22 Aortic Dissection

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Take Home Messages

• Survival and outcome following aortic dissection is dependent on making a rapid and accurate diagnosis.
• Surgery in type A dissection confers overwhelming benefit compared with the natural history of the disease.
• Medical management is recommended initially in type B dissection – complication-specific approach to be followed.
• Close follow-up for aortic dissection by a specialised team to assess the signs of aortic expansion and aneurysm formation by serial imaging is pertinent.
• Endovascular stent graft placement for type B aortic dissection compares favourably with surgical treatment, but further studies to compare it with medical treatment are warranted.

22.1 Introduction

Dissection is the most common catastrophic event affecting the aorta, occurring two to three times more frequently than rupture of a degenerative abdominal aortic aneurysm. A dissection is characterised by the presence of a tear in the intima of the aorta through to the muscular media, allowing cleavage and separation within a medial plane. This creates a false channel or lumen within the aorta. The plane of cleavage may be propagated proximally and distally along the length of the aorta under the force of left ventricular ejection, generating excruciating pain. Those afflicted by dissection are at risk of aortic rupture and malperfusion phenomena as the origin of major branch arteries of the aorta are compromised by dissection propagation. If the ascending aorta is affected, the dissection may compromise coronary perfusion and carotid artery perfusion, aortic valve function and, if rupture occurs, death by cardiac...
tamponade [1]. Thus the disease is associated with high morbidity and mortality. Dissection has an incidence and prevalence of approximately 20–40 per million per annum [2, 3] and is more common in males (male:female ratio 5:1) [4]. The incidence of aortic dissection displays peaks at three age ranges. Proximal dissection (involving the ascending aorta) has a peak incidence at 50–60 years of age, whereas distal dissection (only involving the descending aorta) peaks at 60–70 years [5]. Dissection of the proximal aorta in patients with Marfan’s syndrome or predisposing causes accounts for the third peak at 20–40 years [6, 7].

Despite advances in diagnosis and medical, surgical and endovascular management of aortic dissection, the morbidity and mortality remain significant [8] and early recognition with prompt expert management is necessary if outcomes are to be improved. A thorough understanding of the clinical presentation, classification and pathological anatomy of aortic dissection is essential to planning management.

22.2 Aetiology, pathology and pathophysiology

Aetiology
Risk factors for aortic dissection include certain congenital heart defects, connective tissue disorders, inflammatory diseases and environmental factors (Table 22.1). The greatest risk in pregnancy occurs in the third trimester when hormone-induced changes in connective tissue and hypertension are maximal. Dissection associated with pregnancy is most prevalent in Marfan’s syndrome and consideration should be given to prophylactic aortic root surgery prior to pregnancy. An association with the use of cocaine and pregnancy result at least in part from transient surges in blood pressure [9, 10].

Deceleration trauma usually causes a transection injury of the aorta, but rare cases of dissection have been reported. More commonly trauma is iatrogenic, occurring either at the time of surgical manipulation of the ascending aorta during cardiac surgery or during the passage of intravascular catheters. Chronic dissection at the site of previous aortotomy for cannulation, valve access or proximal coronary bypass anastomosis has been reported.

Histological features
The aortic wall comprises three layers, the intima, tunica media and adventitia, and is approximately 2 mm thick in the ascending portion. The tunica media is the strongest component and comprises layers of elastic and collagen fibres and obliquely (ascending) or circularly (distal) orientated smooth muscle cells (5%). The media is thickest in the ascending aorta due to increased elastic fibres. Elastin appears to be deposited around microfibrillar fibres of the glycoprotein fibrillin which serves as a scaffold for the organised structure of the wall. Absence or mal-production of fibrillin is the basis of Marfan’s syndrome and arises due to an inherited or spontaneous mutation of the fibrillin gene on the long arm of chromosome 22.
In non-Marfan’s patients with an apparent predilection to dissection, there is a degeneration of the medial elastic skeleton termed cystic medial necrosis. In this condition, the elastic lamellae become stretched, depleted and fragmented. The resulting cystic defects are filled with chondroitin sulphate, a mucopolysaccharide. The process appears to be an expression of accelerated degeneration without elastic tissue replacement and is associated with increased levels of matrix metalloproteinase within the aortic wall.

**Dissection propagation**

The ascending aorta, which has the highest fraction of elastic tissue, is the aortic segment that shows the greatest predilection to dissection (50–80%). There remains uncertainty whether the initiating process represents a spontaneous intimal tear with medial cleavage or whether a spontaneous bleed develops in the media breaking through into the lumen. The dissection plane propagates distally, leaving a true lumen and generating a false lumen. The propagation affects mainly the areas of greatest

<table>
<thead>
<tr>
<th>Condition or factor associated with dissection</th>
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<tr>
<td><strong>Congenital, inherited or acquired connective tissue disorders</strong></td>
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<tr>
<td>Marfan’s syndrome</td>
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<td>Ehlers–Danlos syndrome</td>
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<td>Loeys–Dietz syndrome</td>
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<td>Osteogenesis imperfecta</td>
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<td>Pseudoxanthoma elasticum</td>
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<td>Menkes’ syndrome</td>
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<td><strong>Other cardiac associations</strong></td>
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<td>Unicuspid and bicuspid aortic valves</td>
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<td>Coarctation of the aorta</td>
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<td>Pseudo-coarctation</td>
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<td>Supra-valvar aortic stenosis</td>
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<td><strong>Hypertension</strong></td>
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<td>Essential hypertension</td>
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<td>Phaeochromocytoma</td>
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<td>Pregnancy</td>
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<td>Body-building</td>
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<td>Cocaine abuse</td>
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<td><strong>Inflammatory diseases</strong></td>
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<td>Syphilis</td>
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<td>Polymyalgia rheumatica</td>
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<td>Giant cell arteritis</td>
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<td>Behçet’s syndrome</td>
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<td>Takayasu arteritis</td>
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<td>Ormond’s disease</td>
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<tr>
<td><strong>Trauma</strong></td>
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<tr>
<td>Deceleration injury</td>
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<td>Vascular catheter-induced</td>
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<td>Iatrogenic during cardiac surgery</td>
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longitudinal and radial curvature. In the ascending aorta this leads to false luminal expansion to the right and anteriorly, which may disrupt the right coronary ostium and the non-coronary sinus of Valsalva, and along the convexity of the aortic arch, jeopardising the origins of the epi-aortic vessels. The primary entry tear is usually transverse and approximately 60% arise within 2 cm of the left main coronary ostium. The dissection plane usually propagates along the left posterolateral aspect of the descending aorta, creating a false lumen separated by an intimal flap and a relatively thinner adventitial layer on the outside. False luminal patency is maintained by fenestrations in the intima generated by the shearing of intercostal origins or secondary re-entry tears generated by the intra-luminal rupture of the pressurised false channel [1].

**Malperfusion phenomena**

Malperfusion phenomena due to branch artery compromise are seen in a third of cases of aortic dissection and may arise by a variety of mechanisms. The most common form occurs when the true lumen is compressed by a pressurised false lumen. This may compromise branch arteries or even the whole aorta, leading to acute lower body hypoperfusion and an acute Leriche syndrome, manifesting as impotence, fatigue and pallor affecting the lower limbs. Extension of a dissection into a branch artery may also occur, leading to a similar phenomenon without re-entry. Alternatively, the intima of the whole vessel origin may be sheared, leading to intussusceptions of the inner intimo-medial layer. The mobile intimal flap can also occlude a branch vessel, acting like a lid forced closed under pressure. Thrombosis beyond the compromised vessel ostium may further worsen ischaemia. The incidence of clinically important malperfusion phenomena is reported to be: lower limb 12–25%, cerebral 8–12%, coronary 7–10% and renal or mesenteric 5–10%.

### 22.3 Classification

Aortic dissections are classified according to location and acuity (Table 22.2, Figure 22.1). These classifications are pragmatic. A dissection involving the ascending aorta is managed differently from one involving the descending aorta alone. A chronic dissection will have undergone a degree of wall healing and theoretically should be more amenable to surgical intervention.

### 22.4 Clinical presentation

The main challenge in the management of acute aortic dissection is to make an accurate diagnosis as soon as possible [11]. About a third of the patients with dissection are initially diagnosed as having acute coronary syndromes, pulmonary embolism and abdominal emergencies (Table 22.3). As the predominant symptom of dissection is chest pain, there is a large differential diagnosis (Table 22.2). A fraction of patients will have a history
of Marfan’s syndrome or other predisposing risk factors for dissection, while two-thirds of patients will give a history of treated hypertension.

**Symptoms**

Pain is the chief presenting symptom, described as sharp, tearing, ripping or stabbing in nature with a variable location. In proximal dissections the
pain is usually located anteriorly behind the sternum, whereas distal dissections are characterised by interscapular and back pain. Chest pain may occasionally be absent especially in cases of chronic dissection. The absence of pain substantially decreases the probability of acute dissection.

Syncope is seen in 5–10% of cases of acute type A dissection and may occur as a result of severe pain, activation of aortic baroreceptors, leakage from the aorta, cardiac tamponade or involvement of the brachiocephalic vessels with transient brain malperfusion. These patients are more likely to have type A dissection, suffer a stroke and die in hospital. Some patients present with stroke. Breathlessness may follow pain as a manifestation of acute heart failure or pleural effusion. On rare occasions, symptoms such as vocal chord paralysis (caused by compression of the left recurrent laryngeal nerve), haemoptysis or haematemesis (due to haemorrhage into the tracheobronchial tree or perforation into the oesophagus), upper airway obstruction (due to compression), superior vena cava syndrome, Horner’s syndrome (due to compression of the superior cervical sympathetic ganglion) or signs of mesenteric or renal ischaemia may be encountered. Paraplegia or paraparesis from interruption of intercostal vessels occurs more commonly in patients with type B dissection and is seen in 2–3% of cases [5]. Similarly, patients may complain of transient limb weakness or numbness as a manifestation of malperfusion phenomena.

**Physical signs**

*Hypertension* is common at presentation in both type A and B dissection. Hypotension is more common in type A dissection and may be due to acute heart failure as a result of aortic regurgitation or coronary malperfusion or cardiac tamponade [12]. Examination of all pulses is critical and may identify a collapsing pulse with wide pulse pressure due to aortic regurgitation (AR), evidence of pulsus paradoxus or pulse deficits. Pulse or pressure differentials between carotid, radial or femoral arteries are common and occur in 30–50% of the patients. These patients are more likely to develop complications including neurological deficits, coma and hypotension and are at an increased risk of dying. Carotid pulse deficits are

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**Table 22.3** Differential diagnosis of acute dissection.

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<thead>
<tr>
<th>Cardiac causes</th>
<th>Abdominal causes</th>
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<tr>
<td>Myocardial infarction</td>
<td>Acute cholecystitis</td>
</tr>
<tr>
<td>Acute coronary syndrome</td>
<td>Duodenal ulcer</td>
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<td>Aortic regurgitation without dissection</td>
<td>Pancreatitis</td>
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<tr>
<td>Pericarditis</td>
<td>Reflux oesophagitis</td>
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<tr>
<td><strong>Pulmonary causes</strong></td>
<td><strong>Miscellaneous</strong></td>
</tr>
<tr>
<td>Pleurisy</td>
<td>Musculoskeletal pain</td>
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<tr>
<td>Pulmonary embolism</td>
<td></td>
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<tr>
<td>Pneumothorax</td>
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strongly related to fatal strokes. The clinical course of peripheral ischaemia can be quite variable, with a third of these patients demonstrating spontaneous resolution [5]. Refractory hypertension despite medical management is commonly seen in the patients with type B dissection. Involvement of the brachiocephalic vessels can result in differential blood pressure between the arms. In type A dissection, an early diastolic murmur of aortic regurgitation can be heard in about a third of cases.

**Initial investigations**

**Electrocardiogram (ECG)**

Many patients have non-specific T wave or ST segment changes and a few have clear evidence of coronary malperfusion, most usually with inferior ST elevation. Thus, an ECG alone may not distinguish dissection from acute myocardial infarction and a number of patients erroneously receive thrombolysis. A normal ECG is present in one-third of patients with coronary disease, whilst some patients with acute dissection involving the coronary ostia may have ECG evidence of ischaemia or infarction.

**Chest X-ray**

The chest X-ray is normal in about 1 in 8 cases and the classical description of a widened mediastinum only occurs in approximately 60%. Other features may include an abnormal aortic or cardiac contour and there may be apparent displacement of intimal calcification at the aortic knuckle into the vessel lumen. Pleural effusion may be an exudative reaction, a consequence of heart failure or an indication of rupture.

**Laboratory investigations**

Creatine kinase may be elevated due to muscle injury, but cardiac troponin may be more discriminatory provided coronary malperfusion is absent. Patients with dissection may have a moderate leukocytosis, raised C-reactive protein, serum bilirubin and lactic dehydrogenase. An elevated concentration of smooth muscle myosin heavy chain has been associated with aortic dissection [13]. A consumption coagulopathy with prolonged prothrombin and partial thromboplastin times, reduced platelet count, reduced fibrinogen and elevated D-dimers commonly accompanies dissection. At present, no biochemical test has sufficient specificity or sensitivity to aid diagnosis.

**Imaging**

**Transthoracic/transoesophageal echocardiography**

Transthoracic echocardiography (TTE) is a useful screening investigation while definitive investigations are awaited. It will provide information regarding left ventricular function, regional wall motion abnormality, aortic valve competence, and the presence of pericardial fluid, and allows a limited examination of the proximal ascending aortic wall. Approximately 60% of acute type A dissections can be confidently diagnosed using TTE, but the absence of an intimal flap is a poor indicator that dissection is not
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present (high false negative). Transoesophageal echocardiography (TOE) (Figure 22.2) improves the sensitivity and can provide valuable additional advice regarding the site of entry and re-entry tears and false lumen blood flow. Small intimal tears can be detected by colour Doppler, registering jets traversing the dissection membrane. Echo-free spaces around the aorta may be evidence of peri-aortic haematoma. The sensitivity and specificity of TOE for aortic dissection diagnosis are each approximately 90%.

Computed tomography (CT)
Contrast-enhanced fine-cut CT is currently the most commonly used imaging modality in patients with suspected aortic dissection. The diagnosis is based on the demonstration of an intimal flap which separates the true from the false lumen. The flap is identified as a low attenuation linear structure in the aortic lumen (Figure 22.3). Secondary findings include internal displacement of intimal calcifications, delayed enhancement of the false lumen and aortic widening. The finding of greatest significance is the observation of false lumen being larger than true lumen in more than 90% of cases. The sensitivity and specificity of CT scan are each in the region of 90% [14].

Magnetic resonance imaging (MRI)
MRI may have the highest overall sensitivity and specificity for detection of aortic dissection [15]. However, the technique is often not available on an emergency basis and examination of haemodynamically unstable patients

Figure 22.2 Epi-aortic echocardiography of the ascending aorta in a patient with acute aortic dissection (dissection flap arrowed). Note the increased echo-density in the false lumen compared with the smaller true lumen. T, true lumen; F, false lumen.
may be difficult. As with TOE, MRI allows localisation of entry and re-entry tears and, in addition, flow in false and true lumen can be quantified using phase contrast cine MRI or by tagging techniques. MRI may also allow visualisation of the proximal coronary arteries.

Aortography
Aortography used to be the gold standard for diagnosis, but its role has been taken over by less invasive investigations. Its use is now restricted to a component of percutaneous intervention in endovascular management of dissection and malperfusion. Contrast aortography accurately identifies branch vessel involvement, especially of renal or mesenteric vessels. The specificity of aortography for diagnosing aortic dissection is > 95%, but its sensitivity is lower than that of other techniques.

Coronary angiography
Although coronary atherosclerotic disease may be present in a quarter of patients with acute dissection and may worsen the surgical outcome, coronary angiography is rarely used as it may delay emergency surgery and has potential to cause propagation of the dissection. Its use is reserved for those cases with a definite history of ischaemic heart disease or previous surgery, particularly coronary artery bypass surgery. Coronary angiography has not been shown to improve hospital survival [16].

Intravascular ultrasound (IVUS)
IVUS is a novel, invasive technique that can directly visualise the vessel wall architecture from inside the aortic lumen, therefore allowing the accurate
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recognition of aortic wall characteristics and pathology. It provides visualisation of the intimal-medial flap, its movement, its circumferential and longitudinal extent and the degree of luminal compromise. Branch vessel involvement and false lumen thrombosis may be better defined with IVUS than with TOE. Although sensitivities and specificities approaching 100% have been reported, IVUS is not yet in widespread diagnostic use [17].

22.5 Management

Medical treatment
As soon as the diagnosis is made the patient should be moved to a critical care unit and invasive arterial blood pressure monitoring instituted. All type A dissections should be immediately transferred to cardiac surgical centre. There is a strong argument for also transferring cases of type B dissection, but at present, transfer is usually reserved for those patients with complications such as malperfusion phenomena. Systolic blood pressure (BP) should be reduced to ≤ 110 mmHg, but in the presence of aortic regurgitation, a mean BP of 60–75 mmH should also be targeted. Effective pain relief will help to control BP. Beta blockers are the initial drug of choice as they decrease the force of left ventricular contraction (dP/dT). Intravenous beta blockers like labetolol (both alpha and beta blocker) and esmolol are suitable for this purpose (Chapter 24).

Pleural effusions should be drained. The presence of a pericardial effusion in the presence of dissection should be assumed to be due to intra-pericardial rupture and is an indication for emergency surgery. Pericardiocentesis may be used as a holding manoeuvre during transfer in shocked patients, but this is controversial [18].

Surgical management of type A dissection
The presence of ascending aortic involvement is an indication for operative management in all but the highest risk cases [19]. Neurological status at presentation can influence the decision to operate. Obtundation, coma, stroke or paraplegia present for less than a few hours may represent malperfusion phenomena that may improve with reperfusion. However, established deficits with corresponding lesions on CT scanning may become haemorrhagic during cardiopulmonary bypass and so individualised management plans with deferment of surgery may be appropriate. The presence of mesenteric or bilateral renal ischaemia are both treatment priorities, but initial management should be directed at closing the initial entry tear surgically or endovascularly prior to a direct approach such as laparotomy, as re-institution of true luminal flow may lead to reperfusion of the affected viscera [5]. However, endovascular peripheral revascularisation may still be necessary [20].

The objectives of surgery in type A dissection are: prevention of intra-pericardial rupture, protection of the coronary artery ostia and other major vessels from malperfusion, restoration of the competence of the
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Aortic valve and elimination of a distal false lumen (Figure 22.4). The reported outcomes for acute type A dissection repair vary, with reported early mortalities between 6 and 30%. In the International Registry of Aortic Dissection database, the overall hospital mortality was 25.1%. Mortality is higher in patients with instability or malperfusion phenomena. Longer-term outcomes are satisfactory, with 5- and 10-year survival for patients undergoing immediate surgical repair of 71% and 54% respectively [21, 22].

Surgical management of type B dissection

Early surgery is reserved for cases with complications, including leakage, distal malperfusion, prompt expansion, continuing pain and inability to control BP. Such emergency procedures are inevitably high risk and mortality rates are approximately 30% [23]. More recently, endovascular stenting of complicated dissections has shown great promise in reducing mortality and morbidity. Deployment of a covered endovascular stent to exclude the entry tear in the distal aortic arch may restore perfusion to organs in jeopardy. Both surgery and stenting can be combined with other percutaneous catheter-based techniques to deal with branch vessel occlusion including intimal flap fenestration and branch vessel stenting [24]. In a meta-analysis of endovascular stent graft placement in type B dissections, successful stent deployment was achieved in > 95% of cases with a neurological complication rate of 3% [25]. Survival at 2 years was 90%, but stenting failed to abolish the false lumen in about a quarter of patients.
Most uncomplicated type B aortic dissections are treated conservatively, with similar survival rates approaching 90% [26]. Medical therapy is directed towards the control of hypertension and at least 60–70% continue to be free of aortic complications in the long term. The role of endovascular treatment for all cases remains an issue of debate [27].

**Anaesthetic management**

Many patients present as an emergency, which necessitates rapid sequence induction. In these and elective cases, the choice of anaesthetic technique should confer cardiac stability, with particular attention to blunting the haemodynamic response to intubation. In the emergency setting, careful control of the blood pressure with beta blockade and/or intravenous venodilators may prevent progression of the dissection (see Chapter 24).

Line placement is crucial and should be guided by a discussion regarding the proposed surgical technique. Arterial monitoring should be established via a femoral artery (leaving one side for surgical access) and a radial artery. The left radial is preferentially used to avoid loss of the arterial blood pressure wave form if the dissection flap extends to the innominate artery or the surgeon cannulates the right axillary artery for perfusion access. Other routine monitoring includes 5-lead ECG, saturation probe (left hand), central venous pressure (via right internal jugular) and a nasopharyngeal temperature probe. The measurement of core temperature is essential if deep hypothermic arrest is to be used, and the nasopharyngeal route equates most closely with the cerebral temperature. Further evidence of adequate cerebral metabolic suppression may be obtained by monitoring the jugular bulb venous saturation using a retrogradely inserted jugular bulb line. A jugular bulb saturation of $\geq 95\%$ implies that the cerebral metabolic rate is less than 25% of the rate at $37^\circ$C and this can be used as a guide for commencing circulatory arrest. Most centres advocate intra-operative TOE during aortic dissection surgery, which necessitates a ‘second operator’.

Two issues of specific concern during thoracic aortic surgery are cerebral protection during deep hypothermic arrest and the maintenance of spinal perfusion. Deep hypothermic arrest is usually at 16–18°C. Cerebral perfusion may be maintained via anterograde or retrograde flow, though some surgeons advocate neither [28]. Twenty minutes is a safe period of arrest in adults, though longer periods are used in children. An absolute value is difficult to establish. It is advisable to check pupil size and reaction prior to arrest, and institute bispectral index monitoring. A variety of methods have been used to confer cerebral protection including thiopentone, propofol, isoflurane, corticosteroids and topical cooling [29]. Thiopentone will cause burst suppression and does reduce the cerebral metabolic requirement for oxygen, but the doses needed for clinical protection are high (3–10 mg/kg confers 5 minutes of electroencephalography (EEG) suppression) and necessitate increased use of vaspressors and inotropes. Propofol may be used to maintain anaesthesia during bypass and may confer an advantage during
hypothermic arrest. Isoflurane cannot be administered during arrest. Corticosteroids are only of proven value in reducing cerebral oedema, and have not been shown to confer cerebral protection \textit{per se}. The only proven method of protection is ice packing the head. Care must be taken not to cause hypothermic injuries to the face and eyes. Slow cooling and slow rewarming are also advisable during cardio-pulmonary bypass; slow rewarming allows wash-out of metabolites and free radicals at a time when the brain is particularly sensitive to permanent cellular damage. Glucose-containing solutions should be avoided as they may worsen cellular damage.

The spinal cord is largely supplied by a series of arteries which are branches of the descending thoracic aorta. Damage to these vessels may result in cord ischaemia and subsequent paralysis, affecting 5–20% of cases. Injury is also possible when hypoperfusion is prolonged. If the pressure within the cerebrospinal fluid (CSF) containing spaces is reduced, supply to the cord may be preserved even during periods of relative hypoperfusion. It is on this basis that spinal drains are advocated for cord protection during thoracic aortic surgery. The insertion technique is simple and involves dural puncture with a Thuoy needle and placement of an indwelling spinal catheter under strict aseptic conditions. Debate regarding the risk of bleeding and subsequent cord compression during full heparinisation complicates the use of spinal drains. The CSF pressure is measured and maintained using a closed manometer and drain. Both excessive and inadequate drainage of CSF may have disastrous consequences and spinal drains need careful monitoring and vigilance during the intra-operative and post-operative period. The use of shunts or partial bypass during thoracic aortic surgery does not guarantee distal perfusion or confer spinal protection. It is advisable to check motor function in the lower limbs as soon as possible post-operatively, and remove the spinal drain once adequate motor function is documented. In patients who have not had a spinal drain inserted prior to surgery but subsequently develop neurological signs of lower limb motor dysfunction post-operatively, a spinal drain may then be used for cord protection [30]. Corticosteroids may also be used to reduce spinal cord oedema.

Post-operatively, a major concern is maintenance of perfusion whilst protecting the surgical sites from surges in blood pressure. Systolic pressures of 80–100 mmHg should ensure adequate spinal cord perfusion and renal blood flow. Excessive systemic pressures may be controlled by ensuring adequate sedation and analgesia combined with anti-hypertensive therapy, for example intravenous glyceryl trinitrate. Central neuraxial blockade may be complicated by full heparinisation for bypass and the use of a spinal drain. Post-operative complications that may occur and require vigilance include renal, mesenteric and spinal hypoperfusion. Coagulopathy following massive transfusion is common and should be aggressively corrected. Blood glucose should be closely controlled (6–8 mmol/l) as poor control worsens outcome [31].
22.6 Outcomes

Type A dissection
Untreated type A aortic dissection carries a mortality of 20% by 24 hours after presentation, 30% at 48 hours, 40% by day 7 and at least 50% at 1 month with medical management alone (Figure 22.5) [19]. Mortality is attributable to rupture, malperfusion phenomena and intractable heart failure due to severe aortic regurgitation. The most common causes of post-operative mortality are cardiac failure, intra-operative haemorrhage, multiple organ failure and major neurological injury [32]. Surgical repair does not consistently eliminate the flow and pressure in the distal false lumen.

Type B dissection
Patients with uncomplicated type B dissection have a 30-day mortality rate of 10% [8]. Patients with leg ischaemia, renal failure, visceral ischaemia or contained rupture have a 30-day mortality rate of 25%. Advanced age, rupture, shock and malperfusion are important predictors of mortality [26, 33]. The majority of patients with type B dissection are uncomplicated and can be managed medically [8].

Late complications
Both proximal and distal dissection may be complicated by late aneurysm formation, which is seen in 25–40% of the patients surviving the acute event despite adequate treatment [5]. Late aneurysm formation appears to be related to persistence of a patent false lumen, higher initial aortic

![Figure 22.5](image-url)
dimension and poor BP control within the first weeks of the dissection [34]. Long-term beta blockade leads to a reduction in late aneurysmal complications [35]. About 10–20% of patients with dissection experience late aneurysm rupture [36]. Hence, it is essential to follow up such cases with serial imaging.

22.7 Other acute aortic syndromes

**Intra-mural haematoma (IMH)**
Aortic IMH is considered a precursor of dissection, originating from ruptured vasa vasorum in medial wall layers and resulting in an aortic wall infarct that may provoke a secondary tear, causing a classic aortic dissection. IMH may progress to dissection, aneurysm formation or rupture, but a significant fraction will reabsorb with good BP control. Most IMH cases (50–85%) occur in the descending aorta and are typically associated with hypertension. Clinical manifestations of IMH are similar to acute aortic dissection. Chest pain is more common with ascending (type A) IMH; upper or lower back pain is more common with descending (type B) lesions. The diagnosis of IMH versus acute aortic dissection cannot be made clinically and is dependent on tomographic imaging. Type B IMH is treated like a type B dissection. A high fraction of type A IMHs may progress to full dissection or wall rupture and therefore these lesions are managed surgically.

**Penetrating atherosclerotic ulcer (PAU)**
Deep ulceration of atherosclerotic aortic plaques can lead to IMH, aortic dissection or perforation. PAU most often complicates IMH and appears as an ulcer-like projection into the aortic wall haematoma. Symptomatic ulcers or those with signs of deep erosion appear more prone to rupture, and in these patients endovascular stenting is emerging as an attractive therapeutic modality.

22.8 Summary

The possibility of aortic dissection should be considered for every patient presenting with severe chest pain. The key to achieving the best outcome is early recognition and diagnosis and prompt expert management. Patients with type A dissections should be immediately transferred to a cardiac surgical centre. There is a strong argument for also transferring cases of type B dissection, but at present this is usually reserved for those patients with complications such as malperfusion phenomena. Blood pressure control is key to preventing proliferation of the dissection flap and malperfusion syndromes. Type A dissections are managed surgically in most cases, whereas early surgery is reserved for only type B dissections associated with complications.
Case study
A 67-year-old female, a current smoker, presented to the Emergency Department with central anterior chest pain radiating to the back associated with shortness of breath and a left-sided weakness. The patient was found collapsed by a relative who then brought her to hospital, where she had an episode of profound bradycardia which was treated with atropine. Past medical history included cerebral aneurysm treated 13 years ago and she was under investigation for a breast lump. There was a family history of type B aortic dissection affecting her son.

On examination, the patient’s circulation was adequate clinically but a diastolic murmur of severe aortic regurgitation was heard and the radial and brachial pulses on the left were impalpable. There were signs of a left hemiparesis. A CT scan of the head, thorax and abdomen demonstrated a type A dissection (Figure 22.3) and the decision to perform emergency repair was made. Informed consent was secured quoting a mortality of 50% (increased because of the pre-operative malperfusion), stroke rate of 10% and paraplegia of 10%. Ten units of cross-matched blood were requested, 10 units of fresh frozen plasma (FFP), 10 units of platelets and 10 units of cryoprecipitate.

Surgery commenced within 2 hours of referral. At operation a near-circumferential type A dissection with disruption of non-left and non-right commissures leading to aortic regurgitation was evident. A small transverse intimal tear 1 cm distal to the left main coronary artery was identified, with the dissection involving the right coronary ostium and in the arch involving the ostia of the innominate and left carotid artery. The ascending aorta was excised, the aortic valve resuspended and an interposition graft was anastomosed between the transected proximal and distal aorta.

The patient returned to the intensive treatment unit (ITU). Where on waking there was no movement in the legs, cerebrospinal fluid drainage was performed through a drain inserted at the L4/L5 level. Physiotherapy was instigated on the first post-operative day, at which point there were no plantar or knee reflexes. In the pursuing days the patient maintained good haemodynamic parameters and progressively improved from a respiratory aspect, with all drains and invasive monitoring having been removed by the 8th day post surgery.

The main concern was the lack of recovery from neurological symptoms. She maintained a flaccid paralysis of the lower limbs and the impression was that there was significant injury of the spinal cord following a malperfusion injury. Unfortunately due to the cerebral aneurysmal clipping she had performed 13 years ago, an MRI could not be performed to delineate the level of spinal cord injury. Despite a small amount of recovery of power in the left leg there was no significant improvement in the lower limb neurology. The patient was discharged to a long-term rehabilitation centre for intensive neuro-rehabilitation on anti-hypertensive medication and with a planned follow-up surveillance scanning.
References


