

We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,900

Open access books available

186,000

International authors and editors

200M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com



Role of Interventional Treatment of Thoracic Aorta

Ibrahim Akin, Uzair Ansari and
Christoph A. Nienaber

Additional information is available at the end of the chapter

<http://dx.doi.org/10.5772/66520>

Abstract

An aging western and oriental population coupled with breakthrough advances in modern diagnostic imaging modalities has evoked renewed interest in the hitherto underdiagnosed acute and chronic diseases of the aorta, which also include aortic aneurysm and aortic dissection. Although classical surgical strategies still dominate the clinical management of acute and chronic pathologies of the ascending aorta and the proximal arch region, the emergence of novel endovascular concepts has offered an interesting therapy alternative for the treatment of descending aortic pathology in suitable patients and is highly likely to evolve as the primary treatment strategy in majority of the cases. Moreover, the use of hybrid approaches combining surgical head-vessel debranching and interventional stent-graft implantation in an attempt to improve clinical outcome in aortic arch pathologies has helped avoid the high risk of open arch repair or complete replacement. Notwithstanding these recent advancements, the complex nature of the underlying vascular disease still dictates that the proposed management of every diagnosed patient is discussed in a team constituting cardiologists, cardiac surgeons, anesthesiologists, and radiologists, with the conceptualization of individualized therapeutic strategies and conducted in a center with significant surgical and endovascular experience.

Keywords: aortic dissection, aortic aneurysm, stent graft, endoleak, malperfusion

1. Introduction

The pathophysiological underpinnings initiating the development of thoracic aortic disease are complex and not fully understood. Standard treatment options in most instances have included surgical resection and interposition of vascular prostheses despite the risk of severe complications arising from surgical trauma [1–3]. Although significant advancements in surgical procedures and intraoperative management have mitigated the risk of some of these adverse events, the perioperative mortality and morbidity remains high. Additionally, the

changing dynamics of demographic distribution in the western and oriental world have skewed the incidence of disease to an increasingly aging population, a patient group inherently afflicted with a variety of comorbidities, thus foretelling the compounded risk of a sobering surgical outcome.

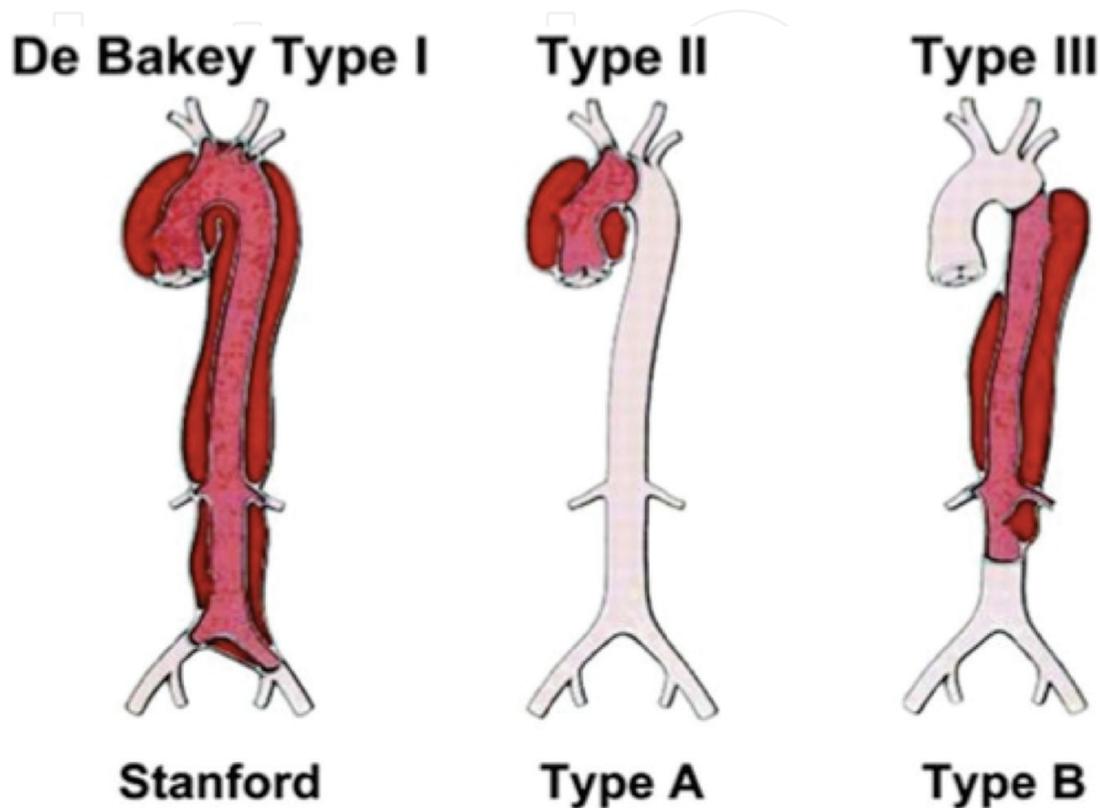
The use of an endoluminal stent-graft prosthesis initiated as a revolutionary treatment concept more than two decades ago for patients with thoracic aortic disease offered an interesting alternative to open surgery by circumventing certain obvious risks. The induction of reconstructive remodeling of the diseased aorta by triggering a natural healing process through exclusion and depressurization of the aneurysmal process was an innovative interventional approach [4–6], with initial reports suggesting encouraging results for the treatment of various aortic pathologies (e.g., degenerative aneurysm, mycotoxins aneurysm, traumatic injuries, aortic dissections, and penetrating aortic ulcers (PAU)). Scarce randomized data as well as the absence of long-term surveillance of treated patients has strengthened critique against this approach, and with the lack of a satisfying rebuttal, its universal adoption has consequently been hindered [7–10]. This comprehensive chapter describes current indications, techniques, and advancements in endovascular strategies in the treatment of thoracic aortic disease.

2. Classification

Aortic dissection and aortic aneurysm have been listed as the most commonly encountered pathologies of the thoracic aorta. The incidence of aortic dissection in the clinical arena is still relatively rare, with an estimated 2.6–4 cases per 100,000 in the general population reported in a year [11–13]. Statistics reveal that only around 0.5% of the patients presenting with chest or back pain in the emergency room (ER) suffer from aortic dissection [14]. The early phase of this disease, associated with significantly high mortality, is labeled an acute aortic dissection as documented symptom-onset is less than 2 weeks on presentation. Patients surviving the initial 2 weeks without any intervention are classified as suffering from a subacute form, while the chronic patients, who constitute about one-third of all patients with aortic dissection, survive longer than 90 days. The Stanford and DeBakey classifications of aortic dissection are further attempts to group different presentations according to the anatomical location of disease (**Figure 1**). The fundamental distinction lies in the presentation of either a proximal (involving the aortic root or ascending aorta) or distal (beyond the subclavian artery) loci. An untreated proximal aortic dissection is characterized by an initial mortality rate of more than 1% per hour, which if left untreated results in death due to cardiac rupture, tamponade, heart failure from acute aortic regurgitation, or from major coronary closure [11–13]. The prognosis of a distal aortic dissection, popularly known as a Type B aortic dissection, is comparatively better clocking a 30-day mortality rate of about 10% [11–13].

In retrospect, little has been elucidated about the true prevalence and mortality rates of thoracic aortic aneurysms (TAA). An older population-based study reported an age- and gender-adjusted incidence of 5.9 new aneurysms per 100,000 people-year in a Midwestern community over a 30-year period. The median ages for men and women were 65 years and 77 years, respectively, with the primary distribution of cases reflecting an affliction of the ascending

aorta in 51% of the patients, of the arch in 11% and of the descending thoracic aorta in 38% [15]. The Crawford classification that attempted to classify these aneurysms based on the origin of distal to the subclavian artery (Types I–IV) has recently been adapted and tweaked by Safi (Type V) [16] (Figure 2).



Acuity:

- Acute: <2 weeks after onset
- Subacute: 2-8 weeks after onset
- Chronic: >8 weeks after onset

Anatomic location:

- Ascending aorta: Stanford Type A, DeBakey Type II
- Ascending and descending aorta: Stanford Type A, DeBakey Type I
- Descending aorta: Stanford Type B, DeBakey Type III

Pathophysiology:

- Class 1: Classical aortic dissection with intimal flap between true and false lumen
- Class 2: Aortic intramural hematoma without identifiable intimal flap
- Class 3: Intimal tear without hematoma (limited dissection)
- Class 4: Atherosclerotic plaque rupture with aortic penetrating ulcer
- Class 5: Iatrogenic or traumatic aortic dissection (intra-aortic catheterization, high-speed deceleration injury, blunt chest trauma)

Figure 1. Classification of thoracic aortic dissection.

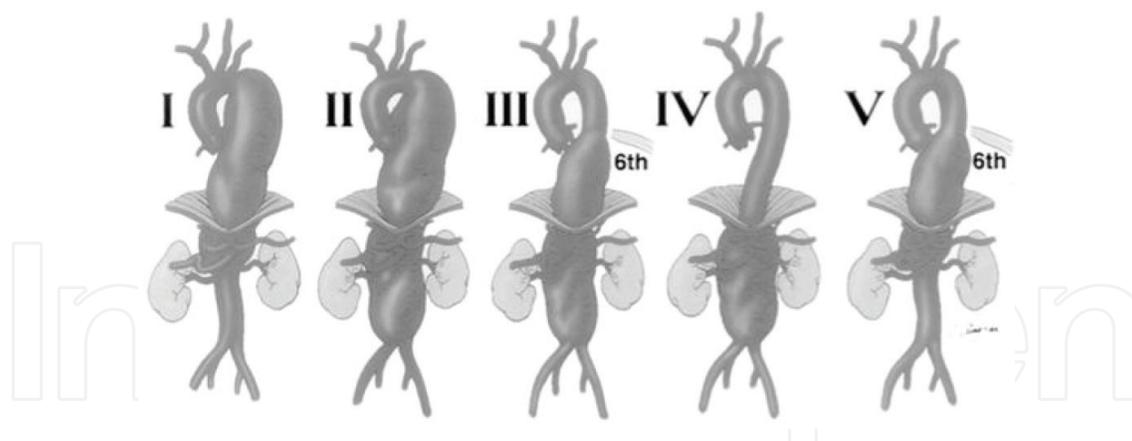


Figure 2. Classification of thoracic aortic aneurysms.

3. Indications for endovascular stent-graft therapy

In the event of an aortic dissection, the sealing of proximal entry tears with a customized stent graft has proven to be the most effective method for excluding a growing aneurysmal false lumen. Although the closure of a distal reentry tear is also desirable, this is not particularly necessary to achieve optimal results [12, 13, 17]. A favorable postinterventional outcome would constitute adequate depressurization and shrinkage of the false lumen, supplemented in an ideal scenario by a complete thrombosis of this lumen with consequent remodeling of the entire dissected aorta [18] (Figure 3).

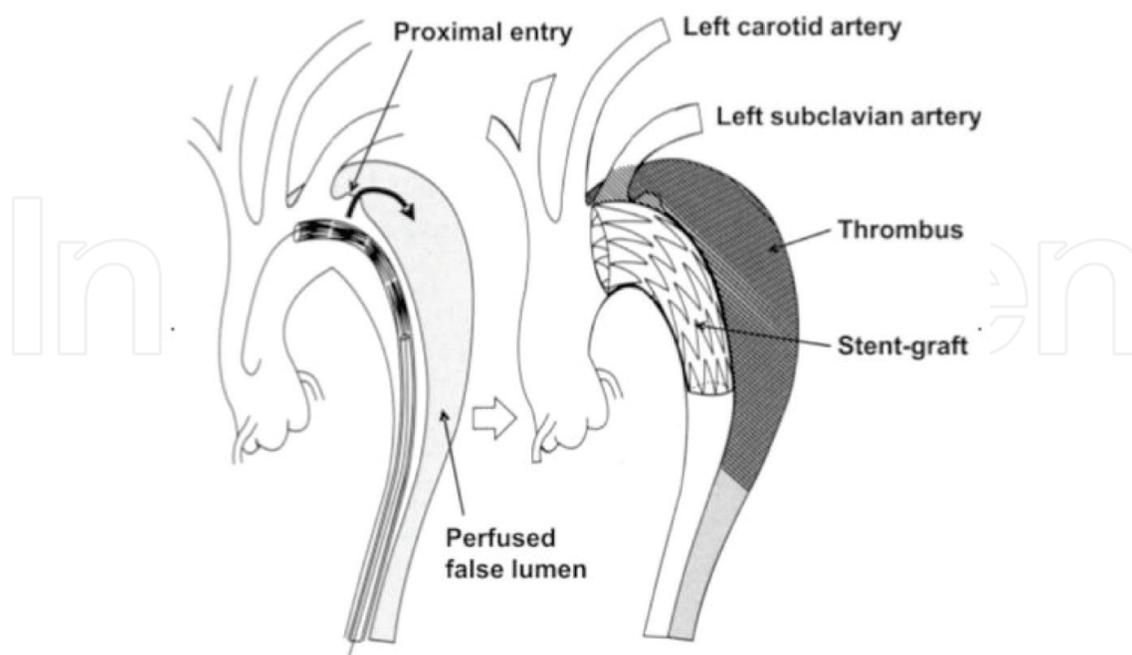


Figure 3. Concept of interventional reconstruction of the dissected aorta with sealing of the proximal entries, depressurization of the false lumen, and initiation of false lumen thrombosis.

Disease etiology

Aortic aneurysms

Atherosclerotic/degenerative

Posttraumatic

Mycotic

Anastomotic

Cystic medial necrosis

Aortitis

Stanford Type B aortic dissection

Acute

Chronic

Giant penetrating ulcer

Traumatic aortic tear

Aortopulmonary fistula

Marfan syndrome

Aneurysm morphology

Aneurysm of the descending aorta

Proximal neck length = 2 cm

<2 cm if supraaortic vessels have been transposed prior stent-graft placement

Distal neck length = 2 cm

Diameter = 6 cm

Patients condition

Preferentially older age

Unfit for open surgical repair or high-risk patients

Chronic obstructive pulmonary disease

Severe coronary heart disease

Severe carotid artery disease

Renal insufficiency

Suitable vascular access site

Life expectancy of more than 6 month

Table 1. Current indication for stent-graft implantation.

The role of percutaneous fenestration in the management of aortic dissection is debatable and should be in all likelihood avoided considering its profile of unproven benefits and interventional risk. The relevance of a team of professionals experienced in treating aortic disease is absolute considering the necessity for a thorough evaluation of the clinical, technical,

and anatomical criteria before a treatment strategy has been defined. The general indication for emergency placement of stent grafts has been extended to scenarios where patients have intractable pain associated with a descending dissection, develop a rapidly expanding false lumen diameter, percolate extra-aortic blood (as a sign of imminent rupture), or show signs of a distal malperfusion syndrome [11–13] (**Table 1**). The use of endovascular stent grafting as the primary strategy may be justified in the event of late-onset complications like malperfusion of vital peripheral aortic branches.

4. Anatomical measurements for thoracic aortic pathology

Multi-slice computed tomography (CT) has special relevance in the diagnosis of aortic disease. In addition to the reconstruction of a three-dimensional perspective, certain descriptive features of the thoracic aorta outlining the condition of the aortic wall (atheroma, calcification, and thrombus) as well as shape and size of the aortic pathology (diameter, length, and shape) need to be elucidated before the formulation of treatment strategies [19].

Although an initial CT angiography/magnetic resonance (MR) angiography is considered as the primary diagnostic tool for aortic pathologies, the use of transesophageal echocardiography and intravascular ultrasound to ascertain additional valuable information has also been advised. For instance, flow-sensitive MR sequences or contrast-enhanced TEE not only shows the communication sites between true and false lumen, but it also highlights the dynamic flow pattern in the false lumen prior to stent-graft placement. The use of contrast angiography has limited potential in this scenario as measured values are generally unreliable.

The measurement of vessel diameters is generally not defined by any set of conventional practice and operators usually refer to calculations derived from the inner vessel wall (endothelial trailing edge) hinging their assumption on the basis that this will guarantee some degree of oversizing, a potentially desirable outcome for endograft placement. Our in-house protocol delineates the diameter of the normal appearing proximal aorta (inner edge to edge) measured from a transverse plane perpendicular to the long axis of the aorta (preferentially from contrast-enhanced multi-slice CT images) (**Figure 4**).

The evaluation of access vessels for size and tortuosity is also pertinent because stent-graft delivery systems are quite large (up to 24F), and there is an associated risk of significant trauma to the femoral access site and iliac arteries. Severe aortic angulation or tortuosity, friable atheroma or thrombus lining the aortic wall, and aortic pathology involving the ascending aorta are some features that preclude the use of thoracic stent grafts. Additionally, the role of appropriate peri-interventional image reconstruction in aortic arch pathology is further cemented when considering the placement of a stent graft for a Type B dissection, where the demarcation of nearby aortic branches (including the left subclavian or left common carotid artery) is vital.

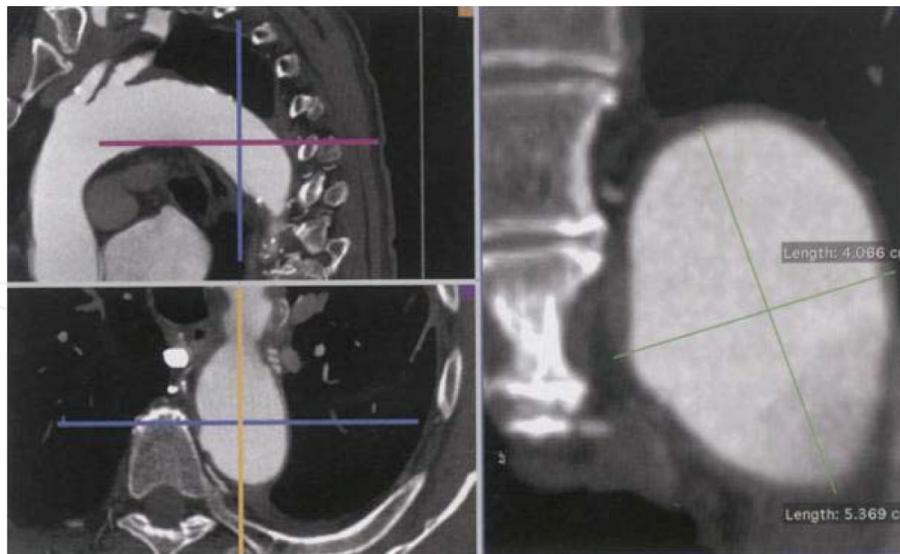


Figure 4. CT scan of a patient with dilated thoracic aorta. Due to the fact that the aorta follows a curved and three-dimensionally tortuous path, the axial scans are inadequate for measuring aortic diameters, as are sagittal and coronal planes for measuring aortic lengths.

5. Initial treatment

An acute aortic disease is a medical emergency and suspected patients require urgent admission to an intensive care or monitoring unit with an emphasis on immediate diagnostic evaluation [20]. Initial management strategies include the treatment of pain and maintenance of a systolic blood pressure of 110 mmHg with the use of morphine sulphate and intravenous beta-blockers (metoprolol, esmolol, or labetalol), respectively. The use of additional anti-hypertensives like angiotensin-converting enzyme inhibitors or vasodilators like sodium nitroprusside has been prescribed in refractory cases, while the intravenous use of verapamil or diltiazem is recommended for patients in whom beta-blockers are contraindicated. Although monotherapy with beta-blockers sufficiently controls cases of mild hypertension, the addition of sodium nitroprusside at an initial dose of 0.3 $\mu\text{g}/\text{kg}/\text{min}$ proves to be an effective combination in a severe hypertensive state. A careful evaluation for blood loss, pericardial effusion, or heart failure (by echocardiography) is a prerequisite before administering volume in normotensive or hypotensive patients. Additionally, the control of heart rate is of utmost importance in this scenario [21]. Hemodynamic instability compounds to the gravity of the clinical situation, with patients often requiring endotracheal intubation, mechanical ventilation, and urgent bedside transesophageal echocardiography or rapid CT for confirmation of the provisional diagnosis. In rare instances, the diagnosis of cardiac tamponade as quantified by transthoracic echocardiography may justify an immediate sternotomy in order to obtain surgical access to the ascending aorta, thus reducing the risk of shock, ischemic brain damage, and circulatory arrest.

6. Technique of endovascular stent-graft placement

The use of individually selected stent grafts to cover up to 20 cm (and sometimes even more) of the diseased aorta relies on measurements derived from different diagnostic modalities. These tests also have certain additional benefits. The transesophageal echocardiography is mandatory for detection of small entries, while contrast-enhanced CT (Computed tomography) angiograms have proven to be the preferred tool for diagnosis of unstable patients in an emergency situation. The use of intravascular ultrasound (IVUS), using a 10 MHz ultrasound catheter-mounted transducer with the potential of manual maneuverability through the diseased aorta, can be used to better identify communications, partial thrombosis, or other anomalies.

The operative placement of an endovascular stent graft is usually performed in a Cathlab or a hybrid operating room equipped with digital angiography and necessary support for general anesthesia (a compulsory prerequisite for all patients). The femoral artery generally serves as the access-site as it could typically accommodate a 20–24 stent-graft system. After the employment of the Seldinger technique to position the initial sheath, a 260 cm stiff wire (e.g., Amplatzer) is placed over a pigtail catheter and navigated along with a soft wire in the true lumen under both fluoroscopic and transesophageal ultrasound guidance. The “embracement technique” augmented by the use of two pigtail catheters could prove useful in maintaining the true lumen during complex cases when multiple reentries into the abdominal aorta are required. Essentially, a catheter inserted via the left brachial artery navigates through the true aortic lumen and meets the femoral pigtail catheter in the true lumen of the abdominal aorta, thence pulling it up into the aortic arch. This technique ensures the definitive positioning of the stiff guide wire within the true lumen necessary for the ensuing endovascular intervention. The subsequent launch of the stent graft is performed by carefully advancing the graft over the stiff wire in the true lumen while briefly maintaining a low blood pressure through rapid right ventricular pacing [22]. To improve apposition of the stent struts to the aortic wall in the event of incomplete sealing of the proximal thoracic communications, a latex balloon may be shortly inflated at the target site postdeployment of graft and sealing of entry flap. Doppler-ultrasound and contrast fluoroscopy are useful techniques instrumental in confirming the immediate result and play a vital role when initiating adjunctive maneuvers. The navigation of wires and instruments is less cumbersome in the setting of a thoracic aortic aneurysm (TAA) or aortic ulcer; however, meticulous intraprocedural ultrasound imaging and fluoroscopy are essential to monitor the interventional progress. The close vicinity of the left subclavian artery origin to the primary tear in Type B dissections presents a unique anatomical problem, often requiring a radical solution. A complete closure of the left subclavian artery (LSA) ostium may be necessary at times to ensure the proper placement and debranching of the stent graft, else use of extra-anatomical bypasses would have to be considered [23]. Observational evidence dictates that prophylactic surgical maneuvers are not always required but could be performed electively after the endovascular intervention in the event of developing ischemia. Additionally, the existence of potential supra-aortic variants (e.g., presence of a lusorian artery, an incomplete vertebra-basilar system, or vertebral arteries directly originating from the arch) must be considered before the intentional occlusion of LSA [24].

7. Device sizing and length

A key factor influencing strategies in aortic dissection and TAA interventions is the length of the true lumen that needs scaffolding with an endoprosthesis. The instinctual choice between covering primarily the proximal entry point and alternatively lining a longer length of the true lumen (where the descending thoracic aorta down to the level of the diaphragm is also treated) can significantly alter the course and prognosis of the intervention. Although a longer scaffold provides potentially greater stability and is associated with lower recurrence, the grave risk of potential paraplegia resulting from compromise in spinal arterial supply serves as the single biggest reason to avoid extensive coverage. Observational evidence indicates that stent-graft coverage exceeding 20 cm, previous abdominal aortic surgery, overstenting of the LSA, or use of the left mammary artery for coronary bypass is associated with an increased risk of spinal ischemia. Essentially, all scenarios leading to compromised collateral flow in the spinal cord arterial network are associated with an increased risk for neurological complications, thus underlining the need to avoid overstenting the LSA or use long stent grafts. The angiographic identification of the anterior spinal artery in this scenario has recently shown prognostic relevance.

The disadvantage of shorter coverage lengths is primarily associated with potential multiple reentries presenting themselves in the distal portions of the descending aorta and maintaining perfusion of the false lumen despite proximal entry-site closure. This deviant presentation can be managed by the use of uncovered bare metal stents ensuring distal reapposition of the dissecting lamella without any compromise to abdominal side branches or spinal arteries, in effect sustaining the distal extension of the scaffolding concept of endoluminal repair.

The proximal aspect of the device should be sized to the internal luminal diameter of the aorta, close to the left subclavian origin in order to achieve attachment and fixation at the proximal "neck." TAA interventions may require a degree of device oversizing by about 10–15%, while the endoprosthesis used in aortic dissection should never be oversized. Fatal complications are associated with excessive device oversizing, typically as a result of ensuing trauma to the aortic wall, thus resulting in either a retrograde dissection into the arch or conversion of a Type B dissection into a Type A dissection. Cases of aortic perforation and formation of pseudoaneurysms have also been reported in literature. The countereffects of oversizing further distally into the descending thoracic aorta include the risk of tears in the membrane between the two channels, thus forming a new reentry point, and consequently sustaining the ongoing pressurized perfusion of the false lumen. However, if absolute measures do dictate the extensive scaffolding of the aortic lumen, the operator could choose to use two different shorter grafts instead of a single long graft. The final decision represents a compromise between the reduced number of modular junctions and additional frictional forces associated with the deployment of a long stent graft. The concurrent employment of two stent grafts, when deemed necessary, requires them being fashioned in a "telescopic" manner, with the amount of overlap exceeding 30 mm in straight anatomic segments and measuring up to 50 mm or more in the angulated or curved segments of the aorta. The geometry of the

presumed junction between the grafts provides some assistance in estimating the length of overlap required at the connecting zones. A key deciding factor in this situation is the length of the radius of the curve, with shorter radii requiring a longer stent overlap on the lesser curve. Another prominent factor influencing the decision regarding overlap is the degree of support provided by the native aorta at the modular junction. If the junction occurs in the sac of a large fusiform aneurysm rather than in a segment of aorta with a normal diameter, the required overlap should be longer. The reason for this approach is a tendency for the graft to move out toward the greater curve of the aneurysmatic aortic segment due to mechanical forces associated with the pulsatile motion of the aorta, potentially causing a migration or a disconnection of the modular components.

8. Extended scaffolding with bare stents

The general consensus on management of entry tears dictates treatment with a covered stent graft, while the remaining distal thoracic or even abdominal aorta may be additionally supported by uncovered stents [25]. In selecting the suitable stent graft, the following principles should be followed:

- An appropriate size and diameter is crucial to avoid erosion through the aortic wall and to assure optimal conformability.
- The flexibility of the endoprosthesis and its release with a deployment mechanism should permit ease of use and provide accurate placement at the desired zone.
- The fragility of the dissected wall directs the use of a stent graft that is not dependent on balloon expansion for deployment or postdeployment modeling. Our experience suggests that the use of a self-expanding endoprosthesis with a nitinol-based architecture using limited radial force (in case of aortic dissection) is helpful in avoiding any untoward ballooning.

9. Landing zone

The three-dimensional mechanical forces associated with the pulsatile flow that “play” on an endoprosthesis in the thoracic aorta are far greater and more complex than those in the abdominal aorta. It is for this reason that an extremely stable graft-anchorage and optimal graft-apposition are prerequisites for a satisfying result. The proximal and distal landing zones should ideally be free of aortic wall atheroma or thrombi and circumscribe 15 mm in length. The contemporary hypothesis suggesting that the left subclavian artery is a natural barrier beyond which it is impossible to deploy stent grafts has been now rendered redundant.

The suitable presentation of the aortic arch between the left subclavian artery and the origin of the left common carotid artery, coupled with its relatively horizontal orientation in comparison to the aorta distal to the LSA, has encouraged interventionists to use this segment

of the aorta as a preferred anchor zone (with or without bypassing the LSA). To ascertain optimal cerebral circulation, it is considered prudent to perform a preinterventional digital subtraction angiography (DSA), CT, or magnetic resonance angiography. This could help identify patients with an incomplete circle of Willis or other limitations or abnormalities of cerebral vascular supply. In patients affected with a cerebrovascular pathology, prophylactic measures such as carotid-subclavian bypass might be considered prior to the stent-associated covering of the LSA. An obvious alternative is the use of bare stents proximally for better alignment while foregoing the risk of occluding a vital branch.

10. Hybrid arch procedures

The relative proximity of supra-aortic branches needing preservation poses a strict interventional challenge, considering that the aortic arch anatomy and pathology is complicated by varying degrees of length and angulation [26–28]. The traditional open arch surgical reconstruction techniques requiring perioperative hypothermic cardiac arrest, extracorporeal circulation, and selective cerebral perfusion have been shown to manage aortic arch pathologies effectively. However, these major surgical procedures carry the risk of significant mortality, paraplegia, and cerebral stroke. Younger patients with a smaller risk-profile qualify for such an open repair, while high-risk patients constitute the group of candidates ill-suited for this surgical approach. Hybrid arch procedures are a combination of methods preserving cerebral perfusion (like the debranching bypass with supra-aortic vessel transposition) on the one hand, while objectively providing patient-centric solutions for complex aortic arch lesions through thoracic endografting on the other (**Figure 5**). Hybrid arch procedures are performed without hypothermic circulatory arrest or extracorporeal circulation, thus expanding the treatable population demographic to older and high-risk patients with severe comorbidities currently not eligible for open surgical repair.

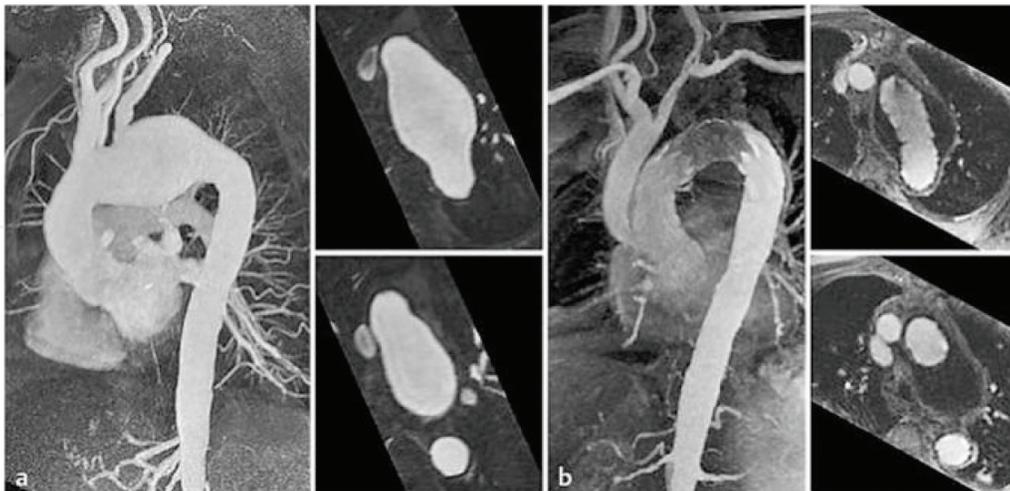


Figure 5. Contrast-medium enhanced MR-angiography of the aorta in a case of an arch aneurysm. (a) Aneurysm of the aortic arch involving the supra-aortic branches. (b) Result after hybrid procedure with debranching of the supraaortic vessels and stent-graft placement in the aortic arch.

11. Aftercare and long-term follow-up

The long-term care of patients successfully treated for an acute aortic dissection is pivoted on the appreciation of this disease as a systemic illness. Estimates suggest that nearly one-third of patients surviving an acute dissection of the thoracic aorta will either experience complications like extension of the dissection and late aortic rupture or require surgical correction of a newly formed aortic aneurysm, within 5 years of initial presentation [29]. All patients merit aggressive medical therapy, follow-up visits, and serial CT-imaging. The medical management of these patients is primarily centered on therapy with beta-blockers, in essence serving as the cornerstone of successful aftercare. High blood pressure values and steeper curves representing changes in the blood volume and pressure (dp/dt) have been shown to accelerate aortic expansion in Marfan's syndrome and also in patients with chronic abdominal aortic aneurysms (AAA). The aim in patients with thoracic aortic disease is to balance blood pressure values to less than 135/80 mmHg, while patients diagnosed with Marfan's syndrome are advised to maintain blood pressure levels below 130/80 mmHg [11–13]. Additionally, an adequate control of heart rate under 60 beats per minute has been associated with significantly fewer secondary adverse events (aortic expansion, recurrent aortic dissection, aortic rupture, and/or need for aortic surgery) in Type B aortic dissection patients [21].

Serial CT imaging of the aorta is an essential component of long-term management (before and after surgery or stent-graft placement). The choice of imaging modality, CT or magnetic resonance, is dependent on institutional availability and expertise. Past recommendations have suggested follow-up imaging at 1-, 3-, 6-, 9-, and 12-month intervals following discharge, and annually thereafter [11–13]. This aggressive strategy, in effect, underlines the observation that both hypertension and aortic expansion/dissection are common and not easily predicted in the first months following hospital discharge. The risk of dissection progression and aneurysm formation anywhere along the entire length of the aorta cements the need for consistent follow-up imaging of the complete thoracic aorta and in exceptional cases, imaging of the thoracoabdominal as well as abdominal aorta.

12. Management of complications

12.1. Endoleaks

An endoleak is a condition defined by the persistence of blood flow outside the endovascular stent-graft lumen, contained within the aneurysm sac, or in certain cases adjacent to the vascular segment treated by the stent graft. Endoleaks generally persist for a long duration, with a few eventually developing a late aneurysmal rupture, while some may also resolve and close spontaneously.

The classification of endoleaks according to time of onset permits their grouping into "primary endoleaks," when it occurs during the perioperative period (lesser than 30 days) or secondary leak when it is detected later. Further subcategorization requires precise information on periprosthetic blood flow. A Type I endoleak is an indicator of a persistent perigraft

channel of blood flow caused by an inadequate seal at either the proximal (I-a) or distal (I-b) stent-graft end or attachment zones. A Type II endoleak is attributed to retrograde flow into the aneurysmal sac via aortic side branches, while a Type III endoleak is caused by component disconnection (III-a) or fabric tear, fabric disruption, or graft disintegration (III-b). Type III-b can be further stratified as minor (<2 mm), or major (>2 mm). The Type IV endoleak is caused by blood flow through an intact but otherwise porous fabric, observed during the first 30 days after stent-graft implantation. If an endoleak is detected on imaging studies but the precise source cannot be determined, the endoleak is categorized as an endoleak of undefined origin.

Various strategies have been outlined for the treatment of endoleaks. The conservative line of management, essentially constituting strict observation and follow-up is especially suggested for Type II and Type IV endoleaks. An endovascular reintervention (e.g., balloon-inflation and/or implantation of an additional stent graft) is suggested for the management of Type I and Type III endoleaks. If these endovascular maneuvers fail in their attempt to exclude the aneurysm from circulation, the resulting increase in TAA-diameters could be corrected by open surgery.

13. Aneurysm evolution after stent-graft treatment

The thoracic aorta is defined by a set of unique anatomical features and habits a distinctive biomechanical and hemodynamic environment, which explains some of the rare late complications specific to device use seen in this region. The descending thoracic aorta, unlike the abdominal aorta, is relatively mobile in the thorax and is subject to complex and vigorous three-dimensional motion. The only points of fixation are the aortic root and sites of origin of major branches, thereby permitting the mobility of a long aortic segment extending from the LSA to the celiac artery. This contributes to the elongation, angulation, and eventual enlargement of the thoracic aorta between these points, thus promoting the development of an aneurysm. The mechanical forces exert a complex pattern of dynamic circumferential, radial, and axial forces on thoracic stent grafts, resulting in a stress field significantly different than that exerted on abdominal stent grafts. It is, however, still relatively rare to see aneurysms develop in the dissected aorta post stent-graft treatment. The relevance of strict clinical and imaging follow-up to monitor anatomical changes in the thoracic aorta is highlighted by the fact that the development of a false lumen thrombosis secondary to thoracic endografting is essential to the prevention of late aortic expansion.

A pertinent issue of concern is the fate of the distal aortic segment despite successful thoracic stent-graft placement. In the presence of large reentry points, the thoracoabdominal segment of the false lumen has a tendency to remain patent and remodel completely, setting the stage for late complications such as aneurysmal enlargement at the proximal or distal end of the stent graft. Some other complications include perforations of the fragile aortic intima by the ends of the metallic stent (especially in the early phase of acute aortic dissection), and injuries caused by stiff guide-wires and device manipulation potentially setting the stage for an

evolving aneurysm. The risk of these complications is noticeably diminished by the introduction of a more flexible and soft tip delivery system aided by minimally traumatic thoracic guide-wires specifically designed for these interventions. Additionally, the time required to perform an endovascular intervention could provide insight to complications such as stroke or bleeding; both of which could be significantly reduced by experienced interventionists requiring less than 30 min to complete a case. It has been also suggested that the patency of the abdominal aortic false lumen may be related to persisting communications between the true and false lumen. Treatment of these communications at the level of distal thoracic and abdominal aorta could potentially obliterate the false lumen and reduce the aortic diameter. In clinical practice, however, the closure is difficult to achieve because of the proximity or involvement of the visceral branches. Another late complication of graft treatment is related to the mechanical weakness of dissected aortic walls causing distention of the aorta beyond the portion covered by the stent graft. Prevention of these complications can be partially achieved during the primary procedure by ensuring adequate landing zones proximal and distal to the stent graft, and closure of large fenestrations along the length of the false lumen. The use of a provisional bare metal stent as a distal extension to the stent graft has been reported to facilitate aortic remodeling and help completely repair the distal dissected segments without compromising important side-branches (Provisional Extension To Induce Complete Attachment after stent-graft implantation (PETTICOAT) technique) [25]. This would address the problem of persistent perfusion within the false lumen as well as any increase in aneurysmal size.

14. Stent-graft infection

The infected prosthetic graft represents a rare complication that could be difficult to diagnose despite extensive work up, with consensus reached only after thorough analysis of imaging, hematological, and clinical parameters. The presence of air, soft tissue accumulation, and progressive enlargement of the aneurysm sac is usually pathognomonic, and suspicious changes spotted in imaging coupled with raised markers of systemic inflammation and clinical symptoms are generally suggestive of an infective process. The diagnostic use of positron emission tomography (PET) radionuclide studies is a helpful tool in most cases. Prior to initiation of treatment against thoracic endograft infections, data concerning diagnostic certainty, pathogenesis of the infecting microbe, the extent of infection, the presenting features, and the medical co-morbidities of a given patient need to be evaluated.

As with the treatment of all prosthetic graft infections, multiple management strategies have been proposed in this scenario. These include a conservative line of treatment with targeted intravenous antibiotics or also alternatively by direct puncture and application into the peri-graft space; insertion of another stent graft into the potentially infected graft or excision of the infected stent graft with debridement of the surrounding tissue; and in-situ/extra-anatomic vascular reconstruction. Irrespective of the applied strategy, the treatment of aortic graft infection remains difficult and problematic. In the absence of management algorithms defining treatment strategies in the varying forms of presentation and degrees of infection, individualized therapies need to be devised while weighing the risks in each scenario. In

general, a decision must be made as to whether treatment is to be potentially curative or palliative. The curative treatment approach encompasses an aggressive open surgery, justified only in certain cases in lieu of the relatively high mortality associated with the procedure.

The palliative approach suggests placement of an endovascular graft (graft-in-graft approach) thereby preventing the risk of life-threatening bleeding or fistulation. Patients with complex graft disease should always be treated in experienced centers equipped to conduct all treatment variations.

15. Retrograde Type A dissection following stent-graft placement

There have been cases reporting a proximal aortic dissection after placement of an endograft; the incidence of this complication, according to existing data, ranges presumably between 1 and 2% and generally occurs shortly after the intervention [30, 31]. Chest pain or symptoms of an ischemic heart or brain should immediately alert the suspicion that a Type B dissection has modified itself to a Type A dissection, with a higher morbidity and mortality risk. An emergency Computed tomography angiography (CTA) and subsequent surgery are the only lifesaving options for the patient. There are several reasons why Type A dissections may occur after stent-graft placement in the descending thoracic aorta (**Table 2**).

The initial misinterpretation of a Type A dissection as a Type B dissection

Spread of the dissection into the ascending aorta or aortic arch due improper placement of the stent graft relatively distant to the LSA, or the possible maneuvering into the false lumen

Under- or oversizing of the stent graft, with consequent leaks or additional injuries to the aortic wall

Malapposition of the stent graft to the aortic wall at the proximal landing zone resulting in subsequent collapse of the proximal stent graft

Ballooning of the proximal end of the stent graft resulting in injury of the diseased aortic wall, thus causing the extension of the dissection in the aortic arch and ascending aorta

Development of a Type A dissection independent of the existing Type B dissection

Table 2. Cause of Type A aortic dissection after stent-graft implantation.

In these cases, it is of absolute importance to advance the guide-wire, catheter, and endograft in the true lumen only. If doubts persist concerning the correct positioning of the guide wire (in the true or false lumen), the use of angiography and transoesophageal echocardiography is instrumental before initiation of the next step. Signs differentiating the true from the false lumen include luminal size, where the false aortic lumen is usually larger than the true lumen, as well as flow at entry, which is generally directed into the false lumen. This explains the damping of the pulse amplitude and a poorly palpable pulse in many of these patients.

Precise measurement of the proximal aortic dimensions is essential for the selection of the size of stent graft and successful sealing of aortic tears. In Type B dissection, the stent graft is

normally placed with its proximal end directly at the origin of the LSA. The proximal landing of a stent graft is usually close to the LSA, even though the tear could be in the middle portion; so performed, when considering the possibility of a dissection membrane developing a new tear between LSA and proximal end of the stent graft. In successful cases, angiography demonstrates a good result with complete sealing of the leak and absence of signs showing renewed opacification of the false lumen. Occasionally, a stiff endograft chafes on the fragile dissection membrane and a new endoleak may develop. This could possibly require additional stent grafting or eventually lead to open surgery.

16. Paraplegia after stent-graft placement

The risk of spinal cord ischemia is of significant concern post stent-graft placement as there is a frequent need to cover multiple intercostal arteries as also the artery of Adamkiewicz (the single prominent intersegmental branch from the aorta at the lower thoracic or upper lumbar level). Interestingly, around 3–12% of the stent-graft patients treated for aneurysms, dissections, ulcers, intramural hematoma, or aortic traumatic transection are at risk to develop spinal cord ischemia. Although occlusion of important radicular arteries that originate from vertebral, intercostal, and lumbar arteries is primarily responsible for ischemia of the spinal cord, associated factors such as ischemia-reperfusion and hypotension may also play a causal role in the development of paraplegia. This is a telling contradiction to the widely held impression that the coverage of the segmental artery or the orifices of the intercostal/interlumbar arteries by the stent graft represents the single possible and most critical cause of spinal ischemia. Measures to prevent occurrence of paraplegia in stent-graft patients should include several aspects (**Table 3**).

Screening for high-risk patients

- o Age > 75
 - o Anticipated endograft coverage between T9 and T12 (location of anterior spinal artery)
 - o Coverage of long segment (>20 cm)
 - o Compromised collateral pathways (e.g., LIMA as coronary artery bypass, infrarenal surgical aortic repair)
 - o Long extent of atherosclerotic lesions
 - Early detection of spinal cord ischemia
 - o Somatosensory-evoked potentials
 - o Motor-evoked potentials
 - Monitoring of cerebral or spinal cord perfusion and drainage (spinal tap decreases intrathecal pressure to 10–15 cm H₂O to generate space for collateral arteries to fill and perfuse better)
 - Prevention of perioperative hypotension
-

Table 3. Factors influencing the rate of paraplegia.

Symptoms of paraplegia or paraparesis could be possibly reversed if a spinal tap is introduced without any delay and arterial perfusion pressure is maintained at a systolic pressure of 140 mmHg by pharmacological means or volume replenishment.

17. Stroke after stent-graft placement

Stroke is a grave complication occurring post stent-graft replacement and reports of incidence vary between 0 and 18%. The instrumentation in the aortic arch has been known to produce an embolic shower in the brain, and the use of guidewires and catheters as well as bulky delivery systems carrying the stent grafts in endovascular interventions in this region supplements to the risk. The need for balloon dilatation of stent grafts adds to the possibility of dislodgement of particles from the aortic arch. It is also well known that proper flushing of the stent-graft delivery system does not guarantee the elimination of air contained in the crimped stent graft. Moreover, bubbles are also released with the deployment of the stent graft. The confirmation of an open contralateral vertebral artery with correct formation of the basilar trunk and absence of intracranial branch anomalies is a basic prerequisite in patients in whom subclavian artery closure is considered.

In summary, there is an absolute need to completely define the left vertebral artery with depiction of its origin, patency, and size, as well as the size and condition of the right vertebral artery including the constitution of the basilar artery and its branches before conducting a procedure involving the closure of the LSA. In patients with compromised cerebral circulation, prior diagnosis of the four vessels with cerebral angiography is the recommended standard. When the left vertebral artery takes off from the aortic arch, occlusion of the LSA can result in severe ischemia of the left arm. If the left vertebral artery takes off from an aneurysm that is being excluded, it could result in a Type II endoleak, a severe steal phenomenon or even present as an ipsilateral posterior stroke. In such cases, a transposition of the LSA prior to the intervention is required.

18. Conclusions

Endovascular stent grafting represents an exciting therapeutic advancement and has emerged as an alternative therapy to open surgical repair in thoracic aortic pathologies. Although it is apparent that high-risk patients will benefit from this technology, the exact role of stent grafting needs to be defined as long-term data is constantly accumulated and analyzed, and further influenced by evolving devices and technology. Rather than replacing conventional surgical treatment, endovascular repair will likely play a complementary role and offer a less invasive option in the treatment armamentarium. Clearly, there are limitations to both approaches; however, while high-risk surgery is defined by clinical parameters, comorbidities and physiological reserve, contraindications for endovascular stent-graft treatment are defined mostly by anatomical criteria such as a too wide aorta to provide landing zones for an endoprosthesis or already irreversible paraplegia. Nevertheless, treatment should be carried out only in a center with experience in both endovascular and surgical procedures, and with adequate technical facilities. Treatment of thoracic aortic pathologies should be subject to prior multidisciplinary

discussion, particularly with regard to risks of conversion and need for cardiopulmonary bypass. All patients should have access to a structural follow-up plan offering both regular clinical assessment and professional imaging follow-up by CT or MR angiography.

Conflict of interest

No conflict of interest for authors.

Author details

Ibrahim Akin^{1*}, Uzair Ansari¹ and Christoph A. Nienaber²

*Address all correspondence to: ibrahim.akin@umm.de

1 Universitätsmedizin Mannheim, Mannheim, Germany

2 Royal Brompton Hospital and Harefield Trust, London, UK

References

- [1] Cooley DA, DeBakey ME. Resection of the thoracic aorta with replacement by homograft for aneurysms and constrictive lesions. *J Thorac Surg* 1955; 29: 66–100.
- [2] Svensson LG, Crawford ES, Hess KR, Coselli JS, Safi HJ. Experience with 1,509 patients undergoing thoracoabdominal aortic operations. *J Vasc Surg* 1993; 17: 357–368.
- [3] Trimarchi S, Nienaber CA, Rampoldi V, Myrmet T, Suzuki T, Bossone E, Tolva V, Deeb MG, Upchurch GR Jr, Cooper JV, Fang J, Isselbacher EM, Sundt TM 3rd, Eagle KA; IRAD Investigators. Role and results of surgery in acute type B aortic dissection: insight from the International Registry of Aortic Dissection (IRAD). *Circulation* 2006; 114(1 Suppl): I357–I364.
- [4] Volodos NL, Shekhanin VE, Karpovich IP, Troian VI, Gur'ev IuA. A self-fixing synthetic blood vessel endoprosthesis. *Vestn Khir Im I I Grek* 1986; 137: 123–125.
- [5] Dake MD, Miller DC, Semba CP, Mitchell RS, Walker PJ, Liddell RP. Transluminal placement of endovascular stent-grafts for the treatment of descending thoracic aortic aneurysms. *N Engl J Med* 1994; 331: 1729–1734.
- [6] Nienaber CA, Fattori R, Lund G, Dieckmann C, Wolf W, von Kodolitsch Y, Nicolas V, Pierangeli A. Nonsurgical reconstruction of thoracic aortic dissection by stent-graft placement. *N Engl J Med* 1999; 340: 1539–1545.
- [7] Coady MA, Rizzo JA, Hammond GL, Pierce JG, Kopf GS, Elefteriades JA. Penetrating ulcer of the thoracic aorta: what is it? How do we recognize it? How do we manage it?. *J Vasc Surg* 1998; 27: 1006–1015.

- [8] Evangelista A, Mukherjee D, Mehta RH, O'Gara PT, Fattori R, Cooper JV, Smith DE, Oh JK, Hutchison S, Sechtem U, Isselbacher EM, Nienaber CA, Pape LA, Eagle KA; International Registry of Aortic Dissection (IRAD) Investigators. Acute intramural hematoma of the aorta: a mystery in evolution. *Circulation* 2006; 111: 1063–1070.
- [9] Ganaha F, Miller DC, Sugimoto K, Do YS, Minamiguchi H, Saito H, Mitchell RS, Dake MD. Prognosis of aortic intramural hematoma with and without penetrating atherosclerotic ulcer: a clinical and radiological analysis. *Circulation* 2002; 106: 342–348.
- [10] Nienaber CA, von Kodolitsch Y, Petersen B, Loose R, Helmchen U, Haverich A, Spielmann RP. Intramural hemorrhage of the thoracic aorta. Diagnostic and therapeutic implications. *Circulation* 1995; 92: 1465–1472.
- [11] Erbel R, Alfonso F, Boileau C, Dirsch O, Eber B, Haverich A, Rakowski H, Struyven J, Radegran K, Sechtem U, Taylor J, Zolikofer C, Klein WW, Mulder B, Providencia LA; Task Force on Aortic Dissection, European Society of Cardiology. Diagnosis and management of aortic dissection. *Eur Heart J* 2001; 22: 1642–1681.
- [12] Svensson LG, Kouchoukos NT, Miller DC, Bavaria JE, Coselli JS, Curi MA, Eggebrecht H, Elefteriades JA, Erbel R, Gleason TG, Lytle BW, Mitchell RS, Nienaber CA, Roselli EE, Safi HJ, Shemin RJ, Sicard GA, Sundt TM 3rd, Szeto WY, Wheatley GH 3rd; Society of Thoracic Surgeons Endovascular Surgery Task Force. Expert consensus document on the treatment of descending thoracic disease using endovascular stent-grafts. *Ann Thorac Surg* 2008; 85(1 Suppl): S1–S41.
- [13] Hagan PG, Nienaber CA, Isselbacher EM, Bruckman D, Karavite DJ, Russman PL, Evangelista A, Fattori R, Suzuki T, Oh JK, Moore AG, Alouf JF, Pape LA, Gaca C, Sechtem U, Lenferink S, Deutsch HJ, Diedrichs H, Marcos y Robles J, Llovet A, Gilon D, Das SK, Armstrong WF, Deeb GM, Eagle KA. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. *JAMA* 2000; 283: 897–903.
- [14] Kodolitsch Y, Schwarz AG, Nienaber CA. Clinical prediction of acute aortic dissection. *Arch Intern Med* 2000; 160: 2977–2982.
- [15] Clouse WD, Hallett JW Jr, Schaff HV, Gayari MM, Ilstrup DM, Melton LJ 3rd. Improved prognosis of thoracic aneurysms: a population-based study. *JAMA* 1998; 280: 1926–1929.
- [16] Crawford ES, Crawford JL, Safi HJ, Coselli JS, Hess KR, Brooks B, Norton HJ, Glaeser DH. Thoracoabdominal aortic aneurysms: preoperative and intraoperative factors determining immediate and long-term results of operations in 605 patients. *J Vasc Surg* 1986; 3: 389–404.
- [17] Swee W, Dake MD. Endovascular management of thoracic dissection. *Circulation* 2009; 117: 1460–1473.
- [18] Tsai TT, Evangelista A, Nienaber CA, Myrmel T, Meinhardt G, Cooper JV, Smith DE, Suzuki T, Fattori R, Llovet A, Froehlich J, Hutchison S, Distane A, Sundt T, Beckman J, Januzzi JL Jr, Isselbacher EM, Eagle KA; International Registry of Acute Aortic Dissection. Partial thrombosis of the false lumen in patients with acute type B aortic dissection. *N Engl J Med* 2007; 357: 349–359.

- [19] Salvolini L, Renda P, Fiore D, Scaglione M, Piccoli G, Giovagnoni A. Acute aortic syndromes: role of multidetector row CT. *Eur J Radiol* 2008; 65: 350–358.
- [20] Estrera AL, Miller CC 3rd, Safi HJ, Goodrick JS, Keyhani A, Porat EE, Achouh PE, Meada R, Azizzadeh A, Dhareshwar J, Allaham A. Outcomes of medical management of acute type B aortic dissection. *Circulation* 2006; 114(Suppl I): 384–389.
- [21] Kodama K, Nishigami K, Sakamoto T, Sawamura T, Hirayama T, Misumi H, Nakao K. Tight heart rate control reduces secondary adverse events in patients with type B acute aortic dissection. *Circulation* 2008; 118(14 Suppl): S167–S170.
- [22] Nienaber CA, Kische S, Rehders TC, Schneider H, Chatterjee T, Büniger CM, Höppner R, Ince H. Rapid pacing for better placing: comparison of techniques for precise deployment of endografts in the thoracic aorta. *J Endovasc Ther* 2007; 14: 506–512.
- [23] Rehders TC, Petzsch M, Ince H, Kische S, Korber T, Koschyk DH, Chatterjee T, Weber F, Nienaber CA. Intentional occlusion of the left subclavian artery during stent-graft implantation in the thoracic aorta: risks and relevance. *J Endovasc Ther* 2004; 22: 659–666.
- [24] Peterson BG, Eskandari MK, Gleason TG, Morasch MD. Utility of left subclavian artery revascularization in association with endoluminal repair of acute and chronic thoracic aortic pathology. *J Vasc Surg* 2006; 43: 433–439.
- [25] Nienaber CA, Kische S, Zeller T, Rehders TC, Schneider H, Lorenzen B, Büniger C, Ince H. Provisional extension to induce complete attachment after stent-graft placement in type B aortic dissection: the PETTICOAT concept. *J Endovasc Ther* 2006; 13: 738–746.
- [26] Schumacher H, von Tengg-Kobligk H, Ostovic M, Henninger V, Ockert S, Böckler D, Allenberg JR. Hybrid arch procedures for endoluminal arch replacement in thoracic aneurysms and type B dissection. *J Cardiovasc Surg (Torino)* 2006; 47: 509–517.
- [27] Saleh HM, Inglese L. Combined surgical and endovascular treatment of aortic arch aneurysms. *J Vasc Surg* 2006; 44: 460–466.
- [28] Czerny M, Gottardi R, Zimpfer D, Schoder M, Grabenwoger M, Lammer J, Wolner E, Grimm M. Transposition of the supraaortic branches for extended endovascular arch repair. *Eur J Cardiothorac Surg* 2006; 29: 709–713.
- [29] Hata M, Shiono M, Inoue T, Sezai A, Niino T, Negishi N, Sezai Y. Optimal treatment of type B acute aortic dissection: long-term medical follow-up results. *Ann Thorac Surg* 2003; 75: 1781–1784.
- [30] Eggebrecht H, Thomson M, Rousseau H, Czerny M, Lönn L, Mehta RH, Erbel R; European Registry on Endovascular Aortic Repair Complications. Retrograde ascending aortic dissection during or after thoracic aortic stent graft placement: insight from the European registry on endovascular aortic repair complications. *Circulation* 2009; 120(Suppl 11): S276–S281.
- [31] Dong ZH, Fu WG, Wang YQ, Guo da Q, Xu X, Ji Y, Chen B, Jiang JH, Yang J, Shi ZY, Zhu T, Shi Y. Retrograde type A aortic dissection after endovascular stent graft placement for treatment of type B dissection. *Circulation* 2009; 119: 735–741.