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Recent changes in the management of aortic dissection

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The first description of aortic dissection was made following the death of King George II of Great Britain in 1760, when Frank Nicolls, the King’s personal physician, was ordered to open and embalm the body. It was not until the 1950s that DeBakey and colleagues in Houston, Texas, performed the first successful surgical treatment of this fatal condition.

The incidence of aortic dissection is 3–4 per 100,000 population in the UK and US. In this issue of the Journal, Gupta et al. neatly described the burden of type A aortic dissection in the Māori and non-Māori population in the Midland DHBs region. They report that the prevalence in Māori was higher than non-Māori and the average age at presentation was 5 year lower in Māori.

Classification of aortic dissection was first described by DeBakey as three distinctive types (I, II and III). A few years later, it was noted that the prognosis and treatment differed depending on if the arch was involved. Hence, a second classification emerged from the Stanford group categorising dissection into two types (A and B). DeBakey type I is when the dissection involves the ascending arch and descending aorta, and type II is when the dissection is confined to the ascending aorta. This corresponds to the Stanford type A. When the dissection starts distal to the left subclavian artery, it is classified as a Stanford B or a DeBakey type III. The Stanford classification is the more commonly used clinically for its relative simplicity. However, the DeBakey classification provides a better anatomical and descriptive classification which influences surgical management and follow-up surveillance.

Risks of dissection starting in the ascending aorta (Stanford A, DeBakey I and II) are aortic rupture into the pericardium with cardiac tamponade and acute aortic regurgitation and death. Untreated, the rate of mortality is 80%, therefore surgical treatment is the ‘gold standard’. In Stanford type B or DeBakey III, the dissection can cause organ malperfusion and aortic wall weakness, which can lead to aortic dilatation, aneurysm formation with risk of rupture. The management depends on the clinical presentation.

The management of aortic dissection has seen significant changes in the past two decades. This is a result of a better understanding of the dissection process and natural history, improvement in radiological imaging, stent graft technology and the advent of endovascular surgery. The natural history of dissection can be complex. The creation of true and false lumens by the dissection flap, the dynamic relationship to each other and the size of the two lumens in conjunction to the effect this has to the blood flow into the head and neck, the viscera and the spinal arteries. These factors play an important role in the management of aortic dissection and are critical to achieving acceptable outcome. It is the understanding of these concepts that shaped an alternative approach to the management of acute dissection, particularly in type A or DeBakey I & II dissections.

The treatment of type A dissections has evolved from medical therapy, with 80% mortality, to surgery, which carries 20% mortality. The goal of treatment is to prevent cardiac tamponade and/or acute aortic regurgitation, which are the primary causes of death. The traditional surgery, as recommended by DeBakey, is replacing the ascending aorta, with or without aortic valve re-suspension. However, this treatment only addresses the proximal part of the dissection and not the distal extension, whether the...
dissection stops in the arch or extends distally. Although treating the ascending aorta may suffice, it is unpredictable how the distal dissection will behave. Progressive collapse of the true lumen and expansion of the false lumen, despite surgery, could cause organ malperfusion in the short term and aneurysm formation in the long-term—both are challenging consequences to manage. As a result, surgeons have varied their approaches to surgical treatment of type A dissections, taking into consideration the new endovascular techniques and dynamic imaging.

In many centres, replacing the ascending aorta is still an acceptable treatment option with excellent outcomes. However, if further treatment is required at a later stage that involves re-do surgery, it becomes major undertaking. Therefore, alternative approaches that facilitate easier surgical or endovascular interventions are considered, should complications or progression of dissection occur, thereby avoiding re-do surgery. “Frozen Elephant Trunk”, De-branching and Hybrid procedures are strategies to address the distal dissection and future intervention (if required) could be performed by endovascular means. The decision on the particular approach depends on the surgeon preference and experience, the centre's expertise, and complications of dissection. The surgeon may weigh the risk and benefits of performing acute complex surgery against potentially higher mortality and morbidity.

Frozen Elephant Trunk and Hybrid procedures have the advantage of dealing with the proximal aortic and arch dissection and the supra-aortic vessels. These methods will direct blood flow into the true lumen, causing collapse of the false lumen, with better aortic remodeling. Moreover, this simplifies the management of the distal dissection by endovascular means in the future. Even the type A dissection that was treated by the traditional surgery of replacing the ascending aorta but continued to progress leading to organ malperfusion or acute aortic aneurysm could be treated by skillful endovascular intervention. This would involve de-branching of the supra-aortic vessels, chimney graft and TEVAR (unpublished data).

Endovascular surgery is advancing and there are numerous options available to the endovascular interventionist. While de-branching of supra-aortic branches with thoracic endovascular aortic repair (TEVAR) is one approach, other approaches include custom-made fenestrated TEVAR for the supra-aortic vessels. One new technology is the Arch Branch graft (Cook Zenith) for the arch aneurysm. This has fenestrations for the brachiocephalic and left common carotid artery. This graft is in trials and the results are awaited. More challenging is the availability of endovascular stent grafts for treatment of primary type A dissections (Ascend Endovascular Graft by Cook Zenith). It is estimated that 10–30% of the type A dissections are not fit for traditional surgery and such patients could be offered endovascular stent-grafts. Moreover, it is estimated that up to 50% of type A dissections would be potential suitable for a stent graft based on CT aortogram.

In Stanford type B or DeBakey III, the presentation is broadly divided into complicated (end organ malperfusion, rupture, refractory pain and uncontrolled hypertension) and uncomplicated dissection. The dissection can cause organ malperfusion and aortic wall weakness leading to aortic dilatation, aneurysm formation and risk of rupture. Its management depends on the clinical presentation.

Type B dissection was treated by surgery, but it was associated with very high mortality. Now, best medical therapy with aggressive antihypertensive treatment is the standard of care in most uncomplicated type B dissection. Should repair be required, TEVAR has become the first line treatment in the majority of cases due to the relative lower morbidity and mortality seen with open aortic repair and cross clamping.

Two recent randomised trials, the INSTEAD (INvestigation of STEnt Grafts in Aortic Dissection) and the ADSORB (Acute Dissection: Stent graft OR Best medical therapy) trials, compared endovascular treatment with medical therapy did not show any survival difference. In the long-term (3–5 years) aortic remodeling is improved in the TEVAR group. Type B dissection requires life-long surveillance targeted to detect the progression of aortic dilatation and aneurysm formation.

Due to the complexity of this condition, aortic dissection has attracted the attention
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of several disciplines to improve patient care and advance our understanding. Increased awareness of the disease and better imaging techniques has helped speed diagnosis and treatment. Despite that, aortic dissection still carries a significant morbidity and mortality.

We have improved our surgical and endovascular skills, but have not made strides in prevention. The two main causes of dissection are hypertension and connective tissue diseases, such as Marfan’s disease and Ehlers Danlos syndrome. A significant number of patients with acute dissection presentations were found to be hypertensive. Moreover, we have failed to identify a genetic mutation for this disease, even in families who were referred to dissection clinic with a strong family history of aortic dissection. Therefore identifying high-risk groups and prophylactic management of risk factors may prevent dissection, but we are still in preliminary stages of understanding and management, and it may take a monumental effort in convincing government agencies to invest in a disease affecting 1 in 100,000 of its population.

**REFERENCES:**


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