European Review for Medical and Pharmacological Sciences 2014; 18: 2562-2574

Interventional treatment strategies of thoracic aortic pathologies

I. AKIN, C.A. NIENABER[1]

Department of Internal Medicine I, Medical Faculty Mannheim, University Heidelberg, Mannheim, Germany
[1]Department of Internal Medicine I, Heart Center Rostock, University of Rostock, Rostock, Germany

Abstract. – Chronic and acute diseases of the thoracic aorta, including aortic dissection and aortic aneurysm are attracting increasing attention both in the light of an ageing Western and Oriental population and with the proliferation of modern diagnostic imaging modalities. While classical surgical strategies still dominate the care for acute and chronic pathology of the ascending aorta and the proximal arch region, new endovascular concepts are emerging and are likely to evolve as primary treatment strategies for descending aortic pathology in suitable patients constituting the majority of cases. Additionally, aortic arch pathologies are becoming the target of hybrid approaches combining surgical head-vessel debranching and interventional stent-graft implantation in the attempt to improve outcome by avoiding the high risk of open arch repair or complete replacement. Nonetheless, due to the complexity of the underlying vascular disease, every patient should be discussed in a team consisting of cardiologists, cardiac surgeons, anaesthesiologists and radiologists and an individualized therapeutic strategy should be carried out in a center with experience in both endovascular and surgical procedures.

Key Words:
Aortic dissection, Aortic aneurysm, Stent graft, Endoleak, Malperfusion.

Introduction

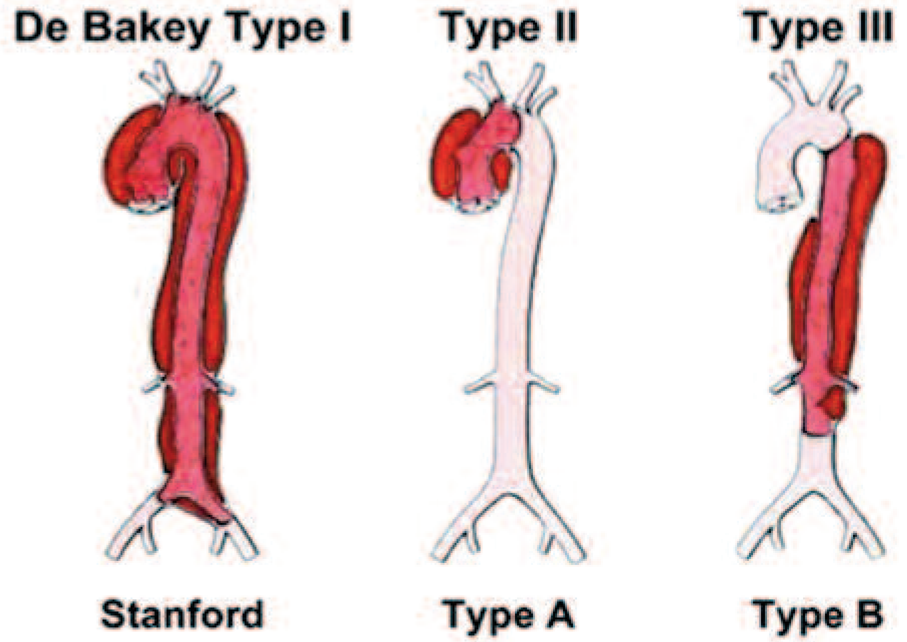
The pathophysiologic processes underlying the development of thoracic aortic disease are complex and until yet not fully understood. Surgical resection and interposition of vascular prostheses have long been considered the standard treatment in most instances, despite a substantial risk of severe complications from surgical trauma[1-3]. Regardless of great strides in the past decades to improve surgical techniques and intraoperative management, both perioperative mortality and morbidity remain high. As a consequence of demographic changes in the Western and Oriental world, the afflicted population is of increasing age and associated with a variety of comorbidities portending an inherent risk and explaining in part the sobering surgical outcomes. As a revolutionary alternative, the concept of an endoluminal stent-graft prosthesis for patients with thoracic aortic disease emerged more than two decades ago in order to avoid risks of open surgery using the interventional approach and to induce reconstructive remodeling of the diseased aorta by initiating a natural healing process through exclusion and depressurization of the aneurismal process[4-6]. Although initial reports on the endovascular stent-graft strategy were encouraging in various pathologies (e.g. degenerative aneurysm, mycotic aneurysm, traumatic injuries, aortic dissections and penetrating aortic ulcers [PAU]), randomized data are scarce, and the smoldering critique has never been fully extinguished with respect to the lack of long-term surveillance[7-10]. This review described current indications, techniques and advancing fields of endovascular strategies in the thoracic aorta.

Classification

The most common thoracic aortic pathologies are aortic dissection and aortic aneurysm. Nevertheless, aortic dissection is a relative rare condition in the clinical arena, with an estimated incidence of approximately 2.6 to 4 cases per 100.000 person/year[11-13]. Around 0.5% of patients presenting to an emergency department with chest or back pain suffer from aortic dissection[14]. Aortic dissection diagnosed within 2 weeks of the onset of symptoms e.g. during the early phase of the disease associated with high mortality, is considered an acute dissection. Patients who survive 2 weeks without treatment are considered subacute, and eventually chronic after eight weeks constituting about one third of patients with aortic dissection. Aortic dissection is also classified according to

Corresponding Author: Ibrahim Akin, MD; e-mail: ibrahim.akin@umm.de

anatomic location described by either the Stanford and the DeBakey classification (Figure 1). The fundamental distinction is whether the dissection is proximal (involving the aortic root or ascending aorta) or distal (beyond the left subclavian artery). Untreated proximal aortic dissection is characterized by mortality rate of more than 1% per hour initially; if left untreated death may occur from rupture, tamponade, heart failure from acute aortic regurgitation or from major coronary closure[11-13]. The natural history of type B aortic dissection is significantly better with a 30-day mortality rate of about 10%[11-13]. Conversely, little is known about the true incidence and mortality rate of thoracic aortic aneurisms (TAA). Accordingly, a population-based study reported an age- and gender-adjusted incidence of 5.9 new aneurysms per 100,000 person-year in a Midwestern community over a 30-year period with median ages of 65 years for men and 77 years for women and a distribution to the ascending aorta in 51%, to the arch in 11%, and to the descending thoracic aorta in 38%[15]. Aneurysms distal to the origin of the left subclavian artery are classified according to the Crawford et al classification (type I to type IV), recently adapted 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.

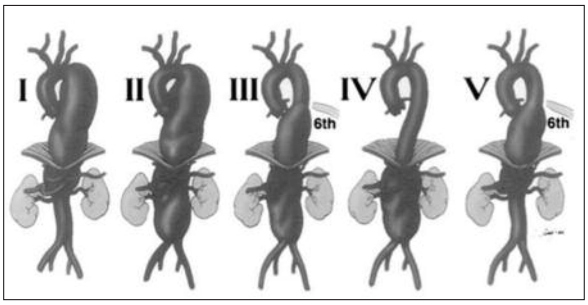


Figure 2. Classification of thoracic aortic aneurysms.

Indications for Endovascular Stent-Graft Therapy

The most effective method for excluding and enlarging an aneurysmal false lumen after dissection is the sealing of proximal entry tears with a customized stent-graft. The closure of a distal reentry tear is desirable but not necessary for optimal results[12,13,17]. Depressurization and shrinking of the false lumen is the most favorable outcome that can be expected, ideally followed by complete thrombosis of the false lumen and remodeling of the entire dissected aorta[18] (Figure 3). The role of percutaneous fenestration in the management of aortic dissection is not fully understood but should be most likely avoided due to unproven benefits and definite risk. Beside clinical criteria, there are also technical and anatomical criteria to be evaluated prior to defining the treatment strategy by an aortic team. Similar to previously accepted indications for surgical intervention scenarios such as intractable pain with descending dissection, rapidly expanding false lumen diameter, extra-aortic blood collection as a sign of imminent rupture or distal malperfusion syndrome are accepted indications for emergency stent-graft placement[11-13] (Table I). Moreover, late onset of complications such as malperfusion of vital aortic side branches may justify endovascular stent-grafting as a first option.

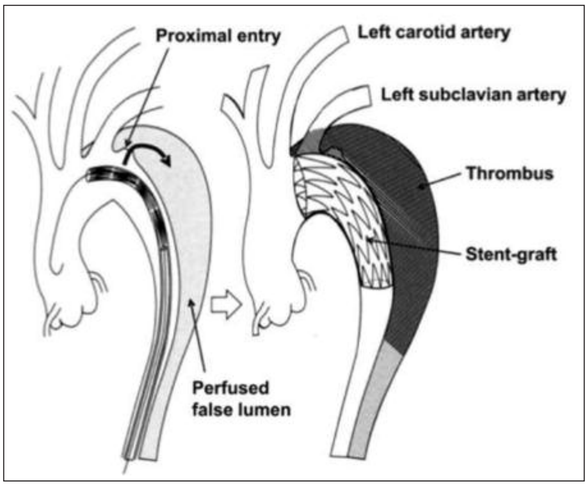


Figure 3. Concept of interventional reconstruction of the dissected aorta with sealing of the proximal entries, depressurization of the false lumen and initiation of false lumen thrombosis.

Standard Case

Anatomical Measurements for Thoracic Aortic Pathology

Important descriptive features of the thoracic aorta are derived from multislice computed tomography (CT) including the shape and size of the aortic pathology (diameter, length and shape), and the condition of the aortic wall (atheroma, calcification, thrombus) as well as a three-dimen-

Table I. Current indications for endovascular treatment of thoracic aortic disease.

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 months

sional reconstruction[19]. Although there is no standard convention for the measurement of vessel diameters, many operators measure the inner wall of the vessel (endothelial trailing edge) on the basis that this will guarantee some degree of oversizing that is considered desirable for endograft placement. Our protocol measures the diameter of the proximal normally appearing aorta (inner edge to edge) from a transversal plane perpendicular to the long axis of the aorta (preferentially from contrast-enhanced multi-slice CT images) (Figure 4). Contrast angiography does not provide reliable measurements. In addition to initial obligatory diagnostic CT angiography or magnetic resonance (MR) angiography, transesophageal echocardiography and intravascular ultrasound may be performed providing additional valuable information. For instance flow sensitive MR sequences or contrast-enhanced TEE not only shows the communication sites between true and false lumen, but also insight to the dynamic flow pattern in the false lumen prior to stent-graft placement. Access vessels must also be evaluated for size and tortuosity as the stent-graft delivery systems are quite large (up to 24F) and can cause significant trauma to the femoral access site and iliac arteries. Features that are unfavorable for thoracic stent-graft use include severe aortic angulation or tortuosity, friable atheroma or thrombus lining the aortic wall, and aortic pathology involving the ascending aorta. Moreover, the vicinity of relevant side branches, usually the left subclavian artery or left common carotid artery is critical while planning to place a stent-graft for type B dissection; appropriate periinterventional image reconstruction of the aortic arch pathology enhances the individual result in any given case.

Initial Treatment

Patients with suspected acute aortic disease should be admitted to an intensive care or monitoring unit and undergo diagnostic evaluation immediately[20]. Pain and blood pressure control to a target systolic pressure of 110 mmHg can be achieved using morphine sulfate and intravenous β-blockers (metoprolol, esmolol or labetalol) or in combination with vasodilating drugs such as sodium nitroprusside or an-

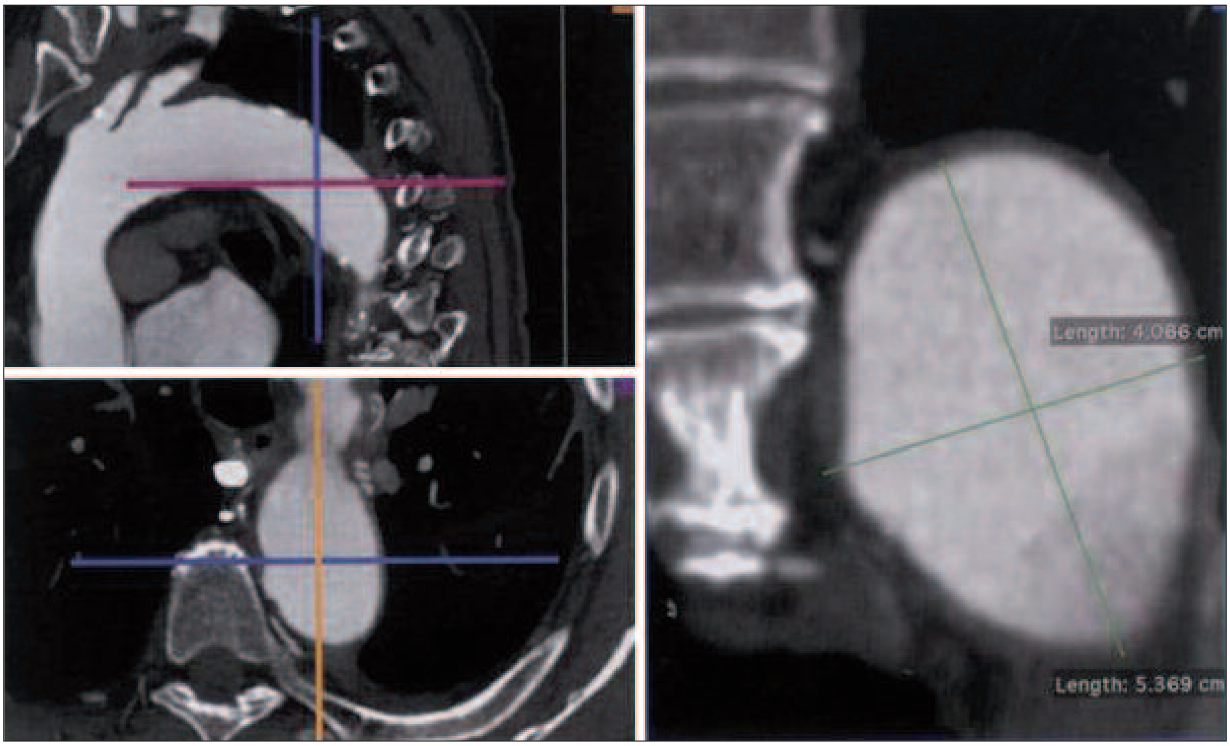


Figure 4. CT scan of a patient with dilated thoracic aorta. Due to the fact that the aorta follows a curved and three-dimensionally tortuous path, the axial scans are inadequate for measuring aortic diameters, as are sagittal and coronal planes for measuring aortic lengths.

giotensin-converting enzyme inhibitors. Intravenous verapamil or diltiazem may also be used if β-blockers are contraindicated. Monotherapy with β-blocking agents may be adequate to control mild hypertension and, in concert with sodium nitroprusside at an initial dose of 0.3 μg/kg/min, is often effective in a severe hypertensive state. In normotensive or hypotensive patients, careful evaluation for loss of blood, pericardial effusion or heart failure (by cardiac ultrasound) is mandatory before administering volume. Additionally, heart rate control is of utmost importance[21]. Patients with profound haemodynamic instability often require intubation, mechanical ventilation and urgent bedside transoesophageal echocardiography or rapid CT for confirmatory imaging. In rare cases, the external ultrasound diagnosis of cardiac tamponade may justify immediate sternotomy and surgical access to the ascending aorta to prevent circulatory arrest, shock and ischaemic brain damage.

Technique of Endovascular Stent-Graft Placement

Based on measurements obtained during transesophageal echocardiography (mandatory for detection of small entries), contrast-enhanced CT angiograms (best technique for unstable patients in an emergency situation), or intravascular ultrasound (IVUS) using a 10 Mhz ultrasound catheter-mounted transducer that should be manually maneuvered through the diseased aorta in the attempt to better identify communications, partial thrombosis or other peculiarities, individually selected stent-grafts should be used to cover up to 20 cm (and sometimes even more) of diseased aorta. The procedure is best performed in the catheterization laboratory or hybrid operating room equipped with digital angiography; general anesthesia is obligatory in all patients. The femoral artery is the most common access-site that usually can accommodate a 20-24 F stent-graft system. Using the Seldinger technique to place a sheath a 260 cm stiff wire (e.g. Amplatzer) is placed over a pigtail catheter navigated with a soft wire in the true lumen under both fluoroscopic and transesophageal ultrasound guidance. In complex cases with multiple reentries in the abdominal aorta the "embracement-technique" with the use of 2 pig-tail catheters is useful to maintain the true lumen. In essence, a catheter navigating true aortic lumen via the left brachial artery picks up the femoral pig-tail catheter in the true lumen of the abdominal aorta and pulls it up into the aortic arch. This procedure ensures definite positioning of the stiff guide wire within the true lumen, that is essential for the subsequent endovascular procedure. Launching of the stent-graft is performed by carefully advancing the graft over the stiff wire in the true lumen with systolic blood pressure briefly lowered by rapid right ventricular pacing[22]. After deployment at the target site and sealing of the entry flap short inflation of a latex balloon may be used to improve apposition of the stent struts to the aortic wall, if proximal sealing of thoracic communications was incomplete. Both Doppler-ultrasound and contrast fluoroscopy are instrumental for documenting the immediate result or for initiating adjunctive maneuvers. In the setting of thoracic aortic aneurysms (TAA) or ulcers the navigation of wires and instruments may be facilitated, yet meticulous intraprocedural ultrasound imaging and fluoroscopy are equally important to monitor the progress of the intervention. A frequent anatomical consideration is the close vicinity between the origin of the left subclavian artery (LSA) and the primary tear in type B dissections; complete coverage of the LSA ostium has to be accepted at times for proper placement and debranching or extra-anatomic bypasses have to be considered[23]. According to observational evidence prophylactic surgical maneuvers are not always required, but may be performed electively after an endovascular aortic intervention if intolerable signs or symptoms of ischemia develop. However, prior to intentional LSA occlusion attention has to be paid to potential supra-aortic variants (e.g. presence of a lusorian artery, an incomplete vertebro-basilar system or vertebral arteries, directly originating from the arch)[24].

Device Sizing and Length

Both for dissections and TAA, decision must be made about the length of the true lumen that should be scaffolded by an endoprosthesis. The key initial decision is whether the strategy should be to cover only the proximal entry point, or alternatively to line a longer length of true lumen, eventually to treat the descending thoracic aorta down to diaphragm level. Although longer scaffolding provides potentially greater stability and lower risk of recurrence, the main reason to avoid extensive coverage is the potential risk of paraplegia resulting from compromising of spinal arterial supply. There is observational evidence that extensive coverage exceeding 20 cm of stent-graft, previous surgery to the abdominal aorta, overstenting of the LSA or use of the left mammary artery for coronary bypass is associated with increased risk of spinal ischemia. In general, all previous measures that led to compromised collateral flow to the spinal cord arterial network are heralding an increased risk for neurological complications; in other words LSA overstenting and long stent-grafts are to be avoided in such scenarios. Conversely, the angiographic identification of the spinalis anterior artery was newer proven to be prognostically relevant. The main disadvantage of shorter lengths of coverage is the tendency of multiple re-entries to be present more distally in the descending thoracic aorta maintaining perfusion of the false lumen even after proximal entry-site closure. In such scenarios distal extension of the scaffolding concept of endoluminal repair could be ensured by uncovered bare metal stents to ensure distal reapposition of the dissecting lamella without any compromise to abdominal side branches or spinal arteries. 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 may require a degree of device oversizing of 10-15%, while endoprosthesis in dissection should not be oversized. Excessive oversizing can result in trauma to the aortic wall resulting in retrograde dissection into the arch or conversion of a type B dissection into a type A dissection, aortic perforation or false aneurysm formation. More distally, oversizing may result in tearing of the membrane between the two channels, resulting in a new re-entry point and ongoing pressurized perfusion of the false channel. If decision was made to scaffold the aortic lumen more extensively, the operator must decide whether to use one long or two different shorter grafts. The ultimate decision represents a compromise between reducing the number of modular junctions, and the additional frictional forces associated with the deployment of a long stent-graft. When two stent-grafts are necessary, then, they are employed in “telescope” fashion; the amount of overlap should be at least of 30 mm in straight anatomic segments, and up to 50mm or more in angulated or curved segments of the aorta. Geometry of the presumed junction between the grafts provides some assistance in estimating the length of the overlap required at connecting zones. The critical factor to note, however, is the length of the radius of the curve; the shorter this radius, the longer the stent overlap is required on the lesser curve. The second factor that comes into play when making a 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 fusiforme aneurysm rather than in a segment of aorta with normal diameter, the required overlap should be longer. The reason for this approach is a tendency of the graft to move out towards the greater curve of the aneurysmatic aortic segment due to mechanical forces associated with the pulsatile motion of the aorta, potentially causing migration or disconnection of modular components.

Extended Scaffolding with Bare-Stents

There is general agreement that entry tears should be treated with a covered stent-graft. The remaining distal thoracic or even abdominal aorta may be supported by additional uncovered stents[25]. In selecting the suitable stent-graft the following principles should be followed:

- The appropriate size and diameters are crucial to avoid erosion through the aortic wall and to assure optimal conformability.
- The endoprosthesis should also be flexible and released with a deployment mechanism that is easy to use and provides accurate placement at the desired zone.
- Due to the fragility of the dissected aortic wall, it is preferred to use a stent graft which is not dependent on balloon expansion for deployment or post deployment modeling. We prefer self-expanding endoprosthesis with a nitinol-based architecture and only limited radial force (in case of aortic dissection) and avoid any ballooning.

Landing Zone

Experience has shown that the 3-dimensional mechanical forces associated to the pulsatile flow that “play” on an endoprosthesis in the thoracic aorta are far greater and more complex than those in the abdominal aorta. Consequently, anchorage of the graft must be as stable as possible and the apposition of the graft should be optimal. Proximal and distal landing zones should ideally encompass 15 mm in length and no aortic wall atheroma or thrombus. The earlier concept of the left subclavian artery being a natural barrier beyond which it was impossible to deploy stent grafts is no longer valid. The comparatively normal nature of the aortic arch between the left subclavian artery and left common carotid artery origins and the horizontal orientation compared with the aorta distal to the LSA, have encouraged interventionalists to use this segment of the aorta as a preferred anchor zone with or without bypassing the LSA. It is considered prudent to visualize the cerebral circulation by DSA, CT or magnetic resonance angiography to identify patients with an incomplete circle of Willis or other limitations or abnormalities of vascular supply to the brain. In these patients protective measurees such as carotid-subclavian bypass might be necessary prior to stent-covering the LSA. Of course, there is always the option of using proximal bare-stents for better alignment without the risk of occluding a vital side branch such as the LSA.

Hybrid Arch Procedures

The aortic arch anatomy and pathology is challenging because of variable degree of length and degree of angulation, and the proximity of the supra-aortic branches that need to be preserved[26-28]. Traditional open arch surgical reconstruction requiring hypothermic cardiac arrest, extracorporal circulation and selective cerebral perfusion has been demonstrated to effectively manage aortic arch pathologies. However, these major surgical procedures carry significant mortality and risk of paraplegia and cerebral stroke. Therefore, such open repair is preferred particularly in low-risk younger patients. Typical high-risk patients are not considered candidates for this surgical approach. Hybrid arch procedures are a combination of debranching bypass (supra-aortic vessel transposition) to preserve cerebral perfusion and subsequent thoracic endografting to provide patient-centred solutions for complex aortic arch lesions (Figure 5). Hybrid arch procedures are performed without hypothermic circulatory arrest and extracorporal circulation and could expand the treatment group to older and high-risk patients with severe co-morbidities currently not eligible for open surgical repair.

Aftercare and Long-Term Follow-up

The long-term care of patients with successful initial treatment of acute aortic dissection begins with the appreciation of a systemic illness. It has been estimated that nearly one-third of patients initially surviving an acute dissection of the thoracic aorta shall experience extension of dissection or late aortic rupture, or require surgery for aortic aneurysm formation within 5 years of presentation[29]. All patients merit aggressive medical therapy, follow-up visits and serial CT imaging.

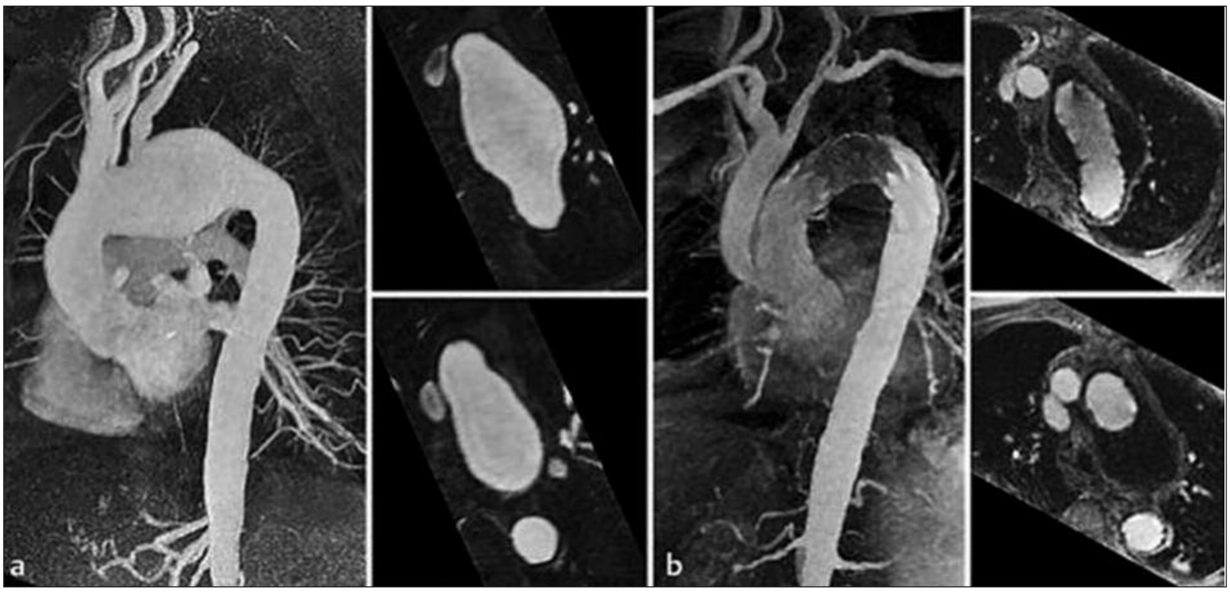


Figure 5. Contrast-medium enhanced MR-angiography of the aorta in a case of an arch aneurysm. ***A,*** Aneurysm of the aortic arch involving the supra-aortic branches. ***B,*** Result after hybrid procedure with debranching of the supraaortic vessels and stent-graft placement in the aortic arch.

Treatment with effective β-blockade is the cornerstone of medical therapy. By lowering both blood pressure and steepness of the pressure rise (dp/dt), β-blockers have been shown to retard aortic expansion in Marfan's syndrome and also in patients with chronic abdominal aortic aneurysms (AAA). Blood pressure should be titrated to less than 135/80 mmHg and even to less than 130/80 mmHg in Marfan's syndrome[11-13]. Additionally, heart rate should be controlled since a heart rate under 60 significantly decreases secondary adverse events (aortic expansion, recurrent aortic dissection, aortic rupture and/or need for aortic surgery) in type B aortic dissection compared to conventional control of > 60 bpm[21]. Serial CT imaging of the aorta is an essential component of long-term management (before and after surgery or stent-graft placement). Choice of imaging modality, CT or magnetic resonance,is dependent on institutional availability and expertise. Previous recommendations suggest follow-up imaging at 1, 3, 6, 9 and 12 months following discharge, and annually thereafter[11-13]. This aggressive strategy underlines the observation that both hypertension and aortic expansion/dissection are common and not easily predicted in the first months following hospital discharge. Imaging should not be confined to the region of initial involvement since both dissection and aneurysm formation may progress anywhere along the entire length of the aorta. The full length of the thoracic and if indicated also thoraco-abdominal and abdominal aorta should be imaged.

Management of Complications

Endoleaks

An endoleak is a condition associated with endovascular stent-graft, defined by persisting blood flow outside the lumen of the stent-graft but within the aneurysm sac or adjacent vascular segment treated by the stent-graft. Most endoleaks persist, but some may resolve and close spontaneously. In endoleaks that persist some shall develop late aneurysm rupture. An endoleak can be classified according to the time of occurrence. When occurring during the perioperative (< 30 days) period it is defined as a "primary endoleak", and an endoleak detected later is termed a "secondary endoleak". Further categorization requires precise information on periprosthesis blood flow. Type I endoleak is indicative of a persistent perigraft channel of blood flow caused by inadequate seal at either the proximal (I-a) or distal (I-b) stent-graft end or attachment zones. Type II endoleak is attributed to retrograde flow into the aneurysmal sac via aortic sidebranches. 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 major (< 2 mm), or major (> 2 mm). 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 visualized on imaging studies but the precise source cannot be determined, the endoleak is categorized as an endoleak of undefined origin.

Endoleaks may be managed by either observation (especially type II and IV), by further endovascular procedure (e.g. balloon-inflation and/or implantation of an additional stent-graft; especially type I and III) or by conversion to open surgery (if further endovascular maneuvers fail to achieve complete exclusion of the aneurysm from circulation resulting in increase of TAA-diameters).

Aneurysm Evolution After Stent-Graft Treatment

The thoracic aorta has unique anatomical features with a distinctive biomechanical and hemodynamic environment, explaining some of the rare late complications specific to devices used in this location. The descending thoracic aorta, unlike the abdominal aorta, is relative mobile in the chest and is subject to a complex and vigorous 3-dimensional motion. Fixation points are the aortic root, sites of origin of major branches with a long mobile aortic segment extending from the LSA to the celiac artery. Thus, the thoracic aorta elongates, angulates and enlarges between these points and aneurysms may develop. 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. Development of aneurysms of the dissected aorta after stent-graft treatment is an infrequent event. False lumen thrombosis secondary to thoracic endografting is crucial to prevent late aortic expansion; therefore, close clinical and imaging follow-up are essential to monitor anatomical changes over time. A still unresolved problem, even after successful thoracic stent-graft placement, is the fate of the distal aortic segment. In the presence of large re-entry points, the thoraco-abdominal 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 have been identified. Other complications include perforations of the fragile aortic intima by the ends of the metallic stent, especially in the early phase of the acute aortic dissection and injuries caused by stiff guidewires and devices manipulation potentially setting the stage for aneurysmal evolution. The risk of these complications seems to be reduced by introducing the more flexible and soft tip delivery systems with the aid of minimally traumatic thoracic guide-wires specifically designed for these interventions. In addition, the duration of an endovascular procedure may also relate to complications such as stroke or bleeding; both of which may of course be reduced by experienced operators requiring less than 30 minutes 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 lumen and false lumen. Treatment of these communications at the level of distal thoracic and abdominal aorta shall potentially obliterate the false lumen and reduce the aortic diameter, yet in practice the closure is difficult to achieve because of proximity or involvement of the visceral branches. Another mechanism of late complication of graft treatment is related to distension of the aorta beyond the portion covered by the stent-graft due to the mechanical weakness of the dissected aortic walls. Prevention of these complications can be partially achieved during the primary procedure by ensuring adequate landing zones proximally and distally to the stent-graft, and closure of large fenestrations along the length of the false lumen. To address the eventual problem of distal false lumen persisting perfusion with aneurismal increase and incomplete true lumen remodeling, use of provisional bare stent as distal extension to a stent-graft has been suggested to facilitate aortic remodeling and completely repair of the distal dissected segments without compromising important side-branches (PETTICOAT technique)[25].

Stent-Graft Infection

Prosthetic graft infection represents a rare complication that is difficult to diagnose. Stent-graft infection is suggested by combining imaging, haematological and clinical parameters. The presence of air in the aneurysm sac should raise the suggestion of stent infection as would excessive soft tissue accumulation and progressive enlargement of the aneurysm sac. Similarly, in the presence of suspicious imaging, the presence of raised markers of systemic inflammation may be informative as would the onset of clinical symptoms of an infective process. In most cases PET radio-nuclide studies may be helpful. Treatment of thoracic endograft infection will depend on the diagnostic certainty, the pathogenesis of the organism, the extent of infection, the presenting features and the medical co-morbidities of a given patient. As with the treatment of all prosthetic graft infections a spectrum of management strategies includes conservative treatment with targeted antibiotics delivered peripherally or alternatively by direct puncture and application into the peri-graft space, insertion of another stent-graft inside the potentially infected graft or excision of the infected stent-graft with debridement of the surrounding tissue and either in situ or extra-anatomic vascular reconstruction. Irrespective of the method, the treatment of aortic graft infection remains difficult and problematic. In absence of management algorithms that can define treatment in particular conditions based on patient presentation and degree of infection individual decisions in each patient weighing the risk of the available options are required. In general, a decision must be made as to whether treatment is to be potentially curative or palliative. In cases of potentially curative treatment an aggressive approach with open surgery associated with a relatively high mortality may be justified. In cases of palliation, placement of an endovascular graft (graft-in-graft approach) to prevent life threatening bleeding or fistulation may be considered. Patients with complex graft disease should always be treated in experienced centers having all required treatment modalities at their disposal.

Retrograde type A Dissection Following Stent-Graft Placement

Few cases of proximal aortic dissection after placement of an endograft are reported; the incidence of this complication is presumably in the range of 1-2% according to previous reports and occurs usually shortly after the intervention[30,31]. Chest pain or ischemic symptoms of heart, or brain should immediately alert the suspicion that a type B dissection has changed to a type A dissection with a high morbidity and mortality. Emergency CTA and subsequent surgery may save the patient. There are several reasons why type A dissections may occur after stent-graft placement in the descending thoracic aorta:

- Initial misinterpretation of a type A dissection as a type B dissection.
- Spreading of the dissection into the ascending aorta or aortic arch due to not placing the stent-graft close enough to the LSA, or maneuvering in the false lumen.
- Under- or oversizing of the stent-graft with a leak or additional injury to the aortic wall.
- Malapposition of the stent-graft to the aortic wall at the proximal landing zone with subsequent proximal collapse of the stent-graft.
- Ballooning of the proximal end of the stent-graft resulting in injury of the diseased aortic wall with extension of the dissection in the aortic arch and ascending aorta.
- Development of a type A dissection independently of the treated type B dissection.

In these cases it is critically important to advance the guide-wire, catheter and endograft in the true lumen only. If the position of the guidewire in the true or false lumen remains uncertain, angiography and transesophageal echocardiography are in experienced hands instrumental to confirm correct positioning. The true lumen can be differentiated from the false lumen by angiography and transesophageal echocardiography since the false aortic lumen is usually larger than the true lumen, and flow at the entry is directed into the false lumen. This explains the damping of the pulse amplitude and the difficult palpation of the pulse in many of these patients. Precise measurement of the proximal aortic dimensions is essential for the selection of the stent-graft size and a successful sealing of the aortic tears. In type B dissection the stent-graft is normally placed with its proximal end directly at the origin of the LSA. Proximal landing of a stent-graft is usually close to the LSA, even when the tear is in the mid-portion; otherwise, the dissection membrane may develop a new tear between LSA and proximal end of the stent-graft. In successful cases angiography shows a good result with complete sealing of the leak and no opacification of the false lumen anymore. Occasionally, however, a stiff endo-graft chafes on the fragile dissection membrane and a new endoleak may develop, possibly requiring additional stent-grafting or eventually conversion to open surgery.

Paraplegia After Stent-Graft Placement

The initial concern after stent-graft placement was expressed in terms of risks of spinal cord ischemia due to the frequent need to cover multiple intercostal arteries and even the artery of Adamkiewitz, usually the only one prominent intersegmental branch from the aorta at the lower thoracic or upper lumbar level. Indeed, 3% to 12% of the stent-graft patients treated for aneurysms, dissections, ulcers, intramural hematoma or aortic traumatic transsection are still at risk of spinal cord ischemia. Besides ischemia of the spinal cord due to occlusion of important radicular arteries that originate from vertebral, intercostals and lumbar arteries, ischemia-reperfusion and hypotension may play a causal role in the development of paraplegia. Thus, the widely accepted impression that the coverage of the segmental artery or the orifices of the intercostal/interlumbar arteries by the stent-graft represents the only and possibly the most critical cause of spinal ischemia should be corrected. Prevention of paraplegia in stent-graft patients should include:

- Screening for high-risk patients
  - Age > 75
  - Anticipated endograft coverage T9-T12 (loacation of anterior spinal artery)
  - Coverage of long segment (> 20 cm)
  - Compromised collateral pathways (e.g. LIMA as coronary artery bypass, infrarenal surgical aortic repair)
  - Long extent of atherosclerotic lesions
- Early detection of spinal cord ischemia
  - Somatosensory evoked potentials
  - Motor evoked potentials
- Cerebral or spinal cord perfusion monitoring and drainage (spinal tap decreases intrathecal pressure to 10-15 cm H_2O to generate space for collateral arteries to fill and perfuse better)
- Prevention of perioperative hypotension

If spinal tap is introduced without any delay and arterial perfusion pressure is elevated to 140 mmHg systolic pressure by pharmacological means or volume resplenishment, symptoms of paraplegia or paraparesis are frequently reversible.

Stroke After Stent-Graft Placement

Reports of strokes after stent-graft placement vary from 0 to 18% and the complication is often devastating. It is well recognized that instrumentation in the arch produces showers of particles embolizing to the brain. In endovascular technique instrumentation in the aortic arch includes not only the use of guidewires or catheters, but also bulky delivery systems that carry these stent-grafts. In addition balloon dilation of stent-grafts is often needed which adds risks of dislodgement of particles from the aortic arch. It is known that even flushing correctly the delivery system of stent-graft cannot always eliminate all the air contained in the crimped stent-graft. Moreover, bubbles are released with the deployment of the stent-graft. A basic prerequisite in patients in whom the subclavian artery closure has been considered is the assurance that the contralateral vertebral artery is open and forming the basilar trunk without anomalies of the intra-cranial branches. This means that the basic assessment that is necessary, before deciding a procedure that will cover the LSA involves the complete definition of the left vertebral artery including the depiction of the origin, patency and size, size and condition of the right vertebral artery and the constitution of the basilar artery and its branches as. In patients with compromised cerebral circulation the four vessel 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 in severe steal phenomenon or even ipsilateral posterior stroke. In those cases a transposition of the LSA prior to the intervention is required.

Conclusions

The emergence of endovascular stent-grafting as an alternative therapy to open surgical repair of thoracic aortic pathologies is an exciting advancement. Although it is apparent that high-risk patients will benefit from this technology, the exact role of stent-grafting remains to be defined as we accumulate long-term data and as devices and technology evolve. 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 of both approaches. However, while high risk for surgery is defined by clinical parameters, co-morbidities and physiological reserve, contraindications for endovascular stent-graft treatment are defined mostly by anatomical criteria such as 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

The Authors declare that there are no conflicts of interest.

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, Myrmel 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): S1-41.

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): S167-170.

22) Nienaber CA, Kische S, Rehders TC, Schneider H, Chatterjee T, Bünger 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, Bünger 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. 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.