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Biomechanical studies on type B aortic dissection

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Introduction to Acute Aortic Type B Dissection

Adapted from; Understanding Acute Aortic Type B Dissection:
Are There New Horizons In Patient Selection?

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HISTORY

The dissection process of blood vessels was first described more than three hundred years ago by Sennertus. However the first detailed descriptions of the clinical entity was published by the British physician named Nicholls in 1760. [1] In 1802 Maunoir described the penetration of blood through the media of a diseased aorta. At the end of the 19th century the first correct ante-mortem diagnosis of aortic dissection was made by Swaine. It took almost half a century before the first surgical intervention with an aortic fenestration procedure was attempted to treat malperfusion syndrome. The first primary open surgical repair of an acute aortic dissection was performed by De Bakey and Cooley in 1954. [2] The first reports of endovascular repair of acute aortic dissection with stent graft technology were at the end of the 20th century. [3]

PATHOFYSIOLOGY

Aortic dissection (AD) begins with an intimal tear which allows blood to enter and split the medial layers. Intimal disease, such as that associated with atherosclerosis, is not a prerequisite, although underlying medial disease due to both elastic fiber and smooth muscle cell degeneration is the rule. [4]

The most frequent site of entry in the descending thoracic aorta is just beyond the insertion of the ligamentum arteriosum, where the relatively mobile arch becomes anchored to the thoracic cage. Aortic dissection results in a false and true lumen. The false lumen having pressures greater than or equal to those in the true lumen. Expansion of the false lumen occurs due to its thin outer wall, which contains only about one-third of the elastin of the total vascular wall. The elastin-poor outer wall of the false lumen then dilates more than the elastin-rich nondissected inner wall to generate the wall tension required to balance a given blood pressure. [5] Dilatation of the false lumen is multi-factorial and depends on the (intra-luminal) blood pressure, residual wall thickness (depth of dissection plane in the media; wall shear stress), percentage of the wall circumference involved in the dissection. The dissection can evolve in either an antegrade or retrograde direction. Because of pressure differences, the false lumen may compress or obstruct the true lumen. [6] In general, after dissection several sequelae can occur. Acute dissection may be complicated by loss of blood supply to a vital organ because of branch arterial obstruction with either dynamic or static mechanisms. [6] In static obstruction, the dissection flap enters the branch vessel with absent or inadequate distal re-entry and causes ischemia by reducing the true lumen diameter. In contrast, the dissection flap in dynamic obstruction intermittently prolapses across the orifice of the branch vessel during the cardiac cycle, and this subsequently results in end-organ ischemia. [6] The false lumen may remain patent, thrombose, recommunicate with the true lumen through fenestrations, or rupture.

AETIOLOGY

Aortic dissection results from an interaction between abnormal hemodynamic circumstances and / or abnormal morphological characteristics of the aortic wall.

The two causes of abnormal hemodynamic factors are hypertension - found in at least two-thirds of all cases - and aortic coarctation. Causes of abnormal morphological characteristics of the aortic wall can be due to atherosclerosis, connective tissue disorders, iatrogenic factors or trauma (Tabel I). [6]

Tabel I. Conditions contributing to abnormal morphological characteristics of the aortic wall

Atherosclerotic Risk factors		Hypertension
		Dyslipidemia
		Smoking
		Cocaine
Connective tissue disorders	Congenital	Bicuspid aortic valve
		Cystic medial necrosis
		Turner's syndrome
		Marfan syndrome
		Ehlers-Danlos syndrome
		Loeys-Dietz syndrome
	Hereditary	Familial thoracic aortic aneurysm
	Acquired	Giant cell arteritis
		Takayasu's arteritis
		Syphilitic aortitis
		Behcet disease
Iatrogenic		Aortic catheterization
		Aortic (valve) surgery
		TEVAR
Trauma		Deceleration injuries

Cocaine serves as both a predisposing factor to aortic dissection due to its effect on aortic connective tissue and as a precipitating factor due to its propensity to produce abrupt and severe hypertension. [7]

Connective tissue disorders can be congenital, hereditary or acquired. Congenital causes includes bicuspid aortic valve and is associated with aortic aneurysm and dissection. This suggests the possibility that a bicuspid valve is an identifiable manifestation of a systemic connective tissue disorder. [8] In cystic medial necrosis there is a degenerative breakdown of collagen, elastin and smooth muscle caused by aging contributing to weakening of the wall of the artery. Turner's syndrome is associated with high blood pressure and aortic dilatation. [9] Structural weakness of the aortic wall is associated with multiple connective tissue disorders such as Marfan syndrome, Ehlers-Danlos and Loeys-Dietz syndrome. [6]

Acquired disorders include inflammatory diseases, which can destroy the medial layers of the aortic wall and lead to weakening, expansion, and dissection of the aortic wall. [9] Autoimmune processes may affect vasa vasorum and promote nutrient deficiency of aortic wall layers. [9] Iatrogenic aorta dissections can result from aortic (valve) surgery, aortic catheterization or after Thoracic EndoVascular Aortic Repair (TEVAR). Rare but not uncommon is aorta dissection after high deceleration trauma.

INCIDENCE OF AORTA DISSECTION

Aortic dissection is the most frequently diagnosed lethal condition of the aorta and occurs nearly three times as frequent as rupture of abdominal aortic aneurysm. [10] The overall incidence of aortic dissection has been estimated to 2.9 to 3.5 cases per 100 000 person-years. [6] The mean age at presentation is reported at around 60 years. [11] Males are more frequently affected than females, the rate is considered between 2:1 and 5:1. [9] Women may be affected less frequently, but have worse outcome as a result of atypical symptoms and delayed diagnosis. [9]

An important risk factor is a positive family history of thoracic aortic diseases. Acute TBAD has a prevalence of 13- 22% in patients who have a first degree relative with a history of descending thoracic aneurysm or AD. 6

CLASSIFICATION

Anatomical

The Stanford classification system of AD, described in 1970, has come to be the most widely used in the literature. Type A affects the ascending aorta proximal of the brachiocephalic artery, type B distally (Figure 1.). The most frequent site of entry in the descending thoracic aorta is just beyond the insertion of the ligamentum arteriosum, where the relatively mobile arch becomes anchored to the thoracic cage.

Recently a new anatomical classification system was introduced by the Society for Vascular Surgery/Society of Thoracic Surgeons (SVS/STS). This is based on the location of the entry tear and the proximal and distal extent and redefines nomenclature associated with Type B Aortic Dissection (TBAD) (Figure 2.). [12]

This new classification provides a clear framework of language that will allow more granular discussions and reporting of aortic dissection in the future. [12]

Complications

Complicated TBAD (cTBAD) is associated with rupture, mal-perfusion syndrome, refractory pain, rapid aortic expansion at onset or during hospital stay and failure of medical management

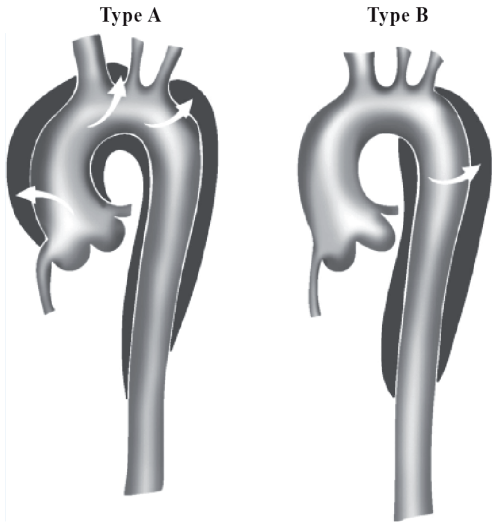
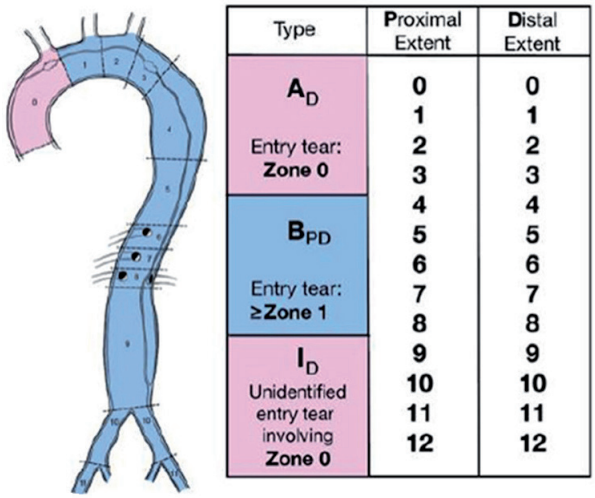


Figure 1. Stanford classification system of aortic dissection



Type	Proximal Extent	Distal Extent
A_D Entry tear: Zone 0	0	0
	1	1
	2	2
	3	3
B_{PD} Entry tear: ≥Zone 1	4	4
	5	5
	6	6
	7	7
	8	8
I_D Unidentified entry tear involving Zone 0	9	9
	10	10
	11	11
	12	12

Figure 2. Society for Vascular Surgery (SVS) and Society of Thoracic Surgeons (STS) reporting standards for type B aortic dissections. [12]

(see paragraph Treatment). Uncomplicated TBAD (uTBAD) occurs without the aforementioned complications.

Time of onset

A subdivision in onset of uTBAD is "acute" and should refer to <2 weeks, "subacute" from 2 to 6 weeks, and "chronic">6 weeks from symptom onset.

CLINICAL PRESENTATION

Acute AD is clinically suspected at initial evaluation in fewer than half of patients ultimately diagnosed with the disease. [9] Acute aortic dissection is frequently confused with acute coronary syndrome, resulting in delayed diagnosis and inappropriate treatment with antiplatelet, antithrombin, and fibrinolytic therapies. On the other hand a variety of symptoms can initially suggest acute aortic dissection but ultimately prove to represent other conditions. The stimulated sensory fibers in acute aortic dissection may share the common spinal segments with those arising from the heart, pericardium, pleura, and esophagus. [13]

The majority of patients with dissections have a previously documented history of hypertension (sensitivity 64%). [13] Most patients with acute aortic dissection present with pain (sensitivity 90%) of severe intensity (sensitivity 90%) with sudden onset (sensitivity 84%). [13.] The presence of a tearing and ripping pain sensation has a specificity around 95%. [14] Clinical findings for thoracic AD during physical examination are present in less than half of all cases. [14] The sensitivity of a clinical finding suggesting a dissection is disappointing with a reported sensitivity of only 31% in case of pulse pressure differential between carotid, radial and femoral arteries. [14]

IMAGING

In the 20th century aortography was used as the best imaging technique for assessing patients with clinically suspected thoracic aortic dissection. Currently, the noninvasive modalities most frequently used to identify aortic dissections are ultrasonography (US), helical computed tomography angiography (CTA) and magnetic resonance angiography (MRA).

US include transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE). The image quality of TTE is adversely affected by obesity, emphysema, mechanical ventilation, chest-wall deformities, or small intercostal spaces resulting in sensitivity between 31%–55% for dissections involving the descending aorta. [5] TEE may be used in the primary care setting to visualize the descending thoracic aorta from the Left Subclavian Artery (LSA) to coeliac artery with sensitivity up to 100%. However TEE in the awake patient can lead to more hypertension introducing more risks. If a patient is intubated for TEE further clinical symptoms are masked by sedation. This has to be balanced in the acute situation. US also have major general shortcomings, the diagnostic accuracy depends largely on the investigator's experience and the images can't be used to plan therapy. [15] This makes US as diagnostic imaging modality for AD not very helpful.

The standard technique for diagnosing and classifying thoracic aortic dissections are contrast-enhanced CTA, 3-dimensional (3D) MRA and 4-dimensional (4D) MRI.

The gold standard for imaging TBAD is Computed Tomography Angiography (CTA). [16] Imaging should include a noncontrast study, followed by an early and late phase contrast study and should examine the part of the body between the thoracic inlet and the common femoral arteries. CTA

has the advantages of shorter acquisition time, wide availability, and high diagnostic accuracy with sensitivity of 100% (CI 95% 96-100) and specificity of 98% (CI 95% 87-99). [17] False-negative CTA results can occur due to inadequate contrast opacification caused by cardiac failure or to the thrombosed lumen being mistaken for an aortic aneurysm with mural thrombus. [5]

Over the last two decades, CTA has become more sophisticated and is more readily available, with an increase in the number of scanners, the use of ECG-gated techniques, and through advances in post-processing software. These advances have resulted in motion free images with higher resolution, reduced scanning times, and better visualisation including three dimensional reconstruction. [18]

A major disadvantage of CTA is the static aspect of images, interpretation of the volume and flow changes in the true and false lumen during cardiac cycle is not possible. Contrast-enhanced 3D MRA has several advantages over CTA, including lack of nonionizing radiation, multiplanar evaluation, "safer" (ie, nonnephrotoxic) contrast material and greater vessel coverage at high resolution with fewer sections. [5] 4D flow MRI can accurately visualize and quantify the functional flow and access hemodynamic information such as as entry tear flow, blood flow patterns in the false and true lumen and Wall Shear Stress (WSS). [19] The WSS expresses the viscous force per unit area applied by the fluid on the wall in a direction at the local interface. [20]

Nevertheless, 3D and 4D MRA has its clinical limitations. It cannot be performed in unstable patients due to longer acquisition time and difficulty in monitoring, and it is also not appropriate for patients with implanted electronic devices or metal implants.

Performing CTA or MRA first for confirming or ruling out thoracic aortic dissection should depend on the availability of each imaging test because time delay increases the mortality rate in untreated patients. [9] However, 3D MRA may prove to be the optimal imaging modality in medically stable patients with aortic dissection. [21]

TREATMENT

Current treatment modality depends on whether the TBAD is categorized as either uncomplicated or complicated. About one third of the TBADs are complicated. [22] Intractable pain, rapid expansion and/or rupture, and organ and/or lower limb ischemia (malperfusion) are signs of cTBAD. The therapeutic options for cTBAD are endovascular therapy (fenestration and / or TEVAR) and open surgery. Endovascular fenestration can be performed in case of cTBAD with malperfusion syndrome due to dynamic compression. [23] Fenestration of the dissection flap decreases the load and pressure inside the false lumen by increasing communication between the true and false lumen. The concept of TEVAR was propelled by the desire to induce aortic remodeling by means of exclusion of the false lumen and thrombosis of the false lumen and, at the same time, avoiding the risks associated with open surgical intervention. Entry tear coverage with endovascular stent grafting and redirection of thoracic aortic flow entirely through the true lumen have been the hall-

marks of endovascular repair. [24] Creation of a sufficient proximal landing zone by over stenting the left subclavian artery during endovascular stent grafting results in increased risk of perioperative stroke, paraplegia and death. [28] Revascularisation of the LSA offers protection against a composite endpoint of stroke, paraplegia and death. [6 25]

A feared complication after TEVAR is the occurrence of a retrograde dissection. The stress yielded by the endograft seems to play a predominant role in its occurrence. It is important to take this stress-induced injury into account during both design and placement of the endograft. [26] Despite these disadvantages TEVAR provides improved survival in cTBAD with mortality rates of 10% to 20% versus 20% to 30% in open surgery. [6 9] TEVAR is now the treatment of choice for these extremely high-risk and complex patients. [6 9]

uTBAD is diagnosed in the absence of complications and has traditionally been managed medically. Optimal Medical Treatment (OMT) includes observation in an intensive care setting with aggressive blood pressure and heart rate control and close surveillance. [6 9 27] Also sedation and pain reducing management should be included. OMT for all acute uTBAD was derived from early studies conducted several decades ago that demonstrated no survival advantage of surgery over OMT. The goal of OMT is to reduce aortic wall stress and False Lumen (FL) pressurization. In the acute phase, intravenous medications are aimed to control heart rate, blood pressure and reduce the maximum change in left ventricular pressure in early systole (maximum dP/dt). [6 9 11] This result in a decrease aortic wall shear stress. [6 27] Alpha- and betablockers such as labetalol are useful first-line agents. If a potent vasodilator such as sodium nitroprusside is to be used, it is imperative to be certain that the patient is on a beta-blocker with good heart rate control to avoid reflex tachycardia. [3] A significantly decrease is observed of secondary adverse events (aortic expansion, recurrent aortic dissection, aortic rupture and/or need for aortic surgery) by reducing the heart rate below 60 bpm. [6] To achieve a systolic blood pressure of 100 to 120 mm Hg calcium-channel-blockers, nitrates and angiotensin converting enzyme inhibitors can be administered. [27] Approximately 20% of acute uTBAD could progress to a complicated state, with malperfusion syndrome constituting a significant portion of delayed complications. [9] Despite medical management overall in-hospital mortality for uTBAD is around 12%. [11 28] Once complications occur the prognosis declines, with hospital mortality greater than 50%. [29] By evolving medicine in particular improved perioperative management and most important the increased experience of TEVAR the morbidity and mortality from intervention has decreased. This resulted in several studies comparing OMT to OMT+TEVAR in subacute and acute uTBAD.

Subacute uTBAD

The INSTEAD (Investigation of Stent Grafts in Aortic Dissection) trial randomized 140 patients with subacute uTBAD into cohorts of elective TEVAR with OMT (n = 72) to OMT alone (n = 68). [28] At 2 years, no mortality difference was found for patients that had OMT+TEVAR compared with OMT alone. OMT+TEVAR was associated with improved favorable remodeling, with a FL thrombosis rate of 91.4%, compared with 19.4% in the OMT cohort ($p \leq 0.001$). [28] Extended

follow-up to 5 years in the INSTEAD-XL trial, the OMT+TEVAR patients had improved 5-year aorta-specific mortality (6.9% vs. 19.3%; p = 0.045), as well as greater rates of favorable remodeling (79.2% vs. 10%; p < 0.001). [30] This survival benefit is attributed to the ability of TEVAR to prevent late complications. In the Clinical Practice Guidelines of the European Society for Vascular Surgery (ESVS) patients at risk of further aortic complictions with suitable anatomy for endografting, endovascular repair of uTBADs in the sub-acute phase should be considered in dedicated centres. [6]

Acute uTBAD

ADSORB is the first and only prospective randomised clinical trial of acute uTBAD. [29] The primary endpoints were false lumen thrombosis, aortic dilatation, and aortic rupture. The original sample size calculation at trial inception in 2002 called for 250 patients to be randomized, but this was subsequently revised because of slow recruitment as well as newer data from the INSTEAD trial. Finally 61 patients with acute uTBAD were randomized into cohorts of elective OMT+TEVAR to OMT alone. OMT was given to both arms of this study and the mortality at 30 days was zero. Given the small sample size and short duration of follow-up, the trial is not powered to detect differences in aortic-related and all-cause mortality. A recent meta-analysis demonstrates that there remains uncertainty whether TEVAR, in addition to OMT, is beneficial in acute and subacute uncomplicated type B aortic dissection. [31] The capability to accurately predict which patients with acute uTBAD will develop cTBAD could transform clinical management by allowing earlier intervention before complications occur.

PREDICTORS OF DISSECTION RELATED EVENTS AFTER INITIAL CONSERVATIVE TREATMENT

Several prognostic predictors of dissection related events (dissection related death or need for intervention) after initial conservative treatment in acute uTBAD have previously been identified.

Predictors of complications in acute uTBAD during admission are aortic diameter ≥40mm, a primary entry tear >10mm, primary entry tear located on the concavity (undersurface) of the distal aortic arch and a FL diameter > 22mm. [31-34]

Prognostic predictors for dissection related events in acute TBAD during admission are a peak CRP level >96.1 mg/L and patency of the false lumen (defined as the concurrent presence of both flow and thrombus).

The peak CRP level is a strong predictor of long-term outcomes in acute uTBAD (Table II). [36] The peak CRP may represent the extent of the inflammatory reaction in the dissected aortic wall and may also reflect the damage to the lesion.

Patent false lumen and in particular partial thrombosis of the false lumen portends a poor outcome. [37] One potential explanation for a poor outcome relates the pressure within the false lumen to the presence of partial thrombosis. A patent false lumen may be perfused by a proximal

entry tear and decompressed by distal reentry tear(s), formation of a partial thrombus may occlude these distal tear(s), obstructing the outflow. An increase in pressure within the false lumen will increase wall tension, which may elevate the risk of aneurysm expansion, redissection and rupture.

Patients with complete thrombosis of the false lumen have improved outcomes, whereas those with a patent false lumen have an increased risk of aortic expansion and death. [6 33]

Antegrade flow through the false lumen is more likely to be associated with chronic false lumen patency than distal tears with retrograde flow. [38] Complete thrombosis of the false lumen excludes the false lumen from the circulation and is a predictor of less aortic enlargement and is related to lower mortality. Complete thrombosis of the false lumen is thought to be a prerequisite for complete healing.

Tabel II. Predictors of adverse dissections related events in ABAD

			HR	*95% Confidence intervals*
At admission	Aortic diameter ≥40mm [6 34]		3.13	1.10 to 8.88
	Primary entry tear >10mm [6 34]			
	Primary entry tear located on the concavity (undersurface) of the distal aortic arch [34 35]			
During admission	Peak CRP level [36]	96.1 to 148.7 mg/L	2.42	1.04 to 5.61
		149.0 to 326.0 mg/L	3.99	1.78 to 8.99
		FL diameter > 22mm [31 33]		
	Patency of the false lumen [6 38]		7.63	2.68 to 21.69

GENERAL OUTLINE AND AIM OF THE THESIS

Although some clinical and image based predictors of adverse outcomes have been identified there is still little basic mechanistic insight in the process of dissection and the behavior of the false lumen. Better understanding of important pathophysiologic elements of the dissection process can help to guide clinical management. This resulted in our interest to study the false lumen in TBAD. Improved understanding of the false lumen might result in new insights to predict and understand the development of dissection related adverse events in TBAD. The false lumen is influenced by dissection morphology, heamodynamics and aortic wall elasticity. An in-vitro or ex-vivo study has the potential to isolate and study one specific parameter in a controlled setting. Previous ex-vivo studies to examine dissection pathofysiology have used a non biological silicon tubing to simulate TBAD. For this thesis we created a porcine TBAD model and implemented it in a previously validated circulation system to have the ability to study several individual factors that influence the false lumen in TBAD.

In TBAD patent false lumen portends a poor outcome. Patent branch vessels originating from the false lumen in an aortic dissection type B are assumed to contribute to persistent blood flow and patent false lumen. Chapter 2 aims to assess the morphologic changes of the false lumen generated by different outflow rates in the created porcine TBAD model.

In contrast to aortic wall elasticity, the influence of haemodynamics and dissection morphology have been investigated often in multiple in-vitro and ex-vivo studies. Chapter 3 focuses on the influence of aortic wall elasticity on the diameter and pressure of the false lumen in aortic dissection by using the porcine TBAD model.

4D flow MRI has in contrast to the gold standard for imaging TBAD the ability to visualize and quantify flow and provide hemodynamic information such as wall shear stress. Chapter 4 examine the influence of heart rate on the volume, mean and peak wall shear stress by 4D flow MRI in the false lumen in the porcine aorta dissection model.

Fenestration is a minimally invasive alternative for the treatment of acute symptomatic TBAD because it may quickly decrease the pressure gradient of the false lumen. Chapter 5 evaluates were the optimal location of these fenestrations should be made.

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