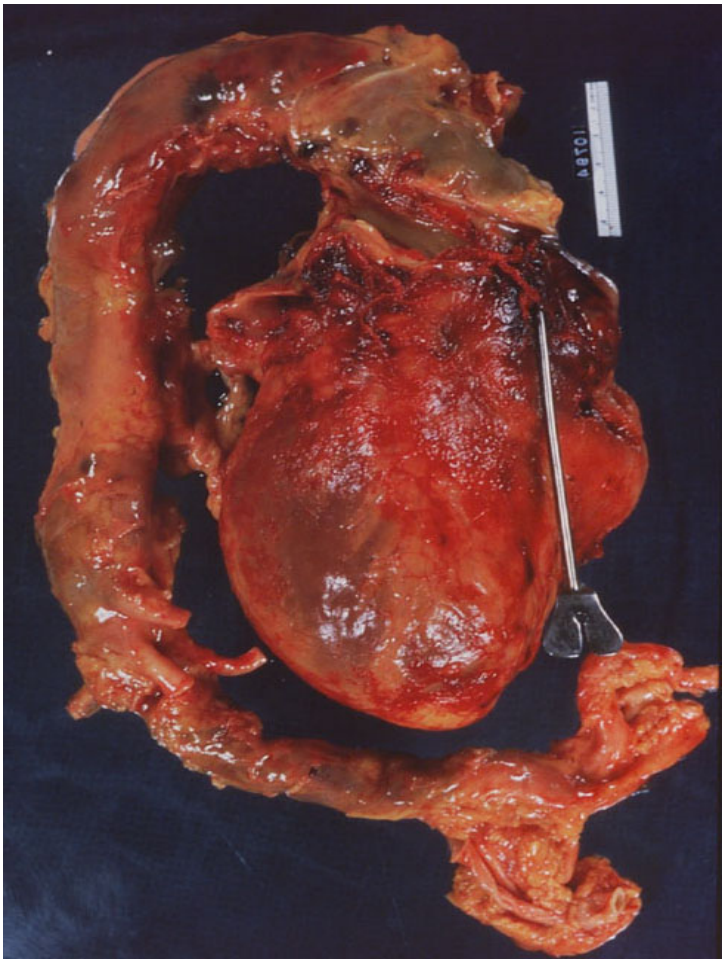
Surgical Strategies in Acute Type A Dissection

An Educational Guide



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1 Epidemiology of Aortic Dissection

1.1 Background

Acute Type A dissection is a catastrophic problem with a dismal outlook without surgery (Figure 1).[1] The overall prevalence is between 5-10 cases per million people worldwide with more than 2000 cases reported per year in the USA and between 400-500 cases reported annually in the UK. More common than ruptured abdominal aortic aneurysm, men suffer 2-3 times more frequently than women with a peak incidence occurring between the ages of 40 and 69 years. Five percent of patients diagnosed with acute Type A aortic dissection have Marfan syndrome with dissection occurring at a mean age of 36 years in this population. Thirty percent of untreated patients are dead within 24 hours and 50% by 48 hours.[2] Left untreated, the mortality rate at 3 months is 90%. Thus, any acute dissection involving the ascending aorta should be considered a surgical emergency.

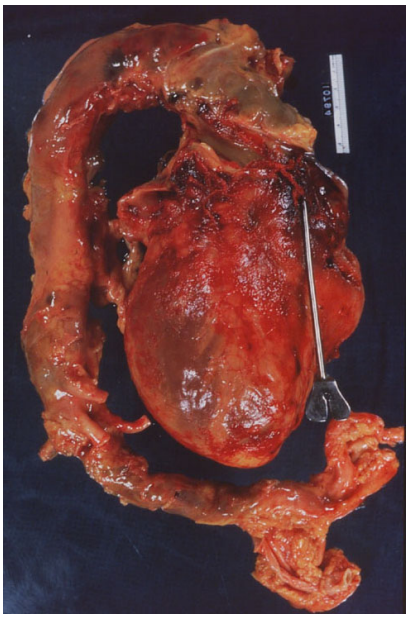


Figure 1. Pathological specimen from a patient who died from rupture of an acute type A dissection into the pericardium (at site of probe). The dissection extends throughout the aorta and into the iliac arteries.

1.2 Clinical Presentation

Myocardial infarction is 20 times more common than aortic dissection. Given the commonality of this event and the symptomatic similarities to aortic dissection, it most often is the cause for the misdiagnosis or delayed diagnosis of aortic dissection. Contributing to the confusion with myocardial infarction, ECG abnormalities are found in more than 50% of dissection patients. These include ST/T wave changes, evidence of left ventricular hypertrophy, conduction defects, pericardial involvement and arrhythmias. Unfortunately, the misdiagnosis of myocardial infarction may have fatal consequences when thrombolysis or antiplatelet therapy is initiated. A severe bleeding diathesis greatly reduces the likelihood of successful surgical repair.

Type A dissection characteristically presents with sudden searing central chest pain with radiation to the neck, back or abdomen.[3] Frequently the patient has a history of hypertension. Typically, early death will occur through rupture of the thinned external layer into the pericardial sac (Figure 2).

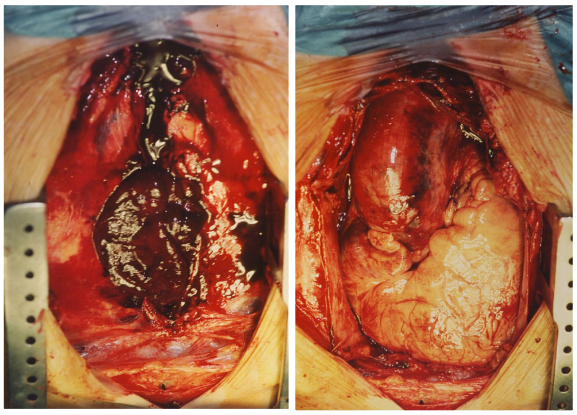


Figure 2. Patient with tamponade and cardiogenic shock. When the pericardium is opened, blood clot extrudes under tension and the blood pressure rises. Further opening reveals the surgeon's characteristic view of acute type A dissection.

Alternatively, dissection may rapidly occlude the carotid or coronary arteries, resulting in the patient presenting with stroke, unconsciousness or myocardial infarction (Figure 3).

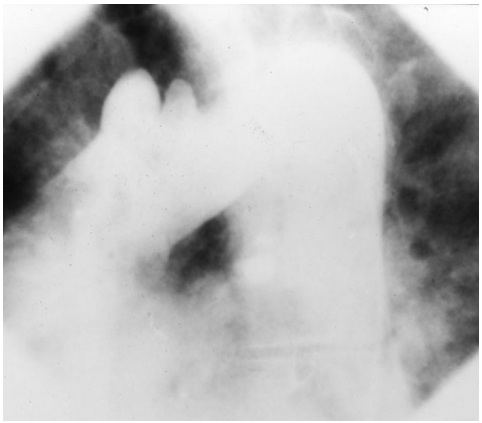
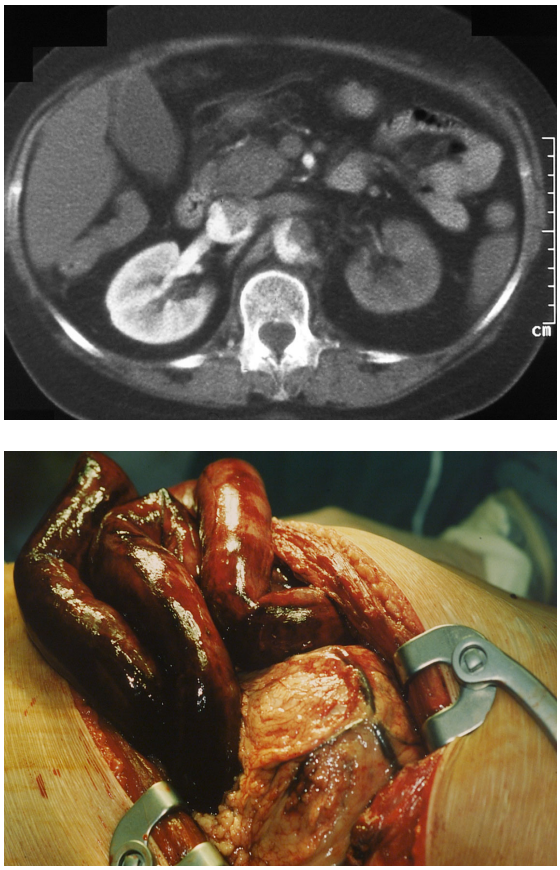


Figure 3. Angiography from the days when it was still applied in this condition. The brachiocephalic vessels are occluded by pressure in the false lumen. The patient presented with stroke.

Aortic regurgitation occurs through prolapse of unsupported right or non-coronary valve cusps in up to 70% of patients. In some instances, the left ventricle may dilate acutely, causing pulmonary oedema. In almost half of patients, diminished, delayed or absent femoral pulses are reported with a spectrum of neurological events such as syncope, visual disturbances, monoplegia, hemiplegia, recurrent laryngeal nerve palsy or Horner's syndrome. Dissection propagated into the descending thoracic and abdominal aorta may cause paraplegia, acute renal failure or mesenteric ischaemia (Figures 4a, b).



Figures 4a, b. (a) Malperfusion in the dissected abdominal aorta. The contrast computerised tomographic scan shows one renal artery to be occluded with acute failure of the involved kidney. (b) Ischemic and necrotic bowel caused by dissection of the mesenteric arteries. This is usually a fatal complication.

Other clinical manifestations include left sided pleural effusion through leakage of blood across the outer layer of the dissected aorta, superior vena caval obstruction, and an ischemic limb or disseminated intra-vascular coagulation.

1.3 Predisposing Factors

Eighty percent of patients diagnosed with aortic dissection are more than 40 years of age and are hypertensive.[4] Those younger than 40 years of age have a predisposing vascular defect such as Marfan's syndrome, a bicuspid aortic valve, or coarctation of the aorta. Other inherited conditions that are also predisposing factors include Turner's syndrome, Noonan's syndrome and the Elhers-Danlos syndrome.

Although Erdhiem coined the phrase "cystic medial necrosis," the aorta in Marfan's syndrome shows neither cysts nor necrosis.[5, 6] In patients diagnosed with Marfan's syndrome, the changes in diameter of the ascending aorta from diastole to systole are significantly reduced owing to decreased distensibility and greater wall stiffness. This results from decreased elasticity of the media through genetic deficiency of fibrillin, which scaffolds the elastic lamellae. Flow-mediated vasodilatation through nitric oxide production is deficient. In Marfan patients presenting with acute aortic dissection, there is separation and fragmentation of the various medial layers of the aorta, but primarily of the elastic fibres. Wall weakening occurs through the accumulation of collagenous and mucoid material which is most prominent in the aortic sinuses and ascending aorta. This part of the aorta is subject to the greatest pulsatile expansion and stress during systole. Rate and degree of aortic expansion are dependent upon the intra-luminal pressure. The normal systolic pulse wave generates shear stresses that cause the aortic intima to move fractionally over the medial layer. The greater the left ventricular ejection velocity and systemic arterial pressure, the greater the shear stress. An eccentric jet from a bicuspid valve may amplify the shear stress as does volume overload and hypertension in pregnancy. Dilatation of the ascending aorta increases wall tension and frequently precedes dissection in both Marfan and hypertensive patients.[6] In the latter the medial layer may be normal in structure but is overwhelmed by the increased mural stress caused by elevated pulse pressure. Autopsy studies show that 9-13% of dissection patients have a bicuspid aortic valve compared with only 1-2% in the general population.[7]

The pathological anatomy of dissection has an important bearing on the surgical strategy. A transverse intimal tear with longitudinally propagated distal dissection through the medial layer is found in 95% of cases.[7] In 70% of patients, the tear is located in the ascending aorta on the convex surface 2-3cm beyond the aortic valve and involves 50-70% of the circumference (Figure 5a, b).

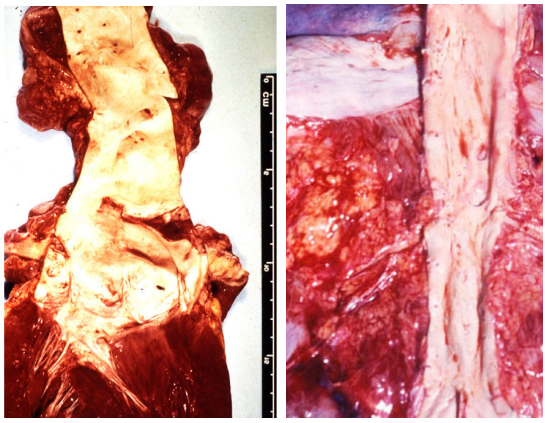


Figure 5a, b. Primary tear in the ascending aorta and propagation of the dissection through the descending thoracic and abdominal aorta.

In 20% of patients, the tear is located in the descending aorta (Type B) and in the remaining 10% of patients, it is in the aortic arch. In all instances, the dissected channel is situated in the outer half of the media, leaving a very thin external wall with a thickness 20% of the original media. Once within the media, the dissection propagates around the right lateral wall and greater curvature of the distal aorta. Atheromatous plaques may interrupt the longitudinal process of dissection in older patients whereas propagation is unhindered in the young with medial disease.

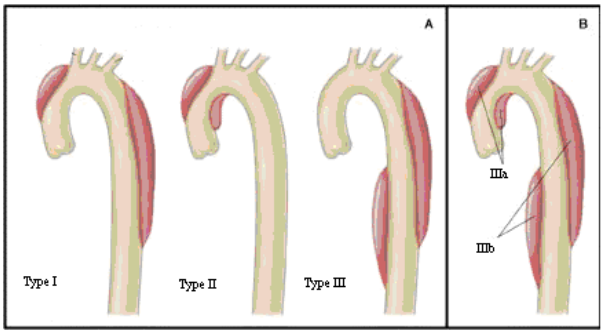


Figure 6. DeBakey Classification System (a) Type I, II, III (b) Type IIIa, b.

1.4 Classification

The extent of a dissection is classified using one of two systems. The DeBakey classification system is an anatomical description of the aortic dissection (Figure 6). This system takes into account the location of the original intimal tear and the extent of the dissection. Dissections are classified as Types I, II, or III. Type I dissections originate in the ascending aorta and extend to at least the aortic arch. Type II dissections originate and are confined to the ascending aorta. Type III dissections originate in the descending aorta (rarely extending proximally) and extend distally. The Stanford classification system divides dissections into two groups (Type A or B dissection) (Figure 7). This system has the advantage of simplicity because it distinguishes between conditions with a different natural history and management.[8] Stanford Type A dissections involve the ascending aorta (same as DeBakey Type I and II) whereas Type B (same as DeBakey Type III) are confined to the descending aorta beyond the left subclavian artery. In Type A dissection the tear is found in the ascending aorta in 85% of patients, in the arch in 6% and in the descending aorta in 5%, but with retrograde propagation to the ascending aorta.

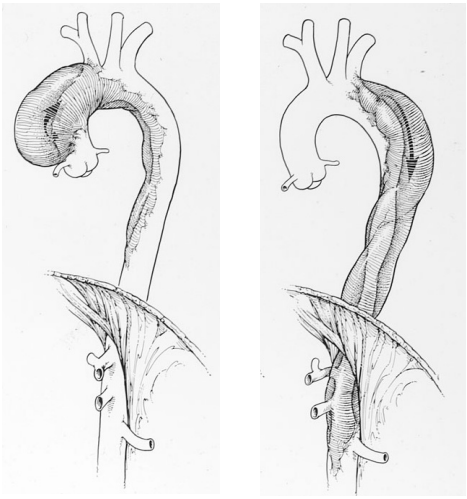


Figure 7. Stanford Classification System.

1.5 Evolution of Diagnosis and Surgical Treatment

Laennec coined the term "l'aneurysme dissequant" in 1826, though the condition had been recognised by Morgagni in the 16th century and King George II was found to have died from aortic rupture into the pericardium as a result of an aortic dissection.[9] Elliotson (1830) described the characteristic transverse intimal tear with distal splitting throughout the length of the aorta after which Peacock suggested that "distal re-entry tears were an imperfect natural cure of the disease." The first diagnosis of dissection in a live patient was by Latham in 1855. A 51-year-old man presented with severe chest pain followed by loss of power in the legs and unconsciousness. Leg pulses were absent and dissection was confirmed at autopsy. Aortic dissection remained poorly understood until the comprehensive review by Shennan in 1934.[10]

Surgical treatment began in the 1950's with attempts to fenestrate the dissected abdominal aorta to salvage ischemic legs. Later, attempts were made to revascularise occluded branch vessels or to wrap the aorta in an attempt to avoid rupture. As for most types of thoracic aneurysm, the pioneering surgery that

evolved into the surgical techniques used today occurred in Houston. DeBakey (1954) directly approached an aneurysmal descending aortic dissection, closed the false lumen and performed an end-to-end anastomosis.[11] He then described the DeBakey classification of aortic dissections – Types I, II and III. It was not until 1953 after the introduction of cardiopulmonary bypass by Gibbon that surgery of the ascending aorta became possible. Spencer (1962) described the first successful repair of a chronic type A dissection with aortic regurgitation.[12] In this instance, the patient's morphologically normal aortic valve was preserved by re-suspension. Morris and colleagues at Baylor (1963) were the first to repair an acute type A dissection.[13] This patient was followed with residual moderate aortic regurgitation until 1977 when elective valve replacement was performed. The false lumen remained patent but without aneurysm formation. In 1965, Wheat made a major contribution to cardiac surgery by emphasizing the need for controlling blood pressure prior to surgery.[14] This soon became the treatment of choice for uncomplicated descending (Type B) aortic dissection. Prosthetic aortic valves emerged in the 1960s and the important concept of composite prosthetic aortic root replacement was described by Bentall and DeBono in 1968.[15] The range of operations for dissection was extended by David (1992) with the introduction of aortic valve sparing root replacement surgery, first used electively for ascending aortic aneurysm repair.[16] This has become the procedure of choice for young Marfan patients who present with dissection.

2 Current Surgical Strategies

2.1 Preparation for Surgery

When acute Type A dissection is suspected, the systolic blood pressure should be carefully regulated and maintained below 110mm Hg using an intravenous beta blocker or glyceryl trinitrate. This will minimise the risk of rupture into the pericardium. Ongoing blood pressure control is particularly important during transoesophageal echocardiography, computerised tomography or magnetic resonance imaging as anxiety during these investigations causes blood pressure to rise.[17] Some patients present in shock with cardiac tamponade and are unable to maintain adequate systemic perfusion. In this case, intravenous adrenaline should be used and the patient urgently transferred to the operating room. The intra-aortic balloon pump is contraindicated in this scenario due to the weakened aortic wall. Transoesophageal echocardiography can then be used to confirm acute Type A dissection as the patient is anaesthetised.[18] Coronary angiography is not recommended unless the patient has a history of angina or previous coronary surgery and remains haemodynamically stable. Rarely, needle pericardiocentesis or an urgent sub-xiphoid window is required to relieve cardiac tamponade.

It is also important to avoid hypertension during anesthesia. Anaesthetic agents such as Fentanyl provide analgesia and suppress noxious reflexes whilst Isoflurane or vasodilators such as glyceryl trinitrate provide effective blood pressure control. It is important to optimise cerebral protection during hypothermic circulatory arrest which can be achieved by careful metabolic management. The pH/CO_2 balance is maintained by the alpha-stat method which preserves coupling between cerebral blood flow, metabolic rate and cerebral oxygen uptake. At our institution, we use a barbiturate (thiopentone) and the brain specific calcium channel blocker Nimodipine to attenuate cerebral reperfusion injury. Others use methyl prednisolone (30mg/kg) to decrease intercranial pressure. Hyperglycaemia is often difficult to avoid as hypothermia causes insulin resistance. Hyperglycaemia increases brain lactate concentration which worsens outcome in stroke victims; it is therefore important to use glucose-free infusions.

Another major issue is the patient's coagulation status. First, the extensive vascular injury and clotting in the false lumen can cause significant defibrination or frank disseminated intravascular coagulation. Second, many patients are mistakenly prescribed aspirin, Clopidogrel or both, according to "chest pain" protocols. The combined use of anti-platelet agents and defibrination can result in uncontrollable bleeding, massive transfusion requirement and multi-organ failure.

Patients with cardiac tamponade often deteriorate during induction of anaesthesia so it is important to treat these patients with adrenaline or noradrenaline accordingly. The author prefers to cannulate the right subclavian artery for arterial perfusion but the femoral artery is used in more urgent circumstances. As it may be difficult to locate the femoral artery in hypotensive patients, it is important to mark its location before skin preparation. The pericardium usually contains free blood and sometimes relief of cardiac tamponade results in a surge of blood pressure and rupture of the false lumen. Because of this, the subclavian or femoral artery is cannulated before the pericardium is opened in tamponade patients. Venous return can then be established quickly with a two stage cannula inserted through the right atrial appendage.

2.2 Surgical Treatment

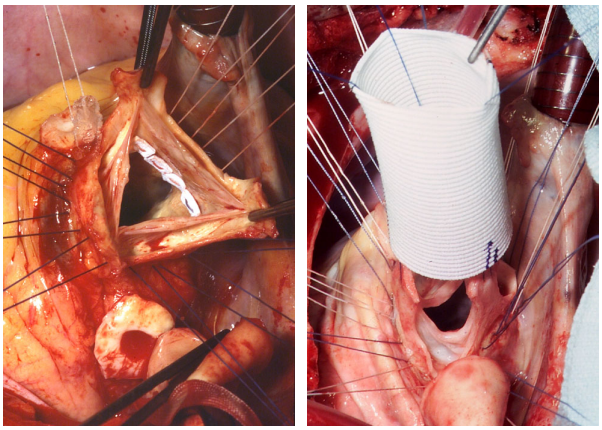
The safety and long-term success of aortic dissection surgery depends upon complete resection of the primary intimal tear with attempted obliteration of the false lumen at both the proximal and distal anastomoses.[19] This may require extension of the

aortic resection into the arch. In principle, total aortic arch replacement is only required in patients with extensive arch disruption and peri-aortic haematoma, though Japanese protagonists have enthusiasm for this radical approach.[20]

The procedure undertaken depends upon the extent of the aortic pathology, whether or not the patient has Marfan's syndrome (or similar aortic disease) and on the experience of the surgeon. Table I details the options for the management of the dissected aortic root. The aortic valve is morphologically normal in more than 85% of patients, but aortic regurgitation (Grade 2-4) occurs in 60% of cases when dissected aortic sinuses cause cusp prolapse. Bicuspid aortic valve is found in 9-13% of patients and can be preserved if not calcified, severely regurgitant or stenotic. Preservation of the native aortic valve avoids prosthesis-related complications in the long term.[21] Valve conservation is equally important in Marfan patients, though the surgeon must have elective experience with David's valve sparing aortic root replacement.

An algorithm summarising management of the aortic tear is shown in Table II. Clearly there should be a good reason to replace the aortic valve. In approximately 40% of patients there is no aortic regurgitation and the sinotubular junction and aortic root are virtually intact. Under these circumstances the native aortic valve is retained for all patients unless there is pre-existing aortic stenosis when dissection originates in the post-stenotic dilatation. If a bicuspid valve is heavily calcified and stenotic it should be replaced. The whole primary tear is resected continuing into the aortic arch if necessary. All patients are operated upon under conditions of hypothermic circulatory arrest without an aortic cross clamp in place so that the arch can be inspected.

For the majority of patients where dissection extends into the aortic root (usually to the non-coronary and right coronary sinuses), the native aortic valve will prolapse, causing aortic regurgitation. The valve must then be re-suspended in order to restore competence. Again the ascending aorta is excised to remove the primary tear and the dissected ends reconstituted. The ascending aorta is then replaced with a supracommisural Dacron® tube graft up to the innominate artery or extending into the inferior aspect of the arch (hemi arch replacement). Only for Marfan patients and those with related aortopathy is full aortic root replacement necessary. Under these circumstances the ideal strategy is to preserve a tricuspid native aortic valve using the David procedure of valve re-implantation (Figures 8a, b, c).[16]



Figures 8a, b, c. David procedure for valve sparing aortic root replacement.

The alternative is prosthetic aortic root replacement using a valved conduit and the classic Bentall procedure (Figure 9).[15]

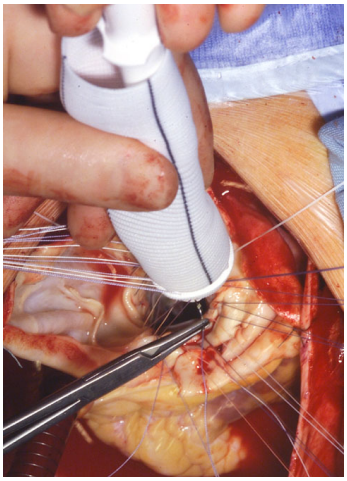


Figure 9. Prosthetic valve conduit aortic root replacement as described by Bentall and DeBono.

Both operations involve mobilisation of the coronary ostia from the dissected aorta and re-implantation into a Dacron graft. Whilst it is preferable to preserve the native Marfan aortic valve and avoid subsequent

prosthetic valve complications, this is a specialised procedure with which the on-call surgeon may not have familiarity; if this is the case, the well-tested Bentall operation is a reliable and durable alternative.

In practice, more than 90% of patients require only the simple approach of ascending aortic resection, reconstitution of the dissected layers and a supracommisural tube graft.[19] With careful peri-operative management, this can be achieved with very low hospital mortality.

2.3 Ascending Aortic Replacement

The key to a secure ascending aortic replacement is complete transection of the dissected aorta proximally and distally together with excision of the intimal tear, reconstitution of the dissected layers, and the application of BioGlue® Surgical Adhesive (CryoLife, Inc.) to expedite the repair process. Different surgeons have clear preferences with regard to the sequence of the operation.[19] The description which follows has resulted in a consistently low mortality rate (6%) over 20 years in more than 150 aortic dissection operations.

Soon after beginning cardiopulmonary bypass via the subclavian or femoral artery, the surgeon should confirm with the anaestheologist that the head is cooling. Should malperfusion be identified within the first few minutes of perfusion, the arterial cannula should be switched immediately to the left ventricular apex and passed through the aortic valve. This is important, as malperfusion may occlude the innominate or left common carotid artery and cause perioperative stroke (Figure 10).

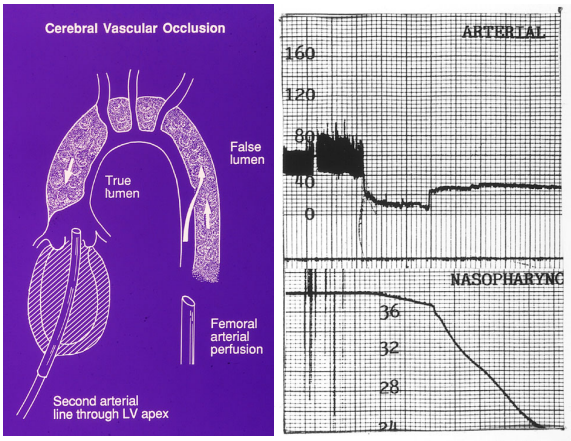


Figure 10. Mechanism of cerebral malperfusion during perfusion from the femoral artery in the dissection patient. The chart shows very low perfusion pressure in the brachiocephalic vessels (measured in the right radial artery) and failure of the head to cool (nasopharyngeal temperature) for virtually 15 minutes until the perfusion cannula was switched to the apex of the left ventricle.

Though rarely necessary, this manoeuvre can decompress a pressurised false lumen and restore cerebral blood flow. Perfusion is used to reduce the systemic temperature to 18°C. During the cooling period, an aortic cross clamp is applied at least 2cm proximal to the innominate artery at a position where the clamped aorta can be completely excised. The rationale for cross clamping is that the proximal repair can be undertaken during cooling following which only a short period of hypothermic circulatory arrest is required for an open ended distal repair. Hemostasis can then be achieved during rewarming. A theoretical danger to clamping is new pressurisation of the false lumen with cerebral malperfusion. This can be ruled out using EEG or cerebral oximetry recordings.

With the cross clamp in place, a longitudinal incision is made through the dissected layers to provide visual confirmation of an ascending aortic tear and clear definition of the level of the sinotubular junction. The aorta is then completely transected at around 0.5cm above the sinotubular junction and commissural pillars. Care is taken not to enter the right pulmonary artery behind the aorta. With the aorta transected, the morphology of the aortic root and native aortic valve are defined and cardioplegia can be delivered directly into the coronary ostia. In approximately 30% of patients, there will be no dissection into the aortic root and no significant aortic regurgitation. In 70% of patients, the dissection enters preferably the right and non coronary sinuses. Usually the left coronary ostium and sinus are not dissected. In contrast, dissection frequently encircles the right coronary ostium. Separation of the dissected layers allows the native aortic valve to prolapse. If the native valve was normal before the dissection or the patient has a well-functioning biscuspid valve, the valve should be conserved by reconstituting the dissected aortic wall. The thinned outer layer tends to expand so careful judgement is required to provide a symmetrical valve resuspension. All blood clot must be removed from between the layers following which a Teflon pledgetted mattress suture is used to approximate the layers above each commissural pillar. Once symmetry of the valve is obtained, a thin layer of BioGlue is applied into the false lumen taking care not to allow glue to enter the aortic sinuses or coronary ostia (Figure 11).

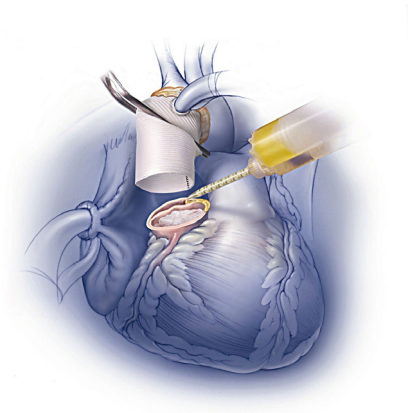


Figure 11. Use of CryoLife's BioGlue to reconstitute the dissected layers.

A gauze swab placed within the root will prevent this. The dissected layers are then held together carefully for approximately for 3-4 minutes while the glue sets and obliterates the space. Unlike many other sealants on the market whose strength is marginal in comparison, BioGlue also serves to increase the suture-holding capacity of the dissected layers. When a Teflon® ring is thought necessary, this is best approximated circumferentially to the aortic root before the graft is sewn into place. The diameter of the Dacron graft is chosen carefully to directly fit the reconstituted sinotubular junction. The graft should not be oversized or significantly undersized. It is sewn to the reconstituted root with continuous 3/0 polypropylene taking care to achieve full thickness bites through secure tissue. Using this technique, completion of the proximal anastomosis is usually achieved in less time than required for the patient to be cooled to 18°C.

At 18°C, the patient is tipped head down, perfusion is stopped and blood drained into the oxygenator. The distal aorta is then completely transected above the site of the cross clamp. This is necessary because the cross clamp often lacerates the intimal layer. In the majority of patients, the primary tear will be restricted to the ascending aorta and excluded by this process. The aortic arch is then carefully inspected. Excision or repair is required should the primary tear extend into the arch. When the primary tear originates in the arch or proximal descending aorta, then complete arch replacement may be required. In practice, around 60% of patients will undergo anastomosis at the level of the distal ascending aorta, a further 30% require replacement of the underside of the arch while less than 10% will undergo full aortic arch replacement. In each case the distal aorta is repaired in the same way. It is completely transected to allow placement of a circumferential cuff of Teflon outside the aortic lumen. The dissected aortic wall is carefully repaired using BioGlue and then the distal end of the Dacron graft is sewn to the reconstituted aorta. The duration of hypothermic circulatory arrest required to perform the distal anastomosis is reproducibly less than 30 minutes rendering cannulation and perfusion of the brachiocephalic vessels unnecessary. An alternative is to cannulate the right subclavian artery then clamp the root of the innominate artery so that cerebral perfusion can be continued when the rest of the circulation is arrested. Systemic cooling can then be limited to 24°C. However, the authors prefer not to clamp the innominate artery because this may also be dissected.

Upon completion of the repair, the aorta is refilled (with the patient tipped head down) and cardiopulmonary bypass recommenced with rewarming. Should initial perfusion be obtained using a femoral artery, it is preferable to switch the arterial cannula to the ascending aortic graft. In this case a graft specifically constructed with a perfusion side limb is employed to replace the ascending aorta. During rewarming, the repair can be inspected for bleeding and extra sutures applied where necessary.

2.4 Complete Aortic Arch Replacement

Only experienced aortic surgeons should undertake complete aortic arch replacement in dissection patients. The objective is to completely excise the primary arch tear by individually transecting the innominate, left common carotid and left subclavian arteries rather than to maintain a dissected Carrell button of aortic arch as is used in aortic arch aneurysm repairs. The proximal descending aorta is carefully reconstituted as previously described and the whole arch replaced with a three branch graft. As an alternative, the authors retain the distal arch and subclavian artery, joining the aortic root to the distal arch and then applying a separate bifurcated graft to the innominate and left common carotid arteries. The bifurcated graft is then joined to the ascending part of the initial graft. As hemostasis is particularly important for the less accessible distal parts of the repair, the application of a thin layer of BioGlue ensures the suture lines are sealed. Following complete aortic arch replacement, a brief period of retrograde cerebral perfusion via the superior vena cava ensures the removal of air from the brachiocephalic vessels.

2.5 Surgical Results/Discussion

A landmark paper from Miller (1979) showed a hospital mortality rate of 38% (range 34-41%) after surgical treatment.[22] For a selected group managed medically, the hospital mortality rate was 83% (CL 77-87%). The next 15 years produced substantial improvements in operative technique, yet the hospital (30 days) mortality rate in even the most experienced centres ranges from 6 to 25%. The mortality rate is least in those requiring replacement of the tubular

portion of the ascending aorta and increases with the need for aortic arch and aortic root replacement.[19, 23,24] Factors contributing to mortality rate are preoperative diabetes and coronary artery disease. The use of streptokinase and delayed operation predispose to postoperative multisystem failure.[25] Other important preoperative risk factors include presentation with cardiogenic shock, use of thrombolysis with a mistaken diagnosis of myocardial infarction, acute myocardial ischemia and acute renal failure caused by the dissection. Specifically, cardiopulmonary bypass prolonged more than 90 minutes is a significant risk factor.

Hospital mortality typically results from left ventricular failure, renal failure and stroke. Coronary occlusion (usually the right coronary artery) occurs in 2-5% of patients and causes myocardial infarction if operation is delayed. Preoperative stroke resolves in up to 50% of patients after successful repair, but postoperative mortality is substantial in these patients. Intraoperative cerebral malperfusion must be detected at an early stage and is addressed by changing the site of the aortic cannula or by establishing retrograde cerebral perfusion. Bleeding is now less of a problem with the use of surgical sealants such as BioGlue and impervious vascular grafts. Established mesenteric ischaemia usually proves fatal and is associated with delayed operation. This should be suspected in severely acidotic patients with a white cell count exceeding 20,000/mL, especially when dissection occurs more than 2 days before surgery.

The International Registry of Acute Aortic Dissection, which draws on data from 12 international referral centres, reports a 26% hospital mortality rate for operative patients. Suggestions that more patients should undergo complete aortic root replacement or valve-conserving aortic root replacement, both of which involve coronary button mobilisation and implantation, must be reviewed with scepticism. The surgeon faced with a dissection patient whilst on call at night must perform an operation which he has the ability to complete successfully within a reasonable time frame. Since random allocation of surgeon by rota is a risk factor for mortality, a specialist team approach for aortic dissection is advisable. Perioperative complications must be managed aggressively and the surviving patient followed regularly with non-invasive imaging to detect aortic valve regurgitation or distal aneurysm formation.

3 Long-term Results

The long-term outlook after successful primary operation remains poor.[26, 27] Ten-year survival for DeBakey type I and II dissection are 29 and 46%, respectively. Sudden death occurs in up to 25% of patients while a further 40% die from myocardial infarction or stroke. About 20% of patients die as a result of a rupture of another aortic segment and this rate is substantially greater in patients with Marfan's syndrome. The late aortic complications that require re-operation (in 15-20% of patients) stem from persistence of the aortic false lumen distal to the repair. MRI studies show persistent blood flow in the false channel in 80% of patients and this may eventually lead to aneurysm formation. Careful follow-up by MRI or CT scans are advisable at 6-month intervals.[28] All dissection patients should be kept on long-term beta-blockade to delay aortic expansion.

4 Conclusion

Despite these dismal statistics, successful patient outcomes (low hospital mortality and reoperation rate) can be achieved with the use of conservative pathology-oriented surgery. This includes an open distal anastomosis and complete excision of the primary tear in all patients. Root repair with preservation of the native valve is achievable in more than 90% of patients. The use of BioGlue can simplify the repair process by reinforcing friable tissues and sealing anastomotic suture lines. Careful heamostasis minimizes the need for excessive red cell and coagulation product usage, which contribute to patient morbidity. In an environment where hospital survival is the single most important goal, more extensive procedures should be performed by surgeons who are more experienced in aortic surgery techniques.

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6 Tables

Table I.

Options for Management of the Dissected Aortic Root

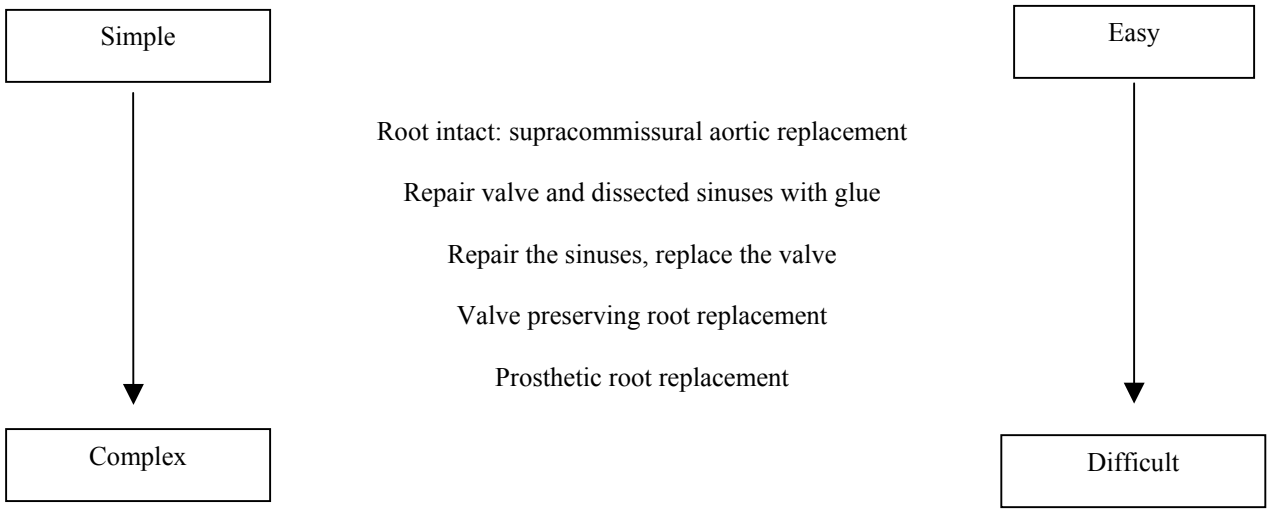
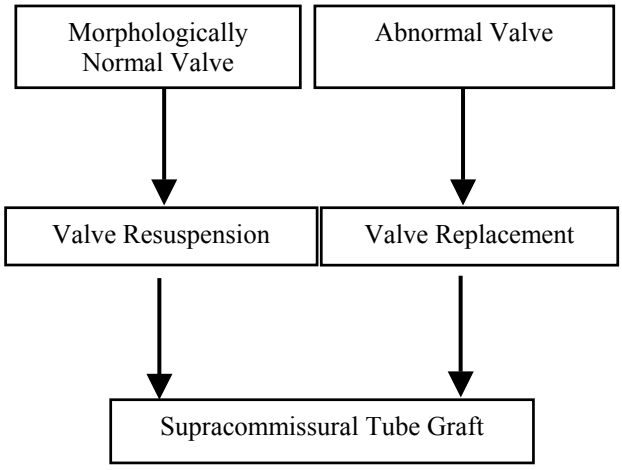


Table II.

Surgical Strategies
In Acute Aortic Root Dissection

SIMPLE



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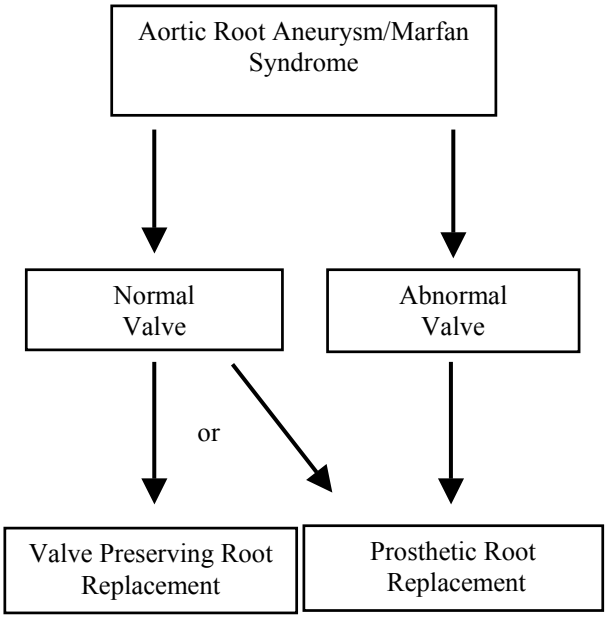

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