CARDIOVASCULAR SYSTEM

MAIN CATALOGUE

CASE 179
The only information available for this man aged 20, who died in 1935, is that he was admitted with right lobar pneumonia and then developed infective endocarditis and septicaemia, an enlarged spleen and petechial haemorrhages in the skin. At post-mortem there was lobar pneumonia in the right lower lobe with red and grey hepatization and commencing organization. The spleen was enlarged (490gm) and there were several infarcts up to 5cm in diameter. There were infarcts in the kidneys and many petechial haemorrhages throughout the tissues and organs.
The specimen consists of the heart opened to display the mitral valve. There is a large crumbling friable vegetation on the under surface of the valve and on the chordae tendineae. The underlying valve leaflets appear normal and there is no evidence of previous chronic rheumatic valve disease. The left atrium is normal. The left ventricle is not dilated or hypertrophied.
Staphylococcus aureus was grown from the vegetations.
Diagnosis: Acute infective endocarditis

CASE 260
No clinical information is available.
The specimen consists of a portion of the spine with the aorta and oesophagus. There is a localised saccular aneurysm of the descending aorta over a distance of about 7cm commencing just distal to the left subclavian artery. The aneurysm projects backwards and is adherent to the vertebral column, displacing the oesophagus anteriorly. Its lining is atherosclerotic and it contains old pale thrombus.
Diagnosis: Aneurysm of the descending aorta. ?syphilitic, ?atherosclerotic

CASE 2709
The patient was a woman aged 50 who died in 1928. There had been breathlessness for 2 weeks before admission and clinical features of aortic regurgitation were present: a diastolic murmur, a collapsing pulse (BP 150/45) and cardiac enlargement. The Wassermann reaction was positive. She died suddenly. At post-mortem there was chronic venous congestion of the liver and spleen, a left hydrosalpinx and syphilitic aortitis.
The specimen consists of the heart and the proximal 15cm of aorta. There is advanced syphilitic aortitis with dilation of the ascending aorta and arch with moderate overlying atherosclerosis. One cusp of the aortic valve is bound firmly down to the aorta by scar tissue. The other cusps show some slight fibrous thickening of their free borders in keeping with the effects of regurgitation. There is scarring around the origin of the right coronary artery. The left ventricle is dilated.
Diagnosis: Syphilitic aortitis
Explain the relationship between the syphilitic aortitis, dilated aorta, aortic incompetence, dilated left ventricle, congestion of the liver and spleen, and clinical symptoms and signs.
In tertiary syphilis there is chronic inflammation and fibrosis of the vasa vasorum in the proximal aorta. The narrowed vessels don't supply sufficient blood to the aortic media, which atrophies and the vessel dilates. With dilation of the aortic root, the valve cusps do not close properly -> incompetence. Additional blood enters the LV via the incompetent aortic valve during diastole creating a murmur from turbulent flow and leading to LV dilation (as the LV stretches to pump additional blood - Frank-Starling mechanism) and an enlarged heart (displaced apex beat) on examination. The left heart failure leads to increased pulmonary venous pressure and pulmonary oedema causing breathlessness, and secondary right heart failure -> blood backing up in the liver and spleen -> congestion. A collapsing pulse is typical of aortic incompetence, the pulse collapses as blood rushes back into the ventricle in diastole.
In what stage of syphilis does the aortitis occur? Tertiary syphilis that develops many years after the initial infection.

CASE 3162
No clinical information is available for this old specimen.
The specimen consists of the heart opened to show dilatation and moderate hypertrophy of the left atrium. The mitral valve is scarred and its orifice narrowed by chronic rheumatic changes. There are possible recent vegetations around the margins of the stenosed mitral valve, suggesting infective endocarditis.
Diagnosis: Thickened mitral valve, probably rheumatic.

CASE 3236
The patient was a man aged 59 who had congestive cardiac failure consequent to stenosis and regurgitation of the aortic valve.
The specimen shows the dilated left ventricle with a thickened wall. There is marked scarring and calcification of the aortic valve with fusion of the cusps. The under surfaces of the valve show friable necrotizing vegetations.
Diagnosis: Subacute infective endocarditis superimposed on chronic rheumatic valve disease.
See also Common and Classic Diseases section

CASE 3347
The patient was a girl aged 18 who died in 1932. She was very pale, propped up in bed with a rapid regular pulse. Systolic and diastolic murmurs were audible in the mitral area and the liver was enlarged. There was a leucocytosis of 20,000/cu.mm.
The specimen consists of the heart opened from the back through both atria. The left atrium contains large masses of brown friable thrombotic vegetations extending upwards from the posterior leaflet of the mitral valve across the posterior wall. Culture of the vegetations at post-mortem yielded colonies of *Staphylococcus aureus* and haemolytic and non-haemolytic *streptococci*.
Diagnosis: Acute infective endocarditis

CASE 3411
The patient was a man aged 52 with acromegaly who died of congestive cardiac failure. At post-mortem the sella turcica was considerably enlarged and was filled with a soft pale adenoma 2.5cm in diameter.
The specimen shows a heart that is massively enlarged and globular and weighs 1200gm. On the back of the jar the tongue is greatly enlarged and the kidney is somewhat enlarged.
Diagnosis: Acromegaly with enlarged organs

CASE 3824
The patient was a woman aged 31 who was reported to have streptococcal septicaemia for some months. The condition allegedly arose from puerperal sepsis. She became much worse a few days before her admission to hospital and there was obvious suppuration in the region of the left hip.
The specimen is the heart opened to display the mitral valve. The line of closure of the valve is covered by a continuous row of small brown rheumatic vegetations. The cavity of the ventricle is not dilated and the myocardium is macroscopically normal.
Histology showed typical rheumatic vegetations, with a cap of fibrin and platelets, overlying macrophages, fibrinoid necrosis and Anitschkow cells. There were a few ill-defined Aschoff nodules in the fibrous tissue of the valve.
Diagnosis: Acute rheumatic valvulitis

CASE 3839
The patient was a woman aged 22 with a 4 month history of loss of weight, cough and night sweats. A sudden pain in the right chest occurred 3 weeks before admission and blood culture grew *Streptococcus viridans*. At post-mortem there was ascites, pleural effusions, pulmonary infarcts and chronic venous congestion of the liver and spleen.

The specimen is the heart and great vessels. It is opened through the right ventricle, pulmonary infundibulum and aorta. A mass of friable vegetations lines the right side of the pulmonary artery just above the valve. There are some small nodular vegetations on one of the pulmonary valve leaflets itself. The aortic valve shows friable vegetations on the valve cusps. A blue rod has been inserted in a patent ductus arteriosus passing between the aorta and the pulmonary artery. The lumen of the ductus is about 2mm in diameter and the vegetations in the pulmonary artery are related to this ductus.
Diagnosis: Patent ductus arteriosus with infective endocarditis
What are the likely causes of the loss of weight, cough, night sweats, sudden pain in the chest, ascites, pleural effusions, pulmonary infarcts and chronic venous congestion of the liver and spleen given the pathology demonstrated?
The loss of weight and night sweats were probably caused by the infective endocarditis.
The cough, pulmonary infarcts and sudden pain in the chest were probably related to pulmonary embolism (in this case arising from the vegetations in the pulmonary artery).
The ascites, pleural effusions and chronic venous congestion of the liver and spleen are likely to be related to severe right heart failure. This could be related to pulmonary hypertension developing as a consequence of multiple pulmonary emboli and/or related to a left ->right shunt from the patent ductus.

CASE 3965
The patient was a woman aged 26 who had joint pains for 2-3 months before admission. There was a high swinging fever, changing cardiac murmurs, and red cells were found in the urine.
The specimen consists of the heart and slices of spleen and kidney. The mitral valve is covered with friable vegetations that extend for some distance down the chordae tendineae but not on to the atrial wall. The underlying mitral valve shows no thickening of the cusps or the chordae tendineae to suggest old rheumatic valvulitis. The left ventricle is not dilated or thickened. The spleen shows a classic wedge-shaped pale infarct and the kidney also shows a pale wedge-shaped infarct with a congested border. Culture grew *streptococci*, but the type is not stated.
Diagnosis: Acute infective endocarditis
See also Common and Classic Diseases section.

CASE 4033
No information is available about this specimen except that the patient died suddenly while confined to bed with a left femoral vein thrombosis. At post-mortem a mass of thrombus was found in the vein, from which a large fragment had become detached and impacted in the heart.
The specimen is the heart viewed from behind with the atria opened. A long antemortem thrombus is visible with its head in one atrium and its tail in the other, through a large patent foramen ovale (not readily visible).
Diagnosis: Patent foramen ovale with paradoxical embolism

CASE 4335
The patient was a man aged 32 who died in 1942. The only information available is that subacute infective endocarditis followed an operation on the nose. At post-mortem there were vegetations in the heart, large infarcts in the spleen and many purpuric spots in the skin of the extremities.
The specimen consists of the heart and portions of the spleen and kidney. There are friable vegetations on the mitral valve, extending upwards over a wide scarred area (MacCallum's patch) on the posterior wall of the left atrium. The underlying valve is mildly thickened. The left ventricle is dilated. There is massive ischaemic infarction in the spleen and there are small haemorrhagic flea-bitten spots (subtle) on the surface of the kidney. Culture grew haemolytic *streptococci*, *staphylococci* and *Streptococcus viridans*.
Diagnosis: Subacute infective endocarditis
What is the pathogenesis of the purpuric spots given the diagnosis? These are probably due to leaky blood vessels caused by an immune complex vasculitis. Circulating immune complexes (antigen-antibody)

(which can form in infective endocarditis) are filtered out in small vessels. Complement is activated and an inflammatory response develops**.**
What causes the 'flea bitten spots' on the kidneys? These are also caused by immune complexes – here leading to a glomerulonephritis in the kidney.

CASE 5312
The patient was a man aged 75 who died from hematemesis and melena from a large duodenal ulcer.
The specimen is of the abdominal portion of the aorta and the origins of the common iliac arteries. There are several irregular saccular atherosclerotic aneurysms of the aorta and iliac arteries that are lined by variable amounts of antemortem thrombus. Note the lines of Zahn.
Diagnosis: Atherosclerotic aorto-iliac aneurysms

CASE 6734
The patient was a woman aged 46 who had her first attack of rheumatic fever at the age of 20 and a further attack at 23. Five weeks before her last admission she was fibrillating and developed anaesthesia of the left side of the body with slurring of speech and some dysphagia. A week later there was a sudden pain in the chest and a small haemoptysis, and a further haemoptysis occurred a few days before admission. At post-mortem, there was mitral and tricuspid stenosis with infarcts in the spleen and a softening in the left lentiform nucleus of the brain.
The specimen is a portion of the heart sectioned to show the lower part of the left atrium and the mitral valve. The muscle coat of the atrium is thickened and the mitral valve is fibrotically thickened. The valve orifice is reduced to a narrow slit 2cm in length and 4mm in diameter.
Some thrombus is present in the atrial appendage.
Diagnosis: Rheumatic mitral stenosis
What are the likely causes of the patient's atrial fibrillation and splenic and cerebral infarctions? The atrial fibrillation was probably related to the mitral stenosis. Atrial fibrillation predisposes to thrombus formation (due to turbulence) in the left atrium. Embolised thrombus had probably caused the cerebral and splenic infarcts. N.B. Infarcted brain becomes softened, hence the description of softening at post-mortem.

CASE 7261
The patient was a man aged 28. The illness began a month before admission with weakness, shivering, sweating and malaise. Clinically there were signs of aortic regurgitation and a rumbling mitral diastolic murmur. The spleen was enlarged, the temperature was 38.5 degrees C. and blood cultures grew *Streptococcus viridans*. He was treated with penicillin with good result, however, after 5 weeks he suddenly developed acute heart failure and died. There was no history of rheumatic fever. At post-mortem there was marked cardiac enlargement, pulmonary oedema, pleural effusions and ascites, together with an infarct in the left kidney. The spleen was slightly enlarged (weight 275gm).
The specimen consists of the heart opened to show the greatly dilated left ventricle. The aortic valve is bicuspid and the cusps show slight fibrous thickening. The ventricular surface of one cusp is covered by pale friable vegetation and the vegetations have spread to the adjacent cusp. The principally affected cusp has perforated and shows a large tear measuring about 2cm in length and 1 cm in breadth. The mitral valve and chordae tendineae are not thickened and show no evidence of previous rheumatic valve disease. The ascending aorta and coronary orifices appear essentially normal.
Diagnosis: Infective endocarditis with perforated bicuspid aortic valve.
Comment: You should be able to explain the relationship between the bicuspid aortic valve, infective endocarditis, valve rupture, dilated left ventricle, renal infarct and the clinical signs and symptoms.

CASE 7493

The patient was a man aged 68 with a history of precordial pain on exertion for 3-4 months. He was admitted with severe precordial pain and shock consequent upon a myocardial infarction. On the 6th day he suddenly became restless and dyspnoeic with severe circulatory failure and died.
The specimen is of the heart opened to show the left ventricle. There has been rupture of two papillary muscles holding the anterior mitral valve leaflet. The myocardium above and anterior to the ruptured papillary muscles shows yellow discolouration indicative of underlying infarction. Histology showed a myocardial infarct of some days' duration, the muscle cells being necrotic with many interstitial neutrophils.
Diagnosis: Recent myocardial infarction with ruptured papillary muscle.
Explain the pathogenesis of the patient's dyspnoea and severe circulatory failure on day 6 post MI.
On day 6, there is little healing of the infarcted area yet, the necrotic muscle being weak and prone to rupture. The patient has developed acute mitral incompetence as a consequence of the ruptured papillary muscles. This has lead to acute pulmonary oedema (causing dyspnoea) and cardiogenic shock.

CASE 8923
The patient was a man aged 51 who died from the effects of disseminated lymphoma. At post-mortem there was generalised lymphoid enlargement and a haemorrhagic pericardial effusion.
The specimen is a heart. The visceral pericardium is covered by dense fibrinous exudate that is readily seen. Fibrin deposition is deepest over the posterior upper surface of the heart. Histology showed marked fibrinous pericarditis with many lymphoma cells among the cellular exudate.
Diagnosis: Fibrinous pericarditis

CASE 9897
The patient was a man aged 63 who had been in hospital 6 months previously with a myocardial infarction. Three months later there was a probable further infarct and 15 hours before his last admission he developed acute pulmonary oedema and shock. On admission the B.P. was 105/80, there was a systolic murmur, an enlarged liver, jaundice and congestive cardiac failure. A little later there was a small haemoptysis. The jaundice deepened and he complained of abdominal pain. Liver function tests suggested obstructive jaundice. Treatment with digoxin was unavailing. At post-mortem the liver was cirrhotic and nodular. The bile ducts were normal.
The specimen is one of the heart sectioned in the sagittal plane to show a large longstanding aneurysm 7cm across the base, projecting forwards for 6cm on the anterior aspect of the left ventricle. The wall of the aneurysm is a thin sheet of fibrous tissue and its cavity is entirely filled with old laminated thrombus. The mitral valve and aortic valves appear normal. The posterior wall of the ventricle shows some blotchiness and thinning of the myocardium suggestive more of fibrosis than recent infarction. Histology showed marked interstitial fibrosis of the posterior wall of the ventricle but no definite histological evidence of recent infarction.
Diagnosis: Aneurysm of the left ventricle
Explain the likely pathogenesis of the aneurysm. The aneurysm has developed in an infarcted area of myocardium. Its formation may even have started via infarct expansion in the first few days-weeks following the infarct, as the dead muscle stretches secondary to the pressure in the left ventricle. The infarct is then replaced by scar tissue that does not contract and the region can expand further -> aneurysm of the left ventricle.

CASE 10759
The patient was a man aged 73 who died of myocardial infarction. He had suffered from generalised atherosclerosis and for several weeks before his death the right foot had been pale and cold. At post-mortem the aorta was moderately atherosclerotic and the right femoral artery was completely occluded.
The specimen consists of several cross-sections of the right common femoral artery as it divides into the superficial and profunda femoris branches. All portions show luminal narrowing from atherosclerosis. There is dark occluding thrombus in the common and superficial femoral arteries. Haemorrhage is noted within the plaque (top right), which may have precipitated the overlying thrombus formation. Histology revealed gross

atherosclerosis of the intima with thrombus (blood cells, fibrin, platelets) filling the lumen. Granulation tissue infiltrated the edge of the thrombus in keeping with organization and the age of the thrombus.
Diagnosis: Thrombosed atherosclerotic femoral artery

CASE 13353
The patient was a man aged 55 who, while driving his car, experienced a sudden severe central chest pain passing through to the back between the shoulders. There was also sudden loss of power in the legs and he had to be driven home in a taxi, but recovered the use of his limbs 5 hours later. Next day he became breathless and was admitted to hospital. He was cyanosed, and the B.P. in the right arm was 150/100 and in the left arm 130/90. The right carotid pulse was reduced. There were basal systolic and diastolic murmurs in the heart. Six days after admission he developed pain at the lower end of the sternum associated with a pericardial friction rub. He became increasingly distressed with evidence of cardiac failure, and died on the 16th day after the onset of the illness.
The specimen is of the base of the heart, the arch of the aorta with the origins of the great vessels and the descending thoracic aorta. There is a transverse intimal tear about 4cm in length on the posterior wall of the aorta, 5cm above the aortic valve. From this point a dissection has tracked down the aorta and around the origin of the innominate artery. Blood can be seen within the dissection track running in the media, well demonstrated in the descending aorta. The dissection extends beyond the end of the specimen (and there was a re-entry rupture into the left common iliac artery). Histology showed some fragmentation of the elastic lamellae in the media and some pools of ground substance. The dissected sac is lined on each side by a zone of granulation tissue and early fibrosis.
Diagnosis: Aortic dissection

CASE 13780
This patient was a woman aged 66, known to have mitral stenosis and congestive cardiac failure. She had lately become orthopneic and was admitted in circulatory failure after having been given morphia 15mg intramuscularly. She remained hypotensive with cardiac arrhythmia and a very low urinary output, and died on the 5th day. At post-mortem there were pleural effusions and chronic venous congestion of organs. The heart was enlarged.
The specimen is the heart opened to show a large old irregular antemortem thrombus filling most of the left atrium. A stenosed mitral valve can just be seen inferiorly. The reverse of the jar shows the left and right ventricles and proximal aorta. The chordae tendineae of the mitral valve are fibrous and thickened. The aortic valve is essentially normal.
Diagnosis: Mitral stenosis with left atrial thrombus
What is the likely cause of the mitral stenosis? By far the commonest cause of mitral stenosis is chronic rheumatic valve disease. The fibrotically thickened valve and chordae seen in this case are typical.
What arrhythmia was likely to have been present in the patient and why? Atrial fibrillation – it is common with mitral valve disease and also, left atrial thrombus is typically associated with it.

CASE 13821
The patient was a male pedestrian aged 46 struck by a car. He was admitted with a fractured skull and fractured left leg and remained unconscious. He died on the 14th day. At post-mortem there was much subarachnoid bleeding over both cerebral hemispheres and extensive old punctate haemorrhages throughout the left cerebral hemisphere.
The specimen is of 15cm of the distal arch and proximal descending thoracic aorta. In the region where the arch meets the descending aorta there is a transverse tear through the full thickness of the vessel almost encircling it. The tear gapes in its centre. There is a moderate amount of bleeding in the mediastinal tissues outside the aorta but there has been no massive external bleed. Some thin irregular antemortem thrombus lines the floor of the tear.
Diagnosis: Traumatic rupture of the aorta

CASE 13894

The patient was a man aged 68. Eight days before admission there was severe pain in the chest passing through to the back, accompanied by shock. His doctor considered this was a coronary occlusion and he was put to bed. The pain persisted and on the day of admission there was very severe abdominal pain, shock and collapse. On admission B.P. was 80/60 and the pulse 120 and irregular. A large ill-defined pulsatile mass could be felt in the central and upper abdomen. X-ray showed calcification of the abdominal aorta and enlargement with dilatation of the thoracic aorta. Aortic dissection was considered but the pulses at the wrist were equal and the blood pressures in each arm were the same. Signs of intermittent cerebral ischaemia continued and he died after 24 hours. At post-mortem about 50ml of fluid blood were present in the pericardial cavity.

The specimen is of the heart, aorta, femoral and iliac arteries. The aortic and mitral valves appear normal. The wall of the left ventricle appears thickened. There is a 4cm intimal tear in the posterior wall of the thoracic aorta about 8cm beyond the origin of the left subclavian artery. From this primary tear there has been proximal dissection within the media up to the aortic valve and around the root of the aorta where recent epicardial haematoma can be seen. Distal dissection has occurred for only a few cm. The abdominal aorta is atheromatous and there is a large atherosclerotic aneurysm, 9cm in diameter and almost completely filled with old thrombus, in the trunk of the left common iliac artery. The reverse of the jar shows an irregular rupture through its anterior wall. From this point a large haematoma comprising about 4 litres of blood had formed in the root of the mesentery and in the mesocolon, extending around the left kidney. The other iliac arteries are dilated and atherosclerotic.

Diagnosis: Aortic dissection and ruptured atherosclerotic iliac aneurysm

Which of the patient's collection of symptoms is due to the dissection and which due to the ruptured iliac aneurysm? The patient's initial presentation (8 days before admission) with chest pain radiating to the back would have been due to the aortic dissection. The later severe abdominal pain with shock and collapse, requiring admission would have been due to the ruptured aneurysm.

From what has the patient died? The patient has died from hypovolemic shock (loss of 4 litres of blood) secondary to rupture of the iliac aneurysm.

What relationship does the thickened LV wall have to the aortic dissection? The patient may well have had systemic hypertension that is a risk factor for both.

CASE 14166

The patient was a man aged 86 who had a gastrectomy 12 months previously. He was readmitted confused and disorientated with nausea, vomiting and diarrhoea and died on the 4th day. An unexpected finding at post-mortem was an aortic dissection that extended from just distal to the left subclavian artery to the coeliac axis.

The specimen of aorta shows the upper end of the dissection. The primary rupture is on the posterior wall just distal to the left subclavian artery. It is an elongated slit with a smooth edge. From this point the dissection has passed around the superior and posterior aspects of the aorta, terminating at the level of the superior mesenteric artery. A re-entry rupture was not identified. Old laminated thrombus is displayed within the dissection track in the wall of the aorta.

Diagnosis: Old aortic dissection

CASE 14348

The patient was a girl aged 7 who had been breathless on slight exertion all her life but was never cyanosed. The heart was explored some years previously for possible coarctation but none was found. The pulse was of low volume. The apex beat was diffuse. There was a right ventricular heave with a systolic thrill and a pansystolic murmur in the left 4th to 6th spaces and along the left sternal edge. A second thoracotomy was performed using bypass. The right atrium was opened and a large atrial septal defect was found, with a split mitral valve. The mitral valve was reconstituted and a patch was sewn over the ASD. The right ventricle was then opened and a ventricular septal defect was oversewn. She did not regain

consciousness and respiratory distress persisted. Intermittent positive pressure respiration was begun but cardiac arrest occurred 5.5 hours after the operation and she could not be revived.
The specimen is of the heart in which some of the abnormalities just described can be seen. Note also a patent foramen ovale. The pericardium is congested due to operative trauma.
Diagnosis: Atrioventricular septal defect

CASE 14390
The patient was a man aged 53 who had been in good health. Two hours before death there was sudden vomiting associated with cyanosis, stertorous breathing and loss of consciousness. The BP was noted to be greater than 260/160. He died of a cerebellar haemorrhage that had extended into the 4th ventricle.
The specimen is of the heart that is enlarged. There is marked thickening (normal thickness is less than 1.5 cm) of the wall of the left ventricle, without dilatation. The right ventricular wall is also thickened. What can be seen of the valves appears normal.
Diagnosis: Concentric hypertrophy of the left ventricle due to systemic hypertension

CASE 14400
The patient was a boy aged 6 who was known to have Fallot's tetralogy with cyanosis and reduced exercise tolerance. On admission the BP was 105/75. There was an apical systolic murmur and thrill and a second sound. The fingers were clubbed. After premedication with Omnopon 10mg and Scopolamine 0.4mg and under oxygen and nitrous oxide anaesthesia the ventricular septal defect was patched and the outflow tract was repaired. Cardiac arrest occurred after completion of the operation but responded to massage and consciousness returned. Eleven hours later there was a further cardiac arrest, again successfully massaged, but 5 hours later he collapsed a third time and did not respond.
The specimen consists of the heart which is enlarged mainly through enlargement of the right ventricle. The pericardial surface is covered with a little adherent fibrin related to operative trauma and inflammation. A large surgical graft 5x2cm has been stitched on the anterior aspect of the right ventricle immediately beneath the pulmonary conus. The tricuspid valve admits 2 fingers and the valve cusps appear normal. The right atrium is somewhat dilated and the right ventricular wall is thickened. There has been excision of muscle beneath the pulmonary valve at the level of the previous stenosis but above this the pulmonary artery is normal. The ductus is obliterated. A further surgical graft has been applied over the site of the previous ventricular septal defect which can be seen best from the left ventricle, opened on the left side. The aortic valve above the repaired defect is normal.
Diagnosis: Tetralogy of Fallot
What is Fallot's tetralogy? Fallot's tetralogy is a congenital defect of the heart characterised by ventricular septal defect (VSD), subvalvar pulmonary stenosis, an overriding aorta (i.e. overriding the VSD and receiving blood from both ventricles) and right ventricular hypertrophy (as a consequence of pulmonary stenosis).
Why are patients with Fallot's tetralogy cyanosed? They are cyanosed as the high right ventricular pressure (as a consequence of pulmonary stenosis) causes deoxygenated blood to be pumped through the VSD (right-> left shunt) and into the systemic circulation.

CASE 14529
The patient was a man aged 23 known to have congenital heart disease. Thoracotomy had been performed at the age of 11 but definitive surgical repair was not made. There was diminished exercise tolerance and incipient left ventricular failure. The BP was 115/80. The heart was enlarged and there were accentuated pulmonary sounds and a pan-systolic murmur indicative of a left-to-right shunt. The patient died on the operating table from haemorrhage after ligation of the patent ductus arteriosus.
The specimen is the heart and great vessels. The ventricular walls are thickened, especially the right. The pulmonary artery shows marked aneurysmal dilatation to a diameter of 6cm and forms a thin walled sac in the concavity of the aortic arch. The very short ligated stump of a large ductus is present at its tip. The other ligated end of the ductus is on the inferior curve of the aortic arch. Both sutures are intact. A short rupture

surrounded by a purse-string suture is visible just to the left of the ligated lower end of the ductus. At post-mortem it leaked blood readily on slight pressure.
Diagnosis: Patent ductus arteriosus
Explain why the right ventricle is hypertrophied. With a patent ductus, blood is shunted from the aorta (higher pressure) to the pulmonary artery (lower pressure). This leads to an increase of blood flow through the lungs. Prolonged severe increases in pulmonary blood flow from large shunts causes thickening of arterioles with subsequent reduction of the cross-sectional area of the vascular bed. This leads to pulmonary hypertension which of course leads to right ventricular hypertrophy. (N.B. Pulmonary HT and RVH can lead to reversal of the shunt.)

CASE 14802
The patient was a man aged 42 at his death. He developed tuberculosis at the age of 9 with involvement of the left knee. This was treated by immobilization and the knee had been completely stiff from the age of 14. At the age of 30, 600ml of yellow caseous material was aspirated from the left knee. The tuberculous infection began to be increasingly active about a year before his final admission and treatment with Streptomycin, P.A.S. and INH was begun. Fever continued. A few weeks later he unexpectedly vomited a considerable amount of bright blood. Bleeding became profuse and he did not respond to resuscitation but became profoundly shocked and died. At post-mortem a large paravertebral tuberculoma was found eroding the aorta and the oesophagus. There were miliary tuberculous lesions in both lungs and both kidneys, as well as tuberculous prostatitis, seminal vesiculitis and epididymitis.
The specimen is a portion of the thoracic aorta and of the oesophagus. A large rounded caseous tuberculous mass 7cm in diameter lies between the aorta and the oesophagus. It has penetrated the oesophagus through an irregular elongated area measuring 3 x 1 cm. In the thoracic aorta there is a horizontal full-thickness split 2cm in length and the lumen of the aorta leads directly into the necrotic tuberculous mass.
Diagnosis: Paravertebral tuberculoma eroding the thoracic aorta and oesophagus
Why has the patient had hematemesis? The tuberculoma has eroded the walls of the aorta and oesophagus leading to the formation of a fistula between them.

CASE 15547
The patient was a man aged 59. At the age of 46 he had a total cystectomy with uretero-colic transplantation for a bladder carcinoma. Six years later there was a severe attack of acute right-sided pyelonephritis. At his last admission he was lethargic, thirsty and had lost weight. There was dyspnoea on exertion, swelling of the ankles and a cough with bloodstained sputum. The urine specific gravity was 1008 with 4+ albumen. The creatinine was elevated. Congestive cardiac failure became worse in spite of treatment. He died on the 10th day. At post-mortem the left kidney was small and both showed gross scars of chronic pyelonephritis. Recent bilateral acute pyelonephritis was also present.
The specimen consists of the heart that is enlarged. Both the visceral and parietal surfaces of the pericardium are covered by dense fibrinous exudate.
Histology showed non-specific fibrinous pericarditis.
Diagnosis: Fibrinous pericarditis
What is the likely cause of the pericarditis in this case? Uraemia (from chronic renal failure)
Why has this patient developed chronic renal failure (CRF)? Recurrent urinary tract infections following the uretero-colic transplantation have lead to recurrent acute pyelonephritis and chronic pyelonephritis, the cause of his CRF.
What is the cause of the patient's lethargy, thirst, loss of weight and cardiac failure? Chronic renal failure.

CASE 15624
The patient was an alcoholic man aged 48 who had been in hospital 2 months previously with an attack of alcoholic delirium tremens. Ten days before his last admission he developed red spots on the skin of the

legs together with cough, weakness and swelling of the ankles. On examination the spleen was enlarged, there was pitting oedema of the legs and there were purpuric spots on the skin. The temperature was 39.2 degrees C and the pulse 112. Serial blood cultures grew *Erysipelothrix rhusiopathiae*. A week after admission he developed headache, vomiting and semi-coma with bloodstained CSF, and a diagnosis of ruptured mycotic aneurysm was made. He died a few days later.

The specimen consists of the heart, a slice of the spleen and a portion of the frontal lobes of the brain. The left ventricle is dilated and there is a line of not very florid haemorrhagic vegetations along the line of closure of the mitral valve. The mitral valve is mildly thickened. The spleen (weight 590gm) shows a large pale infarct in the upper pole and an antemortem thrombus is visible in the large branch of the splenic artery leading to this region. A mycotic aneurysm 8mm in diameter is present on the left anterior cerebral artery. It contains antemortem thrombus and has ruptured into the white matter of the left frontal pole to form a haematoma. Posteriorly the haematoma has spread through the genu of the corpus callosum to the white matter of the right frontal pole. More posteriorly still it had ruptured into the cavities of the anterior horns of both lateral ventricles.

Diagnosis: Infective endocarditis with ruptured mycotic cerebral aneurysm and splenic infarct**.**

What is a mycotic aneurysm? A mycotic aneurysm is one that results from infection in the wall of the vessel. They usually arise in the setting of infective endocarditis, where organisms embolise from the heart and lodge on the intima of a distant vessel or gain access to the wall via the vasa vasorum.

What is the likely reason for the patient's headache, vomiting and semi-coma? Raised intracranial pressure secondary to intracerebral haemorrhage.

CASE 15636

The patient was a man aged 44. At the age of 38 he was admitted to hospital with fever, pain in the left side of the chest, anorexia and insomnia. A diagnosis of subacute bacterial endocarditis of the aortic valve with gross aortic incompetence was made. For 7 months he was treated with penicillin, streptomycin and sulphonamides. Two years later he was digitalised by his local doctor. He remained reasonably well until 10 days before his death when he was again admitted. There had been remittent fever for 2 weeks reaching 39.5 degrees C with malaise, pain in the limbs, vomiting and anorexia. The BP was 175/40. He was pale and there was a small haemorrhage in the right fundus. The JVP was not elevated, there was no cyanosis and no peripheral oedema. The fingers were clubbed. There was a diffuse thrusting apex beat in the 7th left interspace in the anterior axillary line. There was a palpable diastolic thrill in the aortic area propagated to the neck. Heart sounds: M1 present but M2 diminished, A1 soft, A2 not heard, P1 loud. At the apex there was a blowing presystolic murmur, a low-pitched mid-diastolic murmur and a pan-systolic murmur. At the base there was a rough early systolic murmur heard best in the aortic and pulmonary areas, and a hard low-pitched diastolic murmur maximal in the aortic area radiating to the neck and back. The liver was palpable 3 fingers below the right costal margin and the spleen was palpable 2 fingers below the left costal margin. A clinical diagnosis of mitral stenosis and incompetence and aortic incompetence due to subacute bacterial endocarditis was made. The ESR was 37 mm in 1 hour, haemoglobin 113g/L, leucocytes 7800, blood culture grew a microaerophilic streptococcus sensitive to penicillin, streptomycin, chloramphenicol, tetracycline and erythromycin. ECG showed complete left bundle branch block. He was treated with large doses of penicillin and appeared to be making a good recovery but on the 10th day he was found dead in his bed. At post-mortem the heart weighed 895g and the spleen 560gm.

The specimen consists of the heart that is greatly enlarged. The left ventricle shows extreme dilatation and its wall is thickened with patches of pale interstitial fibrosis most eminent near the apex. The aortic valve cusps are a little thickened and fibrous and some pale vegetation adheres to their surface. A perforation almost 1cm in length is present in one cusp. The vegetations spread over the endocardium beneath the aortic valve for a distance of some 3cm. The anterior mitral leaflet is mildly thickened and shows a 1cm aneurysmal bulge with an oval perforation about 2x3mm. There is some irregularity and endocarditis on the wall of the atrium above the perforation. The atrium itself is dilated.

Diagnosis: Subacute infective endocarditis with valvular ruptures.

CASE 15684
The patient was a woman aged 76 who suffered from giant cell arteritis. Symptoms were failure of vision over 3 weeks. Examination showed that vision was limited to movements on the right and light perception on the left. She was treated with prednisolone. Four and a half years later she was readmitted with pain in the left side of the chest associated with fits. ECG suggested posterior infarction. A month later nominal aphasia developed and she was awaiting discharge to a nursing home. After one week she collapsed with cyanosis and inaudible heart sounds and died. At post-mortem there was a massive acute hemopericardium resulting from an unusual ruptured ventricular aneurysm.
The specimen is the heart opened through the left ventricle and aorta. The ventricle is not obviously dilated or its wall thickened and the aortic and mitral valves appear essentially normal. On the posterior wall of the left ventricle is a thin walled aneurysmal sac measuring about 3x2cm and partly filled with antemortem thrombus. The sac reportedly communicated with the left ventricle via a narrow channel (not seen). A point of rupture can be seen near the tip of the sac.
Comment: This aneurysm is not typical of those seen following infarction. It could possibly have formed secondary to myocardial scarring related to ischaemic necrosis secondary to a giant cell coronary arteritis (which can be seen in association with giant cell temporal arteritis).
The aneurysm reportedly communicated with the left ventricle and did not arise from a coronary artery, however, at autopsy one would have had to consider whether it represented a coronary artery aneurysm that formed secondary to a giant cell coronary arteritis.
Diagnosis: Cardiac aneurysm of uncertain pathogenesis.

CASE 15784
The patient was a man aged 68 who died from a ruptured atherosclerotic aneurysm of the abdominal aorta with a very large retroperitoneal haematoma. A vague undefined mass could be felt in the abdomen and in spite of extensive resuscitation he died several hours after admission to hospital. At post-mortem a large atherosclerotic aneurysm was present in the abdominal aorta, occupying the entire length of the aorta between the renal arteries and the aortic bifurcation but not penetrating into the iliac branches.

The specimen is the thoracic aorta including the origin of the great vessels. There is extreme ulcerative atherosclerosis of the intima. Tags of ruptured intima are conspicuous at the margins of the plaques.
Diagnosis: Atherosclerosis
One can easily see how small pieces of this friable plaque can detach and embolise.

CASE 15945
The patient was a man aged 53. The illness began with sudden pain in the throat spreading to the upper thorax and face, followed by breathlessness and collapse, with probable loss of consciousness. He was known to be hypertensive (BP 190/120). Half an hour after the initial attack he collapsed again with lower blood pressure (120/80) and some cardiac irregularity. All pulses were normal and equal. The ECG suggested left ventricular strain and early posterior infarction. He collapsed a third time 10 hours after the onset and died two hours later.
At post-mortem there was a massive recent hemopericardium. The heart was enlarged (weight 510gm).

The specimen consists of part of the heart and the arch of the aorta with the origins of the great vessels. The left ventricular wall is thickened. The mitral and aortic valves appear normal. There is a primary rupture in the aortic arch in the form of a transverse split in the intima and media with a proximal extension for about 1cm towards the aortic valve. This split extends around half the circumference of the aorta. An acute haematoma, in this case probably largely in the adventitia, surrounds the ascending aorta and arch. There has been proximal dissection around the aortic valve ring. The hemopericardium is not apparent.
Diagnosis: Aortic dissection.
What is the main risk factor for aortic dissection? Hypertension.
What is the probable reason for the thickened left ventricle in this patient? Hypertension.

Comment: One can readily see how this haematoma could constrict the major aortic branches, including coronary arteries proximally, and how it could rupture into the pericardium proximally, or into pleural cavity as they sometimes do.

CASE 15968
The patient was a man aged 66 who died from a ruptured myocardial infarction with cardiac tamponade.
The specimen consists of the lower 9cm of aorta with the common iliac arteries and their main branches. There is aneurysmal dilatation 5cm in diameter at the lower end of the aorta. There is a larger dilatation 6x5cm of the trunk of the right common iliac artery and mild aneurysmal dilatation of the trunk of the left common iliac artery.
Diagnosis: Atherosclerotic aneurysms of the aorta and iliac arteries.

CASE 16009
The patient was a man aged 65 who was involved in a car crash. The injuries included fractured right femur, a comminuted depressed skull fracture, fractured ribs with hemothorax, fractured cervical spine and a minor laceration of the liver. Cardiac tamponade was suspected but no blood was found on aspiration. The patient collapsed during bronchoscopy and died. At post-mortem extensive bleeding was found in the mediastinum and a large haematoma enveloped the descending thoracic aorta. The left pleural cavity contained 1800ml of blood that originated from a tear in the aorta.
The specimen is of the arch and proximal descending aorta. It shows a complete transverse tear through all coats of the vessel just beyond the origin of the left subclavian artery. The reverse of the specimen shows recent haemorrhage in the mediastinum between the aorta and trachea and in the adventitia of the vessel.
Diagnosis: Traumatic rupture of the aorta.

CASE 16018
No clinical information is available. The post-mortem was performed by the coroner's pathologist in 1963.
The specimen is of the heart that weighed 65gm. The left ventricle has been opened to display one large vessel (the truncus arteriosus) overriding the interventricular septum, in association with a high septal defect that lies between and below two of the aortic valve cusps. These valve cusps appear normal and so does the mitral valve. The left ventricular wall is thickened. It is not evident in this specimen whether this single vessel communicated with the pulmonary arteries.
Diagnosis: Congenital heart disease: truncus arteriosus

CASE 16317
The patient was a woman aged 78 who was admitted with a history of sudden severe stabbing epigastric pain that became constant. The JVP was raised 2cm and there was a loud apical systolic murmur that conducted to the neck and axilla. The BP was 230/100. Two days later the pain settled. On the 4th day a pulsatile mass could be felt in the epigastrium slightly to the left of the midline.
Next day there was sudden severe abdominal pain passing to the back, and an hour later a further episode of severe abdominal and chest pain. The voice became slow and slurred. She died that day. At post-mortem a massive haematoma was found in the posterior mediastinum, extending through the diaphragm into the superficial layers of the stomach. There was much free blood and clot in the right pleural cavity and the superior vena cava was compressed from behind. The heart was large (560gm) with a thickened left ventricle.
The specimen consists of the arch and descending aorta and the stomach. The aorta shows marked atherosclerosis. A primary rupture is present as a ragged transverse slit extending around two-thirds of the circumference of the vessel at a point 3cm beyond the origin of the left subclavian artery. There has been some proximal dissection towards the arch as well as distal dissection. Blood is readily seen in the media with some in the adventitia also. Ten cm below the primary rupture there is a second transverse slit in the

intima and media. The other specimen shows massive haemorrhage infiltrating around the oesophagus and beneath the peritoneum of the upper fundus, body and antrum of the stomach.
Diagnosis: Ruptured aortic dissection.
What is the main risk factor for aortic dissection? Hypertension.
What is the probable reason for the thickened left ventricle in this patient? Hypertension.

CASE 16327
The patient was a man aged 73. A carcinoma of the left lung was discovered 9 months before his death after a 2-month history of cough, breathlessness and haemoptysis. Palliative radiotherapy was given but he continued to deteriorate and died. At post-mortem there were many metastases in the ribs, large bowel and heart. An unexpected finding was complete thrombotic occlusion of the abdominal aorta.
The specimen is of the abdominal aorta and the trunks of the common iliac arteries. There is a fusiform atherosclerotic aneurysm in the lower 7cm of the aorta producing a distinct bulge. The aneurysm is entirely filled with antemortem thrombus and the thrombus extends up to the level of the coeliac artery. The origins of the coeliac and mesenteric arteries are shown. They are atherosclerotic but are not occluded. The origin of the right renal artery can be seen to be totally filled with antemortem thrombus. The right kidney is also shown, with many areas of anaemic infarction.
Diagnosis: Thrombosis of aorta and common iliac arteries with renal infarction.

CASE 16335
The patient was a man aged 73 who had various neurological symptoms since an attack of meningitis at the age of 20. These included occipital headache, diplopia and falling to the right. He had been admitted 3 times to the Royal Adelaide Hospital in the last few years and was admitted a fourth time with substernal chest pain of 9 hours duration, not relieved by trinitrin. He died 2 weeks later. The heart showed atheroma of the coronary arteries but no obvious infarction.
The specimen consists of the aorta. There is very gross atherosclerosis with focal outpoutchings of the wall with large masses of old antemortem thrombus adherent in places. The plaque in many areas has split and one can easily see how this can give rise to athero-emboli. Antemortem thrombus extends into the origin of the left subclavian artery and occludes it.
Diagnosis: Gross atherosclerosis of the aorta.

CASE 16344
The patient was a woman aged 26. There had been two episodes of illness somewhat suggestive of rheumatic fever at the ages of 5 and 8 years, for which she was treated at the ACH and a country hospital. Later there was cardiac decompensation with breathlessness on mild exertion. She had been pregnant once and was delivered without difficulty. Four months before her death she was admitted with pain in the chest and right costal margin for one week. On examination the BP was 170/70 and the heart was enlarged, with right and left ventricular heaves. There were crepitations at the lung bases, the liver was enlarged and pulsatile, the spleen was palpable and there was ankle oedema. There were aortic diastolic and mitral systolic murmurs with a loud M1 and an atrial gallop. Treatment with diuretics produced some improvement. The clinical evidence was thought to suggest a congenital rather than a rheumatic cardiac lesion. She became febrile without other symptoms, but repeated blood cultures did not show any evidence of bacterial endocarditis. Therapy with penicillin and streptomycin was begun. Cardiac catheterisation then showed a large patent ductus arteriosus. She died unexpectedly a few days later.
At post-mortem there was intense vascular congestion in the lungs. Histologically in the lungs many of the small arteries showed medial hypertrophy and there was marked pulmonary haemosiderosis.
The specimen consists of the heart and great vessels. Anteriorly, the right ventricle has been opened to display the pulmonary outflow tract and proximal pulmonary artery. Near the bifurcation of the pulmonary artery there is a 1cm diameter communication (sectioned through the middle) with the inferior aspect of the aortic arch. This patent ductus arteriosus consists of a large hole rather than a discrete vessel, for the aorta and pulmonary artery are in apposition and only a ridge of connective tissue separates them. There is no

evidence of endocarditis in relation to the ductus. The right ventricular wall is considerably thickened, it should normally be much thinner than the wall of the left ventricle (opened on left). The pulmonary valve appears normal. The tricuspid valve ring is dilated. The mitral valve ring is also enlarged but the valve cusps appear normal. There is no interventricular septal defect.
Diagnosis: Patent ductus arteriosus.
What is the significance of the medial hypertrophy of the small pulmonary arteries found at post mortem? This is seen in pulmonary hypertension**.**
What does pulmonary haemosiderosis indicate? In this case it has been caused by long standing left heart failure. Red blood cells as well as fluid exude from the pulmonary capillaries as a result of the high hydrostatic pressure. The rbcs are phagocytosed by macrophages and the haemoglobin converted to haemosiderin. Pulmonary haemosiderosis may also be seen in other diseases where there is chronic bleeding into the alveoli e.g. Goodpasture's syndrome.
Explain the pathogenesis of this patient's congestive cardiac failure. With a large patent ductus, postnatally there is initially a L->R shunt (systemic/LV pressure > than pulmonary/RV pressure). This results in increased blood flow through the lungs and volume overload of the left heart as it returns. In response to increased pulmonary blood flow, the pulmonary arterioles constrict and hypertrophy resulting in pulmonary hypertension. Right ventricular hypertrophy and right heart failure subsequently develop.

CASE 16437
The patient was a man aged 44 who had been perfectly well until 11 months previously, when he developed quite suddenly a severe pain in the lower central chest while at work, accompanied by paraesthesiae in the left arm and by profuse sweating and vomiting. The pain lasted 45 minutes and was relieved by morphia; ECG and serum enzyme levels were normal. The next day there was a fever of 39.5^{o} C with a non-productive cough and bilateral basal crepitations. Chest x-ray showed patchy consolidation. He was treated with tetracycline and discharged home 11 days later. Nine days thereafter he began to experience breathlessness on exercise and when lying flat. Simultaneously there was a stabbing pain in the left costal margin, worse on deep breathing. These symptoms became progressively worse and then night sweats began. He felt generally tired, weak and lethargic. He began to lose weight and to sleep on 4 pillows. He was admitted to the RAH. On examination the BP was 150/40, pulse 120 regular and collapsing, temp. 40^{o} C. There were visible neck pulses, capillary pulsation and systolic murmurs over the femoral arteries. The apex beat was in the 6th left interspace 14cm from the midline. There was an apical triple rhythm, an aortic systolic murmur and thrill and a long aortic diastolic murmur. There was an added high-pitched systolic sound in the aortic region. The liver and spleen were palpable. X-ray and ECG confirmed left ventricular hypertrophy. The ESR was 45mm. He was treated with digoxin, diuretics and antibiotics and the fever gradually settled, the heart slowed and congestive failure was relieved.
By the time of discharge his exercise tolerance was greatly improved and orthopnea had almost disappeared. The spleen was no longer palpable. Although blood cultures were repeatedly negative a diagnosis of subacute endocarditis was made. On his final admission congestive failure had occurred and there were signs in the chest. Orthopnea increased with a poor response to diuretics and he died with a rising blood urea and oliguria 2 weeks after his final admission.
The specimen consists of the heart, the aorta with its major branches, and the kidneys. The heart is enlarged and there is eccentric hypertrophy of the left ventricle. The mitral valve appears normal but the aortic ring appears stretched. The intima has been torn transversely over a wide area of the ascending aorta just above the aortic valve. A broad fold of intima hangs down over the aortic valve cusps. The proximal and distal margins of the split gape widely, being about 3cm apart. The underlying exposed media is irregular and has become re-endothelialised. From this primary rupture, a dilated dissection track (from which the blood has been removed proximally) extends along the posterior wall of the ascending aorta, around the arch and down the posterior descending aorta. This track has not been opened for most of its length but can be seen as a bulge on the posterior aspect of the true aortic lumen. The origins of the intercostal arteries are surrounded by the dissection track in the thoracic portion of the vessel and can be

seen at the summit of a long smooth ridge, pushed forward by the blood in the dissection track behind. The track extends down to the left common iliac artery where it terminates in a re-entrant rupture 1cm from the origin of the vessel, on its posterior wall. There is a further re-entrant rupture at the level of the renal arteries and the intramural haematoma can be seen within this opening. The dissection also extends into the innominate and then right subclavian arteries where it extends for about 8cm before terminating in a third re-entrant rupture, and also for about 5cm along the right common carotid artery. The dissection does not involve the origins of the coronary arteries. The kidneys macroscopically appear essentially normal. There is little aortic atherosclerosis.
Diagnosis: Old aortic dissection.
Histology showed marked fragmentation of elastic lamellae in the aortic media and partial organization of the intramural haematoma.
When did the dissection occur? 11 months before death.
What is the likely cause of this patient's left heart failure and eccentric hypertrophy of the left ventricle? Aortic incompetence (as a result of the dissection extending proximally around the valve ring).
Comment. Patients may survive for many years with such re-entrant dissections. The dissection track often ultimately dilates.

CASE 16511
The patient was a man aged 59 who died from pulmonary carcinoma with metastases to the spine, producing paraplegia at T4. There had been a left femoro-popliteal bypass for occlusive arterial disease some 6 years previously.
The specimen is 9cm of the left popliteal artery sectioned longitudinally. A saccular aneurysm 2cm in diameter is present in the centre of the specimen; its cavity is completely filled with old laminated antemortem thrombus.
Diagnosis: Atherosclerotic aneurysm of the popliteal artery.

CASE 16600
The patient was a hypertensive woman aged 66 who presented with a six-day history of severe burning retrosternal chest pain radiating to both shoulders and down the right arm. The BP (previously 160/100) dropped to 120/80. ECG confirmed anterior myocardial infarction and anticoagulants were given. Repeated attacks of acute pulmonary oedema occurred while in hospital and she died in one such attack 2 weeks after the onset of the pain. At post-mortem there was a slightly turbid pericardial effusion measuring 300ml, bilateral serous pleural effusions (right 150ml, left 400ml), gross pulmonary oedema and venous congestion of organs.
The specimen is a transverse slice of the left and right ventricles. There is a full thickness extensive myocardial infarction with blotchy pallor and congestion of the anterior myocardium and much of the interventricular septum. There is probably also some involvement of the anterior right ventricle. The involved myocardium is becoming thinned. The overlying pericardium is congested but shows little pericarditis. There is patchy discolouration of the posterior myocardium also, possibly representing old scarring.
Diagnosis: Healing myocardial infarction.
Histology showed infarction infiltrated by granulation tissue in keeping with its age.

CASE 16691
The patient was a man aged 29. At age 23 he was admitted to the RAH with subacute bacterial endocarditis of the aortic valve. There was no previous history of rheumatic fever. Prophylactic penicillin was administered from that time until his death, but he became increasingly disabled and was finally a complete invalid. Cardiac catheterisation towards the end of the illness showed severe aortic incompetence but there was no evidence of mitral disease. Angiography also showed aortic incompetence and an aneurysm of the right coronary artery. The aortic valve was replaced at a bypass operation, and the right coronary aneurysm was ligated. Satisfactory perfusion of the right coronary artery was not obtained and

ventricular fibrillation set in when the bypass was discontinued. Vigorous attempts at resuscitation were unsuccessful.
The specimen consists of the upper portion of an enlarged heart. There is some acute epicardial haemorrhage as a result of the operation. There is marked thickening of the left ventricular wall and enlargement of the right ventricle. The mitral valve is mildly thickened. The aortic valve prosthesis is in a satisfactory position. The left coronary artery and its anterior descending branch have been opened longitudinally.
Diagnosis: Aortic valve prosthesis with left ventricular hypertrophy.

CASE 17150
The patient was a man aged 66 who was involved in a car crash 5 days before his death from a massive pulmonary embolism.
The specimen consists of the upper portion of the heart. A portion of the embolus - which has originated in a leg vein - can be seen trapped in a patent foramen ovale in the inter-atrial septum. The ragged tail of the embolus lies in the right atrium and the rounded head lies in the cavity of the left atrium.
Diagnosis: Patent foramen ovale with paradoxical embolism.

CASE 17177
The patient was a man aged 78 who was admitted with a 12-hour history of severe pain in the chest. The attack lasted for about 1 hour and then the pain moved to the back and abdomen. He vomited 3 times when the pain was at its worst. At the time of admission the pain had almost settled. On examination he was pale with a feeble totally irregular pulse. There was circulatory failure and his hands were cyanosed and cold and his feet were pale and cold. There was guarding of the abdominal wall in the epigastrium, and a pulsatile mass could be felt in the midline. An audible murmur was present over the abdominal mass and a murmur was also heard over the femoral arteries. A diagnosis of ruptured abdominal aneurysm and posterior myocardial infarction was made. The circulatory failure did not respond to treatment and he died 6 hours after admission. At post-mortem no myocardial infarct was found, the coronary arteries were patent and the abdominal aortic aneurysm had not ruptured. The kidneys were small and nephro-sclerotic but showed no infarcts.
The specimen consists of the abdominal aorta and the iliac vessels. There is a large thick-walled cylindrical aneurysm 12cm in length and 9cm in diameter in the lower abdominal aorta. Old laminated thrombus covers the irregular atheromatous wall. The upper end of the aneurysm lies about 2cm below the origin of the renal arteries. The common iliac arteries also show dilatation with gross pultaceous atheroma and patchy adherent thrombus.
Diagnosis: Atherosclerotic aorto-iliac aneurysm.

CASE 17320
The patient was a treated hypertensive man aged 66. There had been previous anginal attacks and he was breathless on exertion. He was admitted with severe retrosternal chest pain and dyspnoea of 4 hours duration. The pain radiated down both arms. On examination the BP had fallen from its previous level of 160/100 to 115/80. There was no fever or cyanosis and the heart sounds were regular. ECG suggested infarction, enzyme levels were raised and there was a leucocytosis of 19,000 (90% neutrophils). He was anticoagulated and appeared to be improving but died suddenly on the 21st day. At post-mortem there was extensive atherosclerosis of the aorta with aneurysmal dilatation of the lower abdominal aorta and the common iliac arteries. The right coronary artery was narrow and the anterior descending branch of the left coronary artery was narrow but not thrombosed.
The specimen consists of a slice through both ventricles. There is an antero-septal myocardial infarction showing patchy haemorrhagic congestion of the muscle. Pearly-grey patches of fibrous organisation are visible in keeping with the history of 3 weeks duration.
Diagnosis: Healing myocardial infarction.

CASE 17394
The patient was a woman aged 33. Two years previously a partial gastrectomy was performed for carcinoma. A year later bilateral Krukenberg tumours of the ovaries were removed and radiotherapy was begun. Later she was treated with cyclophosphamide. She died of general peritonitis after rupture of the ileum. There were many metastatic deposits throughout the abdomen. There was no history of previous rheumatic fever. The BP was 110/90 and no cardiac murmurs were heard.
The specimen is the heart opened to show the left atrium and left ventricle. The muscle coat of the left atrium is thickened. The mitral valve and chordae tendineae show fibrotic thickening and the chordae are shortened. The muscle coat of the left ventricle is of normal thickness.
Diagnosis: Old rheumatic mitral valve disease.
What are Krukenberg tumours? These are metastases to the ovaries of signet ring cell carcinomas, usually arising in the stomach.

CASE 17577
The patient was a woman aged 74. She was uraemic and had complete heart block. The BP was 110/70. At post-mortem the heart weighed 510gm.
The specimen shows two slices through the heart. The left ventricle is dilated and its wall mildly thickened. Many patches of pale fibrous tissue are present in the left ventricular wall, particularly near the apex.
Diagnosis: Patchy myocardial fibrosis.
Comment: While ischaemic heart disease cannot be excluded as a cause, the pattern of fibrosis is not typical of infarctions (larger area of fibrosis in arterial territory) or chronic ischaemia (generally tiny patchy areas of subendocardial fibrosis). Other conditions that can cause patchy fibrosis in the heart include sarcoidosis and dilated and hypertrophic cardiomyopathies.

CASE 17930
The patient was a woman who had been treated for aortic incompetence for 7 years. There was a strongly positive Wassermann reaction. A month before her last admission she developed a left sided pleural effusion for which she was referred to the Chest Clinic, then to the RAH. The pulse was 94/minute and collapsing, and there was a loud blowing to-and-fro aortic murmur transmitted to the neck. The Wassermann. Kahn, Kline, Kolmer and RPCFT tests were all positive. Bronchoscopy showed distortion of the left main bronchus by the dilated aorta. Cytological examination of the pleural aspirate showed malignant cells and left scalene node biopsy revealed a poorly differentiated adenocarcinoma. Irradiation of the lung tumour was planned but she died suddenly after 4 weeks in hospital. At post-mortem a carcinoma of the left upper lobe was found, with spread to the pleura. There was aneurysmal dilatation of the aorta but the report does not state how far it extended.
The specimen is the heart opened to show the dilated syphilitic ascending aorta. The wall is fibrous and there is overlying atherosclerosis. The aortic ring is stretched and the aortic valve leaflets are mildly fibrotic, as is the mitral valve. The commissures of the aortic valve are separated as is classical in syphilis. The left ventricular cavity is mildly dilated.
Diagnosis: Syphilitic aortitis with aneurysm.
Histology showed long-standing aortitis with fibrosis and destruction of elastic laminae.
What is the pathogenesis of syphilitic aortitis? The effects in the aorta are proposed to develop from inflammation of the vasa vasorum, the infiltrate composed mainly of lymphocytes and plasma cells. Fibrosis and narrowing of these small vessels occurs leading to atrophy and fibrosis of the aortic wall.
In what stage of syphilis does the aortitis occur? Tertiary syphilis, which develops many years after the initial infection.

With what other illnesses can aortitis occur? Giant cell (temporal) arteritis, rheumatoid arthritis, systemic lupus erythematosus, ankylosing spondylitis, Takayasu's disease and others.
What are the complications of aortitis? Aortitis most commonly affects the ascending aorta. It typically causes dilation of the aortic root with aortic incompetence. It can also precipitate aortic dissection. Takayasu's disease often involves the main branches of the aortic arch leading to fibrosis and narrowing.

CASE 18071
The patient was a woman aged 70 who was admitted to hospital with gallbladder disease. Investigation showed a malignant lymphoma in the caecum and regional lymph nodes and a right hemicolectomy was performed. Thereafter radiotherapy was given but she slowly deteriorated with a rising creatinine and died after 3 months. At post-mortem there was a single pigment stone in the gallbladder. The bile ducts were patent. An antemortem thrombus was found in the femoral vein.
The specimen shows this thrombus that is at least 19cm in length. It has a smoothly pointed head directed superiorly and its surface is generally pale from the deposition of fibrin and platelets. Extension into a branch can also be seen just below the middle.
Diagnosis: Antemortem thrombus in the femoral vein.

CASE 18207
The patient was a man aged 61 who had had a myocardial infarction 4 years previously. During the last two weeks there had been several attacks of ischaemic chest pain. He then passed into circulatory failure and was admitted to hospital. The BP was low and he died from gross pulmonary oedema half an hour after admission.
The specimen is the heart opened through a large aneurysm 10cm in diameter at the apex of the left ventricle. The wall of the aneurysm is about 2mm in thickness and is composed entirely of fibrous tissue. The cavity is somewhat bilocular and the upper loculus is filled by old laminated antemortem thrombus. There is some congestion of the myocardium of the septum just above the aneurysm, on the left side of the specimen.
Diagnosis: Left ventricular aneurysm.
Explain the pathogenesis of this aneurysm in light of the patient's history. The myocardial infarct 4 years before death would have been in the LAD territory. It has healed leading to scarring and thinning of the myocardium in this area. The scar tissue does not contract and the region expands forming an aneurysm.
Which coronary artery supplies the area of the aneurysm? Left anterior descending.
From what has this patient died and why has it arisen? The patient died from acute pulmonary oedema that arose secondary to acute left heart failure as a result of acute ischaemic heart disease in an already compromised myocardium (old MI with aneurysm). From the history, it sounds as if the patient has suffered acute myocardial ischaemia that may or may not have resulted in an infarct (there is insufficient information from the history and specimen to determine if a recent infarct has occurred).

CASE 18762
The patient was a girl aged 15 with congenital mental retardation and gross thoracic kyphosis. Carotid arteriogram showed hydrocephalus and ventriculography revealed congenital aqueductal stenosis. Bilateral Torkildsen's tubes were inserted but did not function. A ventriculo-cardiac shunt was therefore placed in the right side, and she recovered uneventfully. Four months later she was readmitted with purulent meningitis. The smear showed pneumococci. She died next day.
The specimen shows the jugular vein and superior vena cava with the ventriculo-atrial catheter in situ. A large mass of antemortem thrombus surrounds the lower 10cm of the catheter and extends into the innominate vein across the front of the neck. The greatest mass of thrombus lies within the right atrium.
Diagnosis: Thrombosis around ventriculo-atrial shunt

CASE 18781
The patient was a man aged 59 who was a heavy drinker and smoker. He was admitted with a history of severe epigastric pain and vomiting of 24 hours duration. Bowel sounds were absent and the abdomen was rigid, but there was no fever. An internal hernia was suspected and laparotomy showed a volvulus of the small bowel. This was untwisted and the abdomen was closed. Next day he was febrile and there were crepitations at both lung bases. By the 6th day he had a high swinging fever (to 103º F) that persisted for a week. Subphrenic abscess was suspected but he gradually developed signs of pneumonic consolidation in the right lower lobe and later in the left midzone. Antibiotics were not given until the 12th day after the operation. The temperature then fell, but he developed circulatory failure and cyanotic areas appeared on the left ear, left buttock and right 5th finger. The creatinine level rose. At this stage coagulase positive *Staphylococcus aureus* was grown from the blood. Bacterial endocarditis with septic emboli was diagnosed. The patient gradually improved for a time but then he died after a month in hospital.
The specimen consists of the heart together with slices of spleen and liver. Infective vegetations are present on all three aortic leaflets. The underlying cusps otherwise appear relatively normal. The left ventricular wall appears mildly thickened. There are several wedge-shaped peripheral areas of ischaemic infarction in the spleen with superimposed suppurative change. The liver shows contiguous irregular areas of pale ischaemic infarction, in all about 7cm maximum diameter, with surrounding zonal congestion.
Diagnosis: Acute infective endocarditis.
Histology of the liver showed infarction surrounded by a wide zone of acute inflammatory exudation. Outside this was young granulation tissue. Clumps of cocci were conspicuous throughout the infarct.

CASE 19083
This woman aged 40 presented to her local doctor with a sore throat 3.5 weeks before admission. This was treated with penicillin with apparent relief, but 3 weeks later she saw her doctor again, complaining of fever with aching in the arms and legs. Two days later the temperature was 39.8º C with hyperpnoea, tachycardia, neck rigidity and a positive Kernig's sign. Leucocytes were 14,000. Chest x-ray showed no abnormality. Treatment was begun with penicillin and streptomycin, but she did not improve and was admitted to the RAH. At that time the temperature was 39.8º C, pulse 140, respirations 40 and BP 140/45. There was a petechial rash on the skin. There was marked neck stiffness. Lumbar puncture showed slightly bloodstained CSF with 290 neutrophils and 8000 red cells per cubic mm. No bacteria were seen. There was a leuco-erythroblastic blood picture, Hb was 9.3g/dL, WBC 10,300 with occasional myelocytes and metamyelocytes. Intensive antibiotic treatment was given together with hydrocortisone, but she remained very ill and unrouseable and died on the 3rd day. At post-mortem the brain was swollen and there was an acute haematoma in the parasagittal region of the right parietal lobe.
The specimen is the heart opened to show much acute vegetation on the ventricular aspect of the mitral valve, extending along the chordae tendineae to a papillary muscle. The vegetation appears to erode through the valve on to the atrial aspect of the leaflet. The underlying valve is mildly fibrotic. The kidney shows numerous small subcapsular petechial haemorrhages. **Diagnosis:** Acute infective endocarditis.
What is the likely cause of the neck stiffness and the haemorrhage in the brain? These were probably caused by a ruptured mycotic aneurysm, the neck stiffness from a degree of subarachnoid bleeding, not described at post-mortem.
What has caused the petechial haemorrhages in the kidney? These may be related to microemboli causing foci of necrosis and haemorrhage or to immunologically mediated glomerulonephritis.

CASE 19487
The patient was a hypertensive man aged 49 who had had a myocardial infarction 7 months previously. This left him with a large left ventricular aneurysm and in gross congestive cardiac failure. The aneurysm was excised. Post-operatively his condition was generally satisfactory for 24 hours but then respiration became difficult. He was placed on a respirator but developed gastric dilatation and decreased cerebral

perfusion and died on the second post-operative day. At post-mortem there was a large amount of blood and blood clot in the pericardial cavity.
The specimen shows the anterior surface of the heart. A group of blue surgical stitches can be seen at the point of excision of the aneurysm. Note the fibrosis related to the excised aneurysm in the adjacent myocardium. Clotted blood fills much of the pericardial sac.
Diagnosis: Post-operative haemorrhage after cardiac surgery.

CASE 19676
The patient was a man aged 53. Four years previously he was found to have aortic stenosis. The aortic valve was opened and one cusp was replaced by a teflon Bahnson cusp. After this he was apparently well except for some anginal pain. Further calcification was seen on x-ray and a second operation was contemplated, but he suddenly developed severe chest pain followed by acute left ventricular failure with signs of aortic regurgitation. He died a week later.
The specimen consists of the heart opened to show an aortic valve of two cusps. There is considerable fibrous thickening and nodular calcification of the residual aortic cusp. The teflon aortic cusp has fractured and a large linear split extends through its centre to its insertion. Antemortem thrombus covers this ruptured cusp. The wall of the left ventricle is thickened. There is an irregular mass of calcium in one mitral leaflet. The aortic intima shows very mild atherosclerosis.
Diagnosis: Fractured teflon aortic valve cusp.

CASE 19712
This man aged 60 had aortic stenosis and incompetence and mitral incompetence. The aortic and mitral valves were each replaced with a Starr-Edwards prosthesis. He was anti-coagulated and appeared to be well, but died suddenly 6 weeks after the operation. At post-mortem there was pulmonary oedema and bilateral small pleural effusions.
The specimen is the heart opened to show the two prostheses. The suture lines around both valves and in the aorta appear to be intact. There is very little surrounding antemortem thrombus and no evidence of infective endocarditis. The left atrium and the left ventricle are dilated and their walls thickened.
Diagnosis: Starr-Edwards prostheses replacing aortic and mitral valves.
What is the most likely cause of this man's underlying aortic and mitral valve stenosis/incompetence? Why? Rheumatic valve disease – as it not uncommonly involves both mitral and aortic valves. Other valve diseases generally only affect one valve.

CASE 19774
This woman was a congenital mental defective aged 41 known to have a ventricular septal defect. Profuse bleeding occurred from a polypoid villous tumour of the rectum. At operation the rectum was found to be gangrenous and extensive resection of the large bowel was necessary. She died on the operating table.
The specimen is the heart and proximal aorta opened through the right ventricle. The right ventricle is dilated and its wall thickened. The right ventricular outflow tract (right of specimen) is small, and the pulmonary valve leaflets are deformed. The aorta overrides a ventricular septal defect that measures about 1.5 x 2 cm and communicates directly with both left and right ventricles.
Diagnosis: Fallot's tetralogy.
What is Fallot's tetralogy? Fallot's tetralogy is a congenital defect of the heart characterised by ventricular septal defect (VSD), subvalvar pulmonary stenosis, an overriding aorta (i.e. overriding the VSD and receiving blood from both ventricles) and right ventricular hypertrophy (as a consequence of pulmonary stenosis).

CASE 20003
The patient was a male woodcutter aged 66 who was found in the bush with a right hemiparesis. A loud systolic murmur was heard suggesting mitral incompetence. The BP on admission was 180/95. A consultant neurologist suggested urgent bilateral angiography. The patient died 2 days later. At post-mortem a large softening was found in the left middle cerebral arterial territory.
The specimen is the left common carotid artery. An antemortem thrombus 9cm in length completely occludes the proximal part of the common carotid trunk, terminating a few cm below the bifurcation. A smaller thrombus is also present in the origin of the internal carotid artery. Acute adventitial haemorrhage is present, consequent upon the arterial puncture for angiography.
Diagnosis: Thrombosis of left common carotid artery.

CASE 20337
The patient was a retired university lecturer aged 71. He had a left sided stroke 4.5 years previously with good recovery. Three years later an opacity was found in the chest on an x-ray performed after an unresolved respiratory infection. At his last admission there had been progressive weakness of the left arm and left leg for one week. Two days later he became deeply unconscious with bilateral upgoing plantar reflexes, constricted pupils and Cheyne-Stokes respiration. He died next day. At post-mortem there was gross haemorrhagic infarction of almost the entire right middle cerebral arterial territory. There was no evidence of left hemispheric infarction.
The specimen consists of both common carotid arteries with the origins of the internal and external branches. Both internal carotids are occluded by antemortem thrombus arising on atherosclerotic patches in the carotid bulbs.
Diagnosis: Bilateral thrombosis of internal carotid arteries.

CASE 20366
The patient was a woman aged 41. Six years previously a diagnosis of truncus arteriosus was made after investigation by cardiac catheterisation. She was admitted several times thereafter with paroxysmal tachycardia and congestive cardiac failure. Towards the end, venesection was performed because of polycythaemia. At her final admission there was right and left ventricular failure with atrial fibrillation. On the 6th hospital day she suffered a cardiac arrest and died. At post-mortem the heart was enlarged, weighing 680gm.
The specimen is of the heart and proximal aorta. The wall of the right ventricle is greatly thickened. A large ventricular septal defect 2.5cm in diameter is present below an overriding single large vessel that replaces the normal aorta and pulmonary artery. The pulmonary artery arises from this vessel posteriorly. The leaflets of the single valve at the base of the truncus are thickened and the commissures are adherent. The left coronary orifice is of normal size and arises from the anterior sinus of Valsalva. There is a double right coronary orifice (not seen).
Diagnosis: Truncus arteriosus.

CASE 20717
The patient was a man aged 65 in whom an abdominal aneurysm had been diagnosed clinically 3 years previously. He presented with a 3-day history of acute abdominal pain, beginning around the umbilicus and becoming generalised, with vomiting and dysuria. A large pulsatile abdominal mass could be felt. Both femoral pulses were present. At laparotomy a ruptured diverticulum in the pelvic colon was found, with general peritonitis. The diverticulum was excised. Postoperatively there was moderate fever. He died suddenly on the 4th day. At post-mortem the abdominal aneurysm had ruptured and a large haematoma containing 4 litres of blood was present in the retroperitoneal tissues and in the peritoneal cavity.
The specimen is the abdominal aorta opened to show a fusiform atherosclerotic aneurysm 12cm in length and 9cm in diameter. The aneurysm bulges anteriorly and is filled with antemortem thrombus. Acute haemorrhage is present in the adjacent fat on the reverse of the specimen.
Diagnosis: Ruptured atherosclerotic aortic aneurysm.

CASE 20814
The patient was a woman aged 55 with rheumatoid arthritis whose illness began several years previously with backache and polyarthritis. The Rose test was positive. Later there was tachycardia, with aortic disease and aortic regurgitation. This progressed, and 3.5 years after the onset of the illness the aortic valve was replaced. Thereafter she was reasonably well. A few weeks before her last admission aortic incompetence returned with a regurgitant murmur and a collapsing pulse. It was thought that some minor leakage past the artificial valve had occurred. On the evening of her death she suffered a very acute attack of pulmonary oedema with marked frothing from the nose and mouth and she died quite quickly.
The specimen is the heart opened to show the aortic valve prosthesis, surrounded by antemortem thrombus. The base of this prosthesis has torn away from its bed around some two-thirds of its circumference. There is a further old perforation of the aortic valve ring on the right anterior aspect. There is marked pearly thickening of the aortic intima as a result of rheumatoid aortitis. The left and right ventricles are somewhat dilated.
Diagnosis: Rheumatoid arthritis associated aortitis with avulsed aortic valve prosthesis.
Histology showed marked fibrosis with destruction of elastic laminae in the outer third of the media with lymphocytes and plasma cells. The intima is fibrous.
With what other illnesses can aortitis occur? Giant cell (temporal) arteritis, syphilis, systemic lupus erythematosus, ankylosing spondylitis, Takayasu's disease and others.
What are the complications of aortitis? Aortitis most commonly affects the ascending aorta. It typically causes dilation of the aortic root with aortic incompetence. It can also precipitate aortic dissection. Takayasu's disease often involves the main branches of the aortic arch leading to fibrosis and narrowing.

CASE 20815
The patient was a woman aged 80 who had complained of malaise, anorexia, vomiting and vague atypical pain in the epigastrium, thorax and back. On examination the pulse was 88 and the BP 155/105. There was a loud apical systolic murmur radiating to the neck. A rounded very hard mass continuous with the liver could be felt beneath the right costal margin. In hospital she became progressively more breathless and a left pleural effusion was tapped. She died suddenly on the 5th day after admission. At post-mortem a carcinoma of the gallbladder invading the liver and spreading to the peritoneum and the pleural cavities was found. There was also a massive hemopericardium.
The specimen is the heart sectioned in the coronal plane. There is a haemorrhagic area of infarction in the anterior left ventricle. An irregular split about 3cm in length represents the site of rupture of the infarct. The aortic valve demonstrates marked nodular calcification and fusion of 2 of its cusps. The reverse of the jar shows some small patches of interstitial fibrosis in the myocardium of the septum and in the base of a papillary muscle.
Diagnosis: Ruptured anterior myocardial infarction and stenosed calcified aortic valve.

CASE 21232
The patient was a man aged 57 who had suffered an anterior septal infarction 3 months previously. Thereafter he became progressively weaker and disorientated. A month after the onset the left ventricle was considered to be only slightly enlarged but 2 months later, in the week before his last admission, he became severely breathless and confused and there was slight jaundice. Chest x-ray showed a large heart and pneumonic changes at both lung bases. Myocardial failure persisted and he died after 4 days in hospital.
The specimen is the heart and proximal aorta. There is a very large apical aneurysm in the left ventricle measuring 7cm across the neck and 10cm in its greatest diameter. The sac is largely filled with old laminated antemortem thrombus. The wall of the aneurysm is thin and fibrous and is about 2mm thick.
Diagnosis: Left ventricular aneurysm.

CASE 21451
This patient was a woman aged 69 who was admitted with a history of pain in the upper abdomen and chest for 36 hours, radiating to the angle of the jaw. She vomited soon after the onset of the pain. On the 5th hospital day she suddenly became weak, cold and shocked. The BP was unrecordable and a loud harsh systolic murmur could be heard. Serum enzyme levels were raised. She was considered to have ruptured a papillary muscle. She died 12 hours after onset of shock.
The specimen consists of the heart opened to show the left ventricle and aorta. There is an old scarred infarction with pale fibrous endothelial thickening at the apex of the ventricle, involving also the apical portion of the septum. Some antemortem thrombus adheres to the inner surface of this area. There is massive rupture of the posterior part of the interventricular septum a few cm above this old apical scar. Subtle yellow muscle necrosis is visible around the split.
Diagnosis: Recent myocardial infarct with ruptured interventricular septum.
Which arteries supply the affected areas? What abnormalities would you expect to see in each? The recent infarct is in the territory of the right coronary artery and the old infarct in the territory of the LAD coronary artery. The LAD is likely to demonstrate atherosclerosis and old fibrous occlusion (healed thrombus) proximally and the right coronary artery is likely to demonstrate atherosclerosis and recent thrombotic occlusion proximally.
Correlate the clinical symptoms and signs in light of the pathology demonstrated. The abdominal and chest pain radiating to the jaw was due to the recent infarction. On the 5th day post MI the infarct ruptured through the IV septum leading to a L->R shunt causing the systolic murmur and acute cardiac failure leading to cardiogenic shock.

CASE 21522
The patient was a man aged 76 who presented in casualty with right hemiparesis and aphasia. He was semi-comatose and the left pupil was larger than the right, but both reacted. Loud murmurs could be heard over both carotid arteries. The BP was 180/85. He died 30 hours after admission.
The specimen consists of a portion of the aortic arch with the great vessels arising from it. There is moderate intimal atherosclerosis with patchy calcification. The origin of the innominate artery is narrowed by atheromatous plaque. A large pultaceous atheromatous patch narrows the right common carotid artery for about 3cm at a point 6cm above its origin. There is old pultaceous atheroma and thrombus in the right carotid bulb that is almost completely occluded. The left common carotid artery is atheromatous and its entire trunk is occluded by antemortem thrombus, the upper end of which reaches almost to the bifurcation. The left subclavian artery is large.
Diagnosis: Atherosclerosis and thrombosis of carotid arteries.

CASE 21740
The patient was a man aged 66 with a 3-week history of colicky pain and diarrhoea. Laparotomy revealed marked congestion of 120cm of the terminal ileum extending to within 15cm of the ileo-caecal valve. No resection was performed. His condition did not improve and further laparotomy 6 days later showed a mesenteric thrombosis. He died next day. At post-mortem the small bowel was infarcted from a point 60cm beyond the 3rd part of the duodenum, extending to include the caecum and ascending colon. The transverse, descending and pelvic colons were not involved. There was moderate atheroma of the abdominal aorta but the superior mesenteric artery was free of thrombus. Antemortem thrombus occluded the superior mesenteric vein. The splenic vein was not involved.
The specimen shows this thrombus extending from the superior mesenteric vein into the lumen of the portal vein.
Diagnosis: Thrombosis of the superior mesenteric vein.
What risk factors for this condition (not necessarily given in the history) may have been operative in this patient? Venous thromboses (as opposed to arterial thromboses) are most likely to be precipitated by

hypercoagulability of the blood (e.g. due to malignancy, oestrogens (not relevant here), hereditary conditions) and/or venous stasis (not relevant here).

What are the main causes of small bowel infarction?

Venous occlusion:

- Strangulation in a hernia or around a peritoneal adhesion
- Volvulus
- Intussusception

Arterial occlusion:

- Atherosclerosis and thrombosis
- Embolism

Systemic hypotension

CASE 21868

The patient was a man aged 35. A malignant thymoma was diagnosed after thoracotomy 6 months before his death. Biopsy at operation showed a cellular tumour with the appearance of a spindle-cell thymoma. Local radiotherapy was given. He was readmitted 4 months later with melena and a central abdominal mass was irradiated. At his last admission there was crushing central chest pain and cough, and ECG suggested anterior myocardial infarction. X-ray showed a pericardial effusion and bloodstained fluid was aspirated. Myasthenic symptoms with weakness and lid lag were noticed just before his death. At post-mortem there were many secondary deposits infiltrating the small bowel, the omentum and the liver. There were also many secondaries in the lungs.

The specimen consists of the left lung, the heart, pericardium and mediastinal structures, together with the upper respiratory passages and the thyroid gland. A large mass of pale tumour is present in the superior mediastinum, more on the right side than the left. The tumour surrounds and compresses the aorta and great vessels and has extended in continuity to invade the parietal and visceral layers of the pericardium, which can be seen covered with nodular haemorrhagic tumour. Patches of fibrinous pericardial exudate are also noted. The right lobe of the thyroid is unusually pale and homogeneous in appearance suggesting tumour infiltration. The reverse of the specimen shows the tumour infiltrating through the full thickness of the myocardium of the left and right ventricles near the apex. Irregular pale deposits are also visible in the substance of the lung.

Diagnosis: Malignant thymoma invading the heart.

CASE 22378

The patient was a boy aged 17 with a well-documented history of Marfan's syndrome. He was admitted to hospital with congestive cardiac failure. The pulse rate was 120. The right radial pulse was absent but the right brachial pulse was present. The apex beat was in the mid-axillary line and the BP was 125/50. The heart sounds were characteristic of aortic and mitral incompetence. The condition was attributed clinically to limited aortic dissection, but the immediate problem was left ventricular failure, which did not respond to digitalis and diuretics. He then developed a cold left foot and extensor right plantar response and became progressively oliguric. He died on the 6th day. At post-mortem there was pulmonary oedema and bilateral pleural effusions, together with chronic venous congestion of organs. Histology of the aorta showed patchy loss of medial elastic laminae.

The specimen is the heart and aorta. The heart is greatly enlarged. There is dilatation of the left ventricle with mild thickening of its wall. The aortic valve cusps are thin and stretched. There is dilatation of the right ventricle. The descending and abdominal aortas are narrow. The ascending aorta is dilated and there are large irregular areas of what probably represent old intimal tears. There is no evidence of medial dissection or of external rupture. There is no evidence of infective endocarditis.

Diagnosis: Marfan’s syndrome

What is the likely cause of this patient's congestive cardiac failure? It probably arose as a result of aortic incompetence due to dilation of the aortic valve ring that arises secondary to abnormalities in the wall of the ascending aorta.

What might have caused the mitral incompetence? It may have arisen due to dilatation of the mitral valve ring secondary to severe left ventricular dilation (as a result of aortic incompetence). Marfan's patients sometimes have a condition called floppy (or myxomatous) mitral valve (not apparent here) that can also cause mitral incompetence.
What is Marfan's syndrome? Marfan's syndrome is a genetic (transmitted as autosomal dominant) disorder of elastic tissue. Patients are generally tall with long limbs and digits. They often suffer, amongst other things, kyphoscoliosis, lens dislocations and 'floppy' mitral valves (mitral valve prolapse) and their aortas develop changes predisposing them to dissections and aneurysms of the ascending aorta.
What typical histologic changes occur in the ascending aorta of a patient with Marfan's syndrome? The aorta histologically demonstrates patchy fragmentation and loss of elastic tissue and accumulation of pools of mucopolysaccharide in the media, a change (incorrectly) termed 'cystic medial necrosis'.

CASE 22385
The patient was a woman aged 82. An episode of severe chest pain radiating to the arms occurred two weeks before admission, and there was a 2nd similar episode on the day of admission, lasting about 1 hour and associated with breathlessness, orthopnea and sweating. Twelve hours after admission a 3rd attack of pain occurred, and a loud pansystolic murmur was heard, maximal over the 3rd and 4th left interspaces. She died a few hours later.
The specimen is a slice through both ventricles showing an area of blotchy discolouration anteriorly. This area has ruptured and a false passage can be seen extending from the left to the right ventricle. There is a patch of epicardial haemorrhage over the infarcted area.
Diagnosis: Myocardial infarction complicated by rupture of the interventricular septum.
Histology showed neutrophil infiltration into the infarct but no fibrous organization. With which episode of chest pain is the infarct likely to have occurred and why? The infarct is most likely to have occurred on the day of admission, corresponding to the 2nd episode of pain. If it was 2 weeks old, there is likely to have been few neutrophils and some organisation (not seen histologically) and rupture would have been unlikely. It would not have occurred at the 3rd attack of pain as an infarct that was only a few hours old would show few if any neutrophils and would not have ruptured.

CASE 22393
The patient was a man aged 55. Two years previously aortic stenosis and incompetence developed as a result of bacterial endocarditis. Operation was considered inadvisable because of personality problems. On his last admission the BP was 90/60 and the ECG showed atrial fibrillation and left bundle branch block. He died after 2.5 weeks in hospital. At post-mortem the heart was greatly enlarged (weight 840gm) and there was venous congestion of organs.
The specimen is the heart opened to show the greatly dilated and thickened left ventricle. The myocardium is 2.5cm in thickness. The aortic valve shows gross fibrotic thickening and calcification with fusion of the cusps. Antemortem thrombus adheres to areas of ulceration of the valve. Projecting downwards from the left posterior coronary sinus is an aneurysmal cavity 2cm in diameter completely filled with laminated thrombus (left side of specimen). It communicates with the sinus by a narrow straight channel 2mm in diameter and 1cm in length. The left coronary artery runs anterior to the aneurysm but it does not appear to be involved. The right ventricle is also dilated.
Diagnosis: Healed infective endocarditis with aneurysm of sinus of Valsalva.

CASE 22398
The patient was a man aged 68 who had intermittent claudication for 7 years. A left lumbar sympathectomy was performed at that time. Four years later he developed gangrene of the right hallux and the right common iliac artery was disobliterated. At that time he was found to be diabetic. The operation on the right side was not successful and was followed a few months later by a right femoro-popliteal bypass using a

vein graft. However gangrene in the right foot progressed and the right limb was amputated below the knee. On his last admission 18 months later there was pain and swelling of the left foot at rest and an aorto-femoral bypass was performed. There was little improvement in the blood flow to the limb and a haematoma at the upper end of the wound became infected. Revision of the stump was performed but he died a day later. At post-mortem there was recent antero-septal myocardial infarction and severe calcific and ulcerative atherosclerosis of the entire aorta.
The specimen consists of the distal 10cm of aorta together with left and right common, external and internal iliac arteries and the left common femoral artery with its superficial and profunda femoral branches. There is a left aorto-femoral dacron bypass graft. All vessels are markedly affected by ulcerated calcific atheroma. From the lower aorta distally there is extensive pale and brown thrombus filling the vessels, except for the left profunda femoris artery. The dacron graft 20cm in length extends from the anterior aspect of the aorta, just below the inferior mesenteric artery, to the distal portion of the left femoral artery.
Diagnosis: Left aorto-femoral bypass.

CASE 22476
The patient was a woman aged 70 who developed rheumatic fever at the age of 21. Little further history was available except that she was said to have had episodes of heart failure treated with digitalis, quinidine and diuretics. At her last admission the BP was 120/90 and the pulse rate 60/minute with atrial fibrillation. The JVP was raised and there was gross pitting oedema of the sacrum and lower limbs. The heart was enlarged and systolic and diastolic murmurs were audible, maximal in the mitral area. The liver was large and pulsating. Her condition slowly deteriorated and she died 5 days later. At post-mortem the heart weighed 475gm.
The specimen consists of the heart. The left atrium is greatly dilated. The mitral valve and chordae tendineae are thickened and fibrotic. There is marked calcification and endothelial roughening of the posterior wall of the left atrium over a large area extending upwards from the mitral valve. The other chambers are also dilated.
Diagnosis: Chronic rheumatic mitral valve disease.

CASE 22504
The patient was a man aged 60 who had suffered a myocardial infarction 3 months previously. At his first admission the BP was 160/120 and ECG showed antero-septal infarction. Some hours later there was an episode of ventricular tachycardia with hypotension and unconsciousness. This was treated with intravenous lignocaine and defibrillation. Thereafter he was febrile and a pericardial friction rub was heard for some days.
On the 11th day a loud pansystolic murmur was audible, and rupture of the ventricular septum was diagnosed. Cardiac failure ensued which was treated with digoxin and diuretics. He was discharged after a month in hospital but was readmitted a few days later with further severe chest pain. The JVP was raised 2cm and the apex beat was in the 6th space in the anterior axillary line. A loud pansystolic murmur could be heard all over the anterior chest, maximal at the left sternal edge. He improved after some days on digitalis and diuretics but on the 10th day suddenly became cold and pulseless. ECG showed sinus bradycardia and he was transferred to the Coronary Care Unit, where hypotension and multiple ectopic beats culminated in ventricular fibrillation from which he could not be revived.
The specimen is the heart opened to display the left ventricle and atrium. There is a large antero-septal infarction involving the apex with considerable thinning and some aneurysmal bulging of the wall. A large area of the septum is involved indicated by subendocardial fibrosis and a metal arrow has been placed through a rupture in the septum, to emerge in the cavity of the right ventricle. Some antemortem thrombus surrounds the rupture in the left ventricle. From the right side (on the reverse of the jar), fibrosis of the inferior portion of the septum is more evident and there is a small aneurysmal dilatation about 2cm in diameter at the extreme tip of the right ventricle. This does not communicate with the left side of the heart (an artefactual hole produced by the dissection is visible).
Diagnosis: Myocardial infarction with ruptured interventricular septum.

Comment: The thinning and fibrosis of the infarct indicate that it is not very recent and are in keeping with its age of 3 months.

CASE 22620
The patient was a man aged 62 who was admitted with acute severe chest pain. ECG suggested recent anterior myocardial infarction, which was confirmed by a rise in ESR and of serum enzyme levels. He developed congestive cardiac failure with many ventricular extrasystoles and died on the 14th day. At post-mortem a haemopericardium from rupture of the infarct was found.
The specimen consists of the heart showing pericardial congestion and fibrinous exudate. The left ventricle reveals thinning, stretching and patchy pallor of the apical region of its wall with overlying mural thrombus. The infarct has ruptured through to the exterior anteriorly.
Diagnosis: Recent myocardial infarction with aneurysmal bulging, mural thrombus, pericarditis and rupture.

CASE 22975
The patient was a man aged 51 who was admitted with an 11-day history of breathlessness and central constant crushing chest pain. He had a cough with a little sputum, but no haemoptysis. He smoked 20 cigarettes/day and had recently lost weight. Chest x-ray showed a large right hilar mass and bronchoscopy and biopsy confirmed extensive mediastinal infiltration from a primary bronchogenic carcinoma. Radiotherapy was given with little effect. A malignant pleural effusion developed and he died 12 days after admission. At post-mortem there was a massive right hilar carcinoma with malignant infiltration of the pleura and invasion of the pericardium. The pericardial sac contained 200ml of turbid bloodstained fluid. Histology showed highly anaplastic carcinoma with gross pleomorphism and many giant cells.
The specimen is the heart showing extensive nodular infiltration of the epicardial fat by pale tumour. There is a small area of haemorrhagic fibrinous exudate over the lateral surface of the left atrium.
Diagnosis: Malignant infiltration of the epicardium.

CASE 23060
The patient was a woman aged 73 with a four-day history of epigastric pain. The pain moved to the centre of the chest and was associated with sweating and mild dyspnoea. On examination there was mild right and left cardiac failure and a systolic murmur. ECG was consistent with antero-septal infarction. She died suddenly on the fourth hospital day. At post-mortem the pericardial cavity was filled with fresh blood.

The specimen consists of the anterior half of the heart that has been divided in the coronal plane. A large oblique rupture about 3cm in length can be seen on the anterior wall of the left ventricle a little distance above the apex. On the reverse side there is a large mass of solid antemortem thrombus on the endocardial surface. Blotchy yellow discolouration is noted on the left side of the muscular septum.
Diagnosis: Ruptured myocardial infarct.

CASE 23066
The patient was a 75-year old man who was diagnosed as having multiple myeloma with amyloidosis 8 years previously (1963). He was treated with Melphalan in 1966 but developed aplastic anaemia due to this drug in 1971. He died from septicaemia. At post-mortem there was macroglossia and the heart weighed 605gm. Histology showed masses of amyloid among the muscle fibres of the left atrial wall and in a layer beneath the endocardium. There was no amyloid deposition in the liver, spleen or kidneys.
The specimen shows the opened left ventricle and atrium. There are small waxy patches scattered on the endocardium of the left atrium, typical of amyloid infiltration. The left ventricular wall is mildly thickened. The mitral valve shows focal nodularity.
Diagnosis: Amyloidosis of the heart.
What type of amyloid would this be? Why? This will be primary amyloid (composed of lambda type immunoglobulin light chains) as occurs in some cases of multiple myeloma. The light chains are produced by the malignant plasma cells.

CASE 23077
The patient was an aboriginal labourer aged 26 who had rheumatic fever at the age of 16 years, with recurrences at 18 and 21 years. On his last admission there was tachycardia, great enlargement of the heart, a murmur of aortic incompetence, unilateral pulmonary oedema (right) and congestive cardiac failure. There were also several episodes of epigastric and lower sternal pain that were presumed to be angina. He died unexpectedly a month after admission. At post-mortem the heart weighed 725g.
The specimen consists of an enlarged heart. The left ventricle is markedly dilated and the wall thickened. The aortic valve is of very large circumference and the valve cusps thickened. The free margins are more thickened in keeping with the effects of incompetence. There is some fibrotic thickening of the anterior leaflet of the mitral valve and chordae tendineae. The right atrium is dilated. There is patchy intimal thickening of the aorta above the aortic valve and there is similar longitudinal wrinkling and scarring of the aorta in its thoracic portion.
Histology of the aorta showed intimal fibrosis and thinning of the media with irregular destruction of the elastic laminae.
Diagnosis: Chronic rheumatic aortic and mitral valve disease and aortitis.
Comment: The appearances suggest that the aortic incompetence is due to dilation of the aortic valve ring secondary to aortitis rather than chronic rheumatic valve damage. Rheumatic fever may cause aortitis.

CASE 23171
The patient was a man aged 85 who had a prostatectomy at the age of 76. He was in controlled congestive cardiac failure. On his final admission investigations showed chronic renal failure with acute pre-renal failure. His deterioration was due to an episode of severe vomiting and diarrhoea. He failed to respond to rehydration and died on the 5th hospital day.
The specimen consists of the heart and first few cm of aorta. The aorta is dilated and shows gross calcific atherosclerosis. There is marked fibrinous pericarditis affecting the entire epicardial surface.
Diagnosis: Fibrinous pericarditis (uraemic).

CASE 23191
The patient was a man aged 69. Calcific aortic stenosis was discovered 4 years previously. A little later there was an episode of severe angina, and ECG showed left ventricular strain but no infarct. Nine months before his death he was admitted for investigation of persistent low-grade fever, malaise and night sweats. The ESR was 46. No cause was found and he was discharged, but he continued to lose weight and was finally admitted for a more intensive search for a suspected malignancy. The only significant findings were a persistently high ESR of 100 and raised IgG and IgM. Bone marrow studies were consistent with chronic infection or occult neoplasm. There was no myeloma. Blood cultures were repeatedly negative. He died after some 6 weeks in hospital. At post-mortem the spleen was large and soft (weight 350gm) and contained several infarcts. The liver was congested.
The specimen is the heart and ascending aorta opened to display the left ventricle and the aortic valve. The aortic valve cusps show old fibrous thickening with patchy calcification. Irregular friable vegetations are also present on the valve leaflets. There is focal calcification of the anterior leaflet of the mitral valve but the mitral chordae tendineae appear normal. The left ventricular wall is mildly thickened.
Diagnosis: Subacute infective endocarditis.

CASE 23255
The patient was a man aged 39 who presented with a history of increasing dyspnoea over the previous month. He had a slight cough for three months and had lost 6.4kg in weight during the last month. He had noted that his urine had been dark for three weeks before admission. He was gaunt, sick looking, with both peripheral and central cyanosis. The pulse rate was 124, the BP 140/95 and the respiratory rate 30. The

trachea was in the midline. The chest was over-inflated and the percussion note was resonant. A few crepitations were heard at both lung bases. A large number of non-tender mobile skin nodules were present and the liver was enlarged, firm and irregular. A chest x-ray showed multiple fluffy opacities throughout both lung fields. Biopsy of a pigmented nodule on the right side of the chest showed a malignant melanoma. Microurine examination revealed numerous histiocytes that contained melanin. The patient deteriorated rapidly and died five days after admission. At post-mortem the pericardial sac contained about 100ml of greenish fluid. Melanoma deposits were found in almost all of the organs studied.
The specimen consists of the heart. A large number of pigmented tumour deposits ranging in size up to 7mm are visible scattered throughout the myocardium of all chambers and on the endocardial and pericardial surfaces of the heart.
Diagnosis: Metastatic melanoma.

CASE 23298
The patient was a schizophrenic male vagrant aged 42 who was admitted to hospital from the Adelaide Gaol. He had been unemployed for 6 weeks and in that time had walked from Shepparton, Victoria, to Renmark, South Australia, a distance of more than 640km, without removing his boots. This resulted in patches of ischaemic gangrene on the toes of both feet. On admission he was febrile (39.4^{o} C), the BP was 160/90, the pulse 104 and irregular from ventricular extrasystoles. The heart was enlarged and there was a systolic murmur in the aortic area radiating to the neck. The liver was palpable 3cm below the costal margin. The spleen was not palpable. There was microscopic haematuria and a rose-coloured rash on the trunk. The haemoglobin was 8.1g/dL, leucocyte count 13,000, reticulocytes 3.2%, ESR 35mm. Blood cultures grew *Streptococcus viridans*. On the 6th day there was neck stiffness and lumbar puncture yielded xanthochromic fluid containing 2500 erythrocytes/cubic mm. Two days later there was right hemiplegia affecting the face, arm and leg. His condition fluctuated and on the 13th day he became comatose and died 2 days later. At post-mortem there was an intracerebral haemorrhage resulting from rupture of a mycotic aneurysm on the left middle cerebral artery (see specimen 23298 in the CNS section of the museum).
The specimen consists of the heart opened to display the left ventricle and ascending aorta. The aortic valve cusps are a little thickened and there is fusion across one of the commissures. Nodular vegetations are present on the under surfaces of the cusps which in one area extend down onto the endocardial surface beneath the valve. Another vegetation is present on the under surface of the adjacent anterior mitral leaflet which is mildly thickened and there is fibrous thickening and shortening of the chordae tendineae. The cavity of the left ventricle is dilated. The underlying fibrous thickening of the aortic and mitral valves is strongly suggestive of chronic rheumatic valve disease.
Diagnosis: Infective endocarditis.
What risk factors does this patient have for infective endocarditis? The fibrosis of both mitral and aortic valves is strongly suggestive of chronic rheumatic valve disease. Gangrene of the toes may also have provided a site of entry for bacteria into the blood (although *Strep. viridans* usually comes from the throat).
Explain the relationship between the clinical features and the pathological process demonstrated in the specimen.
-the patient had probably had rheumatic fever as a child with subsequent scarring developing in the aortic and mitral valves.
-gangrenous toes could have been the source of a bacteraemia (although *Strep. viridans* usually comes from the throat) with subsequent colonization of the abnormal valves and development of infective endocarditis. (Alternatively the gangrene could have arisen due to emboli from the heart).
-the high temperature, pulse rate, ESR and white cell count are related to sepsis and subsequent inflammation, mediated by release of inflammatory mediators.
-the low haemoglobin may be related to chronic disease, depending on the duration of the endocarditis. (Alternatively it could be dietary iron or folate etc deficiency in a vagrant).
-the ventricular extrasystoles could just be incidental or they could indicate myocardial ischaemia, in this case possibly due to embolism.

-the systolic murmur may relate to a degree of underlying aortic stenosis 2ndary to the chronic valve damage and commissural fusion. The vegetations may have contributed but they are probably not large enough on their own to have caused it.
-the clinically enlarged heart and liver may be secondary to heart failure.
-the haematuria and rash are probably immunologically mediated (glomerulonephritis in kidney), as can happen in infective endocarditis. Alternatively, the haematuria could be caused by microemboli and infarction in the kidney.
-neck stiffness with high rbc and xanthochromia of the CSF suggest subarachnoid haemorrhage that may have occurred from rupture of the mycotic aneurysm.
-the hemiplegia could have been caused either by a cerebral infarct caused by an embolism from the heart lodging in a cerebral artery, or (as indicated at autopsy) to rupture of a mycotic aneurysm which arises from a septic embolus containing bacteria lodging in an artery and causing inflammation and damage to the wall.

CASE 23335
The patient was a woman aged 61 who was admitted to hospital for the removal of a rodent ulcer (basal cell carcinoma) from the left lower eyelid. She died suddenly 24 hours after this minor operation. At post-mortem the pericardial cavity was filled with fresh blood originating from a ruptured posterior myocardial infarction.
The specimen consists of the greater part of the aorta. There is a transverse intimal tear 2cm in length and 1cm in width on the postero-lateral aspect of the aorta just beyond the origin of the left subclavian artery. It has smooth endothelialised edges and some old antemortem thrombus can be seen within. Extending downwards for about 6cm from this tear is a short dissection filled with old pale thrombus. This is best seen on the left of the specimen. Immediately below this sac is a round hole, which is the endothelialised entry tear of a 2nd dissection extending downwards to the level of the renal arteries. The hole has been opened to display the underlying endothelialised dissection track that contained unclotted blood at post-mortem.
Diagnosis: Two old aortic dissections.
No history was obtained to suggest when these two episodes of dissection or the myocardial infarct occurred.

CASE 23348
The patient was a 78-year old woman who had suffered from angina pectoris for four years before her death. During the four days before her final admission she had experienced many episodes of chest pain, similar to her anginal pain. Some of these had had a more protracted duration. Admission was brought about because of anterior chest pain of nine hours duration radiating down both arms. Physical examination on admission revealed a regular rhythm with BP of 160/90. A fourth heart sound was audible and an early systolic murmur was present. She complained of further chest pain on the fourth and fifth hospital day. This was associated with a fall in blood pressure and the development of signs of biventricular cardiac failure. The murmur also became more intense. She died on the 6th hospital day.
The specimen consists of the heart. There is some mottling of the posterior wall of the left ventricle with congestion of the overlying epicardium in keeping with infarction. In addition the papillary muscle of the posterior cusp of the mitral valve has ruptured.
Diagnosis: Myocardial infarction with ruptured papillary muscle.
Which coronary artery would you expect to be occluded in this case? The right coronary artery.
This patient had episodes of chest pain of increasing duration occurring on and off for 4 days before going on do develop a myocardial infarction. What clinical term would you give to this scenario? Unstable or crescendo angina.
What would be happening in the coronary artery supplying this area over this time? It is likely to show a fissured atherosclerotic plaque with overlying thrombus that may or may not be occlusive.
Comment: The pain of unstable angina varies as the plaque is unstable and narrowing of the artery varies. The plaque has fissured and thrombus forms. However thrombus is not static – it may lyse partially, then

develop some more, changing over time, in some cases eventually occluding the artery for long enough to cause an infarction.
The early systolic murmur heard on admission in this patient may have been due to papillary muscle dysfunction as a result of ischaemia. Certainly the change in character of the murmur can be attributed to rupture of this muscle. Although some necrosis of this papillary muscle is common in posterior myocardial infarction, actual rupture is rare.

CASE 23551
This patient was a woman aged 59 in whom a carcinoma of the upper lobe of the left lung had been discovered 15 months previously. She was given radiotherapy. At her final admission there was massive haemoptysis, hematemesis and melena that were treated by massive transfusion, but she failed to improve and died. At post-mortem there was a rounded lobulated primary carcinoma 4cm in diameter in the lingular segment of the left upper lobe on its medial side. The tumour was directly adherent to the adventitia of the aorta. There were metastases in mediastinal and abdominal lymph nodes, the adrenals, kidneys and liver.
The specimen consists of the arch and descending aorta with attached portions of the oesophagus and trachea. A mass of pale tumour lies between these structures and erodes the aorta with an intimal rupture in the distal arch extending directly by a fistulous track through the tumour tissue to the lumen of the oesophagus. A black probe has been inserted in the fistula. The tumour also erodes the mucosa of the trachea at its bifurcation, seen on the back of the jar.
Diagnosis: Lung carcinoma causing aorto-oesophageal fistula
Histology showed an oat-cell (small cell) carcinoma.

CASE 24132
The patient was a hypertensive woman aged 58. She was admitted with a myocardial infarction from which she seemed to be making a good recovery until 3 days after admission, when the BP fell to 75/65, the pulse rate rose to 140 and a right ventricular heave with a systolic murmur and thrill appeared. A diagnosis of septal rupture was made but surgery was contra-indicated. She was therefore treated conservatively, but the cardiac output gradually fell and she died in a hypotensive state with renal failure.
The specimen is the heart in which the left ventricular wall has been cut to reveal a defect in the interventricular septum. The apical and anterior portions of the wall of the left ventricle appear slightly thinned and mottled consistent with recent myocardial infarction. Near the apex, in the anterior part of the muscular interventricular septum there is an oval defect of 1cm diameter where the septum has ruptured.
Diagnosis: Myocardial infarct with ruptured interventricular septum.

CASE 24229
The patient was a man aged 59 who was admitted with severe central chest pain radiating to the back. The ECG suggested postero-inferior transmural infarction and the serum enzyme levels were markedly raised. On the 4th day his condition deteriorated markedly, with increasing breathlessness, tachypnoea, cyanosis and hypotension. A high-pitched harsh systolic murmur was audible throughout systole at the left sternal edge and the apex. Oxygen and diuretics produced some improvement. Two weeks later there was further sudden severe breathlessness and shock and he died that same day.
The specimen is the heart opened to show the left ventricle. There is blotchy pallor and discolouration of the posterior wall of the left ventricle over a large area. Fragments of antemortem thrombus adhere to the endothelial surface of the infarct. A mitral papillary muscle involved in the area of infarction has ruptured and can be seen hanging loose from the chordae tendineae. The posterior papillary muscle appears necrotic, but has not ruptured.
Diagnosis: Myocardial infarction with ruptured papillary muscle.
What problem does rupture of a papillary muscle cause? Acute mitral incompetence, often with cardiogenic shock.

CASE 24277

The patient was a man aged 77 with a history of 3 previous myocardial infarctions. He was admitted with gangrene of several left toes, which were amputated. After the operation the remainder of the foot also became gangrenous. He died suddenly after 2 weeks in hospital. At post-mortem there was a small old myocardial infarct and severe atherosclerosis of the aorta and its branches.
The specimen is the abdominal aorta and the iliac arteries. There is a long bilocular aortic aneurysm 12cm in length and 5cm in breadth extending downwards from the level of the renal arteries. There is much antemortem thrombus in this aneurysm but a wide channel remains in the centre. The iliac arteries are also dilated and partly occluded by antemortem thrombus.
Diagnosis: Atheromatous aorto-iliac aneurysm.
From what is this patient likely to have died? In the absence of any other information, a likely scenario is that he died from a fatal cardiac arrhythmia as a consequence of ischaemic heart disease.

CASE 24279
A 67-year old man developed severe pain in the back and abdomen. He was noted to be severely shocked (systolic BP 40 mmHg) and a pulsatile abdominal mass was felt. Laparotomy disclosed a ruptured abdominal aortic aneurysm. This was resected and replaced with a dacron graft. The colon became gangrenous and was resected at further operation the next day. The patient became anuric and died 2 days after admission.
The specimen consists of the abdominal aorta and iliac arteries. The aorta shows severe atheroma. A corrugated dacron graft, the surface of which is covered with post-mortem clot, has been inserted 4cm below the renal arterial origins. The bifurcations of the graft are anastomosed to the external iliac arteries. Surrounding the aortic portion of the graft is the grossly atheromatous remainder of the aneurysm, containing old pultaceous atheroma. Haemorrhage is present externally.
Diagnosis: Dacron graft to abdominal aorta and external iliac arteries.
Why might the colon have become gangrenous? There are a number of reasons, several of which may have contributed: the artery supplying it (doesn't say which part of the colon is involved) may be narrowed by atheroma, the blood supply was impaired during the period of shock, emboli from the aorta may have dislodged during the operation and/or it could relate to surgical misadventure.

CASE 24364
The patient was a man aged 73 who had occasional angina on effort for 5 years. For the 5 weeks before admission there had been considerable increase in the pain, with attacks of nocturnal paroxysmal tachycardia. Chest x-ray suggested aortic dissection. After some days he began to cough up blood in increasing amounts and he died suddenly 4 days later. At post-mortem there was much free blood in the left pleural space.
The specimen consists of the aorta and the left lung. About 4cm distal to the left subclavian artery is a transverse endothelialised intimal opening which leads to a false aneurysmal sac some 5cm in diameter, almost completely filled with old thrombus. This aneurysm was adherent to, and ruptured into, the medial aspect of the left lung, which shows massive acute haematoma in the apical segment of the lower lobe, extending downwards into the posterior basal segment. In addition there has been rupture into the pleural cavity in which haematoma can be seen. **Diagnosis:** False aortic aneurysm with rupture into the left lung.
Comment: The false aneurysm has probably been present for at least the 5 weeks before death, eroding into lung 4 days before death. It probably resulted from an aortic dissection. Trauma is another possible cause but no history of this is given.

CASE 24428
This man aged 60 was admitted with a 3-hour history of chest pain radiating to both arms, accompanied by vomiting. He had been hypertensive for 5 years and diabetic for 8 years. There had been episodes of angina pectoris. While in hospital cardiac failure progressed. On the 7th day there was an extension of the infarct and he became anuric. He died suddenly 4 days later, on the 11th hospital day. At post-mortem the

pericardial sac was filled with blood. The left coronary artery was narrow but patent but the right coronary artery was occluded by thrombus 3cm from its origin.
The specimen is a transverse slice of the heart showing mottling and pallor of the posterior wall of the left ventricle. There is some interstitial haemorrhage and a small defect is visible through the top of the jar at the point of external rupture. The posterior papillary muscle is pale and is involved in the infarct and there is overlying fibrinous pericarditis.
Diagnosis: Ruptured myocardial infarct.

CASE 24459
This 71-year old male alcoholic was admitted with an inferior myocardial infarction and died in severe biventricular failure.
The specimen shows a large heart with left ventricular dilation and hypertrophy. The infarct is difficult to see bit there is slight haemorrhage and mottling in the myocardium posterolaterally. The aortic valve is bicuspid, thickened and calcified, with areas of calcification extending into the top of the interventricular septum and mitral valve. The mitral valve otherwise does not appear fibrotic. The aortic valve orifice was grossly stenotic, admitting only the tip of one finger.
Diagnosis: Calcific degenerative changes in a bicuspid aortic valve and myocardial infarction.

CASE 24585
The patient was a diabetic man, aged 62 at his death in February 1973,who had had many admissions for arterial disease and ischaemic heart disease. A left femoral thrombosis occurred in 1961. A bypass graft was performed, but this too thrombosed after 4 months and the left leg was amputated above the knee. In 1963 intermittent claudication occurred in the right leg and arteriogram showed that the right femoral artery was occluded. Episodes of ischaemic heart disease with congestive failure and pulmonary oedema occurred in 1971-2. In January 1973 there was transient giddiness. A month later he developed severe giddiness with falling to the left and vomiting. Weakness progressing rapidly to total paralysis of the right arm and right leg was noted. There was anaesthesia to pain and temperature of the right half of the body and the left side of the face. The BP was 240/110. He was drowsy and his speech was slurred. The cough reflex was depressed and sputum accumulated. The pupils were equal and reacted normally. There was no obvious weakness of the 3rd, 4th, 5th and 6th nerves but there was a left upper motor neurone 7th weakness. He was deaf in the left ear and there was gross rotatory nystagmus. The gag reflex was depressed, the uvula deviated to the right and the protruded tongue to the left. There was difficulty in swallowing. Sweating was absent on the left forehead and face. He died of respiratory paralysis and bronchopneumonia on the 3rd day in hospital (21 February 1973). At post-mortem a lateral medullary syndrome on the left side was found, corresponding to the neurological deficit. The heart weighed 560gm, and there was hypertrophy of the left ventricle.
The specimen is the aorta with its iliac branches and the right kidney. There is gross ulcerating atherosclerosis of the entire specimen. The common iliac trunks are dilated, the left containing old antemortem thrombus. Note the extent of the yellow pultaceous lipid filled atheroma, much of which is ulcerated, in the aortic wall. The right kidney is small and there is a cyst 3cm in diameter at its upper pole.
Diagnosis: Gross atherosclerosis of the aorta and its branches.
Why is the kidney small? It is probably chronically ischaemic from atherosclerotic narrowing of its artery or its origin. Tubules and glomeruli become atrophied and there is microscopic interstitial scarring.
Correlate the patient's neurological signs and symptoms with the site of the CNS lesion found at autopsy.
CASE 24646
The patient was a man aged 36 who had an anterior resection of the colon for a carcinoma of the recto-colonic junction. Re-operation was necessary next day to control haemorrhage. On the 12th day he developed a faecal fistula and a right subphrenic abscess, which was drained. He then became jaundiced and developed pneumonia. On the 20th day a transverse colostomy was performed and later an abdomino-perineal resection. Massive haemorrhage again complicated the post-operative period. On the 30th day a

small bowel fistula developed and the wound broke down. A week later blood culture grew *Candida albicans* and he was treated with Amphotericin B. A few days later a systolic murmur maximal in the pulmonary area was heard. Subsequently tracheostomy was performed and the right subphrenic abscess was again drained. By this time there was obstructive jaundice and he became comatose and died. At post-mortem multiple foci of abdominal sepsis were present and there were multiple large white metastatic nodules in the liver. The heart weighed 370gm.
The specimen is the heart opened from behind to display the cavities of the left and right sides. Large friable crumbling vegetations are present on the tricuspid valve. Each measures about 2.5cm in diameter. Some smaller bead-like vegetations are present on the line of closure of the valve. The underlying valve and chordae tendineae appear normal. The cavity of the right ventricle is somewhat dilated but its muscle is not obviously hypertrophied.
Diagnosis: Candida endocarditis of the tricuspid valve.
Histology showed branching hyphae of Candida in the vegetations, embedded in fibrin and inflammatory exudate. The liver nodules were secondary adenocarcinoma.

CASE 24651
The patient was a man aged 80 who was admitted with a resolving pneumonia that had been present for 2 weeks. He was known to be hypertensive and on examination was breathless, wasted and slightly demented. The BP was 150/70 and systolic murmurs were audible in the mitral and aortic areas. Chest x-ray showed an enlarged heart, aneurysmal dilatation of the ascending aorta and evidence of resolving pneumonia. He slowly deteriorated until his death after 3 weeks in hospital. Serological tests were positive for syphilis.
The specimen consists of the heart and the greater portion of the aorta. The left ventricle is slightly dilated but its myocardium is not thickened. The aortic valve cusps are a little thickened and scarred. The mitral valve appears normal. A large transverse patch of atherosclerosis is present 2cm above the aortic valve on the posterior wall of the aorta. Above this there is marked dilatation of the ascending and transverse portions of the arch, including the origins of the great vessels. The aneurysm measures about 10x10cm and its wall shows gross calcific and ulcerative atherosclerosis. Similar atherosclerotic change is present throughout the remainder of the aorta, decreasing somewhat in severity in the lower descending aorta.
Diagnosis: Aneurysm of the ascending aorta.
Comment: The aneurysm probably represents the effects of tertiary syphilis. Histology of non-atheromatous areas in the ascending aorta would be necessary to confirm if there was underlying syphilitic aortitis. Atherosclerosis is a very uncommon cause of aneurysms of the ascending aorta.

CASE 24765
The patient was a woman aged 62 who was admitted to hospital with acute myeloid leukaemia. A blowing systolic ejection murmur was readily heard over the back. Her course in hospital was stormy and after a week she developed a gram-negative septicaemia from which she died. At post-mortem there was a small pericardial effusion.
The specimen is the heart and the aorta. A coarctation of the aorta about 6mm in diameter is present at a point 3cm distal to the origin of the left subclavian artery. There is very mild atherosclerosis of the aorta.
Diagnosis: Coarctation of the aorta.

CASE 24884
The patient was a man aged 83 who presented with a 7-hour history of sharp abdominal pain accompanied by anorexia and nausea. A large palpable mass could be felt in the mid-line of the abdomen and plain x-ray showed an aortic aneurysm. He died on the 3rd day after admission. At post-mortem there was a very large retroperitoneal haematoma surrounding both kidneys and suprarenals and extending into the root of the mesentery.

The specimen is the aorta and iliac arteries opened from behind to show the large spheroidal aneurysm measuring 10x12cm extending from a level 2cm below the origins of the renal arteries down to the iliac bifurcation. A thick layer of antemortem thrombus adheres to the inner wall of the aneurysm and haemorrhage is present externally. The left iliac artery is somewhat dilated. There is gross calcific and ulcerative atherosclerosis of the aorta above the aneurysm.
Diagnosis: Ruptured atherosclerotic aneurysm.

CASE 25193
The patient was a man aged 75 who died from a cerebellar infarct and myocardial infarction. At post-mortem the aorta was grossly atherosclerotic throughout.
The specimen consists of the lower 6cm of abdominal aorta and the iliac branches. There is gross ulcerated and calcific atherosclerosis of these vessels. Both internal iliac arteries are dilated and totally occluded by antemortem thrombus. The proximal portion of the thrombus on the left side appears old and is very degenerate and pultaceous.
Diagnosis: Atherosclerosis with thrombosis of internal iliac arteries.

CASE 25211
The patient was a woman aged 83 known to be hypertensive for 10 years (BP 240/120). She was admitted in congestive cardiac failure uncontrolled by digitalis and diuretics. She died on the 5th day. At post-mortem an adenocarcinoma was found in the tail of the pancreas with metastases in the liver. There was a blood stained left pleural effusion and there were recent haemorrhagic infarcts in the lower lobes of both lungs. Both innominate veins were occluded by thrombus that extended to the left jugular vein. The right femoral and right long saphenous veins also contained antemortem thrombus.

The specimen is of the right femoral vein measuring 33cm in length with 17cm of adjacent femoral artery. The femoral vein and its saphenous tributary are occluded by recent antemortem thrombus. Pale laminae of fibrin and platelets stand out against darker areas where red cells predominate. The head of the thrombus is pointed and lies free in the lumen of the vein. The distal portion of the thrombus seems firmly attached to the venous wall. The artery shows intimal atherosclerosis.
Diagnosis: Antemortem thrombus in the femoral vein.
What risk factors did this patient have for deep venous thrombosis (DVT)? How does each predispose to this condition? This patient had a number of risk factors for this condition: pancreatic adenocarcinoma, congestive cardiac failure and quite possibly limited mobility related to severe cardiac failure. Adenocarcinomas of the pancreas probably produce some factor that causes the blood to be hypercoagulable. As the right heart fails in CCF, venous flow is slowed, predisposing to coagulation. Similarly, with limited mobility, the venous flow in the legs is slowed as the muscle pump is not working.
Explain the pathogenesis of the infarcts in the lungs. Why are they haemorrhagic? Portions of thrombus broke off from the thrombus in the femoral vein and travelled to branches of the pulmonary arteries in the lungs, occluding them. Due to CCF, the bronchial arterial supply to the lungs was not sufficient to maintain viability of the areas supplied by the occluded pulmonary arteries so areas of the lung infarcted. The blood from the bronchial arteries haemorrhaged into the infarcted areas.

CASE 25253 (a)
The patient was a man aged 84 who was known to have an abdominal aortic aneurysm for which he had refused surgery. He was admitted because he was bedridden, in gross congestive cardiac failure and was incontinent of urine and faeces. He died on the 2nd hospital day. At post-mortem he was found to be a true hermaphrodite with a left testis, uterus, both tubes and right ovary present. Chromosomal sex of the polymorphs was of the female type. The liver and kidneys were congested. There was active pulmonary tuberculosis.

The specimen consists of the abdominal aorta with its iliac branches, together with both kidneys. There is a long aneurysm of the aorta measuring 13 x 8 x 6cm, extending from the level of the renal arteries to the bifurcation. The rear of the specimen shows a thick layer of laminated antemortem thrombus in the cavity of the aneurysm. The common iliac arteries are also dilated and thrombus is present in a left iliac aneurysm. There is no surrounding haemorrhage to suggest rupture. The kidneys are slightly shrunken and nephrosclerotic.
Diagnosis: Atherosclerotic aneurysms.

CASE 25253 (b)
The specimen is a portion of the abdominal aorta opened to show gross ulcerative and calcific atherosclerosis. In many of the patches the pultaceous atheromatous material is obviously breaking down.
Diagnosis: Atherosclerosis.

CASE 25261
The patient was a man aged 59 who presented with sudden chest pain accompanied by sweating and breathlessness. That same day he developed ventricular fibrillation from which he was resuscitated 3 times. Later, venous thrombosis occurred in the legs and he died on the 18th day from a further episode of ventricular fibrillation.
The specimen consists of a horizontal slice through the ventricles. The anterior wall of the left ventricle and anterior two thirds of the interventricular septum show transmural yellow discolouration and congestion in keeping with recent infarction. A small amount of pale thrombus adheres to the overlying endocardium. There is no evidence of associated pericarditis.
Diagnosis: Antero-septal myocardial infarction**.**
Which artery supplies the affected area? Left anterior descending coronary artery.

CASE 25272
The patient was a woman aged 22 with a history of congenital heart disease. She also had a cleft palate that was repaired at the age of 3 years. A thoracotomy was performed in Melbourne when she was 9, but no record is available of what was found. Four weeks before her last admission she developed an upper respiratory infection which cleared after a few days, but left her increasingly breathless and weak. On the day of admission she had a grand mal fit. Examination showed central and peripheral cyanosis with severe clubbing of fingers and toes and polycythaemia. The haemoglobin was 23.6g/dL, PCV 73%, ESR 0. The heart was enlarged with a double impulse at the apex and an early systolic murmur in the pulmonary area. ECG showed right atrial enlargement with right ventricular hypertrophy and strain. Chest x-ray showed cardiac enlargement, particularly of the right ventricle but also of the right atrium and the pulmonary outflow tract. Pulmonary vascular markings were increased. A provisional diagnosis of Eisenmenger's syndrome with patent ductus arteriosus was made. While in hospital she continued to be severely breathless and slow digitalisation and venesection were begun. She died on the 8th day.
The specimen is of the heart and the right lung. The cavities of the left and right ventricles are dilated and the right ventricular wall is considerably thickened. A large opening in the muscular interventricular septum is indicated by the green arrow. This VSD measures 1.5 x 1cm. There is no overriding of the aorta and a patent ductus arteriosus almost 1cm in diameter, indicated by the black arrow, joins the aorta to the left pulmonary artery near its origin. No vegetations are present on the valves that appear normal. The foramen ovale is closed (not well seen). The lung shows patchy congestion and a small haemorrhagic infarct at the apex of the lower lobe. **Diagnosis:** Patent ductus arteriosus with ventricular septal defect.
Histology showed marked intimal and medial thickening of small branches of the pulmonary arteries in the lung in keeping with pulmonary hypertension. In the lung parenchyma there was interstitial fibrosis and a few small organising infarcts.
Explain why this patient has developed right ventricular hypertrophy, central cyanosis and polycythaemia. With a large patent ductus and VSD, postnatally there is initially a L->R shunt (systemic/LV pressure > than pulmonary/RV pressure). This results in increased blood flow through the lungs. In

response to increased pulmonary blood flow, the pulmonary arterioles constrict and hypertrophy resulting in pulmonary hypertension. Right ventricular hypertrophy develops to improve pumping of blood into the lungs against this increase in afterload. However, right ventricular and pulmonary pressures eventually become higher than left ventricular pressure and the shunt reverses (now R -> L) and blood bypasses the lungs, allowing deoxygenated blood to pass into the systemic circulation causing cyanosis. Polycythaemia develops as the patient is chronically hypoxic, and more erythropoietin is released from the kidneys that stimulates increased red blood cell production in the bone marrow in an attempt to increase the oxygen carrying capacity of the blood.
What is Eisenmenger syndrome? This is the combination of clinical features (including central cyanosis and fatigue) resulting from the pulmonary hypertension and shunt reversal in an adult with congenital heart diseases that cause chronic L -> R shunts.

CASE 25356
The patient was a man aged 74 who had had a myocardial infarction 3 years previously. He was admitted with sudden onset severe retrosternal and left sided chest pain, sweating and nausea. The ECG showed a developing pattern of anterior infarction. He became breathless and moderate pulmonary oedema developed, followed by a fall in blood pressure and death on the 14th day.
The specimen is a slice through the ventricles. The scar of a massive old posterior infarction is visible in the posterior myocardium and the posterior part of the interventricular septum. Blotchy pallor and congestion mark the site of a recent antero-septal infarction. A large mass of laminated thrombus bridges the cavity between the two infarcts.
Diagnosis: Old and recent myocardial infarctions.
What would be expected to be seen microscopically in each infarcted area? The posterior infarct would demonstrate mature fibrous tissue with scattered capillaries and fibroblasts. The recent infarct, being of 14 days of age, would demonstrate necrotic muscle being phagocytosed by macrophages and surrounded by vascular and cellular granulation tissue depositing collagen.

CASE 25372
The patient was a man aged 82 who suffered sudden severe central chest pain at rest, with sweating but no vomiting. The pain did not radiate. There had been no previous episode. The BP was 120/80 initially and the pulse 105 and regular. After a few hours the BP fell and the pulse rate rose and he died in what was considered to be cardiogenic shock. At post-mortem there were about 300ml of fresh blood in the pericardial cavity and there was a rupture on the anterior wall of the left ventricle 4cm above the apex. An antemortem thrombus was present in the trunk of the left anterior descending coronary artery.
The specimen consists of a transverse slice through both ventricles. There is a small area of haemorrhagic infarction in the anterior wall of the left ventricle, through which blood has penetrated to the exterior.
Diagnosis: Ruptured myocardial infarct.

CASE 25423
The patient was a man aged 76 who had suffered a myocardial infarct 5 years previously. On his last admission there was a 2 hour history of slurred speech and left hemiparesis. His condition steadily deteriorated and he died on the same day. He was diabetic and had been taking oral hypoglycaemic agents.
The specimen consists of the heart opened to display the left ventricle. There is marked thinning and scarring of the apico-septal region of the left ventricle in keeping with previous old infarction with slight bulging some 6cm in diameter. The endocardial aspect is fibrotic. The left ventricular myocardium is not obviously thickened elsewhere. The mitral and aortic valves appear normal.
Diagnosis: Old apical myocardial infarction with aneurysmal bulging.

CASE 25472

This woman aged 68 had a long history of mitral valve disease, which was detected during her first pregnancy. Valve replacement was performed 8 years before her death, but afterwards she suffered increasing congestive cardiac failure. When last seen at the cardiac clinic there was hepatomegaly, splenomegaly, tricuspid incompetence and moderate congestive cardiac failure. She was admitted but died 2 days later. At post-mortem the heart was very large and weighed 620gm.
The specimen of heart shows a greatly dilated left atrium. There is some irregular scarring of the left atrial endocardium above the artificial mitral ring. The valve prosthesis is well situated and it has become partially covered by fibrous tissue and endocardium. There is no evidence of infective vegetations. The left ventricle is not obviously dilated or its wall thickened, but its endocardium shows patchy fibrosis. The aortic valve appears normal. The right atrium and ventricle are also dilated. The tricuspid valve is not visible because of the way the specimen is mounted.
Diagnosis: Mitral valve replacement.
What is the most likely cause of this patient's mitral valve disease? Rheumatic fever.

CASE 25488
This man aged 69 was first admitted to the RAH with myocardial infarction 6 months before his death. Congestive cardiac failure developed which responded well to treatment. At his last admission there was severe chest pain with acute pulmonary oedema, central and peripheral cyanosis and acute dyspnoea. The JVP was elevated 5cm, ECG showed left bundle branch block, and chest x-ray a large heart and a right pleural effusion. He died the next day.
The specimen consists of the heart opened to show the left ventricle that is moderately dilated. An apical aneurysm 6cm in diameter is present. Its wall is thin (about 4mm) and fibrous. Pale antemortem thrombus almost fills the cavity of the aneurysm. The posterior myocardium appears slightly congested, possibly representing a recent infarction suggested by the history.
Diagnosis: Old apical myocardial infarction with aneurysm.

CASE 25494
The patient was a man of 37 whose illness lasted 10 months. In the 3rd month there was pericarditis with effusion, which settled within two months. Six months later he was still tired and unwell, and x-ray showed some small circular opacities in both lungs. These rapidly increased in size during the next month, and open thoracotomy and biopsy showed angiosarcoma. Biochemical investigations showed moderate disorder of liver function, but on liver biopsy there was only congestion with non-specific reactive changes. He died a few days later.
The specimen consists of the heart opened from behind. The right atrium is greatly enlarged, the cavity almost entirely filled with a lobulated tumour arising from the lateral and superior walls and measuring 8cm in diameter. The cut surface is divided by coarse fibrous septa and shows areas of necrosis and haemorrhage. The tumour partly obstructs the tricuspid valve. The interatrial septum is thickened. The right ventricle is not dilated or hypertrophied. The left ventricle, left atrium and the mitral valve also appear within normal limits.
Diagnosis: Angiosarcoma of the right atrium.
What is the likely cause of the liver congestion? Tumour related tricuspid stenosis or incompetence causing right heart failure.
What is the commonest primary tumour of the heart? Atrial myxoma (benign).
Comment: The tumour in this specimen is macroscopically seen to be infiltrative and necrotic so is obviously malignant. Histology showed the tumour cells to be spindled in shape and very pleomorphic and they formed numerous capillary channels in the fashion characteristic of angiosarcoma.
Primary neoplastic lesions of the heart are uncommon. The most common is the benign myxoma. These tend to arise in the atria and often present with embolic complications from bits of tumour breaking off. Rhabdomyomas are the most frequent primary tumour of the heart in infants and children. Angiosarcoma is the most common primary malignant tumour of the heart.

CASE 25511
The patient was a man aged 63. He presented a month previously with a myocardial infarct, and while recovering from this he had two episodes of pulmonary embolism. He was discharged but returned a few days later with severe breathlessness, central cyanosis, and crepitations at the lung bases which were thought to be due to extension of the myocardial infarct complicated by pulmonary oedema. He did not respond to treatment and died that same day. At his previous admission a cavitating lesion was found in the lower lobe of the right lung. At post-mortem this proved to be a mass of emphysematous bullae.
The specimen consists of the heart opened to display the left ventricle and aorta. The ventricle is dilated and the wall possibly mildly thickened. The opening of a large aneurysmal sac can be seen posteriorly. The anterior papillary muscle and its chordae tendineae bridge the opening, which measures 6x5cm, which opens into a large thin walled unilocular cavity some 8cm in diameter at the posterior aspect of the interventricular septum. The cavity is free of thrombus. The aortic valve is essentially normal. The right ventricle can be seen on the reverse side of the specimen. It is essentially normal.
Diagnosis: Left ventricular aneurysm.

CASE 25515
The patient was a woman aged 66. Two years previously a squamous cell carcinoma of the left side of the tongue was treated by radon needle insertion and radical dissection of the posterior triangle of the left side of the neck, followed by radiotherapy. At post-mortem the pericardium was tense and the cavity contained 300ml of blood. There was a metastasis in the body of the 2nd cervical vertebra.
The specimen consists of the heart that is enlarged. The visceral pericardium is markedly congested and shows florid fibrinous exudate.
Histology showed pleomorphic poorly differentiated squamous cell carcinoma infiltrating the visceral pericardium.
Diagnosis: Fibrinous pericarditis (from malignant infiltration).

CASE 25559
The patient was a woman aged 70 with a history of angina pectoris and hypertension. On her last admission there was severe chest pain radiating down both arms, accompanied by nausea and vomiting. The BP was 220/80, the pulse 80 and regular, and ECG showed recent left bundle branch block. Enzyme changes were consistent with myocardial infarction. A week later she suffered a cardiac arrest and died. At post-mortem the heart weighed 670gm.
The specimen consists of a horizontal slice through the heart. The posterior and lateral walls of the left ventricle and posterior one third of the interventricular septum show transmural mottled pallor and haemorrhage in keeping with recent infarction. There is some thickening of the left ventricular wall and septum, but the right ventricle is of normal size.
Diagnosis: Recent myocardial infarction.
Histology showed areas of vascular granulation tissue infiltrating between necrotic muscle.

CASE 50335/80
The patient was a man aged 56. He had a 30-year history of episodes of drenching sweats, nausea, dizziness, slurred speech and dull chest ache lasting hours to days. These occurred once a year initially but increased with age. He was found to have ventricular tachycardia during these episodes. He died 5 days after a period of central chest pain. At post-mortem, the left circumflex coronary artery was found to be occluded by atheroma and thrombus.
The specimen is a transverse slice of heart through both ventricles. The right ventricular wall is normal. There is old thinning, scarring and subendocardial fibrosis of the anterior wall of the left ventricle and anterior two thirds of the interventricular septum in keeping with previous infarction in the territory of the left anterior descending coronary artery. There is overlying laminated mural thrombus. In the lateral wall of the left ventricle in the territory of the left circumflex coronary artery is an area of yellow discolouration with surrounding haemorrhage in keeping with recent myocardial infarction.

Diagnosis: Old and recent myocardial infarctions.

CASE 50085/81
The patient was a man aged 53 who underwent an aortic valve replacement.
The specimen is a portion of heart opened to show the left ventricle and aorta. A prosthetic aortic valve covered in thrombus is evident. At post-mortem the suture line between the prosthesis and the aortic ring had broken down around 20% of the circumference. *Staphylococcus aureus* was cultured. The mitral valve is possibly mildly thickened. The left ventricular wall is thickened and shows small patches of fibrosis.
Diagnosis: Infected aortic valve prosthesis.
Why might the left ventricular wall be thickened? The patient may have had aortic stenosis.

CASE 50226/82
This patient had a subphrenic abscess.
The specimen consists of the heart. The pericardial surface is extensively covered in pale strands of fibrin.
Diagnosis: Fibrinous pericarditis (infective).
Comment: Fibrinous pericarditis is classically referred to as 'bread-and-butter pericarditis', it being likened to the buttery surface of two slices of bread that have been buttered, placed together and then pulled apart. The similarity is obvious here.

CASE 50242/82
The patient was a man aged 73 who had had a myocardial infarction 4 years prior to his death and had angina since that episode. He died from a ruptured aneurysm of the abdominal aorta.
The specimen consists of a slice of heart through both ventricles. There is marked fibrotic thinning and subendocardial fibrosis of the anterior wall of the left ventricle with fibrosis in the posterior wall also. These appearances are consistent with old myocardial infarcts.
Diagnosis: Old myocardial infarcts.

CASE 50322/82
The specimen consists of the abdominal aorta opened from behind. The lumen of the distal portion is occluded by laminated antemortem thrombus. The proximal part of the thrombus arises from a small 2cm saccular atherosclerotic aneurysm on the right side (left side of pot). A small area of ulcerated atheromatous plaque with adherent thrombus is seen just proximal to the main thrombus and distal to the origins of the renal arteries.
Diagnosis: Aortic atherosclerosis and thrombus.

CASE 50342/82
The patient, a previously fit man aged 76, was admitted after a short episode of severe central chest pain. He died two weeks after admission after a recurrence of chest pain and the onset of ischaemia of the bowel. At post-mortem, all of the coronary arteries were found to be severely affected with atherosclerosis and there was total occlusion of the right coronary artery.
The specimen consists of a slice through the right and left ventricles. The right and left ventricles have walls of normal thickness. There is circumferential dark mottling of the left ventricular myocardium mainly in a subendocardial location consistent with recent subendocardial infarction. Small scattered areas of pale early fibrosis are also present and the features are in keeping with an infarct of 2 weeks of age.
Diagnosis: Subendocardial myocardial infarction.
What are the causes of subendocardial myocardial infarction?
- severe atherosclerotic narrowing of all main coronary arteries that may cause a circumferential infarct.
- lysis of a thrombus in a single coronary artery with reperfusion such that there is insufficient time for a transmural infarct to develop (infarcts begin subendocardially and then extend outwards) which will cause a regional subendocardial infarct.
- shock leading to reduced perfusion.

- Severe concentric left ventricular hypertrophy

Several factors may contribute together.

CASE 50433/82

The specimen consists of the abdominal aorta with the proximal segment of the iliac vessels. There is a saccular aneurysm present involving the abdominal aorta from 3cm below the origin of the renal arteries to its bifurcation. The aneurysm is filled with laminated thrombus. There is fresh blood clot amongst the tissues outside the aorta. This is a portion of a much larger haematoma. The aorta is severely atheromatous, showing fatty streaks and ulcerated plaques.

Diagnosis: Ruptured atherosclerotic aortic aneurysm.

What clinical signs and symptoms related to this pathology would have been expected in this patient immediately before death? Pallor, tachycardia, sweatiness, hypotension, pulsatile abdominal mass.

CASE 50437/82

The patient was a man aged 85.

The specimen consists of a slice from the apex of the heart. There is fibrotic thinning and slight bulging of the anterior and interventricular left ventricular myocardium at the apex.

Diagnosis: Left ventricular aneurysm.

How has this aneurysm developed? This aneurysm has developed in an area of scarring following previous myocardial infarction. The scarred area does not contract and it often stretches forming an aneurysm.

CASE 50442/82

The patient was a man aged 65 who 5 years before his death was admitted to hospital with a myocardial infarction. Following that episode he experienced increasing angina. Eleven days before his death he was admitted with a story of severe continuous chest pain and an electrocardiogram showed changes indicating recent myocardial infarction. The patient died from pulmonary embolism.

The specimen consists of a horizontal slice of right and left ventricles. The lateral wall of the left ventricle shows an old myocardial infarct indicated by fibrotic thinning of the myocardium. There is also extensive transmural yellow discolouration and congestion of the anterior left ventricular myocardium including the papillary muscle in keeping with recent myocardial infarction. Overlying mural thrombus is present. There are also yellow areas of necrosis in the posterior interventricular septum and right ventricle.

Diagnosis: Recent and old myocardial infarctions.

CASE 50463/82

The specimen consists of the lower thoracic and abdominal aorta with the iliac arteries. The aorta and the iliac arteries have a double lumen due to previous aortic dissection. The dissection arose adjacent to the origin of the left subclavian artery (not present) and has extended down the thoracic and abdominal aorta to involve the iliac vessels. The false channel can be distinguished from the true aortic lumen by the fact that the true lumen contains atheromatous plaques. The coeliac and superior mesenteric arteries can be seen traversing the false lumen that has a smooth endothelialised lining. A re-entry point can be seen in one iliac artery.

Diagnosis: 'Double barrelled aorta' from previous dissection.

What are the risk factors for aortic dissection? The main ones are systemic hypertension and Marfan's syndrome. Others include aortic coarctation and bicuspid aortic valve.

Comment: This pathology could have been present for many years and may not have been symptomatic. Blood would have filled the false lumen during life.

Marfan's syndrome is a genetic disorder of elastic tissue. The aorta histologically demonstrates fragmentation and loss of elastic tissue and accumulation of mucopolysaccharide in the media, a change (incorrectly) termed 'cystic medial necrosis'.

CASE 50473/82
The patient was a man aged 76 who died after prolonged congestive cardiac failure with shortness of breath on exertion, raised JVP and bilateral ankle oedema. He terminally developed deep venous thrombosis with multiple pulmonary emboli. At post-mortem, all the coronary arteries were grossly narrowed by advanced atheroma.
The specimen shows a pale liver with a recognisable nutmeg pattern. The left ventricle is dilated but the wall not significantly thickened. There are patchy small areas of subendocardial fibrosis in keeping with chronic ischaemia.
Diagnosis: Chronic myocardial ischaemia with congestive cardiac failure and nutmeg liver.
You should be able to describe the relationship between these pathological abnormalities and the clinical information given.

CASE 50559/82
The specimen consists of the heart. On the anterior surface are 2 vein grafts. The proximal end of one anastomoses with the aorta, its distal end being anastomosed to the distal left anterior descending coronary artery. This artery has been opened longitudinally more proximally to reveal atherosclerosis. A second graft inserts into the first about 2cm from its proximal end. It courses around the right ventricle, its distal end inserting into the posterior descending interventricular artery on the right side of the heart. The blue sutures are easily visible. Note how small branches of the venous grafts have been tied off.
Diagnosis: Coronary artery bypass grafts.
Comment: Insertion of grafts to bypass narrowed atherosclerotic coronary arteries is a very common operation. Often 3 (triple bypass) or 4 (quadruple bypass) grafts are inserted. The graft material used may be saphenous vein taken from the legs or segments of internal mammary artery. The operation involves making a midline incision in the chest, division of the sternum and retraction of the rib cage to expose the heart. The patient is connected to a cardiopulmonary bypass pump that takes over for the heart and lungs during the procedure. The body is cooled to reduce the need for oxygen and the heart is stopped. Once the grafts are in place, electric shocks start the heart pumping again, the heart-lung machine is turned off, the blood slowly returns to normal body temperature and the surgeon closes the chest cavity.
The grafts themselves, even the veins, often eventually develop atherosclerosis.

CASE 50003/83
The patient was a 78 year old man who presented with an atheromatous abdominal aortic aneurysm. The aneurysm was repaired by inserting the teflon graft as seen. However, he died of myocardial infarction shortly after the operation.
The specimen consists of the abdominal aorta including the bifurcation. The graft traverses a dilation of the aorta, with old pale thrombus between the graft and wall. The back of the specimen shows bleeding into the interstitial tissues. Elsewhere the aorta and iliac arteries show atheromatous plaques that are ulcerated in areas.
Diagnosis: Grafted aortic aneurysm.

CASE 50069/83
The specimen consists of a short segment of aorta showing severe ulcerated atheroma.
Diagnosis: Atheromatous aorta.

CASE 50101/83
This patient died of a massive pulmonary embolus one month following an operation to repair an abdominal aortic aneurysm. The graft had been functioning well.
The specimen consists of the abdominal aorta. There is a large, fusiform aneurysm of the lower abdominal aorta and a dacron graft can be seen in place within the lumen of the vessel. The graft has been laid down the centre of the aneurysm leaving the wall in situ. The aneurysm contains old thrombus and has developed

on the basis of atheroma. A small amount of thrombus is adherent to the luminal aspect of the graft. There is patchy atheroma with focal overlying thrombus elsewhere.
Diagnosis: Grafted aortic aneurysm.

CASE 50128/83
The specimen consists of the anterior aspect of the heart with proximal ascending aorta. A vein graft can be seen lying across the pericardial surface. The aortic orifice of the graft can be seen from the back of the jar and its attachment to the left anterior descending coronary artery distally is also evident. Alongside the graft is the severely atheromatous bypassed coronary artery that has been sliced transversely in several places. The shaggy surface of the heart is largely due to fibrous adhesions that develop as a result of operative trauma.
Diagnosis: Coronary bypass graft.
Comment: Insertion of grafts to bypass narrowed atherosclerotic coronary arteries is a very common operation. Often 3 (triple bypass) or 4 (quadruple bypass) grafts are inserted. The graft material used may be saphenous vein taken from the legs or segments of internal mammary artery. The operation involves making a midline incision in the chest, division of the sternum and retraction of the rib cage to expose the heart. The patient is connected to a cardiopulmonary bypass pump that takes over for the heart and lungs during the procedure. The body is cooled to reduce the need for oxygen and the heart is stopped. Once the grafts are in place, electric shocks start the heart pumping again, the heart-lung machine is turned off, the blood slowly returns to normal body temperature and the surgeon closes the chest cavity.
The grafts themselves, even the veins, often eventually develop atherosclerosis.

CASE 50431/85
The specimen consists of a portion of the left ventricle with the posterior mitral valve leaflet. There is an exophytic vegetation which has a central perforation sited near the base of the valve. This vegetation was associated with an underlying abscess that penetrated the myocardium. The underlying valve is mildly thickened and there is some suggestion of 'hooding' of the valve. The chordae tendineae are normal.
Diagnosis: Infective endocarditis with perforation of the mitral valve.
What do the thickening and 'hooding' of the mitral valve suggest? The appearances suggest the condition of mitral valve prolapse (also known as 'floppy' mitral valve or myxomatous degeneration of the mitral valve). This condition is a risk factor for infective endocarditis.
Comment: An endocarditis which is causing destruction of the valve and myocardial abscess formation is most likely to be caused by a very virulent organism such as *Staphylococcus aureus* and run an acute course.

CASE 50163/93
This was an incidental finding at autopsy.
The specimen is part of a heart that demonstrates a congenital anomaly. It has been opened through the mitral valve. A second mitral valve orifice is present through the anterior leaflet of the valve.
Diagnosis: Double mitral valve orifice.

CASE 50057/97 (2 specimens)
The specimen is a slice of heart through both ventricles. There is myocardial and subendocardial fibrosis in the lateral left ventricular wall in keeping with old infarction in the territory of the left circumflex coronary artery. The right and left ventricles otherwise appear within normal limits.
Diagnosis: Old myocardial infarction.