Causes of an elevated central venous pressure
Right ventricular failure
Tricuspid stenosis or regurgitation
Pericardial effusion or constrictive pericarditis
Superior vena caval obstruction
Fluid overload
Hyperdynamic circulation
Wave form
Causes of a dominant *a* wave
Tricuspid stenosis (also causing a slow *y* descent)
Pulmonary stenosis
Pulmonary hypertension
Causes of cannon *a* waves
Complete heart block
Paroxysmal nodal tachycardia with retrograde atrial conduction
Ventricular tachycardia with retrograde atrial conduction or atrioventricular dissociation
Cause of a dominant *v* wave
Tricuspid regurgitation
***x* descent**
Absent: atrial fibrillation
Exaggerated: acute cardiac tamponade, constrictive pericarditis

***y*descent**
Sharp: severe tricuspid regurgitation, constrictive pericarditis
Slow: tricuspid stenosis, right atrial myxoma

The abdominojugular reflux test (*hepatojugular reflux*) is a way of testing for right or left ventricular failure or reduced right ventricular compliance.[17] Pressure exerted over the middle of the abdomen for 10 seconds will increase venous return to the right atrium. The JVP normally rises transiently following this manoeuvre.[u] If there is right ventricular failure or left atrial pressures are elevated (left ventricular failure), it may remain elevated (>4cm) for the duration of the compression—a positive hepatojugular reflux. The sudden fall in the JVP (>4 cm) as the pressure is released may be easier to see than the initial rise. It is not necessary to compress the liver and so the older name, *hepatojugular reflux*, is not so appropriate. It is important that the patient be relaxed, breathe through the mouth and not perform a Valsalva manoeuvre. The examiner should press firmly with the palm over the middle of the abdomen. It is not necessary to apply pressure for more than 10 seconds.

Cannon a waves occur when the right atrium contracts against the closed tricuspid valve. This occurs intermittently in complete heart block where the two chambers beat independently.

Giant a waves are large but not explosive *a* waves with each beat. They occur when right atrial pressures are raised because of elevated pressures in the pulmonary circulation or obstruction to outflow (tricuspid stenosis).

The *large v waves* of tricuspid regurgitation should never be missed. They are a reliable sign of tricuspid regurgitation and are visible welling up into the neck during each ventricular systole.

The praecordium

Now at last the examiner has reached the praecordium.

Inspection

Inspect first for scars. Previous cardiac operations will have left scars on the chest wall. The position of the scar can be a clue to the valve lesion that has been operated on. Most valve surgery requires cardiopulmonary bypass and for this a *median sternotomy* (a cut down the middle of the sternum) is very commonly used. This type of scar is occasionally hidden under a forest of chest hair. It is not specifically helpful, as it may also be a result of previous

coronary artery bypass grafting. Alternatively, left- or even right-sided lateral thoracotomy scars, which may be hidden under a pendulous breast, may indicate a previous closed mitral valvotomy. In this operation a stenosed mitral valve is opened through an incision made in the left atrial appendage; cardiopulmonary bypass is not required. Coronary artery bypass grafting and even valve surgery are now sometimes performed using small lateral 'port' incisions for video-assisted instruments.

Skeletal abnormalities such as *pectus excavatum* (funnel chest, page 121) or *kyphoscoliosis* (Greek *kyphos* 'hunchbacked', *skolios* 'curved'), a curvature of the vertebral column (page 121), may be present. Skeletal abnormalities such as these, which may be part of Marfan's syndrome, can cause distortion of the position of the heart and great vessels in the chest and thus alter the position of the apex beat. Severe deformity can interfere with pulmonary function and cause pulmonary hypertension (page 81).

Another surgical 'abnormality' that must not be missed, if only to avoid embarrassment, is a pacemaker or cardioverter-defibrillator box. These are usually under the right or left pectoral muscle just below the clavicle, are usually easily palpable and obviously metallic. The pacemaker leads may be palpable under the skin, leading from the top of the box. The box is normally mobile under the skin. Fixation of the skin to the box or stretching of the skin over the box may be an indication for repositioning. Erosion of the box through the skin is a serious complication because of the inevitable infection that will occur around this foreign body. Rarely, a loose lead connection will lead to twitching of the muscles of the chest wall around the box. Penetration of the right ventricular lead into or through the right ventricular wall may lead to disconcerting paced diaphragmatic contractions (hiccups) at whatever rate the pacemaker is set. Defibrillator boxes are larger than pacemakers. They are currently about 10 × 5 cm and a little less than 1 cm thick.

Look for the apex beat. Its normal position is in the fifth left intercostal space, 1 cm medial to the midclavicular line (Figure 4.23). It is due primarily to recoil of the heart as blood is expelled in systole. There may be other visible pulsations—for example, over the pulmonary artery in cases of severe pulmonary hypertension.

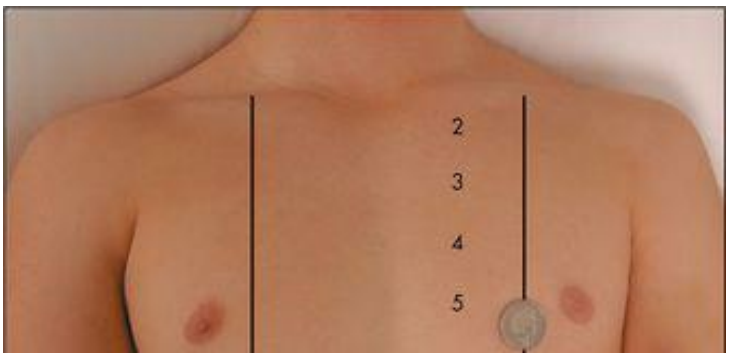


Figure 4.23 The apex beat
Coin is over the apex. Intercostal spaces are numbered. Vertical lines show right and left midclavicular and left anterior axillary lines. Care must be taken in identifying the midclavicular line; the inter-observer variability can be as much as 10 cm!

Palpation

The *apex beat* must be palpated (Figures 4.23 and 4.24).[18] It is important to count down the number of interspaces. The first palpable interspace is the second. It lies just below the manubriosternal angle. The position of the apex beat is defined as the most lateral and inferior point at which the palpating fingers are raised with each systole. The normal apex is felt over an area the size of a 20 cent (50 p) coin (Figure 4.23). Use firm pressure with the tips of the fingers into the rib interspaces. The heel of the examiner's hand is lifted off the patient's sternum. Note that the apex beat is palpable in only about 50% of adults.

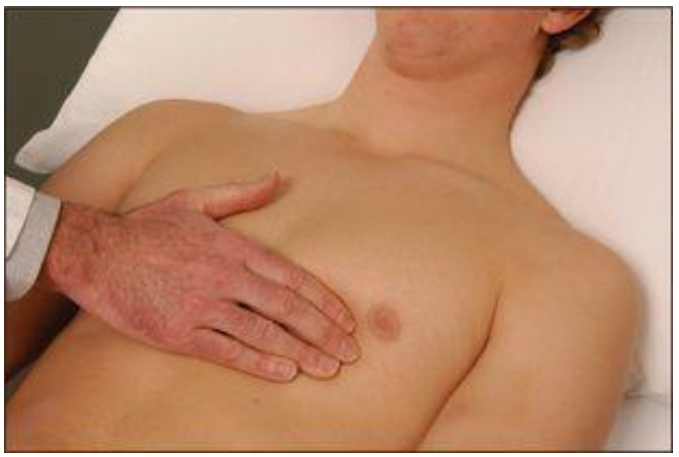


Figure 4.24 Feeling for the apex beat

It is worth noting that the palpable apex beat is not the anatomical apex of the heart but a point above it. At the time the apex beat is palpable,[v] the heart is assuming a more spherical shape and the apex is twisting away from the chest wall. The area above the apex, however, is moving closer to the chest and is palpable. If the apex beat is displaced laterally or inferiorly, or both, this usually indicates enlargement,[18] but may sometimes be due to chest wall deformity, or pleural or pulmonary disease (page 121).

wall deformity, or pleural or pulmonary disease (page 121).

The character of the apex beat may provide the examiner with vital diagnostic clues. The normal apex beat gently lifts the palpating fingers. There are a number of types of abnormal apex beats. The *pressure loaded* (heaving, hyperdynamic or systolic overloaded) apex beat is a forceful and sustained impulse. This occurs with aortic stenosis or hypertension. The *volume loaded* (thrusting) apex beat is a displaced, diffuse, non-sustained impulse. This occurs most commonly in advanced mitral regurgitation or dilated cardiomyopathy. The *dyskinetic* apex beat is an uncoordinated impulse felt over a larger area than normal in the praecordium and is usually due to left ventricular dysfunction (e.g. in anterior myocardial infarction). The *double impulse* apex beat, where two distinct impulses are felt with each systole, is characteristic of hypertrophic cardiomyopathy (page 91). The *tapping* apex beat will be felt when the first heart sound is actually palpable (heart sounds are not palpable in health) and indicates mitral or very rarely tricuspid stenosis. The character, but not the position, of the apex beat may be more easily assessed when the patient lies on the left side.

In many patients the apex beat may not be palpable. This is most often due to a thick chest wall, emphysema, pericardial effusion, shock (or death) and rarely to dextrocardia (where there is inversion of the heart and great vessels). The apex beat will be palpable to the right of the sternum in many cases of dextrocardia.

Other praecordial impulses may be palpable in patients with heart disease. A *parasternal impulse* may be felt when the heel of the hand is rested just to the left of the sternum with the fingers lifted slightly off the chest (Figure 4.25). Normally no impulse or a slight inward impulse is felt. In cases of right ventricular enlargement or severe left atrial enlargement, where the right ventricle is pushed anteriorly, the heel of the hand is lifted off the chest wall with each systole. Palpation with the fingers over the pulmonary area may reveal the palpable tap of pulmonary valve closure (*palpable* P2) in cases of pulmonary hypertension (Figure 4.26).

Figure 4.25 Feeling for the parasternal impulse

Figure 4.25 Feeling for the parasternal impulse

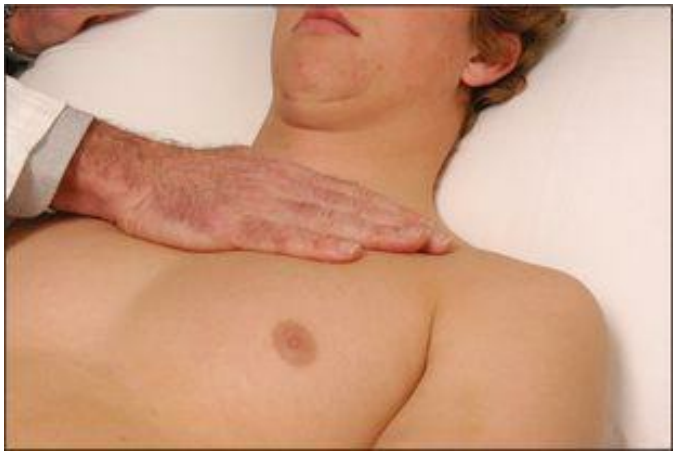


Figure 4.26 Palpating the base of the heart

Turbulent blood flow, which causes cardiac murmurs on auscultation, may sometimes be palpable. These palpable murmurs are called *thrills*. The praecordium should be systematically palpated for thrills with the flat of the hand, first over the apex and left sternal edge, and then over the *base of the heart* (this is the upper part of the chest and includes the aortic and pulmonary areas) (Figure 4.26).

Apical thrills can be more easily felt with the patient rolled over to the left side (the left lateral position) as this brings the apex closer to the chest wall. Thrills may also be palpable over the base of the heart. These may be maximal over the pulmonary or aortic areas, depending on the underlying cause, and are best felt with the patient sitting up, leaning forwards and in full expiration. In this position the base of the heart is moved closer to the chest wall. A thrill that coincides in time with the apex beat is called a *systolic thrill*; one that does not coincide with the apex beat is called a *diastolic thrill*.

The presence of a thrill usually indicates an organic lesion. Careful palpation for thrills is a useful, but often neglected, part of the cardiovascular examination.

Percussion

It is possible to define the cardiac outline by means of percussion[w] but this is not routine (page 124).[19] Percussion is most accurate when performed in the fifth intercostal space. The patient should lie supine and the examiner percusses from the anterior axillary line towards the sternum. The point at

which the percussion note becomes dull represents the left heart border. A distance of more than 10.5 cm between the border of the heart and the middle of the sternum indicates cardiomegaly. The sign is not useful in the presence of lung disease.

Auscultation

Now at last the stethoscope is required.[20] However, in some cases the diagnosis should already be fairly clear. In the viva voce examination, the examiners will occasionally stop a candidate before auscultation and ask for an opinion.

Auscultation of the heart begins in the mitral area with the bell of the stethoscope (Figures 4.1, page 45, and 4.27). The bell is designed as a resonating chamber and is particularly efficient in amplifying low-pitched sounds, such as the diastolic murmur of mitral stenosis or a third heart sound. It must be applied to the chest wall lightly, because forceful application will stretch the skin under the bell so that it forms a diaphragm. Some modern stethoscopes do not have a separate bell; the effect of a bell is produced when the diaphragm is placed lightly on the chest, and of a diaphragm when it is pushed more firmly.

Figure 4.27 Auscultation in the mitral area with the bell of the stethoscope Listening for mitral stenosis in the left lateral position.

Next, listen in the mitral area with the diaphragm of the stethoscope (Figure 4.28), which best reproduces higher-pitched sounds, such as the systolic murmur of mitral regurgitation or a fourth heart sound. Then place the stethoscope in the tricuspid area (fifth left intercostal space) and listen. Next inch up the left sternal edge to the pulmonary (second left intercostal space) and aortic (second right intercostal space) areas (Figures 4.29 and

space) and aortic (second right intercostal space) areas (Figures 4.29 and 4.30), listening carefully in each position with the diaphragm.

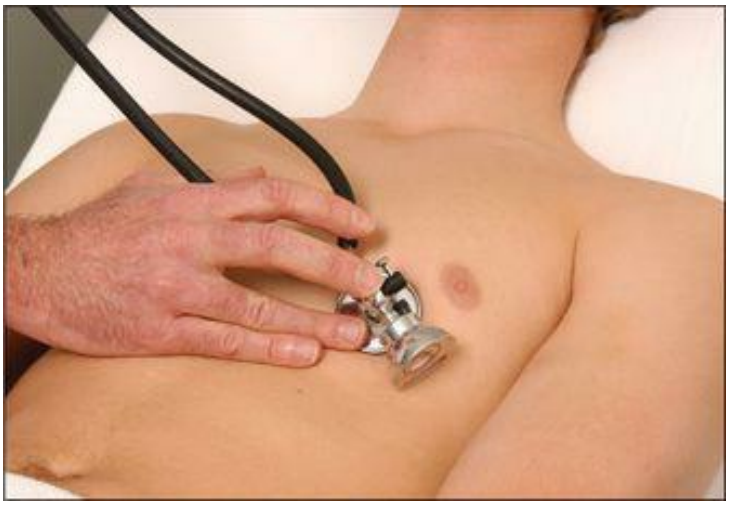


Figure 4.28 Auscultation at the apex with the diaphragm of the stethoscope

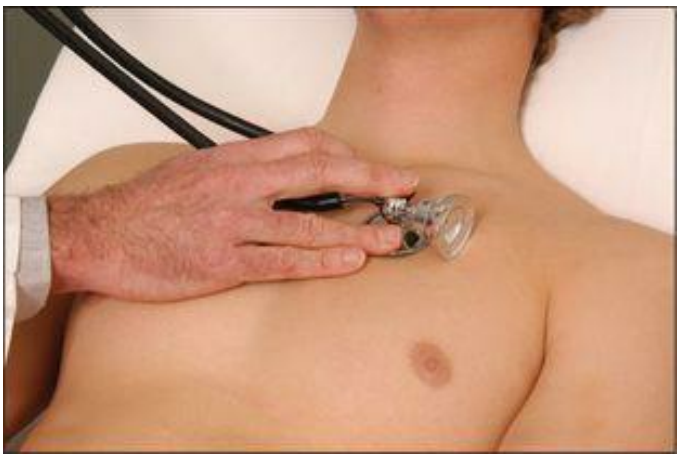


Figure 4.29 Auscultation at the base of the heart (pulmonary area)

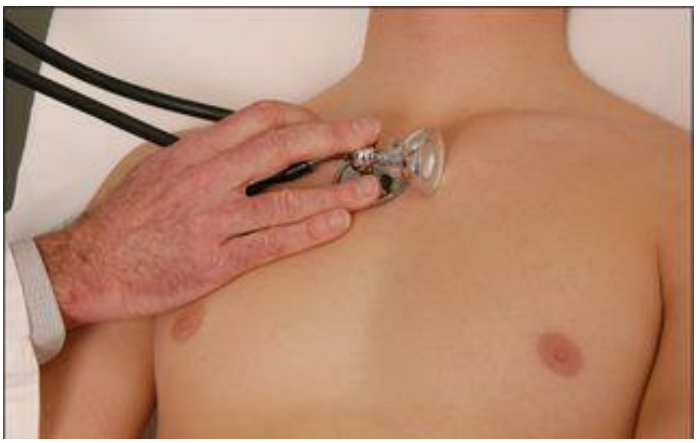


Figure 4.30 Auscultation at the base of the heart (aortic area)

For accurate auscultation, experience with what is normal is important. This can be obtained only through constant practice. Auscultation of the normal heart reveals two sounds called, not surprisingly, the first and second heart sounds. The explanation for the origin of these noises changes from year to year; the sounds are probably related to vibrations caused by the closing of the heart valves in combination with rapid changes in blood flow and tensing within cardiac structures that occur as the valves close.

The *first heart sound* (S1) has two components: mitral and tricuspid valve closure. Mitral closure occurs slightly before tricuspid, but usually only one sound is audible. The first heart sound indicates the beginning of ventricular systole.

The *second heart sound* (S2), which is softer, shorter and at a slightly higher pitch than the first and marks the end of systole, is made up of sounds arising from aortic and pulmonary valve closures. In normal cases, although left and right ventricular systole end at the same time, the lower pressure in the pulmonary circulation compared with the aorta means that flow continues into the pulmonary artery after the end of right ventricular systole. As a result, closure of the pulmonary valve occurs later than that of the aortic valve. These components are usually (in 70% of normal adults) sufficiently separated in time so that splitting of the second heart sound is audible. Because the pulmonary component of the second heart sound (P2) may not be audible throughout the praecordium, splitting of the second heart sound may best be appreciated in the pulmonary area and along the left sternal edge. Pulmonary valve closure is further delayed (by 20 or 30 milliseconds) with inspiration because of increased venous return to the right ventricle; thus, splitting of the second heart sound is wider on inspiration. The second heart sound marks the beginning of diastole, which is usually longer than systole.

It can be difficult to decide which heart sound is which. Palpation of the carotid pulse will indicate the timing of systole and enable the heart sounds to be more easily distinguished. It is obviously crucial to define systole and diastole during auscultation so that cardiac murmurs and abnormal sounds can be placed in the correct part of the cardiac cycle. Students are often asked to time a cardiac murmur; this is not a request to measure its length, but rather to say in which part of the cardiac cycle it occurs. Even the experts can mistake a murmur if they do not time it. It is important, during auscultation, to concentrate separately on the components of the cardiac cycle. The clinician should attempt to identify each and listen for abnormalities. There can be more than 12 components to identify in patients

with heart disease. An understanding of the cardiac cycle is helpful when interpreting the auscultatory findings (Figure 4.31).

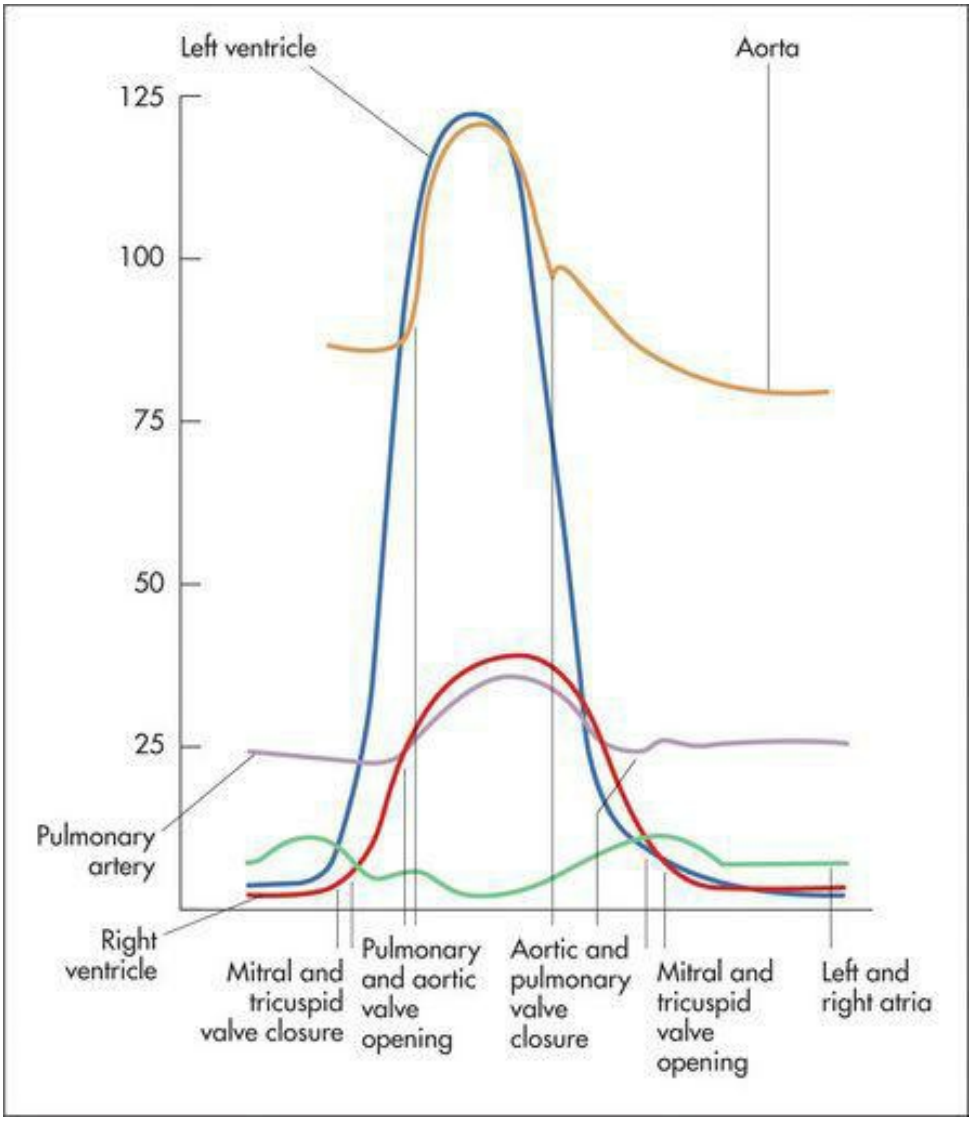


Figure 4.31 The cardiac cycle
Normally the onset of left ventricular systole precedes the onset of pressure rise in the right ventricle. The mitral valve, therefore, closes before the tricuspid valve. Because the pulmonary artery diastolic pressure is lower than aortic diastolic pressure, the pulmonary valve opens before the aortic valve. Therefore, pulmonary ejection sounds occur closer to the first heart sound than do aortic ejection sounds. During systole the pressure in the ventricles slightly exceeds the pressure in the corresponding great arteries. Towards the end of systole, the ventricular pressure falls below the pressure in the great arteries, and when diastolic pressure is reached the semilunar valves close. Normally, aortic valve closure precedes pulmonary valve closure. The mitral and tricuspid valves begin to open at the point at which the ventricular pressures fall below the corresponding atrial pressures.

Adapted from Swash M, ed. Hutchison's clinical methods, 20th edn. Philadelphia: Baillière Tindall, 1995, with permission.

Abnormalities of the heart sounds

Alterations in intensity

The **first heart sound** (S1) is *loud* when the mitral or tricuspid valve cusps remain wide open at the end of diastole and shut forcefully with the onset of ventricular systole. This occurs in mitral stenosis because the narrowed valve orifice limits ventricular filling so that there is no diminution in flow towards the end of diastole. The normal mitral valve cusps drift back towards the closed position at the end of diastole as ventricular filling slows down. Other causes of a loud S1 are related to reduced diastolic filling time (e.g. tachycardia or any cause of a short atrioventricular conduction time).

Soft first heart sounds can be due to a prolonged diastolic filling time (as with first-degree heart block) or a delayed onset of left ventricular systole (as with left bundle branch block), or to failure of the leaflets to coapt normally (as in mitral regurgitation).

The **second heart sound** (S2) will have a *loud* aortic component (A2) in patients with systemic hypertension. This results in forceful aortic valve closure secondary to high aortic pressures. Congenital aortic stenosis is another cause, because the valve is mobile but narrowed, and closes suddenly at the end of systole. The pulmonary component of the second heart sound (P2) is traditionally said to be *loud* in pulmonary hypertension, where the valve closure is forceful because of the high pulmonary pressure. In fact, a palpable P2 correlates better with raised pulmonary pressures than a loud P2.[21]

A *soft* A2 will be found when the aortic valve is calcified and leaflet movement is reduced, and in aortic regurgitation when the leaflets cannot coapt.

Splitting

Splitting of the heart sound is usually most obvious during auscultation in the pulmonary area. Splitting of the **first heart sound** is usually not detectable clinically; however, when it occurs it is most often due to the cardiac conduction abnormality known as complete right bundle branch block.

Increased normal splitting (wider on inspiration) of the **second heart sound** occurs when there is any delay in right ventricular emptying, as in right bundle branch block (delayed right ventricular depolarisation), pulmonary stenosis (delayed right ventricular ejection), ventricular septal

pulmonary stenosis (delayed right ventricular ejection), ventricular septal defect (increased right ventricular volume load), and mitral regurgitation (because of earlier aortic valve closure, due to more rapid left ventricular emptying).

In the case of *fixed splitting* of the second heart sound, there is no respiratory variation (as is normal) and splitting tends to be wide. This is caused by an atrial septal defect where equalisation of volume loads between the two atria occurs through the defect. This results in the atria acting as a common chamber.

In the case of *reversed splitting*, P2 occurs first and splitting occurs in expiration. This can be due to delayed left ventricular depolarisation (left bundle branch block), delayed left ventricular emptying (severe aortic stenosis, coarctation of the aorta) or increased left ventricular volume load (large patent ductus arteriosus). However, in the last-mentioned, the loud machinery murmur means that the second heart sound is usually not heard.

Extra heart sounds

The **third heart sound** (S3) is a low-pitched (20–70 Hz) mid-diastolic sound that is best appreciated by listening for a triple rhythm.[22] Its low pitch makes it more easily heard with the bell of the stethoscope. It has been likened (rather accurately) to the galloping of a horse and is often called a *gallop rhythm*. Its cadence is similar to that of the word 'Kentucky'. It is more likely to be appreciated if the clinician listens not to the individual heart sounds but to the rhythm of the heart. It is probably caused by tautening of the mitral or tricuspid papillary muscles at the end of rapid diastolic filling, when blood flow temporarily stops. A physiological left ventricular S3 sometimes occurs in children and young people and is due to very rapid diastolic filling. A pathological S3 is due to reduced ventricular compliance, so that a filling sound is produced even when diastolic filling is not especially rapid. It is strongly associated with increased atrial pressure.

A *left ventricular* S3 is louder at the apex than at the left sternal edge, and is louder on expiration. It can be associated with an increased cardiac output, as occurs in pregnancy and thyrotoxicosis. Otherwise, it is an important sign of left ventricular failure and dilatation, but may also occur in aortic regurgitation, mitral regurgitation, ventricular septal defect and patent ductus arteriosus.[23]

A *right ventricular* S3 is louder at the left sternal edge and with inspiration. It occurs in right ventricular failure or constrictive pericarditis.

The **fourth heart sound** (S4) is a late diastolic sound pitched slightly higher than the S3.[24] The cadence of an S4 is similar to that of the word 'Tennessee'. Again, this is responsible for the impression of a triple (gallop)

rhythm. It is due to a high-pressure atrial wave reflected back from a poorly compliant ventricle. It does not occur if the patient is in atrial fibrillation, because the sound depends on effective atrial contraction, which is lost when the atria fibrillate. Its low pitch means that unlike a split first heart sound it disappears if the bell of the stethoscope is pressed firmly onto the chest.

A *left ventricular* S4 occurs whenever left ventricular compliance is reduced due to aortic stenosis, acute mitral regurgitation, systemic hypertension, ischaemic heart disease or advanced age. It is often present during an episode of angina or with a myocardial infarction, and may be the only physical sign of that condition.

A *right ventricular* S4 occurs when right ventricular compliance is reduced in pulmonary hypertension or pulmonary stenosis.

If the heart rate is greater than 120 per minute, S3 and S4 may be superimposed, resulting in a **summation gallop**. In this case, two inaudible sounds may combine to produce an audible one. This does not necessarily imply ventricular stress, unless one or both of the extra heart sounds persists when the heart rate slows or is slowed by carotid sinus massage. When both S3 and S4 are present, the rhythm is described as a quadruple rhythm. It usually implies severe ventricular dysfunction.

Additional sounds

An **opening snap** is a high-pitched sound that occurs in mitral stenosis at a variable distance after S2. It is due to the sudden opening of the mitral valve and is followed by the diastolic murmur of mitral stenosis. It can be difficult to distinguish from a widely split S2, but normally occurs rather later in diastole than the pulmonary component of the second heart sound. It is pitched higher than a third heart sound and so is not usually confused with this. It is best heard at the lower left sternal edge with the diaphragm of the stethoscope. Use of the term ‘opening snap’ implies the diagnosis of mitral or rarely of tricuspid stenosis.

A **systolic ejection click** is an early systolic high-pitched sound that is heard over the aortic or pulmonary and left sternal edge areas, and which may occur in cases of congenital aortic or pulmonary stenosis where the valve remains mobile; it is followed by the systolic ejection murmur of aortic or pulmonary stenosis. It is due to the abrupt doming of the abnormal valve early in systole.

A **non-ejection systolic click** is a high-pitched sound heard during systole and is best appreciated at the mitral area. It is a common finding. It may be followed by a systolic murmur. The click may be due to prolapse of one or more redundant mitral valve leaflets during systole. Non-ejection

clicks may also be heard in patients with *atrial septal defects* or *Ebstein's anomaly* (page 89).

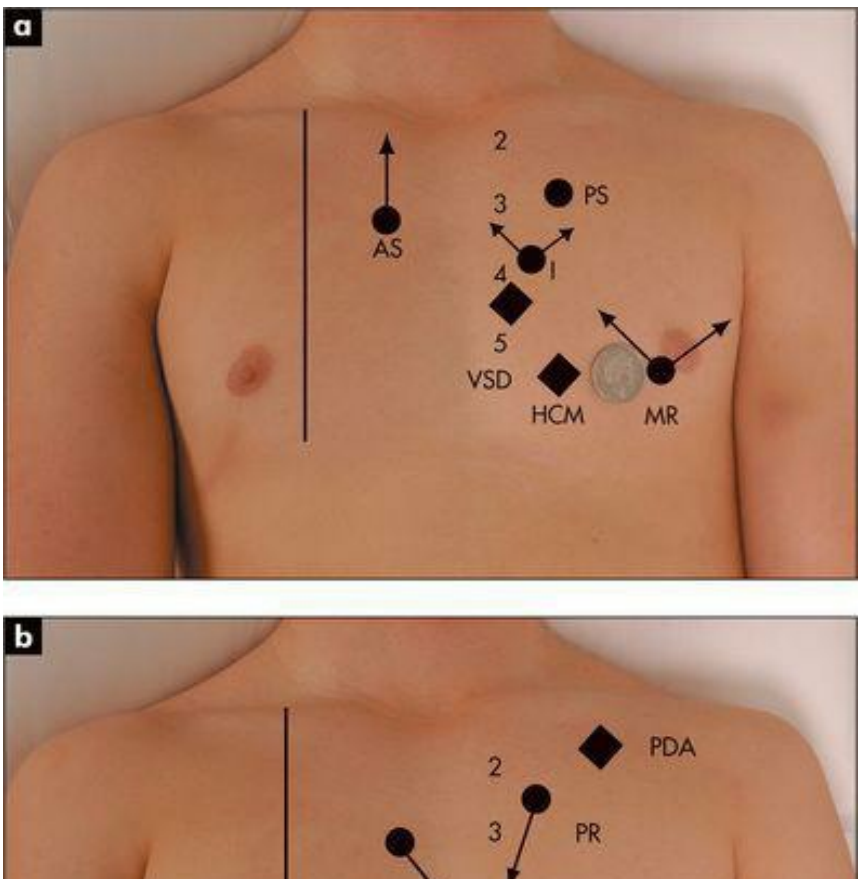
An *atrial myxoma* is a very rare tumour which may occur in either atrium. During atrial systole a loosely pedunculated tumour may be propelled into the mitral or tricuspid valve orifice causing an early diastolic plopping sound, a **tumour plop**. This sound is only rarely heard even in patients with a myxoma (about 10%).

A **diastolic pericardial knock** may occur when there is sudden cessation of ventricular filling because of constrictive pericardial disease.[25]

Prosthetic heart valves produce characteristic sounds (page 90). Rarely, a right ventricular pacemaker produces a late diastolic high-pitched click due to contraction of the chest wall muscles (the **pacemaker sound**).[26]

Murmurs of the heart

In deciding the origin of a cardiac murmur, a number of different features must be considered. These are: timing, the area of greatest intensity, the loudness and pitch, associated features (peripheral signs), and the effect of dynamic manoeuvres, including respiration and the Valsalva manoeuvre (Figure 4.32). The presence of a characteristic murmur is very reliable for the diagnosis of certain valvular abnormalities, but for others less so.



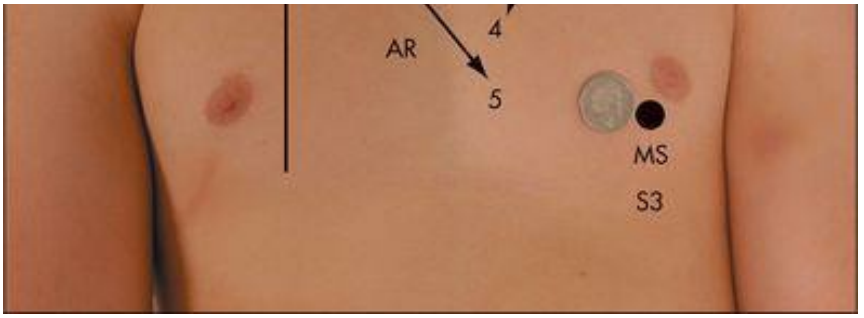


Figure 4.32 Sites of maximum intensity and radiation of murmurs and heart sounds

(a) Systolic murmurs:
AS = aortic stenosis;
MR = mitral regurgitation;
HCM = hypertrophic cardiomyopathy;
PS = pulmonary stenosis;
VSD = ventricular septal defect;
I = innocent.

(b) Diastolic murmurs and sounds:
AR = aortic regurgitation;
MS = mitral stenosis;
S3 = third heart sound;
PR = pulmonary regurgitation;
PDA = patent ductus arteriosus (continuous murmur).

Associated features

As already mentioned, the cause of a cardiac murmur can sometimes be elicited by careful analysis of the peripheral signs.

Timing (Table 4.15)

Systolic murmurs (which occur during ventricular systole) may be pansystolic, ejection systolic or late systolic.

TABLE 4.15 Cardiac murmurs

Timing	Lesion
Pansystolic	Mitral regurgitation
	Tricuspid regurgitation
	Ventricular septal defect
	Aortopulmonary shunts
Midsystolic	Aortic stenosis
	Pulmonary stenosis
	Hypertrophic cardiomyopathy
	Pulmonary flow murmur of an atrial septal defect

Late systolic	Mitral valve prolapse
	Papillary muscle dysfunction (due usually to ischaemia or hypertrophic cardiomyopathy)
Early diastolic	Aortic regurgitation
	Pulmonary regurgitation
Mid-diastolic	Mitral stenosis
	Tricuspid stenosis
	Atrial myxoma
	Austin Flint* murmur of aortic regurgitation
	Carey Coombs† murmur of acute rheumatic fever
Presystolic	Mitral stenosis
	Tricuspid stenosis
	Atrial myxoma
Continuous	Patent ductus arteriosus
	Arteriovenous fistula (coronary artery, pulmonary, systemic)
	Aortopulmonary connection (e.g. congenital, Blalock‡ shunt)
	Venous hum (usually best heard over right supraclavicular fossa and abolished by ipsilateral internal jugular vein compression)
	Rupture of sinus of Valsalva into right ventricle or atrium
	'Mammary souffle' (in late pregnancy or early postpartum period)

Note: The combined murmurs of aortic stenosis and aortic regurgitation, or mitral stenosis and mitral regurgitation, may sound as if they fill the entire cardiac cycle, but are not continuous murmurs by definition.

* See footnote mm, page 87.

† Carey F Coombs (b. 1879), Bristol physician.

‡ Alfred Blalock (1899–1965), Baltimore physician.

The *pansystolic murmur* extends throughout systole, beginning with the

The *pansystolic murmur* extends throughout systole, beginning with the first heart sound, then going right up to the second heart sound. Its loudness and pitch vary during systole. Pansystolic murmurs occur when a ventricle leaks to a lower pressure chamber or vessel. As there is a pressure difference from the moment the ventricle begins to contract (S1), blood flow and the murmur both begin at the first heart sound and continue until the pressures equalise (S2). Causes of pansystolic murmurs include mitral regurgitation,[x] tricuspid regurgitation and ventricular septal defect.

With an *ejection (mid)systolic* murmur, the murmur does not begin right at the first heart sound; its intensity is greatest in midsystole or later, and wanes again late in systole. This is described as a *crescendo-decrescendo* murmur. These murmurs are usually caused by turbulent flow through the aortic or pulmonary valve orifices or by greatly increased flow through a normal-sized orifice or outflow tract.

With a *late systolic* murmur it is possible to distinguish an appreciable gap between the first heart sound and the murmur, which then continues right up to the second heart sound. This is typical of mitral valve prolapse or papillary muscle dysfunction where mitral regurgitation begins in midsystole.

Diastolic murmurs occur during ventricular diastole. They are more difficult for students to hear than systolic murmurs and are usually softer. A loud murmur is unlikely to be diastolic.

The *early diastolic* murmur begins immediately with the second heart sound and has a decrescendo quality (it is loudest at the beginning and extends for a variable distance into diastole). These early diastolic murmurs are typically high pitched and are due to regurgitation through leaking aortic or pulmonary valves. The murmur is loudest at the beginning because this is when aortic and pulmonary artery pressures are highest.

Mid-diastolic murmurs begin later in diastole and may be short or extend right up to the first heart sound. They have a much lower-pitched quality than early diastolic murmurs. They are due to impaired flow during ventricular filling and can be caused by mitral stenosis and tricuspid stenosis, where the valve is narrowed, or rarely by an atrial myxoma, where the tumour mass obstructs the valve orifice. In severe aortic regurgitation, the regurgitant jet from the aortic valve may cause the anterior leaflet of the mitral valve to shudder, producing a diastolic murmur. Occasionally, normal mitral or tricuspid valves can produce flow murmurs, which are short and mid-diastolic, and occur when there is torrential flow across the valve. Causes include a high cardiac output or intracardiac shunting (atrial or ventricular septal defects).

Presystolic murmurs may be heard when atrial systole increases blood flow across the valve just before the first heart sound. They are extensions of the mid-diastolic murmurs of mitral stenosis and tricuspid stenosis, and usually do not occur when atrial systole is lost in atrial fibrillation.

As the name implies, **continuous murmurs** extend throughout systole and diastole. They are produced when a communication exists between two parts of the circulation with a permanent pressure gradient so that blood flow occurs continuously. They can usually be distinguished from combined systolic and diastolic murmurs (due, for example, to aortic stenosis and aortic regurgitation), but this may sometimes be difficult. The causes are presented in Table 4.15.

A **pericardial friction rub** is a superficial scratching sound; there may be up to three distinct components occurring at any time during the cardiac cycle. They are not confined to systole or diastole. A rub is caused by movement of inflamed pericardial surfaces; it is a result of pericarditis. The sound can vary with respiration and posture; it is often louder when the patient is sitting up and breathing out. It tends to come and go, and is often absent by the time students can be found to come and listen for it. It has been likened to the crunching sound made when *walking on snow*.

A **mediastinal crunch** (Hamman's sign[v]) is a crunching sound heard in time with the heartbeat but with systolic and diastolic components. It is caused by the presence of air in the mediastinum, and once heard it is not forgotten. It is very often present after cardiac surgery and may occur associated with a pneumothorax or after aspiration of a pericardial effusion.

Area of greatest intensity

Although the place on the praecordium where a murmur is heard most easily is a guide to its origin, this is not a particularly reliable physical sign. For example, mitral regurgitation murmurs (*GOOD SIGNS GUIDE 4.1*) are usually loudest at the apex, over the mitral area, and tend to radiate towards the axillae, but they may be heard widely over the praecordium and even right up into the aortic area or over the back. Conduction of an ejection murmur up into the carotid arteries strongly suggests that this arises from the aortic valve.

GOOD SIGNS GUIDE 4.1 Characteristic murmurs and valvular heart disease

Sign	Positive LR	Negative LR
Characteristic systolic murmur		
Aortic stenosis	3.3	0.1
Mild mitral regurgitation or worse (moderate or severe)	5.4	0.4
Mild tricuspid regurgitation or worse	14.6	0.8
Moderate to severe tricuspid regurgitation	10.1	0.4
Characteristic diastolic murmur		
Mild aortic regurgitation or worse	9.9	0.3
Pulmonary regurgitation	17.4	NS

NS = not significant.

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

Loudness and pitch

Unfortunately, the *loudness* of the murmur is not always helpful in deciding the severity of the valve lesion. For example, in the severest forms of valve stenosis, murmurs may be soft. However, murmurs are usually graded according to loudness. Cardiologists most often use a classification with six grades (Levine's grading system):[27]

Grade 1/6: very soft and not heard at first (often audible only to consultants and to those students who have been told the murmur is present)

Grade 2/6: soft, but can be detected almost immediately by an experienced auscultator

Grade 3/6: moderate; there is no thrill

Grade 4/6: loud; thrill just palpable

Grade 5/6: very loud; thrill easily palpable

Grade 6/6: very, very loud; can be heard even without placing the stethoscope right on the chest.

This grading is useful, particularly because a change in the intensity of a murmur may be of great significance—for example, after a myocardial infarction.

It requires practice to appreciate the *pitch* of the murmur, but this may be of use in identifying its type. In general, low-pitched murmurs indicate turbulent flow under low pressure, as in mitral stenosis, and high-pitched murmurs indicate a high velocity of flow, as in mitral regurgitation.

Dynamic manoeuvres (***GOOD SIGNS GUIDE 4.2***)

All patients with a newly diagnosed murmur should undergo dynamic manoeuvre testing (Table 4.16).[28]

Respiration. Murmurs that arise on the right side of the heart become louder during inspiration as this increases venous return and therefore blood flow to the right side of the heart. Left-sided murmurs are either unchanged or become softer. Expiration has the opposite effect. This can be a sensitive and specific way of differentiating right- and left-sided murmurs.

Deep expiration. A routine part of the examination of the heart (Figure 4.33) includes leaning a patient forward in full expiration and listening to the base of the heart for aortic regurgitation, which may otherwise be missed. In

base of the heart for aortic regurgitation, which may otherwise be missed. In this case the manoeuvre brings the base of the heart closer to the chest wall. The scraping sound of a pericardial friction rub is also best heard in this position.

The Valsalva manoeuvre.[z] This is a forceful expiration against a closed glottis. One should ask the patient to hold his or her nose with the fingers, close the mouth, breathe out hard and completely so as to pop the eardrums, and hold this for as long as possible. Listen over the left sternal edge during this manoeuvre for changes in the systolic murmur of hypertrophic cardiomyopathy, and over the apex for changes when mitral valve prolapse is suspected.

GOOD SIGNS GUIDE 4.2 Dynamic auscultation

Sign	Positive LR	Negative LR
Louder on inspiration—right-sided murmur	7.8	0.2
Louder with Valsalva strain—hypertrophic cardiomyopathy	14.0	0.3
Louder squatting to standing—hypertrophic cardiomyopathy	6.0	0.1

Softer with isometric handgrip—hypertrophic cardiomyopathy	3.6	0.1
Louder with isometric handgrip—mitral regurgitation	5.8	0.3

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

TABLE 4.16 Dynamic manoeuvres and systolic cardiac murmurs

Manoeuvre	Lesion			
	Hypertrophic cardiomyopathy	Mitral valve prolapse	Aortic stenosis	Mitral regurgitation
Valsalva strain phase (decreases preload)	Louder	Longer	Softer	Softer
Squatting or leg raise (increases preload)	Softer	Shorter	Louder	Louder
Hand grip (increases afterload)	Softer	Shorter	Softer	Louder

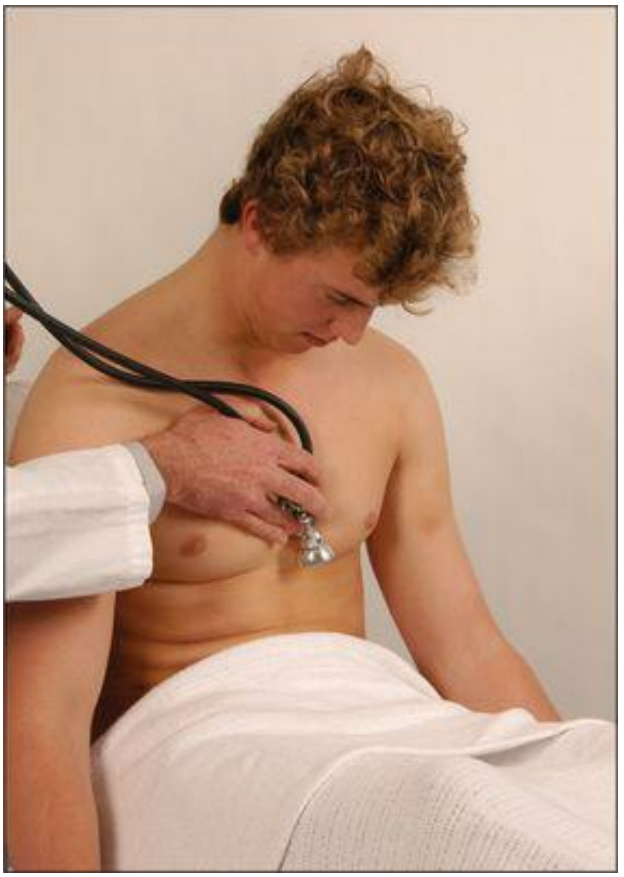


Figure 4.33 Dynamic auscultation for aortic regurgitation or a pericardial friction rub;

patient in deep expiration

The Valsalva manoeuvre has four phases. In phase 1 (beginning the manoeuvre), a rise in intrathoracic pressure and a transient increase in left ventricular output and blood pressure occurs. In phase 2 (the straining phase), systemic venous return falls, filling of the right and then the left side of the heart is reduced, and stroke volume and blood pressure fall while the heart rate increases. As stroke volume and arterial blood pressure fall, most cardiac murmurs become softer; however, because the left ventricular volume is reduced, the systolic murmur of hypertrophic cardiomyopathy becomes louder and the systolic click and murmur of mitral valve prolapse begins earlier. In phase 3 (the release of the manoeuvre), first right-sided and then left-sided cardiac murmurs become louder briefly before returning to normal. Blood pressure falls further because of pooling of blood in the pulmonary veins. In phase 4, the blood pressure overshoots as a result of increased sympathetic activity as a response to the previous hypotension. Changes in heart rate are opposite to the blood pressure changes.

The blood pressure responses can be measured by inflating a cuff to 15 mmHg over the systolic pressure before the manoeuvre. Korotkoff sounds will then appear in phases 1 and 4 in a normal patient. Absence of the phase 4 overshoot is a sign of cardiac failure. The left ventricle is unable to increase cardiac output despite increased sympathetic activity.

Standing to squatting. When the patient squats rapidly from the standing position, venous return and systemic arterial resistance increase simultaneously, causing a rise in stroke volume and arterial pressure. This makes most murmurs louder. However, left ventricular size is increased, which reduces the obstruction to outflow and therefore reduces the intensity of the systolic murmur of hypertrophic cardiomyopathy, while the midsystolic click and murmur of mitral valve prolapse are delayed.

Squatting to standing. When the patient stands up quickly after squatting, the opposite changes in the loudness of these murmurs occurs.

Isometric exercise. Sustained hand grip or repeated sit-ups for 20 for 30 seconds increases systemic arterial resistance, blood pressure and heart size. The systolic murmur of aortic stenosis may become softer because of a reduction in the pressure difference across the valve but often remains unchanged. Most other murmurs become louder, except the systolic murmur of hypertrophic cardiomyopathy, which is softer, and the mitral valve prolapse murmur, which is delayed because of an increased ventricular volume.

Auscultation of the neck

This is often performed as a part of dynamic auscultation for valvular heart disease, but certain aspects of the examination may be considered here. Abnormal sounds heard over the arteries are called *bruits*. These sounds are low-pitched and may be more easily heard with the bell of the stethoscope. Carotid artery bruits are most easily heard over the anterior part of the sternomastoid muscle above the medial end of the clavicle. Ask the patient to stop breathing for a brief period to remove the competing noise of breath sounds. It may be prudent to ask the patient not to speak. The amplified voice is often painfully loud when heard through the stethoscope.

A systolic bruit may be a conducted sound from the heart. The murmur of aortic stenosis is always audible in the neck and a soft carotid bruit is sometimes audible in patients with severe mitral regurgitation or pulmonary stenosis. A bruit due to carotid stenosis will not be audible over the base of the heart. Move the stethoscope from point to point onto the chest wall; if the bruit disappears, it is likely the sound arises from the carotid. It is not possible to exclude a carotid bruit in a patient with a murmur of aortic stenosis that radiates to the neck. Carotid artery stenosis is an important cause of a carotid bruit. More severe stenosis is associated with a noise that is longer and of increased pitch. Total obstruction of the vessel leads to disappearance of the bruit. It is not possible to make a diagnosis of significant (>60% obstruction) carotid stenosis clinically. The bottom line is that a carotid bruit poorly predicts significant carotid stenosis or stroke risk.Thyrotoxicosis can result in a systolic bruit (page 303) due to the increased vascularity of the gland.

A continuous noise is sometimes audible at the base of the neck. This is usually a venous hum, a result of audible venous flow. It disappears if light pressure is applied to the neck just above the stethoscope. Occasionally a loud machinery murmur (page 93) or severe aortic regurgitation (page 86) may cause a similar sound. Haemodialysis patients frequently have an audible bruit transmitted from their arterio-venous fistula.

The back

It is now time to leave the praecordium. Percussion and auscultation of the *lung bases* (Chapter 5) are also part of the cardiovascular examination. Signs of cardiac failure may be detected in the lungs; in particular late or pan-inspiratory crackles or a pleural effusion may be present. The murmur associated with coarctation of the aorta may be prominent over the upper back.

back.

While the patient is sitting up, feel for pitting oedema of the sacrum, which occurs in severe right heart failure, especially in patients who have been in bed. This is because the sacrum then becomes a dependent area and oedema fluid tends to settle under the influence of gravity.

The abdomen

Lay the patient down flat (on one pillow) and examine the abdomen (Chapter 6). You are looking particularly for an enlarged tender *liver* which may be found when the hepatic veins are congested in the presence of right heart failure. Distension of the liver capsule is said to be the cause of liver tenderness in these patients. When tricuspid regurgitation is present the liver may be *pulsatile*, as the right ventricular systolic pressure wave is transmitted to the hepatic veins. Test for hepatojugular reflux.[17,29] *Ascites* may occur with severe right heart failure. *Splenomegaly*, if present, may indicate infective endocarditis.

Feel for the pulsation of the abdominal aorta, to the left of the middle line. It is often palpable in normal thin people but the possibility of an abdominal aortic aneurysm should always be considered when the aorta's pulsations are palpable and expansile (page 173).[30,31]

The lower limbs (Table 4.17)

Palpate behind the medial malleolus of the tibia and the distal shaft of the tibia for *oedema* by compressing the area for at least 15 seconds with the thumb. This latter area is often tender in normal people, and gentleness is necessary. Oedema may be pitting (the skin is indented and only slowly refills —Figure 4.34) or non-pitting. Oedema due to hypoalbuminaemia often refills more quickly.

TABLE 4.17 Lower limb examination

1 Inspection—anterior and lateral surfaces, sole of foot, between

toes

• Amputation

• Ulcers

• Erythema

• Varicosities

• Atrophy

• Scars

• Discoloration (e.g. venous staining)

• Loss of hair

2 Palpation

• Temperature: run the dorsum of the hand from the hips to the foot on each side. Note reduction in temperature peripherally and compare left and right

• Test capillary refill: press on great toenail and release. The blanched nail bed should turn pink within 3 seconds

• Test venous filling: occlude the dorsal venous arch of each foot in turn using two fingers; release the distal finger and look for venous refilling. Absence of venous refilling suggests poor arterial supply to the foot

• Pulses: feel for an abdominal aortic aneurysm, feel for a femoral pulse, the popliteal pulses (flex the patient's leg), then feel the posterior tibial and dorsalis pedis pulses

3 Auscultation

• Listen for abdominal, renal, and femoral bruits

4 Perform Buerger's test (see text)
5 Measure the ankle–brachial index
6 Test lower limb sensation. Diabetes may cause sensory loss in a 'stocking' distribution.
7 Test for glucose in the urine

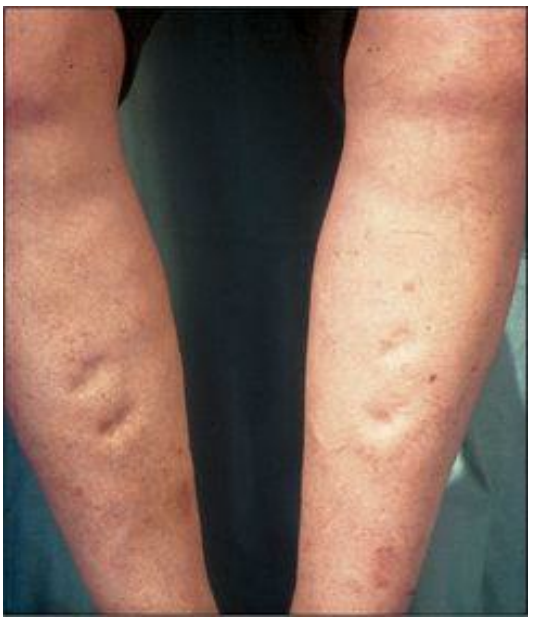


Figure 4.34 Severe pitting oedema of the legs

Pitting oedema occurs in cardiac failure unless the condition has been present for a long time and secondary changes in the lymphatic vessels have occurred. If oedema is present, note its upper level (e.g. 'pitting oedema to mid-calf' or 'pitting oedema to mid-thigh'). Severe oedema can involve the skin of the abdominal wall and the scrotum as well as the lower limbs. Causes of oedema are listed in Table 4.18.

TABLE 4.18 Causes of oedema

Pitting lower limb oedema
Cardiac: congestive cardiac failure, constrictive pericarditis
Drugs: calcium antagonists
Hepatic: cirrhosis causing hypoalbuminaemia
Renal: nephrotic syndrome causing hypoalbuminaemia
Gastrointestinal tract: malabsorption, starvation, protein-losing enteropathy causing hypoalbuminaemia
Beri-beri (wet)
Cyclical oedema
Pitting unilateral lower limb oedema
Deep venous thrombosis
Compression of large veins by tumour or lymph nodes
Non-pitting lower limb oedema
Hypothyroidism
Lymphoedema • Infectious (e.g. filariasis) • Malignant (tumour invasion of lymphatics) • Congenital (lymphatic development arrest) • Allergy • Milroy's* disease (unexplained lymphoedema which appears at puberty and is more common in females)

* William Milroy (1855–1914), Professor of Medicine, University of Nebraska, described the disease in 1928.

Non-pitting oedema suggests chronic lymphoedema which is due to lymphatic obstruction. 'Lipidoedema' is a term used to describe fat

lymphatic obstruction. Lipidoedema is a term used to describe fat deposition in the ankles. It typically spares the feet and affects obese women.

Look for evidence of Achilles[aa] tendon xanthomata due to hyperlipidaemia. Also look for cyanosis and clubbing of the toes (this may occur without finger clubbing in a patient with a patent ductus arteriosus, because a rise in pulmonary artery pressures, sufficient to reverse the direction of flow in the shunt, has occurred).

Peripheral vascular disease

Examine both *femoral arteries* by palpating and then auscultating them. A bruit may be heard if the artery is narrowed. Next palpate the following pulses: *popliteal* (behind the knee—Figure 4.35a: if this is difficult to feel when the patient is supine, try the method shown in Figure 4.35b), *posterior tibial* (under the medial malleolus, Figure 4.36a) and *dorsalis pedis* (on the forefoot, Figure 4.36b) on both sides.[32]

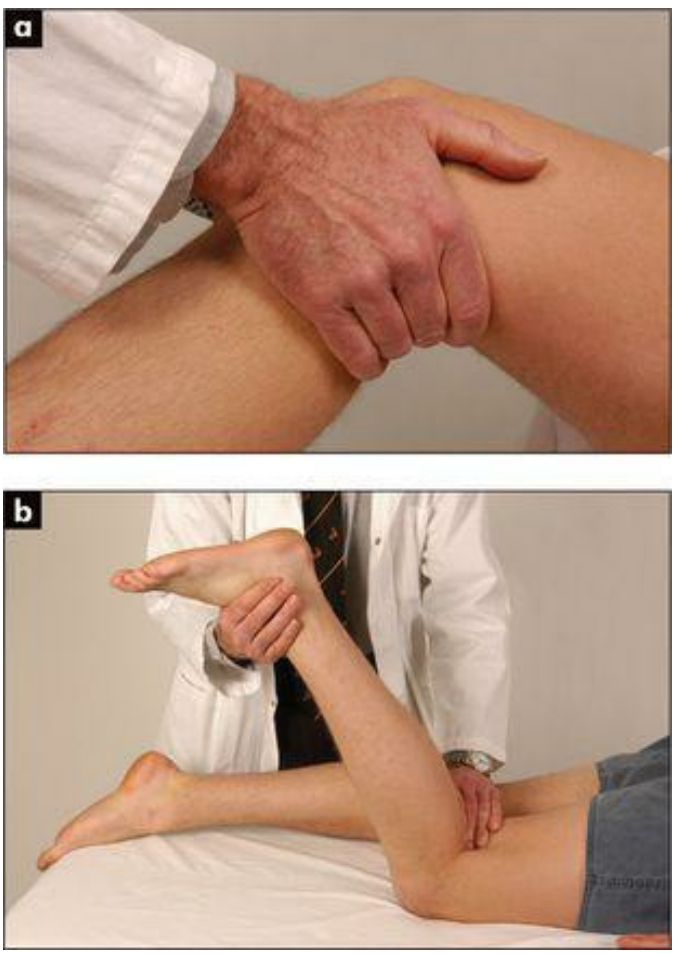


Figure 4.35 Palpating the popliteal artery: (a) patient supine; (b) patient prone

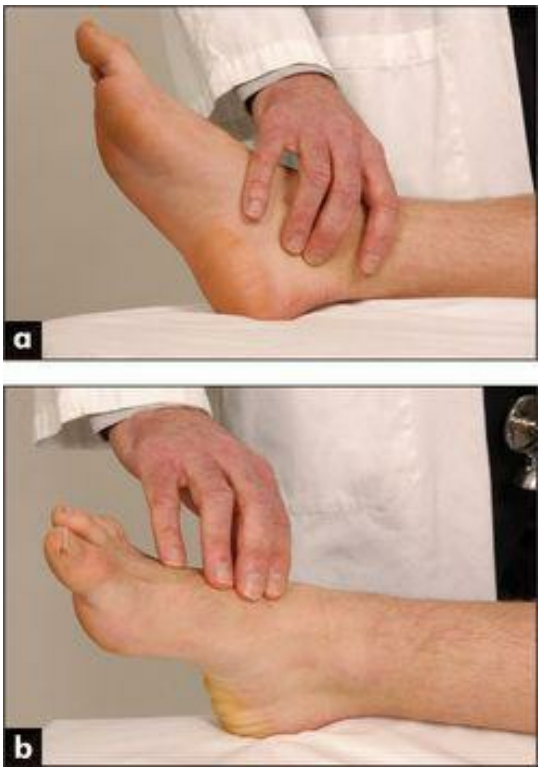


Figure 4.36 Feeling (a) the posterior tibial artery and (b) the dorsalis pedis artery

Patients with exertional calf pain (intermittent claudication) are likely to have disease of the peripheral arteries. More severe disease can lead to pain even at rest and to ischaemic changes in the legs and feet (*GOOD SIGNS GUIDE 4.3*). Look for atrophic skin and loss of hair, colour changes of the feet (blue or red) and ulcers at the lower end of the tibia.[4] Venous and diabetic ulcers can be distinguished from arterial ulcers (Figures 4.37-4.39).

GOOD SIGNS GUIDE 4.3 Peripheral vascular disease

Sign	Positive LR	Negative LR
Sores or ulcers on feet	7.0	NS
Feet pale, red or blue	2.8	0.7
Atrophic skin	1.7	NS
Absent hair	1.7	NS
One foot cooler	6.1	0.9
Absent femoral pulse	6.1	NS
Absent dorsalis pedis and posterior tibial pulses	14.9	0.3
Limb bruit present	7.3	0.7
Capillary refill time >5 seconds	1.9	NS
Capillary refill >20 seconds	3.6	NS

NS = not significant.

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

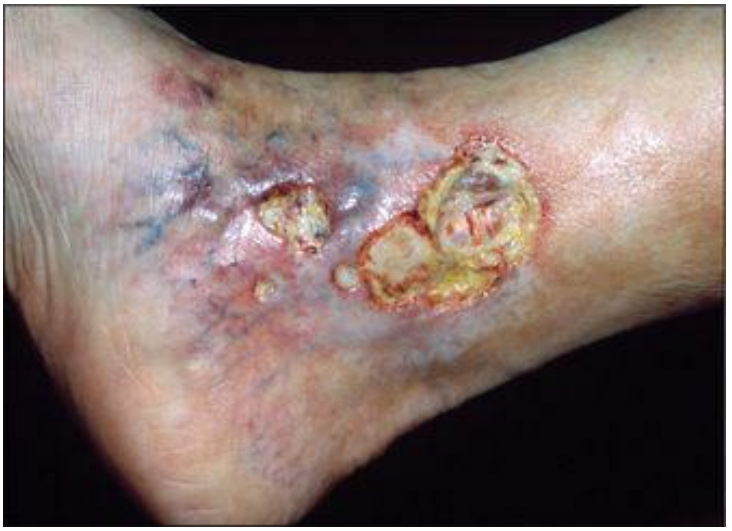


Figure 4.37 Venous ulcer
This venous ulcer has an irregular margin, pale surrounding neo-epithelium (new skin), and a pink base of granulation tissue. There is often a history of deep venous thrombosis. The skin is warm and oedema is often present. (See Table 4.19.)

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

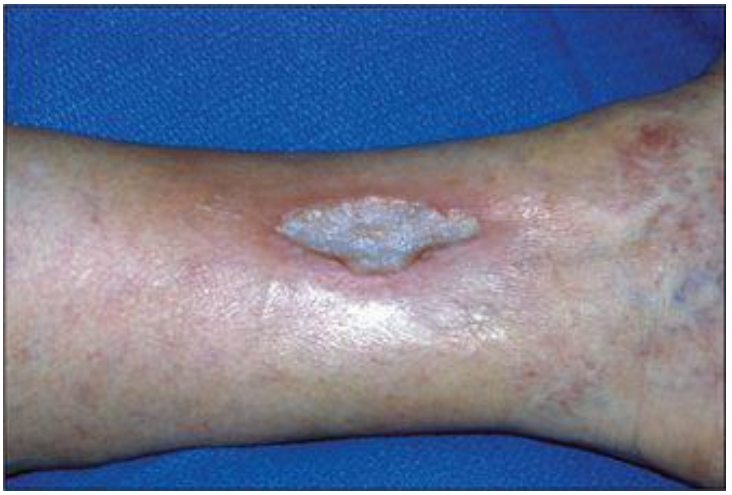


Figure 4.38 Arterial ulcer
This arterial ulcer has a regular margin and 'punched out' appearance. The surrounding skin is cold. The peripheral pulses are absent. (See Table 4.19.)

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

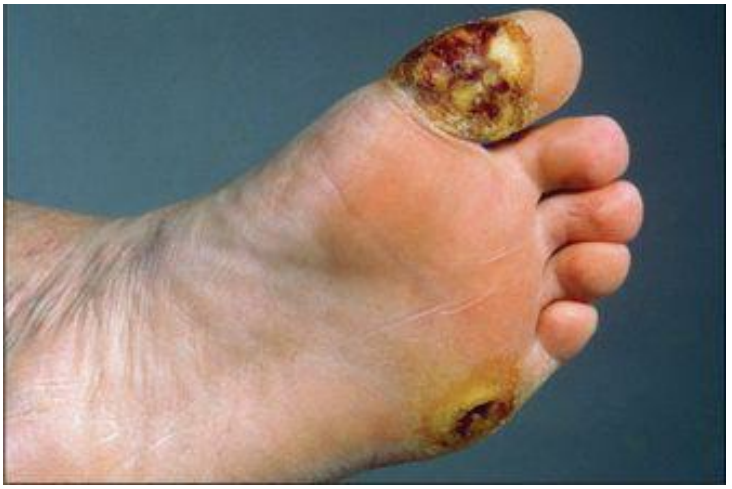


Figure 4.39 Diabetic (neuropathic) ulcer
Neuropathic ulcers are painless and are associated with reduced sensation in the surrounding skin. (See Table 4.19.)

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

Look for reduced capillary return (compress the toenails—the return of the normal red colour is slow).[33] In such cases, perform *Buerger's test*[bb] to help confirm your diagnosis: elevate the legs to 45 degrees (pallor is rapid if there is a poor arterial supply), then place them dependent at 90 degrees over the edge of the bed (cyanosis occurs if the arterial supply is impaired). Normally there is no change in colour in either position.

The **ankle–brachial index** (ABI) is a measure of arterial supply to the lower limbs, and an abnormal index indicates increased cardiovascular risk. The systolic blood pressure in the dorsalis pedis or posterior tibial artery is measured using a Doppler probe and a blood pressure cuff over the calf. This is divided by the systolic blood pressure measured in the normal way at the brachial artery. An ABI of less than 0.9 indicates significant arterial disease and an ABI of between 0.4 and 0.9 is associated with claudication. ABIs of less than 0.4 are associated with critical limb ischaemia. An ABI greater than 1.3 occurs with a calcified (non-compressible) artery.

Acute arterial occlusion

Acute arterial occlusion of a major peripheral limb artery results in a painful, pulseless, pale, 'paralysed' limb which is perishingly cold and has paraethesiae (the six P's).

It can be the result of embolism, thrombosis or injury. Peripheral arterial embolism usually arises from thrombus in the heart, where it is often secondary to (i) myocardial infarction, (ii) dilated cardiomyopathy, (iii) atrial

fibrillation or (iv) infective endocarditis.[34]

Deep venous thrombosis

Deep venous thrombosis is a difficult clinical diagnosis (*GOOD SIGNS GUIDE 4.4*).[35] The patient may complain of calf pain. On examination, the clinician should look for swelling of the calf and the thigh, and dilated superficial veins. Feel then for increased warmth and squeeze the calf (gently) to determine if the area is tender. Homans' sign[cc] (pain in the calf when the foot is sharply dorsiflexed) is of limited diagnostic value and is theoretically dangerous because of the possibility of dislodgment of loose thrombus.

GOOD SIGNS GUIDE 4.4 Deep venous thrombosis

Sign	Positive LR	Negative LR
Asymmetrical calf swelling >2 cm difference	2.1	0.6

Thigh swelling	2.5	0.6
Superficial venous dilatation	1.9	NS
Tenderness and erythema	NS	NS
Asymmetrical skin warmth	1.4	NS
Homans' sign	NS	NS

NS = not significant.

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

The causes of thrombosis were described by Virchow[dd] in 1856 under three broad headings (the famous *Virchow's triad*): (i) changes in the vessel wall, (ii) changes in blood flow, and (iii) changes in the constitution of the blood. Deep venous thrombosis is usually caused by prolonged immobilisation, cardiac failure (stasis) or trauma (vessel wall damage), but may also result from occult neoplasm, disseminated intravascular coagulation, the contraceptive pill, pregnancy and a number of inherited defects of coagulation (the *thrombophilias*: e.g. Factor V Leiden, anti-thrombin III deficiency).

Varicose veins

If a patient complains of 'varicose veins', ask him or her to *stand* with the legs fully exposed.[36] *Inspect* the front of the whole leg for tortuous, dilated branches of the long saphenous vein (below the femoral vein in the groin to the medial side of the lower leg). Then inspect the back of the calf for varicosities of the short saphenous vein (from the popliteal fossa to the back of the calf and lateral malleolus). Look to see if the leg is inflamed, swollen or pigmented (subcutaneous haemosiderin deposition secondary to venous stasis).

Palpate the veins. Hard leg veins suggest thrombosis, while tenderness indicates thrombophlebitis. Perform the *cough impulse test*. Put the fingers over the long saphenous vein opening in the groin, medial to the femoral vein. (Don't forget the anatomy—femoral vein [medial], artery [your landmark], nerve [lateral].) Ask the patient to cough: a fluid thrill is felt if the saphenofemoral valve is incompetent.

The following supplementary tests are occasionally helpful (and surgeons like to quiz students on them in examinations).

Trendelenburg test:[ee] with the patient lying down, the leg is elevated.

Firm pressure is placed on the saphenous opening in the groin, and the patient is instructed to stand. The sign is positive if the veins stay empty until the groin pressure is released (incompetence at the saphenofemoral valve). If the veins fill despite groin pressure, the incompetent valves are in the thigh or calf, and Perthes' test[ff] is performed.

Perthes' test: repeat the Trendelenburg test, but when the patient stands, allow some blood to be released and then get him or her to stand up and down on the toes a few times. The veins will become less tense if the perforating calf veins are patent and have competent valves (the muscle pump is functioning).

If the pattern of affected veins is unusual (e.g. pubic varices), the clinician should try to exclude secondary varicose veins. These may be due to an intrapelvic neoplasm which has obstructed deep venous return. Rectal and pelvic examinations should then be performed.

Finally, chronic venous stasis is one cause of ulceration of the lower leg. This is often associated with pigmentation and eczema, which are due to venous stasis.

The differential diagnosis of leg ulcers is summarised in Table 4.19.

TABLE 4.19 Causes of leg ulcers

1 Venous stasis ulcer—most common (Figure 4.37)
Site: around malleoli
Character: irregular margin, granulation tissue in the floor. Surrounding tissue inflammation and oedema
Associated pigmentation, stasis eczema
2 Ischaemic ulcer (Figure 4.38) • Large-artery disease (atherosclerosis, thromboangiitis obliterans): usually lateral side of leg (pulses absent)

obliterans): usually lateral side of leg (pulses absent) • Small-vessel disease (e.g. leucocytoclastic vasculitis, palpable purpura)
Site: over pressure areas, lateral malleolus, dorsum and margins of the feet and toes
Character: smooth, rounded, ‘punched out’ pale base which does not bleed
3 Malignant ulcer, e.g. basal cell carcinoma (pearly translucent edge), squamous cell carcinoma (hard everted edge), melanoma, lymphoma, Kaposi’s sarcoma
4 Infection, e.g. *Staphylococcus aureus*, syphilitic gumma, tuberculosis, atypical *Mycobacterium*, fungal
5 Neuropathic (painless penetrating ulcer on sole of foot: peripheral neuropathy, e.g. diabetes mellitus, tabes, leprosy) (Figure 4.39)
6 Underlying systemic disease • Diabetes mellitus: vascular disease, neuropathy or necrobiosis lipoidica (front of leg) • Pyoderma gangrenosum • Rheumatoid arthritis • Lymphoma • Haemolytic anaemia (small ulcers over malleoli), e.g. sickle cell anaemia

Correlation of physical signs and cardiovascular disease

> When a disease is named after some author, it is very likely that we don’t know much about it.

August Bier (1861–1949)

Cardiac failure

This is one of the commonest syndromes: the signs of cardiac failure should be sought in all patients admitted to hospital, especially if there is a complaint of dyspnoea (see *Questions box 5.2*, page 111).[37] Cardiac failure has been defined as a reduction in cardiac function such that cardiac output is reduced relative to the metabolic demands of the body and compensating mechanisms have occurred. The specific signs depend on whether the left, right or both ventricles are involved. It is important to note that the absence of definite signs of cardiac failure may not exclude the diagnosis. Patients with compensated, chronic cardiac failure may be normal on cardiac examination.

Left ventricular failure (LVF)

• **Symptoms:** exertional dyspnoea, orthopnoea, paroxysmal nocturnal dyspnoea.

• **General signs:** tachypnoea, due to raised pulmonary pressures; central cyanosis, due to pulmonary oedema; Cheyne-Stokes breathing (see Table 5.10, page 112), especially in sedated elderly patients; peripheral cyanosis, due to low cardiac output; hypotension, due to low cardiac output; cardiac cachexia.

• **Arterial pulse:** sinus tachycardia, due to increased sympathetic tone; low pulse pressure (low cardiac output); pulsus alternans (alternate strong and weak beats; it is unlike a bigeminal rhythm caused by regular ectopic beats, in that the beats are regular; see Figure 4.40)—this is an uncommon but specific sign of unknown aetiology.

• **Apex beat:** displaced, with dilatation of the left ventricle; dyskinetic in anterior myocardial infarction or dilated cardiomyopathy; palpable gallop rhythm. The absence of these signs does not exclude left ventricular failure.

• **Auscultation:** left ventricular S3 (an important sign); functional mitral regurgitation (secondary to valve ring dilatation).

• **Lung fields:** signs of pulmonary congestion (basal inspiratory crackles) or pulmonary oedema (crackles and wheezes throughout the lung fields), due

to raised venous pressures (increased preload). The typical middle to late inspiratory crackles at the lung bases may be absent in chronic, compensated heart failure, and there are many other causes of basal inspiratory crackles. This makes crackles a rather non-specific and insensitive sign of heart failure.

- **Other signs:** abnormal Valsalva response, positive abdominojugular reflux test, right ventricular failure may complicate left ventricular failure, especially if this is severe and chronic.

- **Signs of the underlying or precipitating cause:**

  Causes of LVF: (i) myocardial disease (ischaemic heart disease, cardiomyopathy); (ii) volume overload (aortic regurgitation, mitral regurgitation, patent ductus arteriosus); (iii) pressure overload (systolic hypertension, aortic stenosis).

  Signs of a precipitating cause: anaemia, thyrotoxicosis (page 301), rapid arrhythmia (usually atrial fibrillation). (See *GOOD SIGNS GUIDE 4.5*.)

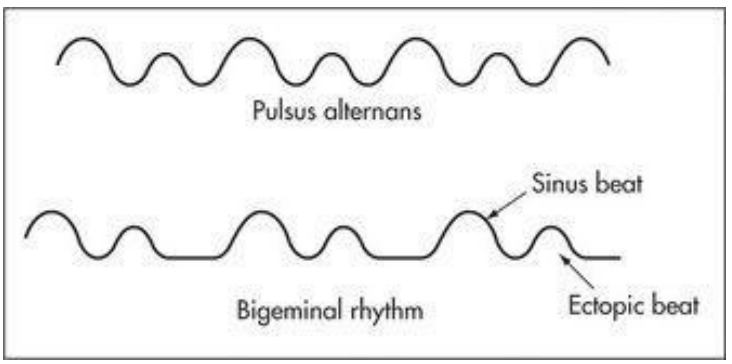


Figure 4.40 Pulsus alternans

GOOD SIGNS GUIDE 4.5 Left ventricular failure

General signs	**Positive LR**	**Negative LR**
Heart rate >100 beats per minute at rest	5.5	NS
Valsalva manoeuvre abnormal	7.6	0.1
Lungs		
Crackles	NS	NS
Cardiac examination		
JVP elevated	3.9	NS
Abdominojugular test positive	8.0	0.3
Apex displaced lateral to midclavicular line	5.8	NS
S3	5.7	NS

S3	[illegible]	NS
S4	NS	NS
Other findings		
Oedema	NS	NS

NS = not significant.

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

Right ventricular failure (RVF)

• **Symptoms:** ankle, sacral or abdominal swelling, anorexia, nausea.

• **General signs:** peripheral cyanosis, due to low cardiac output.

• **Arterial pulse:** low volume, due to low cardiac output.

• **Jugular venous pulse:** raised, due to the raised venous pressure (right heart preload); Kussmaul's sign, due to poor right ventricular compliance (e.g. right ventricular myocardial infarction); large *v* waves (functional tricuspid regurgitation secondary to valve ring dilatation).

• **Apex beat:** right ventricular heave.

• **Auscultation:** right ventricular S3; pansystolic murmur of functional tricuspid regurgitation (absence of a murmur does not exclude tricuspid regurgitation).

• **Abdomen:** *tender hepatomegaly*, due to increased venous pressure transmitted via the hepatic veins; *pulsatile liver* (a useful sign), if tricuspid regurgitation is present.

• **Oedema:** due to sodium and water retention plus raised venous pressure, may be manifested by pitting ankle and sacral oedema, ascites, or pleural effusions (small).

• **Signs of the underlying cause:**

Causes of RVF: (i) chronic obstructive pulmonary disease (commonest cause of cor pulmonale); (ii) left ventricular failure (severe chronic LVF causes raised pulmonary pressures resulting in secondary

right ventricular failure); (iii) volume overload (atrial septal defect, primary tricuspid regurgitation); (iv) other causes of pressure overload (pulmonary stenosis, idiopathic pulmonary hypertension); (v) myocardial disease (right ventricular myocardial infarction, cardiomyopathy).

Chest pain

Many of the causes of chest pain represent a medical (or surgical) emergency. The appropriate diagnosis or differential diagnosis is often suggested by the history, and urgent investigations (e.g. ECG, chest X-ray, lung scan or pulmonary angiogram) may be indicated. However, a careful and rapid physical examination may add important information in many cases. In all cases the general inspection and measurement of the vital signs will help with the assessment of the severity and urgency of the problem. Certain specific signs may help with the diagnosis.[38,39]

Myocardial infarction

• **General signs:** there are few specific signs of myocardial infarction but many patients appear obviously unwell and in distress from their chest pain. Sweating (often called diaphoresis by accident and emergency staff), an appearance of anxiety (angor animi or sense of impending doom), and restlessness may be obvious. It is important that all this information be recorded so that changes to the patient's condition can be assessed as the infarct evolves.

• **Pulse and blood pressure:** tachycardia and/or hypotension (25% with anterior infarction from sympathetic hyperactivity); bradycardia and/or hypotension (up to 50% with inferior infarction from parasympathetic hyperactivity). Other arrhythmias including atrial fibrillation (due to atrial infarction), ventricular tachycardia and heart block may be present.

• **The JVP:** increased with right ventricular infarction; Kussmaul's sign is a specific and sensitive sign of right ventricular infarction in patients with a recent inferior infarct.

• **Apex beat:** dyskinetic in patients with large anterior infarction.

• **Auscultation:** S4; S3; decreased intensity of heart sounds; transient apical midsystolic or late-systolic murmur (in 25% from mitral regurgitation

secondary to papillary muscle dysfunction), or a pericardial friction rub (with transmural infarction).

• **Complications:** arrhythmias (ventricular tachycardia, atrial fibrillation, ventricular fibrillation or heart block); heart failure; cardiogenic shock; rupture of a papillary muscle; perforation of the ventricular septum; ventricular aneurysm; thromboembolism or cardiac rupture. Signs of these complications (which do not usually occur for a few days after the infarct) include the development of a new murmur, recurrent chest pain, dyspnoea, sudden hypotension or sudden death.

The **Killip Class**[gg] can be calculated from the examination. It gives considerable prognostic information.[38]

Killip Class I—no evidence of heart failure

Killip Class II—mild heart failure; crackles over lower third or less of the lungs; systolic BP > 90 mmHg

Killip Class III—pulmonary oedema, crackles more than one-third of chest; systolic BP > 90 mmHg

Killip Class IV—cardiogenic shock, pulmonary oedema, crackles more than one-third of chest, systolic BP < 90 mmHg.

Killip Class III or IV is associated with a >5-fold mortality risk and Class II with a >3-fold risk compared with Class I.

Pulmonary embolism

There may be no physical signs of this condition, but dyspnoea which may be profound and make the patient exhausted is often the most obvious indication of a large pulmonary embolism. There is usually a resting tachycardia. Signs of shock—hypotension and cyanosis—indicate a very large and life-threatening embolus. There may be signs of a DVT in the legs but absence of these by no means excludes the diagnosis.

Acute aortic dissection

This is a difficult diagnosis which cannot usually be excluded on clinical grounds. A tear in the intima leads to blood surging into the aortic media, separating the intima and adventitia; this may present acutely or chronically. There are three different types: *type I* begins in the ascending aorta and extends proximally and distally; *type II* is limited to the ascending aorta and

extends proximally and distally; *type II* is limited to the ascending aorta and aortic arch (this is particularly associated with Marfan's syndrome); *type III* begins distal to the left subclavian artery and has the best prognosis.

• **Symptoms:** chest pain (typically very severe, it radiates to the back and is maximal in intensity at the time of onset due to either the aortic tear or associated myocardial infarction); stroke; syncope (associated with tamponade); symptoms of left ventricular failure; and, rarely, limb pain (ischaemia), paraplegia (spinal cord ischaemia), or abdominal pain (mesenteric ischaemia).

• **Signs:** the examination can reveal signs that make the diagnosis very likely (specific but not sensitive). There may be signs of a body habitus associated with dissection (e.g. Marfan's, Ehlers-Danlos syndromes). The pulses and blood pressures in the two arms must be assessed. A diminution of the radial pulse on one side or difference in blood pressure of 20 mmHg or more between the two arms is significant and suggests dissection has progressed to involve the origin of the arm vessels. Examine the patient for signs of pericardial tamponade (page 79), which occurs if the aorta ruptures into the pericardial sac. Examine the heart for signs of aortic regurgitation caused by disruption of the aortic valve annulus. A neurological examination may reveal signs of hemiplegia, due to dissection of one of the carotid arteries. Rare signs that have been described include a pulsatile sternoclavicular joint, hoarseness (recurrent laryngeal nerve compression) and dysphagia (oesophageal compression).

Pericardial disease

Acute pericarditis

• **Signs:** fever; dyspnoea; pericardial friction rub—sit the patient up and listen to the heart with the patient holding the breath in deep expiration.

• **Causes of acute pericarditis:** (i) viral infection (coxsackievirus A or B, influenza); (ii) after myocardial infarction—early, or late (10–14 days, termed Dressler's syndrome[hh]); (iii) after pericardiotomy (cardiac surgery); (iv) uraemia; (v) neoplasia—tumour invasion (e.g. bronchus, breast, lymphoma) or after irradiation for tumour; (vi) connective tissue disease (e.g. systemic lupus erythematosus, rheumatoid arthritis); (vii) hypothyroidism; (viii) other infections (e.g. tuberculosis, pyogenic pneumonia or septicaemia); (ix) acute

rheumatic fever.

Chronic constrictive pericarditis

- **General signs:** cachexia.

- **Pulse and blood pressure:** pulsus paradoxus (more than the normal 10 mmHg fall in the arterial pulse pressure on inspiration, because increased right ventricular filling compresses the left ventricle); low blood pressure.

- **The JVP:** raised; Kussmaul's sign—lack of a fall or even increased distension on inspiration (50%); prominent *x* and *y* descents (brisk collapse during diastole).

- **Apex beat:** impalpable.

- **Auscultation:** heart sounds distant, early S3; early pericardial knock (rapid ventricular filling abruptly halted).

- **Abdomen:** hepatomegaly, due to raised venous pressure; splenomegaly, due to raised venous pressure; ascites.

- **Peripheral oedema**.

- **Causes of chronic constrictive pericarditis:** (i) cardiac operation or trauma; (ii) tuberculosis, histoplasmosis or pyogenic infection; (iii) neoplastic disease; (iv) mediastinal irradiation; (v) connective tissue disease (especially rheumatoid arthritis); (vi) chronic renal failure.

Acute cardiac tamponade

- **General signs:** tachypnoea; anxiety and restlessness; syncope. Patients look very ill.

- **Pulse and blood pressure:** rapid pulse rate; pulsus paradoxus; hypotension.

- **The JVP:** raised; prominent x but an absent *y* descent.

- **Apex beat:** impalpable.

• **Auscultation:** soft heart sounds.

• **Lungs:** dullness and bronchial breathing at the left base, due to lung compression by the distended pericardial sac.

Infective endocarditis

• **General signs:** fever; weight loss; pallor (anaemia).

• **Hands:** splinter haemorrhages; clubbing (within six weeks of onset); Osler's nodes (rare); Janeway lesions (very rare).

• **Arms:** evidence of intravenous drug use (Figure 4.41)—right (and left) heart endocarditis can result from this.

• **Eyes:** pale conjunctivae (anaemia); retinal or conjunctival haemorrhages —Roth's spots[ii] are fundal vasculitic lesions with a yellow centre surrounded by a red ring (Figure 4.42).

• **Heart**—signs of *underlying heart disease*: (i) acquired (mitral regurgitation, mitral stenosis, aortic stenosis, aortic regurgitation); (ii) congenital (patent ductus arteriosus, ventricular septal defect, coarctation of the aorta); (iii) prosthetic valves.

• **Abdomen:** splenomegaly.

• **Peripheral evidence of embolisation** to limbs or central nervous system.

• **Urinalysis:** haematuria (a fresh urine specimen will then show dysmorphic red cells and red cell casts on microscopy).

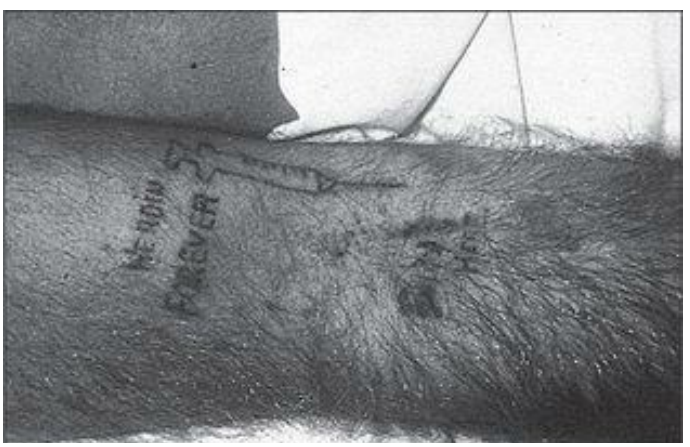


Figure 4.41 Example of an intravenous drug addict's forearm

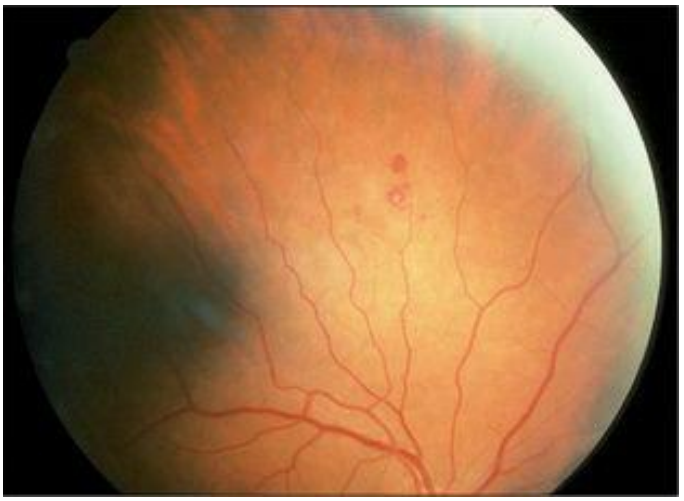


Figure 4.42 Roth's spot on fundoscopy

Systemic hypertension

It is important to have in mind a method for the examination of a patient with systemic hypertension. The examination aims to measure the blood pressure level, determine if there is an underlying cause present, and assess the severity as determined by signs of end-organ damage. It is a common clinical problem.

On **general inspection** the signs of the rare causes of secondary hypertension must be sought: for example, Cushing's syndrome,[ii] acromegaly, polycythaemia (page 236) or chronic renal failure.

Take the **blood pressure**, with the patient lying and standing, using an appropriately sized cuff. A rise in diastolic pressure on standing occurs typically in essential hypertension; a fall on standing may suggest a secondary cause, but is usually an effect of anti-hypertensive medications. Palpate for radiofemoral delay, and check the blood pressure in the legs if coarctation of the aorta is suspected or if severe hypertension is discovered before 30 years of age.

Next examine the **fundi** for Keith-Wagener retinal changes of hypertension (Figures 4.43 and 4.44) which can be classified from grades 1 to 4:

Grade 1—'silver wiring' of the arteries only (sclerosis of the vessel wall

reduces its transparency so that the central light streak becomes broader and shinier)

Grade 2—grade 1 plus arteriovenous nipping or nicking (indentation or deflection of the veins where they are crossed by the arteries)

Grade 3—grade 2 plus haemorrhages (flame-shaped) and exudates (soft—cottonwool spots due to ischaemia, or hard—lipid residues from leaking vessels)

Grade 4—grade 3 plus papilloedema.

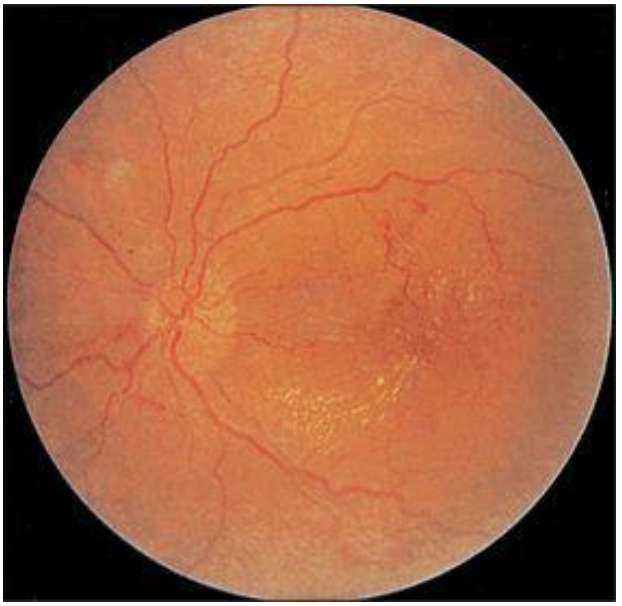


Figure 4.43 Hypertensive retinopathy grade 3
Note flame-shaped haemorrhages and cottonwool spots.

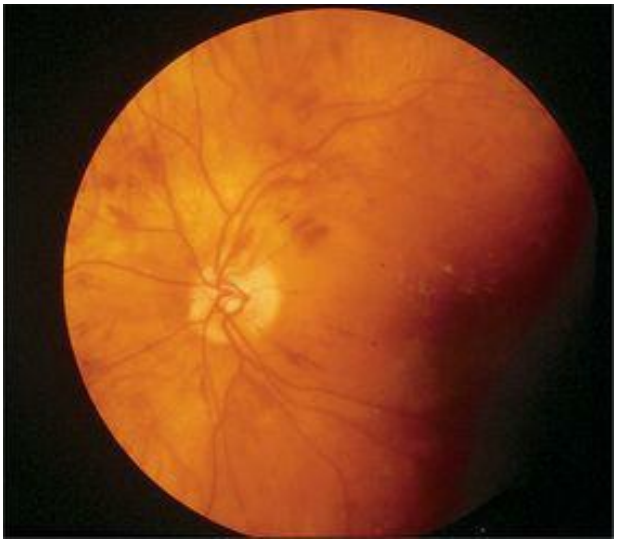


Figure 4.44 Hypertensive retinopathy grade 4
Note AV nipping, silver wiring and papilloedema.

It is important to describe the changes present rather than just give a grade.

Now examine the **rest of the cardiovascular system** for signs of left ventricular failure secondary to hypertension, and for coarctation of the aorta. A fourth heart sound is frequently detectable if the blood pressure is greater than 180/110 mmHg.

Then go to the **abdomen** to palpate for renal or adrenal masses (possible causes), and for the presence of an abdominal aortic aneurysm (a possible complication). Auscultate for a renal bruit (page 211) due to renal artery stenosis.[40] Remember that most left-sided abdominal bruits arise from the splenic artery and are of no significance. A bruit is less likely to be significant if it is short, soft and midsystolic. A loud systolic–diastolic bruit that is prominent in the epigastrium is more likely to be associated with renal artery stenosis.

Examine the **central nervous system** for signs of previous cerebrovascular accidents, and palpate and auscultate the **carotid arteries** for bruits (stenosis may be a manifestation of vascular disease, and may be associated with renal artery stenosis). **Urinalysis** should also be performed to look for evidence of renal disease (page 213).

Causes of systemic hypertension

Hypertension may be essential or idiopathic (more than 95% of cases), or secondary (less than 5%). Immoderate alcohol and salt consumption and obesity (see *Questions box 4.5*) are associated with hypertension. Obstructive sleep apnoea is also an association.

Secondary causes include: (i) renal disease—renal artery stenosis, chronic pyelonephritis, analgesic nephropathy, connective tissue disease, glomerulonephritis, polycystic disease, diabetic nephropathy, reflux nephropathy; (ii) endocrine disorders—Cushing's syndrome, Conn's syndrome (primary aldosteronism), phaeochromocytoma, acromegaly, thyrotoxicosis, hypothyroidism, hyperparathyroidism; (iii) coarctation of the aorta; and (iv) other, such as the contraceptive pill, polycythaemia rubra vera, toxaemia of pregnancy, neurogenic causes (increased intracranial pressure, lead poisoning, acute porphyria), or hypercalcaemia.

Complications of hypertension