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Cardiovascular investigations

Plain films

Evaluate heart size and chamber enlargement. On a standard PA chest projection, the ratio of the cardiac diameter to that of the maximum internal diameter of the chest should be no greater than 50% on a full inspiratory film. Expiratory films may falsely give the impression of cardiomegaly and pulmonary congestion. Supine films may also give a similar appearance.

Ultrasound

Echocardiography and Doppler examination reveal anatomical abnormalities as well as flow disturbances and assist in the study of incompetent and stenotic valves and ventricular function; aortic arch aneurysms, dissecting aneurysms, cardiomyopathy and pericardial effusions can also be diagnosed using echocardiography.

Figure 3.1 Ultrasound of the heart showing all four chambers.
Isotope scanning

Technetium-99m pyrophosphate accumulates in damaged myocardium whereas thallium-201 produces a deficient uptake in territories supplied by occluded or narrowed arteries. Thallium is most commonly used as a screening technique in patients with suspected coronary artery disease.

Computed tomography (CT)

Relevant applications include the further evaluation and diagnosis of dissecting thoracic aneurysms, pericardial effusions and myocardial tumours. A recent non-invasive application of CT calculates the total calcium deposition in the coronary arteries giving a predictive value of coronary artery disease.

Figure 3.2 CT abdomen. Leaking abdominal aortic aneurysm with peri aortic haematoma (arrow).

Figure 3.3 Dissection of the descending aorta. Note the intimal flap in the contrast-filled aorta (arrow) and blood in the mediastinum.
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Magnetic resonance imaging (MRI)

MRI can be gated to the cardiac cycle to reduce motion artefact. It can examine the heart in any plane and is of value in many clinical situations including pericardial effusions, hypertrophic cardiomyopathy, and congenital and valvular heart disease. Magnetic resonance angiography (MRA) has the capability of providing a non-invasive method of imaging many vascular abnormalities such as aneurysm, dissection, stenoses, occlusions and congenital anomalies.

Venography

The venous system can be studied by proximal contrast injection. The most common indications are injection into:
- a foot vein to look at the lower limb venous system;
- antecubital vein to assess the axillary and subclavian vein and the superior vena cava;
- femoral vein to study the inferior vena cava.

Figure 3.4 Right-upper limb venography showing thrombus in the axillary and subclavian veins (arrows) with a large collateral circulation.
Arteriography

Vascular access is usually obtained using a percutaneous approach via the femoral artery. Any major vessel or blood supply to an organ can be studied by selective arterial cannulation with contrast injection. Radial, brachial, axillary or popliteal arteries can also be punctured percutaneously, if femoral artery access is unsuitable. Anatomical detail is excellent; haematoma, haemorrhage and arterial thrombus are recognized rare local complications.

Coronary angiography is a commonly performed examination. In this study contrast is initially injected into the left ventricle to evaluate function, and subsequently into the left and right main coronary arteries to detect the extent of any stenoses; angioplasty and stent insertion may be carried out on suitable stenoses.

Figure 3.5 Left subclavian angiogram: short occlusion of the axillary artery (arrows).

Figure 3.6 Normal right coronary angiogram.

Figure 3.7 Normal left coronary angiogram.

Figure 3.8 Aortogram: lower abdominal aortic aneurysm (arrows).
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Figure 3.9  Cardiomegaly the heart size is measured by comparing the cardiac diameter ($a + b$) to the maximum internal diameter of the chest.

Figure 3.10  Pericardial effusion: large globular shaped cardiac shadow with clear lung fields.
Cardiomegaly

On a standard PA chest, the heart size can be expressed as the cardiothoracic ratio. Generally, a ratio of over 50% of the heart size to the maximum internal diameter of the chest indicates cardiac enlargement. This measurement is only an approximate guide, and is helpful for serial measurement. Specific chamber enlargement is often difficult to identify, but a plain chest film may show the following:

- **Left atrium:** the only chamber that can be reliably diagnosed when it is enlarged; it may feature a double contour to the right heart border, splaying of the carina with upward displacement of the left main bronchus or posterior bulging the chamber on a lateral chest X-ray.
- **Right atrium:** prominence of the right heart border.
- **Right ventricle:** upward displacement of the cardiac apex with anterior enlargement of the heart border on a lateral projection.
- **Left ventricle:** increased convexity of left heart border.

Echocardiography is more accurate in the assessment of specific chamber and cardiac size.

Pericardial effusion

A pericardial effusion is a collection of fluid in the pericardial sac, the fluid being either serous, blood or lymphatic in origin.

Radiological features

- **Chest film:** illustrates a symmetrically enlarged and globular cardiac shadow only when there is a significant effusion (>250ml). Pericardial effusion should be suspected if there has been a rapid serial increase in the cardiac shadow, with normal pulmonary vasculature.
- **Echocardiography:** the investigation of choice. Effusions are visible as echo-free areas surrounding the heart.
- **CT:** may also identify the aetiology, e.g. mediastinal malignancy.
- **MRI:** accurate for diagnosis and also images the chest and mediastinum.

Causes

Infective (viral, bacterial, tuberculous); uraemia; postmyocardial infarction (Dressler’s syndrome); myxoedema; malignancy: bronchial and mediastinal tumours with pericardial invasion; collagen vascular diseases (systemic lupus erythematosus (SLE), rheumatoid arthritis).
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Figure 3.11  Cardiac failure: interstitial pulmonary oedema. Note fluid in the right horizontal fissure.

Figure 3.12  Alveolar pulmonary oedema; fluid accumulating predominantly in the perihilar region; left pleural effusion.
Cardiac failure

Cardiac failure is said to be present when tissue demands cannot be adequately supplied by the heart. It is usually due to low output from ischaemic heart disease but, paradoxically, may rarely result from high output as a consequence of excessive tissue needs in conditions such as thyrotoxicosis or Paget’s disease.

Radiological features

On a chest X-ray the following may be seen:

- Cardiac enlargement.
- Upper-lobe vascular prominence: from raised pulmonary venous pressure.
- Pleural effusions: seen as blunting at the costophrenic angles, but as the effusions become larger, there is a homogeneous basal opacity with a concave upper border.
- Interstitial pulmonary oedema: initially, prominence of the upper-lobe and narrowing of the lower-lobe vessels. As venous pressure rises, interstitial oedema develops and fluid accumulates in the interlobular areas with peripheral septal lines (Kerley ‘B’ lines).
- Alveolar pulmonary oedema. With further increases in venous pressure, fluid transgresses into the alveolar spaces (alveolar shadowing) with haziness and blurring in the perihilar regions; in severe cases, pulmonary oedema develops throughout both lung fields. The outer thirds of the lungs may be spared, the bilateral central oedema being described as ‘bat’s wing’.

Figure 3.13  Manifestations of cardiac failure.
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Figure 3.14 Doppler examination of the femoral vein with a normal Doppler signal and blood flow.

Figure 3.15 Lower limb venogram: recent thrombus in the femoral vein seen as a filling defect with contrast around the thrombus (arrows). The contrast filled common femoral and iliac vein appear normal.
Deep-vein thrombosis

Thrombus formation in the deep veins of the calf is a common clinical problem. Predisposing causes include recent surgery, contraceptive use, prolonged bed rest, neoplastic disease and hypercoagulability states.

Presentation

- Calf swelling.
- Calf pain.
- Pulmonary embolus may be the first sign, the calves being asymptomatic.

Radiological investigations

- Colour Doppler ultrasound.
- Venography.

Radiological features

Colour Doppler ultrasound is the initial investigation of choice but difficulty may be encountered in visualizing calf veins especially in obese patients. If there is a strong clinical suspicion, in the presence of a normal Doppler examination, venography is suggested.

- Colour Doppler ultrasound. Accurately images vascular flow patterns and the presence of thrombus in the lower limbs. Blood clot may be seen within the vein lumen, often accompanied by a reduction in blood flow.
- Venography. Contrast is injected into a foot vein to visualize the lower-limb circulation. Tourniquets applied at the ankle and above the knee force the contrast into the deep veins, thrombus being visualized as filling defects in the vein lumen. Extensive thrombus formation may lead to poor or complete lack of contrast filling of the veins.

Complications

- Pulmonary embolus.
- Postphlebitic syndrome.

Treatment

- Heparin.
- Anticoagulants.
- Vena cava filter insertion in recurrent pulmonary embolization. Introduced percutaneously via the femoral or internal jugular vein and positioned in the inferior vena cava, just below the renal veins.
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Figure 3.16 Ventilation and perfusion isotope scans in both the frontal and oblique projections showing mismatched defects suggesting pulmonary emboli.

Figure 3.17 Selective right pulmonary angiogram: thrombus in the right main pulmonary artery (arrows) with poor distal perfusion.
Pulmonary embolus

Pulmonary embolism

Pulmonary embolism occurs when a blood clot detaches from the peripheral venous system and lodges in the pulmonary artery or its branches.

Pulmonary infarction

Pulmonary infarction is the lesion that develops secondary to pulmonary embolus. Predisposing causes include prolonged bed rest, recent surgery, pregnancy, hypercoagulable states and lower limb deep-vein thrombosis.

Radiological features

The blood clot usually originates from the pelvic or lower-limb veins and migrates into the pulmonary circulation. The chest X-ray is normal in the majority of cases, but if pulmonary infarction develops as a consequence of embolus, any of the following may be seen:

- raised diaphragm;
- small pleural effusions;
- basal collapse or plate-like atelectasis;
- consolidation, often segmental, peripherally situated and wedge-shaped.
- Isotope scan. Pulmonary embolus results in a segmental defect in perfusion with preserved ventilation (ventilation/perfusion mismatch).
- CT pulmonary arteriography (CTPA). A rapid series of scans are taken through the lungs after intravenous injection of a large bolus of contrast; emboli are seen as filling defects in the contrast column. This is proving to be a highly accurate technique and is now often employed as the first line of investigation for a suspected pulmonary embolus.
- Pulmonary angiography. This is an invasive investigation; direct contrast injection into the pulmonary arteries reveals blood clot as intraluminal filling defects with obstruction and attenuation of the pulmonary arterial branches. Infusion of thrombolytics through the catheter may lyse the clot.

Complications

Pulmonary hypertension: resolves in the acute stage when thrombi disintegrate. However, it may persist with recurrent embolization.

Types of embolism

- Fat embolism. Usually seen after severe skeletal trauma with fat globules entering the circulation and obstructing pulmonary vessels.
- Septic embolism. Arising from tricuspid endocarditis or infected material from central venous pressure (CVP) lines, pacing wires, etc.
- Amniotic fluid embolism. Commonest cause of postpartum maternal death. Amniotic debris may gain access to the maternal circulation with subsequent embolization.
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Figure 3.18 Plain abdominal film showing curvilinear calcification (arrows) in a large abdominal aortic aneurysm.

Figure 3.19 CT abdomen after contrast shows filling of the lumen (↑) and thrombus in an aneurysm (↓).

Figure 3.20 Transverse ultrasound of the abdomen showing the lumen (black) surrounded by thrombus (arrows).
Abdominal aortic aneurysm

An ‘aneurysm’ refers to a localized dilatation of the vessel wall. Aneurysms may arise in any part of the aorta, but are most frequently seen in the abdominal aorta below the level of the renal arteries. Degenerative vascular disease with subsequent weakening of the vessel wall is the usual cause.

Presentation

Asymptomatic finding; abdominal pain or back pain from vertebral erosion; pulsatile abdominal mass; acute abdomen.

Radiological features

- **Plain abdominal films** may show curvilinear calcification in the wall of an aneurysm, especially when due to atherosclerosis. Calcification is more clearly visualized on a lateral abdominal film.
- **Ultrasound** is the best initial investigation to determine the presence of an aneurysm, measurement of its diameter and to assess subsequent progress. An increased threat of rupture exists with those >6 cm in diameter and elective surgery is recommended.
- **CT/MRI** are both useful to localize the exact site of an aneurysm; assessment of renal artery involvement is essential to determine the type of operative approach.
- **Arteriography**: abdominal aneurysms may not necessarily show a widened lumen as the majority contain thrombus. Arteriography will demonstrate the distal circulation and relation of the renal arteries to the aneurysm.

Types of aneurysm

- Traumatic.
- Congenital: most commonly affects the intracranial circulation in the region of the circle of Willis (‘berry aneurysm’).
- Inflammatory: infection or abscess around the aorta leads to weakening of the wall.
- Dissecting: usually due to a tear in a weakened intimal wall in the thoracic aorta; predisposing factors include hypertension and Marfan’s syndrome. Retrograde dissection can involve the coronary arteries, aortic valve and the pericardial sac. CT or MRI may detect an intimal flap separating the two lumina, MRI being the more sensitive investigation.
- Degenerative: commonest sites are the abdominal aorta, iliacs, femorals and popliteals.
- Poststenotic: distal to arterial narrowing, such as coarctation.
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Figure 3.21  Arteriogram: occluded segment (arrows) in the right femoral artery.

Figure 3.22  Femoral embolus: sharp contrast cut off in the left femoral artery (arrow) due to embolus. Note the poor collateral circulation.
Peripheral vascular disease

Arterial insufficiency commonly develops in the lower limbs from atheromatous involvement of the aorta and lower-limb arteries. Pain in the calves or buttocks on exercise (intermittent claudication), cold limbs and ulceration are the commonest clinical features. Predisposing causes include diabetes and smoking.

Radiological investigations

Doppler ultrasound; arteriography; MRI.

Radiological features

Ultrasound will diagnose major occlusions but arteriography is required for the accurate visualization of diseased vessels, stenoses and occlusions; resolution of MR angiography has recently shown such significant improvements that it may eventually replace conventional arteriography.

Treatment

- Balloon angioplasty.
- Metallic stent insertion under radiological control.
- Surgical bypass grafts: aorto-iliac, femoropopliteal and femorofemoral.

Arterial embolus

An arterial embolus occurs when blood clot, originating elsewhere in the cardiovascular system, travels more peripherally and occludes an artery. The lower limbs are affected in the majority of cases. Symptoms are of rapid onset and consist of a cold, pale, numb leg with absent pulses distal to the occlusion. Predisposing factors include recent myocardial infarction with mural thrombus and atrial fibrillation. If there is co-existing vascular disease, a diagnosis of acute thrombosis should be considered.

Radiological features

Peripheral arteriography demonstrates the contrast column in the artery with a sharp, well-defined cut-off point, usually a convex upper border projecting into the lumen of the vessel. Further evidence of an acute episode is provided by a deficient collateral circulation.

Treatment

- Surgical embolectomy.
- Thrombolysis: perfusion of streptokinase or tissue plasminogen activator (TPA) directly into the arterial thrombus in order to lyse the clot.
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**Figure 3.23** Pulmonary hypertension: bilateral hilar vascular enlargement and prominence of the pulmonary outflow tract.

**Figure 3.24** Venogram with contrast injection in the right femoral vein (➜) showing a complete occlusion of the inferior vena cava and a collateral circulation via a hypertrophied ascending lumbar vein (↩).
Pulmonary arterial hypertension

Pulmonary arterial hypertension refers to increased pulmonary artery pressure from its normal value of 25/10 mmHg to greater than 30/15 mmHg.

Radiological features

Hypertension has to be quite marked before changes on a chest X-ray.

- Cardiac enlargement with right ventricular hypertrophy.
- Dilatation of the pulmonary hilar vessels with distal attenuation.
- Distension of the main pulmonary artery with a bulge below the aortic knuckle.

Causes

- Increased pulmonary blood flow in congenital heart disease.
- Obstruction of the pulmonary circulation, e.g. pulmonary emboli, parenchymal lung disease.
- Secondary to pulmonary venous hypertension from left-heart failure or mitral stenosis.

Superior and inferior vena cava obstruction

The superior (SVC) and inferior vena cava (IVC) may obstruct from many causes resulting in distal venous distension.

Presentation

- SVC obstruction: facial and neck oedema, visible collateral veins.
- IVC obstruction: lower limb oedema, scrotal oedema.

Radiological investigations

- Doppler ultrasound: verifies decreased or lack of a blood flow pattern.
- CT/MRI: confirms occlusion and often identifies the cause.
- Venography: demonstrates anatomical detail, especially useful if stenting is to be considered.

Causes of SVC obstruction

- Neoplastic: bronchial carcinoma, lymphoma, radiotherapy.
- Benign: mediastinal disease due to tuberculosis, sarcoid.

Causes of IVC obstruction

- Tumour invasion from abdominal neoplasms, most commonly renal.
- Retroperitoneal fibrosis; radiotherapy.
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Figure 3.25  Pericardial calcification (arrow).

Figure 3.26  Enlarged heart resulting from mitral valvular disease showing valve calcification (arrow).
Cardiac calcification

Pericardial calcification

Usually follows pericarditis: tuberculosis, rheumatoid arthritis, pyogenic, viral or rheumatic fever; the aetiology may be unknown.

Myocardial calcification

Occurs typically at the apex of the left ventricle; the common causes are myocardial infarction and ventricular aneurysm.

Valve calcification

Calcification in the valves is common, but has to be quite extensive before being evident on plain films. Calcification usually means an element of stenosis, with the aortic and mitral valves most commonly affected. Causes include atheroma, rheumatic valvular disease and congenital bicuspid valve.

Aortic-wall calcification

May be present in atheroma, in the wall of an aneurysm or represent syphilitic aortitis (ascending aorta).

Figure 3.27  Aortic-wall calcification (arrows).
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Figure 3.28 Swan-Ganz catheter in the right pulmonary artery (arrow) and endotracheal tube in a patient after cardiac surgery (note ECG leads).

Figure 3.29 Tip of the central line in the right atrium (arrow).
Tube and catheter placement

Endotracheal tube
The tip should be positioned 5–7 cm above the carina. When sited too far, the endotracheal tube may advance into a main bronchus, causing collapse of the opposite lung.

Central line
Inserted via the jugular or subclavian veins into a large intrathoracic vein. For accurate measurement of right atrial pressure, the tip of the catheter must lie in a large intrathoracic vein such as the superior vena cava.

Swan-Ganz catheter
The catheter is inserted via the jugular, subclavian or femoral vein and manipulated through the right heart into either the right or left pulmonary artery. The end-diastolic left-ventricular pressure is estimated from a reading taken at the distal tip of the catheter.

Nasogastric tube
The radio-opaque tip should be visualized in the region of the stomach on plain films. An X-ray is usually necessary to ensure that the tube is not malpositioned, especially into the trachea or bronchus.

Pacing wire
Pacemaker leads are placed through the subclavian or internal jugular veins into the right side of the heart, with the tip implanted at the apex of the right ventricle, whereas dual-lead pacemakers have their ends positioned in the right atrium and ventricle.