# Aortic Dissection

# 8

CONTENTS



## 8.1 Summary


- Aortic dissection is one of the "great masqueraders," so always suspect this diagnosis in any acute painful illness with a pulse deficit.
- A practical classification in the emergency situation is type A, involving the ascending thoracic aorta and the arch and type B involving the aorta distal to the left subclavian artery.
- Treatment of type A dissection is always surgical.
- Treatment of type B dissection is medical in most cases and surgical if there is complicating organ ischemia or bleeding.
- Alert the attending thoracic or vascular surgeon on call early during management, especially in type A dissections.


## 8.2 Background

Dissection of the thoracic aorta represents a major clinical problem that is extremely demanding to manage even for experienced surgeons. Once it is diagnosed this condition is usually managed by an experienced specialist in thoracic or vascular surgery. The responsibility for the diagnosis and its primary management, however, mostly belongs to the surgical or medical emergency physicians.

### 8.2.1 Magnitude of the Problem

The true prevalence and incidence of aortic dissection are unknown, but it has been reported to have an annual occurrence of 5–10 cases per

million and to affect between 10,000 and 25,000 patients annually in the United States. Autopsy studies in the United States and Denmark report dissections in 0.2–0.8% and 0.2% of cases, respectively. An age-adjusted mortality rate from aortic dissections of 0.5–2.7% per 100,000 inhabitants was calculated from 1950 to 1981. The overall incidence of aortic dissections consequently is in the same range or possibly up to two to three times greater than that for ruptured abdominal aortic aneurysm. It is two to five times more common in men than in women, and maximum occurrence is in the 5th decade of life. Still, in our era of modern diagnostic methods, a majority of patients probably die with this disease undetected. Aortic dissection is a dramatic and dangerous condition with a very high mortality: 20–50% of patients die within the first 24–48 h, and up to 75% within the first 2 weeks. It is considered as one of the "great masqueraders," with a wide range of presenting symptoms. Because the diagnosis is difficult, awareness of aortic dissection in the differential diagnosis is essential, as is rapid and correct management.

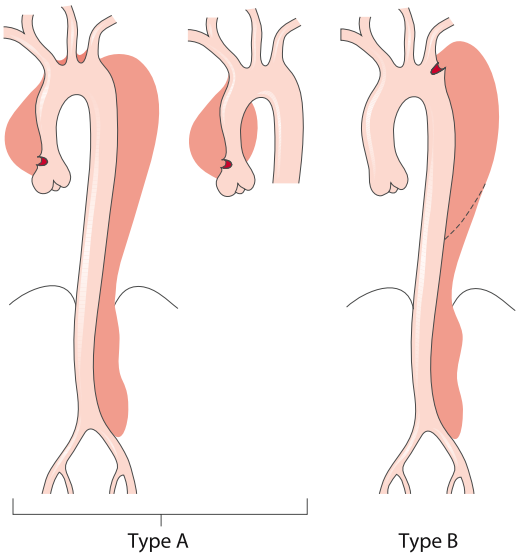


**Fig. 8.1.** Classification of aortic dissection in types A and B, according to Daily (Stanford)

## 8.2.2 Classification and Definition

Aortic dissection is characterized by two or more communicating flow channels originating from a proximal intimal tear, with propagation of the bloodstream within the medial layer. It should be distinguished from an intramural hematoma, which is a hemorrhage into the medial layer of the aortic wall without an intimal tear. Intramural hematomas have a natural history similar to aortic dissection and are treated similarly.

The most useful classification of aortic dissections in the acute situation is the one proposed by Daily (Stanford classification), as shown in Fig. 8.1.

Other classic classifications are by DeBakey and Crawford (thoracoabdominal aneurysms and chronic dissections). The information in this chapter is based on the Stanford classification because it simplifies the acute management. A type-A dissection always involves the ascending aorta, regardless of the distal extension. A type-B dissection does not involve the ascending aorta. There is consensus in the literature that an aortic dissection is considered acute if the onset of symptoms occurred within 14 days of presentation, and chronic if more than 14 days have elapsed.

**NOTE**

**The most practical classification in the emergency situation is**

- **Type A: involving the ascending thoracic aorta and the arch**
- **Type B: involving the aorta distal to the left subclavian artery**

## 8.2.3 Etiology

Aortic dissection is usually related to some kind of degenerative changes in the aortic wall, in particular the media. Even if a dissection primarily starts with a tear in the intima, its propagation within the media varies considerably from almost none to rapid progression along the entire length of the aorta. The variation is related to the condition of the medial layer. Some congenital connective tissue defects are known to cause such degeneration, including Marfan's syndrome, Turner syndrome, and Ehlers–Danlos' syndrome. Cystic media necrosis is another predisposing condition. The role of arteriosclerosis is often discussed. It is present in older patients with hypertension pre-

senting with dissection and might constitute a rare mechanism by penetration of atherosclerotic ulcers extending through the intima into the media. This is, however, mostly considered coincidental rather than causative, and some authors even argue that atherosclerotic changes within the aortic wall might be a barrier to the extension of a dissection.

Arterial hypertension is the most important predisposing factor. It is noteworthy that the sudden extreme hypertension associated with severe physical exercise may cause aortic dissection in younger persons. Also, pregnancy with its hypercirculation and hormonal changes affecting connective tissue, is a certain risk factor particularly during the last trimester and during labor. Fortunately, aortic dissections in women are rare, but 50% of dissections occurring in women younger than 40 years old do occur during pregnancy.

Iatrogenic injuries during coronary diagnostic and therapeutic procedures with catheter manipulations can also cause aortic dissection. Blunt chest trauma in otherwise healthy persons may cause aortic dissection, but such dissections are usually very limited due to the minimal degeneration in these structurally normal aortas.

**NOTE**

**A degenerative process causing weakening of the aortic wall in combination with hypertension is the most important etiologic factor.**

### 8.2.4 Pathophysiology

Type A dissection, constituting 60–70% of all aortic dissections, is mostly seen in younger patients with some elastic and connective tissue abnormality. It characteristically starts with a primary intimal tear just distal to the sinotubular ridge in the ascending aorta. This location is in the vicinity of the cephalad extension of the aortic valve commissures. The tear is commonly transverse and has a length corresponding to 50–60% of the aortic circumference. The dissection process starts in the intimal tear and its extension and direction vary, as does the speed with which it propagates.

Typically, a type A dissection affects the right lateral wall of the greater curvature of the ascending aorta. The dissection is usually directed antegradely, but retrograde extension is also relatively common. A primary entry in the ascending aorta is associated with a great risk for bleeding into the pericardium, causing cardiac tamponade.

Type B dissection usually starts with a primary intimal tear in the descending thoracic aorta just distal to the origin of left subclavian artery. This type constitutes approximately 25% of all aortic dissections. A patient with a type-B dissection is typically older, in the 6th–7th decade of life, and has thoracic aortic degeneration and hypertension.

Other possible but less common sites of the primary tear are the aortic arch, occurring in approximately 10% of cases, and the abdominal aorta, occurring in only 2%. As already mentioned, the dissection in the aortic media can travel in a retrograde as well as an antegrade direction, causing two flow channels with a false and a true lumen. Secondary tears and reentries usually occur distally, allowing flow from the false into the true lumen.

Rupture is the most common cause of death in patients with aortic dissection and is mostly located near the site of the primary intimal tear. Consequently, a type-A dissection usually ruptures into the pericardial sac, causing cardiac tamponade, or an aortic arch rupture that bleeds into the mediastinum. In addition, the close relation to the aortic valve commisures can result in acute valve regurgitation due to prolapse of the commissural attachments. Dissection into the aortic root may also involve the coronary arteries, leading to myocardial ischemia or infarction. A type-B descending aortic dissection typically ruptures into the left pleural cavity, and less frequently into the right.

As the dissection extends along the aorta it will subsequently engage major important cerebral and visceral branches, possibly resulting in threatening end-organ ischemia. The mechanisms behind this are compression of the true lumen by the false lumen or shearing of the branch by the dissection process. A third possibility is disruption of an important dissection membrane, causing an intimal flap covering the orifice of a branch. Such peripheral vascular complications occur in 25–30% of patients with aortic dissection and can critically affect cerebral, renal, visceral, and lower

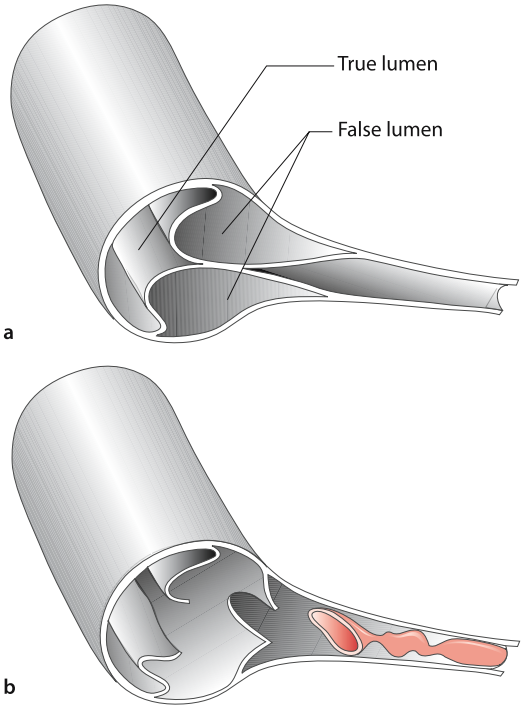


**Fig. 8.2.** Mechanisms of branch occlusion and organ malperfusion in aortic dissection. **a** False lumen expansion causes compression of a side branch. **b** The orifice of a side branch is disrupted by dissection, and its inner layers are impacted distally

extremity perfusion. Because the dissection in the descending aorta mainly engages its left perimeter, the left renal and left iliac arteries are at higher risk than the right ones.

**NOTE**

**Peripheral vascular complications occur in 25–30% of cases of dissection of the aorta.**

## 8.3 Clinical Presentation

### 8.3.1 Signs and Symptoms

Acute aortic dissection patients can display a variety of symptoms, and affected individuals can develop symptoms mimicking those of almost any other acute medical or surgical condition. Aortic dissection must be considered in patients presenting with symptoms indicating acute arterial occlusion and an acute illness that seems to involve unrelated organ systems.

### 8.3.2 Medical History

The most dominant symptom is severe pain, which is migrating or nonmigrating and experienced by more than 90% of patients. When analyzing the pain its typical characteristics are evaluated; if it is: sudden, severe, new, ripping or tearing, and constant. The pain is typically related to the location of the dissection and its propagation distally into different aortic segments. In proximal dissection, the most common pain location is the anterior chest. The pain frequently radiates into the neck and jaws and can be associated with swallowing difficulties. As the dissection propagates distally, the pain migrates to an interscapular location followed by pain in the midback, lumbar, and groin regions (Fig. 8.3).

Abdominal pain might be severe in patients suffering from visceral or renal ischemia. As previously mentioned, the left renal artery is more likely to be compromised, which may explain why severe left flank pain mimicking ureteral colic is often included in the reported history. One should always include questions about hypertension, cardiac disease, peripheral vascular disease, connective tissue abnormalities (such as Marfan's, Turner, and Ehlers–Danlos' syndromes), cystic media ne-

**Table 8.1.** Differential diagnoses in aortic dissection

| Possible differential diagnoses |
|---|
| Coronary ischemia |
| Myocardial infarction |
| Aortic regurgitation without dissection |
| Aortic aneurysm with dissection |
| Musculoskeletal pain |
| Mediastinal tumors or cysts |
| Pericarditis |
| Gall bladder disease |
| Pulmonary embolism |
| Stroke |
| Visceral or lower extremity ischemia without dissection |

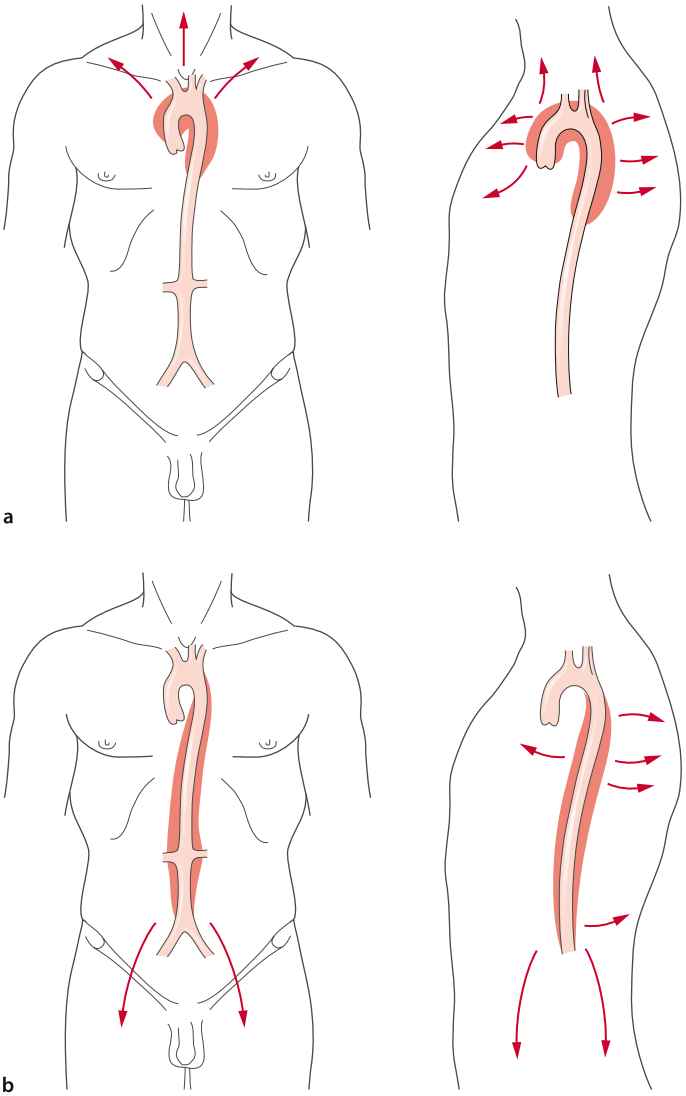

**Fig. 8.3.** **a** Radiation of pain in type A dissection. The pain is usually referred to the neck, anterior chest, and interscapular area. **b** Radiation of pain in type B dissection. Pain is primarily interscapular, but with distal progression of dissection, pain is often referred to the lower back and groin

crosis, diagnostic or therapeutic catheter manipulations, or intense exercise.

Secondary effects of aortic dissection with organ malperfusion necessitate a thorough and complete history to include previous, present, and undulating symptoms of the following:

- Cerebral ischemia: stroke, loss of consciousness, focal neurological symptoms
- Spinal ischemia: paraplegia or parapareses
- Renal ischemia: flank pain, hematuria, diminished urinary output
- Visceral ischemia: severe abdominal pain (for further details of typical symptoms, see Chapter 6, p. 67)
- Lower-extremity ischemia: loss of pulse, loss of sensory or/and motor function, severe pain and coldness (see Chapter 10, p. 122)
- Cardiac malperfusion: angina pectoris or symptoms of acute congestive heart failure

Possible differential diagnoses are listed in Table 8.1.

**NOTE**

**Be aware of aortic dissection as an important differential diagnosis in any acute case presenting with sudden painful illness, in particular if it is associated with symptoms or signs of organ ischemia.**

### 8.3.3 Physical Examination

Complete and repeated physical examinations are of paramount importance in diagnosing and managing patients with suspected or verified acute aortic dissection since this condition can affect so many different organ systems and has a dynamic course.

The typical patient presents with paradoxical physical findings. He or she is frequently pale, restless, and in preshock or shock, and has an appearance indicating poor peripheral perfusion but with a paradoxically high blood pressure. Eighty percent of the patients have arterial hypertension at admission. The high blood pressure is secondary to underlying essential hypertension, elevated catecholamine levels due to severe pain, or occlusion of the renal arteries or even the thoracic or abdominal aorta.

Twenty percent of the patients have a low blood pressure instead. This is usually secondary to cardiac tamponade or rupture, or to acute congestive heart failure secondary to acute aortic insufficiency. Another possible explanation is pseudohypotension secondary to mechanical obstruction from the dissection of one or both subclavian arteries.

Auscultation of the chest is of vital importance. A cardiac murmur indicates aortic regurgitation. The first heart sound is diminished or absent due to elevated end diastolic ventricular pressure. There might be an S3 gallop rhythm. A continuous murmur usually indicates rupture into the right atrium. A pericardial friction rub indicates leakage into the pericardial sac. Auscultation of the lungs might reveal signs of pulmonary edema. Loss of alveolar breath sounds can be found after leakage or rupture into one or both of the pleural cavities. Jugular venous distension is also a common finding.

A complete and repeated neurological examination is mandatory. Horner's syndrome, loss of consciousness, loss of sensory or motor function, paraparesis, paralysis, or paraplegia might be present. Acute cerebral vascular occlusion is for obvious anatomic reasons, more common in proximal dissection, but fortunately neurological deficits occur in only about 20% of those patients.

Lower extremity paralysis in the examination is caused by shearing off or compression of major arteries feeding the spinal cord (intercostal-T8–L1). Another possible explanation is occlusion of the thoracic or abdominal aorta, causing ischemia of the lower body including peripheral nerves. The clinical distinction is important because spinal cord ischemia has a poor prognosis, while a peripheral nerve ischemia has a better prognosis if treated. This distinction can be made by examining peripheral pulses. The latter condition is usually combined with loss of pulses in the groins and distally in the affected lower extremities.

Repeated examination of peripheral pulses as well as blood pressures in the arms and ankles are important indicators of the extension of a dissection and its consequences of organ malperfusion. Repeated examinations are important in order to follow the development. A peripheral pulse may disappear, or a pulse deficit may be dynamic and resolve spontaneously, which is reported to occur in one-third of the patients. Such a dynamic course is probably related to redirection of flow from the false into the true lumen after spontaneous fenestration of the aortic septum known as the reentry phenomenon.

A new pulse deficit is found in approximately 60% of the patients.

## 8.4 Diagnostics

An electrocardiogram (ECG) should be obtained in the emergency department. Low voltage might indicate pericardial tamponade, and ST–T wave changes could indicate myocardial ischemia.

The following blood tests should be ordered: complete blood cell count, arterial blood gases, protrombin and thromboplastin times, serum electrolytes, creatinine, blood urea nitrogen, liver enzymes and lactate.

As with the physical examination, repeated blood tests according to the patient's clinical course might be of great diagnostic value during the acute stage of the disease. Mild anemia is common, while severe anemia indicates rupture and bleeding. Hemolysis with elevated bilirubin or lactic acid concentrations can also be found. A leukocytosis with a count of 10,000–15,000 is common. Blood gases might reveal a metabolic acidosis due to anaerobic metabolism in ischemic tissue. Urinary tests showing hematuria indicates renal involvement.

A plain chest x-ray in standard anteroposterior and lateral projections is rarely diagnostic, but the following findings indicates the presence of aortic dissection:

- Abnormal shadow adjacent to the descending thoracic aorta
- Deformity of the aortic knob
- Density adjacent to the brachiocephalic trunk
- Enlarged cardiac shadow
- Displaced esophagus, trachea, or bronchus
- Abnormal mediastinum
- Irregular aortic contour
- Loss of sharpness of the aortic shadow
- Pleural effusion
- Expanded aortic diameter

Helical CT is accurate for determining the presence of an aortic dissection and provides information for classification. The identification of an intimal tear is, however, difficult and motion artifacts of the ascending aorta are sometimes misinterpreted as dissection. MRI is highly accurate and gives valuable information about the pathoanatomy. Unfortunately it cannot be performed in hemodynamically unstable patients who are on ventilator support.

TEE (Transesophageal echocardiography) is often considered as one of the most valuable diagnostic tools, making it possible to determine the type and extent of the aortic dissection, especially distally. It has limitations in visualization of the distal ascending aorta and the arch. TTE (Transthoracic echocardiography) is, on the other hand, superior for evaluating involvement of the proximal part of the descending aorta in the dissection. Together, TEE and TTE yield a sensitivity and specificity approaching 100% for diagnosing dissection and are thus probably the best – but unfortunately often not available – diagnostic modalities.

Aortography is the old gold standard and is highly accurate in diagnosing aortic dissection, but it can fail to recognize a thrombosed false lumen. It also provides better information than CT or MRI about the condition and involvement of the aortic branches. Furthermore, aortography can be combined with therapeutic endovascular management. However, the modern CT scanners with up to 64 detectors can produce extremely detailed images and, when available, should be the first imaging study after the chest x-ray.

## 8.5 Management

### 8.5.1 Treatment in the Emergency Department

As soon as aortic dissection is clinically suspected, aggressive medical treatment must be started immediately. The goals are to (1) stabilize dissection, (2) prevent rupture, and (3) prevent organ ischemia.

These goals can be achieved by diminishing the stress on the aortic wall. Consequently, the therapeutic cornerstone is to reduce blood pressure in order to minimize the force of the left ventricular ejection (dP/dT). The reduction in blood pressure must, however, be balanced against what is needed for adequate cerebral, coronary, renal, and visceral perfusion. A useful guideline is that the systolic arterial blood pressure should be kept around 100–110 mmHg and mean arterial pressure between 60 and 75 mmHg, provided that urinary output and neurology are unaffected.

In the emergency department the following measures can be employed:

1. Insert one or two large-bore intravenous (IV) lines for administering antihypertensive drugs and fluids.
2. Obtain an ECG.
3. Order blood tests as stated above.
4. Obtain a plain chest x-ray.
5. Administer oxygen by mask
6. Consider injection of a strong analgesic IV, such as morphine 5–10-mg.
7. Insert an arterial catheter for blood pressure monitoring.

8. Start administration of a beta-blocker as described below.

The recommended agents for medical management of acute aortic dissection are direct vasodilators, beta-blockers, nitroglycerin and calcium channel blockers if beta blockers cannot be used.

Beta-blockers orally are recommended for all patients. Contraindications for beta-blockers are heart failure, bradyarrhythmias, atrioventricular blocks, and bronchospastic disease.

Suggested emergency medical treatment (local variations in drug choices are of course common) is as follows:

Start propranolol treatment, 1 mg IV, every 3–5 min until achieving a systolic blood pressure around 100 mmHg and a heart rate of 60–80 beats/min (maximum dose, up to 0.15 mg/kg). Continue thereafter with 2–6 mg IV every 4–6 h. In patients with severe hypertension an IV infusion of nitroglycerin is started and the dose titrated after blood pressure and heart rate.

**NOTE**

**The main objective of the medical treatment is to lower the blood pressure to a level of 100–110 mmHg. It is mandatory to check the patient for the development of new complications of the dissection during medical treatment.**

### 8.5.2 Emergency Surgery

Emergency surgery should be considered in type A dissections involving the intrapericardial ascending aorta and the aortic arch. A distal type B dissection with retrograde dissection involving the aortic arch is also a case for acute operation. A double aortic lumen in the pericardial portion of the ascending aorta is an absolute indication for emergency operation. Depending on the patient's general condition prior to the dissection there are, as usual, exceptions from these basic rules. Contraindications include very advanced age and severe debilitating or terminal illnesses.

Surgical repair of the condition requires thoracic surgical expertise and includes replacing the ascending aorta and resecting the primary intimal tear. The operation involves cardiopulmonary by pass. In type A dissection with persistent organ ischemia despite open surgical repair and replacement of the ascending aorta, endovascular treatment of the rest of the dissection is often a successful complement.

### 8.5.3 Type B dissection

The management of acute distal aortic dissection is initially always medical because this results in lower morbidity and mortality than emergent surgical repair. Consequently, the continued regimen for these patients follows the previously given recommendations regarding beta blockade and vasodilators started in the emergency department.

The medical treatment must be combined with careful observation for complications. Surgical or endovascular intervention should be considered for the following situations:

- Aortic rupture
- Increasing periaortic or intrapleural fluid (suggesting aneurysmal leakage)
- Rapidly expanding aortic diameter
- Uncontrolled hypertension
- Persistent pain despite adequate medical therapy
- Organ malperfusion – ischemia of brain, spinal cord, abdominal viscera, or limbs

The goal of surgical repair in a type B dissection is, as with all other treatment options, to prevent rupture and restore visceral and limb perfusion. Because a common site of rupture is associated with the site of primary dissection, at least the upper half of the descending thoracic aorta needs to be replaced in most cases. Graft replacement in the acute setting should be limited and replacement of the entire thoracic aorta avoided if possible. An abdominal fenestration procedure is sometimes necessary to restore flow to the lower extremities. Extraanatomical by pass is another possible way to reestablish flow to the legs.

### 8.5.4 Endovascular Treatment

In patients with peripheral vascular complications due to extension of the dissection into a branch, causing compression and obstruction of its true

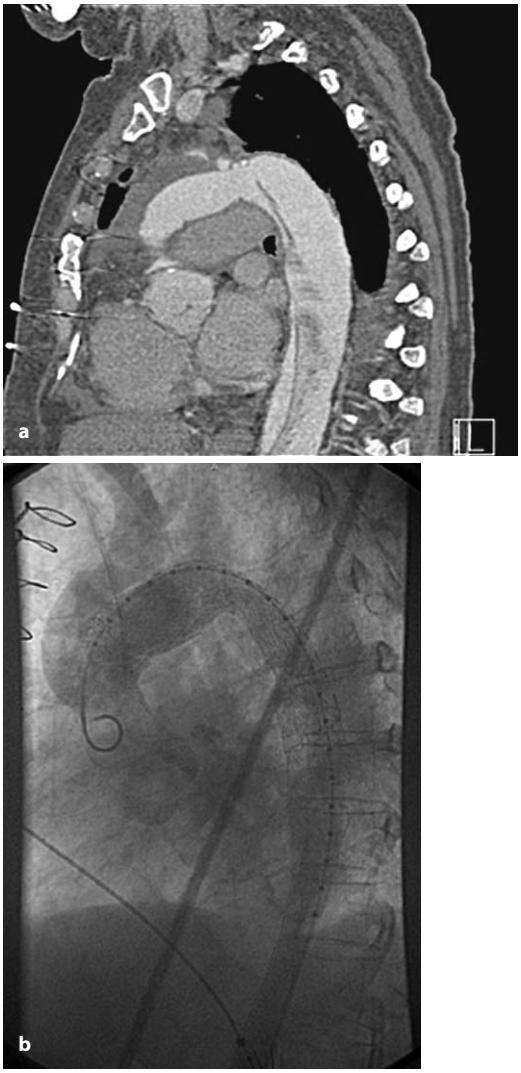


**Fig. 8.4.** **a** Computed tomography showing a type B dissection and its entry in the first part of the descending aorta in a patient with a previous reconstruction of the arch and the brachiocephalic trunk after a type A dissection. The true anterior aortic lumen is severely compressed causing obstruction of the main visceral branches and leading to visceral ischemia. **b** Flow into the true aortic lumen and all branches is restored after deploying a covered stent over the entry site in the descending aorta

lumen, as well as in patients with central aortic true lumen collapse, the endovascular option should be considered. Provided, of course, that the institution has technically skilled physicians, the necessary equipment and back-up support. It is possible to create a fenestration through the intimal flap from the false into the true lumen with endovascular techniques. As shown in Fig. 8.2 a, stenting of the entry site to occlude flow into the false lumen will probably be successful in restoring flow into a branch with its orifice obstructed by the false lumen and the dissection membrane. If there is an avulsion of the intima of that branch as in Fig. 8.2 b, this is not an option.

Endovascular management is developing as an attractive alternative to surgical repair. Patients with an acute type B dissection who are not good candidates for surgery can be considered for endovascular management. Stenting has also been reported to give successful results in aortic collapse with severe ischemia of the lower part of the body.

An endovascular approach can also be used as the initial treatment by performing aortic fenestration and stenting. Most centers prefer to delay either surgical or endovascular repair until after the patient has recovered from the acute phase of malperfusion, whereas others advocate early prophylactic stenting and coverage of the intimal tear to occlude the false lumen and prevent further dissection (Fig. 8.4).

## 8.6 Results and Outcome

A recent article from 12 international centers covering 464 patients with aortic dissection reported, in-hospital mortality rates for type-A dissections treated surgically of 28%, and medically of 58%. The corresponding figures for type B were 31% and 10%, respectively.

Successful closure of the intimal tear with endovascular stent grafts and subsequent thrombosis of the false lumen is reported in up to 75% of patients. Branch occlusions with ischemic symptoms were relieved in 75–95% of the cases. Survival after 30 days was 75–85 %, and long-term results are good, with <1% related deaths and verified thrombosis of the false lumen in 100% of the survivors. No thromboembolic complications oc-

cured. In general, survival is lower for patients with paraplegia or visceral or renal ischemia. In cases with type B dissections and indications for surgical intervention, the results of endovascular intervention seem more favorable compared with conventional surgical repair, but the number of reported cases from any single center is still low. In all cases, long-term follow-up regarding development of aneurysms and continued antihypertensive medication is essential.

## Further Reading

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