important risk factor for coronary artery disease. Find out when hypertension was first diagnosed and what treatment, if any, has been instituted. The treatment of hypertension probably does reduce the risk of ischaemic heart disease, and certainly reduces the risk of hypertensive heart disease, cardiac failure and cerebrovascular disease. Treatment of hypertension has also been shown to reverse left ventricular hypertrophy.

A family history of coronary artery disease increases a patient's risk, particularly if it has been present in first-degree relatives (parents or siblings) and if it has affected these people below the age of 60. Not all heart disease, however, is ischaemic; a patient whose relatives suffered from rheumatic heart disease is at no greater risk of ischaemic heart disease than anybody else.

A history of *diabetes mellitus* increases the risk of ischaemic heart disease very substantially. A diabetic without a history of ischaemic heart disease has the same risk of myocardial infarction as a non-diabetic who has had an infarct. It is important to find out how long a patient has been diabetic and whether insulin treatment has been required. Good control of the blood sugar level of diabetics reduces this risk. An attempt should therefore be made to find out how well a patient's diabetes has been controlled.

Chronic kidney disease is associated with a very high risk of vascular disease. This is possibly related to high calcium-times-phosphate product and may be reduced by dietary intervention, 'phosphate binders', efficient dialysis, in renal transplant. Ischaemic heart disease is the most common cause of death in renal failure patients on dialysis.

The presence of multiple risk factors makes control of each one more important. Aggressive control of risk factors is often indicated in these patients.

It is interesting to note that in the diagnosis of angina the patient's description of typical symptoms is more discriminating than is the presence of risk factors which only marginally increase the likelihood that chest pain is ischaemic. Previous ischaemic heart disease is an exception. Certainly a patient who has had angina before and says he or she has it again, is usually right.

Teeth

A history of dental decay or infection is important for patients with valvular heart disease, since it puts them at risk of infective endocarditis. Dental caries may also be associated with an increased risk of ischaemic heart disease. Ask about the regularity of visits to the dentist and the patient's awareness of the need for antibiotic prophylaxis before dental (and some surgical) procedures.

Treatment

The medications a patient is taking often give a good clue to the diagnosis. Find out about any ill-effects from current or previous medications. The surgical history must also be elicited. The patient may have had a previous angioplasty or coronary artery bypass grafting, and may know how many arteries were dilated or bypassed. If the patient is unable to provide a history, a midline sternotomy scar and scars consistent with previous saphenous vein harvesting support this diagnosis.

Past history

Patients with a history of definite previous angina or myocardial infarction remain at high risk for further ischaemic events. It is very useful at this stage to find out how a diagnosis of ischaemic heart disease was made and in particular what investigations were undertaken. The patient may well remember exercise testing or a coronary angiogram, and some patients can even remember how many coronary arteries were narrowed, how many coronary bypasses were performed (having more than three grafts often leads to a certain amount of boasting). The angioplasty patient may know how many arteries were dilated and whether *stents* (often called coronary stunts by patients and cardiac surgeons) were inserted. Acute coronary syndromes are now usually treated with early coronary angioplasty.

Patients may recall a diagnosis of rheumatic fever in their childhood, but many were labelled as having 'growing pains'.[11] A patient who was put to bed for a long period as a child may well have had rheumatic fever. A history of rheumatic fever places patients at risk of rheumatic valvular disease.

Hypertension may be caused or exacerbated by aspects of the patient's activities and diet (*Questions box 4.5*). A high salt intake, moderate or greater alcohol use, lack of exercise, obesity and kidney disease may all be factors contributing to high blood pressure. Non-steroidal anti-inflammatory drugs cause salt and fluid retention and may also worsen blood pressure. Ask about these, about previous advice to modify these factors, and about any drug treatment of hypertension when interviewing any patient with high blood pressure.

pressure.

Questions box 4.5

Questions to ask the patient with hypertension

1. Do you use much salt in your diet, or eat salty prepared or snack foods?
2. Have you put on weight recently?
3. How much alcohol do you drink?
4. What sort of exercise do you do and how much?
5. Have you had any kidney problems?
6. Do you take your blood pressure at home? What readings do you get?
7. Are you taking any blood pressure tablets and do these cause you any problems?

Social history

Both ischaemic heart disease and rheumatic heart disease are chronic conditions that may affect a patient's ability to function normally. It is therefore important to find out whether the patient's condition has prevented him or her from working and over what period. Patients with severe cardiac failure, for example, may need to make adjustments to their living arrangements so that they are not required to walk up and down stairs at home.

Most hospitals run cardiac rehabilitation programmes for patients with ischaemic heart disease or chronic heart failure. They provide exercise classes that help patients regain their confidence and physical fitness, along with information classes about diet and drug treatment, and can help with psychological problems. Find out if the patient has been enrolled in one of these and whether it has been helpful. Is this service used as a point of contact for the patient if he or she has concerns about new symptoms or the management of medications?

The return of confidence and self-esteem are very important issues for patients and for their families after a life-threatening illness.

Examination anatomy

The contraction of the heart results in a wringing or twisting movement that is often palpable (the *apex beat*) and sometimes visible on the part of the chest that lies in front of it—the *praecordium* (from the Latin *prae* 'in front of', and *cor* 'heart'). The passage of blood through the heart and its valves and on into the great vessels of the body produces many interesting sounds, and causes pulsation in arteries and movement in veins in remote parts of the body. Signs of cardiac disease may be found by examining the praecordium and the many accessible arteries and veins of the body.

The surface anatomy of the heart and of the cardiac valves (Figure 4.1) and the positions of the palpable arteries (Figure 4.2) must be kept in mind during the examination of the cardiovascular system. In addition the physiology of blood flow through the systemic and pulmonary circuits need to be understood if the cardiac cycle and causes of cardiac murmurs are to be understood (Figure 4.3).

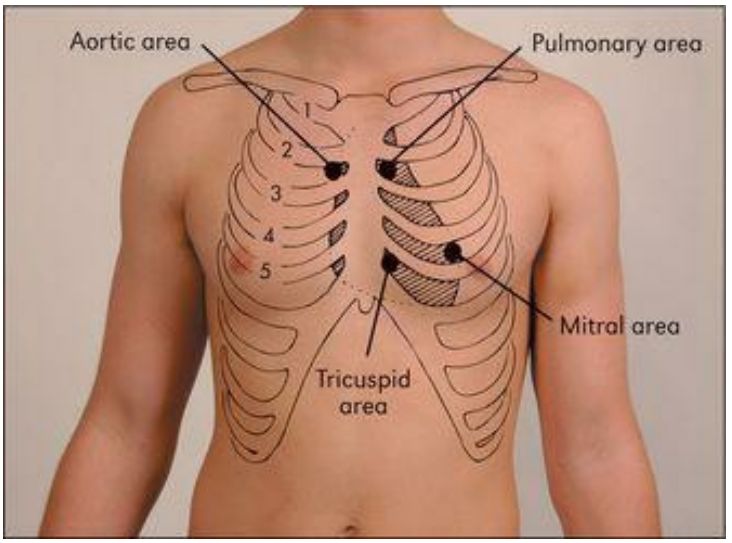
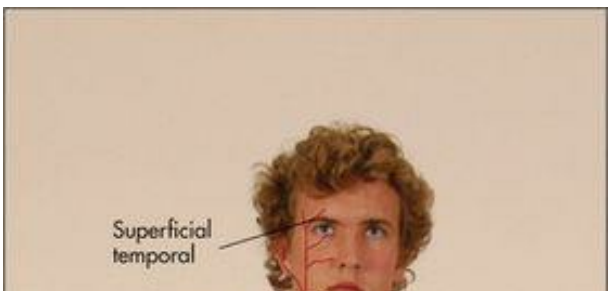


Figure 4.1 The areas best for auscultation do not exactly correlate with the anatomical location of the valves



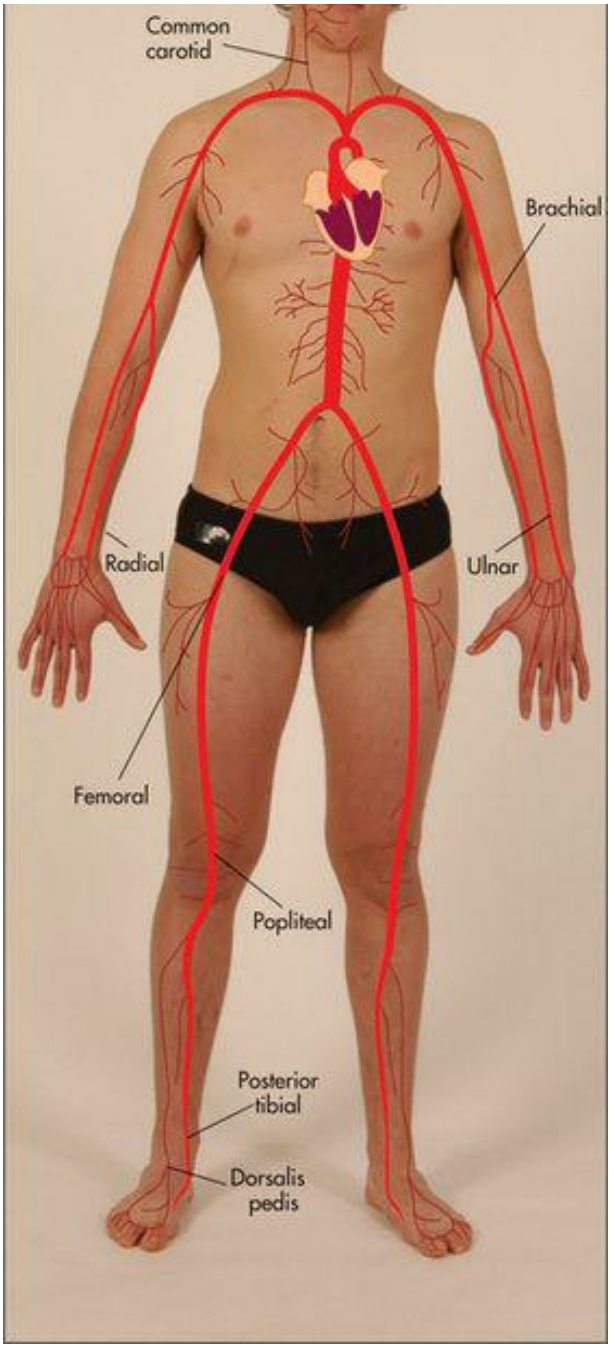
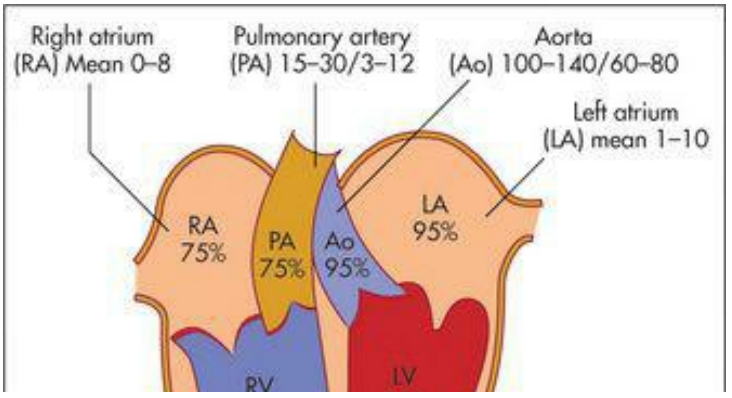


Figure 4.2 Palpable arteries



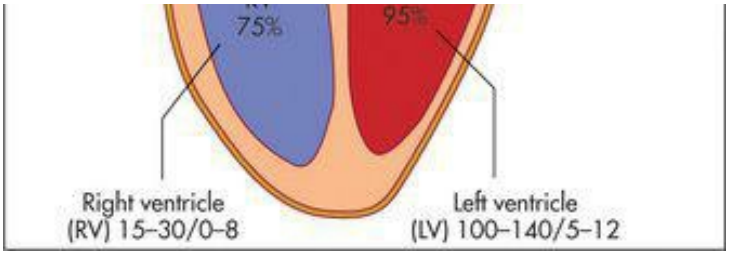


Figure 4.3 Normal pressures (mmHg) and saturations (%) in the heart

The cardiac valves separate the atria from the ventricles (the atrioventricular or mitral and tricuspid valves) and the ventricles from their corresponding great vessels. Figure 4.4 shows the fibrous skeleton that supports the four valves and their appearance during systole (cardiac contraction) and diastole (cardiac relaxation).

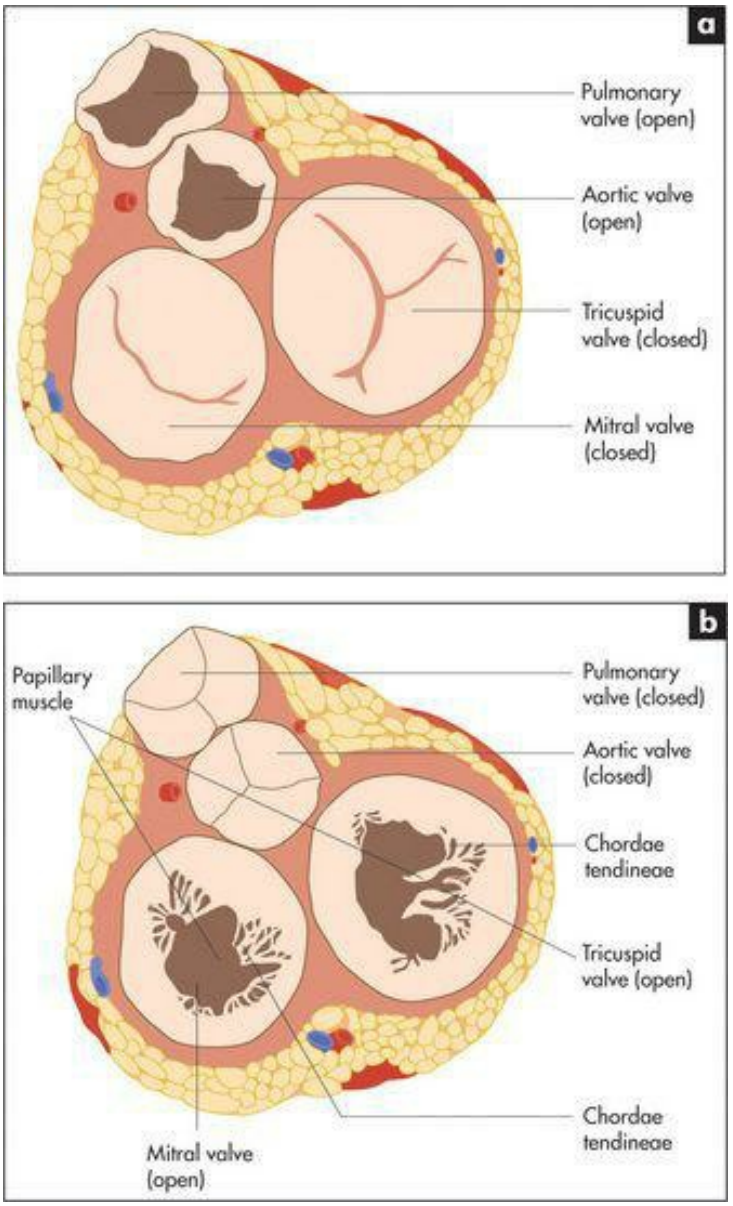


Figure 4.4 The cardiac valves in systole (a) and diastole (b)

The filling of the right side of the heart from the systemic veins can be assessed by inspection of the jugular veins in the neck (Figure 4.5) and by palpation of the liver. These veins empty into the right atrium.

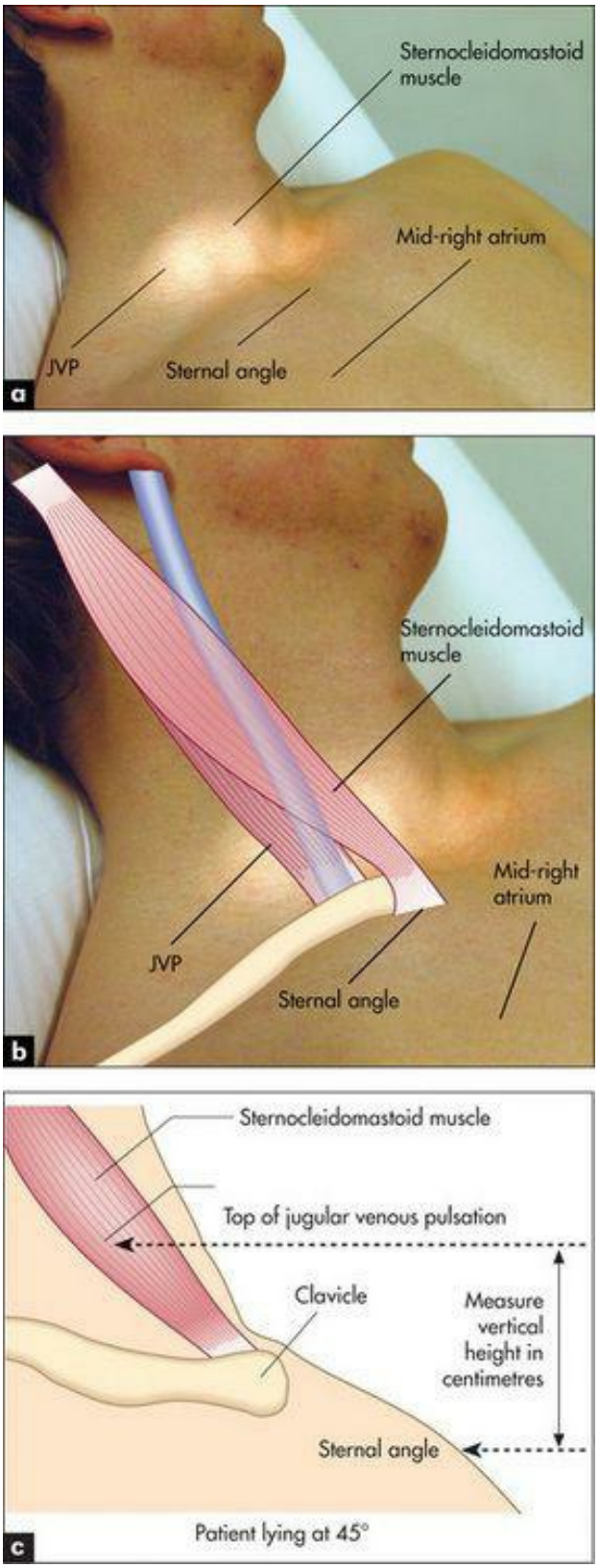


Figure 4.5 The jugular venous pressure (JVP) (a) Assessment of the JVP. The patient

Figure 4.5 The jugular venous pressure (JVP) (a) Assessment of the JVP. The patient should lie at 45 degrees. The relationships between the sternomastoid muscle, the JVP, the sternal angle and the mid-right atrium are shown. (b, c) The anatomy of the neck showing the relative positions of the main vascular structures, clavicle and sternocleidomastoid muscle. See also Figure 4.6.

Figures (b) and (c) adapted from Douglas G, Nicol F, Robertson C, Macleod's Clinical Examination, 11th edn. Edinburgh: Churchill Livingstone, 2005.

The internal jugular vein is deep in the sternomastoid muscle, while the external jugular vein is lateral to it. Traditionally, use of the external jugular vein to estimate venous pressure is discouraged, but the right internal and external jugular veins usually give consistent readings. The left-sided veins are less accurate because they cross from the left side of the chest before entering the right atrium. Pulsations that occur in the right-sided veins reflect movements of the top of a column of blood that extends directly into the right atrium. This column of blood may be used as a manometer and enables us to observe pressure changes in the right atrium. By convention, the sternal angle is taken as the zero point and the maximum height of pulsations in the internal jugular vein, which are visible above this level when the patient is at 45 degrees, is measured in centimetres. In the average person the centre of the right atrium lies 5 cm below this zero point (Figures 4.5a and 4.6).

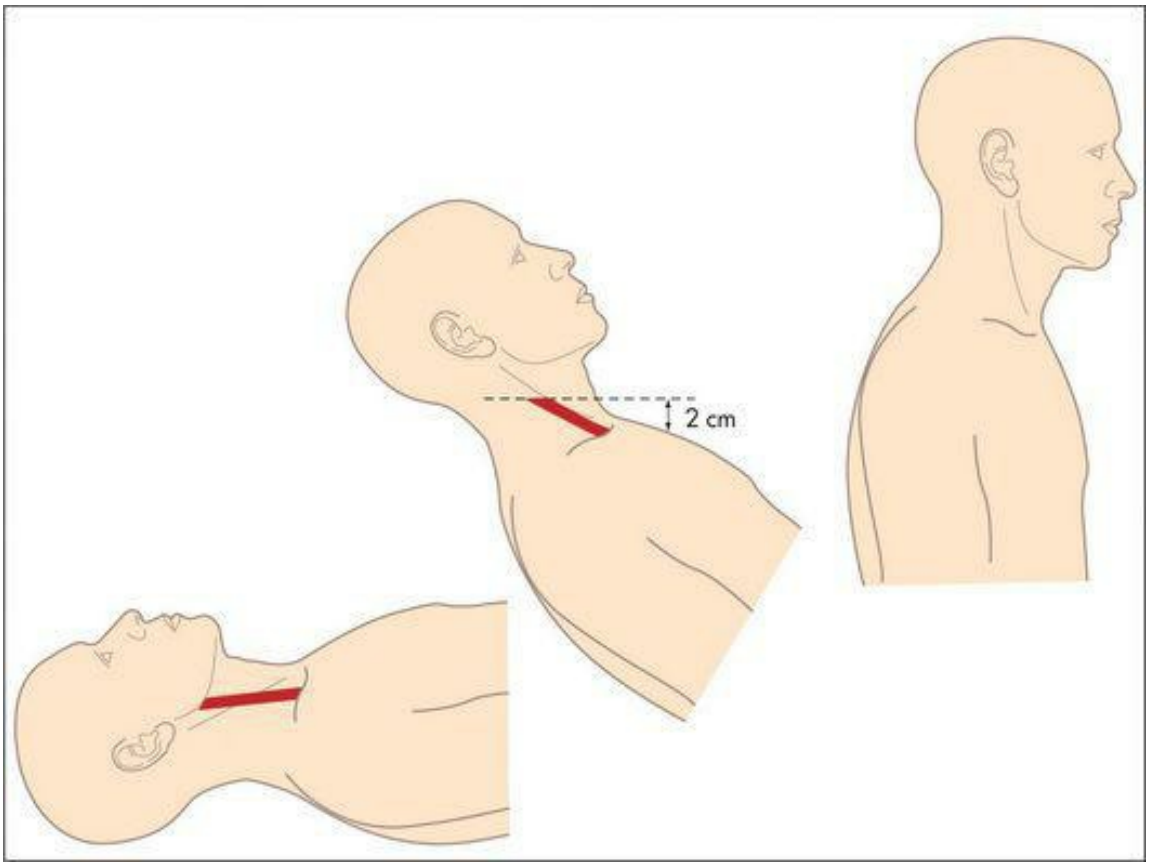


Figure 4.6 Changes in the height of the JVP as the patient sits up

Adapted from McGee S, Evidence-based physical diagnosis, 2nd edn. St Louis: Saunders, 2007.

The cardiovascular examination

The cardiovascular system lends itself particularly well to the formal examination approach. There are a number of equally satisfactory methods, but the precise approach used is not as important as having a method which is comprehensive, gives the impression of being (and is) proficient, and ensures that no important part of the examination is omitted.

First, one should appropriately expose and position the patient properly and pause to get an impression of the general appearance. Then detailed examination begins with the hands and pulses and progresses smoothly to the neck, face, and then on to the praecordium. A summary of a suggested method of examination is found at the end of this chapter.

Positioning the patient

It is important to have the patient lying in bed with enough pillows to support him or her at 45 degrees (Figure 4.7). This is the usual position in which the jugular venous pressure (JVP) is assessed. Even a 'targeted' cardiovascular examination in an outpatients' clinic or surgery can only be performed adequately if the patient is lying down and an examination couch should be available. During auscultation, optimal examination requires further positioning of the patient, as discussed later.

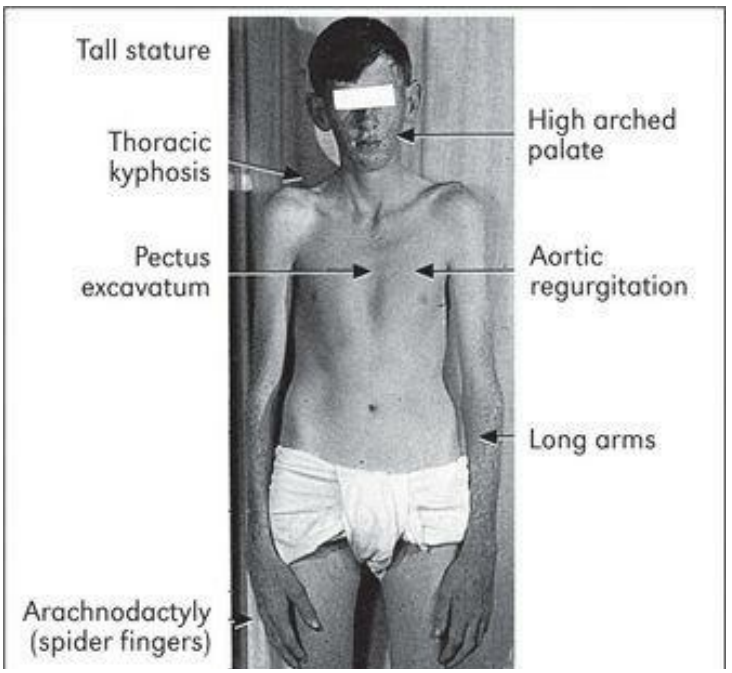
Figure 4.7 Cardiovascular examination: positioning the patient

General appearance

Look at the general state of health. Does the patient appear to be ill? If he or she looks ill, try to decide why you have formed that impression. Note whether the patient at rest has rapid and laboured respiration, suggesting dyspnoea (see Table 5.6, page 110).

The patient may look *cachectic*: that is, there may be severe loss of weight and muscle wasting. This is commonly caused by malignant disease, but severe cardiac failure may also have this effect (*cardiac cachexia*). It probably results from a combination of anorexia (due to congestive enlargement of the liver), impaired intestinal absorption (due to congested intestinal veins) and increased levels of inflammatory cytokines such as TNF-α.

There are also some syndromes that are associated with specific cardiac disease. Marfan's syndrome[f] (Figure 4.8, page 50), Down syndrome[g] (page 314) and Turner's syndrome[h] (page 314) are important examples.



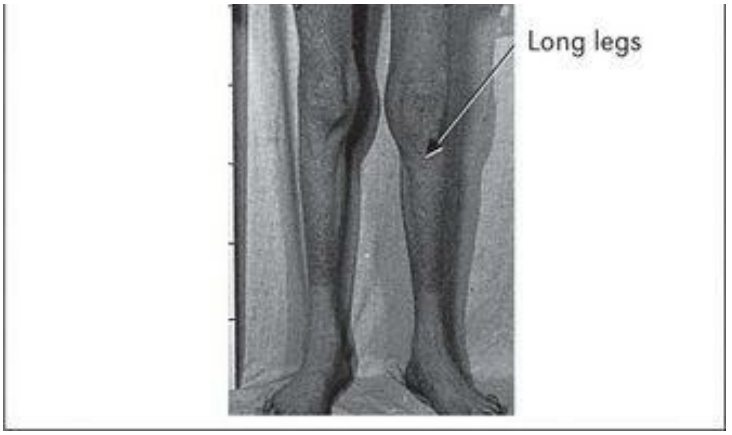


Figure 4.8 Marfan's syndrome
Tall stature, thoracic kyphosis, pectus excavatum, arachnodactyly (spider fingers), long limbs, aortic regurgitation and a high, arched palate

The hands

Pick up the right hand. Look first at the nails. Now is the time for a decision as to the presence or absence of *clubbing*. Clubbing is an increase in the soft tissue of the distal part of the fingers or toes. The causes of clubbing are surprisingly varied (Table 4.9). The mechanism is unknown but there are, of course, several theories. One current theory is that platelet-derived growth factor (PDGF), released from megakaryocyte and platelet emboli in the nail beds, causes fibrovascular proliferation. Megakaryocytes and clumps of platelets do not normally reach the arterial circulation. Their large size (up to 50 μm) prevents their passing through the pulmonary capillaries when they are released from the bone marrow. In conditions where platelets may clump in the arterial circulation (infected cardiac valve) or bypass the pulmonary capillaries (right to left shunt associated with congenital heart disease), they can reach the systemic circulation and become trapped in the terminal capillaries of the fingers and toes. Damage to pulmonary capillaries from various lung disorders can have the same effect.

TABLE 4.9 Causes of clubbing

Common
Cardiovascular
Cyanotic congenital heart disease
Infective endocarditis
Respiratory
Lung carcinoma (usually *not* small cell carcinoma)
Chronic pulmonary suppuration: • Bronchiectasis • Lung abscess • Empyema
Idiopathic pulmonary fibrosis
Uncommon
Respiratory
Cystic fibrosis
Asbestosis
Pleural mesothelioma (benign fibrous type) or pleural fibroma

Gastrointestinal
Cirrhosis (especially biliary cirrhosis)
Inflammatory bowel disease
Coeliac disease
Thyrotoxicosis
Familial (usually before puberty) or idiopathic
Rare
Neurogenic diaphragmatic tumours
Pregnancy
Secondary hyperparathyroidism
Unilateral clubbing
Bronchial arteriovenous aneurysm
Axillary artery aneurysm

Proper examination for clubbing involves inspecting the fingernails (and toenails) from the side to determine if there is loss of the angle between the nail bed and the finger—the *hyponychial angle* (Figure 4.9). One accepted measurement is the *interphalangeal depth ratio*. The anteroposterior (AP) dimension of the finger is measured at the distal interphalangeal joint and compared with the AP diameter at the level of the point where the skin joins the nail. A ratio of more than 1 means clubbing.[12,i] Eventually, the distal phalanx becomes enlarged, due to soft-tissue swelling. This angle can be measured with a *shadowgraph*, which projects the silhouette of the finger so that it can be measured with a protractor. It is not in common use. If the angle is greater than 190°, clubbing is generally agreed to be present. Patients hardly ever notice that they have clubbing, even when it is severe. They often express surprise at their doctor's interest in such an unlikely part of their anatomy.

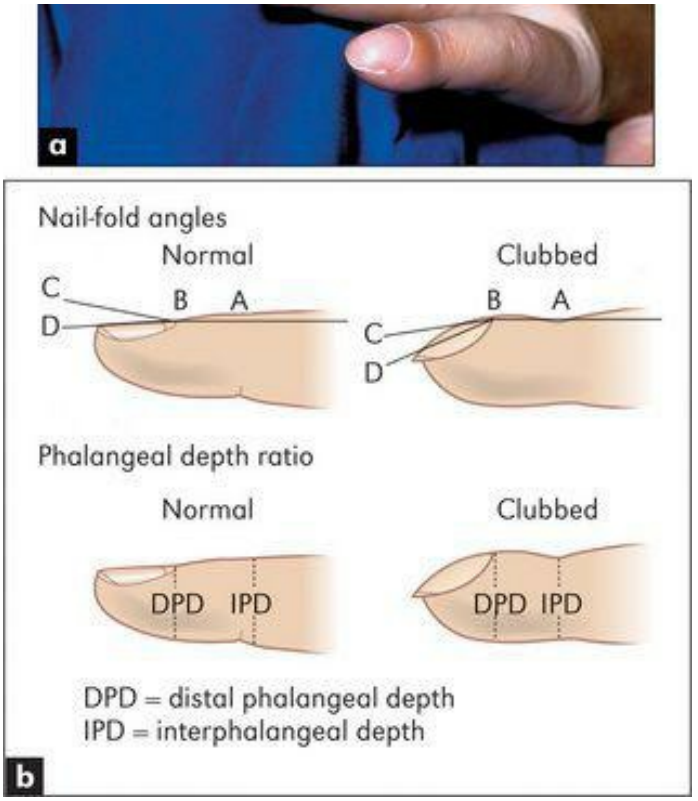


Figure 4.9 Finger clubbing: (a) appearance; (b) phalangeal depth ratio

Before leaving the nails, look for *splinter haemorrhages* in the nail beds (Figure 4.10). These are linear haemorrhages lying parallel to the long axis of the nail. They are most often due to trauma, particularly in manual workers. However, an important cause is infective endocarditis (page 79), which is a bacterial or other infection of the heart valves or part of the endocardium. In this disease splinter haemorrhages are probably the result of a vasculitis in the nail bed, but this is controversial. Other rare causes of splinter haemorrhages include vasculitis, as in rheumatoid arthritis, polyarteritis nodosa or the antiphospholipid syndrome, sepsis elsewhere in the body, haematological malignancy or profound anaemia.

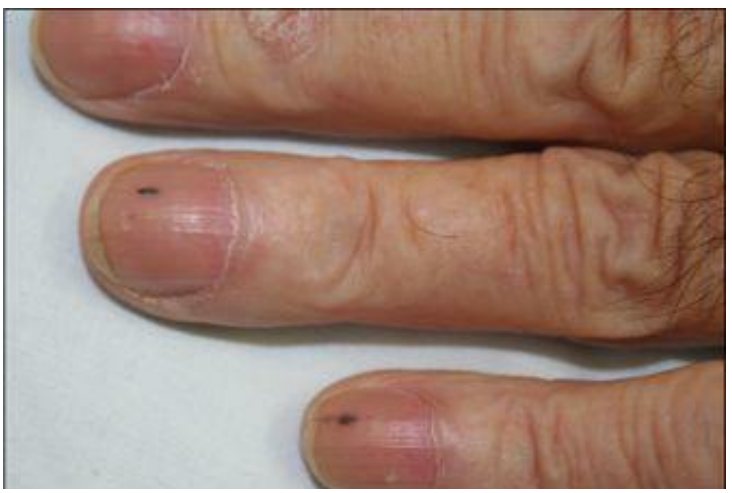


Figure 4.10 Splinter haemorrhages in the fingernails of a patient with staphylococcal aortic valve endocarditis

From Baker T, Nikolić G, O'Connor S, Practical Cardiology, 2nd edn. Sydney: Churchill Livingstone, 2008, with permission.

Osler's nodes[j] are a rare manifestation of infective endocarditis. These are red, raised, tender palpable nodules on the pulps of the fingers (or toes), or on the thenar or hypothenar eminences. They are reported to have occurred in 50% of patients before antibiotic treatment of endocarditis became available. Currently they are seen in fewer than 5% of patients. *Janeway lesions*[k] (Figure 4.11) are non-tender erythematous maculopapular lesions containing bacteria, which occur very rarely on the palms or pulps of the fingers in patients with infective endocarditis.[l]

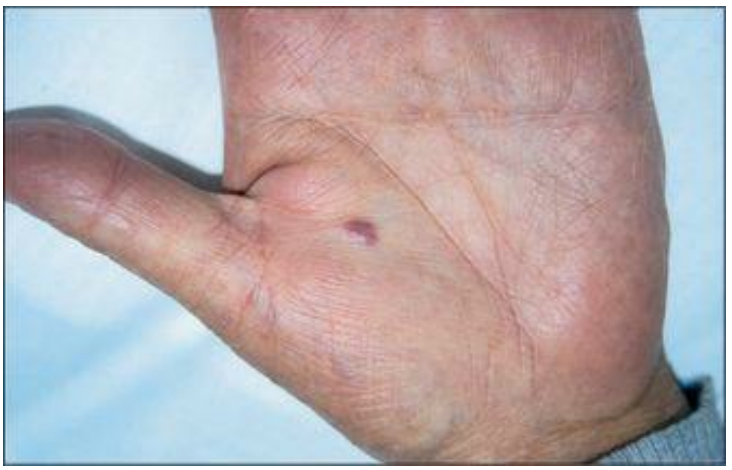


Figure 4.11 Janeway lesion

Tendon xanthomata are yellow or orange deposits of lipid in the tendons that occur in type II hyperlipidaemia. These can be seen over the tendons of the hand and arm. *Palmar xanthomata*, and *tuboeruptive xanthomata* over the elbows and knees, are characteristic of type III hyperlipidaemia (Figure 4.12).

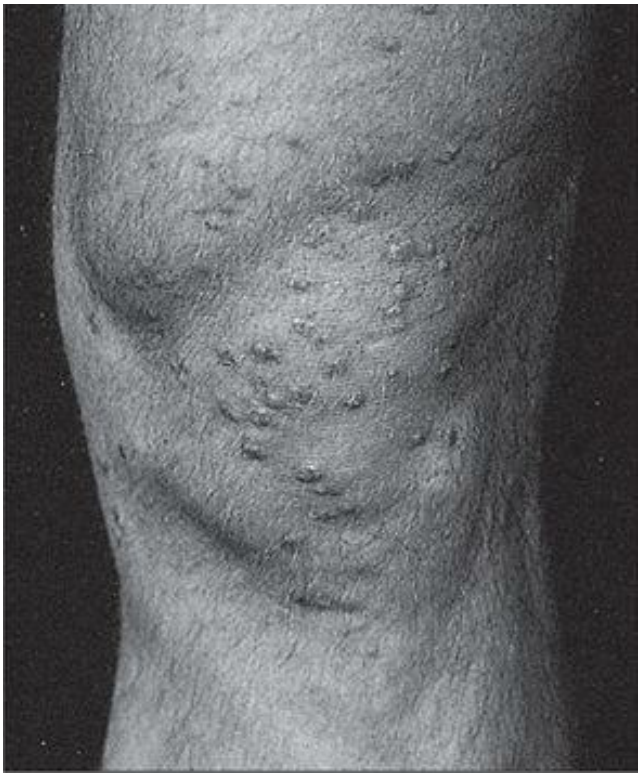


Figure 4.12 Tuboeruptive xanthomata of the knee

The arterial pulse

The accomplished clinician is able, while inspecting the hands, to palpate the radial artery at the wrist. Patients expect to have the pulse taken as part of a proper medical examination. The clinician can feel the pulse while talking to the patient and while looking for other signs. When this traditional part of the examination is performed with some ceremony, it may help to establish rapport between patient and doctor.

Although the radial pulse is distant from the central arteries, certain useful information may be gained from examining it. The pulse is usually felt just medial to the radius, using the forefinger and middle finger pulps of the examining hand (Figure 4.13). The following observations should be made: (i) rate of pulse, (ii) rhythm and (iii) presence or absence of delay of the femoral pulse compared with the radial pulse (*radiofemoral delay*). The character and volume of the pulse are better assessed from palpation of the brachial or carotid arteries.

Figure 4.13 Taking the radial pulse

Rate of pulse

Practised observers can estimate the rate quickly. Formal counting over 30 seconds is accurate and requires only simple mathematics to obtain the rate per minute. The normal resting heart rate in adults is usually said to be between 60 and 100 beats per minute but a more sensible range is probably 55 to 95 (95% of normal people). *Bradycardia* (Greek *bradys* 'slow', *kardia* 'heart') is defined as a heart rate of less than 60 beats per minute. *Tachycardia* (Greek *tachys* 'swift', *kardia* 'heart') is defined as a heart rate over 100 beats per minute. The causes of bradycardia and tachycardia are listed in Table 4.10.

TABLE 4.10 Causes of bradycardia and tachycardia

Bradycardia	
Regular rhythm	**Irregular rhythm**
Physiological (athletes, during sleep: due to increased vagal tone)	*Irregularly irregular*
	Atrial fibrillation (in combination with conduction system disease or AV nodal blocking drugs) due to:

Drugs (e.g. beta-blockers, digoxin, amiodarone)	• alcohol, post-thoracotomy, idiopathic • mitral valve disease or any cause of left atrial enlargement
Hypothyroidism (decreased sympathetic activity secondary to thyroid hormone deficiency)	Frequent ectopic beats
Hypothermia	
Raised intracranial pressure (due to an effect on central sympathetic outflow)—a late sign	*Regularly irregular rhythm*
Third degree atrioventricular (AV) block, or second degree (type 2) AV block	Sinus arrhythmia (normal slowing of the pulse with expiration)
Myocardial infarction	Second degree AV block (type 1)
Paroxysmal bradycardia: vasovagal syncope	Apparent
Jaundice (in severe cases only, due to deposition of bilirubin in the conducting system)	Pulse deficit* (atrial fibrillation, ventricular bigeminy)
Tachycardia	
Regular rhythm	**Irregular rhythm**
Hyperdynamic circulation, due to: • exercise or emotion (e.g. anxiety) • fever (allow 15–20 beats per minute per °C above normal)	Atrial fibrillation, due to: • myocardial ischaemia • mitral valve disease or any cause of left atrial enlargement • thyrotoxicosis

• pregnancy • thyrotoxicosis • anaemia • arteriovenous fistula (e.g. Paget's disease or hepatic failure) • beri-beri (thiamine deficiency)	• hypertensive heart disease • sick sinus syndrome • pulmonary embolism • myocarditis • fever, acute hypoxia or hypercapnia (paroxysmal) • other: alcohol, post-thoracotomy, idiopathic
Congestive cardiac failure	Multifocal atrial tachycardia
Constrictive pericarditis	Atrial flutter with variable block
Drugs (e.g. salbutamol and other sympathomimetics, atropine)	
Normal variant	
Denervated heart e.g. diabetes (resting rate of 106–120 beats per minute)	
Hypovolaemic shock	
Supraventricular tachycardia (usually >150)	
Atrial flutter with regular 2:1 AV block (usually 150)	
Ventricular tachycardia (often >150)	
Sinus tachycardia, due to: • thyrotoxicosis • pulmonary embolism	

• myocarditis • myocardial ischaemia • fever, acute hypoxia or hypercapnia (paroxysmal)	
Multifocal atrial tachycardia	
Atrial flutter with variable block	

* This is the difference between the heart rate counted over the praecordium and that observed at the periphery. In beats where diastole is too short for adequate filling of the heart, too small a volume of blood is ejected during systole for a pulse to be appreciated at the wrist.

Rhythm

The rhythm of the pulse can be regular or irregular. An irregular rhythm can be completely irregular with no pattern (*irregularly irregular* or *chaotic* rhythm); this is usually due to atrial fibrillation (Table 4.10). In atrial fibrillation coordinated atrial contraction is lost, and chaotic electrical activity occurs with bombardment of the atrioventricular (AV) node with impulses at a rate of over 600 per minute. Only a variable proportion of these is conducted to the ventricles because (fortunately) the AV node is unable to conduct at such high rates. In this way, the ventricles are protected from very rapid rates, but beat irregularly, usually at rates between 150 and 180 per minute (unless the patient is being treated with drugs to slow the heart rate). The pulse also varies in amplitude from beat to beat in atrial fibrillation because of differing diastolic filling times. This type of pulse can occasionally be simulated by frequent irregularly occurring supraventricular or ventricular ectopic beats.

Patients with atrial fibrillation or frequent ectopic beats may have a detectable *pulse deficit*. This means the heart rate when counted by listening to the heart with the stethoscope is higher than that obtained when the radial pulse is counted at the wrist. In these patients the heart sounds will be audible with every systole, but some early contractions preceded by short diastolic filling periods will not produce enough cardiac output for a pulse to be palpable at the wrist.

An irregular rhythm can also be *regularly irregular*. For example, in patients with *sinus arrhythmia* the pulse rate increases with each inspiration and decreases with each expiration; this is a normal finding. It is associated

with changes in venous return to the heart.

Patterns of irregularity (Figure 4.14) can also occur when patients have frequent ectopic beats. These may arise in the atrium (atrial ectopic beats—AEBs) or in the ventricle (ventricular ectopic beats—VEBs). Ectopic beats quite commonly occur in a fixed ratio to normal beats. When every second beat is an ectopic one, the rhythm is called *bigeminy*. A bigeminal rhythm caused by ectopic beats has a characteristic pattern: normal pulse, weak pulse, delay, normal pulse, … . Similarly, every third beat may be ectopic —*trigeminy*. A pattern of irregularity is also detectable in the *Wenckebach phenomenon*.[m] Here the AV nodal conduction time increases progressively until a non-conducted atrial systole occurs. Following this, the AV conduction time shortens and the cycle begins again.

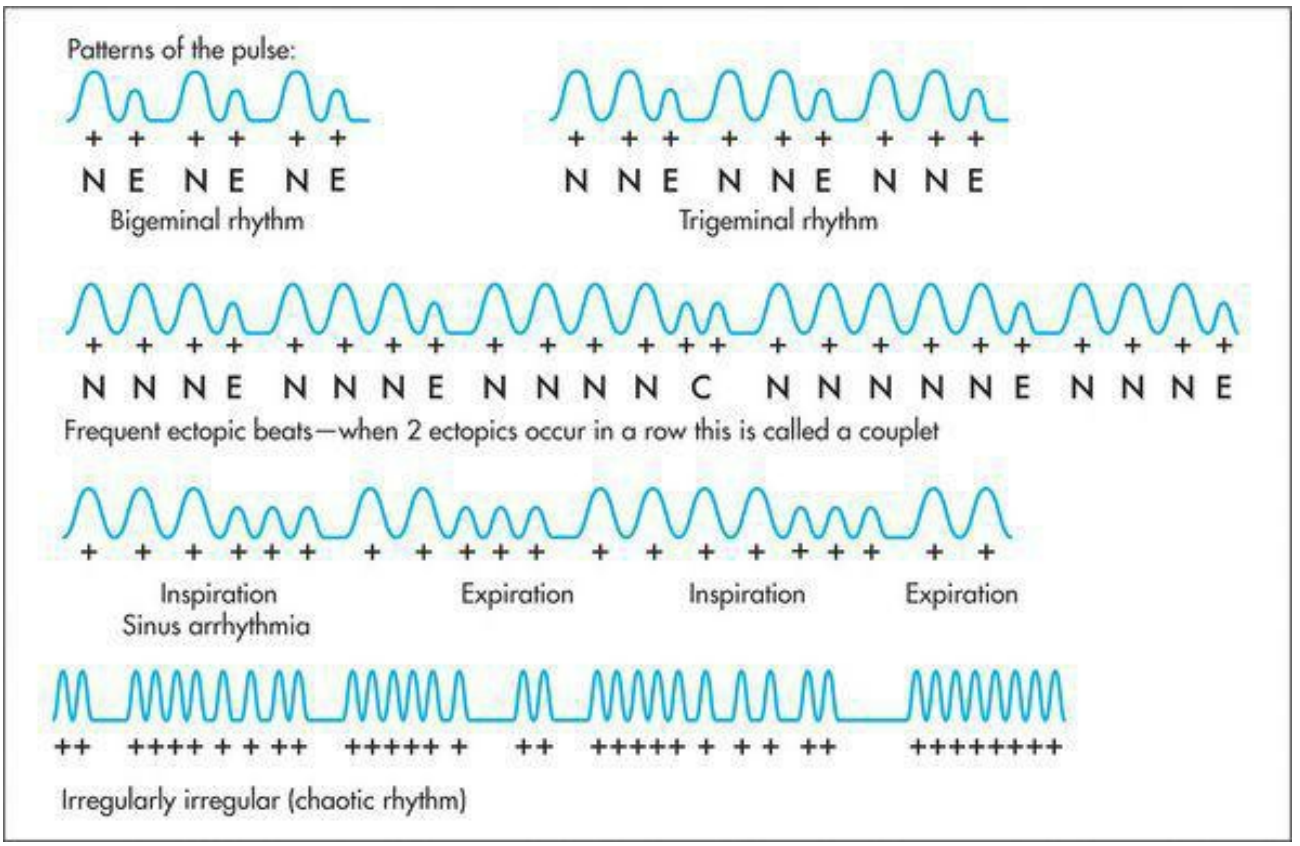


Figure 4.14 Common pulse patterns
N = normal; E = ectopic; C = couplet

Radiofemoral and radial–radial delay

This is an important sign and often neglected. While palpating the radial pulse, the clinician places the fingers of the other hand over the femoral pulse, which is situated below the inguinal ligament, one-third of the way up

from the pubic tubercle (Figure 4.15). A noticeable delay in the arrival of the femoral pulse wave suggests the diagnosis of *coarctation of the aorta*, where a congenital narrowing in the aortic isthmus occurs at the level where the ductus arteriosus joins the descending aorta. This is just distal to the origin of the subclavian artery. This lesion can cause upper limb hypertension.

Figure 4.15 Feeling for the radiofemoral delay

It can also be useful to palpate both radial pulses together to detect radial–radial inequality in timing or volume, usually due to a large arterial occlusion by an atherosclerotic plaque or aneurysm, or to subclavian artery stenosis on one side. It can also be a sign of dissection of the thoracic aorta.

Character and volume

These are poorly assessed by palpating the radial pulse; the carotid or brachial arteries should be used to determine the character and volume of the pulse, as these more accurately reflect the form of the aortic pressure wave. However, the collapsing (bounding) pulse of *aortic regurgitation*, and *pulsus alternans* (alternating strong and weak pulse) of advanced left ventricular failure, may be readily apparent in the radial pulse.

Condition of the vessel wall

Only changes in the medial layer of the radial artery can be assessed by palpation. Thickening or tortuosity will be detected commonly in the arteries of elderly people. These changes, however, do not indicate the presence of luminal narrowing due to atherosclerosis. Therefore, this sign is of little

clinical value.

The blood pressure

Measurement of the arterial blood pressure[n] is an essential part of the examination of almost any patient. Usually, indirect measurements of the systolic and diastolic pressures are obtained with a *sphygmomanometer* (Greek *sphygmos* 'pulsing', *manos* 'thin').[13] The systolic blood pressure is the peak pressure that occurs in the artery following ventricular systole, and the diastolic blood pressure is the level to which the arterial blood pressure falls during ventricular diastole. Normal blood pressure is defined as a systolic reading of less than 140 mmHg *and* a diastolic reading of less than 90 mmHg. In some circumstances, lower pressures may be considered normal (e.g. in pregnancy) or desirable (e.g. for diabetics).

Measuring the blood pressure with the sphygmomanometer

The usual blood pressure cuff width is 12.5 cm. This is suitable for a normal-sized adult forearm. However, in obese patients with large arms (up to 30% of the adult population) the normal-sized cuff will overestimate the blood pressure and therefore a large cuff must be used. A range of smaller sizes are available for children. Use of a cuff that is too large results in only a small underestimate of blood pressure.

The cuff is wrapped around the upper arm with the bladder centred over the brachial artery (Figure 4.16). This is found in the antecubital fossa, one-third of the way over from the medial epicondyle. For an approximate estimation of the systolic blood pressure, the cuff is fully inflated and then deflated slowly (3–4 mmHg per second) until the radial pulse returns. Then, for a more accurate estimation of the blood pressure, this manoeuvre is repeated with the diaphragm of the stethoscope placed over the brachial artery, slipped underneath the distal end of the cuff's bladder.

Figure 4.16 Measuring the blood pressure, with the patient lying at 45 degrees

The patient's brachial artery should be at about the level of the heart which is at the level of the fourth intercostal space at the sternum. If the arm is too high, e.g. at the level of the supraclavicular notch, the blood pressure reading will be about 5 mmHg lower; and if the arm is too low the reading will be higher than is accurate.

Five different sounds will be heard as the cuff is slowly released (Figure 4.17). These are called the Korotkoff[o] sounds. The pressure at which a sound is first heard over the artery is the systolic blood pressure (Korotkoff I). As deflation of the cuff continues, the sound increases in intensity (KII), then decreases (KIII), becomes muffled (KIV) and then disappears (KV). Different observers have used KIV and KV to indicate the level of the diastolic pressure. KV is probably the best measure. However, this provides a slight underestimate of the arterial diastolic blood pressure. Although diastolic pressure usually corresponds most closely to KV, in severe aortic regurgitation KIV is a more accurate indication. KV is absent in some normal people and KIV must then be used.

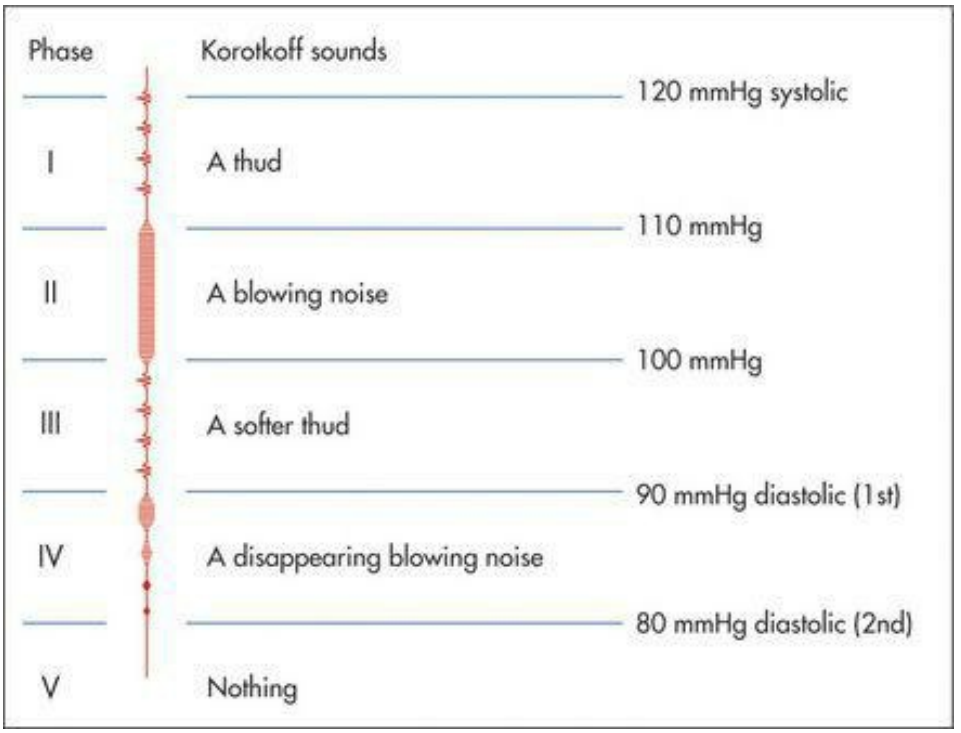


Figure 4.17 Korotkoff sounds
Systolic pressure is determined by the appearance of the first audible sound, and diastolic pressure is determined by its disappearance

pressure is determined by its disappearance.

Occasionally, there will be an auscultatory gap (the sounds disappear just below the systolic pressure and reappear before the diastolic pressure) in healthy people. This can lead to an underestimate of the systolic blood pressure if the cuff is not pumped up high enough.

The systolic blood pressure may normally vary between the arms by up to 10 mmHg; in the legs the blood pressure is normally up to 20 mmHg higher than in the arms, unless the patient has coarctation of the aorta. Measurement of the blood pressure in the legs is more difficult than in the arms. It requires a large cuff that is placed over the mid-thigh. The patient lies prone and the stethoscope is placed in the popliteal fossa, behind the knee.

During inspiration, the systolic and diastolic blood pressures normally decrease (because intrathoracic pressure becomes more negative, blood pools in the pulmonary vessels, so left-heart filling is reduced). When this normal reduction in blood pressure with inspiration is *exaggerated*, it is termed *pulsus paradoxus*. Kussmaul meant by this that there was a fall in blood pressure and a paradoxical rise in pulse rate. A fall in arterial pulse pressure on inspiration of more than 10 mmHg is abnormal and may occur with *constrictive pericarditis, pericardial effusion*, or severe asthma. To detect this: lower the cuff pressure slowly until KI sounds are heard intermittently (expiration) and then until KI is audible with every beat. The difference between the two readings represents the level of the pulsus paradoxus.

Variations in blood pressure

When blood pressure is measured with an intra-arterial catheter it becomes clear that blood pressure varies from minute to minute in normal people. Short-term changes of 4 mmHg in the systolic and 3 mmHg in the diastolic readings are common. Hour-to-hour and day-to-day variations are even greater. The standard deviation between visits is up to 12 mmHg for systolic pressure and 8 mmHg for diastolic. This means that when there is concern about an abnormal reading, repeat measurements are necessary.

When the heart is very irregular (most often because of atrial fibrillation), the cuff should be deflated slowly, and the point at which most of the cardiac contractions are audible (KI) taken as the systolic pressure and the point at which most have disappeared (KV) taken as diastole.

High blood pressure

This is difficult to define.[13] The most helpful definitions of hypertension are based on an estimation of the level associated with an increased risk of vascular disease. There have been many classifications of blood pressure, as what is considered normal or abnormal changes as more information comes to hand. Table 4.11 gives a useful guide to current definitions. If recordings above 140/90 mmHg are considered abnormal, high blood pressure may occur in up to 20% of the adult population.[p] Blood pressure measured by the patient at home, or by a 24-hour monitor, should be up to 10/5 mmHg less than that measured in the surgery.

TABLE 4.11 A classification of blood pressure readings[*]

Category	Systolic (mmHg)	Diastolic (mmHg)
Optimal	< 120	< 80
Normal	120–129	80–84
High normal	130–139	85–89
Mild hypertension (grade 1)	140–159	90–99

Moderate hypertension (grade 2)	160–179	100–109
Severe hypertension (grade 3)	> 180	> 110

* Khan NA, Rahim SA, Avand SS et al. Does the clinical examination predict lower extremity peripheral arterial disease? *JAMA* 2006 Feb 1; 295(5):536–546.

Postural blood pressure

The blood pressure should routinely be taken with the patient both lying down and standing (Figure 4.18).[15] A fall of more than 15 mmHg in systolic blood pressure or 10 mmHg in diastolic blood pressure on standing is abnormal and is called *postural hypotension* (Table 4.12). It may cause dizziness or not be associated with symptoms. The most common cause is the use of antihypertensive drugs, α-adrenergic antagonists in particular.

Figure 4.18 Measuring the blood pressure, with patient standing

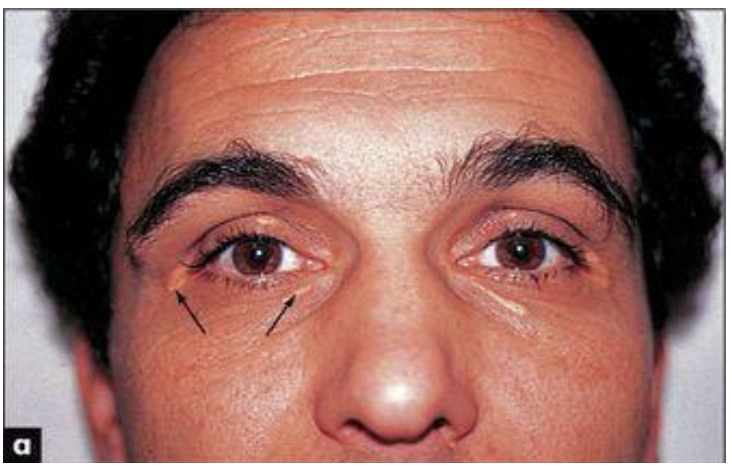
TABLE 4.12 Causes of postural hypotension (HANDI)

Hypovolaemia (e.g. dehydration, bleeding); **H**ypopituitarism
Addison's* disease
Neuropathy—autonomic (e.g. diabetes mellitus), amyloidosis, Shy-Drager syndrome)
Drugs (e.g. vasodilators and other antihypertensives, tricyclic antidepressants, diuretics, antipsychotics)
Idiopathic orthostatic hypotension (rare progressive degeneration of the autonomic nervous system, usually in elderly men)

* Thomas Addison (1793–1860), London physician.

The face

Inspect the sclerae for *jaundice* (page 25). This can occur with severe congestive cardiac failure and hepatic congestion. Prosthetic heart valve induced haemolysis of red blood cells, due to excessive turbulence, is an uncommon but cardiac cause of jaundice. *Xanthelasmata* (Figure 4.19) are intracutaneous yellow cholesterol deposits around the eyes and are relatively common. These may be a normal variant or may indicate type II or III hyperlipidaemia, though they are not always associated with hyperlipidaemia.



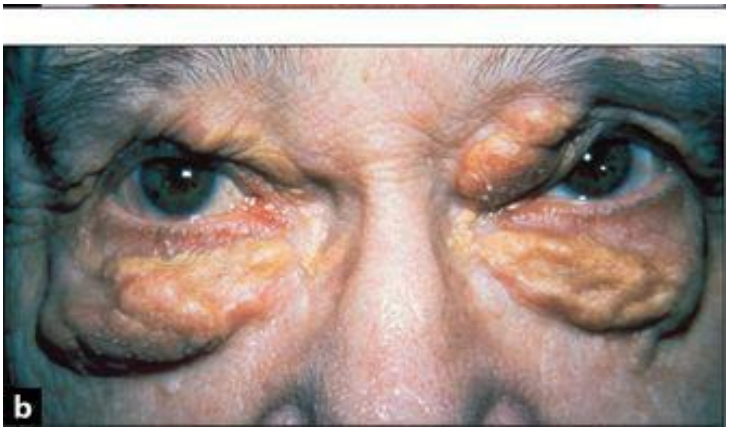


Figure 4.19 Xanthelasmata

Figure b from McDonald FS, ed., Mayo Clinic images in internal medicine, with permission. ©Mayo Clinic Scientific Press and CRC Press.

Look at the pupils for an *arcus senilis* (Figure 4.20). This half or complete grey circle is seen around the outer perimeter of the pupil and is probably associated with some increase in cardiovascular risk.[9]

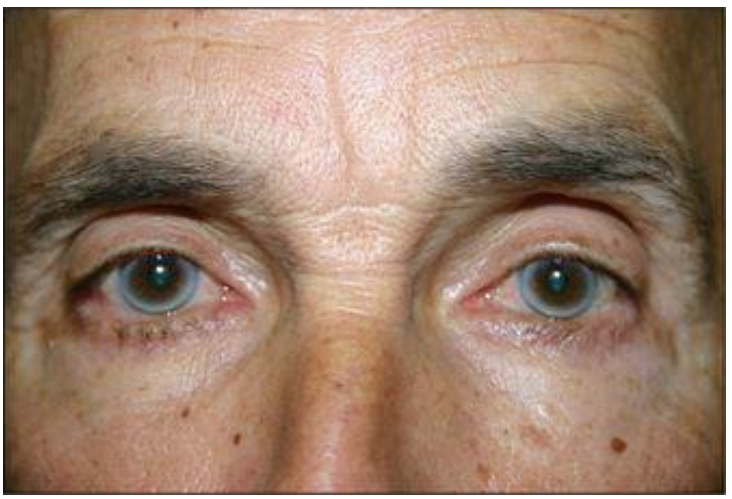


Figure 4.20 Arcus senilis

Next look for the presence of a *mitral facies*, which refers to rosy cheeks with a bluish tinge due to dilatation of the malar capillaries. This is associated with pulmonary hypertension and a low cardiac output such as occurs in severe mitral stenosis, and is now rare.

Now look in the mouth using a torch to see if there is a *high arched palate*. This occurs in Marfan's syndrome, a condition that is associated with congenital heart disease, including aortic regurgitation secondary to aortic root dilatation, and also mitral regurgitation due to mitral valve prolapse. Notice whether the teeth look diseased, as they can be a source of organisms responsible for infective endocarditis. Look at the tongue and lips for central

cyanosis. Inspect the mucosa for petechiae that may indicate infective endocarditis.

The neck

Oddly enough, this small area of the body is packed with cardiovascular signs which must be elicited with great care and skill.

Carotid arteries

The carotids are not only easily accessible, medial to the sternomastoid muscles (Figure 4.21), but provide a great deal of information about the wave form of the aortic pulse, which is affected by many cardiac abnormalities. Never palpate both carotid arteries simultaneously as they provide much of the blood supply to the *brain* (a vital organ).

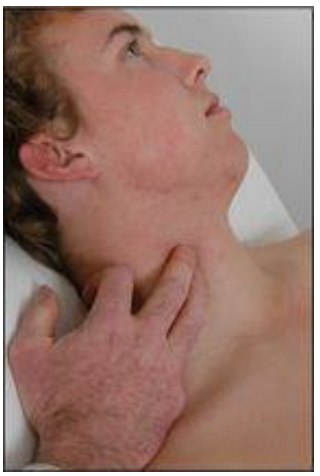


Figure 4.21 Palpating the carotid pulse

Evaluation of the pulse wave form (the amplitude, shape and volume) is important in the diagnosis of various underlying cardiac diseases and in assessing their severity. It takes considerable practice to distinguish the different important types of carotid wave forms (Table 4.13). Auscultation of the carotids may be performed now or in association with auscultation of the praecordium.

TABLE 4.13 Arterial pulse character

Type of pulse	**Cause(s)**
Anacrotic	Aortic stenosis
	Small volume, slow uptake, notched wave on upstroke
Plateau	Aortic stenosis
	Slow upstroke
Bisferiens	Aortic stenosis *and* regurgitation
	Anacrotic and collapsing
Collapsing	Aortic regurgitation
	Hyperdynamic circulation
	Patent ductus arteriosus
	Peripheral arteriovenous fistula
	Arteriosclerotic aorta (elderly patients in particular)
Small volume	Aortic stenosis
	Pericardial effusion

Alternans	Left ventricular failure
	Alternating strong and weak beats

Jugular venous pressure (JVP)—pulsation

Just as the carotid pulse tells us about the aorta and left ventricular function, the *jugular venous pressure* (JVP) (Figure 4.5, page 47) tells us about right atrial and right ventricular function.[16] The positioning of the patient and lighting are important for this examination to be done properly. The patient must be lying down at 45 degrees to the horizontal with his or her head on pillows and in good lighting conditions. This is a difficult examination and there is considerable inter- (and intra-)observer variation in the findings.

When the patient is lying at 45 degrees, the sternal angle is also roughly in line with the base of the neck (Figure 4.5c). This provides a convenient zero point from which to measure the vertical height of the column of blood in the jugular vein. The jugular venous pulsation (movement) can be distinguished from the arterial pulse because: (i) it is visible but not palpable and has a more prominent *inward* movement than the artery; (ii) it has a complex wave form, usually seen to flicker twice with each cardiac cycle (if the patient is in sinus rhythm); (iii) it moves on respiration—normally the JVP decreases on inspiration; and (iv) it is at first obliterated and then filled from above when light pressure is applied at the base of the neck.

The JVP must be assessed for *height* and *character*. When the JVP is more than 3 cm above the zero point, the right-heart filling pressure is raised (a normal reading is less than 8 cm of water: 5 cm + 3 cm). This is a sign of right ventricular failure, volume overload or of some types of pericardial disease.

The assessment of the *character* of JVP is difficult even for experienced clinicians. There are two positive waves in the normal JVP.[r] The first is called the *a wave* and coincides with right atrial systole.[s] It is due to atrial contraction. The *a* wave also coincides with the first heart sound and precedes the carotid pulsation. The second impulse is called the *v wave* and is due to atrial filling, in the period when the tricuspid valve remains closed during ventricular systole. Between the *a* and *v* waves there is a trough caused by atrial relaxation. This is called the *x descent*. It is interrupted by the *c point*, which is due to transmitted carotid pulsation and coincides with tricuspid valve closure; it is not usually visible. Following the *v* wave, the tricuspid valve opens and rapid ventricular filling occurs; this results in the *y descent* (Figure 4.22).

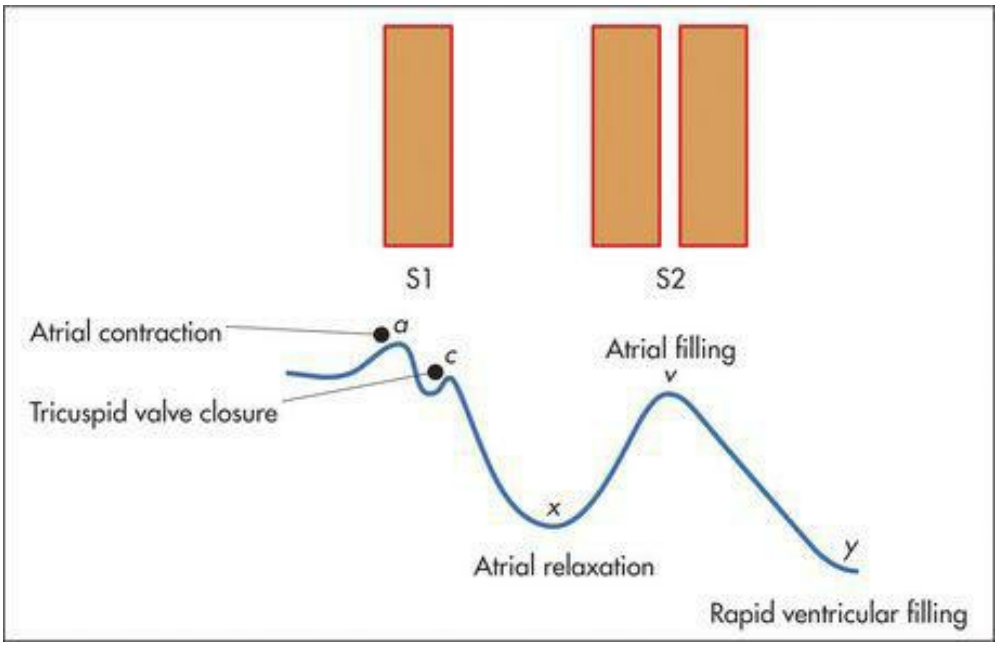


Figure 4.22 The jugular venous pressure, and its relationship to the first (S1) and second (S2) heart sounds

In Table 4.14, characteristic changes in the JVP are described. Any condition in which right ventricular filling is limited (e.g. *constrictive pericarditis, cardiac tamponade* or *right ventricular infarction*) can cause elevation of the venous pressure, which is more marked on inspiration when venous return to the heart increases. This rise in the JVP on inspiration, called *Kussmaul's*[†] sign, is the opposite of what normally happens. This sign is best elicited with the patient sitting up at 90 degrees and breathing quietly through the mouth.

TABLE 4.14 Jugular venous pressure (pulse)