

# Guidelines for surgery of aortic diseases from Brazilian Society of Cardiovascular Surgery

*Diretrizes para o tratamento cirúrgico das doenças da aorta da Sociedade Brasileira de Cirurgia Cardiovascular*

Editores: Luciano Cabral ALBUQUERQUE<sup>1</sup>, Domingo Marcolino BRAILE<sup>2</sup>, José Honório PALMA<sup>3</sup>, Eduardo Keller SAADI<sup>4</sup>. Revisores: Walter José GOMES<sup>5</sup>, Enio BUFFOLO<sup>6</sup>

RBCCV 44205-881

**Descriptors:** Dissecting aneurysm, surgery. Aorta, surgery. Thoracic aorta, surgery. Aortic aneurysm, surgery. Aortic rupture, surgery. Practice guideline [publication type].

**Descritores:** Aneurisma dissecante, cirurgia. Aorta, cirurgia. Aorta torácica, cirurgia. Aneurisma aórtico, cirurgia. Ruptura aórtica, cirurgia. Guia de prática médica [tipo de publicação].

## DESCRIPTION OF METHODS TO COLLECT EVIDENCES

Periodic meetings were carried out to elaborate the text, including the relevant bibliographic citations provided by the official committee members designated by the Brazilian Society of Cardiovascular Surgery Board of Directors. The members, divided into working groups, supplemented their contribution, corrections and recommendations accepted based on a consensus, what allowed editing the preliminary text from a basic reference text. In different moments, we sought after cross-references and more relevant related papers, to edit the text. The Cardiovascular Surgery Board of Directors such as meta-analyses, systematic reviews, and classic multicentric studies. It was attempted to suggest relevant studies written by Brazilian authors, especially those published by the Brazilian Journal of Cardiovascular Surgery and the Brazilian Archives of Cardiology, official publication organs of the Brazilian cardiology. This guideline is the original version of the abovementioned consensus. It was adapted to fit the format suggested by

the Technical Committee of the Project Guidelines of the Brazilian Medical Association and the Federal Council of Medicine.

## DEGREE OF RECOMMENDATION AND STRENGTH OF THE EVIDENCE

- A: Large randomized clinical trails and meta-analyses.
- B: Well-designed observational and clinical trials.
- C: Case Reports and Clinical cases series.
- D: Publications based on consensus and expert opinions.

## OBJECTIVES

- To provide basic information to recognize the different diseases of abdominal and thoracic aorta;
- To allow the early identification of the entities involving the aorta that represent surgical emergencies;
- To describe the surgical and endovascular treatment options to the diverse segments of abdominal and thoracic aorta;

1. Full member of the Brazilian Society of Cardiovascular Surgery. Master in Cardiology by the Federal University of Rio Grande do Sul. Cardiovascular surgeon of Hospital São Lucas- PUCRS, Porto Alegre-RS  
 2. Adjunct Director of the Postgraduation Course of the Medicine School in São José do Rio Preto (Famerp) Professor Livre Docente of Famerp and Unicamp - Editor of BJCVS  
 3. Professor Livre Docente of Cardiovascular Surgery of Unifesp - President of the Endovascular Section of the BSCVS  
 4. PhD. Professor of Cardiovascular Surgery of the Medicine School/ UFRGS. President of the Commission in favor of the Profession in BSCVS

5. Adjunct Professor Livre Docente of Cardiovascular Surgery in EPM, Unifesp. Editor of the Site of the BSCVS  
 6. Full Professor and Head of the Cardiovascular Surgery Department of Unifesp.

Correspondence address:  
 Luciano Cabral Albuquerque  
 Centro Clínico do Hospital da PUCRS. Av. Ipiranga 6690, conj. 615. Porto Alegre, RS. CEP 90610-000. Phone/Fax: (51)-3336-8190. E-mail: alb.23@terra.com.br

To emphasize the long-term requirements to control the atherosclerosis risk factors and the periodic follow-up through imaging methods in all patients surgically managed due to aortic diseases

## INTRODUCTION

However the advances in diagnostic examinations, in monitoring and hemodynamic support methods and in the surgical repair techniques that have occurred over the last few years, aorta diseases are still a significant cause of cardiovascular mortality and morbidity and a continuous challenge for cardiologists and surgeons.

While in cases of acute dissections (AD) the consensus regarding the requirement of immediate surgery may have been established, at least, three decades ago, the correct moment to intervene in degenerative diseases, which determine the asymptomatic dilation of the ascending aorta as well as the management of the aortic root and the aortic valve, in the ascending aorta diseases is still under discussion and has been the subject of a number of recent publications.

When the transverse arch is involved, the controversies are focused on the choice of the best method of cerebral protection among the hypothermic cardiocirculatory arrest, venous retrograde perfusion, selective cerebral perfusion, and more recently the antegrade perfusion through subclavian-axillary axis, and in the definition of when and to what extent the arch must be included in reconstruction.

In the treatment of the descending aorta and thoracoabdominal diseases, spinal cord ischemia is still the main concern, with variable but significant involvement, in the different approaches described. This was one motivation for the development of self-expanding stents. Additionally, in cases of degenerative aneurysms, in the light of the results from recent studies on the prediction of dissection or rupture rates, the decision of intervention based exclusively on the diameter is being replaced by more individualized decisions, including factors such as age, comorbidities and outcomes of the surgical team, among others.

Concerning abdominal aorta, the classic indication for surgical repair of asymptomatic infra-renal aneurysms, larger than 5 cm in diameter, may be modified by the results of recent European and North-American trials. Similarly, the experience with endoluminal exclusion by using recovered prostheses – stents, described in many studies comparing this technique to the open surgery has been the subject of discussions and it is far away from a consensus.

## ACUTE AORTIC DISSECTIONS

### Acute Type-A Dissections

The first endpoint involving dissections of the

ascending aortic is that surgical intervention must be immediate aiming mainly at avoiding rupture and death by cardiac tamponade. Additionally, the second endpoint aims at repairing aortic regurgitation when present; avoiding myocardial ischemia; excluding the site of tunica intima laceration, and re-directing the flow through the true lumen towards the supra-aortic branches and to the descending aorta [1-7] (D).

In the choice of the surgical reconstruction technique, three questions must be taken into account: 1. – Which are the diameter and the condition of the aortic root and sinus (sinus of Valsalva) at the moment of the intervention, and if it is possible, prior to the acute event?; 2. – Which is the condition of the aortic valve?; 3. – Is there any extent or existence of injury to the tunica intima of the transverse arch? (aortic arch)

If the diameters of the ascending aorta and of the aorta root are normal and if there is no misalignment of the aortic valve commissural plane nor there is any distortion of the openings of coronary sinuses (coronary ostia), the repair usually involves the use of a straight Dacron graft anastomosed near to the aortic sinotubular junction. If there is loss of support of one or more commissures in the aortic valve, they should be resuspended during the repair of the commissural angles before the insertion of the straight graft. However, if it is impossible to repair the aortic insufficiency by valvoplasty or if the aortic valve is bicuspid, the replacement must be performed using prosthesis before the supra-commissural implant of the graft [6-8], although some authors report success with the repair of bicuspid valves [9].

The conventional approach to surgical repair of type-A dissections is median sternotomy with the CPB circuit established from cannulation of the right atrium (RA) and the femoral artery, generally utilizing moderate hypothermia between 28 °C to 32 °C. Recently, the perfusion route through the axillary artery has been proposed by many centers with potential advantages compared to the femoral artery.

When aortic dissection occurs in a previously dilated ascending aorta and/or aortic root, or in patients with annulus-aortic ectasia associated or not with Marfan's syndrome, the surgical repair necessarily requires the replacement of the aortic valve, the portion of sinotubular junction and the aortic sinus (sinus of Valsalva), and the re-implantation of the openings of coronary sinuses (coronary ostia) using a valvar prosthesis of composite graft and a Dacron mesh, known as a valved tube [10,11] (B).

In the technique originally described by Bentall & De Bono [12], the openings of coronary sinuses (coronary ostia) are included in the ascending portion of the Dacron tube by means of direct anastomoses performed after valve implantation (B). The significant incidence of late

pseudoaneurysms associated with the difficulty of direct reimplantation of the coronary arteries, in cases in which the dilation of the aortic sinus (sinus of Valsalva) are not so important, supports a modification proposed by Kouchoukos et al. [13] in which the openings of coronary sinuses (coronary ostia) are excised and implanted in the tube in the form of buttons (B); this technique denominated 'button Bentall' has presented low in-hospital mortality and a lower probability of late events [14-16] (B). Alternatively, the modification proposed by Cabrol et al. [17] in which a PTFE graft is connected to the ascending portion of the Dacron tube and anastomosed end-to-end to the openings of coronary sinuses (coronary ostia) may be useful in elderly patients, in surgical reoperations, or in cases in which there is a necessity of a very complex reconstruction of the other segments of the thoracic aorta (C).

Alternatively, stentless biological valves also can be used to reconstruct the aortic root [16] (B).

The utilization of autograft or homograft of pulmonary valve for the reconstruction of the aortic root, although recommended by some authors [18-21] (B), has shown a high incidence of reoperations due to delayed degeneration [22] (B) and must be used in specific cases, especially in elderly patients with or without associated endocarditis [23] (D).

Recently, techniques of preservation and remodeling of the aortic valve and the aortic root have been suggested by authors including David & Feindel [24] and Sarsam & Yacoub [11]. In these techniques, the openings of coronary sinuses (coronary ostia) are excised and the portion of sinotubular junction is trimmed between 3 and 5 mm above the annulus, maintaining the cusps and the insertion line of the commissural angles intact; a line of individual sutures using Teflon pledgets strength the remaining annulus-aortic junction, which is anastomosed to the Dacron graft; and finally, the coronary buttons are re-implanted similarly to the Bentall button technique (B). Because the remodeling techniques are more complex and generally require more time than others which use valved tubes, they must be employed by very experienced surgeons in elective situations, leaving procedures that use composite grafts as is recommended in annulus-aortic ectasia [23] (D). When the occasionally delamination of the aortic wall affects the openings of coronary sinuses (coronary ostia) preventing satisfactory reconstruction, the alternative is limited to the use of arterial graft(s) or saphenous vein graft(s) [23] (D).

In type-A dissections, for a better hemostasis of the proximal and distal sutures, Teflon sheets and tissue adhesives, such as the gelatin-resorcine-formol biological glue (GRF) or both can be used; traditionally, the leaflets separated by a false lumen are joined by a suture line which includes a Teflon sheet inserted between the leaflets or

inserted as an external reinforcement. Even though there is no consensus concerning the benefit of the biological adhesives and this product has not yet been approved to be used in the USA, the GRF is recommended by many authors, as an adjuvant technique or in isolation [25] (C). With the same purpose and now with the recent approval for clinical use in the USA the Cryolife Bioglue adhesive can be used.

Despite of the excellent surgical results of some groups that have reported a in-hospital mortality between 6% and 12% [3,4,6,7,16,26-28], the International Registry of Acute Aortic Dissection [29], in a retrospective survey of 464 cases of acute aortic dissections attended in 12 reference centers in the USA between 1996 and 1998 showed a 26%-surgical mortality in acute type-A dissections, a level closer to the average in our setting (B).

When the aortic dissection involves the aortic arch (transverse arch), the discussion regarding the different approaches proposed usually is focused on: 1) when to include the aortic arch in the surgical repair?; 2) how and to what extent to reconstruct it?; and 3) what is the best method of cerebral protection to be employed?

It is generally accepted that when the tunica intima injury does not affect the into of the aortic arch (transverse arch), it can be repaired through an open approach (without clamping), holding the leaflets of the aortic wall together, anastomosing the ascending aorta graft, and redirecting the flow to the true lumen, a technique described as hemi-arch repair [30-36] (D). However, around 10% to 20% of the aortic dissections, the tunica intima injury occur into the transverse portion of the aortic arch, making the complete reconstruction indispensable including the reimplantation of the supra-aortic trunks, as a block or individually. This decision making is frequently difficult, as the morbidity and mortality rates of total repair of the arch seem to be substantially higher than hemi-arch repair because of the necessary longer period of time of the cardiocirculatory arrest (CCA) and the more intense hypothermia level, which means a significant incidence of permanent brain injury and bleeding due to dyscrasia. In a historical series of the Texas Heart Institute between 1976 and 1982, 60 cases of aortic dissection involving the arch using hypothermic CCA, Livesay et al. [37] reported re-intervention rates for bleeding and postoperative stroke of 19% and 10% respectively (C).

In the recent experience of the Mount Sinai Medical Center Group, total replacement of the aortic arch (transverse arch) was employed in 11 out of 19 cases of type-A aortic dissection, with a mean CCA time under deep hypothermia of 56 minutes, with an incidence of excessive bleeding (which have justified the use of peri-aortic shunts to the right atrium) in 40% of the patients [38] (C).

Borst et al. [34] using hemi-arch repair or total arch reconstruction techniques in 92 patients with aortic dissection, have demonstrated that the mean CCA time doubled and the mortality tripled using the second procedure (17 minutes versus 34 minutes, and 12% versus 36%, respectively). Similarly, Grawford et al. [33] studying 82 patients with type-A aortic dissections have reported a significantly higher mortality rate in total replacement of the arch (31%) than in intervention restricted to the ascending aorta or the partial repair of the aortic arch (17%) (B).

These findings, as well as other reports [39,40], seem to justify a careful approach of the aortic arch with preference for the hemi-arch repair technique with an open anastomosis, and the adjuvant use of biological adhesive except for cases with much intra-arch destruction or discontinuity of the descending portion of the thoracic aorta (B); in these situations the best alternative appears to be to implant a Dacron tubular graft in the descending aorta, with the distal end without anastomosis ("elephant's trunk") and the reimplantation of the supra-aortic branches [41,42] (C). In a possible outcome, if it is necessary to repair the descending aorta, the free end of the graft can be directly anastomosed to the aortic wall or extended by means of an additional tube to a more distal segments using left thoracotomy. As a more simplified alternative it may be valid to implant the covered stent to treat the descending aorta by inserting it under direct visualization into the aortic arch under the CCA [43] (C).

Among the methods described for cerebral protection during interventions of the aortic arch (transverse arch), the most used have been the CCA under deep hypothermia; the hypothermic CCA with retrograde cerebral perfusion; and the hypothermic CCA with antegrade cerebral perfusion through the carotid artery or more recently, through the subclavian-axillary axis.

Yet in the 1970s and 1980s, hypothermic CCA was confirmed as the method of choice of cerebral protection as it is easy to inspect the arch as for tunica intima injury, to allow repair completely along its length, including the proximal portion of the descending aorta and to avoid aorta clamping near the arch from creating sites of dissection near the distal anastomosis [30,37,38,44] (D). The technique consists of establishing CPB by cannulization of the femoral artery, on slow cooling up to 18 or 20° C (temperature at which the metabolic rate is less than 18% of the normal), in the use of iced bags for topical cooling of the head and neck, and occasionally in the administration of CO<sub>2</sub> in the surgical area to reduce the risk of air embolism. Rewarming must also be slow, somewhere close to 1° C each 3 minutes to minimize hemolysis and the deleterious effects to coagulation factors. Several groups have shown that hypothermic CCA is effective and safe as for the

neurological morbidity, if the period of CCA is no more than 45 minutes with a stroke rate between 3% and 12% [34,37,45] (B). Even though, authors such as Ergin et al. [46] report favorable outcomes with up to 60 minutes of CCA and suggest that neurologic injury is more related to the advanced age and preexistent cerebral atherosclerotic disease than to the CCA itself (C).

However, sound data from Svensson et al. [47], in an analysis of 656 cases of hypothermic CCA show a significant increase of the postoperative stroke rate when the CCA time exceeds 40 minutes, and an early mortality with more than 60 minutes (B).

Recently, the control of the cerebral metabolic rate has been proposed by measuring the oxygen saturation in the jugular bulb as a parameter to start hypothermic CCA, but its applicability and safety still need to be proved [48] (C).

The cerebral retrograde perfusion (CRP) through the superior vena cava during CCA, first introduced by Ueda et al. [49], has advantages of offering a sustained brain cooling, eliminating metabolites resulting from ischemia, removing the air or even debris with emboligenic potential, as well as allow nutritional substrate infusion during CCA (B). However, potential limitations such as nonhomogeneous intracerebral distribution, presence of valves in the jugular system [50] (C), cerebral edema [51] (D), insufficient cerebral blood flow [52] (D), and bleeding in the surgical area has made its use controvertible.

Coselli et al. [53] in a recent study of 479 cases of aortic arch reconstructions, in which hypothermic CCA with CRP was performed in 290 patients (60%) and isolated hypothermic CCA in 189 (40%) demonstrated a significantly lower in-hospital mortality in the group submitted to CRP (3.4% versus 6.3%). As to the efficacy of the cerebral protection, favorable findings of CRP were also described by Safi et al., in a retrospective study of 161 patients submitted to aortic arch surgery: protection effect against stroke was three times higher when CRP was employed (3% versus 9%), and this benefit was more obvious in patients over the age of 70. However, there was no significant difference in the stroke or death rates among the groups when the CCA time was less than 60 minutes. It may be presumed that benefits of CRP were indeed involved only using a profound hypothermia (B).

In a multicentric Japanese study, coordinated by Ueda et al. [48], the results of 249 cases of aortic arch replacement utilizing CRP, between 1994 and 1996, were analyzed. In this study the mean CRP time was 46 minutes, the postoperative stroke rate was 4%, and the in-hospital mortality was 13%. A multivariable analysis identified prolonged CPB time, the advanced age, and the urgency of the surgery as the most significant risk factors related to strokes or death (B).

Another cerebral protection technique includes anterograde perfusion during CRP, when cooled blood infusion via the supra-aortic arteries (cerebroplegia) or maintenance of perfusion by the axillary artery with clamping of the brachiocephalic trunk can be used. In the technique originally described by Guilmet et al. [55], the temperature of the patient is kept at 25° C during CRP, part of the blood of the oxygenator is cooled between 6 and 12° C and infused directly into the carotid arteries at a rate of 200 to 250 mL/min, at a pressure of 60 to 70 mmHg (C). Between 1984 and 1998 the authors used CCA and cerebroplegia in 171 cases of aortic arch replacement, 42 of which due to type-A dissections. In these cases the mean CPB time was 121 minutes, the mean selective cerebral perfusion time was 60 minutes, the in-hospital mortality was 17%, and the occurrence of postoperative neurological damage was 13% [56] (B). In Brazil, Souza et al., reported an experiment of nine cases of aortic arch replacement using carotid arteries selective perfusion without any event of postoperative neurological deficit (C). Although other studies have also suggested the superiority of anterograde perfusion over CRP and hypothermic CCA both clinically [58] (C) and experimentally [52] (D) complications, such as bleeding at the site of cannulation and intra-cerebral bleeding have restricted its use to only a few centers.

Cannulation of the axillary artery and the maintenance of the flow through one of the carotid arteries during CCA, as described by Sabik et al. [59], has the following advantages: to avoid the handling of femoral artery, which is often affected by dissections; to maintain the flow in the anterograde direction through the true lumen (important when the elephant trunk is used); and have no need of deep hypothermia, which minimizes dyscrasic complications (C). Additionally, problems of malperfusion of important organs, and new reentry points are created by a pressure increase in the false lumen are eliminated, as was demonstrated by Van Arsdell et al. [60]. In this technique, the axillary artery is dissected near the deltopectoral groove, preferable to use the right side when possible, CPB is maintained through the D axillary-atrial artery and the temperature is maintained at approximately 24° C; once CCA is established, the brachiocephalic (arterial) trunk (innominate artery) is clamped at its root, and the flow is reduced from 150 to 300 mL/min/m<sup>2</sup> to reconstruct the arch.

According to a first positive experiment addressed to small series [61, 62], the anterograde perfusion through subclavian-axillary axis has been tested in more solid studies. Svensson et al., comparing in-hospital outcomes of 299 patients submitted to aortic arch reconstruction with right axillary cannulation, to 471 cases of aortic perfusion and 375 cases of femoral cannulation have observed significantly lower death and cerebral infarct rates in the

first group (B). Similar result was observed by other authors in studies case-control comparing both sites of axillary and femoral cannulation [64-67] (B). Despite these evidences, other authors point out the risks of severe complications due to axillary cannulation, such as thrombosis and brachial plexus injury [68] (C), but its efficacy still need to be proved [69] (B).

Recommendations for surgical treatment of acute type-A dissections are shown in Table 1.

Table 1. Recommendations for the treatment of acute type-A dissections

Recommendations	Recommendation Level
1. Immediate surgery to avoid rupture / tamponing / death	A
2. Straight graft in the ascending aorta, if aortic root and aortic valve are normal	B
3. Straight graft in the ascending aorta and aortic valvar resuspension, if aortic root is normal and the valve is insufficient due to loss of support	B
4. Valved tube, if ascending aorta is dilated or annulus/aorta suffers ectasia and aortic valve is insufficient	B
5. Auto or homograft, if situation number 4 is associated with endocarditis	C
6. Aortic valvar resuspension and remodeling of the aortic root in Marfan's syndrome	C
7. Partial repair of the aortic arch (hemi-arch repair), if dissection involves the arch, but there is no destruction or lesion to the tunica intima	D
8. Total reconstruction of the arch if it presents with tunica intima destruction or injury	D
9. In case of aortic arch intervention, open reconstruction using cerebral protection (hypothermic CCA - cerebral retrograde perfusion - cerebroplegia - axillary perfusion) must be performed	A
10. Saphenous vein graft(s), if the openings of coronary sinuses (coronary ostia) are affected with delamination and the re-implantation is not possible	D

### Acute Type-B Dissections

There is still general agreement that surgical treatment of acute type-B dissections depends on the presence of complications, such as signs of aortic rupture (hemothorax, rapid expansion of the aortic diameter, mediastinal widening), pseudoaneurysm formation, severe visceral ischemia or ischemia of the extremities, or progression of the dissection during drug therapy characterized by persistent or recurrent pain. Also cases of previously aneurysmatic aortas which are submitted to acute dissection must be considered for urgent surgery.

However, groups such as the one from Stanford University have demonstrated in a cohort of 136 patients with acute type-B dissections, that a more aggressive approach might be employed with young, low-operative risk patients using early surgery without increasing in-hospital mortality (11%), when compared to drug therapy, thereby avoiding chronic aneurysmatic degeneration of the descending aorta [7] (C). Even though these results were extremely favorable, they were not considered reproducible by other centers. Indeed, the International Registry of Acute Aortic Dissection [70], which is considered to be an expression of the real world, reveals that in cases of type-B dissections treated conservatively, the mortality over 30 days was only 10%, while in the patients surgically treated the mortality rate was 31% and the incidence of paraplegia was 18%. After a three-year follow-up, there has been no difference statically significant in the mortality rate between the groups treated with drug, endovascular or surgical therapies (B).

The surgical approach in descending aortic dissections normally consists of the replacement of the affected portion using a tubular Dacron graft by a left thoracotomy through the 4th or 7th intercostal spaces, which can be performed with simple proximal clamping [71] with atrial-femoral CPB either with or without hypothermia [72, 73] (C), with femoro-femoral CPB with cannulation, LA-femoral bypass without using oxygenator [74] (B), temporary arterial bypasses or even using CCA under deep hypothermia for a proximal open anastomosis [75] (B). Depending on the chosen technique, the dose of heparin and the effects of the hypothermia level in the cascade of coagulation could determinate variable effects on the hemostasis or in the production of dyscrasia.

Another reported technique to repair type-B dissections is the “elephant trunk” method, initially proposed by Borst et al. [42] for staged surgical treatment of complex aneurysms. After median sternotomy and conventional CPB, hypothermic, CCA is induced and the aortic arch (transverse arch) is opened lengthways; a Dacron tubular graft is introduced into the proximal portion of the descending aorta and anastomosed only in the proximal segment after the left subclavian artery, with the distal end continuing free in the thoracic aorta (C).

Subsequently, Palma et al. [76] also used this technique in all cases of acute type-B dissections, in spite of the existence of complications; the authors performed 70 consecutive insertions of the “elephant trunk” graft, between 1988 and 1995 by median sternotomy with a short period of hypothermic CCA (mean time of 31 minutes) and presented a in-hospital mortality rate of 20% and an overall survival over 5 years of 62% (C).

For the management of renal and mesenteric ischemia or ischemia of the extremities, specifically when the origin of these vessels is in the false lumen [77], it is generally accepted that fenestration by catheter is the method of choice (C), which is justified by the high mortality that follows type-B aortic dissection surgery in these situations: from 50 to 70% in the case of renal ischemia [78,79], 89% in mesenteric ischemia [80, 82] (B), and up to 87% in peripheral ischemia [81] (C).

Since the first successes using fenestration by balloon catheter to communicate false and true lumens in a case of type-B aortic dissections with mesenteric ischemia, which were described by Williams et al. [76] in 1990 (D), several studies have tried to validate this approach [84-90] (D); Additionally, the advent of aortic stents originally used to exclude infra-renal abdominal aneurysms, provided a new alternative in the management of complicated descending aortic dissections.

In general, the branches affected by static obstructions at their root are better treated by the intraluminal implantation of stents, while vessels obstructed by dynamic displacement of the blood column through a false lumen may be reperfused by fenestration using a balloon catheter, with or without stent implantation in the true lumen. In other situations, stents can be used in the true lumen to maintain the original position of some branches [89] (D), or to maintain the fenestration open [91] (C). Another indication for endovascular fenestration may be to create a reentry in cases in which a “dead-end” false lumen significantly compresses the true lumen, even though this maneuver can increase the risk of peripheral embolization [91] (D), [92] (C), or the long-term aneurysmatic dilations maintaining the false lumen patent [93] (C).

The technical objective of fenestration is to create a ‘window’ between the false and true lumens, by “opening” the tunica intima layer, normally near to the artery to be saved; preferentially from the smallest lumen (generally the true lumen) towards the biggest lumen (generally the false lumen), using a special needle [91, 91] (D) and, if possible, using vascular ultrasound (IVUS), followed by a 12- to 15-mm balloon catheter, which is insufflated to promote enlargement of the ‘window’ [86] (D). If necessary, a stent of 10 to 14 mm can be implanted to avoid the possibility of occlusion of the fenestration, due to a ‘flap’ mechanism or thrombosis [91] (D).

The results of reperfusion after percutaneous fenestration of branches obstructed by aortic dissection, even those reported from non-controlled studies [89-91] (C) have been consistent: the flow restoration rate ranges between 90 and 100%, the mean mortality rate over 30 days is 10%, and no additional revascularization procedures were necessary over an average 12-month follow-up. Additionally, all deaths were related to the irreversibility of the ischemia at the moment of the intervention, to the progression of the dissection, or surgical complications of combined interventions.

The implantation of coated endoprosthesis with tissues in the descending aorta involves a greater diversity of devices and techniques, being especially important the margin of a healthy aorta in respect to the left subclavian artery origin, the length of dissection, the existence of reentry point(s) and the involvement of visceral trunks. Usually, the diameter of the endoprosthesis estimated by magnetic resonance, computed tomography, transesophageal echocardiography or by IVUS, ranges from 24 to 42 mm, in general being 10 to 15% larger the aorta diameter, being the length or even the use of more than one device decided according to the length of the aorta injury to be coated. Depending on the proximity of the visceral branches, one part of the prosthesis should not be coated to maintain the patency of the openings (ostia) involved. Occasionally, the carotid-subclavian bypass can be previously manufactured in cases in which the proximity of the left subclavian artery opening can determine its obstruction. The right femoral artery is the commonest insertion site with the positioning of the endoprosthesis being guided by angiography, IVUS or both methods. Arterial pressure monitoring is important during implantation, normally tending to increase during the expansion of the devices and to decrease significantly after its placement. It is recommended to keep the mean arterial pressure between 50 and 60 mmHg, which requires the application of sodium nitroprusside [23] (D).

Preliminary results indicate that the use of stents in complicated type-B dissections is more reliable and causes a smaller risk implanted percutaneously when compared to the implantation by open surgery [95] (C), and that the incidence of paraplegia may be significant with the use of prostheses longer than 15 cm [95,96] (D).

Thus, when it is necessary to treat longer segments of the aorta as in case(s) of distal reentry(ies), it is preferable to use multiple shorter prostheses, which has the additional advantage of better modeling in cases of aortae that present with much curvature. It may also be useful to perform hybrid surgeries, such as the carotid-subclavian bypass before implantation in many cases where the treated portion is very close to the emergence of the left subclavian artery, a maneuver, which in the experience of Grabenwöger et al.

[97], is employed in nearly 40% of patients (C). The short-term follow-up has demonstrated that the obliteration of the tunica intima injury remains and the aortic diameter is reduced with the thrombosis of the false lumen; in cases in which periprosthetic leaks are discovered and in those in which reentries not seen in the first diagnosis are identified, the treatment, in general, may be the implantation of another stent [98] (C).

Recently, in Brazil, an expressive contribution has been proposed by the group of the Escola Paulista de Medicina, (Paulista Medical School) by using percutaneous self-expanding stents in all cases of descending aorta dissections. Palma et al. [99] submitted 70 patients with type-B dissections (60% of which were true dissections and 40% corresponded to intramural hematomas or penetrating ulcers) to implantation via femoral artery of polyester-covered endoprosthesis (Braile Biomedica®), under general anesthesia, systemic heparinization, and induced hypotension. The procedure was considered to be a success in 65 (93%) patients with the exclusion of the false lumen documented using aortography. In 5 cases (7%) conversion to surgery was necessary; there were no events of paraplegia or death. In a mean follow-up of 29 months (from 1 to 55 months), 91% of the patients were alive but the insertion of additional stents was required in 49% of the cases (C). In a more recent report on this series [100] with 120 cases of type-B dissections, the results were consistent: the in-hospital mortality was 10%, surgical conversion occurred in 6 (5%) cases, there were no cases of paraplegia (even though 2/3 of the descending aorta were excluded in 38 cases and the treated region was between T9 and T12 in 34 patients) and the long-term survival rate was 87%. The necessity of implantation of additional stents occurred in 51% of the series, in 14 patients the left subclavian artery was intentionally occluded using an endoprosthesis and in only one case, a carotid-subclavian surgical shunt was necessary (C).

The death of the patients submitted to surgery with complicated type-B dissection was 29.3% according to the international Registry of Acute Aortic Dissection (IRAD) [101], and the clinical preoperative conditions have had significant influence in the treatment outcome (B).

A recent meta-analysis [102] has shown that the endovascular treatment in type-B dissections is technically viable with success rates higher than 95% in selected cohorts. Although the technique is invasive, the complications have occurred between 14% and 18% of the patients with a much lower incidence of paraplegia. The short- and medium-term mortality rates of this new approach can be favorably compared to the conventional surgery (A). Further studies will be necessary to compare the endoprosthesis to medical treatment in uncomplicated type-B dissection.

In summary, there are not doubts about the benefits of percutaneous intervention techniques in the management of complications involving the acute descending aortic dissections when compared to conventional surgery. Even so, issues such as whether the endovascular technique will replace the initial drug therapy in uncomplicated cases, as well as the role of endoprostheses implantation in the descending aorta during ascending aorta surgeries remain unclear, in spite of the preliminary promising results.

Table 2 summarizes the recommendations and forms of interventions in acute type-B dissections.

Table 2. Recommendations for the treatment of acute type-B dissections

Recommendations	Recommendation Level
1. Clinical management with analgesics and aggressive control of the arterial pressure	A
2. Surgical treatment, if persistent/recurrent pain, signs of expansion, rupture or bad perfusion of the extremities	A
3. Implantation of covered endoprosthesis, if persistent/recurrent pain, signs of expansion, rupture or bad perfusion of the extremities and appropriated anatomy	A
4. Stent to unblock the visceral branch root, or to maintain the fenestration open	C
5. Fenestration by balloon and stent implantation, if there is severe compression of the true lumen, with or without distal reentry	C
6. Implantation of covered endoprosthesis in true lumen to avoid chronic aneurysmatic dilation of aorta	C
7. Implantation of covered endoprosthesis in true lumen to occlude the tunica intima injury and to promote thrombosis of the false lumen	C

The long-term follow-up of patients with acute aortic dissections, operated or treated using conservative methods in the acute phase demonstrates that the false lumen remains patent in about 80% of the cases [103] (D); additionally, it is known that in only 10% of patients operated by De Bakey dissections type I, the false lumen remains obliterated [104-107] (B). However, this patency should not be seen as an imminent catastrophic event and often it may be the main source of blood of some noble organs [103] (C). In the experience of the Mount Sinai Medical Center [108], the persistence of a false lumen was associated with a low incidence of aneurysmatic degeneration and no significant differences were seen in

the long-term survival rate: two out of 18 cases (11%) of patent false lumen needed interventions and the event-free survival rate over 5 years was 83% for false lumen with thrombosis, and 64% in cases of patent false lumen (B).

The patency of the false lumen after aortic dissection surgery seems to be more related to the high frequency of distal reentries than to the success of the exclusion of the initial injury site, what reinforces the necessity of a follow-up with extremely rigorous clinical control, in particular in respect to systemic arterial hypertension (SAH) and imaging method in the 1st, 3rd, 6th and 12th post-procedure months and thereafter annually [23] (D).

There are evidences that the method of choice for long-term monitoring and occasional indication of intervention should be the nuclear magnetic resonance (NMR) because it avoids exposure to radiation or to nephrotoxic contrasts used in computed tomography (CT) and is less invasive than transesophageal echocardiogram (TEE); a comparison of serial images facilitates the early detection of increases in the false lumen or the entire aorta, and can be hold responsible for the possible identification of the important visceral branches and their relationships to the dilated portion [109] (C), [110] (B), and [23] (D).

There is clear consensus that the aggressive management of SAH with negative inotropic agents is the most important independent factor in the prevention of chronic aneurysmatic degeneration or rupture; although the use of beta-blockers are the first-line therapy of choice because they decrease the dp/dt, in general, the association of more than one drug is required [23] (D). As was demonstrated by DeBakey et al. [111] in 527 patients after acute aortic dissection, aneurysms developed in 46% of the cases with uncontrolled arterial pressure, but only in 17% of normotensive patients (B).

Of all the cases of death in the experience of Stanford University [112], at least 15% occurred due to aortic rupture, while a 20-year follow-up performed by Baylor College of Medicine [111] revealed that 30% of late deaths were due to the rupture of chronic aneurysms. In fact, it is estimated that about 30% of the surgical cases operated due to acute aortic dissection will suffer aneurysmatic degeneration, more frequently on the thoracoabdominal axis (35%), followed by proximal descending aorta (16%), and ascending aorta (14%) [113] (B). Similar data were published in Europe, where a survival rate of 70 to 85% is reported in the first year and 60% over two years in cases of type-B dissections, with the best prognosis in cases with non-communicant false lumens (80% of survival over 2 years) [105] (B).

Patients with Marfan's syndrome must adhere to a stricter familiar follow-up to anticipate catastrophic events or delayed recurrences, although cases of multiple interventions are not rare [114, 115] (B); Thus, the recommendation to intervene in aneurysmatic dilation of



the thoracic aorta or in an annulus-aortic ectasia, in this specific group, follows earlier criteria that shall be commented in the following topic, although the definitive cutoff point should have been defined in yet unavailable longitudinal studies, which would determine the normal diameter of the aortic root in young patients with Marfan's syndrome [116, 117] (D). A moderate restriction of physical exercise also seems to be beneficial in children and young adults, because of the potential association of hypertension and rupture induced by exercise [118] (D).

Information about the natural history of intramural hematomas (IMH) is limited and sometimes contradictory in the literature. Generally, this can be considered a condition as potentially catastrophic as acute aortic dissections concluding that the mortality rate is significant – from 20 to 80% [119] (B) [120] (C); the evolution to true dissections occurs in 15 of 41% of the cases [121-124] (B), ruptures in 5% to 26% of the patients and evolution to spontaneous cure varies [125] (A) [126] (B). In a recent prospective survey of 360 cases of acute aortic dissections, Nienaber et al. [121] observed that 25 (12%) cases presented evident false lumen, but without identified tunica intima injury in the diagnostic examinations. The mortality in 30 days when there was involvement of the ascending aorta was 80% in clinically treated cases and zero in operated patients, similar to the type-A aortic dissection (B). Vilacosta et al. [126] analyzed the evolution of 21 cases of IMH, of which 15 were spontaneous – 8 involving the ascending aorta and 7 confined to the descending portion – and 6 were caused by trauma. In the group considered type-A, 3 (37%) cases evolved to sudden death, 3 (37%) were successfully operated, and 2 (25%) evolved to a cure. In the type-B group 1 (15%) sudden death occurred and 6 (85%) had favorable clinical courses by drug therapy, while in the trauma group there were spontaneous cures in 3 (50%) patients, and other 3 cases (50%) in this group evolved to death, but due to causes not related to ruptures. Even though the sample was small, the authors suggest that the natural history of IMH is better in trauma or cases restricted to the descending aorta than cases that involve the ascending aorta (C).

The evolution of ulcerated aortic plaque is also not completely known and controversial, in the few reports available. While some authors consider that it is as dangerous as true acute aortic dissections with a high possibility of rupture, others point out its benign clinical course that does not require immediate surgical treatment [127, 128] (C). However, the existence of sub-intimal hematomas or penetrating ulcers should be viewed as possible imminent ruptures. In a retrospective survey of 198 aortic dissection cases, the group from Yale University [129] detected the presence of 15 penetrating ulcers (8%) identified in imaging examinations prior to the acute event,

of which 13 (87%) were found in the descending aorta. The mean age and the size of the aorta in this group were significantly higher than in the overall sample and an association with infra-renal abdominal aortic aneurysms was seen in 40% of these patients, with ruptures seen in the hospital evolution in approximately 40% of the cases (C). The development of pseudoaneurysms in these ulcerated plaques is another long-term complication observed [130, 131] with the development of true dissections estimated from 10% to 20% of the cases (C).

The recommendations for the long-term follow-up of patients after suffering from acute aortic dissections are illustrated in Table 3.

Table 3. Recommendations to the follow-up of patients with aortic dissection

Recommendations	Recommendation Level
1. Continuous control of systemic arterial hypertension with beta-blockers	A
2. Imaging examinations (NMR, CT etc)	D
3. Moderate restriction of physical exercises	D

## CHRONIC DISSECTIONS – THORACIC ANEURYSMS

### Ascending Aorta - Aortic Arch

The aorta is considered pathologically dilated when its diameter exceeds the normal for a specific age and body surface area. When the diameter is more than 50% of the expected size, the segment is considered aneurysmal.

In the ascending aorta, the progressive dilation can lead to aortic valve insufficiency (even in anatomically normal valves), acute dissection or spontaneous rupture. These are events which dramatically change the natural history and the survival curve with the amount of risk being related to the diameter and to the type of structural disease of the aortic wall.

Indications for the surgical replacement of the ascending aorta in patients with Marfan's syndrome, acute dissections, intramural hematomas or endocarditis with extensive annular damage are supported by consistent evidence. However, the moment of the intervention in asymptomatic patients with degenerative dilations, as well as in the association of dilation of the aorta with the bicuspid aortic valve remain uncertain.

In Marfan's syndrome, there is an agreement that prophylactic surgical repair is indicated when the diameter reaches 5.5 cm, although it may be smaller (4.5 to 5 cm) in

patients with familiar history of dissections, ruptures or sudden death [116, 132-136] (B). In the other cases, although the presence of symptoms or the severity of aortic insufficiency can indicate intervention independent of the aortic size, it is generally accepted that a diameter of 6 cm is indicative of surgery in asymptomatic aneurysms [137] (A). Therefore, groups such as that of the Mount Sinai Medical Center based in its recent experience have proposed differentiated criteria for intervention, such as diameters of 4.3 cm in under 40-year-old adults with Marfan's syndrome ranging from 4.8 to 5 cm in cases found by chance during heart surgery for different diseases and 4.5 cm in surgeries of bicuspid aortic valves [16] (B).

The aortic root and/or descending aorta surgical reconstruction options have already been described previously, but in cases of chronic aneurysms, some aspects can contribute to the choice of the technique:

- Age and life expectation in very old or high surgical risk patients, valve replacement and the reduction in the aorta diameter by a lengthways suture with support may be a good alternative [138] (C); likewise, valve replacement followed by the implantation of a Dacron straight graft, separately, can be appropriate for patients with limited life expectation [139] (B).

- Quality of aortic wall: a weakened aorta as in acute dissections or in Marfan's syndrome generally requires removal of all friable tissue of the aortic root and the ascending portion; in these cases, the Bentall button technique can be useful [14] (B) or valve remodeling [140] (C).

- Anatomy of the aortic valvar apparatus – aortic sinuses (sinuses of Valsalva) - Sinotubular portion: when there is dilation of the apparatus, valved tube implantation or exceptionally auto/homografts. If the valve is normal but there is dilation of the sinuses or of sinotubular region, valve remodeling techniques as suggested by Tirone David can be employed [23] (D).

- Anticoagulation risks: in patients at considerable risk of bleeding, the remodeling techniques or auto/homografts must be given preference [23] (D).

- Association with aortic valvar endocarditis: although there is no conclusive evidence, the majority of groups recommend auto or homografts in this situation [23] (D).

Using this systematization, Mount Sinai Medical Center group has recently published their experience of 497 cases of annulus-aortic or ascending aorta reconstruction, specially in elective surgeries (n=310), corrected using the Bentall button technique (n= 250). The overall in-hospital mortality was 8%, and when excluding urgent surgeries 5.5%; in the 250 cases of the modified Bentall technique the hospital mortality was only 4%, and the reoperation-free survival rate was 79% over 5 years, and 62% over 8 years, results that substantiate the authors recommendation of this technique [16] (B).

In respect to aneurysms located in the aortic arch (transverse arch) the currently accepted indications for surgical resection include: 1) an absolute diameter > 6 or 7 cm, or greater than two times the expected diameter for that patient; 2) growth in the diameter greater than 7 or 10 mm per year; 3) pain or symptoms of compression; and 4) saccular aneurysms [141] (B). Patients with Marfan's syndrome generally are indicated for earlier surgery, with growth greater than 3 to 5 mm per year, or absolute diameters of 5 cm [142] (D). Technical details referring to the surgical repair methods and cerebral protection described in acute dissections involving the transverse arch are applicable here too.

### **Descending Aorta – Thoracic-abdominal**

In asymptomatic patients with descending aorta aneurysms (DAA) or thoracoabdominal aorta aneurysms (TAAA), the identification of predictors of dissection, rupture and/or death have been exhaustively studied, attempting to establish a safe cutoff point to indicate surgery, as the mortality and the operative morbidity rates are significantly higher than in ascending aortic surgery. Thus, some complicated exponential equations have been proposed to calculate an estimate of acute events in cases of DAA [143,144] (B). However, the group from Yale University has performed consistent and simplified studies [141, 145] pointing out the diameter as the most relevant independent risk factor for complications. Based on a database of 721 patients with DAA, the authors followed the evolution of 304 asymptomatic cases with a minimum diameter of 3.5 cm and clearly demonstrated that the incidence of associated complications – rupture, dissection or death – was 15.6% for aneurysms with diameters of 5 to 6 cm and only 3.9% for diameters between 4.0 and 4.9 cm (p=0.004). When the rupture were analyzed separately, the Odds ratio (OD) increased 27 times when the diameter reached 6 cm when compared to cases of 4.0 cm or less (p=0.002). Additionally, the 5-year survival curve of patients with diameters > 6 cm was only 56%, and 85% for elective operations (p=0.003) similar survival to that found in the normal population with the same mean age (B). These findings associated with operative mortality of 11% presented by the group have led the authors to choose 6 cm as the parameter for the surgical indication of DAA, a conduct considered consensual by specialists [23] (D).

The principal concern in the surgical repair of descending or thoracoabdominal aneurysms is still paraplegia, with a reported incidence at between 4 and 32% in recent works [146-157] (B). Indeed, medullar injury still is a devastating event for the patient, family and surgical staff. Although paraplegia is clearly multifactorial, it occurs due to one or

more of the following conditions: 1) duration and degree of medullar ischemia; 2) failure to reestablish the blood flow to the spinal column after repair and 3) biochemical-mediated reperfusion injury.

To reduce medullar ischemia, many techniques have been recommended, with conflicting clinical results.

When no method of distal perfusion is used during the reconstruction, the aorta should only be clamped proximally, as recommended by Crawford et al. [158], to avoid increases in the CSF pressure, sequentially repositioning the clamping cephalocaudally as the reconstruction proceed forward and the main intercostal branches, and the visceral branches are anastomosed into the prosthesis. While ischemia times of less than 30 minutes are relative safe, this approach presents an unacceptable incidence of paraparesis or paraplegia with times greater than 60 minutes (B). In contrast, when distal perfusion is maintained to optimize the nutrition of the intercostal and lumbar arteries, short periods of sequential ischemia can be performed; in this technique, CPB is established by left atrium-femoral or femur-femoral vessels which allows the maintenance of perfusion up to the T5 or T6 level during the first clamping of the proximal aorta and of the left subclavian artery. Once the proximal anastomosis is concluded, the distal clamp is usually placed just above the celiac trunk and the main intercostal vessels are included in the graft. In the next step, the clamp of the prosthesis is placed below the intercostal arteries, and the distal aorta clamp below the renal arteries, which are included as a block or individually. Subsequently, the aortic clamping is transferred to the infra-renal portion and the distal repair is concluded. With this approach of sequential ischemia there has been a significant reduction in the rates of paraplegia and renal insufficiency [71] (B).

Other methods of distal protection may include CPB with hypothermic cardiocirculatory arrest or the use of both temporary internal or external shunts. While Kouchoukos et al. [75] supported by incidences of 6.5% for paraplegia and 10% for death over 30 days recommended total CPB with CCA under deep hypothermia, Crawford et al. [159] considered this technique particularly useful in aneurysms that involve part of the arch or in arches extensively calcified in their proximal portions, presenting paraplegia and mortality rates of 9.5% and 16%, respectively (B).

Alternatively, several types of temporary shunts have been proposed justified by the necessity of smaller doses of heparin and because they do not exclude hypothermia, thereby minimizing bleeding complications. The greatest experience in the literature was published by Verdant et al. [160] who used aorto-aortic or aorto-femoral bypasses in 366 consecutive cases of DAA with a in-hospital mortality of 12% and no cases of paraplegia; however and curiously, even with these highly positive results, the authors now recommend atrio-femoral CPB due to the significant

incidence of strokes and aortic injuries at the sites of the shunts, besides the inappropriate control of both the flow and the distal perfusion pressure (B).

Drainage of cerebrospinal liquid (CSF) to reduce the intrathecal pressure constitutes another form of medullar protection, widely tested in several animal models, associated or not with infusion of neurotropic-negative drugs [161-166] (C) [167] (D).

Preliminary studies performed in the Mayo Clinic were favorable to the protective effect of CSF drainage, but the subsequent results did not demonstrate significant benefits [168] (C). Other groups have advocated CSF drainage associated with the local infusion of naloxone [149] (C) or during atriolfemoral CPB [169] (B).

Although Acher et al. [143] in a non-controlled study have suggested that naloxone associated with CRL drainage can even rule out the re-implantation of intercostal arteries (C), no benefits of this technique were evidenced in subsequent randomized trials [170] making its use empirical (A).

The effect of epidural administration of papaverine on the function of spinal cord has been referred to as having a promising effect. In experimental models of thoracic aorta clamping for up to 60 minutes at normothermia, an intrathecal injection of papaverine was highly effective in preventing paraplegia, demonstrating a significant increase in the blood supply in the anterior portion of the spinal medulla by the use of radionuclides [164] (D).

At a clinical level, the removal of a small volume of CSF followed by the intrathecal application of papaverine also showed potential benefits in some non-controlled prospective studies [171] (B).

Other pharmacological agents proposed as adjuvants in the prevention of paraplegia include corticosteroids [150] (C), mannitol [172] (C), magnesium sulphate [166] (C), prostaglandins [173] (C), allopurinol [174] (C), and flunarizine [175] (D) among others, but none has proved to be effective in the clinical practice.

A consensual recommendation discusses the necessity of re-implantation of the maximum number of main intercostal arteries, particularly in the lower 1/3 of the thorax and upper abdomen between T7 and L1 [176] (D). Several studies have already demonstrated that the blood supply to the spinal cord at the thoracic level is predominantly supplied by the intercostal arteries between T4 and T12 and by the lumbar arteries between L1 and L4 [177] (A), [178] (D), and [179] (C), and that between T7 and L1, in more than 90% of patients emerge the most important anterior radicular artery, referred to as the Adamkiewicz artery. Even though, in chronic processes such as atherosclerotic aneurysms there can be development of collateral circulation capable of maintaining the medullar functioning even when there is extensive sacrifice of

intercostal branches, or when early thrombosis of some intercostal patches is an arteriographically documented event [180] (C), all the effort must be made to protect these vessels. Thus, the preservation of the posterior wall of the terminal thoracic aorta as proposed by Williams [181] can be a valid maneuver (C).

Due to the diversity of anatomic variations in the medullar blood supply that can include from a well-developed and easily identified anterior radicular artery, or many large caliber terminal intercostal arteries, or even many small caliber arterioles, intraoperative mapping models of the segmental arteries have been tested, aiming at selecting arteries to be re-implanted reducing the aortic clamping time, such as the intraoperative infusion of hydrogen to identify intercostal branches that effectively contribute to medullar perfusion [182] (C).

Similarly, many experimental and clinical studies [183-188] have been developed trying to define the role of the motor-evoked or somatosensorial potentials in monitoring the activity of the segmental medulla and in the prevention of the paraplegia; although with very variable results and with recommendations still not consensual in respect to the efficacy as an isolated method in the prevention of medullar ischemia (C).

Crawford et al. [177] prospectively analyzed the role of the somatosensory evoked potentials (SSEPs) in a randomized trial of 198 patients submitted to DAA or thoracoabdominal aortic aneurysms (TAAA) correction. As not only did monitoring SSEP not demonstrate a protecting neurological effect, there were incidences of 13% false-negative and 67% false-positive cases in the responses of the monitored group leading the authors to believe that there is no sustenance for this technique (A).

As mentioned in the section about acute dissections, an innovative and highly promising approach in the treatment of DAA is the implantation of self-expanding stents, developed by the Stanford University group. Preliminarily, the authors used the percutaneous implantation of self-expanding stents covered by Dacron in the femoral artery in 13 cases of chronic thoracic aneurysms with a mean diameter of 6.1 cm. An immediate exclusion was observed in 12 patients and no deaths or cases of paraplegia in the hospital phase were reported, and not even over a mean follow-up period of 11.6 months [189] (C). In a clinical assay developed subsequently [190], the same authors amplified the experiment to 103 implantations, the majority of which had prohibitive surgical risk, and obtained immediate success rates of 83%. The early mortality was 9%, paraplegia was observed in 3% of the patients, and 7% developed ischemic strokes during the hospital stay. The long-term follow-up of 3.7 years has demonstrated an event-free survival rate of 53%, and the group started to recommend this technique for selected

high operative risk patients (A). However, the greatest experience with the percutaneous exclusion of DAA was published recently by Buffolo et al. [100]. Between 1996 and 2002, 191 stent implantations were performed in cases of type-B dissections (n=120), true thoracic aneurysms (n=61), deep ulcer hematomas (n=6) and trauma of the descending aorta (n=4). In all patients the authors used general anesthesia, induced hypotension (mean arterial pressure between 50 and 60 mmHg), heparinization only during the procedure (5000 IU/IV), and polyester-covered stents (Braile Biomédica ®), the sizes of which were calculated as 10% to 20% bigger than the diameter of the aorta. The success rate of the implantation, defined as the obliteration of the tunica intima laceration, or the complete exclusion of the aneurysms without extravasation was 91%, the in-hospital mortality was 10%, conversion to surgery was necessary in 6 cases (3%), and no cases of paraplegia were observed (C).

In a multicentric clinical assay [191] the early outcomes of the endovascular treatment were better than the surgical outcomes: mortality rate 2.1% (endovascular) vs 11.7 (conventional surgery) –  $p < 0.001$ , even in low risk patients (A). Nevertheless, in the same study, in a 2-year follow-up there were a considerable incidence of leaks (endoleaks) and re-interventions in the endoprosthesis group. It is recommended the continued follow-up of these patients.

Undoubtedly, these results may represent the most important advance in the treatment of a cardiovascular disease which has extremely high morbidity and mortality rates and is one of the most challenging for surgeons.

Table 4 presents recommendations for the management of chronic aneurysms of the thoracic aorta.

Table 4. Recommendations for the surgical treatment of chronic thoracic aorta/ thoracic-abdominal aneurysms

Recommendations	Recommendation Level
<b>Ascending aorta</b>	
1. Surgery, if there are compression symptoms, aortic insufficiency or aortic diameter > 6.0 cm	A
2. In Marfan's syndrome, prophylactic surgery if diameter > 5.5 cm or > 5.0 cm in cases with familiar history of dissection or sudden death	D
<b>Descending aorta</b>	
1. Surgery, if symptoms or aortic diameter > 6.0 cm	A
2. Stent implantation, if aortic diameter > 6.0 cm and favorable anatomy	A

### **Abdominal Infra-renal Aorta**

In few situations in medicine, a surgical intervention called “prophylactic” have had such a great impact in the modification of the natural history of a disease, as in the case of infra-renal abdominal aneurysms (AAA), not only due to its high prevalence (from 90% to 95% of all cases of aortic aneurysms), but also due to the increase in morbidity and mortality that follow its emergency repair (the mortality risk is 10 times greater than in elective surgeries). To establish guidelines for the indication of elective repair of AAAs, aiming at corroborating the decision making, some aspects about the pattern of this disease must be considered:

- Abdominal aneurysms are often found by chance, mainly in elderly people. Many studies have estimated that AAA are found in 2% of people aged between 60 to 69 years, and in approximately 5% of people over 70-year-olds, being 2 to 3 times more common in men than in woman [192, 193] (B).

- The association of AAA with some diseases is well-known and relatively predictable: it is known that AAA is found in about 5% of patients with coronary atherosclerosis [194] (A), 9% of peripheral artery disease cases [195] (A), and in 30% to 50% of the patients with popliteal or femoral aneurysms [196] (B).

- AAAs are easily detectable by clinical examination and by non-invasive diagnostic methods. In general, an experienced physician can detect aneurysms of 5 cm in diameter directly by palpation, but the precision of the diagnosis only by touch is < 50%, while ultrasound is able to diagnose AAAs of any diameter in 100% of cases [192] (B).

- Ruptures of AAAs are considered a serious public health problem. It is estimated at 15,000 deaths per year in the USA the cases that reach hospital and maybe two or three times this number if all cases of sudden death by AAA that occur out of the hospital are included [197] (B).

- Ruptures of AAAs are no longer related to large, rapid growth aneurysms, or to the early onset of symptoms. The risk of rupture over 5 years for aneurysms smaller than 5 cm in diameter is estimated as less than 5%, while in aneurysms larger than 5 cm, the accumulated risk is 25% to 43% [197, 198] (B), [199] (D). A multivariate analysis of some studies have identified rapid expansion and the presence of significant abdominal, or low back pain as the most important predictors for rupture, independently of the size of the AAA [200] (A), [201] (B).

- Small aneurysms grow at a variable speed: although it is estimated that the mean expansion of AAAs must be about 0.4 cm/year, there is a great variability making impossible to predict the evolution in a specific patient [200] (A).

- The risk of death or major complications with elective

surgery of AAA is dependent on the experience of the surgical group and on the numbers of surgeries in the hospital, but currently the mortality rate must be lower than 5%. Although meta-analysis of recent studies point to a mean mortality rate up to 3.5% [202-206] (B), a variation up to 10% is observed between surgeons and institutions [207] (B).

The presence of symptoms in AAAs is a consensual surgical indication independent of the diameter. These symptoms include pain/lumbar or abdominal discomfort, distal embolization or signs of compression of neighboring structures. Obviously, emergency surgery is essential in the cases when rupture is suspected.

Also aneurysms of an inflammatory etiology are indicative of elective repair independent of the size, as they are followed by significant systemic manifestations, such as fever and weight loss [193] (D).

For asymptomatic patients, the indication of intervention must consider the risk of rupture versus the individual operative risk and the life expectancy. In the 90s, the International Society for Vascular Surgery recommended elective surgeries for AAAs with diameter exceeds 5.0 cm, or even 4.0 cm in patients with chronic obstructive bronchopulmonary disease based on the low postoperative mortality rate found in this group (1%). Also, there was a clear recommendation to contraindicate the surgical repair of AAAs of any size when metastatic neoplasias, severe heart failure, or other conditions that limit survival to no more than 2 to 3 years [199] (D).

However, the results of 2 recent large clinical trials may modify these criteria.

In the United Kingdom Small Aneurysm Trial [208], 1090 patients with asymptomatic aneurysms with diameters between 4.0 and 5.5 cm were randomized for conservative treatment with serial ultrasound examinations or elective surgical repair and follow-up over a period of from 6 to 10 years (mean: 8 years). The surgical mortality rate was 5.5% and survival over 8 years was similar in both groups, although slightly better in the intervention group after this period. No differences were observed in this pattern in respect to age, gender, or the initial size of the aneurysm and the authors concluded that asymptomatic AAAs patients with diameters up to 5.0 cm can be conservatively followed-up without additional risks (A).

The findings were also confirmed in the Aneurysm Detection and Management Veterans Affairs Cooperative Study [209], in which 1136 patients with infra-renal aneurysms with diameters between 4.0 and 5.4 cm were randomized for surgical treatment (n=569) or for periodic echographic control (n=567). The in-hospital mortality in the operated group was 2.7% and there was no difference in the 5-year survival rate between the two groups. There was no reduction in the death rate related to complications

of the AAA in the intervention group (3.0% vs 2.6%) and the rupture risk in the non-operated patients was minimal (0.6% per year). The authors, however, did not recommend elective surgery for AAAs with diameters of up to 5.4 cm, even when the surgical mortality of the group was low (A).

On the other hand, the high incidence of ruptures of the AAAs with diameters > 5.5 cm has been well documented by the same authors in a cohort of 198 patients with absolute contra-indications for surgical repair. The annual rupture rate was 9.4% for diameters between 5.5 and 5.9 cm; 10.2% for cases between 6.0 and 6.5 cm; 19% between 6.6 and 7.0 cm; and 32.5% in patients with diameters > 7.0 cm, results that corroborate those of previous studies (A).

The interventionist therapy using stent implantation has recently been proposed as an alternative to the surgical treatment of AAAs. Since Parodi et al. [211] have performed the first percutaneous exclusion of an intra-renal aneurysm 12 years ago many devices have been developed and tested without arriving at an agreement that this technique may be an occasional substitute to conventional surgery. The current results point to an immediate success rate near 95%, with conversion to surgery being necessary around 3% to 5% of the cases and the related in-hospital mortality rate of approximately 3%. The most common early complications are inguinal hematomas (7%), arterial thrombosis (3%), and rupture of the iliac artery (1.5%). At long-term, endoleaks have been demonstrated in 10% to 20% of the cases, with spontaneous solutions in only 40% to 50% of them [212]. When stent implantation and conventional surgery are compared, as in the studies by Hallet et al. [213] (B) and May et al. [214] (B), it may be observed that there are significant differences in the early mortality and morbidity, however the event-free survival rate over 2 years is notably better in surgical group (93% vs 67%).

In Brazil, Saadi et al. [215] have recently reported an experiment with a percutaneous exclusion of AAA in 25 cases free of intra-hospital mortality and with a 96% event-free survival in a maximum 27-month follow-up (C).

Several randomized prospective studies are being conducted to compare both conventional to endovascular technique in AAA. Two of these studies have already been published; the EVAR-1 [216] and the DREAM [217] have demonstrated a lower surgical mortality with endovascular treatment even in patients of low risk, providing a viable alternative and a lower morbidity rate compared to the conventional surgery (A). The 2- and 4-year follow-up of DREAM [218] and EVAR-1 [219] has demonstrated a trend to approximate the mortality curve, which is similar in both groups after this period. It is interesting to observe that the majority of the deaths in the middle term are not related to rupture of the aneurysm, but to other causes such as myocardial infarct, stroke, and cancer. These data only strength the diffuse characteristic

and the involvement of several organs in the atherosclerotic process in this group of patients.

In conclusion, based on the most recent Guidelines of the American Association for Vascular Surgery and the Society for Vascular Surgery we may state that: 1) Low risk asymptomatic patients should be considered for elective surgery with a minimum diameter of 5.5 cm (in women the diameter may be 5.0 cm); 2) Significant perioperative risk cases with 6.0 cm; and 3) the optimal clinical management must include control of hypertension and cessation of smoking. Endovascular therapy may be a valid alternative in high risk surgical patients [216, 217, 220] (A, D).

Table 5 presents the current criteria of intervention in cases of AAA.

Table 5. Criteria of intervention in intra-renal aortic aneurysms

Recommendations	Recommendation Level
1. Surgical treatment – if symptoms – lumbar or abdominal pain, compression of vertebral body or adjacent structures	D
2. In asymptomatic cases, surgery, if diameter > 5.5 cm and low operative risk / long life expectation	A
3. Consider diameters of 6 cm, if high operative risk (over 5%)	D
4. Lined endoprosthesis implantation, if high surgical risk patient and favorable anatomy	A

#### REFERENCES

1. Daily PO, Trueblood HW, Stinson EB, Wuerflein RD, Schumway NE. Management of acute aortic dissections. *Ann Thorac Surg.* 1970;10(3):237-47.
2. Wolfe WG, Moran JF. The evolution of medical and surgical management of acute aortic dissection. *Circulation.* 1997;56(4 pt 1):503-5.
3. Jamieson WR, Munro AI, Miyagishima RT, Allen P, Tyers FG, Gerein AN. Aortic dissection: early diagnosis and surgical management are the keys to survival. *Can J Surg.* 1982;25(2):145-9.

4. Svensson LG, Crawford ES, Hess KR, Coselli JS, Safi HJ. Dissection of the aorta and dissecting aortic aneurysms: improving early and long-term surgical results. *Circulation*. 1990;82(5 suppl):IV24-38.
5. Pretre R, Von Segesser LK. Aortic dissection. *Lancet*. 1997;349(9063):1461-4.
6. Borst HG, Laas J, Haverich A. A new look at acute type-A dissection of the aorta. *Eur J Cardiothorac Surg*. 1987;1(3):186-9.
7. Miller DC. Surgical management of acute aortic dissection: new data. *Semin Thorac Cardiovasc Surg*. 1991;3(3):225-37.
8. Culliford AT, Ayvaliotis B, Schemin R, Colvin SB, Isom OW, Spencer FC. Aneurysms of the ascending aorta and transverse arch: surgical experience in 80 patients. *J Thorac Cardiovasc Surg*. 1982;83(5):701-10.
9. Fraser Jr CD, Wang N, Mee RB, Lytle BW, McCarthy PM, Sapp SK, et al. Repair of insufficient bicuspid aortic valves. *Ann Thorac Surg*. 1994;58(2):386-90.
10. Gott VL, Cameron DE, Pyeritz RE, Gillinov AM, Greene PS, Stone CD, et al. Composite graft repair of Marfan aneurysm of the ascending aorta: results in 150 patients. *J Card Surg*. 1994;9(5):428-9.
11. Sarsam MA, Yacoub M. Remodeling of the aortic valve annulus. *J Thorac Cardiovasc Surg*. 1993;105(3):435-8.
12. Bentall H, De Bono A. A technique for complete replacement of the ascending aorta. *Thorax*. 1968;23(4):338-9.
13. Kouchoukos NT, Karp RB, Blackstone EH, Kirklin JW, Pacifico AD, Zorn GL. Replacement of the ascending aorta and aortic valve with a composite graft: results in 86 patients. *Ann Surg*. 1980;192(3):403-13.
14. Kouchoukos NT, Marshall WG Jr, Wedige-Stecher TA. Eleven-year experience with composite graft replacement of the ascending aorta and aortic valve. *J Thorac Cardiovasc Surg*. 1986;92(4):691-705.
15. Kouchoukos NT, Wareing TH, Murphy SF, Perrillo JB. Sixteen-year experience with aortic root replacement: results in 172 operations. *Ann Surg*. 1991;214(3):308-20.
16. Ergin MA, Spielvogel D, Apaydin A, Lansman SL, McCullough JN, Galla JD, et al. Surgical treatment of the dilated ascending aorta: when and how? *Ann Thorac Surg*. 1999;67(6):1853-6.
17. Cabrol C, Pavie A, Mesnildrey P, Gandjbakhch I, Laughlin L, Bors V, et al. Long-term results with total replacement of the ascending aorta and reimplantation of the coronary arteries. *J Thorac Cardiovasc Surg*. 1986;91(1):17-25.
18. Kouchoukos NT, Davila-Roman VG, Spray TL, Murphy SF, Perrillo JB, et al. Replacement of the aortic root with a pulmonary autograft in children and young adults with aortic valve disease. *N Engl J Med*. 1994;330(1):1-6.
19. Chambers JC, Somerville J, Stone S, Ross DN. Pulmonary autograft procedure for aortic valve disease: long-term results of the pioneer series. *Circulation*. 1997;96(7):2206-14.
20. Oury JH, Hiro SP, Maxwell JM, Lamberti JJ, Duran CM. The Ross procedure: current registry results. *Ann Thorac Surg*. 1998;66(6 suppl):S162-5.
21. Doty JR, Salazar JD, Liddicoat JR, Flores JH, Doty DB. Aortic valve replacement with cryopreserved aortic allograft: ten-year experience. *J Thorac Cardiovasc Surg*. 1998;115(2):379-80.
22. Kouchoukos NT. Aortic allografts and pulmonary autografts for replacement of the aortic valve and aortic root. *Ann Thorac Surg*. 1999;67(6):1846-8.
23. Erbel R, Alfonso F, Boileau C, Dirsch O, Eber B, Haverich A, et al. Diagnosis and management of aortic dissection. *Eur Heart J*. 2001;22(18):1642-81.
24. David TE, Feindel CM. An aortic valve-sparing operation for patients with aortic incompetence and aneurysm of the ascending aorta. *J Thorac Cardiovasc Surg*. 1992;103(4):617-22.
25. Bachet J, Gigou F, Laurian C, Bical O, Goudot B, Guilmet D. Four-year clinical experience with the gelatin-resorcine-formol biological glue in acute aortic dissection. *J Thorac Cardiovasc*. 1982;83(2):212-7.
26. Najafi H, Dye WS, Javid H, Hunter JA, Goldin MD, Julian OC. Acute aortic regurgitation secondary to aortic dissection: surgical management without valve replacement. *Ann Thorac Surg*. 1972;14(5):474-82.
27. Crawford ES, Svensson LG, Coselli JS, Safi HJ, Hess KR. Surgical treatment of aneurysm and/or dissection of the ascending aorta, transverse aortic arch, and ascending aorta and transverse aortic arch. Factors influencing survival in 717 patients. *J Thorac Cardiovasc Surg*. 1989;98(5 pt 1):659-73.
28. Galloway AC, Colvin SB, Grossi EA, Parish MA, Culliford AT, Asai T, et al. Surgical repair of type A aortic dissection by the circulatory arrest-graft inclusion technique in sixty-six patients. *J Thorac Cardiovasc Surg*. 1993;105(5):781-90.
29. Hagan PG, Nienaber CA, Isselbacher EM, Bruckman D, Karavite DJ, Russman PL, et al. The International Registry of Acute Aortic Dissection (IRAD). *JAMA*. 2000;283(7):897-903.
30. Ergin MA, O'Connor J, Guinto R, Griep RB. Experience with profound hypothermia and circulatory arrest in the treatment of aneurysms of the aortic arch. Aortic arch replacement for acute aortic arch dissections. *J Thorac Cardiovasc Surg*. 1982;84(5):649-55.

31. Heinemann M, Laas J, Jurmann M, Karch M, Borst HG. Surgery extended into the aortic arch in acute type A dissection: indications, techniques and results. *Circulation*. 1991;84(5 suppl):III25-30.
32. Cooley DA. Aortic aneurysm operations: past, present, and future. *Ann Thorac Surg*. 1999;67(6):1959-62.
33. Crawford ES, Kirklin JW, Naftel DC, Svensson LG, Coselli JS, Safi HJ, et al. Surgery for acute dissection of ascending aorta: should the arch be included? *J Thorac Cardiovasc Surg*. 1992;104(1):46-59.
34. Borst HG, Buhner B, Jurmann M. Tactics and techniques of aortic arch replacement. *J Car Surg*. 1994;9(5):538-47.
35. Yun KL, Glower DD, Miller DC, Fan JI, Mitchell RS, White WD, et al. Aortic dissection resulting from tear of transverse arch: Is concomitant arch repair warranted? *J Thorac Cardiovasc Surg*. 1991;102(3):355-70.
36. Moon MR, Miller C. Aortic arch replacement for dissection. In: *Operative techniques in thoracic and cardiovascular surgery*;1999;4(1):33-57.
37. Livesay JJ, Cooley DA, Reul GJ, Walker WE, Frazier OH, Duncan JM, et al. Resection of aortic arch aneurysms: a comparison of hypothermic techniques in 60 patients. *Ann Thorac Surg*. 1983;36(1):19-28.
38. Griep RB, Ergin MA, McCullough JN, Nguyen KH, Juvonen T, Chang N, et al. Use of hypothermic circulatory arrest for cerebral protection during aortic surgery. *J Card Surg*. 1997;12(2 suppl):312-21.
39. Haverich A, Miller DC, Scott WC, Mitchell RS, Dyer PE, Stinson EB. Acute and chronic aortic dissections: determinants of long-term outcome for operative survivors. *Circulation*. 1985;72(3pt2):II22-34.
40. Okita Y, Takamoto S, Ando M, Morota T, Matsukawa R, Kawashima Y, et al. Mortality and cerebral outcome in patients who underwent aortic arch operations using deep hypothermic circulatory arrest with retrograde cerebral perfusion: no relation of early death, stroke, and delirium to the duration of circulatory arrest. *J Thorac Cardiovasc Surg*. 1998;115(1):129-38.
41. Borst HG, Walterbusch G, Schaps D. Extensive aortic replacement using "elephant trunk" prosthesis. *Thorac Cardiovasc Surg*. 1983;31(1):37-40.
42. Borst HG, Frank F, Schaps D. Treatment of extensive aortic aneurysms by a new multiple-stage approach. *J Thorac Cardiovasc Surg*. 1988;95(1):11-3.
43. Palma JH, Carvalho AC, Buffolo E, Almeida DR, Gomes WJ, Brasil LA. Endoscopic placement of stents in aneurysms of the descending thoracic aorta. *Ann Thorac Surg*. 1998;66(1):256-8.
44. Griep RB, Stinson EB, Hollingsworth JF, Buehler D. Prosthetic replacement of the aortic arch. *J Thorac Cardiovasc Surg*. 1975;70(6):1051-63.
45. Galloway AC, Colvin SB, LaMendola CL, Hurwitz JB, Baumann FG, Harris LJ, et al. Ten-year operative experience with 165 aneurysms of the ascending aorta and aortic arch. *Circulation*. 1989;80(3pt1):249-56.
46. Ergin MA, Galla JD, Lansman L, Quintana C, Bodian C, Griep RB. Hypothermic circulatory arrest in operations on the thoracic aorta. Determinants of operative mortality and neurologic outcome. *J Thorac Cardiovasc Surg*. 1994;107(3):788-99.
47. Svensson LG, Crawford ES, Hess KR, Coselli JS, Raskins S, Shenag SA, et al. Deep hypothermia with circulatory arrest: determinants of stroke and early mortality in 656 patients. *J Thorac Cardiovasc Surg*. 1993;106(1):19-31.
48. McCullough JN, Galla JD, Ergin A, Griep RB. Central nervous system monitoring during operations on the thoracic aorta. In: *Operative techniques in thoracic and cardiovascular surgery* 1999;4(1):87-96.
49. Ueda Y, Miki S, Kushuhara K, Okita Y, Tahata T, Yamanaka K, et al. Surgical treatment of aneurysm or dissection involving the ascending aorta and aortic arch, utilizing circulatory arrest and retrograde cerebral perfusion. *J Cardiovasc Surg*. 1990;31(5):553-8.
50. Dresser LP, McKinney WM. Anatomic and pathophysiologic studies of the human internal jugular valve. *Am J Surg*. 1987;154(2):220-4.
51. Ye J, Yang J, Del Bigio MR, Figueira CL, Ede M, Summers R, et al. Neuronal damage after hypothermic circulatory arrest and retrograde cerebral perfusion in the pig. *Ann Thorac Surg*. 1996; 61(5):1316-22.
52. Sakurada T, Kazui T, Tanaka H, Komatsu S. Comparative experimental study of cerebral protection during aortic arch reconstruction. *Ann Thorac Surg*. 1996;61(5):1348-54.
53. Coselli JS. Retrograde cerebral perfusion. Is it valuable adjunct during circulatory arrest? In: Kawashima U, Takamoto S, eds. *Brain protection in aortic surgery*. Amsterdam: Elsevier;1997. p.167-81.
54. Safi HJ, Letsou GV, Lliopoulos DC, Subramaniam MH, Miller CC 3rd, Hasson H, et al. Impact of retrograde cerebral perfusion on ascending aortic and arch aneurysm repair. *Ann Thorac Surg*. 1997;63(6):1601-7.
55. Guilmet D, Roux PM, Bachet J, Goudot B, Tawil N, Diaz F. Nouvelle technique de protection cérébrale: chirurgie de la crosse aortique. *Presse Med*. 1986;15(23):1096-8.
56. Bachet J, Guilmet D, Goudot B, Dreyfus GD, Delentdecker



- P, Brodaty D, et al. Anterograde cerebral perfusion with cold blood: a 13-year experience. *Ann Thorac Surg.* 1999;67(6):1874-8.
57. Souza JM, Rojas SO, Berlinck MF, Mazzieri R, Oliveira PAF, Martins JRM, et al. Circulação extracorpórea pela artéria carótida comum direita na correção de doenças da aorta ascendente, arco aórtico e aorta descendente. *Rev Bras Cir Cardiovasc.* 2003;18(2):137-41.
58. Swain JA, McDonald Jr TJ, Griffith PK, Balaban RS, Clark RE, Ceckler T, et al. Low-flow hypothermic cardiopulmonary bypass protects the brain. *J Thorac Cardiovasc Surg.* 1991;102(1):76-84.
59. Sabik JF, Lytle BW, McCarthy PM, Cosgrove DM. Axillary artery: an alternative site of arterial cannulation for patients with extensive aortic and peripheral vascular disease. *J Thorac Cardiovasc Surg.* 1995;109(5):885-91.
60. Van Arsdell GS, David TE, Butany J. Autopsies in acute type A aortic dissection: surgical implications. *Circulation.* 1998;98(19 suppl):II299-304.
61. Neri E, Massetti M, Capannini G, Carone E, Tucci E, Dicieolla F, et al. Axillary artery cannulation in type A aortic dissection operations. *J Thorac Cardiovasc Surg.* 1999;118(2):324-9.
62. Whitlark JD, Goldman SM, Sutter FP. Axillary artery cannulation in acute ascending aortic dissections. *Ann Thorac Surg.* 2000;69(4):1127-9.
63. Svensson LG, Blackstone EH, Rajeswaran J, Sabik JF, Lytle BW, Gonzalez-Stawinski G, et al. Does the arterial cannulation site for circulatory arrest influence stroke risk? *Ann Thorac Surg.* 2004;78(4):1274-84.
64. Reuthebuch O, Schurr U, Hellermann J, Prêtre R, Künzli A, Lachat M, et al. Advantages of subclavian artery perfusion for repair of acute type A dissection. *Eur J Cardiothorac Surg.* 2004;26(3):592-8.
65. Strauch JT, Spielvogel D, Lauten A, Lansman SL, McMurtry K, Bodian CA, et al. Axillary artery cannulation: routine use in ascending aorta and aortic arch replacement. *Ann Thorac Surg.* 2004;78(1):103-8.
66. Moizumi Y, Motoyoshi N, Sakuma K, Yoshida S. Axillary artery cannulation improves operative results for acute type A aortic dissection. *Ann Thorac Surg.* 2005;80(1):77-83.
67. Olsson C, Thelin S. Antegrade cerebral perfusion with a simplified technique: unilateral versus bilateral perfusion. *Ann Thorac Surg.* 2006;81(3):868-74.
68. Schachner T, Nagiller J, Zimmer A, Laufer G, Bonatti J. Technical problems and complications of axillary artery cannulation. *Eur J Cardiothorac Surg.* 2005;27(4):634-7.
69. Fusco DS, Shaw RK, Tranquilli M, Kopf GS, Eleftheriades JA. Femoral cannulation is safe for type A dissection repair. *Ann Thorac Surg.* 2004;78(4):1285-9.
70. Tsai TT, Fattori R, Trimarchi S, Isselbacher E, Myrmet T, Evangelista A, et al. Long-term survival in patients presenting with type B acute aortic dissection. Insights from the International Registry of Acute Aortic Dissection. *Circulation.* 2006;114(21):2226-31.
71. Scheinin AS, Cooley DA. Graft replacement of the descending thoracic aorta: results of "open" distal anastomosis. *Ann Thorac Surg.* 1994;58(1):19-23.
72. Read RA, Moore EE, Moore FA, Haanel JB. Partial left heart bypass for thoracic aorta repair: survival without paraplegia. *Arch Surg.* 1993;128(7):746-52.
73. Chiesa R, Melissano G, Ruettimann LM, Civilini E, Tshomba Y. Surgical treatment of thoracic and thoracoabdominal aortic aneurysms: technical notes and the use of left heart bypass. *J Vasc Bras.* 2002;1(3):207-18.
74. Laschinger JC, Izumoto H, Kouchoukos NT. Evolving concepts in prevention of spinal cord injury during operations on the descending thoracic and thoracoabdominal aorta. *Ann Thorac Surg.* 1987;44(6):667-74.
75. Kouchoukos NT, Daily BB, Rokkas CK, Murphy SF, Bauer S, Abbou DN, et al. Hypothermic bypass and circulatory arrest for operations on the descending thoracic and thoracoabdominal aorta. *Ann Thorac Surg.* 1995;60(1):67-77.
76. Palma JH, Almeida DR, Carvalho AC, Andrade JC, Buffolo E. Surgical treatment of acute type B aortic dissection using an endoprosthesis (elephant trunk). *Ann Thorac Surg.* 1997;63(4):1081-4.
77. Slonim SM, Nyman U, Semba CP, Miller DC, Mitchell RS, Dake MD. Aortic dissection: percutaneous management of ischemic complications with endovascular stents and balloon fenestration. *J Vasc Surg.* 1996;23(2):241-53.
78. Cambria RP, Brewster DC, Gertler J, Moncure AC, Gusberg R, Tilson MD, et al. Vascular complications associated with spontaneous aortic dissection. *J Vasc Surg.* 1988;7(2):199-209.
79. Laas J, Heinemann M, Schaeffers HJ, Daniel W, Borst HG. Management of thoracoabdominal mal perfusion in aortic dissection. *Circulation.* 1991;84(5 suppl):III20-4.
80. Walker PJ, Miller DC. Aneurismal and ischaemic complications of type B (type III) aortic dissections. *Semin Vasc Surg.* 1992;5:198-214.
81. Eleftheriades JA, Hartleroad J, Gusberg RJ, Salazar AM, Black HR, Kopf GS, et al. Long-term experience with descending

- aortic dissection: the complication-specific approach. *Ann Thorac Surg.* 1992;53(1):11-21.
82. Miller DC, Mitchell RS, Oyer PE, Stinson EB, Jamieson WS, Shumway NE. Independent determinants of operative mortality for patients with aortic dissection. *Circulation.* 1984;70(3pt 2):1153-64.
83. Williams DM, Brothers TE, Messina LM. Relief of mesenteric ischaemia in type III aortic dissection with percutaneous fenestration of the aortic septum. *Radiology.* 1990;174(2):450-2.
84. Cowling MG, Redwood D, Buckenham TM. Case report: critical lower limb ischaemia due to aortic dissection relieved by percutaneous transfemoral fenestration. *Clin Radiol.* 1995;50(9):654-7.
85. Faykus MH Jr, Hiette P, Koopot R. Percutaneous fenestration of a type I aortic dissection for relief of lower extremity ischemia. *Cardiovasc Intervent Radiol.* 1992;15(3):183-5.
86. Trerotola SO. Use of a stone basket as a target during fenestration of aortic dissection. *J Vasc Interv Radiol.* 1996;7(5):687-90.
87. Kato N, Sakuma H, Takeda K, Hirano T, Nakagawa T. Relief of acute lower limb ischemia with percutaneous fenestration of intimal flap in a patient with type III aortic dissection - a case report. *Angiology.* 1993;44(9):755-9.
88. Saito S, Arai H, Kim K, Aoki N, Tsurugida M. Percutaneous fenestration of dissecting intima with a transeptal needle. *Cathet Cardiovasc Diagn.* 1992;26:130-5.
89. Walker PJ, Dake MD, Mitchell RS, Miller DC. The use of endovascular techniques for the treatment of complications of aortic dissection. *J Vasc Surg.* 1993;18(6):1042-51.
90. Williams DM, Lee DY, Hamilton BH, Marx MV, Narasimham DL, Kazanjian SN, et al. The dissected aorta: percutaneous treatment of ischemic complications - principles and results. *J Vasc Interv Radiol.* 1997;8(4):605-65.
91. Lee DY, Williams DM, Abrams GD. The dissected aorta. Part II. Differentiation of the true from the false lumen with intravascular US. *Radiology.* 1997;203(1):32-6.
92. Moon MR, Michell RS, Dake MD, Zarins CK, Fann JI, Miller DC. Simultaneous abdominal aortic replacement and thoracic stent-graft placement for multilevel aortic disease. *J Vasc Surg.* 1997;25(2):332-40.
93. Elefteriades JA, Hammond GL, Gusberg RJ, Kopf GS, Baldwin JC. Fenestration revisited: a safe and effective procedure for descending aortic dissection. *Arch Surg.* 1990;125(6):786-90.
94. Shennan T. Dissecting aneurysm. *Medical Research Council Special Report Series, N° 193.* London: Her Majesty's Stationery Office;1984.
95. Nienaber CA, Fattori R, Lund G, Dieckmann C, Wolf W, von Kodolitsch Y, Nicolas V, et al. Nonsurgical reconstruction of thoracic aortic dissection by stent-graft placement. *N Engl J Med.* 1999;340(20):1539-45.
96. Dake MD, Miller DC, Mitchell RS, Semba CP, Moore KA, Sakai T. The 'first generation' of endovascular stent-grafts for patients with aneurysms of the descending thoracic aorta. *J Thorac Cardiovasc Surg.* 1998;116(5):689-704.
97. Grabenwöger M, Hutschala D, Ehrlich MP, Cartes-Zumelzu F, Thurnner S, Lammer J, et al. Thoracic aortic aneurysms: treatment with endovascular self-expandable stents grafts. *Ann Thorac Surg.* 2000;69(2):441-5.
98. Fann JI, Miller DC. Endovascular treatment of descending thoracic aortic aneurysms and dissections. *Surg Clin North Am.* 1999;79(3):551-74.
99. Palma JH, Souza JAM, Alves CMR, Carvalho AC. Self-expandable aortic stent-grafts for treatment of descending aortic dissections. *Ann Thorac Surg.* 2002;73:1138-42.
100. Buffolo E, Fonseca JHP, Souza JAM, Alves CMR. Revolutionary treatment of aneurysms and dissections of descending aorta: the endovascular approach. *Ann Thorac Surg.* 2002;74(5):S1815-7.
101. Trimarchi S, Nienaber CA, Rampoldi V, Myrmet T, Suzuki T, Bossone E, et al. Role and results of surgery in acute type B aortic dissection. Insights from the International Registry of Acute Aortic Dissection (IRAD). *Circulation.* 2006;114(1 Suppl):I-357-64.
102. Eggebrecht H, Nienaber CA, Neuhauser M, Baumgart D, Kische S, Schmermund A, et al. Endovascular stent-graft placement in aortic dissection: a meta-analysis. *Eur Heart J.* 2006;27(4):489-98.
103. Guthaner DF, Miller DC, Silverman JF, Stinson EB, Wexler L. Fate of the false lumen following surgical repair of aortic dissections: an angiographic study. *Radiology.* 1979;133(1):1-8.
104. Erbel R, Oelert H, Meyer J, Puth M, Mohr-Katoly S, Hausmann D, et al. Influence of medical and surgical therapy on aortic dissection evaluated by transesophageal echocardiography: implication for prognosis and therapy. *Circulation.* 1993;87(5):1604-15.
105. Di Cesare E, Di Renzi P, Pavone P, Marsili L, Castaldo F, Passariello R. Postsurgical follow-up of aortic dissections by MRI. *Eur J Radiol.* 1991;13(1):27-30.
106. Hara KM, Yamaguchi T, Wanibuchi Y, Kurokawa K. The role of medical treatment of distal type aortic dissection. *Int J Cardiol.* 1991;32(2):231-40.

107. Masani ND, Banning AP, Jones RA, Ruttley MS, Fraser AG. Follow-up of chronic thoracic aortic dissection: comparison of transesophageal echocardiography and magnetic resonance imaging. *Am Heart J.* 1996;131(6):1156-63.
108. Ergin MA, Phillips RA, Galla JD, Lansman SL, Mendelson DS, Quinatana CS, et al. Significance of distal false lumen after type A dissection repair. *Ann Thorac Surg.* 1994;57(4):820-5.
109. Neufang KF, Theissen P, Deider S, Sechtem U. Thoracic aorta dissection - the place of MRT and CT in the follow-up after prosthetic aortic replacement. *Rofo.* 1989;151(6):659-65.
110. Rizzo JA, Darr U, Fischer M, et al. Multimodality serial follow-up of thoracic aortic aneurysms. *Int J Angiol.* 1997;6:153-6.
111. DeBakey ME, McCollum CH, Crawford ES, Morris GC, Howell J, Noon GP, et al. Dissection and dissecting aneurysms of the aorta: twenty-year follow-up of five hundred twenty-seven patients treated surgically. *Surgery.* 1982;92(6):1118-34.
112. Miller DC, Mitchell RS, Oyer PE, Stinson EB, Jamieson SW, Shumway NE. Independent determinants of operative mortality for patients with aortic dissections. *Circulation.* 1984; 70(3 PT 2):1153-64.
113. Glower DD, Speier RH, White WD, Smith LR, Rankin JS, Wolfe WG. Management and long term outcome of aortic dissection. *Ann Surg.* 1991;214(1):31-41.
114. Crawford ES, Coselli JS. Marfan's syndrome: combined composite valve graft replacement of the aortic root and transaortic mitral valve replacement. *Ann Thorac Surg.* 1988;45(3):296-302.
115. McDonald GR, Schaff HV, Pyeritz RE, McKusick VA, Gott VL. Surgical management of patients with the Marfan syndrome and dilatation of the ascending aorta. *J Thorac Cardiovasc Surg.* 1981;81(2):180-6.
116. American Academy of Pediatrics Committee on Genetics. Health supervision for children with Marfan syndrome. *Pediatrics.* 1996;98(5):978-82.
117. Taylor JFN. Clinical pediatric cardiology. *Curr Opin Cardiol* 1993; 8:108-13.
118. Braverman AC. Exercise and the Marfan syndrome. *Med Sci Sports Exerc.* 1998;30(10 suppl):S387-95.
119. Alfonso F, Goicolea J, Aragoncillo P, Hernandez R, Macaya C. Diagnosis of aortic intramural hematoma by intravascular ultrasound imaging. *Am J Cardiol.* 1995;76(10):735-8.
120. Ide K, Uchida H, Otsuji H, Nishimine K, Tsushima J, Ohishi H, et al. Acute aortic dissection with intramural hematoma: possibility of transition to classic dissection or aneurysm. *J Thorac Imaging.* 1996;11(1):46-52.
121. Nienaber CA, von Kodolitsch Y, Petersen B, Loose R, Helmchen U, Haverich A, et al. Intramural hemorrhage of the thoracic aorta: diagnostic and therapeutic implications. *Circulation.* 1995;92(6):1465-72.
122. Murray JG, Manisali M, Flamm SD, VanDyke CW, Lieber ML, Lytle BW, et al. Intramural hematoma of the thoracic aorta: MR image findings and their prognostic implications. *Radiology.* 1997;204(2):349-55.
123. Bolognesi R, Manca C, Tsiatas D, Vasini P, Zeppellini R, De Domenico R, et al. Aortic intramural hematoma: an increasingly recognized aortic disease. *Cardiology.* 1998;89(3):178-83.
124. Kaji S, Nishigami K, Akasaka T, Hozumi T, Takagi T, Kawamoto T, et al. Prediction of progression or regression of type A aortic intramural hematoma by computed tomography. *Circulation.* 1999;100(19 suppl):II281-6.
125. Pepi M, Campodonico J, Galli C, Tamborini G, Barbier P, Doria E, et al. Rapid diagnosis and management of thoracic aortic dissection and intramural haematoma: a prospective study of advantages of multiplane vs. biplane transoesophageal echocardiography. *Eur J Echocardiogr.* 2000;1(1):72-9.
126. Vilacosta I, San Roman JA, Ferreiros J, Aragoncillo P, Mendez R, Castillo JA, et al. Natural history and serial morphology of aortic intramural hematoma: a novel variant of aortic dissection. *Am Heart J.* 1997;134(3):495-507.
127. Cooke JP, Kazmier FJ, Orszulak TA. The penetrating aortic ulcer: pathologic manifestations, diagnosis and management. *Mayo Clin Proc.* 1988;63(7):718-25.
128. Kazerooni EA, Bree RL, Williams DM. Penetrating atherosclerotic ulcers of the descending thoracic aorta: evaluation with CT and distinction from aortic dissection. *Radiology.* 1992;183(3):759-65.
129. 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 so we manage it? *J Vasc Surg.* 1998;27(6):1006-16.
130. Coady MA, Rizzo JA, Elefteriades JA. Pathologic variants of thoracic aortic dissections: penetrating atherosclerotic ulcers and intramural hematomas. *Cardiol Clin.* 1999;17(4):637-57.
131. Hussain S, Glover JL, Bree R, Bendick PJ. Penetrating atherosclerotic ulcers of the thoracic aorta. *J Vasc Surg.* 1989;9(5):710-7.
132. Groenink M, Rozendaal L, Naeff MS, Hennekam RC, Hart AA, van der Wall EE, et al. Marfan syndrome in children and adolescent: predictive and prognostic value of aortic root growth for screening for aortic complications. *Heart.* 1998;80(2):163-9.

133. Gott VL, Greene PS, Alejo DE, Cameron DE, Naftel DC, Miller DC, et al. Replacement of the aortic root in patients with Marfan's syndrome. *N Engl J Med*. 1999;340(17):1307-13.
134. Finkbohner R, Johnston D, Crawford ES, Coselli J, Milewicz DM. Marfan syndrome. Long-term survival and complications after aortic aneurysm repair. *Circulation*. 1995;91(3):728-33.
135. Silverman DI, Burton KJ, Gray J, Bosner MS, Kouchoukos NT, Roman MJ, et al. Life expectancy in the Marfan syndrome. *Am J Cardiol*. 1995;75(2):157-60.
136. Legget ME, Unger TA, O'Sullivan CK, Zwink TR, Bennett RL, Byers PH, et al. Aortic root complications in Marfan's syndrome: identification of a lower risk group. *Heart*. 1996;75(4):389-95.
137. Davies RR, Goldstein LJ, Coady MA, Tittle SL, Rizzo JA, Kopf GS, et al. Yearly rupture or dissection rates for thoracic aortic aneurysms: simple prediction based on size. *Ann Thorac Surg*. 2002;73(1):17-28.
138. Carrel T, Von Segesser L, Jenni R, Gallino A, Egloff L, Bauer E, et al. Dealing with dilated ascending aorta during aortic valve replacement: advantages of conservative surgical approach. *Eur J Cardiothorac Surg*. 1991;5(3):137-43.
139. Yun KL, Miller DC, Fann JI, Mitchell RS, Robbins RC, Moore KA, et al. Composite valve graft versus separate aortic valve and ascending aortic replacement: is there still a role for the separate procedure? *Circulation*. 1997;96(suppl. 1):II368-75.
140. David TE. Current practice in Marfan's aortic root surgery: reconstruction with aortic valve preservation or replacement? What to do with the mitral valve? *J Card Surg*. 1997;12 (suppl 2):147-50.
141. Coady MA, Rizzo JA, Hamond GL, Mandapati D, Darr U, Kopf GS, et al. What is the appropriate size criterion for resection of thoracic aortic aneurysms? *J Thorac Cardiovasc Surg*. 1997;113(3):476-91.
142. Hwa J, Richards JG, Huang H, McKay D, Pressley L, Hughes CF, et al. The natural history of aortic dilatation in Marfan syndrome. *Med J Aust*. 1993;158(8):558-62.
143. Juvonen T, Ergin MA, Galla JD, Lansman SL, Nguyen KH, McCullough JN, et al. Prospective study of the natural history of thoracic aortic aneurysms. *Ann Thorac Surg*. 1997;63(6):1533-45.
144. Griep RB, Ergin MA, Galla JD, Lansman SL, McCullough JN, Nguyen KH, et al. Natural history of descending thoracic and thoracoabdominal aneurysms. *Ann Thorac Surg*. 1999;67(6):1927-30.
145. Coady MA, Davies RR, Roberts M, Goldstein LJ, Rogalski MJ, Rizzo JA, et al. Familial patterns of thoracic aortic aneurysms. *Arch Surg*. 1999;134(4):361-7.
146. Cambria RO, Davison JK, Carter C, Brewster DC, Chang Y, Clark KA, et al. Epidural cooling of spinal cord protection during thoracoabdominal aneurysm repair: a five- year experience. *J Vasc Surg*. 2000;31(6):1093-102.
147. Grabitz K, Sandmann W, Stuhmeier K, Mainzer B, Godehardt E, Ohle B, et al. The risk of ischemic spinal cord injury in patients undergoing graft replacement for thoracoabdominal aortic aneurysms. *J Vasc Surg*. 1996;23(2):230-40.
148. Coselli JS. Recent advances in surgical treatment of thoracoabdominal aortic aneurysms. In: Chiesa R, Melissano G, eds. *Gli aneurismi dell'aorta addominale*. Milano:Europa Scienze Umane Editrice;1996. p.269-84.
149. Acher CW, Wynn MM, Hoch JR, Popic P, Archibald J, Turnipseed WD. Combined use of cerebral spinal fluid drainage and naloxone reduces the risk of paraplegia in thoracoabdominal aneurysm repair. *J Vasc Surg*. 1994;19(2):236-48.
150. Svensson LG, Crawford ES, Hess KR, Coselli JS, Safi HJ. Experience with 1509 patients undergoing thoracoabdominal aortic operations. *J Vasc Surg*. 1993;17(2):357-70.
151. Hollier LH, Money SR, Naslund TC, Proctor CD Sr, Buhman WC, Marino RJ, et al. Risk of spinal cord dysfunction in patients undergoing thoracoabdominal aortic replacements. *Am J Surg*. 1992;164(3):201-4.
152. Safi HJ, Campbell MP, Miller III CC, Iliopoulos DC, Khoynzhad A, Letsou GV, et al. Cerebral spinal fluid drainage and distal aortic perfusion decrease the incidence of neurological deficit: the results of 343 descending and thoracoabdominal aortic aneurysm repairs. *Eu J Vasc Endovasc Surg* 1997;14(2):118-24.
153. Coselli JS, LeMaire SA. Left heart bypass reduces paraplegia rates after thoracoabdominal aortic aneurysm repair. *Ann Thorac Surg*. 1999;67(6):1931-4.
154. Schepens MA, Defauw JJ, Hamerlijnck RP, Vermeulen FE. Use of left heart bypass in the surgical repair of thoracoabdominal aortic aneurysms. *Ann Vasc Surg*. 1995;9(4):327-38.
155. Svensson LG, Stewart RW, Cosgrove DM 3rd, Lytle BW, Antunes MD, Beven EG, et al. Intrathecal papaverine for the prevention of paraplegia after operation in the thoracic or thoracoabdominal aorta. *J Thorac Cardiovas Surg*. 1998(5);96:823-9.
156. Svensson LG, Crawford ES, Hess KR, Coselli JS, Safi HJ. Thoracoabdominal aortic aneurysms associated with celiac, superior mesenteric, and renal artery occlusive disease: methods and analysis of results in 271 patients. *J Vasc Surg*. 1992;16(3):378-90.
157. Cooley DA, Golino A, Frazier OH. Single-clamp technique

- for aneurysms of the descending thoracic aorta: report of 132 consecutive cases. *Eur J Cardiothorac Surg.* 2000;18(2):162-7.
158. Crawford ES, Crawford JL, Safi HJ, Coselli JS, Hess KR, Brooks B, et al. Thoracoabdominal aortic aneurysms: preoperative and intraoperative factors determining immediate and long-term results of operation in 605 patients. *J Vasc Surg.* 1986;3(3):389-404.
159. Crawford ES, Coselli JS, Safi HJ. Partial cardiopulmonary bypass, hypothermic circulatory arrest, and posterolateral exposure for thoracic aortic aneurysm operation. *J Thorac Cardiovasc Surg.* 1987;94(6):824-7.
160. Verdant A, Cossette R, Page A, Baillot R, Dontigny L, Page P. Aneurysms of the descending thoracic aorta: three hundred sixty-six consecutive cases resected without paraplegia. *J Vasc Surg.* 1995;21(3):385-91.
161. Miyamoto K, Ueno A, Wada T, Kimoto S. A new and simple method of preventing spinal cord damage following temporary occlusion of the thoracic aorta by draining the cerebrospinal fluid. *J Cardiovasc Surg.* 1960;1:188-97.
162. Blaisdell FW, Cooley DA. The mechanism of paraplegia after temporary thoracic aortic occlusion and its relationship to spinal fluid pressure. *Surgery.* 1962;51:351-5.
163. Wadough F, Lindemann EM, Arndt CF, Hetzer R, Borst HG. The arteria radicularis magna anterior as a decisive factor influencing spinal cord damage during aortic occlusion. *J Thorac Cardiovasc Surg.* 1984;88(1):1-10.
164. Svensson LG, Von Ritter CM, Groeneveld HT, Rickards ES, Hunter SJ, Robinson MF, et al. Cross-clamping of the thoracic aorta: Influence of aortic shunts, laminectomy, papaverine, calcium channel blockers, allopurinol, ad superoxide dismutase on spinal cord blood flow and paraplegia in baboons. *Ann Surg.* 1986;204(1):38-47.
165. Kazama S, Masaki Y, Maruyama S, Ishihara A. Effect of altering cerebrospinal fluid pressure on spinal cord blood flow. *Ann Thorac Surg.* 1994;58(1):112-5.
166. Simpson JI, Eide TR, Schiff GA, Clagnaz JF, Hossain I, Tverskoy A et al. Intrathecal magnesium sulfate protects the spinal cord from ischemic injury during thoracic aortic cross-clamping. *Anesthesiology.* 1994;81(6):1493-9.
167. Follis F, Miller K, Scremin OU, Pett S, Kessler R, Wernly J. NMDA receptor blockade and spinal cord ischemia due to aortic cross clamping in the rat model. *Can J Neurol Sci.* 1994;21(3):227-32.
168. Murray MJ, Bower TC, Oliver Jr WC, Werner E, Glociczki P. Effects of cerebrospinal fluid drainage in patients undergoing thoracic and thoracoabdominal aortic surgery. *J Cardiothorac Vasc Anesth.* 1993;7(3):266-72.
169. Safi HJ, Bartoli S, Hess KR, Shenaq SS, Viets JR, Butt GR, et al. Neurologic deficit in patients at high risk with thoracoabdominal aortic aneurysms: the role of cerebral spinal fluid drainage and distal aortic perfusion. *J Vasc Surg.* 1994;20(3):434-44.
170. Svensson LG, Patel V, Robinson MF, Ueda T, Roehm JO Jr, Crawford ES. Influence of preservation or perfusion intraoperatively identified spinal cord blood supply on spinal motor evoked potentials and paraplegia after aortic surgery. *J Vasc Surg.* 1991;13(3):355-65.
171. Svensson LG, Grum DF, Bednarski M, Cosgrove DM 3rd, Loop FD. Appraisal of cerebrospinal fluid alterations during aortic surgery with intrathecal papaverine administration and cerebrospinal fluid drainage. *J Vasc Surg.* 1990;11(3):423-9.
172. Mutch WA, Graham MR, Halliday WC, Thiessen DB, Girling LG. Use of neuroanesthesia adjuncts (hyperventilation and mannitol administration) improves neurological outcome after thoracic aortic cross-clamping in dogs. *Stroke.* 1993;24(8):1204-11.
173. Grabitz K, Freye E, Prior R, Schror K, Sandmann W. Does prostaglandin E1 and superoxide dismutase prevent ischaemic spinal cord injury after thoracic aortic cross-clamping? *Eur J Vasc Surg* 1990;4(1):19-24.
174. Svensson LG, Loop FD. Prevention of spinal cord ischemia in aortic surgery. In: Bergan JJ, Yao JST, eds. *Arterial surgery: new diagnostic and operative techniques.* New York:Grune & Stratton;1988. p.273-85.
175. Johnson SH, Kraimer JM, Graeber GM. Effects of flunarizine on neurological recovery and spinal cord blood flow in experimental spinal cord ischemia in rabbits. *Stroke.* 1993;24(10):1547-53.
176. Svensson LG. New and future approaches for spinal cord protection. *Semin Thorac Cardiovas Surg.* 1997;9(3):206-21.
177. Crawford ES, Mizrahi EM, Hess KR, Coselli JS, Safi HJ, Patel VM. The impact of distal aortic perfusion and somatosensory evoked potential monitoring on prevention of paraplegia after aortic aneurysm operation. *J Thorac Cardiovasc Surg.* 1988;95(3):357-67.
178. Svensson LG, Klepp P, Hinder RA. Spinal cord anatomy of the baboon: comparison with man and implications on spinal cord blood flow during thoracic aortic cross-clamping. *S Afr J Surg.* 1986;24(1):32-4.
179. Dommissse GF. *The arteries and veins of the human spinal cord from birth.* Edinburgh:Churchill Livingstone;1975.
180. Svensson LG, Hess KR, Coselli JS, Safi HR. Influence of segmental arteries, extent, and atriopulmonary bypass on postoperative paraplegia after thoracoabdominal aortic operations. *J Vasc Surg.* 1994;20(1):255-62.

181. Williams GM. Treatment of chronic expanding dissecting aneurysms of the descending thoracic and upper abdominal aorta by extended aortotomy, removal of the dissected intima, and closure. *J Vasc Surg.* 1993;18(3):441-9.
182. Svensson LG. Intraoperative identification of spinal cord blood supply during repairs of descending aorta and thoracoabdominal aorta. *J Thorac Cardiovasc Surg.* 1996;112(6):1455-61.
183. Svensson LG, Rickards E, Coull A, Rogers G, Fimmel CJ, Hinder RA. Relationship of spinal cord blood flow to vascular anatomy during thoracic aortic cross-clamping and shunting. *J Thorac Cardiovasc Surg.* 1986;91(1):71-8.
184. Svensson LG, Crawford ES, Patel V, McLean TR, Jones JW, DeBakey ME. Spinal oxygenation, blood supply localization, cooling, and function with aortic clamping. *Ann Thorac Surg.* 1992;54(1):74-9.
185. Coselli JS, LeMaire SA, Buket S, Berzin E. Subsequent proximal aortic operations in 123 patients with previous infrarenal abdominal aortic aneurysm surgery. *J Vasc Surg.* 1995;22(1):59-67.
186. Osenbach RK, Hitchon PW, Mouw L, Yamada T. Effects of spinal cord ischemia on evoked potential recovery and post-ischemic regional spinal cord blood flow. *J Spinal Disord.* 1993;6(2):146-54.
187. Reuter DG, Tacker Jr WA, Badylak SF, Voorhees WD 3rd, Konrad PE. Correlation of motor-evoked potential response to ischemic spinal cord damage. *J Thorac Cardiovasc Surg.* 1992;104(2):262-72.
188. Matsui Y, Goh K, Shiiya N, Murashita T, Miyama M, Ohba J, et al. Clinical application of evoked spinal cord potentials elicited by direct stimulation of the cord during temporary occlusion of the thoracic aorta. *J Thorac Cardiovasc Surg.* 1994;107(6):1519-27.
189. 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 Eng J Med.* 1994;331(26):1729-34.
190. Dake MD, Miller DG, Mitchell RS, Semba CP, Moore KA, Sakai T. The "first generation" of endovascular stent-grafts for patients with aneurysms of the descending thoracic aorta. *J Thorac Cardiovasc Surg.* 1998;116(5):689-704.
191. Bavaria JE, Appoo JJ, Makaroun MS, Verter J, Yu ZF, Mitchell RS. Endovascular stent grafting versus open surgical repair of descending thoracic aortic aneurysms in low-risk patients: a multicenter comparative trial. *Gore TAG Investigators. J Thorac Cardiovasc Surg.* 2007;133(2):369-77.
192. Leopold GR, Goldberger LE, Bernstein EF. Ultrasonic detection and evaluation of abdominal aortic aneurysms. *Surgery.* 1972;72(6):939-45.
193. Nehler MR, Taylor Jr LM, Moneta GL, Porter JM. Indications for operation for infrarenal abdominal aortic aneurysms: current guidelines. *Semin Vasc Surg.* 1995;8(2):108-14.
194. Thurmond AS, Semler HJ. Abdominal aortic aneurysm: Incidence in a population at risk. *J Cardiovasc Surg* 1986;27(4):457-60.
195. MacSweeney ST, O'Meara M, Alexander C, O'Malley MK, Powell JT, Greenhalgh RM. High prevalence of unsuspected abdominal aortic aneurysm in patients with confirmed symptomatic peripheral or cerebral arterial disease. *Br J Surg.* 1993;80(5):582-4.
196. Taylor Jr LM, Porter JM. Basic data related to clinical decision-making in abdominal aortic aneurysms. *Ann Vasc Surg.* 1987;1(4):502-4.
197. Glimaker H, Holmberg L, Elvin A, Nybacka O, Almgren B, Bjorck CG, et al. Natural history of patients with abdominal aortic aneurysm. *Eur J Vasc Surg.* 1991;5(2):125-30.
198. Nevitt MP, Ballard DJ, Hallet Jr JW. Prognosis of abdominal aortic aneurysms: a population-based study. *N Engl J Med.* 1989;321(15):1009-14.
199. Hollier LH, Taylor LM, Ochsner J. Recommended indications for operative treatment of abdominal aortic aneurysms. Report of a subcommittee of the Joint Council of the Society for Vascular Surgery and the North American Chapter of the International Society for Cardiovascular Surgery. *J Vasc Surg.* 1992;15(6):1046-56.
200. Cronenwett JL, Murphy TF, Zelenock GB, Whitehouse WM Jr, Lindenauer SM, Graham LM, et al. Actuarial analysis of variables associated with rupture of small abdominal aortic aneurysms. *Surgery.* 1985;98(3):472-83.
201. Hoffman M, Avellone JC, Plecha FR, Rhodes RS, Donovan DL, Beven EG, et al. Operations for ruptured abdominal aortic aneurysms: a community-wide experience. *Surgery.* 1982;91(5):597-602.
202. Perry MO, Calcagno D. Abdominal aortic aneurysm surgery: the basic evaluation of cardiac risk. *Ann Surg.* 1988;208(6):738-42.
203. Leather RP, Shah DM, Kaufman JL, Fitzgerald KM, Chang BB, Feustel PJ. Comparative analysis of retroperitoneal and transperitoneal aortic replacement for aneurysm. *Surg Gynecol Obstet.* 1989;168(5):387-93.
204. Sicard GA, Allen BT, Munn JS, Anderson CB. Retroperitoneal versus transperitoneal approach for repair of abdominal aortic aneurysms. *Surg Clin North Am* 1989;69(4):795-806.
205. Sullivan CA, Rohrer MJ, Cutler BS. Clinical management of the symptomatic but unruptured abdominal aortic aneurysm. *J Vasc Surg.* 1990;11(6):799-803.

- 
206. AbuRahma AF, Robinson PA, Boland JP, Lucente FC, Stuart SP, Neuman SS, et al. Elective resection of 332 abdominal aortic aneurysms in a southern West Virginia community during a recent five-year period. *Surgery*. 1991;109(3 pt 1):244-51.
207. Hannan EL, O'Donnell JF, Kilburn H, Bernard HR, Yazici A. Investigation of the relationship between volume and mortality for surgical procedures performed in New York State hospitals. *JAMA*. 1989;262(4):503-10.
208. United Kingdom Small Aneurysm Trial Participants. Long-term outcomes of immediate repair compared with surveillance of small abdominal aortic aneurysms. *N Eng J Med*. 2002;346(19):1445-52.
209. Lederle FA, Wilson SE, Johnson GR, Reinke DB, Littooy FN, Acher CW, et al. Immediate repair compared with surveillance of small abdominal aortic aneurysms. *N Eng J Med*. 2002;346(19):1437-44.
210. Lederle FA, Johnson GR, Wilson SE, Ballard DJ, Jordan WD Jr, Blebea J, et al. Rupture rate of large abdominal aortic aneurysms in patients refusing or unfit for elective repair. *JAMA*. 2002;287(22):2968-72.
211. Parodi JC, Palmaz JC, Barone HD. Transfemoral intraluminal graft implantation for abdominal aortic aneurysms. *Ann Vasc Surg*. 1991;5(6):491-9.
212. Hallett Jr JW. Management of abdominal aortic aneurysms. *Mayo Clin Proc*. 2000;75(4):395-9.
213. Hallett Jr JW, Marshall DM, Petterson TM, Gray DT, Bower TC, Cherry Jr KJ, et al. Graft-related complications after abdominal aortic aneurysm repair: reassurance from a 36-year population-based experience. *J Vasc Surg*. 1997;25(2):277-86.
214. May J, Woodburn K, White G. Endovascular treatment of infrarenal abdominal aortic aneurysms. *Ann Vasc Surg*. 1998;12(4):391-5.
215. Saadi EK, Gastaldo F, Dussin LH, Zago AJ, Barbosa G, Moura L. Tratamento endovascular dos aneurismas de aorta abdominal: experiência inicial e resultados a curto e médio prazo. *Rev Bras Cir Cardiovasc*. 2006; 21(2): 211-6.
216. Greenhalgh RM, Brown LC, Kwong GP, Powell JT, Thompson SG. The EVAR 1 participants. Comparison of endovascular aneurysm repair with open repair in patients with abdominal aortic aneurysm (EVAR trial 1):30 day operative mortality results: randomized controlled trial. *Lancet*. 2004;364(9437):843-8.
217. Prinssen M, Verhoeven EL, Buth J, Cuypers PW, van Sambeek, Balm R, et al. The DREAM participants. A randomized trial comparing conventional and endovascular repair of abdominal aortic aneurysms. *N Engl J Med*. 2004;351:1607-18.
218. Blankensteijn JD, de Jong SE, Prinssen M, van der Ham AC, But J, van Sterkenburg et al. Dutch Randomized Endovascular Aneurysm Management (DREAM) Trial Group. Two-year outcomes after conventional or endovascular repair of abdominal aortic aneurysms. *N Engl J Med*. 2005;352(23):2398-405.
219. EVAR Trial participants. Endovascular aneurysm repair versus open repair in patients with abdominal aortic aneurysms (EVAR Trial1): randomized controlled trial. *Lancet*. 2005;365(9478):2179-86.
220. Brewster DC, Cronenwett JL, Hallett Jr JW, Johnston KW, Krupski WC, Matsumura JS, et al. Guidelines for the treatment of abdominal aortic aneurysms. Report of a subcommittee of the Joint Council of the American Association for Vascular Surgery and Society for Vascular Surgery. *J Vasc Surg*. 2003;37(5):1106-17.