These include left ventricular failure, cerebrovascular ischaemic events (strokes), renal failure, and eye disease (blindness). Hypertension is also a risk factor for ischaemic heart disease and peripheral vascular disease including abdominal aortic aneurysm and arterial dissections.

### Malignant (accelerated) hypertension

This can be defined as the presence of flame-shaped haemorrhages, cottonwool spots and/or papilloedema (≥grade 3 Keith-Wagener retinal changes) as a result of severe hypertension. These patients need admission to hospital for urgent treatment.

### Pulmonary hypertension

Systolic pulmonary artery pressures higher than 30 mmHg are abnormal and constitute pulmonary hypertension. Symptoms of pulmonary hypertension do not usually occur until the pressures are about twice normal (i.e. >50 mmHg). Exertional dyspnoea and fatigue are then common, and chest pain probably due to right ventricular ischaemia occurs in up to 50% of patients. It is important to know what signs to look for in a patient who may have pulmonary hypertension.

- **General signs** (usually only in patients with severe hypertension): tachypnoea; peripheral cyanosis and cold extremities, due to low cardiac output; hoarseness (very rare, due to pulmonary artery compression of the left recurrent laryngeal nerve).

- **The pulse:** usually of small volume, due to the low cardiac output (only in severe disease).

- **The JVP:** prominent *a* wave, due to forceful right atrial contraction.

- **Apex beat/praecordium:** right ventricular heave; palpable P2.

- **Auscultation:** systolic ejection click, due to dilatation of the pulmonary artery; loud P2, due to forceful valve closure because of high pulmonary artery pressures; S4; pulmonary ejection murmur, due to dilatation of the pulmonary artery resulting in turbulent blood flow; murmur of pulmonary regurgitation if dilatation of the pulmonary artery occurs.

- **Signs of right ventricular failure** (late: termed *cor pulmonale*).

### Causes of pulmonary hypertension

Pulmonary hypertension may be primary (idiopathic) or secondary.

Secondary causes include: (i) pulmonary emboli—e.g. blood clots, tumour particles, fat globules; (ii) lung disease—chronic obstructive pulmonary disease (page 133), obstructive sleep apnoea, interstitial lung disease (e.g. pulmonary fibrosis); (iii) left ventricular failure resulting in back-pressure into the pulmonary circulation; (iv) congenital heart disease causing a left-to-right shunt—atrial septal defect, ventricular septal defect, patent ductus arteriosus; and (v) severe kyphoscoliosis.

### Innocent murmurs

The detection of a systolic murmur on routine examination is a common problem. It can cause considerable alarm to both the patient and the examining clinician. These murmurs in asymptomatic people are often the result of normal turbulence within the heart and great vessels. When no structural abnormality of the heart or great vessels is present these are called *innocent, functional* or *organic* murmurs. They probably arise from vibrations within the aortic arch near the origins of the head and neck vessels or from the right ventricular outflow tract. They are more common in children and young adults. They are louder just after exercise and during febrile illnesses (a common time for them to be detected).

Innocent murmurs are always systolic. (A venous hum, which is not really a murmur, has both systolic and diastolic components.) They are usually soft and ejection-systolic in character. Those arising from the aortic arch may radiate to the carotids and be heard in the neck. Those arising from the right ventricular outflow tract are loudest in the pulmonary area and may have a scratchy quality.

These outflow tract murmurs must be distinguished from the pulmonary flow murmur of an atrial septal defect. Therefore it is important to listen carefully for wide or fixed splitting of the second heart sound before pronouncing a murmur innocent (see *Questions box 4.6*).

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**Questions box 4.6**

## Questions to ask the patient with a heart murmur

1. Has anyone noticed this murmur before? Were any tests done?
2. Did you have rheumatic fever as a child?
3. Have you been told you need antibiotics before dental work or surgical operations?
4. Have you become breathless when you exert yourself?
5. Have you had chest tightness during exercise?—Aortic stenosis
6. Have you had dizziness or a blackout during heavy exercise?—Severe aortic stenosis
7. Have you been breathless lying flat?—Heart failure complicating valve disease

## Valve diseases of the left heart

### Mitral stenosis

The normal area of the mitral valve is 4 to 6 $cm^2$. Reduction of the valve area to half normal or less causes significant obstruction to left ventricular filling, and blood will flow from the left atrium to the left ventricle only if the left atrial pressure is raised.

• **Symptoms:** dyspnoea, orthopnoea, paroxysmal nocturnal dyspnoea (increased left atrial pressure); haemoptysis (ruptured bronchial veins); ascites, oedema, fatigue (pulmonary hypertension).

• **General signs:** tachypnoea; 'mitral facies'; peripheral cyanosis (severe mitral stenosis).

• **Pulse and blood pressure:** normal or reduced in volume, due to a reduced cardiac output; atrial fibrillation may be present because of left atrial enlargement.

• **The JVP:** normal; prominent *a* wave if pulmonary hypertension is present; loss of the *a* wave if the patient is in atrial fibrillation.

• **Palpation:** tapping quality of the apex beat (palpable S1); right ventricular heave and palpable P2 if pulmonary hypertension is present; diastolic thrill rarely (lay patient on the left side).

• **Auscultation** (Figure 4.45): loud S1 (valve cusps widely apart at the onset of systole)—this also indicates that the valve cusps remain mobile; loud P2 if pulmonary hypertension is present; opening snap (high left atrial pressure forces the valve cusps apart, but the valve cone is halted abruptly); low-pitched rumbling diastolic murmur (best heard with the bell of the stethoscope with the patient in the left lateral position, and quite different in quality and timing from the murmur of aortic regurgitation); a late diastolic accentuation of the diastolic murmur may occur if the patient is in sinus rhythm, but is usually absent if atrial fibrillation has supervened—this is best heard in the left lateral position; exercise accentuates the murmur (ask the patient to sit up and down quickly in bed several times).

• **Signs indicating severe mitral stenosis** (valve area less than 1 $cm^2$): small pulse pressure; soft first heart sound (immobile valve cusps); early opening snap (due to increased left atrial pressure); long diastolic murmur (persists as long as there is a gradient); diastolic thrill at the apex; signs of pulmonary hypertension.

• **Causes of mitral stenosis:** (i) rheumatic (following acute rheumatic fever); (ii) congenital parachute valve (all chordae insert into one papillary muscle—rare).

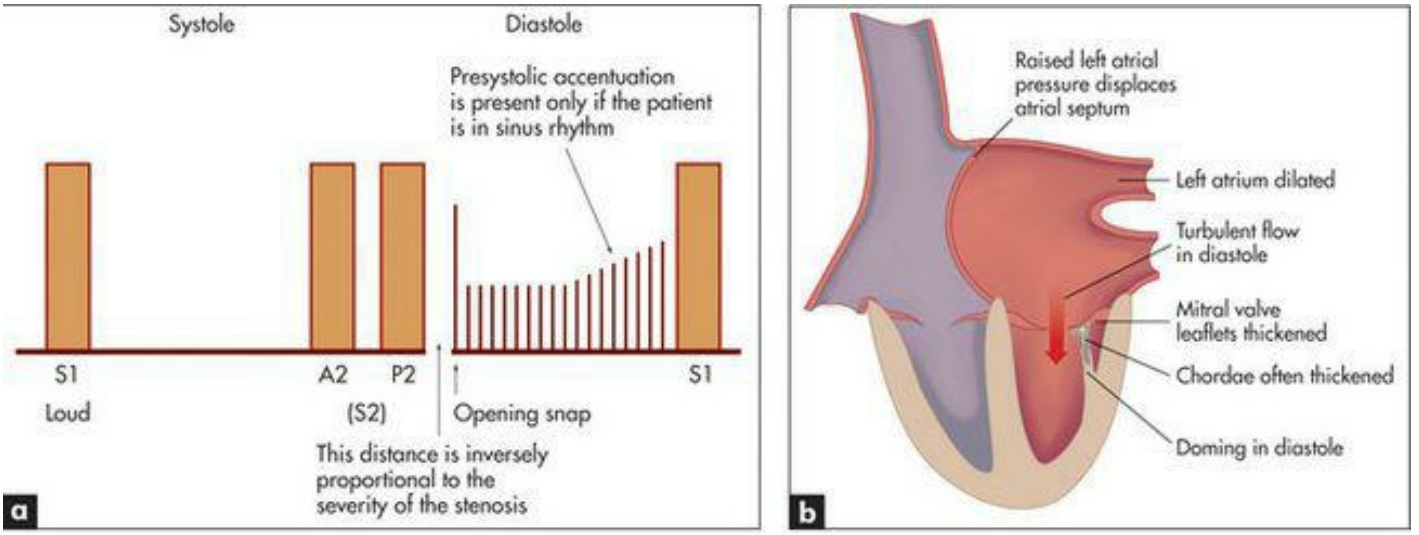



**Figure 4.45** Mitral stenosis, at the apex: (a) murmur; (b) anatomy

## Mitral regurgitation (chronic)

A regurgitant mitral valve allows part of the left ventricular stroke volume to regurgitate into the left atrium, imposing a volume load on both the left

regurgitate into the left atrium, imposing a volume load on both the left atrium and the left ventricle.

• **Symptoms:** dyspnoea (increased left atrial pressure); fatigue (decreased cardiac output).

• **General signs:** tachypnoea.

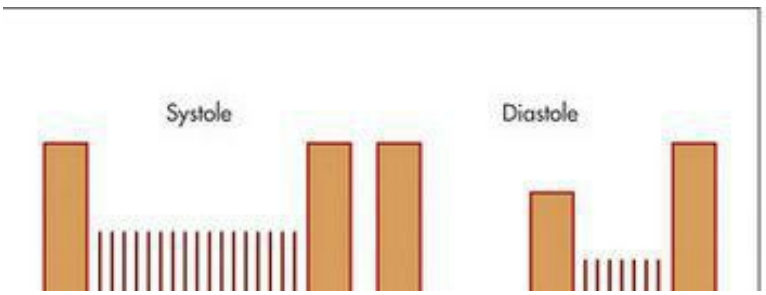
• **Pulse:** normal, or sharp upstroke due to rapid left ventricular decompression; atrial fibrillation is relatively common.

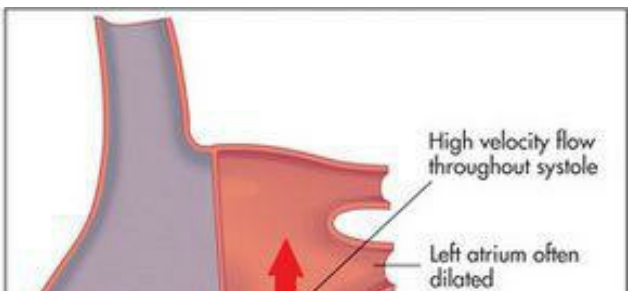
• **Palpation:** the apex beat is displaced, diffuse and hyperdynamic; a pansystolic thrill is occasionally present at the apex; a parasternal impulse (due to left atrial enlargement behind the right ventricle—the left atrium is often larger in mitral regurgitation than in mitral stenosis and can be enormous).

• **Auscultation** (Figure 4.46): soft or absent S1 (by the end of diastole, atrial and ventricular pressures have equalised and the valve cusps have drifted back together); left ventricular S3, which is due to rapid left ventricular filling in early diastole and, when soft, does not imply severe regurgitation; pansystolic murmur maximal at the apex and usually radiating towards the axilla.

• **Signs indicating severe chronic mitral regurgitation:** small volume pulse; enlarged left ventricle; loud S3; soft S1; A2 is early, because rapid left ventricular decompression into the left atrium causes the aortic valve to close early; early diastolic rumble; signs of pulmonary hypertension; signs of left ventricular failure (*GOOD SIGNS GUIDE 4.6*).

• **Causes of chronic mitral regurgitation:** (i) mitral valve prolapse; (ii) 'degenerative'—associated with ageing; (iii) rheumatic; (iv) papillary muscle dysfunction, due to left ventricular failure or ischaemia; (v) cardiomyopathy —hypertrophic, dilated or restrictive cardiomyopathy; (vi) connective tissue disease—e.g. Marfan's syndrome, rheumatoid arthritis, ankylosing spondylitis; (vii) congenital (e.g. atrioventricular canal defect).



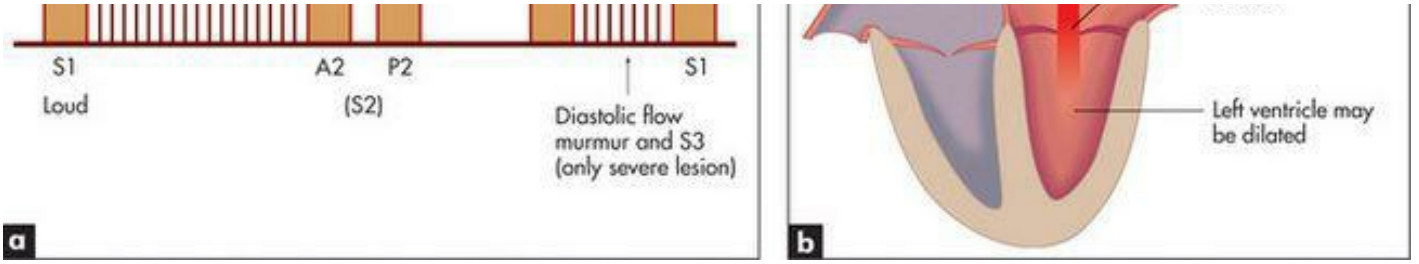



**Figure 4.46** Mitral regurgitation: (a) murmur, at the apex; (b) anatomy

**GOOD SIGNS GUIDE 4.6** Moderate to severe mitral regurgitation

| | Positive LR | Negative LR |
|---|---|---|
| Characteristic murmur grade 3 or more | 4.4 | 0.2 |
| S3 (third heart sound) | 1.8 | 0.8 |

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

### Acute mitral regurgitation

In this case patients can present with pulmonary oedema and cardiovascular collapse. There is usually a systolic apical thrill and a loud apical systolic murmur present (it is short because atrial pressure is increased).

With anterior leaflet chordae rupture the murmur radiates to the axilla and back; with posterior leaflet rupture the murmur radiates to the cardiac base and carotids.

- **Causes:** (i) myocardial infarction (dysfunction or rupture of papillary

• **Causes:** (i) myocardial infarction (dysfunction or rupture of papillary muscles); (ii) infective endocarditis; (iii) trauma or surgery; (iv) spontaneous rupture of a myxomatous chord (sometimes during exercise).

### Mitral valve prolapse (systolic-click murmur syndrome)

This syndrome can cause a systolic murmur or click, or both, at the apex. The presence of the murmur indicates that there is some mitral regurgitation present.

• **Auscultation** (Figure 4.47): systolic click or clicks at a variable time (usually midsystolic) may be the only abnormality audible, but a click is not always audible; the midsystolic click varies 'all over systole' with changing the position of the patient (unlike the ejection click of aorta or pulmonary stenosis)—when supine the click occurs later than when standing; systolic murmur—high-pitched late systolic murmur, commencing with the click and extending throughout the rest of systole.

• **Dynamic auscultation:** murmur and click occur earlier and may become louder with the Valsalva manoeuvre and with standing, but both occur later and may become softer with squatting and isometric exercise.

• **Causes of mitral valve prolapse:** (i) myxomatous degeneration of the mitral valve tissue—it is very common, especially in women, and the severity may increase with age, particularly in men, so that significant mitral regurgitation may supervene; (ii) may be associated with atrial septal defect (secundum), hypertrophic cardiomyopathy, or Marfan's syndrome.

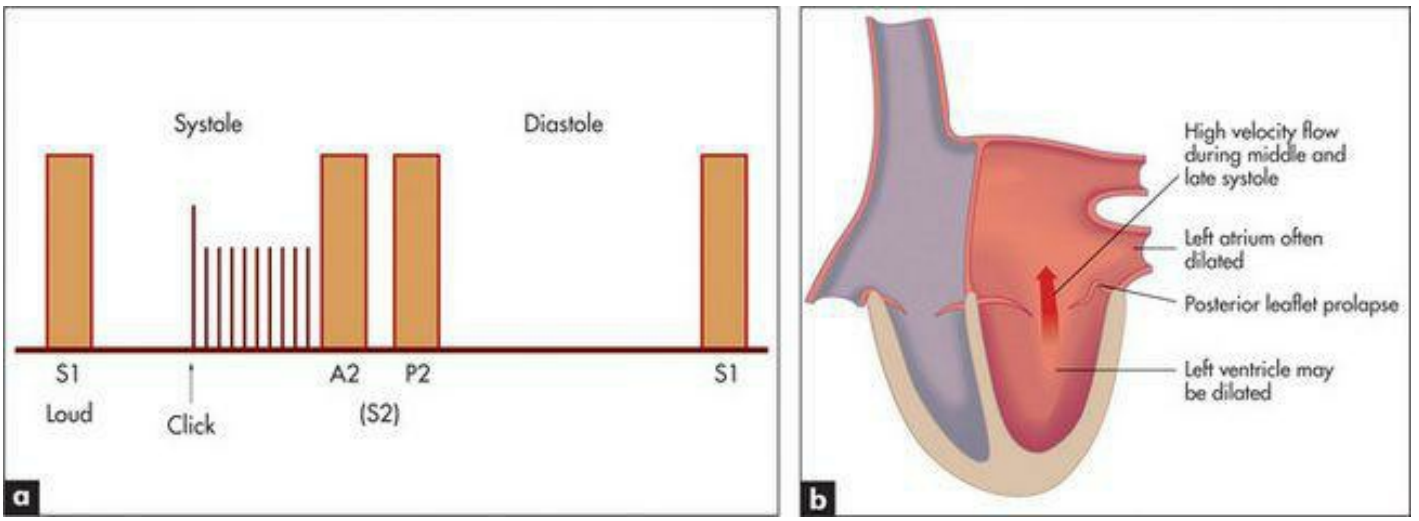



**Figure 4.47** Mitral valve prolapse (MVP): (a) murmur, at the apex; (b) anatomy

## Aortic stenosis

The normal area of the aortic valve is more than 2 $cm^2$. Significant narrowing of this valve restricts left ventricular outflow and imposes a pressure load on the left ventricle.

• **Symptoms:** exertional chest pain (50% do not have coronary artery disease), exertional dyspnoea and exertional syncope.

• **General signs:** usually there is nothing remarkable about the general appearance.

• **The pulse:** there may be a plateau or anacrotic pulse, or the pulse may be late peaking (tardus) and of small volume (parvus).[41]

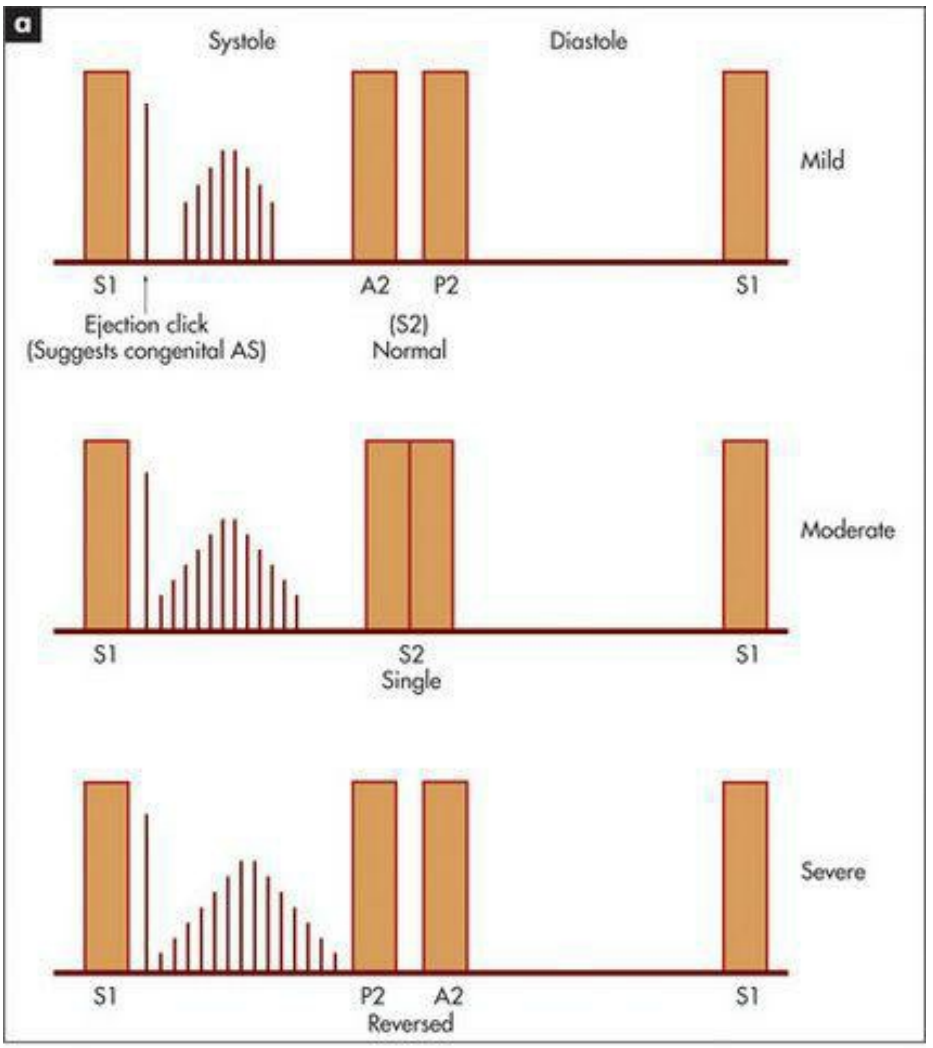
• **Palpation:** the apex beat is hyperdynamic and may be slightly displaced; systolic thrill at the base of the heart (aortic area).

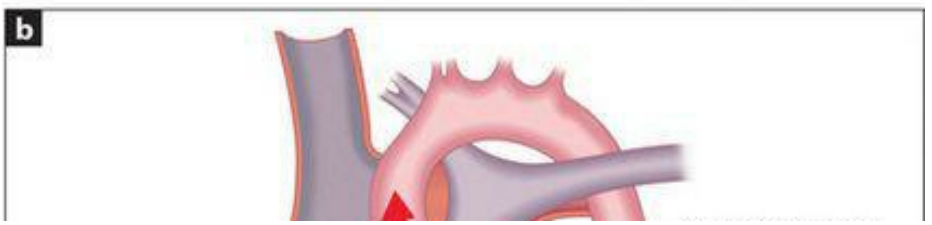
• **Auscultation** (Figure 4.48): a narrowly split or reversed S2 because of delayed left ventricular ejection; a harsh midsystolic ejection murmur, maximal over the aortic area and extending into the carotid arteries (Figure 4.49), is characteristic. However, it may be heard widely over the praecordium and extend to the apex. The murmur is loudest with the patient sitting up and in full expiration; associated aortic regurgitation is common; in congenital aortic stenosis where the valve cusps remain mobile and the dome of the valve comes to a sudden halt, an ejection click may precede the murmur—the ejection click is absent if the valve is calcified or if the stenosis is not at the valve level but above or below it.

• **Signs indicating severe aortic stenosis** (*GOOD SIGNS GUIDE 4.7*) (valve area less than 1 $cm^2$, or valve gradient greater than 50 mmHg): plateau pulse, carotid pulse reduced in force; thrill in the aortic area; length of the murmur and lateness of the peak of the systolic murmur, soft or absent A2; left ventricular failure (very late sign); pressure-loaded apex beat. These signs are not reliable for distinguishing moderate and severe disease. It is important to remember that the signs of severity of aortic stenosis are less reliable in the elderly.[42]

• **Causes of aortic stenosis:** (i) degenerative calcific aortic stenosis, particularly in elderly patients; (ii) calcific in younger patients, usually on a congenital bicuspid valve; (iii) rheumatic.

• **Other types of aortic outflow obstruction are also possible:** (i) supravalvular obstruction, where there is narrowing of the ascending aorta or a fibrous diaphragm just above the aortic valve—this is rare and may be associated with a characteristic facies (a broad forehead, widely set eyes and a pointed chin); there is a loud A2 and often a thrill in the area of the sternal notch; (ii) subvalvular obstruction, where there is a membranous diaphragm or fibrous ridge just below the aortic valve; aortic regurgitation is associated and is due to a jet lesion affecting the coronary cusp of the valve; (iii) dynamic left ventricular outflow tract obstruction may occur in hypertrophic cardiomyopathy; here there may be a double apical impulse. Atrial contraction into a stiff left ventricle may be palpable before the left ventricular impulse (only in the presence of sinus rhythm, of course).



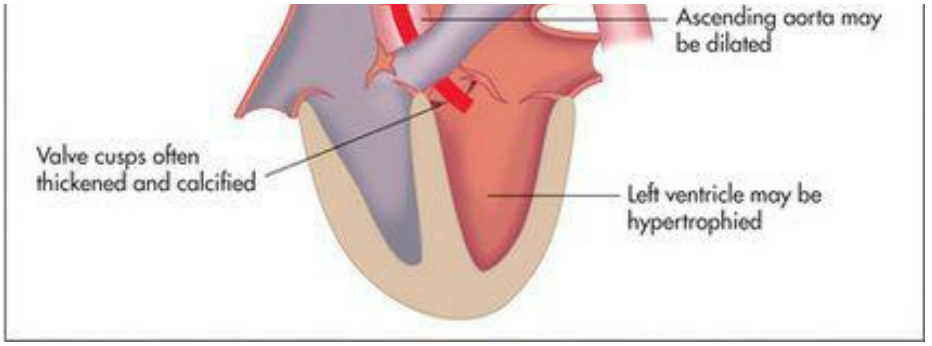



**Figure 4.48** Aortic stenosis (AS): (a) murmur, at the aortic area; (b) anatomy

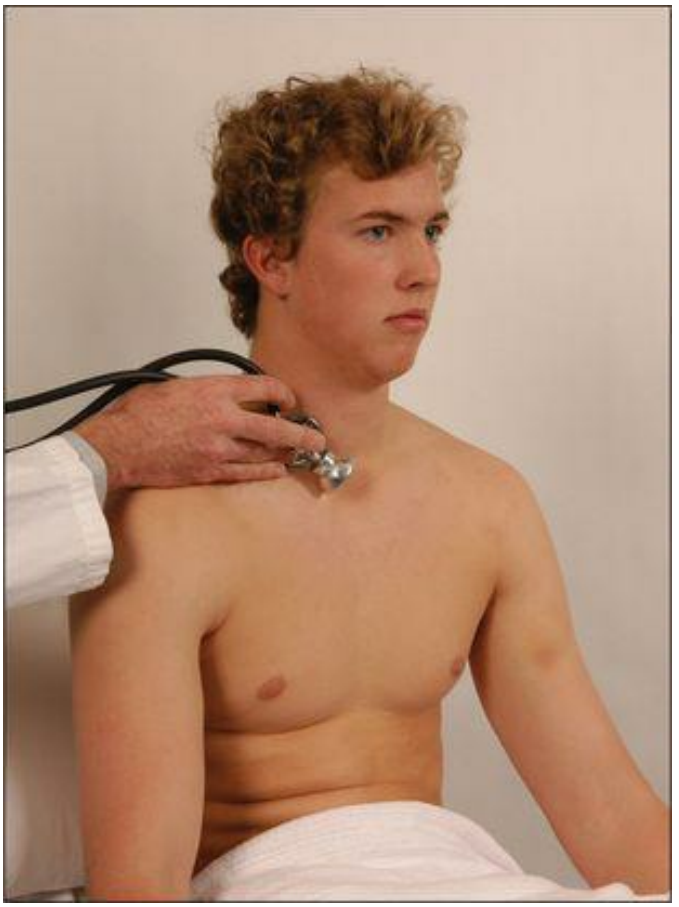


**Figure 4.49** Aortic stenosis: listening over the carotid

**GOOD SIGNS GUIDE 4.7** Severe aortic stenosis

| Sign | Positive LR | Negative LR |
|---|---|---|
| Delayed carotid upstroke | 3.7 | 0.4 |
| Diminished carotid pulse on palpation | 2.3 | 0.3 |
| Apical impulse sustained (pressure-loaded) | 4.1 | 0.3 |
| Absent or decreased A2 | 3.6 | 0.4 |
| S4 gallop | NS | NS |
| Late peaking murmur | 4.4 | 0.2 |
| Long murmur | 3.9 | 0.2 |
| Murmur radiates to the neck | 1.4 | 0.1 |

NS = not significant.

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

Aortic sclerosis presents in the elderly; there are none of the peripheral

signs of aortic stenosis. The diagnosis implies the absence of a gradient across the aortic valve despite some thickening and a murmur.

### Aortic regurgitation

The incompetent aortic valve allows regurgitation of blood from the aorta to the left ventricle during diastole for as long as the aortic diastolic pressure exceeds the left ventricular diastolic pressure.[43]

• **Symptoms:** occur in the late stages of disease and include exertional dyspnoea, fatigue, palpitations (hyperdynamic circulation) and exertional angina.

• **General signs:** Marfan's syndrome, ankylosing spondylitis or one of the other seronegative arthropathies or, rarely, Argyll Robertson pupils may be obvious.

• **Pulse and blood pressure:** the pulse is characteristically collapsing, a 'water hammer'[kk] pulse (Table 4.20); there may be a wide pulse pressure. This sign is most obvious if the clinician raises the patient's arm while feeling the radial pulse with the web spaces of the lifting hand. A *bisferiens* pulse (from the Latin, to beat twice) may be a sign of severe aortic regurgitation or of combined aortic regurgitation and aortic stenosis. It is best assessed at the carotid artery, where two beats can be felt in each cardiac cycle. It is probably caused by a Venturi effect in the aorta related to rapid ejection of blood and brief indrawing of the aortic wall, leading to a diminution of the pulse followed by a rebound increase. It was a particular favourite of Galen's.[ll]

• **Neck:** prominent carotid pulsations (Corrigan's sign).

• **Palpation:** the apex beat is characteristically displaced and hyperkinetic. A diastolic thrill may be felt at the left sternal edge when the patient sits up and breathes out.

• **Auscultation** (Figure 4.50): A2 (the aortic component of the second heart sound) may be soft; a decrescendo high-pitched diastolic murmur beginning immediately after the second heart sound and extending for a variable time into diastole—it is loudest at the third and fourth left intercostal spaces; a systolic ejection murmur is usually present (due to associated aortic stenosis or to torrential flow across a normal diameter aortic valve). Aortic stenosis is distinguished from an aortic flow murmur by the presence of the

peripheral signs of significant aortic stenosis, such as a plateau pulse. An *Austin Flint murmur*[mm] should also be listened for. This is a low-pitched rumbling mid-diastolic and presystolic murmur audible at the apex (the regurgitant jet from the aortic valve causes the anterior mitral valve leaflet to shudder). It can be distinguished from mitral stenosis because S1 (the first heart sound) is not loud and there is no opening snap. Many other signs have been described, but they are interesting rather than helpful (Table 4.20; *GOOD SIGNS GUIDE 4.8*).

• **Signs indicating severe chronic aortic regurgitation:** collapsing pulse; wide pulse pressure (systolic pressure 80 mmHg more than the diastolic); long decrescendo diastolic murmur; left ventricular S3 (third heart sound); soft A2; Austin Flint murmur; signs of left ventricular failure.

• **Causes of aortic regurgitation:** disease may affect the valvular area or aortic root, and may be acute or chronic.

• **Causes of chronic aortic regurgitation:** (i) valvular—rheumatic (rarely the only murmur in this case), congenital (e.g. bicuspid valve; ventricular septal defect—an associated prolapse of the aortic cusp is not uncommon), seronegative arthropathy, especially ankylosing spondylitis; (ii) aortic root dilatation (murmur may be maximal at the right sternal border)—Marfan's syndrome, aortitis (e.g. seronegative arthropathies, rheumatoid arthritis, tertiary syphilis), dissecting aneurysm.

• **Acute aortic regurgitation:** presents differently—there is no collapsing pulse (blood pressure is low) and the diastolic murmur is short.

• **Causes of acute aortic regurgitation:** (i) valvular—infective endocarditis; (ii) aortic root—Marfan's syndrome, dissecting aneurysm of the aortic root.

**TABLE 4.20** Eponymous signs of aortic regurgitation[44]

| |
|---|
| **1** Quincke's sign: capillary pulsation in the nail beds—it is of no value, as this sign occurs normally. |
| **2** Corrigan's sign: prominent carotid pulsations; the Corrigan water hammer pulse sign is present when the patient lies supine with the arms beside the body, the radial pulse is compressed until it disappears, the arm is then lifted perpendicular to the body, the pulse then becomes palpable again even though the same pressure has been maintained on the radial artery. |
| **3** De Musset's sign: head nodding in time with the heartbeat. |
| **4** Hill's sign: increased blood pressure (>20 mmHg) in the legs compared with the arms. |
| **5** Mueller's sign: pulsation of the uvula in time with the heartbeat. |
| **6** Duroziez's sign: systolic and diastolic murmurs over the femoral artery on gradual compression of the vessel. |
| **7** Traube's sign: a double sound heard over the femoral artery on compressing the vessel distally; this is *not* a 'pistol shot' sound that may be heard over the femoral artery with very severe aortic regurgitation. |
| **8** Mayne's sign: a decrease in diastolic pressure of 15 mmHg when the arm is held above the head compared with that when the arm is at the level of the heart. |
| **9** Rosenbach's liver pulsation sign: liver pulsates in time with the heartbeat (in the absence of tricuspid regurgitation). |
| **10** Austin Flint murmur: short rumbling diastolic murmur, thought by Flint to be due to functional mitral stenosis caused by impinging of the aortic regurgitant jet on the anterior mitral valve leaflet. |

| |
|---|
| **11** Becker's sign: accentuated retinal artery pulsations. |
| **12** Gerhard's sign: pulsatile spleen. |
| **13** Landolfi's sign: prominent alternating constriction and dilatation of the pupils (hippus, from the Greek *hippos*—'horse'—and its rhythmical galloping). |
| **14** Lincoln's sign: an easily palpable popliteal pulse. |
| **15** Sherman's sign: an easily palpable dorsalis pedis pulse in a patient over the age of 75 years. |
| **16** Watson's water hammer pulse. |
| **17** Ashrafian's sign: pulsatile pseudo proptosis. |

*Note:* These signs are amusing, but not often helpful. The signs were named after the following people: Heinrich Quincke (1842–1922), German neurologist; Dominic Corrigan (1802–80), Edinburgh graduate who worked in Dublin and is credited with discovering aortic regurgitation; Alfred de Musset, 19th century French poet who suffered from aortic regurgitation (the sign was noticed by his brother, a physician); Sir Leonard Hill (1866–1952), English physiologist who also described the physiology of the cerebral circulation; Frederick Von Mueller (1858–1941), German physician who also noted an increase in metabolism in exophthalmic goitre; Paul Duroziez (1826–97), French physician; Ludwig Traube (1818–76), Hungarian physician who worked in Germany; Otto Heinrich Becker (1828–1890), professor of ophthalmology, University of Heidelberg, who also described this sign in patients with Graves' disease; Lincoln's sign is like de Musset's sign in being named after the patient with the condition; Thomas Watson, English physician, described this sign in 1844; Hutan Ashrafian, cardiothoracic surgeon, St Mary's Hospital, London, described this in 2006—proof that the hunt for more signs of aortic regurgitation goes on.

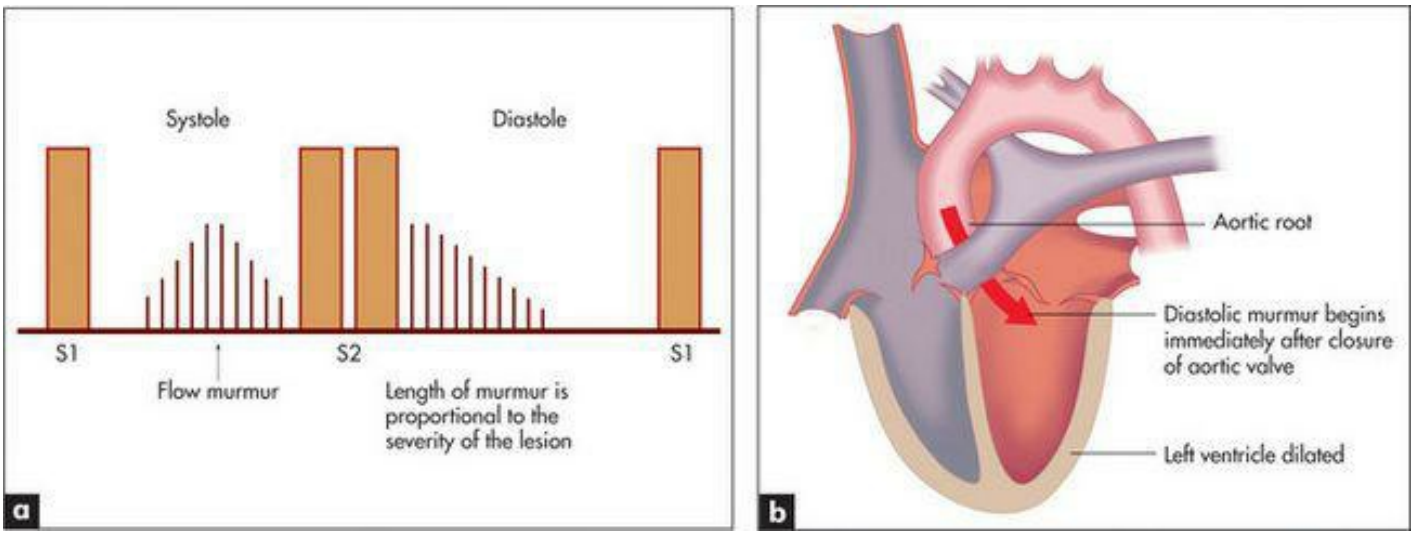



**Figure 4.50** Aortic regurgitation: (a) murmur, at the left sternal edge; (b) anatomy

**GOOD SIGNS GUIDE 4.8** Severity of aortic regurgitation (AR)

| Finding | Positive LR | Negative LR |
|---|---|---|
| **Typical murmur** | | |
| Mild AR or worse | 9.9 | 0.3 |
| Moderate to severe AR | 4.3 | 0.1 |
| **Murmur grade 3 or more** (moderate to severe AR) | 8.2 | 0.6 |
| **Pulse pressure** | | |
| >80 mmHg | 10.9 | – |
| **Other signs—moderate to severe AR** | | |
| Sustained or displaced apex | 2.4 | 0.1 |
| Duroziez's sign, pistol shot femorals, water hammer pulse | NS | 0.7 |

NS = not significant.

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

## Valve diseases of the right heart

### Tricuspid stenosis

This is very rare.

• **The JVP:** raised; giant *a* waves with a slow *y* descent may be seen.

• **Auscultation:** a diastolic murmur audible at the left sternal edge, accentuated by inspiration, very similar to the murmur of mitral stenosis except for the site of maximal intensity and the effect of respiration (louder on inspiration); tricuspid regurgitation and mitral stenosis are often present as well; no signs of pulmonary hypertension.

• **Abdomen:** presystolic pulsation of the liver, caused by forceful atrial systole.

• **Cause of tricuspid stenosis:** rheumatic heart disease.

### Tricuspid regurgitation (Figure 4.51)

• **The JVP:** large *v* waves; the JVP is elevated if right ventricular failure has occurred.

• **Palpation:** right ventricular heave.

• **Auscultation:** there may be a pansystolic murmur maximal at the lower end of the sternum that increases on inspiration, but the diagnosis can be made on the basis of the peripheral signs alone.

• **Abdomen:** a pulsatile, large and tender liver is usually present and may cause the right nipple to dance in time with the heart beat; ascites, oedema and pleural effusions may also be present.

• **Legs:** dilated, pulsatile veins.

• **Causes of tricuspid regurgitation:**[nn] (i) functional (no disease of the valve leaflets)—right ventricular failure; (ii) rheumatic—only very rarely does rheumatic tricuspid regurgitation occur alone, usually mitral valve disease is also present; (iii) infective endocarditis (right-sided endocarditis in intravenous drug addicts); (iv) tricuspid valve prolapse; (v) right ventricular papillary muscle infarction; (vi) trauma (usually caused by a steering wheel injury to the sternum): (vii) congenital—Ebstein's anomaly.[oo]

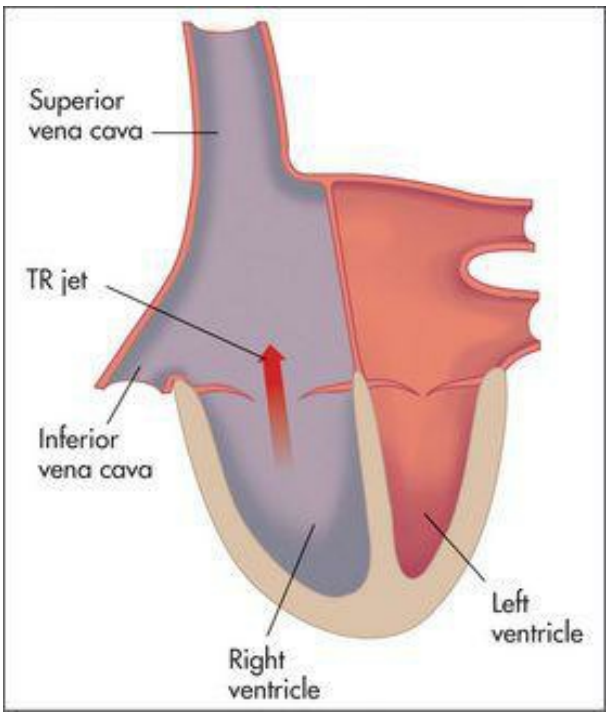



**Figure 4.51** Tricuspid regurgitation (TR): anatomy

## Pulmonary stenosis (in adults) (Figure 4.52)

• **General signs:** peripheral cyanosis, due to a low cardiac output, but only in severe cases.

• **Pulse:** normal or reduced if cardiac output is low.

• **The JVP:** giant *a* waves because of right atrial hypertrophy; the JVP may be elevated.

• **Palpation:** right ventricular heave; thrill over the pulmonary area.

• **Auscultation:** the murmur may be preceded by an ejection click; a harsh and usually loud ejection systolic murmur, heard best in the pulmonary area and with inspiration, is typically present; right ventricular S4 may be present (due to right atrial hypertrophy). It is not well heard over the carotid arteries.

• **Abdomen:** presystolic pulsation of the liver may be present.

• **Signs of severe pulmonary stenosis:** an ejection systolic murmur peaking late in systole; absence of an ejection click (also absent when the pulmonary stenosis is infundibular—i.e. below the valve level); presence of S4; signs of right ventricular failure.

• **Causes of pulmonary stenosis:** (i) congenital; (ii) carcinoid syndrome (rare).

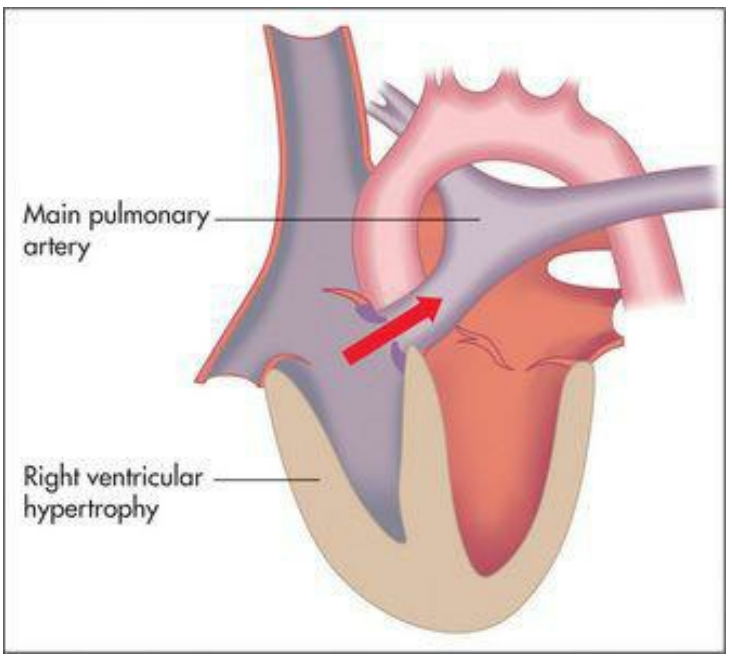



**Figure 4.52** Valvar pulmonary stenosis: anatomy

## Pulmonary regurgitation

This is an uncommon pathological condition; trivial pulmonary regurgitation is often found at echocardiography and is considered physiological.

• **Auscultation:** a decrescendo diastolic murmur which is high-pitched and audible at the left sternal edge is characteristic—this typically but not always increases on inspiration (unlike the murmur of aortic regurgitation). It is

called the Graham Steell murmur[pp] when it occurs secondary to pulmonary artery dilatation caused by pulmonary hypertension. (*Note:* If there are no signs of pulmonary hypertension, a decrescendo diastolic murmur at the left sternal edge is more likely to be due to aortic regurgitation than to pulmonary regurgitation.)

• **Causes of pulmonary regurgitation:** (i) pulmonary hypertension; (ii) infective endocarditis; (iii) following balloon valvotomy for pulmonary stenosis or surgery for pulmonary atresia; (iv) congenital absence of the pulmonary valve.

### Prosthetic heart valves

The physical signs with common types of valves are presented in Table 4.21. Mechanical prosthetic valves should have a crisp sound. Muffling of the mechanical sounds may be a sign of thrombotic obstruction of the valve or chronic tissue ingrowth (pannus). After replacement of the aortic valve the presence of audible aortic regurgitation may indicate a *paravalvular leak*, often through a stitching hole in the valve sewing ring. As tissue valves age and degenerate they may develop signs of regurgitation or stenosis, or both.

**TABLE 4.21** Prosthetic heart valves: physical signs

| | | |
|---|---|---|

| Type | Mitral | Aortic |
|---|---|---|
| Ball valve (e.g. Starr-Edwards)* | Sharp mitral opening sound after S2, sharp closing sound at S1 Systolic ejection murmur, no diastolic murmur | Sharp aortic opening sound Systolic ejection murmur (harsh), no diastolic murmur unless a paravalvular leak has occurred, early diastolic murmur indicates AR usually due to a paravalvular leak** |
| Disc valve (e.g. Bjork-Shiley)† | Sharp closing sound at S1, soft systolic ejection murmur and diastolic rumble (diastolic murmur occasionally) | Sharp closing sound at S2, systolic ejection murmur (soft) |
| Porcine or bovine pericardial valve‡ | Usually sound normal, diastolic rumble mitral opening sound occasionally | Closing sound usually heard, systolic ejection murmur (soft), no diastolic murmur |
| Bileaflet valve (e.g. St Jude) | — | Aortic valve opening and closing sounds common, soft systolic ejection murmur common |
| Homograft (human) valve | Normal heart sounds, occasional soft systolic murmur; early diastolic murmur if AR has occurred | |

AR = aortic regurgitation.

* Modern mechanical valves (e.g. St Jude) make softer opening and closing sounds than older valves. The Starr-Edwards valve is often very noisy and sounds like a ball rattling around in a cage (which is what it is).

**

An aortic regurgitation murmur present after aortic valve replacement suggests regurgitation of the valve ring. It is not uncommon. Less often a mitral regurgitation murmur suggests the same problem with a prosthetic mitral valve.

† Severe prosthetic dysfunction causes absence of the opening or closing sounds. Ball and cage valves cause more haemolysis than other types and make the most noise, while disc valves are more thrombogenic.

‡ Bioprosthetic obstruction or patient–prosthetic mismatch cause diastolic rumbling. These valves are used less often in the mitral position because they often have a very limited life there. A degenerated bioprosthetic valve may cause murmurs of regurgitation or stenosis or both.

*Modified from Smith ND, Raizada V, Abrams J. Auscultation of the normally functioning prosthetic valve. Ann Intern Med 1981; 95:594.*

## Cardiomyopathy

### Hypertrophic cardiomyopathy (Figure 4.53)

This is abnormal hypertrophy of the muscle in the left ventricular or right ventricular outflow tract, or both. It can obstruct outflow from the left ventricle late in systole when the hypertrophied area contracts. Systolic displacement of the mitral valve apparatus into the left ventricular outflow tract also occurs, causing mitral regurgitation and contributing to the outflow obstruction. Although the outflow tract is narrowed by the hypertrophied septum, the major contribution to the dynamic increase in obstruction comes from the systolic movement of the mitral valve. Variants of hypertrophic cardiomyopathy may involve the mid-ventricle or apex with varying degrees of obstruction.

• **Symptoms:** dyspnoea (increased left ventricular end-diastolic pressure due to abnormal diastolic compliance), angina, syncope or sudden death (secondary to ventricular fibrillation or a sudden increase in outflow obstruction).

• **Pulse:** sharp, rising and jerky. Rapid ejection by the hypertrophied ventricle early in systole is followed by obstruction caused by the displacement of the mitral valve into the outflow tract. This is quite different from the pulse of aortic stenosis.

• **The JVP:** there is usually a prominent *a* wave, due to forceful atrial contraction against a non-compliant right ventricle.

• **Palpation:** double or triple apical impulse, due to presystolic expansion of the ventricle caused by atrial contraction.

• **Auscultation:** late systolic murmur at the lower left sternal edge and apex (due to the obstruction) and a pansystolic murmur at the apex (due to

apex (due to the obstruction) and a pansystolic murmur at the apex (due to mitral regurgitation); S4.

• **Dynamic manoeuvres:** the outflow murmur is increased by the Valsalva manoeuvre, by standing and by isotonic exercise; it is decreased by squatting and isometric exercise.

• **Causes of hypertrophic cardiomyopathy:** (i) autosomal dominant (sarcomeric heavy chain or troponin gene mutation) with variable expressivity; (ii) idiopathic; (iii) Friedreich's ataxia[qq] (page 396).

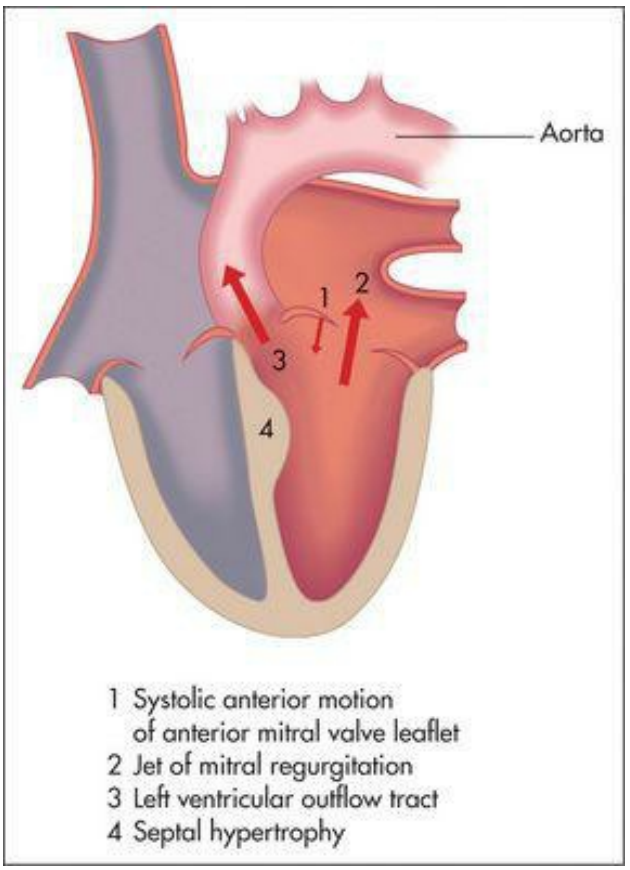



**Figure 4.53** Hypertrophic pulmonary stenosis: anatomy

## Dilated cardiomyopathy

This heart muscle abnormality results in a global reduction in cardiac function. Coronary artery disease is excluded as a cause by definition. (Ischaemic cardiomyopathy is a term often used to describe severe myocardial dysfunction secondary to recurrent ischaemic events.) The signs are those of congestive cardiac failure, including those of mitral and tricuspid regurgitation. The heart sounds themselves may be very quiet. Ventricular

arrhythmias are common. It is a common indication for cardiac transplantation.

• **Causes of dilated cardiomyopathy:** (i) idiopathic and familial; (ii) alcohol; (iii) post-viral; (iv) postpartum; (v) drugs (e.g. doxorubicin); (vi) dystrophia myotonica; (vii) haemochromatosis.

### Restrictive cardiomyopathy

This causes similar signs to those caused by constrictive pericarditis, but Kussmaul's sign is more common and the apex beat is usually easily palpable.

• **Causes of restrictive cardiomyopathy:** (i) idiopathic; (ii) eosinophilic endomyocardial disease; (iii) endomyocardial fibrosis; (iv) infiltrative disease (e.g. amyloid); (v) granulomas (e.g. sarcoid).

## Acyanotic congenital heart disease

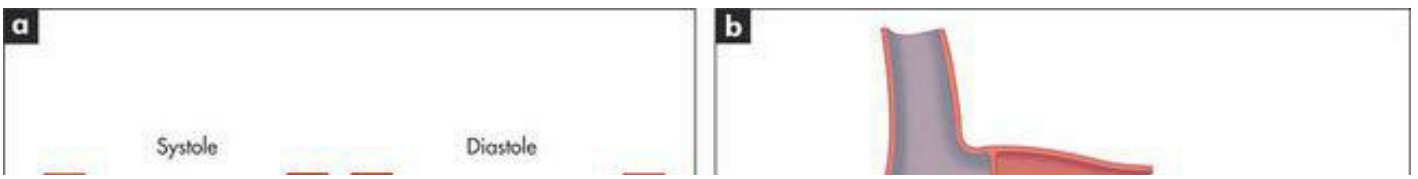
### Ventricular septal defect

In this condition one or more holes are present in the membranous or muscular ventricular septum.

• **Palpation:** hyperkinetic displaced apex if the defect is large; and a thrill at the left sternal edge.

• **Auscultation** (Figure 4.54): a harsh pansystolic murmur maximal at, and almost confined to, the lower left sternal edge with a third or fourth heart sound—the murmur is louder on expiration; sometimes a mitral regurgitation murmur is associated. There is often a palpable systolic thrill. The murmur is often louder and harsher when the defect is small.

• **Causes of ventricular septal defect:** (i) congenital; (ii) acquired—e.g. myocardial infarction involving the septum.



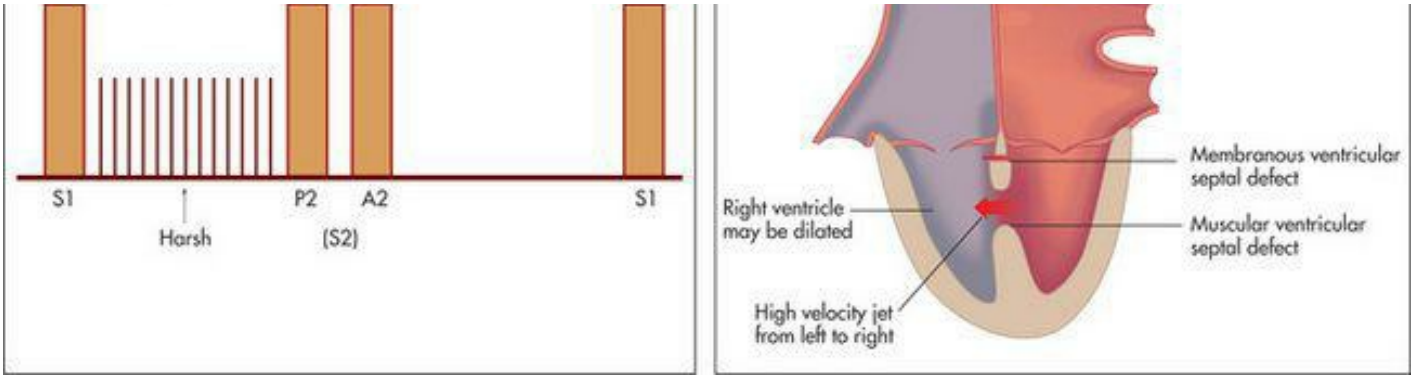



**Figure 4.54** Ventricular septal defect (VSD): (a) murmur, at the left sternal edge; (b) anatomy

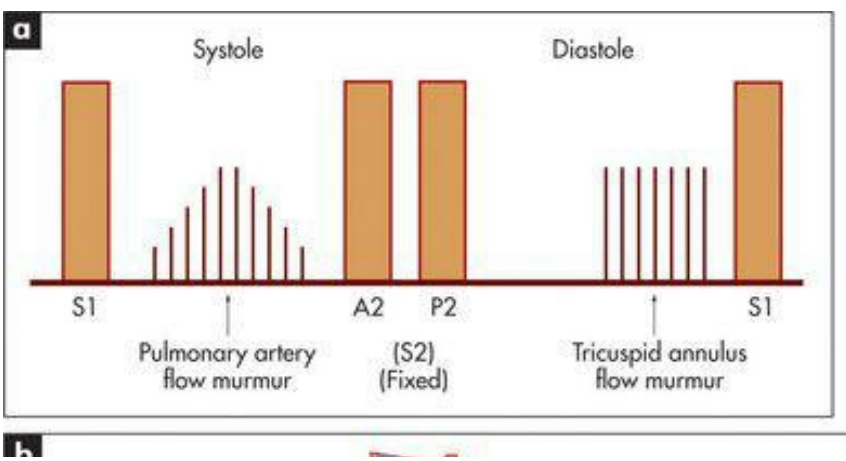
## Atrial septal defect

There are two main types: *ostium secundum* (90%), where there is a defect in the part of the septum which does not involve the atrioventricular valves, and *ostium primum*, where the defect does involve the atrioventricular valves.

- **Palpation:** normal or right ventricular enlargement.

- **Auscultation** (Figure 4.55): fixed splitting of S2; the defect produces no murmur directly, but increased flow through the right side of the heart can produce a low-pitched diastolic tricuspid flow murmur and more often a pulmonary systolic ejection murmur—these are both louder on inspiration.

- **Signs:** The signs of an ostium primum defect are the same as for an ostium secundum defect, but associated mitral regurgitation, tricuspid regurgitation or a ventricular septal defect may be present. The left ventricular impulse is often impalpable.



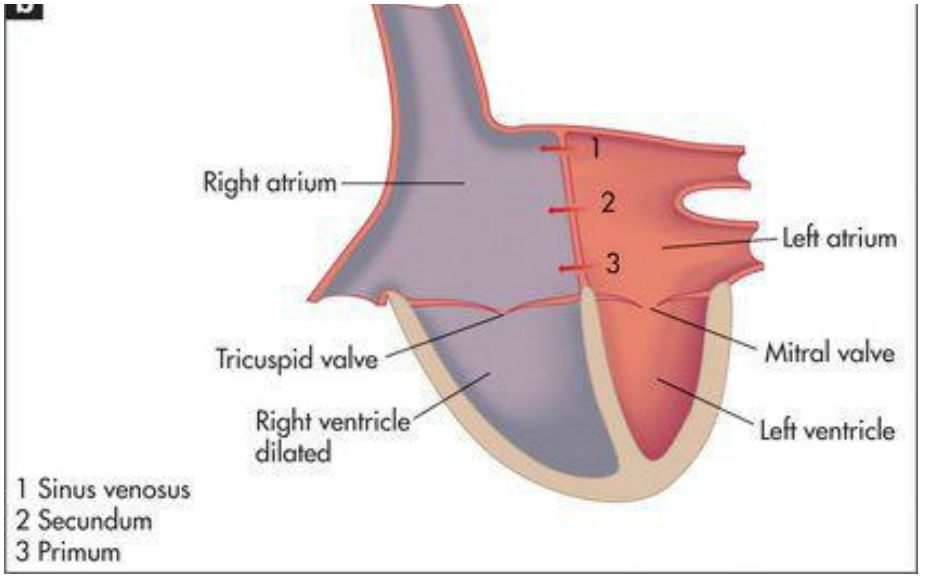



**Figure 4.55** Atrial septal defect (ASD): (a) murmur, at the left sternal edge; (b) anatomy

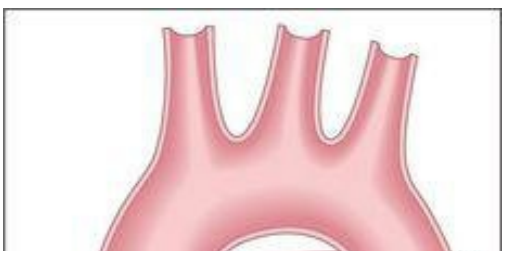
### Patent ductus arteriosus (Figure 4.56)

This is a persistent embryonic vessel which connects the pulmonary artery and the aorta. The shunt is from the aorta to the pulmonary artery unless pulmonary hypertension has supervened.

• **Pulse and blood pressure:** a collapsing pulse with a sharp upstroke (due to ejection of a large volume of blood into the empty aorta with systole); low diastolic blood pressure (due to rapid decompression of the aorta).

• **Palpation:** often there is a hyperkinetic apex beat.

• **Auscultation:** if the shunt is of moderate size a single second heart sound is heard, but if the shunt is of significant size reversed splitting of the second heart sound occurs (due to a delayed A2 because of an increased volume load in the left ventricle); a continuous loud 'machinery' murmur maximal at the first left intercostal space is usually present; flow murmurs through the left side of the heart, including a mitral mid-diastolic murmur, may be heard.



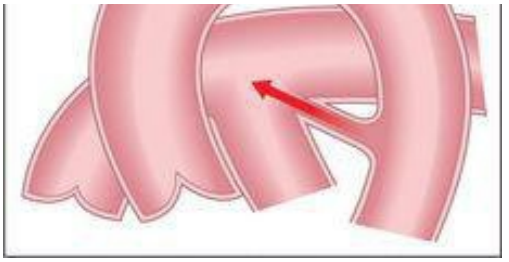


**Figure 4.56** Patent ductus arteriosus: anatomy

## Coarctation of the aorta (Figure 4.57)

This is congenital narrowing of the aorta usually just distal to the origin of the left subclavian artery. It is more common in males. The underlying cause is uncertain but seems related to abnormal placement of tissue involved in the closing of the ductus arteriosus. There is an association with bicuspid aortic valve and Turner's syndrome.

• **Signs:** the upper body may be better developed than the lower; radiofemoral delay is present, and the femoral pulses are weak; hypertension occurs in the arms but not in the legs; a midsystolic murmur is usually audible over the praecordium and the back, due to blood flow through collateral chest vessels and across the coarct itself.

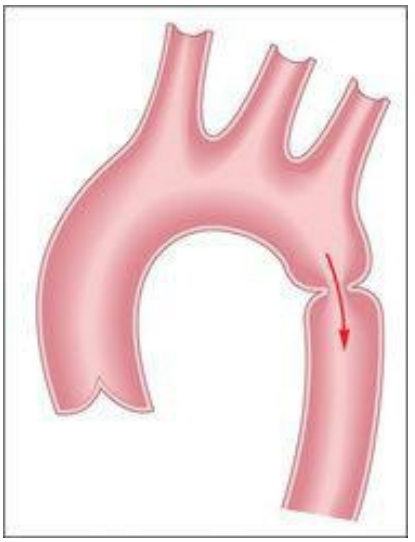


**Figure 4.57** Coarctation of the aorta: anatomy

## Ebstein's anomaly

This is a very rare lesion. The abnormality is a downward displacement of the tricuspid valve apparatus into the right ventricle so that the right atrium becomes very large and consists partly of ventricular muscle, while the right ventricle becomes small. An atrial septal defect is commonly associated. Characteristically, multiple clicks occur due to asynchronous closure of the tricuspid valve. Tricuspid regurgitation is usually present.

## Cyanotic congenital heart disease

This is a difficult area. The causes are listed in Table 4.22. The important point to determine is whether or not signs of pulmonary hypertension are present. Congenital heart disease in which a shunt from the left to the right side of the circulation occurs leads to an increase in pulmonary blood flow. This can cause reactive pulmonary hypertension so that pulmonary pressures eventually exceed systemic pressures. When that happens, the systemic to pulmonary (left to right) shunt will reverse. This right-to-left shunt leads to deoxygenated blood being mixed in the systemic circulation, resulting in cyanosis. This is called Eisenmenger's syndrome.[rr]

**TABLE 4.22** Classification of congenital heart disease

| **Acyanotic** |
| --- |
| **With left-to-right shunt** |

| |
|---|
| Ventricular septal defect |
| Atrial septal defect |
| Patent ductus arteriosus |
| **With no shunt** |
| Bicuspid aortic valve, congenital aortic stenosis |
| Coarctation of aorta |
| Dextrocardia |
| Pulmonary stenosis, tricuspid stenosis |
| Ebstein's anomaly |
| **Cyanotic** |
| Eisenmenger's syndrome (pulmonary hypertension and a right-to-left shunt) |
| Tetralogy of Fallot |
| Ebstein's anomaly (if an atrial septal defect and right-to-left shunt are also present) |
| Truncus arteriosus |
| Transposition of the great vessels |
| Tricuspid atresia |
| Total anomalous pulmonary venous drainage |

### Eisenmenger's syndrome (pulmonary hypertension and a right-to-left shunt)

• **Signs:** central cyanosis; clubbing; polycythaemia; signs of pulmonary hypertension.

It may be possible to decide at what level the shunt occurs by listening to the second heart sound (S2). If there is wide fixed splitting, this suggests an atrial septal defect. If a single second heart sound is present, this suggests truncus arteriosus or a ventricular septal defect. A normal or reversed S2 suggests a patent ductus arteriosus.

### Tetralogy of Fallot[33]

There are four features which are due to a single developmental abnormality: (i) ventricular septal defect (VSD); (ii) right ventricular outflow obstruction, which determines the severity of the condition, and can be at the pulmonary valve or infundibular level; (iii) an aorta which overrides the VSD and is responsible for the cyanosis; and (iv) right ventricular hypertrophy secondary to outflow obstruction.

• **Signs:** central cyanosis—this occurs without pulmonary hypertension because venous mixing is possible at the ventricular level, where pressures are balanced. The aorta overrides both ventricles and so receives right and left ventricular blood. Clubbing and polycythaemia are usually present. There may be evidence of right ventricular enlargement—a parasternal impulse at the left sternal edge. A systolic thrill caused by pulmonary valve or right ventricular outflow obstruction may be present. There is no overall cardiomegaly. On auscultation the second heart sound is single and there are no signs of pulmonary hypertension; a pulmonary systolic ejection murmur is present.

## 'Grown-up' congenital heart disease

Patients who have been treated for serious congenital cardiac conditions now frequently survive into adult life. Many of the surgical procedures undertaken for these conditions, especially 20 years ago, were palliative rather than curative. The patients present with specific symptoms and signs.

### Tetralogy of Fallot

Patients who have had repair of this condition in infancy may present with particular problems. Repair of the right ventricular outflow obstruction and enlargement of the pulmonary valve annulus may leave severe pulmonary regurgitation. This may lead eventually to exertional dyspnoea. The surgery itself has, until recently, required a right ventriculotomy (cutting into the right ventricle). This leaves a scar that can be associated with cardiac rhythm abnormalities in later life. Patients may present with palpitations or syncope.

• **Signs:** may include a median sternotomy scar, a long diastolic murmur of pulmonary regurgitation, and signs of right ventricular enlargement (parasternal impulse) and later of tricuspid regurgitation (big *v* waves in the neck and a pulsatile liver).

**Table 4.23** Features of important valve lesions and congenital abnormalities

| | Site | Timing | Radiation | Character | Accentuation and manoeuvres | Other features |
|---|---|---|---|---|---|---|
| Aortic regurgitation | Aortic area | Early diastolic | Lower left sternal edge | Decrescendo | Expiration, patient leaning forward | Wide pulse pressure, eponymous signs |
| Aortic stenosis | Aortic area | Systolic | Carotids | Ejection | Expiration | Separate from heart sounds, slow-rising pulse |
| Mitral stenosis | Apex | Middle and late diastolic | – | Low-pitched (use stethoscope bell) | Presystolic accentuation, left lateral position, exercise | Loud S1, opening snap |
| Mitral regurgitation | Apex | Pansystolic or middle and late systolic (mitral valve prolapse) | Axilla or left sternal edge | Blowing (MVP) | Longer and louder with Valsalva (MVP) | Para-sternal impulse (enlarges left atrium) |
| Ventricular septal defect | Lower left sternal edge | Pansystolic | None | Localised | | Often associated with a thrill |
| Tricuspid regurgitation | Lower left and right sternal edge | Pansystolic | | | Louder on inspiration | Big V waves, pulsatile liver |
| Hypertrophic cardiomyopathy | Apex and left sternal edge | Late systolic at left sternal edge, pan-systolic at apex | | | Louder with Valsalva, softer with squatting | S4, double-impulse apex beat, jerky carotid pulse |

MVP = mitral valve prolapse.

## Transposition of the great arteries

Most adults who have had surgery for this abnormality have had a palliative operation called a Mustard procedure. In this abnormality, the pulmonary artery is connected to the left ventricle and the aorta to the right ventricle. Thus the systemic and pulmonary circulations are in parallel. This is not compatible with life unless some connection between the two circulations is present. Neonates with the condition will have an atrial septal defect (ASD) created soon after birth with a catheter-based balloon (balloon septostomy). This allows mixing of the circulations. Later 'baffles' are created surgically in the atria to direct blood returning from the body into the right atrium across the ASD and into the left atrium, where it is pumped into the pulmonary artery and into the lungs. Blood returning from the lungs into the left atrium is directed across into the right atrium and into the morphological right ventricle and on into the aorta. This means that the morphological right ventricle is working as the systemic ventricle. This arrangement works very well, but there are long-term concerns about the ability of the right ventricle to cope with systemic workloads.

- **Symptoms:** symptoms that commonly occur include palpitations caused

by supraventricular arrhythmias, dizziness caused by bradycardias and breathlessness related to failure of the systemic ventricle. Occasionally, obstruction of the baffles may occur. The most common problem is with the superior vena caval baffle which leads to facial swelling and flushing.

• **Signs:** include the usual scar, facial flushing and oedema, cyanosis, peripheral oedema from inferior caval baffle obstruction and signs of tricuspid regurgitation. On auscultation there may be a gallop rhythm and the murmurs of mitral and tricuspid regurgitation.

## The chest X-ray: a systematic approach

Analysis of the chest X-ray is complementary to the patient's physical examination. It provides much information about the heart and lungs.

The interpretation of the chest X-ray is not easy. It requires knowledge of anatomy and pathology, appreciation of the whole range of normal appearances (Figure 4.58), and knowledge of the likely X-ray changes occurring with pathological processes. The clinician should feel personally responsible for viewing a patient's radiographs.

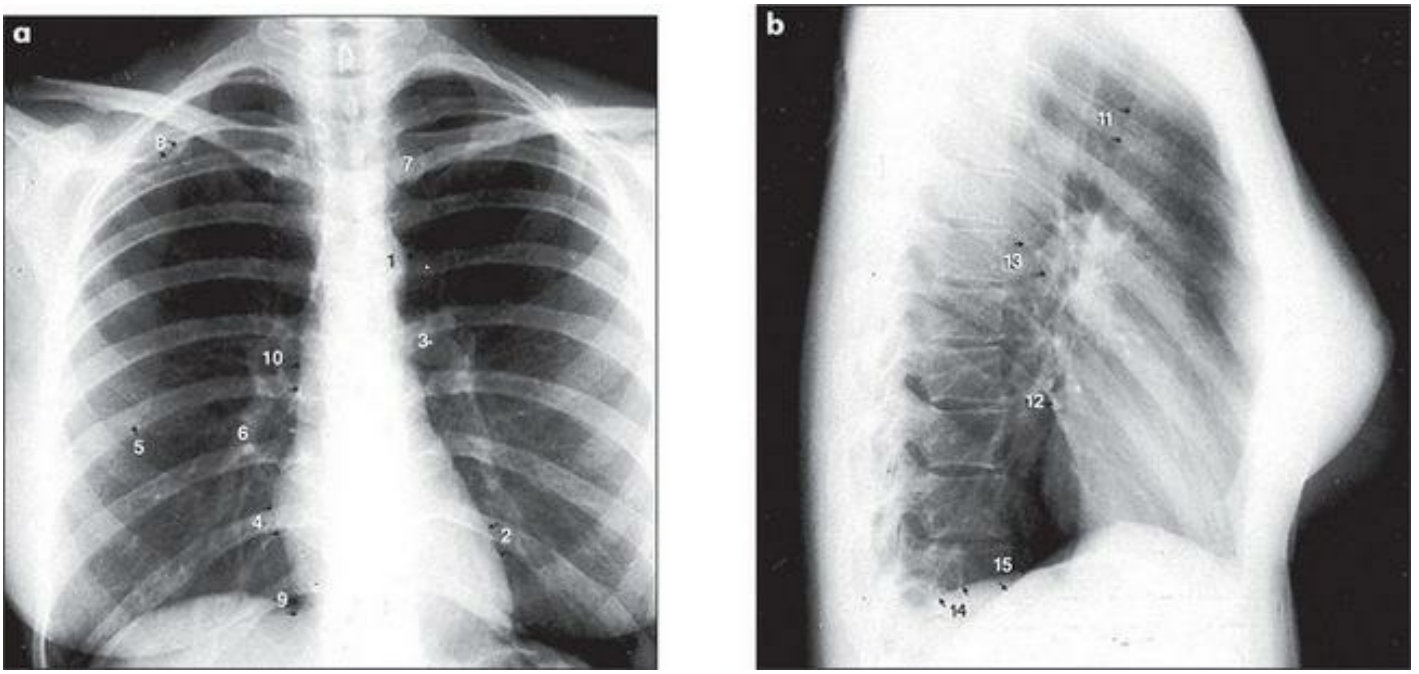



**Figure 4.58** Normal chest X-ray
(a) The posteroanterior view shows (1) aortic knuckle; (2) left heart border formed by the lateral border of the left ventricle; (3) left hilum, formed mostly by the left main pulmonary artery and partly by the left upper pulmonary veins; (4) right heart border formed by the right atrium; (5) inferior angle of the scapula; (6) right basal pulmonary artery; (7) medial aspect of the left clavicle; (8) spine of the scapula; (9) right cardiophrenic angle; (10) superimposition of right lateral margins of the superior vena cava and the ascending aorta

of right lateral margins of the superior vena cava and the ascending aorta.
(b) The lateral view shows: (11) anterior border of trachea; (12) pulmonary vein, entering left atrium; (13) oblique fissure; (14) left hemidiaphragm; (15) right hemidiaphragm.

Most medical students faced with giving their interpretation of a chest X-ray either opt for a 'spot diagnosis' (usually wrong) or raise their eyes to heaven, hoping for divine inspiration. However, a systematic approach is generally more useful! More is missed by not looking than by not knowing.

## Frontal film

### Name, date and projection

First, it is important to check the name and date, to be sure that it is the correct patient's film. Checking the left or right marker prevents missing dextrocardia. The film markings will also indicate the projection and patient position. The standard frontal film is taken by a posteroanterior (PA; back to front) projection of an erect patient. Anteroposterior (AP) and supine films are only second-best. On a supine film there is distension of all the posterior (gravity-dependent) vessels and thus the lung fields appear more plethoric. A small pleural effusion may not be visible if it is lying posteriorly and the heart often appears large on a supine film.

### Centring

The medial ends of the clavicles should be equidistant from the midline spinous processes. If the patient is rotated, this will accentuate the hilum that is turned forwards.

### Exposure

The quality of the film is important. There should be enough X-ray penetration for the spine to be just seen through the mediastinum, otherwise the film will be too white. With good radiographic technique, the scapulae are projected outside the lung fields.

The film needs to be exposed on full inspiration so that there is no basal crowding of the pulmonary vessels and so that estimation of the cardiothoracic ratio is accurate.

On full inspiration, the diaphragm lies at the level of the tenth or

eleventh rib posteriorly or at the level of the sixth costal cartilage anteriorly. The right hemidiaphragm usually lies about 2 cm higher than the left.

### Correct orientation

Do not miss dextrocardia—the heart apex will be to the right and the stomach gas to the left. Do not be misled by left or right markers wrongly placed by a radiographer.

### Systematic film interpretation

#### Mediastinum

The trachea should lie in the midline. It may be deviated by a goitre or mediastinal mass. It is normally deviated a little to the left as it passes the aortic knuckle. (The aortic arch becomes wider and unfolded with age because of loss of elasticity.)

The mediastinum, including the trachea, can be deviated by a large pleural effusion, a tension pneumothorax or pulmonary collapse.

Rotation of the patient may make the mediastinum appear distorted.

#### Hila

The hila are mostly formed by the pulmonary arteries with the upper lobe veins superimposed. The left hilum is higher than the right. The left has a squarish shape whereas the right has a V shape.

A hilum can be more prominent if the patient is rotated. Lymphadenopathy or a large pulmonary artery will cause hilar enlargement.

#### Heart

The heart shape is ovoid with the apex pointing to the left. Characteristically, about two-thirds of the heart projects to the left of the spine.

The right heart border is formed by the outer border of the right atrium, and the left heart border by the left ventricle. The left margin of the right ventricle lies about a thumb's breadth in from the left heart border. (On the surface of the heart, this is marked by the left anterior descending coronary artery.)

The cardiothoracic diameter is a rather approximate way of determining whether the heart is enlarged. If the heart size is more than 50% of the transthoracic diameter, enlargement may be present. Apparent slight cardiac enlargement can occur because of a relatively small AP diameter of the chest. A cardiothoracic ratio at the upper limit of normal should not cause alarm if the patient has no reason to have cardiac failure and no symptoms of it.

Valve calcification, if present, is better seen on the lateral view. On the frontal view, the valve calcification cannot be visualised over the spine.

## Diaphragm

The hemidiaphragms visualised on the frontal films are the top of the domes seen tangentially. Much lung in the posterior costophrenic angles is not seen on the frontal film.

If the hemidiaphragms are low and flat, emphysema may be present. A critical look must be made beneath the diaphragm to see if there is free peritoneal gas (Figure 6.38, page 193).

## Lung fields

On the frontal field, it is convenient to divide the lung fields into zones. It is easy then to compare one zone with another for density differences and the distribution of the vascular 'markings'.

The apices lie above the level of the clavicles. The upper zones include the apices and pass down to the level of the second costal cartilages. The mid-zones lie between the second and fourth costal cartilage levels. The lower zones lie between the fourth and sixth costal cartilages.

The radiolucency of the lung fields is due to the air filling the lung. The 'greyness' is due to blood in the pulmonary vessels.

The upper zones of the lungs are normally less well perfused, resulting in smaller blood vessels. With raised left atrial pressure, there is upper zone blood diversion and the vessels are congested.

An increase in lung radiolucency occurs with pulmonary vessel loss, as also happens with emphysema. Lung radiolucency is lost with an effusion or consolidation.

Terms such as opacity, consolidation and patchy shadowing are used to describe the lung fields. It is usually unwise to attempt to make too precise a diagnosis of the underlying pathology.

The lungs are divided into lobes by reflections of the visceral pleura. The right lung is composed of the upper, middle and lower lobes. On the left, there are only the upper and lower lobes.

there are only the upper and lower lobes.

The right upper lobe has three segments: anterior, posterior and apical. The right middle lobe has a lateral and medial segment. Apical, medial basal, lateral basal, anterior basal and posterior basal segments compose the lower lobe.

There are three differences in the segmental anatomy of the left lung (Figure 5.15, page 137). The left upper lobe has four segments: an apicoposterior, anterior and two lingular segments. The superior and inferior lingular segments are the equivalent of the right middle lobe. The left lower lobe has four segments: it does not contain a medial basal segment.

The fissures are seen as hairline shadows. The horizontal fissure is at the level of the right fourth costal cartilage. The oblique fissures are not seen on the frontal view.

## Bones and soft tissue

Nipple shadows are often seen over the lower zones and are about 5 mm in diameter. They can be confused with a 'coin' lesion. In such a case, nipple markers may be helpful.

Look carefully for a missing breast shadow in a female patient. A mastectomy may provide a diagnostic clue to explain bony or pulmonary metastases, or upper zone postradiation fibrosis.

Soft tissue gas may accompany a pneumothorax or be present after a thoracotomy.

Calcified tuberculous glands in the neck should be looked for in patients with lung scarring or calcified hilar lymph nodes.

Check that there are no rib fractures or space-occupying lesions. Look for rib notching, due to increased blood flow through intercostal vessels (e.g. coarctation of the aorta). Cervical ribs or a thoracic scoliosis should be noted. Erosions or arthritis around the shoulder joints should be looked for.

## Review

Certain parts of the film should be double-checked if the radiograph appears normal.

The retrocardiac region should be looked at again. A collapsed left lower lobe will reveal itself as a triangular opacity behind the heart shadow.

Both apices should be rechecked for lesions, especially Pancoast tumours or tuberculosis.

Has the patient a pneumothorax? There will be a difference between the translucency of the two lungs.

## Lateral film

The lateral view is used largely for localisation of an already visible lesion on the frontal film. Examine it just as carefully. Sometimes a lesion is seen only on the lateral view. If there is clinical evidence of heart or lung disease, frontal and lateral views should always be obtained.

• **Points to remember:** (i) the retrosternal and retrocardiac triangles are normally of a similar radiodensity; (ii) the thoracic vertebrae become less opaque lower down the spine, unless there is pulmonary or pleural disease; (iii) the posterior costophrenic angle is sharp unless there is fluid or adjacent consolidation.

The hemidiaphragms are well defined unless there is pleural or pulmonary disease.

The oblique fissure placement is '4 to 4'. It passes from approximately 4 cm behind the anterior costophrenic angle, through the hilum to the T4 vertebral body level.

## Heart

The right ventricle forms the anterior heart border on the lateral film. The left atrium forms the upper posterior border.

Mitral valve calcification is seen below an imaginary line drawn from the anterior costophrenic angle to the hilum, whereas aortic valve calcification lies above this line.

## Examples of chest X-rays in cardiac disease

The radiological changes seen in pulmonary venous congestion, interstitial pulmonary oedema and alveolar pulmonary oedema are shown in Figures 4.59 to 4.61, respectively. Mitral valve disease is shown in Figure 4.62, while a ventricular aneurysm is seen in Figure 4.63. The characteristic notching of the inferior aspects of the ribs, due to hypertrophy of the intercostal arteries, appears in Figure 4.64, while the pulmonary plethora that is characteristic of a left-to-right shunt is obvious in Figure 4.65. A prosthetic aortic valve, which was inserted when a regurgitant valve was replaced in a patient with Marfan's syndrome, is illustrated in Figure 4.66; and a pacemaker and defibrillators are shown in Figure 4.67.

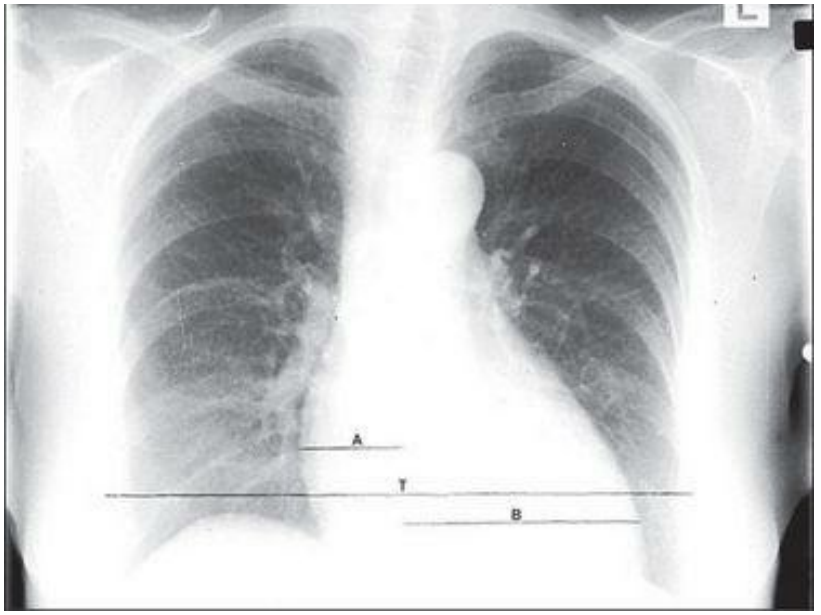



**Figure 4.59** Pulmonary venous congestion
The heart is enlarged due to failure. This failure is not severe enough to cause pulmonary oedema. However, the increased pulmonary venous pressure has caused upper zone blood diversion so the vessels above the hilum appear wider than those below. (The mechanism of the blood diversion is not fully understood.) These changes are seen when the pulmonary venous pressure is about 15 to 20 mmHg.
The cardiothoracic ratio A + B is a useful indicator of cardiac enlargement if it is greater than 50%. The thoracic measurement (T) is the widest diameter above the costophrenic angles, usually at the level of the right hemidiaphragm. The cardiac diameter is the addition of the two widths A and B.

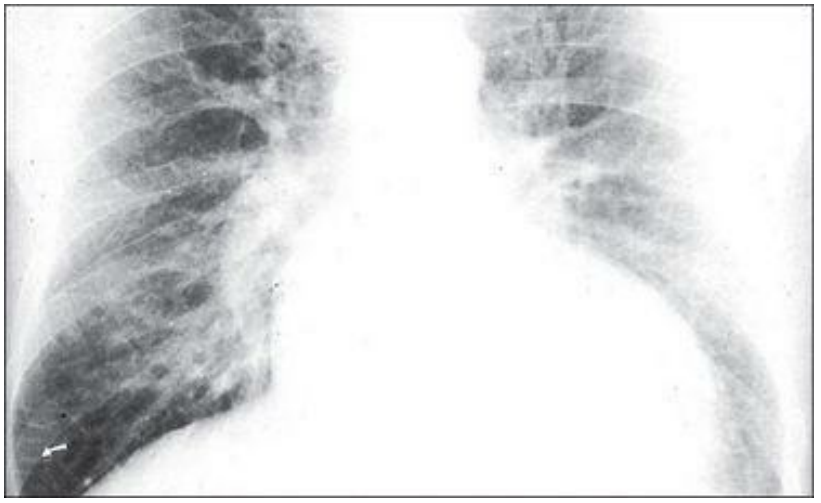


**Figure 4.60** Interstitial pulmonary oedema
The heart is moderately enlarged. The interstitial oedema causes fine, diffuse shadowing in the lung fields with blurring of the vessel margins. The escape of fluid into the interstitial tissue occurs when the capillary pressure exceeds the plasma osmotic pressure of 25 mmHg.
The interstitial oedema is characterised by Kerley 'B' lines, which are oedematous interlobular septa. They are best seen peripherally in the right costophrenic angle (arrow),

where they lie horizontally, and are about 1 cm long. They contain the engorged lymphatics, which were originally thought by Kerley to be the sole cause of the 'B' lines.
Sternal sutures are present from previous cardiac surgery.

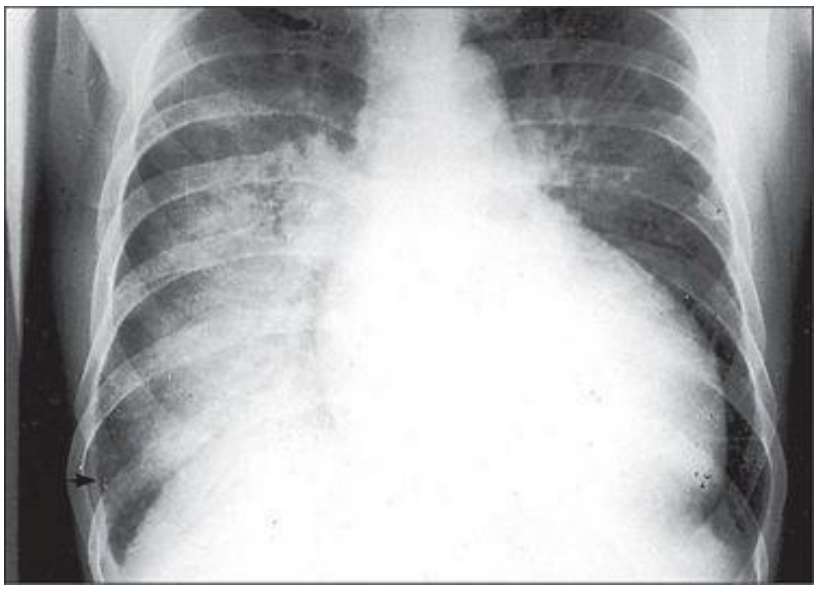


**Figure 4.61** Alveolar pulmonary oedema
When the pulmonary venous pressure reaches 30 mmHg, oedema fluid will pass into the alveoli. This causes shadowing (patchy to confluent depending on the extent) in the lung fields. This usually occurs first around the hila and gives a bat's wing appearance. These changes are usually superimposed on the interstitial oedema.
A lamellar pleural effusion (arrow) is seen at the right costophrenic angle where Kerley 'B' lines are also evident.

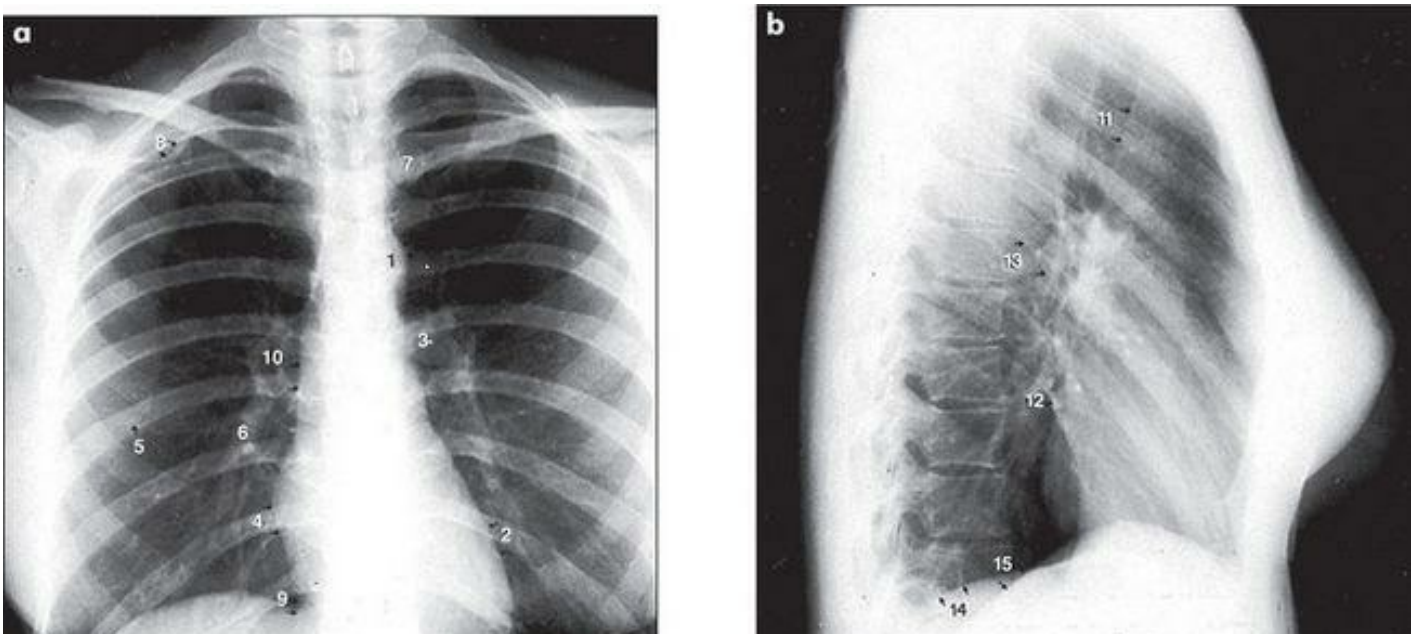


**Figure 4.62** Mitral valve disease
The left atrium enlarges because of the pressure and volume load. It bulges posteriorly and to both sides (arrows). The atrial appendage bulges out below the left hilum. The prominent right border of the atrium causes the 'double right heart border' appearance.
To distinguish the valves if calcification is present, draw imaginary lines. On the PA view (a) the line passes from the right cardiophrenic angle to the inferior aspect of the left hilum. The line on the lateral view (b) passes from the antero-inferior angle through the midpoint of the hilum. The aortic valve lies above this line whereas the mitral valve lies below it.

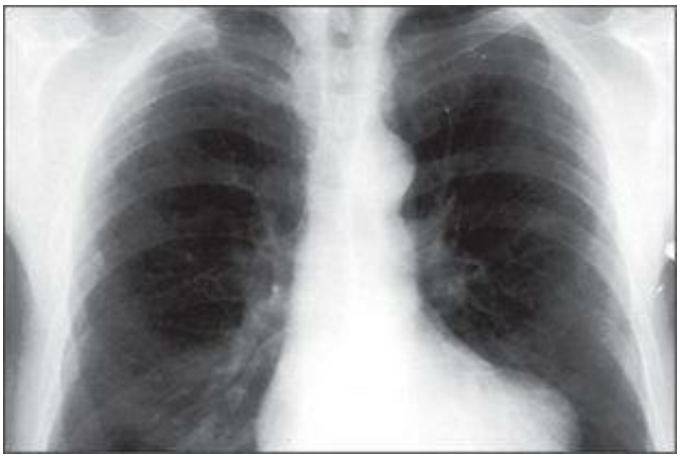
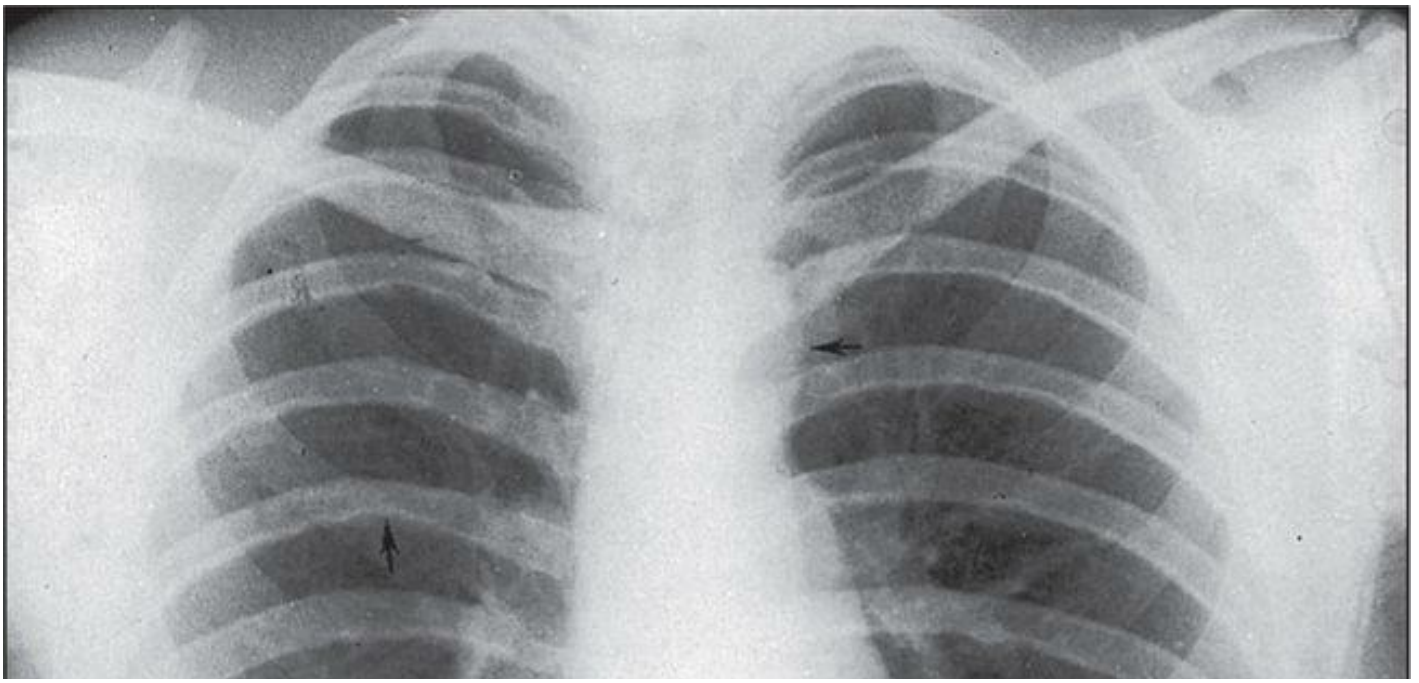


**Figure 4.63** Ventricular aneurysm
There is a bulge of the left cardiac border (arrow), which indicates an aneurysm of the left ventricular wall. The most common cause is weakness following myocardial infarction.



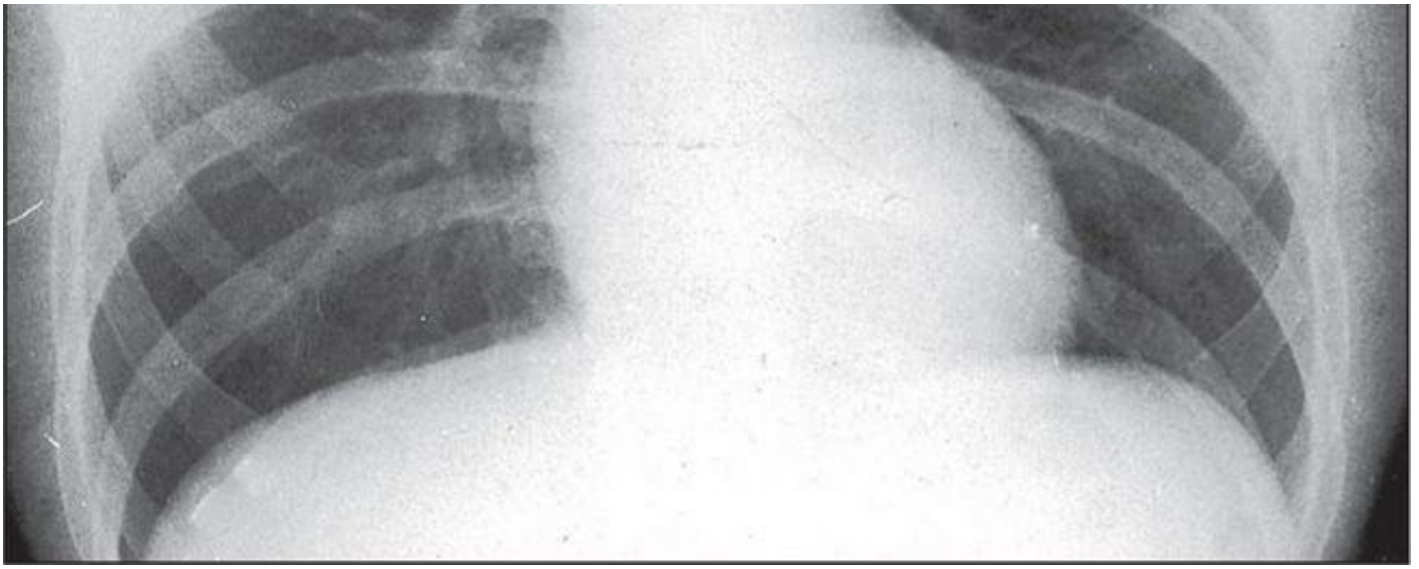


**Figure 4.64** Aortic coarctation

The classical sign in aortic coarctation is notching of the inferior aspects of the ribs (arrow on left). This is due to hypertrophy of the intercostal arteries in which retrograde flow from the axillary collaterals is taking blood back to the descending aorta.

Because of the increased resistance to the left heart flow, left ventricular hypertrophy and then failure can occur. Failure causing cardiac enlargement has not yet occurred in this patient. The arrow on the right indicates a smaller than normal aortic knuckle.

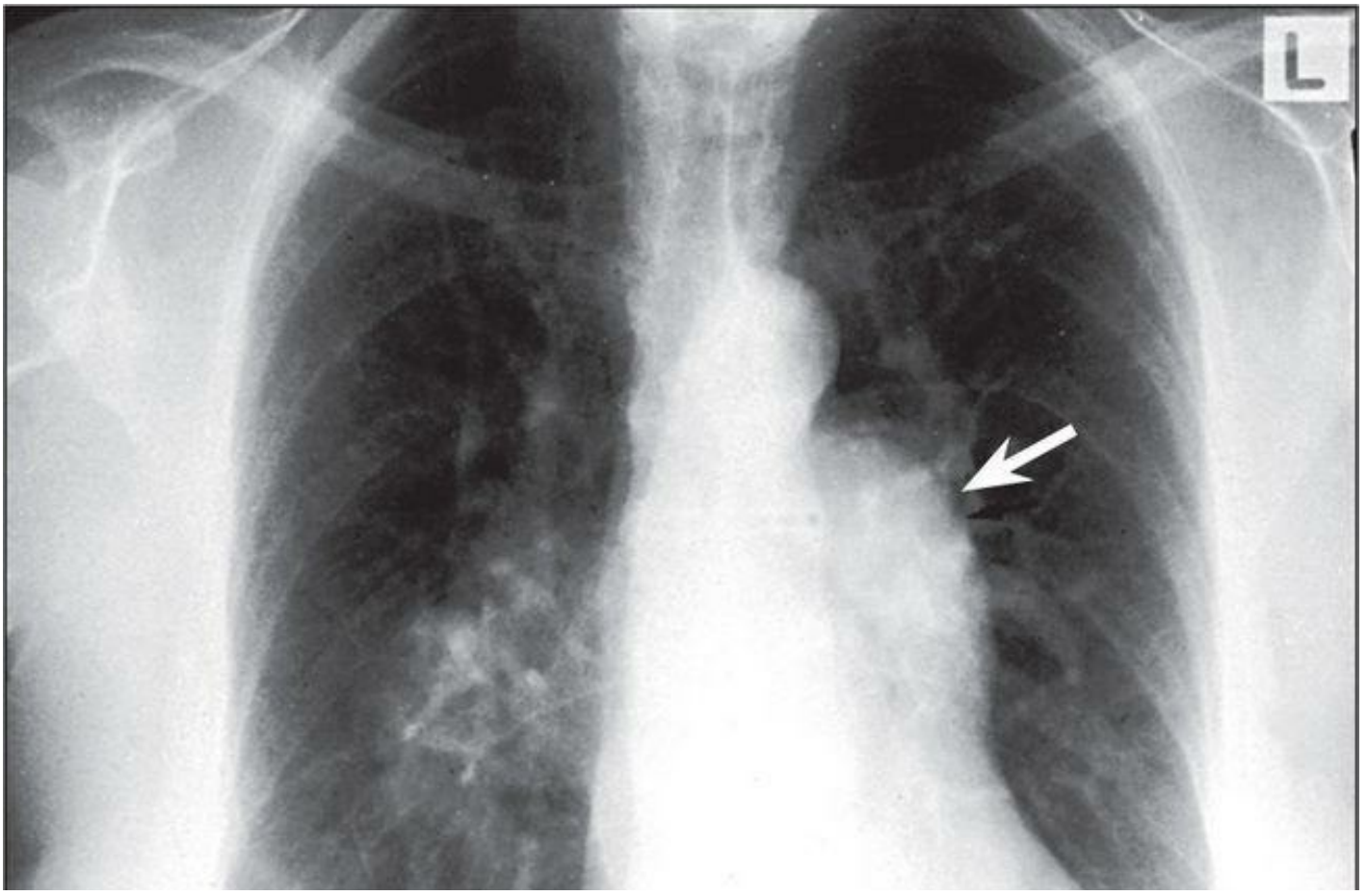


**Figure 4.65** Atrial septal defect (ASD)

The most important thing to recognise is that there is pulmonary plethora indicating a left-to-right shunt. Left-to-right shunts occur in ASD, ventricular septal defect (VSD) and patent ductus arteriosus (PDA).

The shunted flow causes enlargement of the main pulmonary artery and its branches. The right hilum is enlarged because of the very dilated right pulmonary artery. The left hilum is hidden by the very dilated main pulmonary artery (arrow).

The ascending aorta is small (in contrast to its enlargement in PDA). The left atrium and ventricle are not enlarged, as they are in VSD and PDA.

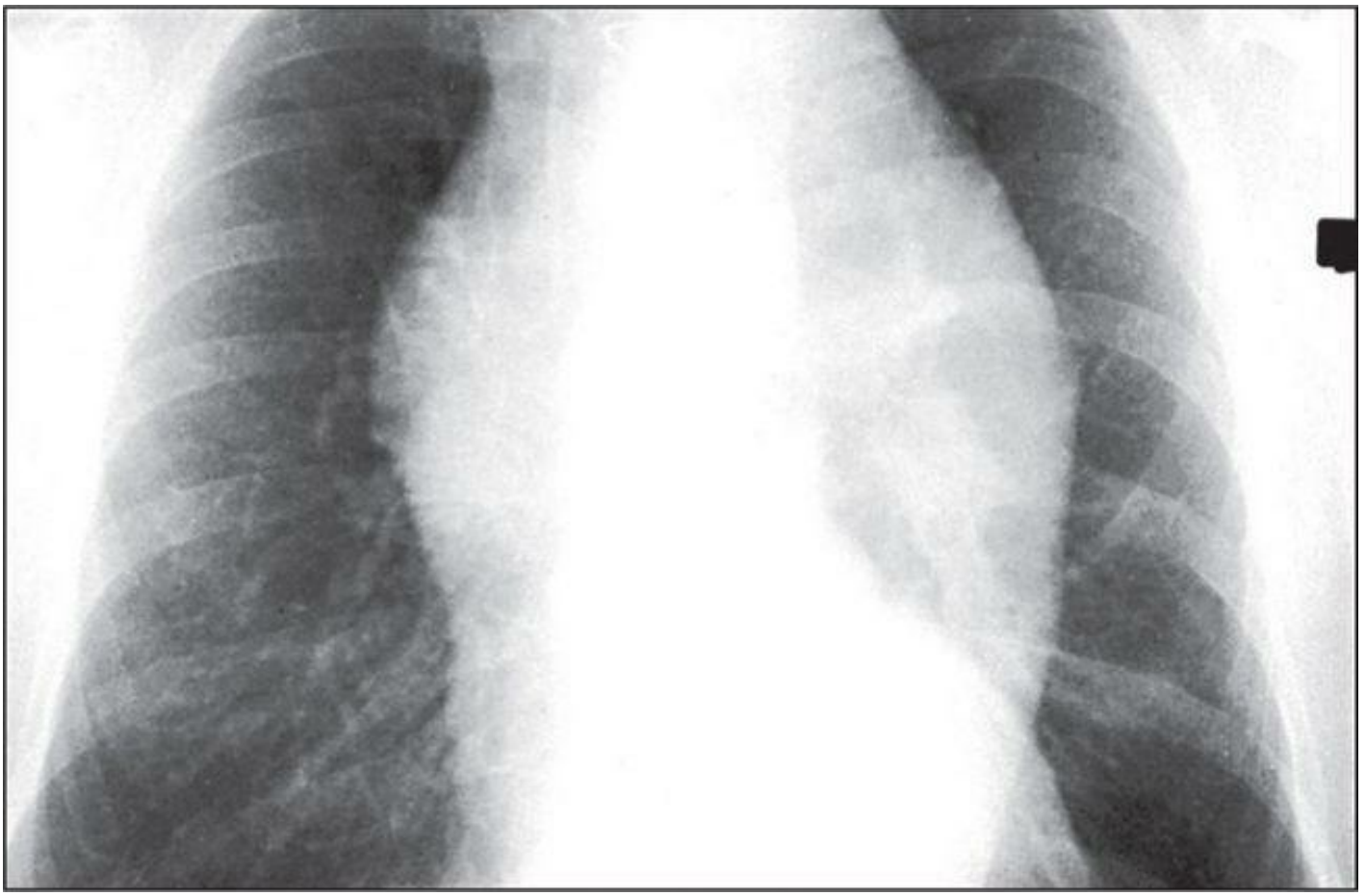


**Figure 4.66** Marfan's syndrome

The mediastinum is widened by uniform dilatation of the ascending aorta, the aortic arch and the descending aorta. This patient had Marfan's syndrome. Dissecting aneurysms can also occur and have a similar appearance.


**Figure 4.67** Pacemaker and defibrillators

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

## Summary

### The cardiovascular examination: a suggested method (Figure 4.68)

**Position** the patient at 45 degrees and make sure his or her chest and neck are fully exposed. Cover the breasts of a female patient with a towel or loose garment.

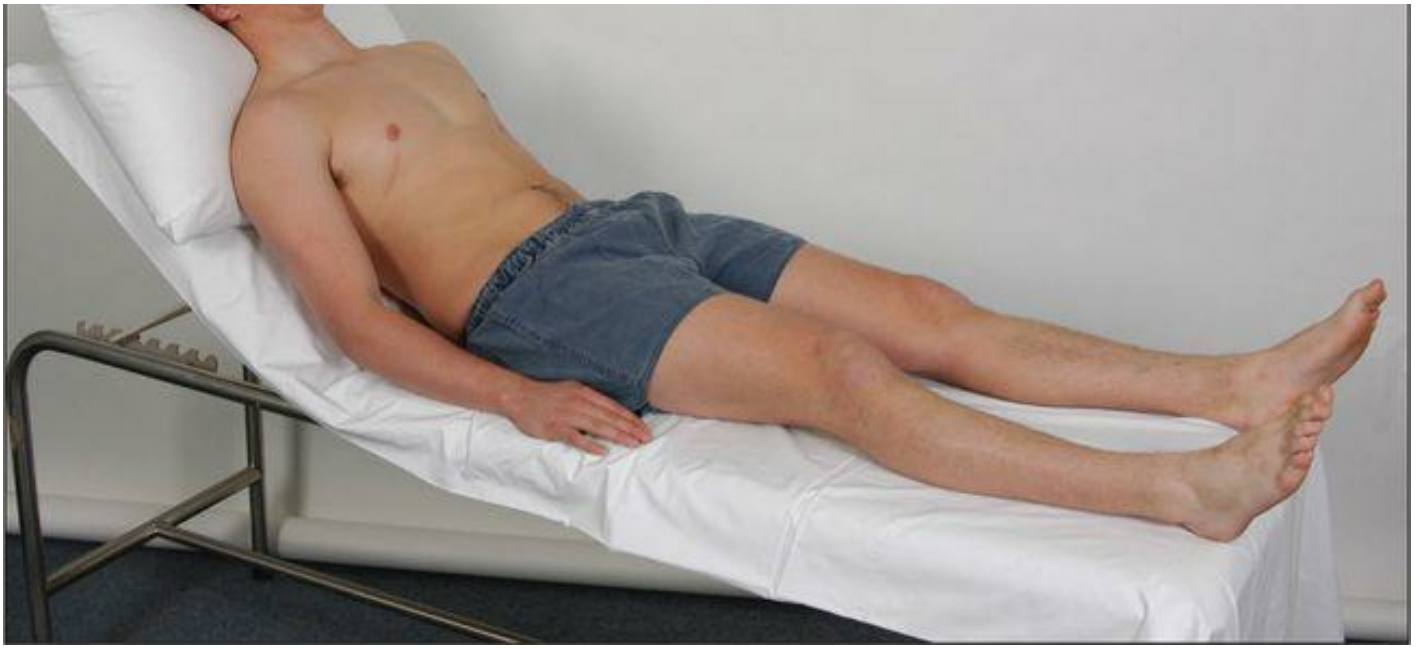


**Figure 4.68** Cardiovascular system
Patient lying at 45 degrees

1. **General inspection**
   Marfan's, Turner's, Down syndrome
   Rheumatological disorders, e.g. ankylosing spondylitis (aortic regurgitation)
   Acromegaly etc
   Dyspnoea

2. **Hands**
   Radial pulses—right and left
   Radiofemoral delay
   Clubbing
   Signs of infective endocarditis—splinter haemorrhages etc
   Peripheral cyanosis
   Xanthomata

3. **Blood pressure**

4. **Face**
   Eyes
   - Sclerae—pallor, jaundice
   - Pupils—Argyll Robertson (aortic regurgitation)
   - Xanthelasma

   Malar flush (mitral stenosis, pulmonary stenosis)
   Mouth
   - Cyanosisc
   - Palate (high arched—Marfan's)

- Dentition

5. **Neck**
   Jugular venous pressure
   - Central venous pressure height
   - Wave form (especially large *n* waves)

   Carotids—pulse character

6. **Praecordium**
   Inspect
   - Scars—whole chest, back
   - Deformity
   - Apex beat—position, character
   - Abnormal pulsations

   Palpate
   - Apex beat—position, character
   - Thrills
   - Abnormal impulses

   NB: Beware of dextrocardia

7. **Auscultate**
   Heart sounds
   Murmurs
   Position patient
   - Left lateral position
   - Sitting forward (forced expiratory apnoea)

   NB: Palpate for thrills again after positioning
   Dynamic auscultation
   - Respiratory phases
   - Valsalva
   - Exercise (isometric, e.g. hand grip)
   - Carotids

8. **Back (sitting forward)**
   Scars, deformity
   Sacral oedema
   Pleural effusion (percuss)
   Left ventricular failure (auscultate)

9. **Abdomen (lying flat—1 pillow only)**
   Palpate liver (pulsatile etc.), spleen, aorta
   Percuss for ascites (right heart failure)
   Femoral arteries—palpate, auscultate

10. **Legs**
    Peripheral pulses
    Cyanosis, cold limbs, trophic changes, ulceration (peripheral vascular disease)
    Oedema
    Xanthomata
    Calf tenderness

11. **Other**
    Urine analysis (infective endocarditis)
    Fundi (endocarditis)
    Temperature chart (endocarditis)

**Inspect** while standing back for the appearance of Marfan's, Turner's or Down syndromes. Also look for dyspnoea, cyanosis, jaundice and cachexia.

Pick up the patient's **hand**. Feel the radial pulse. At the same time inspect the hands for clubbing. Also look for the peripheral stigmata of infective endocarditis: splinter haemorrhages are common (and are also caused by trauma), while Osler's nodes and Janeway lesions are rare. Look quickly, but carefully, at each nail bed, otherwise it is easy to miss these signs. Note any tendon xanthoma (type II hyperlipidaemia).

The **pulse** at the wrist should be timed for rate and rhythm. Feel for radiofemoral delay (which occurs in coarctation of the aorta) and radial–radial inequality. Pulse character is best assessed at the carotids.

Take the **blood pressure** (lying and standing or sitting—postural hypotension).

Next inspect the **face**. Look at the eyes briefly for jaundice (e.g. valve haemolysis) or xanthelasma (type II or type III hyperlipidaemia). You may also notice the classical mitral facies. Then inspect the mouth using a torch for a high arched palate (Marfan's syndrome), petechiae and the state of dentition (endocarditis). Look at the tongue or lips for central cyanosis.

The **neck** is very important. The jugular venous pressure (JVP) must be assessed for height and character. Use the right internal jugular vein for this assessment. Look for a change with inspiration (Kussmaul's sign). Now feel each carotid pulse separately. Assess the pulse character.

Proceed to the **praecordium**. Always begin by inspecting for scars, deformity, site of the apex beat and visible pulsations. Do not forget about pacemaker boxes. Mitral valvotomy scars (usually under the left breast) can be quite lateral and very easily missed.

Palpate for the position of the **apex beat**. Count down the correct

number of interspaces. The normal position is the fifth left intercostal space, one centimetre medial to the midclavicular line. The character of the apex beat is important. There are a number of types. A *pressure-loaded* (hyperdynamic, systolic overloaded) apex beat is a forceful and sustained impulse that is not displaced (e.g. aortic stenosis, hypertension). A *volume-loaded* (hyperkinetic, diastolic overloaded) apex beat is a forceful but unsustained impulse that is displaced down and laterally (e.g. aortic regurgitation, mitral regurgitation). A dyskinetic apex beat (cardiac failure) is palpable over a larger area than normal and moves in an uncoordinated way under the examiner's hand. Do not miss the tapping apex beat of mitral stenosis (a palpable first heart sound). The double or triple apical impulse of hypertrophic cardiomyopathy is very important too. Feel also for an apical thrill, and time it.

Then palpate with the heel of your hand for a left parasternal impulse (which indicates right ventricular enlargement or left atrial enlargement) and for thrills. Now feel at the base of the heart for a palpable pulmonary component of the second heart sound (P2) and aortic thrills. Percussion may be helpful if there is uncertainty about cardiac enlargement.

**Auscultation** begins in the mitral area with both the bell and the diaphragm. Listen for each component of the cardiac cycle separately. Identify the first and second heart sounds, and decide if they are of normal intensity and whether the second heart sound is normally split. Now listen for extra heart sounds and for murmurs. Do not be satisfied at having identified one abnormality.

Repeat the approach at the left sternal edge and then the base of the heart (aortic and pulmonary areas). Time each part of the cycle with the carotid pulse. Listen over the carotids.

It is now time to **reposition** the patient. First put him or her in the left lateral position. Again feel the apex beat for character (particularly tapping) and auscultate. Sit the patient up and palpate for thrills (with the patient in full expiration) at the left sternal edge and base. Then listen in those areas, particularly for aortic regurgitation or a pericardial rub.

Dynamic auscultation should always be done if there is any doubt about the diagnosis. The Valsalva manoeuvre should be performed whenever there is a pure systolic murmur. Hypertrophic cardiomyopathy is easily missed otherwise.

The patient is now sitting up. Percuss the **back** quickly to exclude a pleural effusion (e.g. due to left ventricular failure), and auscultate for inspiratory crackles (left ventricular failure). If there is a radiofemoral delay, also listen for a coarctation murmur over the back. Feel for sacral oedema and note any back deformity (e.g. ankylosing spondylitis with aortic regurgitation).

Next lay the patient flat and examine the **abdomen** properly for

Next lay the patient flat and examine the **abdomen** properly for hepatomegaly (right ventricular failure) and a pulsatile liver (tricuspid regurgitation). Test for the abdomino-jugular reflux sign if relevant. Feel for splenomegaly (endocarditis) and an aortic aneurysm.

Move on to the **legs**. Palpate both femoral arteries and auscultate here for bruits. Go on and examine all the peripheral pulses. Look for signs of peripheral vascular disease, peripheral oedema, clubbing of the toes, Achilles tendon xanthomata and stigmata of infective endocarditis.

Finally, examine the **fundi** (for hypertensive changes, and Roth's spots in endocarditis) and the **urine** (haematuria in endocarditis). Take the **temperature**.

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