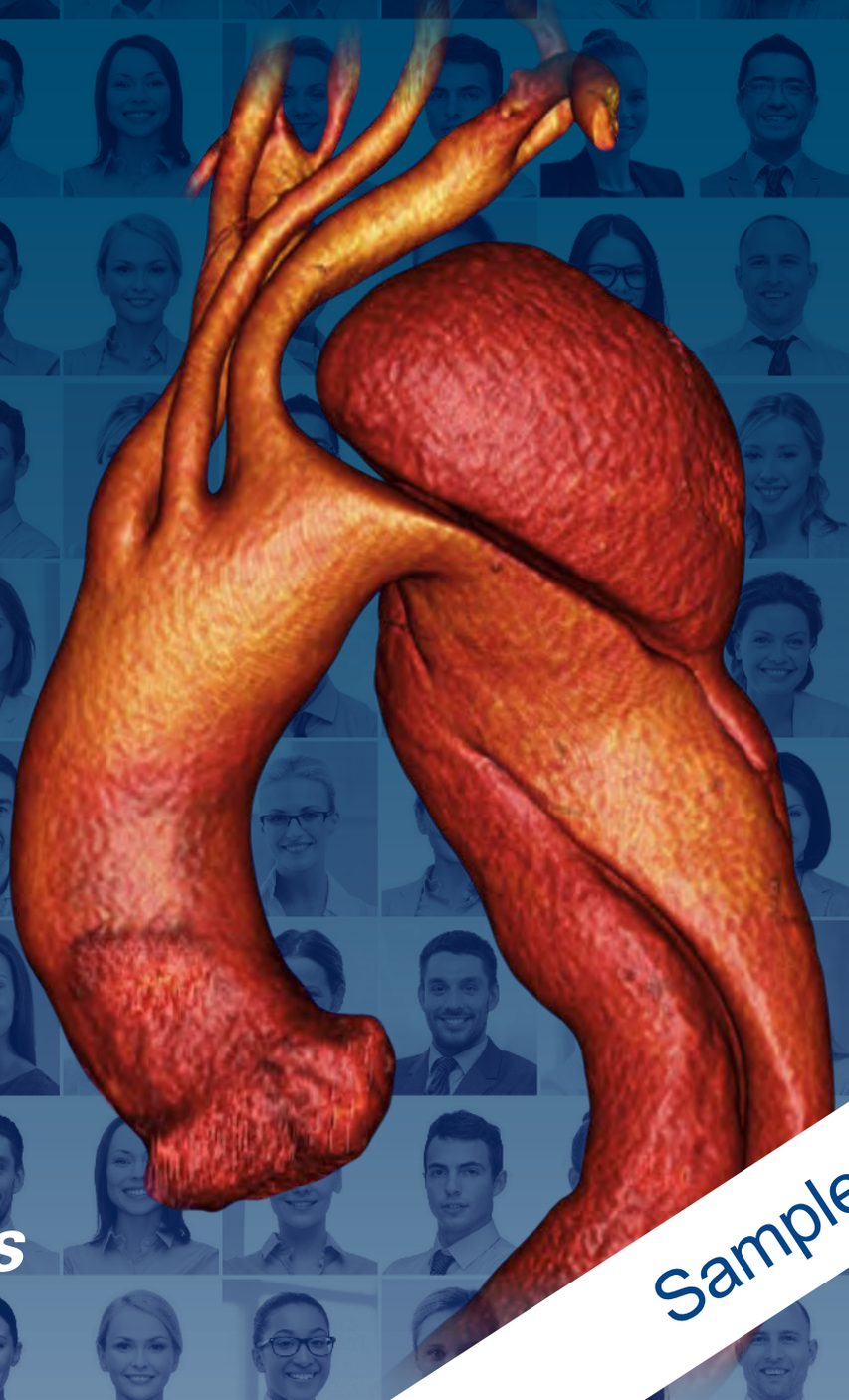
Germano Melissano · Roberto Chiesa

AORTIC DISSECTION

patients true stories
and the innovations that saved their lives



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Sample Chapters

Germano Melissano • Roberto Chiesa
Editors

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AORTIC DISSECTION patients true stories and the innovations that saved their lives
Germano Melissano • Roberto Chiesa (Editors)

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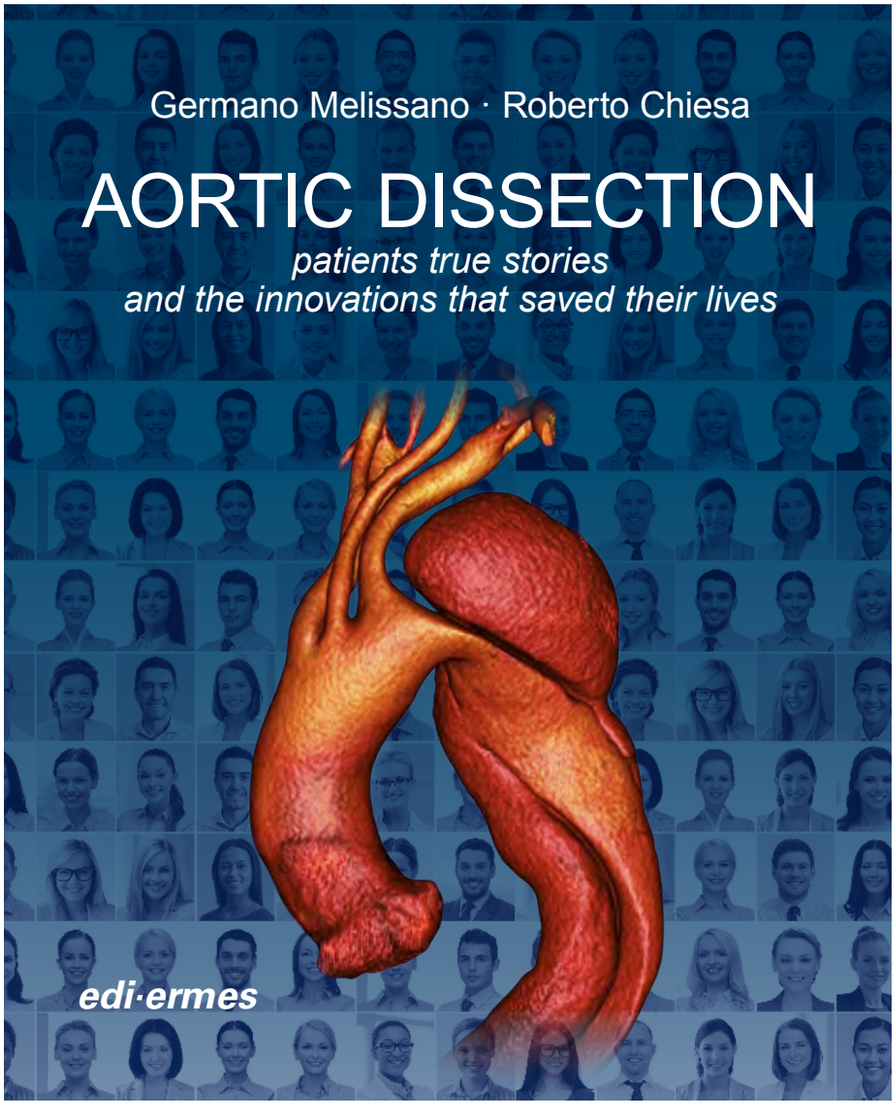

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Foreword

This gem of a book on aortic dissection brings together as authors all the superstars in the field and is edited by two of them, Germano Melissano and Roberto Chiesa. The volume contains the most thorough and up-to date information on this disease entity and its treatment. Interestingly it also includes two chapters which provide perspectives of patients suffering from this serious disease which is so often unrecognized and poorly treated. Accordingly this text is a "must have" for all specialists and generalists interested in the challenging disease process of aortic dissections.

This valuable text is published on the occasion of the highly esteemed "Aortic Surgery – How To Do It" international meeting, which has been running for 14 years and enjoys a superb international reputation. The meeting has a stellar faculty and has been endorsed by many scientific societies, as well as by the Marfan Foundation (a prominent U.S.-based patients association). It is attended annually by more than 1,000 delegates and is further described at www.aorticsurgery.it.

Books that were published for previous "How To Do It" meetings, such as Thoraco Abdominal Aorta: Surgical and Anesthetic Management (2012) and History of Aortic Surgery in the World (2014), were favorably reviewed and remain an important part of most libraries – especially ones that are used by vascular and cardiac surgeons and others interested in these difficult-to-treat entities. This volume is another in this outstanding series.

In the current exemplary volume, Drs. Melissano and Chiesa, have recruited as contributors all the world innovators and leaders who are advancing knowledge in the natural history and exciting new treatments of Types A and B aortic dissections. All the contributors are widely acknowledged to be world leaders and experts in the field. Their research and clinical excellence have led to the rapid advances in the treatment of these aortic dissection disease entities – advances which are slowly being adopted around the world. Clearly the achievements of these experts has been one of the most exciting developments in vascular disease treatment of the last 3 decades. All these achievements are beautifully summarized and illustrated in this outstanding book which is without equal and an indispensable addition to the library of anyone interested in the management of aortic dissections.

New York, December 2016

Frank J. Veith

Preface

Aortic Dissection is an exceptionally complex disease and remains one of the most misdiagnosed diseases across the globe. Although it is rare, doctors with an expertise in identifying and treating this disease are critical to our field. Diagnosis, even when correctly made, may bring ominous consequences for patients, especially when it is delayed. Treatment can also be extremely challenging, and correct timing is crucial.

Endovascular treatment of Aortic Dissection has been possible for more than 20 years, but can only be offered to select patients by skilled physicians. We must make do with inadequate tools since most endografts are not disease-specific, and an ideal graft to treat Aortic Dissection will not be available for many years. Open surgery can be very complex and challenging, and results are often unsatisfactory, especially for acute, complicated cases.

Most textbooks teach the clinical presentation of disease by simply listing symptoms, signs, and their relative frequency. However, a doctor becomes familiar with clinical presentation by actually observing cases in her clinical practice. Given the rarity of Aortic Dissection and its extremely variable presentations, it can be very difficult for an inexperienced doctor to make a correct diagnosis. It is, therefore, more important than ever for physicians to build their knowledge base of this area.

Aortic Dissection is a massive trauma for patients. In most cases, treated patients are not cured, they merely receive a palliation that takes care of the immediate risk of life and complications, but the more distal part of the aorta is still dissected, requiring the patient to submit to lifelong surveillance. This may cause a great deal of psychological distress, particularly when general practitioners are ill-equipped to handle their doubts, questions, and resulting psychological disorders. Surgeons often think their task is finished when a patient is dismissed from the hospital and a 6-month follow-up consultation is scheduled, but from the patient's perspective, the operation is not something that can be readily dismissed. Patients must live with the consequences of their illness every day. They want to know how their actions and their environment might affect the fate of their their bodies, and ultimately their lives.

Although the aorta is one organ and Aortic Dissection is one disease, Type A aortic dissection is usually treated by cardiac surgeons, and Type B aortic dissection, by vascular surgeons. This book discusses both types for the benefit of both specialists. Moreover, this book provides a unique glimpse into the lives of the patients who suffer with this disease. In addition to scientific chapters written by the most prominent experts in the field, we have included stories shared by Aortic Dissection survivors. All the stories are true, although they may have been slightly edited for length, language (they come from different parts of the world), and to make them anonymous. It is well known that facts are easily forgotten but stories and emotions stick in our minds, and our hope is that these stories will help readers remember and recognize Aortic Dissection when a similar case enters the ER. Maybe these stories will save a few lives.

We would like to gratefully acknowledge all the former patients who shared their aortic dissection stories and in particular our good friend Timo Söderlund who collected them and is hugely active in promoting awareness for this disease throughout the world. Many thanks also to all the very distinguished colleagues who took time from their extra-busy schedules to write their chapters and share their very valuable experience; special thanks to Martin Czerny who backed us up in this project from the very beginning. We would also like to thank Ms. Brittany Terwilliger for her assistance and expertise in the English language and Mrs. Adriana Lombardi who followed us patiently on behalf of the publisher Edi.Ermes.

Milan, December 2016

Germano Melissano *Roberto Chiesa*

THE MARFAN FOUNDATION

Know the signs. Fight for victory.

Professor Roberto Chiesa
Professor Germano Melissano
Ospedale San Raffaele, Milano
Universita Vita – Salute San Raffaele, Milano

Dear Professors Chiesa and Melissano,

I am writing in support of the 7th International Congress on the Aorta, which will highlight Aortic and Peripheral Surgery, scheduled for December 15-17, 2016. This meeting will bring global expertise to share new developments in clinical practice and research.

As the Chief Science Officer of The Marfan Foundation, a US based non-profit patient organization, it is extremely important to our patient population that cutting edge topics in aortic surgery and research are shared among physicians who care for patients that need these life-saving surgeries. I had the opportunity to attend a similar Congress in Milan in 2015 and the program was excellent. Inclusion of the leaders of patient organizations globally is needed so that we can bring our membership up-to-date and state-of-the-art information in life-saving aortic surgery.

I am happy to recommend this International Congress which has great potential and significance to the scientific and patient community.

Sincerely,

Josephine Grima

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01.40.25.80.66

Paris, July 25th 2016,

To Prof. Chiesa and Prof. Melissano,

Subject : VASCern Support to the Aortic and Peripheral Surgery "HOW TO DO IT" Congress 2016

Dear Prof. Chiesa and Prof. Melissano,

European Reference Networks (ERNs) are part of an EU initiative to connect healthcare specialists across Europe to tackle complex or rare medical conditions that require highly specialised healthcare and a concentration of knowledge and resources. In line with this ambition, the VASCern network have applied to the ERN 2016 Call for proposal and submitted the VASCern Project Proposal for the ERN on Rare Multisystemic Vascular Diseases, together with 31 Healthcare Provider Member Applicants, including the SS Centro Malattie Rare – MarfanClinic represented by Dr. Alessandro Pini.

ERNs project proposals are currently under the European Commission's assessment, which will be followed by a technical assessment which should take place between September-November 2016. Afterwards, the first ERNs will obtain the label in January 2017.

I, Pr. Guillaume Jondeau, as the Network Coordinator of the ERN Project Proposal on Rare Multisystemic Vascular Diseases (VASCern), would like to support by this letter the world Congress on Aorta: Aortic and Peripheral Surgery "HOW TO DO IT" 2016, that you are organizing and which will take place in Milan between December 15-17th, 2016.

This 7th international Congress will tackle cutting-edge topics, explore new developments in clinical practice and relevant research, and access the latest information on aortic, peripheral, carotid and venous diseases. World-class speakers will discuss recent achievements in the vascular and endovascular field, industrial innovations and novel treatment approaches . An educational program will be proposed as well.

For these reasons, as VASCern Network Project Coordinator, I support the development of this Aorta Congress, and will communicate about it to our Network members as well as on our website.

Yours faithfully,

Pr. Guillaume Jondeau
VASCern project Network Coordinator
CRMR Marfan Syndrome and Related Disorders
FAVA-Multi, French Rare Multi-Systemic Vascular Diseases Network
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Contents

2

An introduction to Type B aortic dissection

Germano Melissano

2.1 Introduction

Aortic dissection (AD) is a very complex disease. Its pathophysiology is not yet fully understood and the clinical presentation can be extremely variable and deceiving so that a straightforward diagnosis is not always the rule. Surgical therapy, both open and endovascular, is also challenging; the greatest challenge, however, may be life after AD and dissection survivors are to be commended for their ongoing efforts in coping with everyday life.

The complexity of the disease has its roots in the complexity of the structure of the aortic wall itself. The aortic wall structural characteristics are mostly related to the lamellar structure of the tunica media. In normal aortas, the lamellae consist of an orderly pattern of layers of elastic fibers alternated with layers of smooth muscle cells. The number of lamellae is highest (≈40) in the ascending aorta and decreases in the thoracic (≈30) and abdominal aorta (≈20); it should be noted that vasa vasorum within the tunica media are observed only when a larger number of lamellae are present; otherwise the aortic wall tissue is nourished directly from the lumen of the aorta [1].

Elastic fibers, unfortunately, are not produced after the first years of life and with age there is an ongoing tendency toward elastic fiber degeneration and loss, making the aorta a much stiffer structure (basically, the same loss of elasticity that we observe in the skin of aging persons occurs also in the aorta, but with more functional and less esthetic effects). Moreover, atherosclerotic changes with severe disarrangement of the medial lamellar structure and histological alterations such as cystic medionecrosis greatly alter the structural properties of the aortic tunica media [2].

It is important to realize that dissection affects the aorta within the layers of the tunica media (inner 1/3 – outer 2/3). Therefore, when terms such as “intimal tear” or “intimal lamella” are used, they do not actually refer to the histological tunica intima which is a mono-cellular layer (and, in spite of its great biological importance, has little or no structural significance); they refer, rather, to the tunica intima plus an important portion of the tunica media. Therefore, the outer wall of a dissected aorta is left with only the adventitia and part of the media and is clearly quite fragile [3]. This is responsible for frank ruptures of dissected aortas but also for ongoing aneurysmatic degeneration. It also explains the great technical difficulty with

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G. Melissano - R. Chiesa (eds), Aortic Dissection patients true stories and the innovations that saved their lives

surgical suturing of acutely dissected aortas, and the higher risks of performing endovascular repair in the first hours or days of the acute phase.

2.2 Epidemiology

Type A AD is an uncommon disorder with an estimated annual incidence of between 2.9 and 4.5 per 100,000 [4, 5]; the incidence of the disease seems to have increased over time [6]. Type B AD accounts for 25 to 40% of all aortic dissections and the ratio of Type A to Type B was approximately 5:1 in different studies [7]. In the light of recent advances in diagnostics and medical management, and given the often more benign course of acute Type B AD with transition into a chronic form of disease [8], it is likely that the calculated annual incidence of 0.5-0.6/100,000 of Type B AD might be underestimated.

Approximately 75% of Type A AD occurs in people aged 40-70 years, with a peak in the 50-65 years range. In patients with Marfan syndrome or a bicuspid valve, the onset is earlier, usually in the third or fourth decades of life [9]. The male to female ratio in Type B AD patients varies among studies, ranging from 2 to 5:1 [10]. The highest incidence rates were found in men aged 65-74 years (14.6/100,000 person-years) and women aged 75-84 years (19.0/100,000 person-years) [11].

Limited information is available regarding the geographical, racial, and ethnic distribution of AD. However, different studies have stated that Type A AD is more common in the black than in the white population, and less common in Asians than in Caucasians [12]. Moreover, in the black population Type B AD is more common than Type A (52.4% vs. 47.6%, respectively) [13].

Different risk factors have been associated with AD:

- hypertension is the predominant cardiovascular risk factor in patients with aortic dissection, with 70–85% of individuals being affected. Whether hypertension is more common with Type B than Type A AD is unclear;
- tobacco use prevalence in patients with Type A AD is highly inconsistent, ranging from 38 to 70%. In addition, whether smoking is more common among patients with Type B than with Type A AD is still an open issue;
- several congenital cardiovascular disorders may be associated with Type A AD, most notably bicuspid valve and aortic coarctation. Bicuspid valve is found in 3% of patients with Type B AD. Similarly, 1% of individuals affected by aortic coarctation develop Type A AD;
- inflammatory diseases (giant cell arteritis, Takayasu's arteritis, rheumatoid arthritis, systemic lupus erythematosus, and Behçet disease) are considered a risk factor for Type A AD. Approximately 4% of individuals with Type A AD have aortitis, and Type A AD occurs in 1–5% of aortitis-affected individuals;
- syndromic conditions associated to Type A AD are Marfan syndrome, Loeys–Dietz syndrome, Ehlers-Danlos syndrome and Turner syndrome. Overall, about 5% of patients with Type A AD have Marfan syndrome and the syndrome is more common among those with Type A than with Type B AD. Conversely, up to 70% of patients with Marfan syndrome develop aortic dissection, which is Type A in 64–86% of cases and Type B in 14–36%;
- non-syndromic familial conditions have been associated to Type A AD. Approximately 16% of non-syndromic familial cases are Type A, while 11% are Type B AD; the remaining 73% have thoracic aneurysms without dissection;
- dilatation of the aorta is a well-established risk factor for thoracic dissection. For the descending thoracic aorta, the critical hinge point diameter at which there is an increased risk of either rupture or dissection is 7 cm [14];
- iatrogenic injuries can also cause AD, more commonly Type A. The most common procedures resulting in iatrogenic dissections are cardiac catheterization and cardiac surgery;
- use of drugs, particularly cocaine, appears to be temporally related to the development of Type A AD; the mean interval between cocaine use and onset of Type A AD is about 12 h.

2.3 Diagnosis

The cornerstone of AD treatment is a precise understanding of the specific anatomo-pathologic characteristics of each individual case. The best diagnostic tool to use depends on the type of dissection and the stage of the disease. Acute Type A dissection is usually diagnosed by ultrasound, preferably with transesophageal echocardiography (TEE, Fig. 2.1), and since the disease is highly lethal in the first hours, many patients undergo emergency surgery on the basis of TEE alone[15].

The management of Type B AD, on the other hand, requires a far more thorough assessment, which is usually obtained by means of computed tomography (CT) angiography (angio-CT) [16]. Magnetic resonance (MR) angiography is also an excellent diagnostic and research tool [17]; however, mainly due to logistic difficulties, it has limited clinical application in the acute setting.

When looking at the CT scan of a patient with Type B AD, there are several features that we need to consider:

- location and size of the primary proximal entry tear (Fig. 2.2): this is crucial to determine the type of dissection. The proximity to the supra-aortic trunks dictates the feasibility of thoracic endovascular aortic repair (TEVAR) with or without surgical de-branching or other means of revascularization;

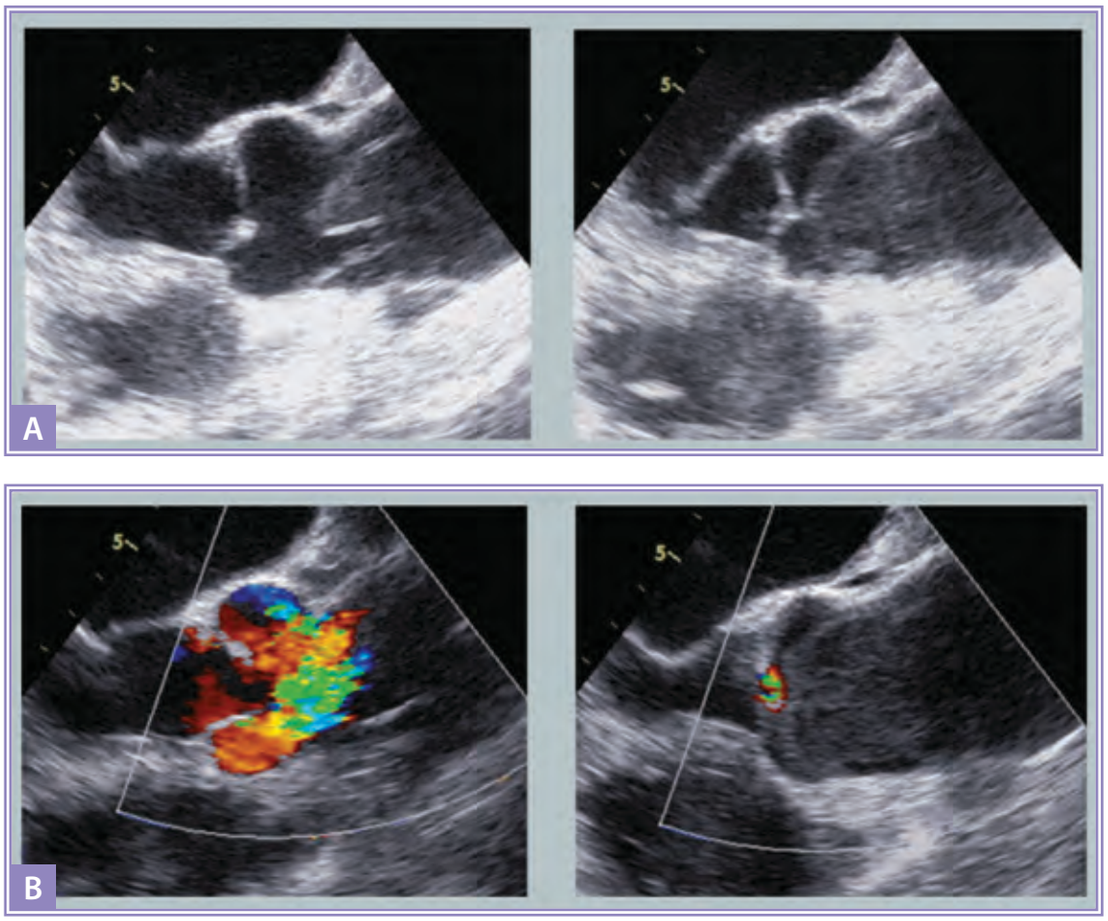

Fig. 2.1 Trans-esophageal long-axis image of a Type A AD. **A**, 2D-mode. The lamella is mobile in the ascending aorta. In the second picture, the false lumen compression reaches the valve and the coronary ostia. **B**, Color flow image of the same phases showing flow alteration.

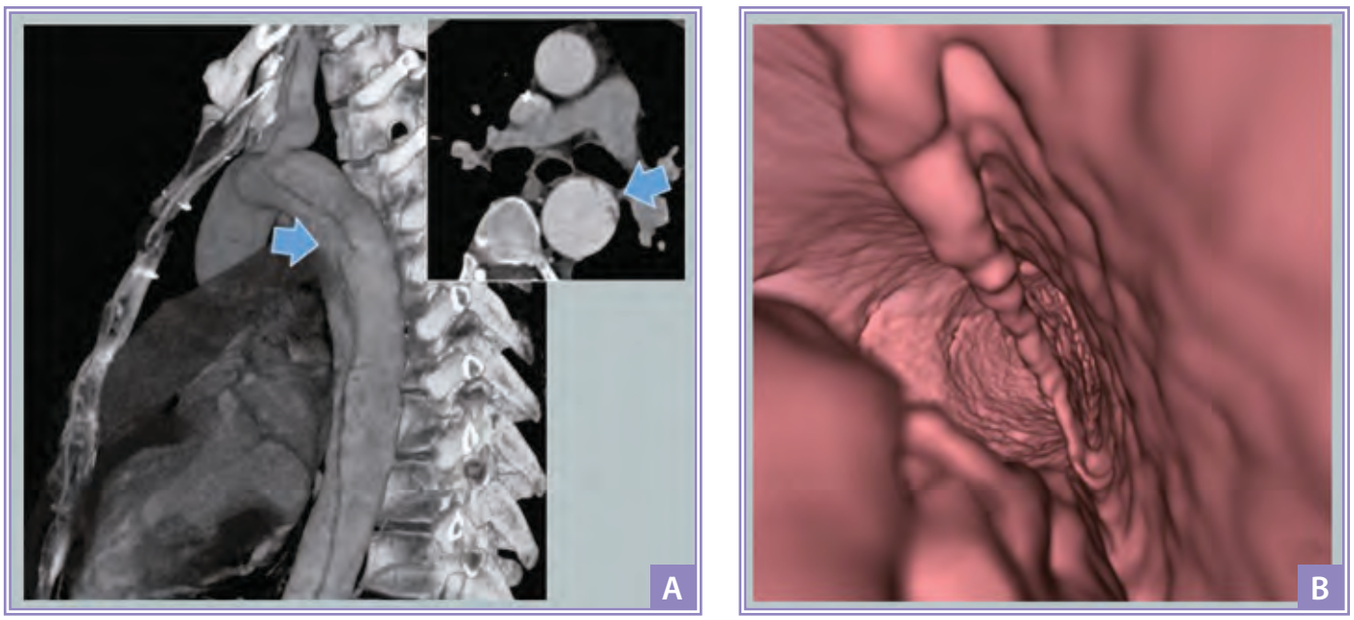

Fig. 2.2 Angio-CT scan of a Type B AD. **A**, Multi-planar reformatting with volume rendering. The proximal entry tear is located in the proximal part of the descending thoracic aorta (blue arrow). In the axial scan, the entry tear and the lamella are clearly visible (blue arrow) with compression of the true lumen. **B**, Virtual angioscopy enables one to determine the longitudinal extension of the entry tear and the two lumina.

- location and size of additional tears: these may be difficult to detect and intraoperative TEE (see Chapter 39), or intravascular ultrasound (IVUS, see Chapter 38) may be more helpful (Fig. 2.3);
- diameter of the non-dissected proximal landing zone: this will be used for sizing the stent-graft (Fig. 2.4);
- total diameter of the aorta and respective diameters of the true and false lumen: a tapered true lumen with significant mismatch between proximal and distal landing zones is common and requires an appropriate strategy (Fig. 2.5);

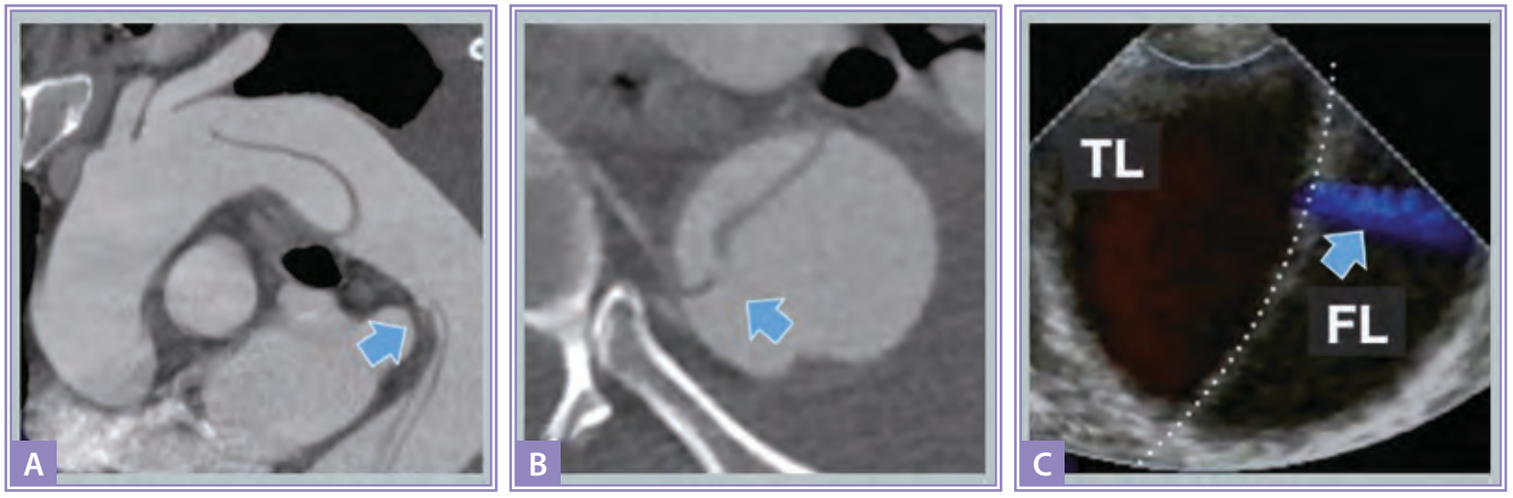

Fig. 2.3 **A**, Angio-CT- Multi-planar reformatting. In the descending aorta, the intimal tear typically originates within a few centimeters of the left subclavian artery because this segment of the aorta is subject to the greatest pressure fluctuations. However, additional tears (blue arrow) can be located anywhere in the thoracoabdominal aorta. **B**, Angio-CT – Axial scan. A small additional tear is visualized. **C**, Intra-operatively, small additional tears can be difficult to detect. TEE is very helpful to visualize them in the descending thoracic aorta.

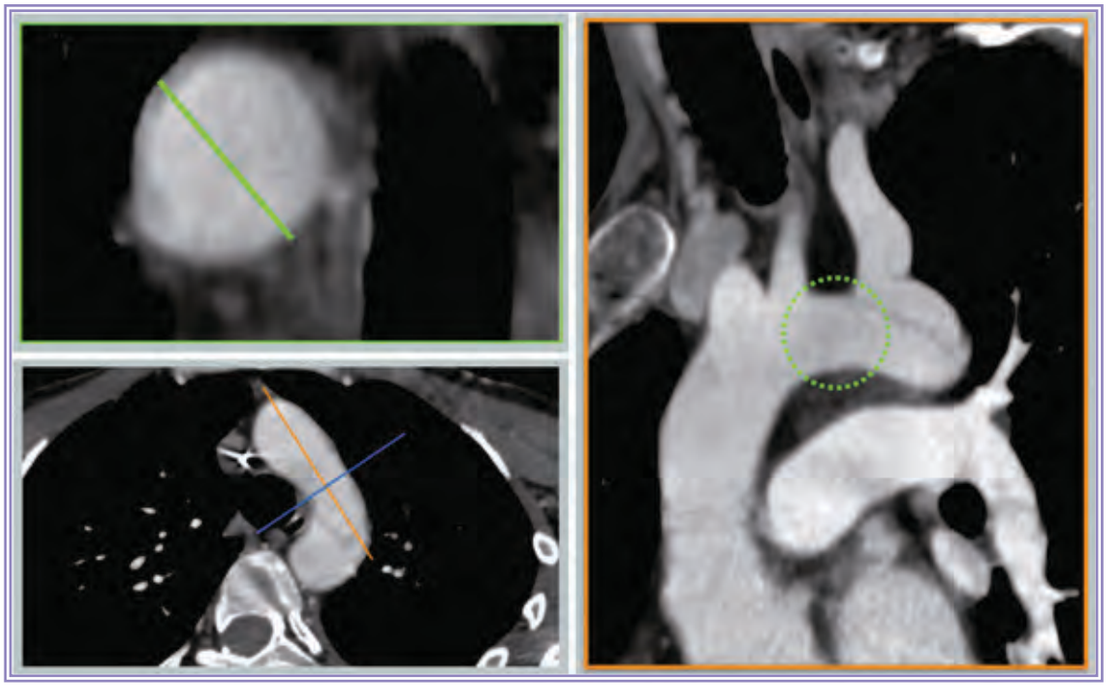

Fig. 2.4 Angio-CT – Multi-planar reformatting. The non-dissected proximal landing zone (green circle) is used for sizing the stent-graft.

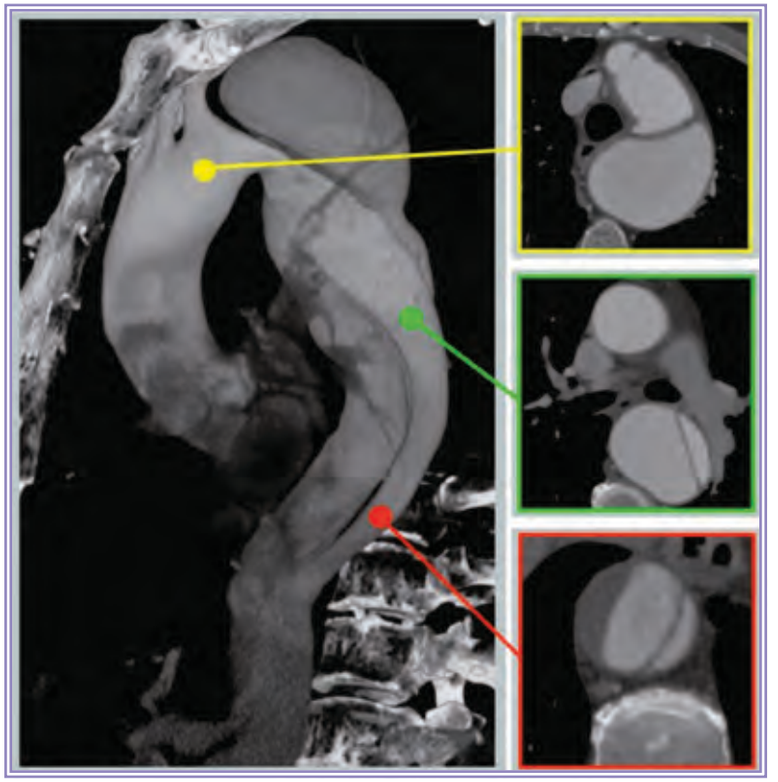

Fig. 2.5 Diameter of the entire aorta and of the true and false lumen in three different parts of the thoracic aorta: aortic arch (yellow), proximal (green) and distal (red) descending thoracic aorta. In the descending thoracic aorta, the true lumen is compressed by the dilated false lumen.

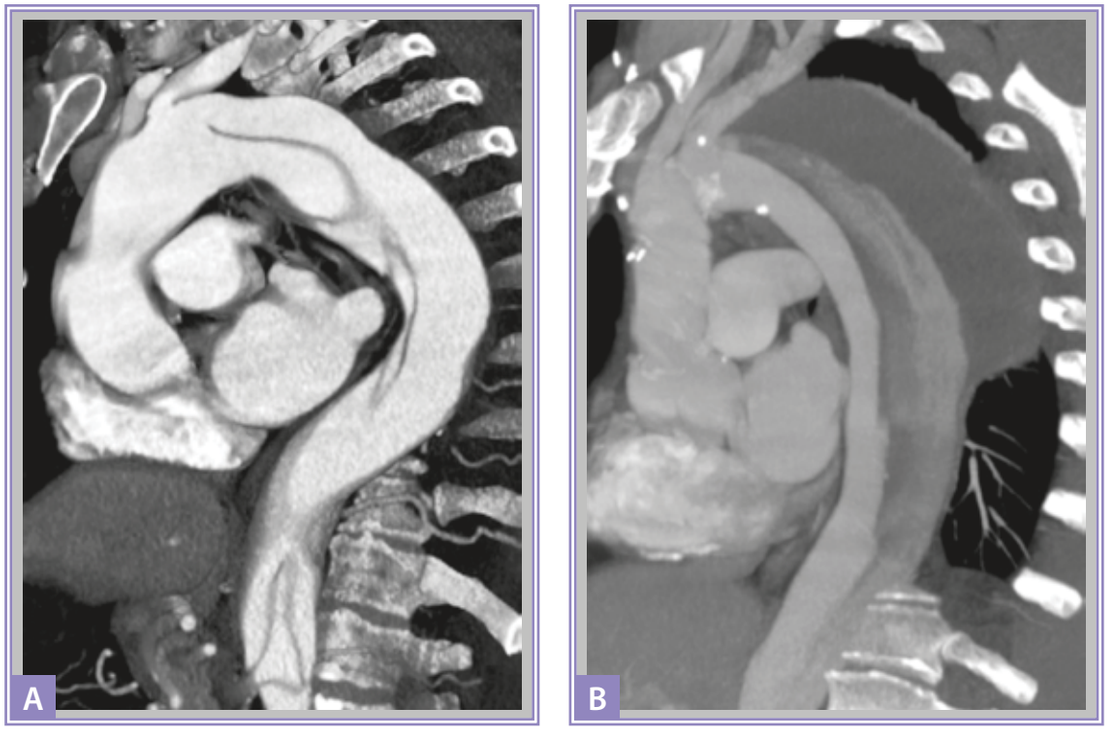

Fig. 2.6 **A**, Acute Type B AD with a thin mobile lamella and no significant thrombosis in the false lumen. **B**, Chronic Type B AD with thickened and stable lamella and aneurysmatic evolution of the partially thrombosed false lumen.

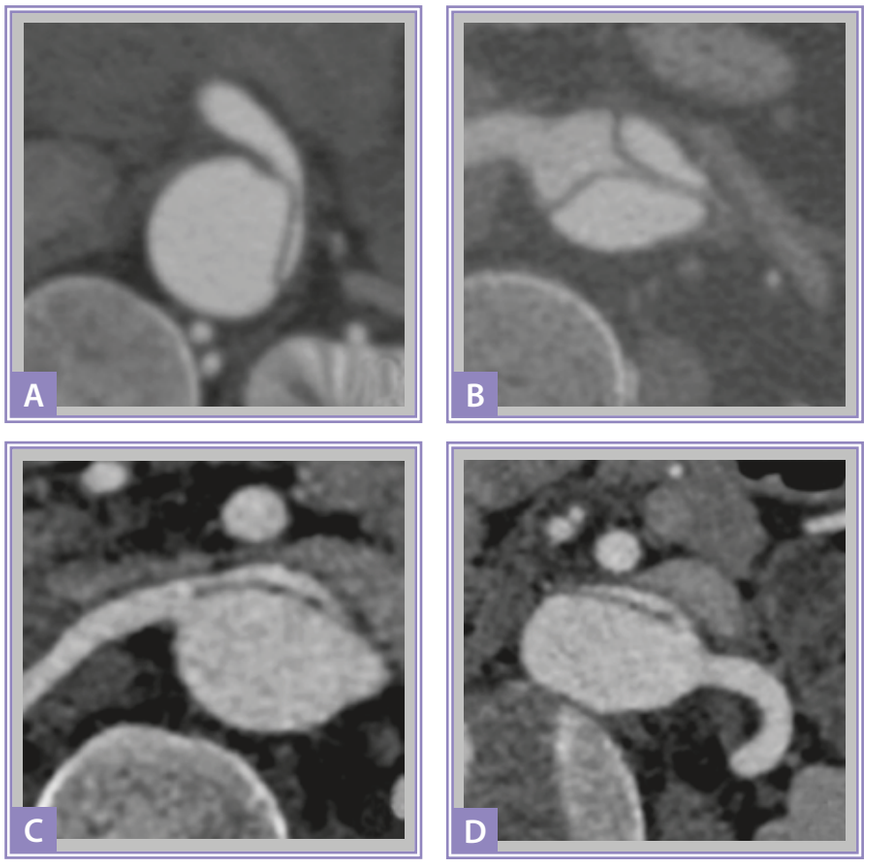

Fig. 2.7 Angio-CT - Axial scans. Various patterns of perfusion of splanchnic and renal arteries during Type B AD are shown. **A**, Dynamic malperfusion of the coeliac trunk. The true lumen is compressed by the false lumen with visceral malperfusion. **B**, Static malperfusion of the left renal artery. The dissection involves the vessel with compression of the true lumen and complete occlusion of the artery. **C**, The right renal artery is partially dissected and arises from both the true and false lumen. **D**, The left renal artery arises from the false lumen.

- degree of thrombosis of the false lumen (Fig. 2.6);
- patency (dissection) of splanchnic and renal arteries and origin from true/false lumen, organ perfusion (Fig. 2.7);
- diameter, patency, and dissection of the iliac arteries (Fig. 2.8);
- additional information is gathered regarding associated organ diseases and variant anatomy of the arteries (particularly the supra-aortic trunks, and the renal and splanchnic arteries), veins and kidneys (single, pelvic, horseshoe, etc.);
- in addition, electrocardiographic (EKG)-gated CT offers the opportunity to evaluate the coronary arteries; this is a crucial step before a thoracic aorta operative procedure, because treatment of critical coronary lesions helps prevent perioperative hemodynamic instability, which is one of the major causes of spinal cord ischemia [18].

Together with the axial scans, oblique multiplanar reconstructions (MPR) may be very useful due to the aortic tortuosity and the non-orthogonal path of the dissection plane. On the other hand, volumetric algorithms such as maximum intensity projections (MIP) should be used with caution since much of the intra-aortic detail, such as the intimal lamella or thrombus, could be lost.

In conclusion, Type B AD, even in an acute setting, requires a thorough CT diagnostic evaluation to disclose all the anatomic and pathologic details needed to plan the most appropriate treatment.

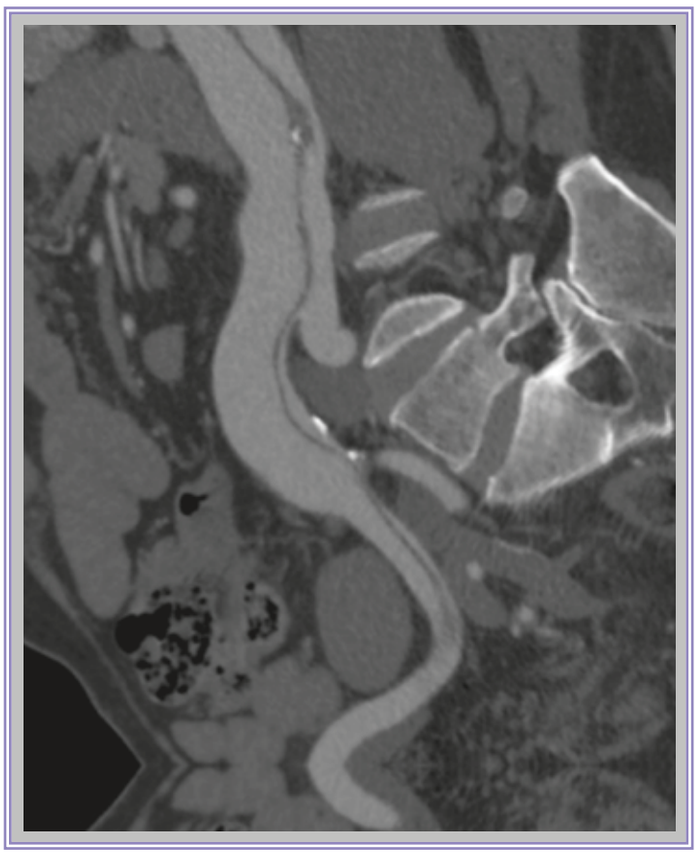

Fig. 2.8 Angio-CT – Multi-planar oblique reformatting showing involvement of the iliac arteries. This Type B AD involves the infra-renal aorta, the right common iliac artery and the proximal portion of the external iliac artery.

2.4 Pathophysiology and principles of therapy

Outcomes today regarding the stent-graft repair of aneurysms of the descending thoracic aorta are encouraging. Stent-grafts, in fact, were initially designed and approved for use in aneurysmal disease, where the technical and clinical goals are clear: to exclude the aneurysmal sac from arterial pressurization and thus reduce or, hopefully, abolish the risk of rupture. However, aneurysms currently represent only one-half or less of patients with indications for Thoracic EndoVascular Aortic Repair (TEVAR) [19]. Other indications include: traumatic injury, aorto-esophageal or aorto-bronchial *fistulae*, penetrating aortic ulcer (PAU), intramural hematoma (IMH) and, last but not least, complicated Type B AD. The goals of TEVAR for acute and sub-acute Type B AD are far less clear than for aneurysms. Remodeling and complete healing of the dissected aortic wall is certainly a desirable outcome but it is actually attained in only a fraction of cases [20].

The initial event of Type B AD is usually an intimal tear that allows blood to break into the aortic wall layers with creation of two lumina: a false lumen (FL) that frequently enlarges and a true lumen (TL) that may contract or even collapse. Blood flows in both lumina, as may be observed with TEE; however, malperfusion can occur as a consequence of static or dynamic events. Moreover, the reduced strength of the dissected aortic wall makes it more prone to progressive dilatation and eventually rupture (Fig. 2.9).

Endovascular treatment of acute and sub-acute Type B AD aims at excluding the FL from the circulation, avoiding malperfusion and preventing aortic enlargement and rupture. This may be obtained with both regular stent-grafts used to cover the intimal tears and with bare stents specifically designed to treat TL collapse and malperfusion (see Chapter 25).

From a technical point of view, this may be achieved by a stent-graft that:

1. obliterates the primary entry tear;
2. re-directs the blood flow into the TL; and
3. produces low turbulent flow in the FL and eventually promotes thrombosis of the FL (Fig. 2.10).

Another consequence of re-directing blood flow into the TL and decompressing the FL is that a compressed or collapsed TL may expand, thus preventing or relieving dynamic malperfusion.

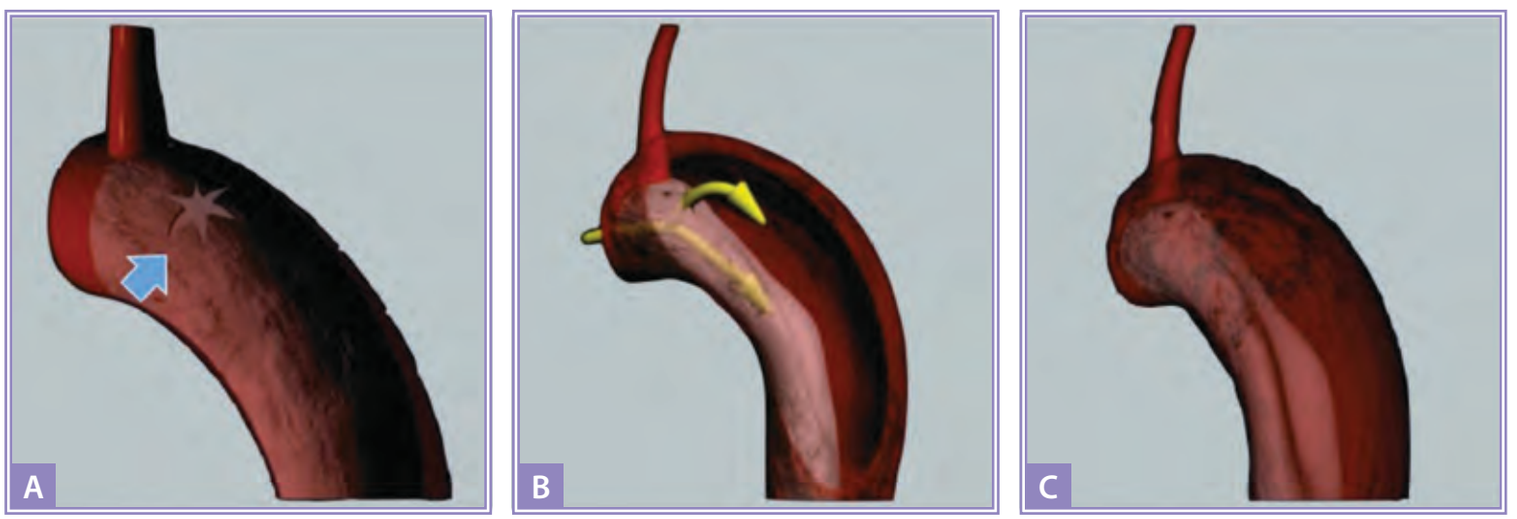

Fig. 2.9 Pathophysiology of Type B AD. **A**, The proximal entry tear (blue arrow) allows blood to break into the aortic wall layers with creation of two lumina. **B**-**C**, The false lumen enlarges and the true lumen collapses.

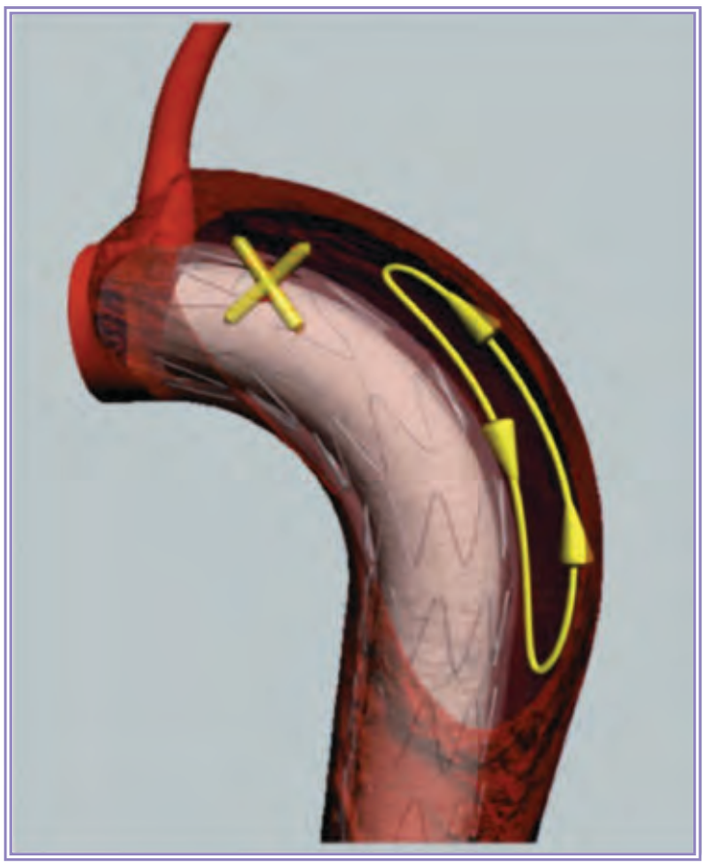

Fig. 2.10 Endovascular exclusion. The fabric covered stent-graft obliterates the primary entry tear, redirects the blood flow into the true lumen and produces low turbulent flow in the false lumen promoting thrombosis.

Possible additional problems include:

1. retrograde flow into the FL from the left subclavian artery; this may be treated percutaneously with a plug;
2. presence of high-flow secondary tears in the thoracic aorta; these are assessed intra-operatively with TEE, and may be treated with additional stent-grafts; this, however, has to be balanced against the increased risk of spinal cord ischemia;
3. additional secondary tears in the abdominal aorta (often adjacent to the splanchnic vessels) are commonplace and are usually left untreated;
4. flapping movements of the intimal lamella may prevent thrombosis of the FL;
5. a collapsed TL may fail to adequately expand with persistent distal malperfusion.

The two latter problems can be addressed by the Provisional Extension To Induce Complete Attachment (PETTICOAT) technique.

Other more complex techniques are nowadays available for specific problems and will be addressed later in dedicated chapters.

From a more clinical perspective, an endovascular approach is accepted by most authors for the treatment of acute and sub-acute Type B AD. However, for uncomplicated cases, optimal medical treatment is still considered the treatment of choice by most authors. This open issue will be discussed in Chapters 9.

In Type B AD, bowel malperfusion is one of the most difficult complications to interpret when only soft and non-specific symptoms are present; waiting for hard signs and symptoms,

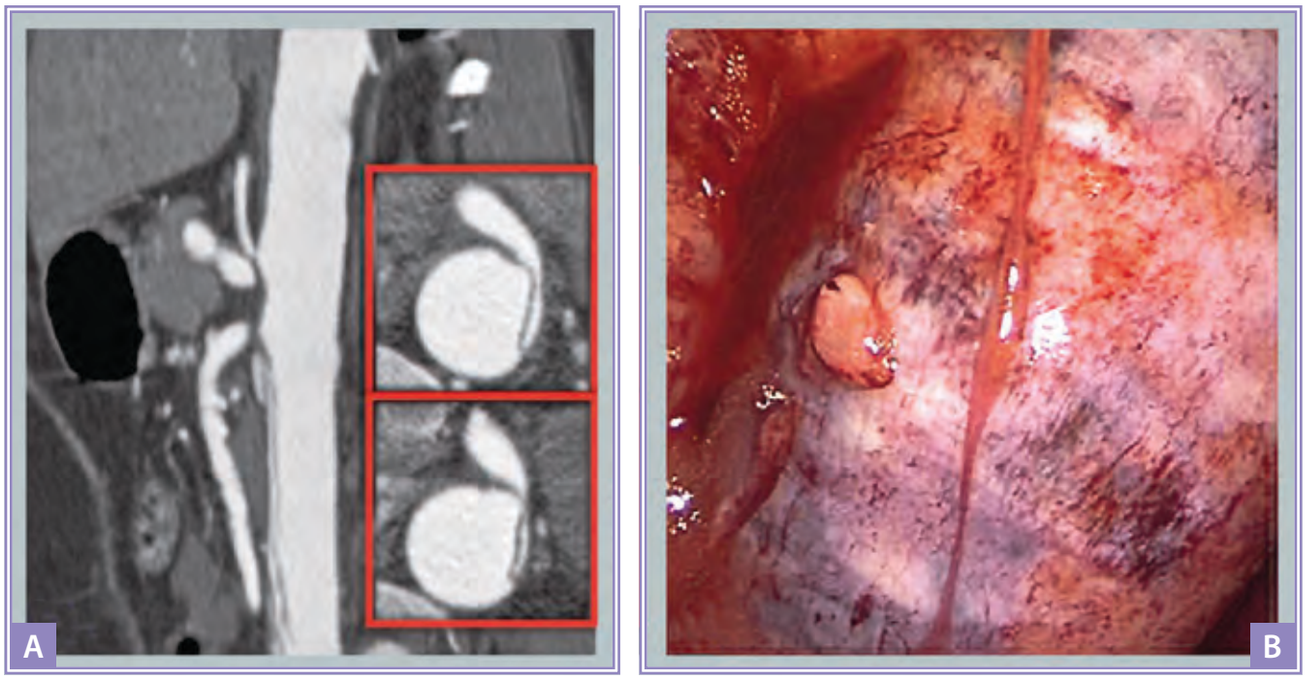

Fig. 2.11 **A**, Pre-operative Angio-CT showing dynamic malperfusion of both the coeliac trunk and superior mesenteric artery. **B**, Diagnostic laparoscopy demonstrates small bowel ischemia with sub-serosal hemorrhage and pallor.

however, may result in fatal bowel necrosis (Fig. 2.11). After an initial positive experience, in the last four years we have routinely used laparoscopy both preoperatively in patients with unclear abdominal symptoms and after TEVAR in all patients with unclear angiographic findings related to intestinal perfusion.

In conclusion, TEVAR has been shown to have a satisfactory degree of safety and efficacy in most acute and sub-acute complicated Type B AD cases, while the PETTICOAT technique may be used in selected cases to promote TL expansion and relieve malperfusion.

Chronic Type B AD with aneurysmal dilatation clearly requires treatment in reasonably fit subjects, when a critical diameter threshold is reached. Open surgery offers very good results when performed by experienced operators in high volume centers (see also Chapter 31). Recently, however, endovascular options that aim at total exclusion of the FL with complete sealing of the TL have also been proposed, and these will be discussed in Chapter 28.

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23

The PETTICOAT technique for endovascular treatment of Type B aortic dissection

Luca Bertoglio, Germano Melissano, Enrico Rinaldi, Andrea L. Kahlberg, Sthefano Atique Gabriel and Roberto Chiesa

23.1 Introduction

Approximately 25% of patients presenting with acute Type B aortic dissection (AD) meet the definition for complicated status (impending rupture, end-organ malperfusion, refractory hypertension or pain) with an associated higher 30-day mortality rate [1]. An obstruction of flow of one or more aortic side branches is present in one-third of complicated patients who are consequently affected by the so-called "malperfusion syndrome". This syndrome is characterized by a nearly three-fold increased likelihood of in-hospital mortality in acute Type B AD [2]: regardless of the treatment success, the presence of both mesenteric ischemia and limb ischemia has been associated to a poor prognosis [2, 3].

Identifying the mechanisms of branch compromise is a critical step in planning effective treatment strategies. Williams et al. [4] introduced the radiological classification of malperfusion mechanisms based on branch-vessel involvement, identifying two types of obstruction:

- dynamic obstruction (Fig. 23.1): the compressed true lumen (TL) is unable to provide adequate flow because the aortic flap prolapses across the branch-vessel origin covering it like a curtain but without entering into it. Because of the constantly changing position of the intimal flap, particularly in the acute phase, these obstructions can be total or subtotal with persistent or intermittent features. This is the most common mechanism of branch compromise and it is responsible for some 80% of malperfusion syndromes [5];
- static obstruction (Fig. 23.2): the dissection flap intersects or enters the branch-vessel origin propagating into the vessel wall. Typically, flap progression into a branch is tolerated because a distal re-entry develops allowing perfusion of the distal vessel from both the lumens (static malperfusion with re-entry). However, a potential profound malperfusion exists when the false lumen (FL) within the branch does not have a re-entry point in the vessel (static malperfusion with no re-entry). As a result, the FL is a blind cul-de-sac which enlarges and compresses the TL. In all cases of static malperfusion, the obstruction is unlikely to be resolved with the restoration of aortic TL flow alone [6, 7].

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G. Melissano - R. Chiesa (eds), Aortic Dissection patients true stories and the innovations that saved their lives

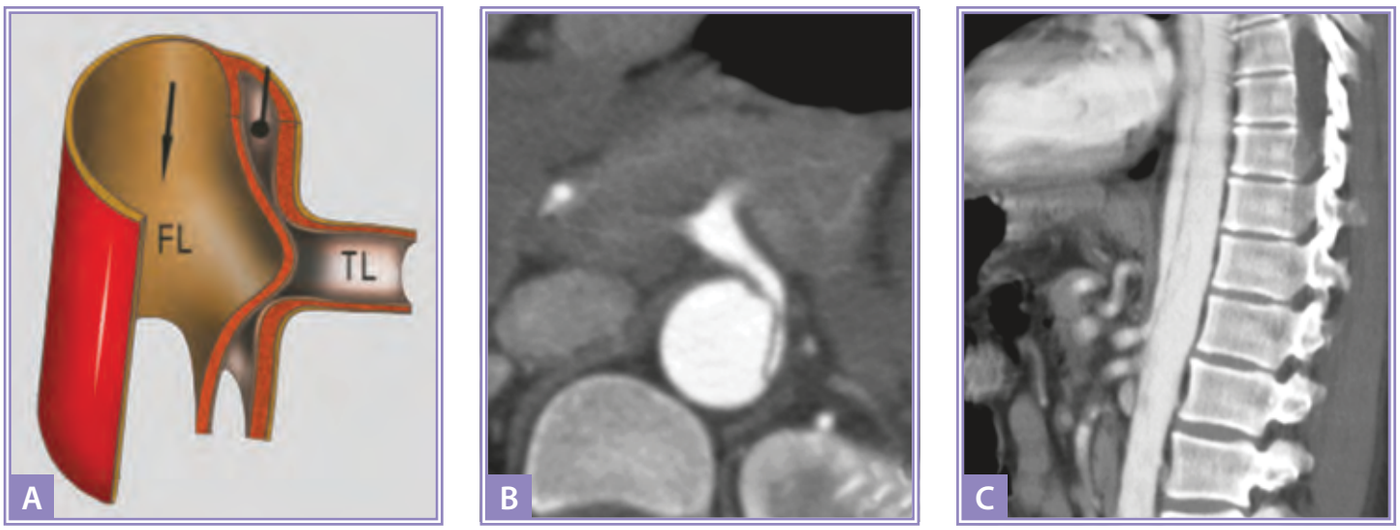

Fig. 23.1 Dynamic malperfusion (**A**): the true lumen (TL) is compressed by the false lumen (FL) and is unable to provide adequate volume flow to the aortic branch. In this example: the axial computed tomography (CT) scan (**B**) demonstrates the collapsed TL due to the FL compression, and the multi-planar reformatting (MPR) view (**C**) shows the extension of the TL collapse to the whole thoracoabdominal aorta.

Nowadays, dynamic obstruction is often managed by means of stent-graft covering of the entry tear, with the following goals [8]:

- covering of the proximal entry tear and creating a seal to stop the flow of blood from entering the FL and prevent the transmission of systemic pressure across the major intimal defect;
- redirecting flow into the TL with FL decompression which may relieve dynamic obstruction of branches supplied by the collapsed TL;
- promoting thrombosis and remodelling of the thoracic FL with the aim of reducing long-term aortic related reinterventions.

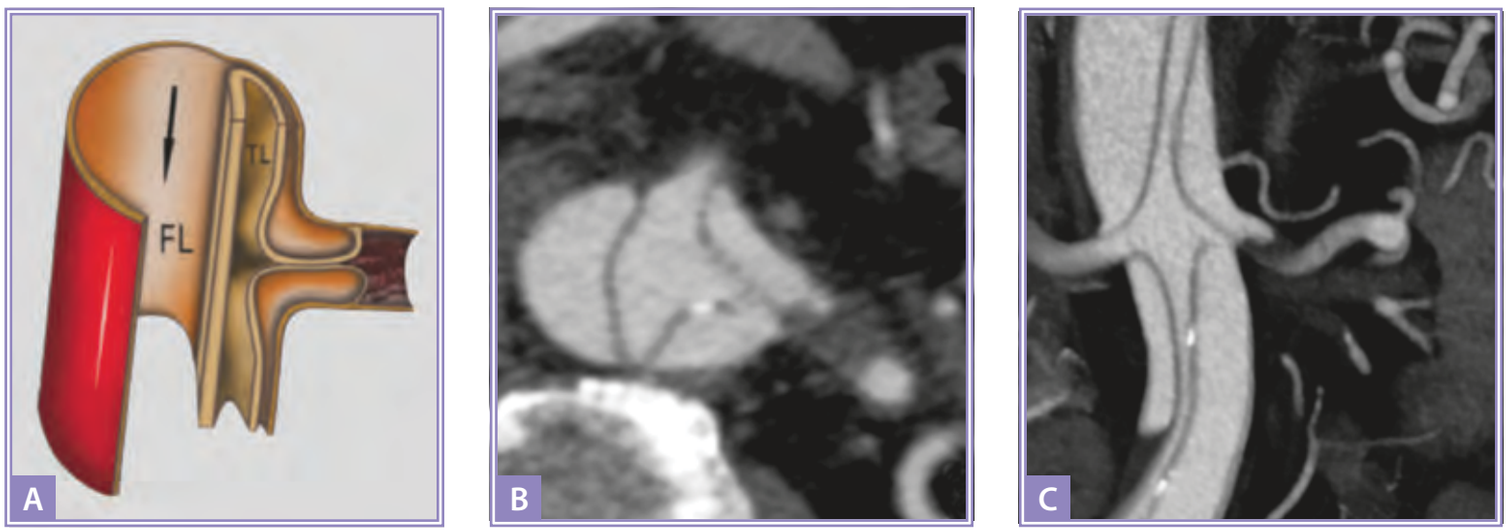

Fig. 23.2 Static malperfusion (**A**): the dissection enters directly and obstructs the ostium of a branch vessel; organ injury can occur as a result of thrombosis or hypoperfusion of the involved vessel. In this example: the axial computed tomography (CT) scan demonstrates the dissection entering into the ostium of the left renal artery (**B**) and the compression of the FL without re-entry causes the partial thrombosis at the ostium of the vessel with end-organ malperfusion (**C**).

Although emergent Thoracic EndoVascular Aortic Repair (TEVAR) for patients with complicated acute Type B AD has shown acceptable 1-year mortality and morbidity (estimated mortality: 10.8%) [9], the fate of the distal thoracoabdominal aorta might remain unaddressed. Distal re-entry tears may sustain distal pressurization of the FL with TL collapse and unsolved dynamic malperfusion. Moreover, the FL may fail to thrombose, thus jeopardizing the aortic remodelling with the consequent risk of an aneurysmal degeneration. Furthermore, the perfusion patterns of visceral and renal branches could change after TEVAR in an unpredictable way leading to persistent or even new malperfusion patterns. For these reasons, additional interventional procedures might be necessary to solve persistent dynamic malperfusion after the initial TEVAR [10].

Some authors have proposed endovascular fenestration alone or in combination with TEVAR aimed at artificially creating a distal abdominal re-entry channel in order to equalize the pressure between the TL and FL [8, 10]. The subsequent FL decompression may relieve TL dynamic obstruction of the aorta and its branch vessels. However, fenestrations may create unpredictable alterations in intimal flap anatomy and flow dynamics. In addition, the effect of fenestration on the long-term outcome of FL expansion in patients with distal dissections is unknown because the FL remains pressurized and therefore at risk of continued progression to aneurysm.

Hence, other solutions have been proposed to solve persistent dynamic malperfusion after TEVAR and to enhance the remodelling of the thoracoabdominal aorta without disrupting the dissection lamellae architecture. Today, aortic scaffolding with bare metal stents, known as the PETTICOAT technique, has been added to the armamentarium of vascular surgeons and it is claimed to improve the short and mid-term results of complicated acute Type B AD.

23.2 The PETTICOAT concept

In 2003, Ito et al. [11] first reported the use of a Gianturco bare Z-stent released in the distal abdominal aorta in order to re-expand a collapsed TL and to solve a visceral dynamic malperfusion. Two years later, Mossop et al. [12] performed a staged endovascular treatment for complicated Type B AD using, first, a standard TEVAR to close the proximal entry tear and, 1 week later, a bare Z-stent deployed in the distal aorta to re-expand the distal collapsed TL. They described the procedure with the acronym of STABLE (Staged ThoracoAbdominal and Branch vesseL Endoluminal repair). In 2006, Nienaber et al. [13] published the first series of 12 patients treated with a proximal TEVAR combined with the use of distal bare stents deployed in the TL in order to help its re-expansion. The authors proposed the acronym of PETTICOAT (Provisional ExTension To Induce COmplete ATtachment) to describe the procedure and, nowadays, this is the term most commonly used to describe it.

The PETTICOAT technique involves the closure of the primary entry tear, performed with standard TEVAR, in order to re-direct blood flow into the TL and depressurize the FL. The expected consequence of the proximal TEVAR is that the thoracic and abdominal TL will tend to expand and the FL to shrink. Usually, thoracic FL thrombosis is obtained at least in the covered stented area and further shrinkage and positive remodelling is achieved. However, in spite of successful deployment of the standard stent graft over the proximal entry tear, the TL distal to the stent graft may fail to dilate satisfactorily with possible persistent dynamic malperfusion. This problem can be addressed by deploying bare metal stents distally to a standard stent graft down into the thoracic and abdominal TL (Fig. 23.3). Not only will the described PETTICOAT technique re-expand the thoracoabdominal TL solving the dynamic malperfusion, but also it will, possibly, address other unmet goals of TEVAR in acute Type B AD.

The intimal lamella distal to a standard TEVAR undergoes continuous movements with each systole and diastole, which keeps the blood in motion in the FL preventing thrombosis occurring at this level. With the introduction of a bare stent beyond the stent graft and down

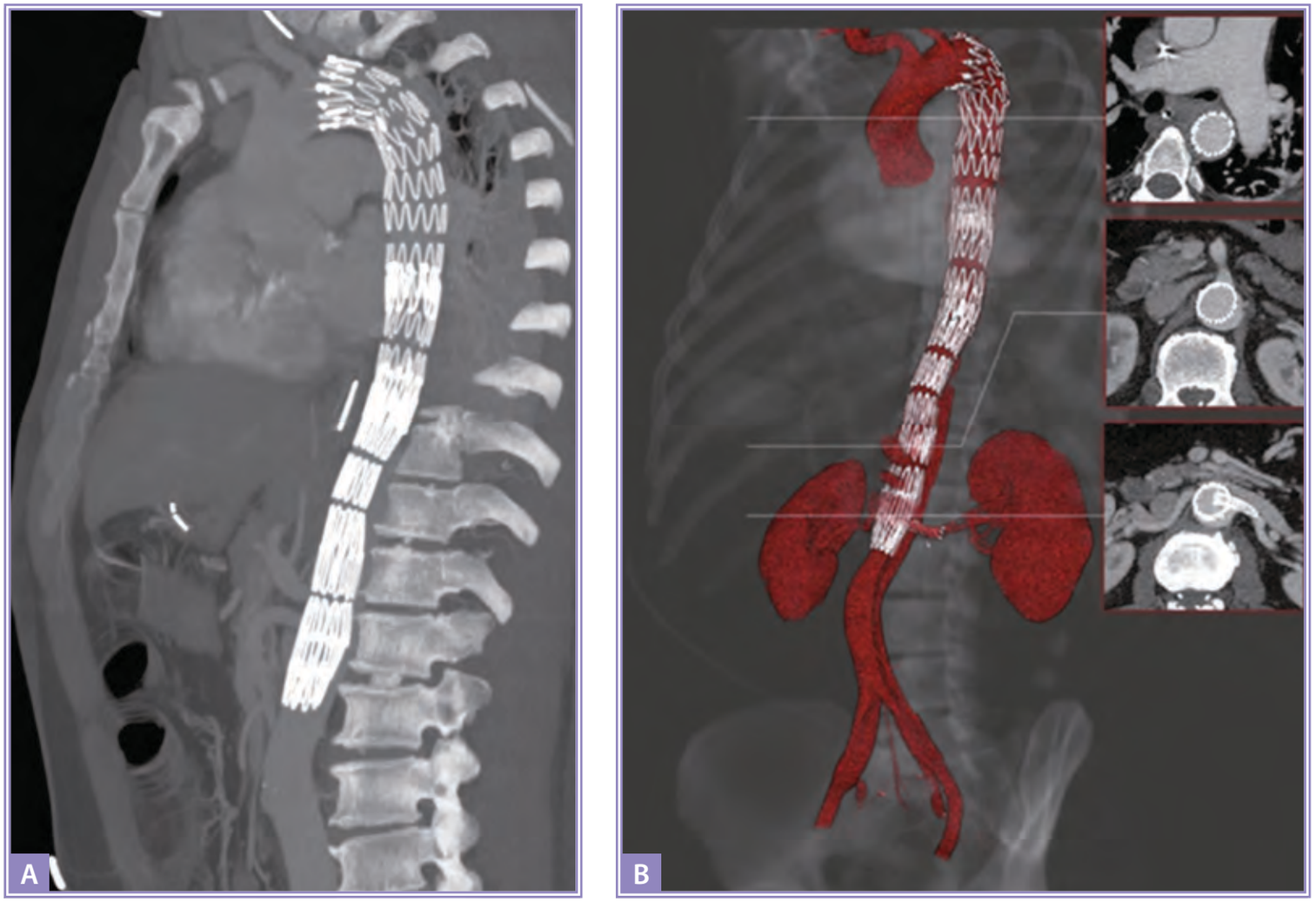

Fig. 23.3 The Zenith® Dissection Endovascular System (ZDES) proposed by Cook (Cook Medical, Bloomington, IN). **A**, The system employed to treat a Type B AD with stent-graft (TX2® graft) coverage of the proximal entry tear and distal extension with two bare stents (Dissection Stent) in order to expand and stabilize the true lumen (TL). **B**, Postoperative angio-CT scan (3D-volume rendering): endovascular treatment of aortic Type B AD with proximal entry tear coverage with a Cook TX2® stent graft and distal extension with two dissection stents (ZDES). Axial scans show exclusion of the thoracic false lumen (FL) and complete re-expansion of the TL in the thoracic and abdominal segments with restoration of perfusion of the right kidney (left renal stenting was performed in this case to perfuse the left renal artery from the re-expanded TL).

to the dissected distal aorta, it is possible both to support the TL and to fix the lamella. The full expansion of the distal TL associated with the stabilization of the dissecting lamella can promote FL thrombosis and remodelling. Moreover, the possibility to use an uncovered stent in the distal thoracic and abdominal aorta reduces the need for extensive aortic coverage with traditional stent grafts, limiting the risk of spinal cord ischemia.

With regard to visceral and renal perfusion, the PETTICOAT technique is helpful not only in solving dynamic malperfusion without interfering with visceral and renal perfusion but also in addressing static obstruction. Static obstruction of aortic branch vessels can persist despite depressurization of the FL and elimination of dynamic obstruction, because of the extension of the FL into branch-vessel ostia. PETTICOAT facilitates subsequent selective stenting of malperfused aortic branches because the expansion of the TL allows realignment of the intimal ostia of dissected branches (Fig. 23.4). The open design of the bare stent mesh allows relatively easy access and cannulation of the aortic branches as well as bridging of the TL to target

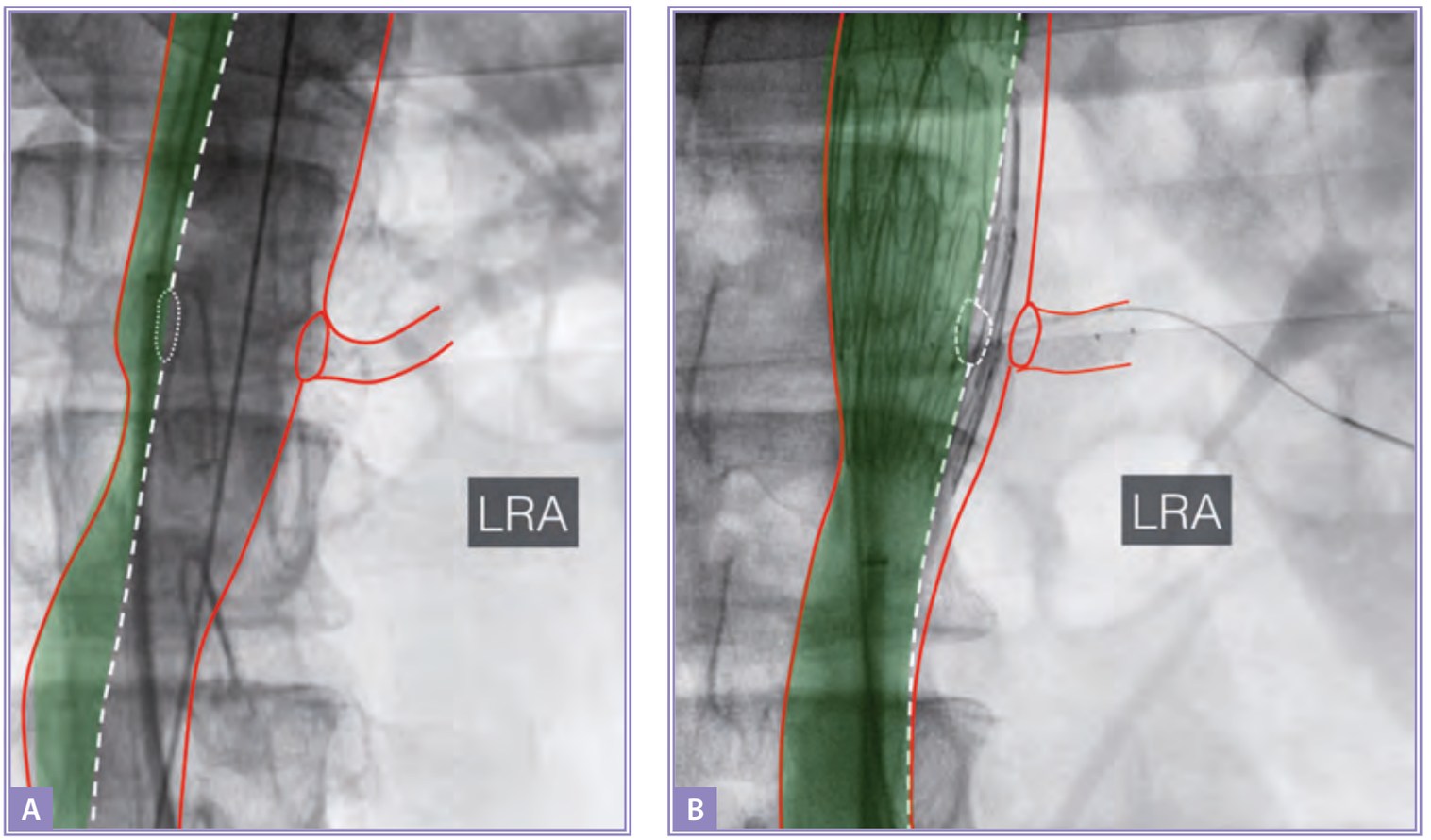

Fig. 23.4 The bare stent-induced true lumen (TL) re-expansion allows realignment of the intimal ostia of dissected branches that could be subsequently stented in order to relieve residual malperfusion. In this case, the left renal artery (LRA) ostium was realigned and then selectively stented with a self-expanding stent. Please note the shorter distance of dissecting lamella (white dotted line) from the renal artery ostium before (**A**) and after (**B**) the deployment of the bare stent into the TL.

vessels with a self-expanding stent (Fig. 23.5). To enhance complete thrombosis of the FL, this bridge stenting can be performed with a covered stent graft if a distal entry tear has to be sealed into the target vessel, as described by Bel et al. [14]. Interestingly, an ex vivo study demonstrated a significant pressure gradient drop (>15 mmHg) across visceral and renal arteries supplied by the FL after PETTICOAT (in 54.5% of cases). These experimental findings might support extensive stenting of visceral or renal vessels arising from the FL after PETTICOAT to reconnect the vessel to the TL and perfectly realign the ostium with the dissecting lamella [15].

Notably, the first published series of PETTICOAT was performed with an off-label use of different self-expanding stents aimed to treat either vena cava or aortic occlusive pathology [13]. In the last decade, new dedicated bare stents have been designed and approved with CE mark to treat AD. The largest series published to date employed two different commercially available stents:

- Zenith® Dissection Endovascular Stent (Cook Medical, Bloomington, IN): this became available in 2005 as a custom-made device. At present, it is commercially distributed with the CE mark obtained in 2010. It is made of very low radial force self-expandable stainless steel Z stents sewn in series. The device is available in two diameters (36 and 46 mm) and three lengths (82, 123 and 164 mm). The delivery system is 20 Fr (outer diameter). Since two diameters of the bare stent component are available (36 mm and 46 mm), the size is chosen according to the diameter of the proximal covered stent graft. A diameter of 36 mm should

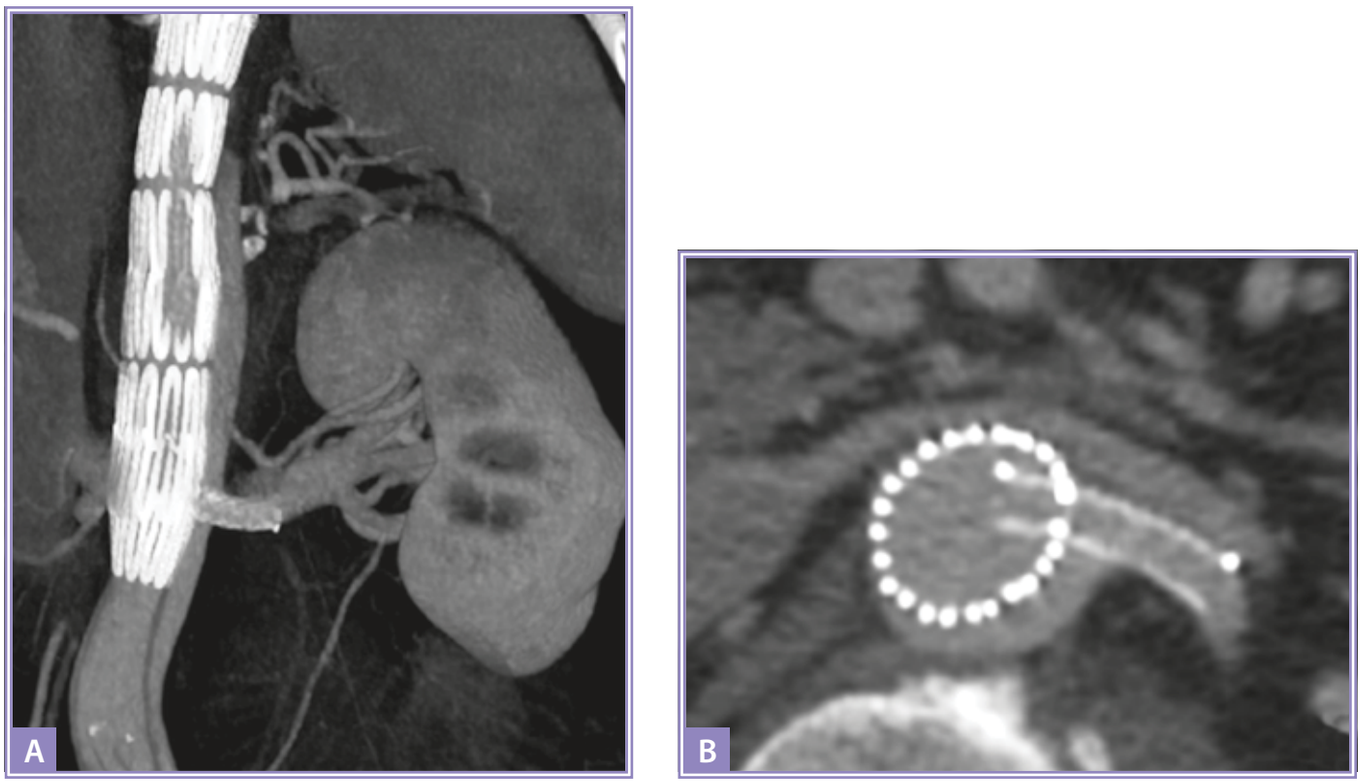

Fig. 23.5 **A**, **B**, Multi-planar reformatting (MPR) of a Zenith® Dissection Endovascular Stent (Cook Medical, Bloomington, IN) deployed to relieve dynamic malperfusion; then the left renal artery was stented through the PETTICOAT to solve residual static malperfusion with a self-expanding stent.

be combined to proximal stent grafts <36 mm, and a diameter of 46 mm to stent grafts >36 mm. The device has been recently modified by the manufacturer with a new configuration. Compared to the previous one, a suture has been added between each peak of the first and second stent of the proximal and distal ends of the device, changing the configuration with a peak-to-peak design. This modification is intended to improve the apposition to the aortic wall by improving the columnar support of the proximal and distal ends of the stent, avoiding the misalignment that might occur during deployment or during inadvertent engagement of the distal loops [16]. A new nitinol version with the same design recently received the CE mark and will soon be commercialized;

- Sinus-XL stent (Optimed, Ettlingen, Germany): this is a self-expanding nitinol stent system. It is commercially distributed with the CE mark obtained in 2007. It has a closed cell design and it is available in different diameters from 16 to 34 mm and different lengths from 30 to 100 mm. The delivery system is 10 Fr (outer diameter).

Anecdotal reports have been published regarding other bare stents, such as the E-xl (JOTEC GmbH, Hechingen, Germany) [17] and the Fortress stent (Curative Medical Devices Inc., Dresden, Germany) [13].

23.3 PETTICOAT literature review

A literature review search was conducted to identify all published studies reporting the combined use of a proximal stent graft with a distal bare metal stent for the management of AD.

Table 23.1 Literature studies

Author [ref]	Year	Study design*	No.	Stanford classification (%)		Malperfusion (%)	Onset (%)		
				Type A	Type B		Acute	Sub-acute	Chronic
Conzelmann et al. [25]	2013	R	13	100%	–	100%	100%	–	–
Hsu et al. [26]	2016	R	9	100%	–	56%	100%	–	–
Nienaber et al. [13]	2006	R	12	16%	84%	0%	16%	42%	42%
Hofferberth et al. [18]	2012	R	40	40%	60%	63%	75%	–	25%
Melissano et al. [19]	2012	P	25	–	100%	20%	20%	52%	28%
Liu et al. [20]	2013	R	33	–	100%	–	100%	–	–
Feng et al. [27]	2013	R	154	–	100%	–	86	–	68
Lombardi et al. [21]	2014	P,T	86	–	100%	65%	64%	36%	–
Alsac et al. [22]	2014	P	15	–	100%	100%	100%	–	–
He et al. [23]	2015	R	35	–	100%	34%	100%	–	–
Kische et al. [24]	2015	P	17	–	100%	71%	71%	29%	–
Overall			439	40 (9.1%)	399 (90.9%)	138 (48.4%)	290 (66.1%)	59 (13.4%)	90 (20.5%)

* R, retrospective; P, prospective; T, prospective trial.

MEDLINE and SCOPUS databases were searched for papers published in English between January 2006 and June 2016, using as keywords: "dissection", "bare stent", "thoracic", "endovascular", and "PETTICOAT". In addition, references to selected studies were screened manually for further identification of relevant studies. Studies were considered for review on the basis of the following criteria: they had to include more than 5 cases, provide full information on the type of dissection treated and the onset of the dissection, as well as report clinical and technical outcomes. Studies containing duplicate (partial or complete) data were excluded and the most recent data coming from the same research group were used. Eleven studies were analysed after literature search (Table 23.1) [13, 18-27].

23.3.1 Short-term results

Notwithstanding 439 patients received a so-called "PETTICOAT" procedure, the materials used, type of dissection treated and time interval from onset of dissection were too heterogeneous to be able to present grouped short-term clinical results. For these reasons, two studies [25, 26] reporting outcomes of 22 PETTICOAT procedures in combination with open surgical repair for Type A AD were excluded. Moreover, two studies [13, 18] presenting cases treated with PETTICOAT for both Type A and B AD were excluded because of the impossibility to extract only the data regarding the treatment of the Type B AD cohort. Finally, three studies were excluded [20, 23, 27] in which a short (<100 mm) bare stent was predeployed into the distal thoracic neck without reaching the visceral and renal artery in order to avoid stent graft-induced distal entry tear.

Table 23.2 Studies reporting the PETTICOAT procedure for acute/sub-cute Type B AD

Outcome	Melissano et al. [19] (2012)	Lombardi et al. [21] (2014)	Alsac et al. [22] (2014)	Kische et al. [24] (2015)	Overall
Patient (no.)	25	86	15	17	143
Device used	ZDES	ZDES	ZDES	ZDES	ZDES
30-day mortality	0 (0%)	4 (4.7%)	0 (0%)	3 (17.6%)	7 (4.9%)
Type I endoleaks	2 (8.0%)	1 (1.2%)	0 (0%)	4 (23.5%)	7 (4.9%)
30-day clinical success	23 (92%)	81 (94.2%)	15 (100%)	10 (58.8%)	129 (90.2%)
Surgical conversion	0 (0%)	0 (0%)	0 (0%)	na	0 (0%)
Retrograde dissection	0 (0%)	7 (8.1%)	0 (0%)	na	7 (5.6%)
Stroke	0 (0%)	6 (7.0%)	1 (6.7%)	na	7 (5.6%)
Any spinal cord ischemia	1 (4.0%)	2 (2.3%)	1 (6.7%)	na	4 (3.2%)
Permanent paraplegia or paraparesis	0 (0%)	1 (1.2%)	0 (0%)	na	1 (0.8%)
Renal failure	5 (25%)	7 (8.1%)	2 (13.3%)	na	14 (11.1%)
Any end-organ resection	0 (0%)	5 (5.8%)	1 (6.7%)	na	6 (4.8%)
1-year survival	92.0%	88.3%	93.3%	80%	–

ZDES, Zenith® Dissection Endovascular Stent; na, not available.

Perioperative (30-day results) of the four remaining papers reporting the PETTICOAT procedure for treatment of Type B AD have been summarized in Table 23.2. All 143 patients were treated with the Zenith® Dissection Endovascular Stent (Cook Medical, Bloomington, IN) for Type B AD either in the acute (89 cases within the first 14 days) or sub-acute phase (54 cases after the first 14 days but before 2 months). The pooled data demonstrated that the procedure is feasible and safe with an overall 30-day mortality rate of 4.9% and a clinical success rate of 90.2%. Notably, 30-day clinical events were recorded in 28.7% of the cases and no different outcomes were recorded between the acute and subacute cases in the two studies presenting data regarding a sub-acute treatment of Type B AD [18-20]. One-year survival ranged from 80 to 93.3%.

23.3.2 TEVAR + PETTICOAT versus TEVAR alone

Six out of the 11 papers proposed a clinical comparison between TEVAR alone (TEVAR) and TEVAR + PETTICOAT (PETTICOAT) in the same dissection setting [18, 23, 24, 26-28]. Although none of these comparative studies were randomized and the decision to employ a distal bare stent was either left to the surgeon or determined by the presence of malperfusion or TL collapse, they do give some insight into the possible benefits of this technique.

Kische et al. [24] did not observe any significant differences in the clinical success rate; however, they did not report separately the morbidity rates in the two different groups.

Hofferberth et al. [18] compared TEVAR versus PETTICOAT in a heterogeneous cohort of patients with both Type A and B AD and with acute and chronic onset, although the incidence of visceral and limb malperfusion in the two groups was similar. No differences were observed in terms of clinical success between the two methods. However, a significantly lower incidence of bowel infarction was recorded (0 vs. 17%) as well as a lower need for late distal aortic reinterventions (0 vs. 19%).

Sobocinsky et al. [28] compared the outcomes of the first 39 patients enrolled in the STABLE trial [21] with 45 cases treated with a standard TEVAR in three European centres for acute complicated Type B AD. No differences were observed within the first year in terms of rupture, conversion, and reintervention rates.

Feng et al. [27] compared 311 cases of simple TEVAR versus 154 cases of modified PETTICOAT (pre-placement of a bare stent in the distal thoracic landing zone) for acute and chronic Type B AD. Despite the fact that the indication of the modified PETTICOAT was a TL collapse and that the bare stent coverage was shorter as well as limited to the thoracic aorta, no significant differences in the peri-operative clinical outcomes were observed between the two arms. However, lesser secondary reinterventions were observed in the PETTICOAT group (3.9% vs. 9.3%). Notably, causes of reintervention in the TEVAR group were related to stent-induced distal re-dissection (0% vs. 2.9%). He et al. [23], in a smaller cohort, obtained the same findings.

Hsu et al. [26] used the PETTICOAT in a subgroup of 9 patients with acute Type A AD after an initial proximal open repair and they did not observe any clinical difference in the perioperative outcomes rate when the comparison was made with a simple TEVAR procedure.

Interestingly, in terms of mid-term survival, no significant differences were observed at 1 year between the TEVAR cohort and the PETTICOAT ones [18, 24, 27].

23.3.3 PETTICOAT and aortic remodelling

The results discussed above justify a selective use of the PETTICOAT technique only in acute TBD complicated by persistent dynamic malperfusion after TEVAR. The lack of evident benefits in terms of short-term survival of the PETTICOAT cohorts does not justify a widespread use of it. However, the analysis of mid-term aortic remodelling of the PETTICOAT series might be helpful in providing valuable insights into other potential benefits of this procedure. Nine out of the 11 papers reported the behaviour of TL and FL after PETTICOAT in the short- or mid-term [13, 18, 19, 21, 23, 24, 26-28]. The different authors proposed either diametric [13, 18, 21, 24, 26-28] or volumetric analysis [19, 23, 28] of thoracic and abdominal lumens and only six studies compared the outcomes with a simple TEVAR control group [18, 23, 24, 26-28].

With regards to TL behaviour, all studies observed that the PETTICOAT technique led to an early significant expansion of the TL not only in the thoracic aorta but also in the distal abdominal aorta if the baseline CT scans were compared to the postprocedural ones. At follow-up, the TL continued to expand significantly in the thoracic aorta, whereas its size remained relatively stable in the abdominal aorta. Interestingly, in the two studies [23, 27] in which a short (<100 mm) bare stent was predeployed only in the distal thoracic aorta in order to prevent distal stent-induced new entry tears, the TL expansion was limited to the stent-grafted thoracic segment with no further expansion in the distal thoracoabdominal aorta. These considerations might be useful in those cases in which a distal TL expansion is necessary to solve dynamic malperfusion.

When the fate of FL is taken into consideration, the data are more heterogeneous. The common finding is that after the procedure both the thoracic and abdominal FL decreased in size significantly due to the redistribution of the lumens in favour of the TL re-expansion. Moreover, a slight increase of the overall thoracoabdominal aorta dimensions was observed after surgery [19, 21, 28]. During follow-up, four studies [18, 19, 21, 24] recorded the same behaviour: the FL continued to decrease in size in the thoracic aorta (usually the stent-grafted area), while the FL in the abdominal aorta remained stable with no shrinkage. Furthermore, two studies, analysing the 2-year follow-up data, observed a tendency towards an increase in the abdominal FL in a small proportion of cases [19, 21]. In those cases, continued pressurization in the FL due to distal entry tears may lead to FL expansion as the aortic wall continues to weaken during follow-up.

A comparative study of the volumetric behaviour of TL and FL in patients enrolled in the STABLE trial [21] versus a cohort of patients who had received a simple TEVAR for compli-

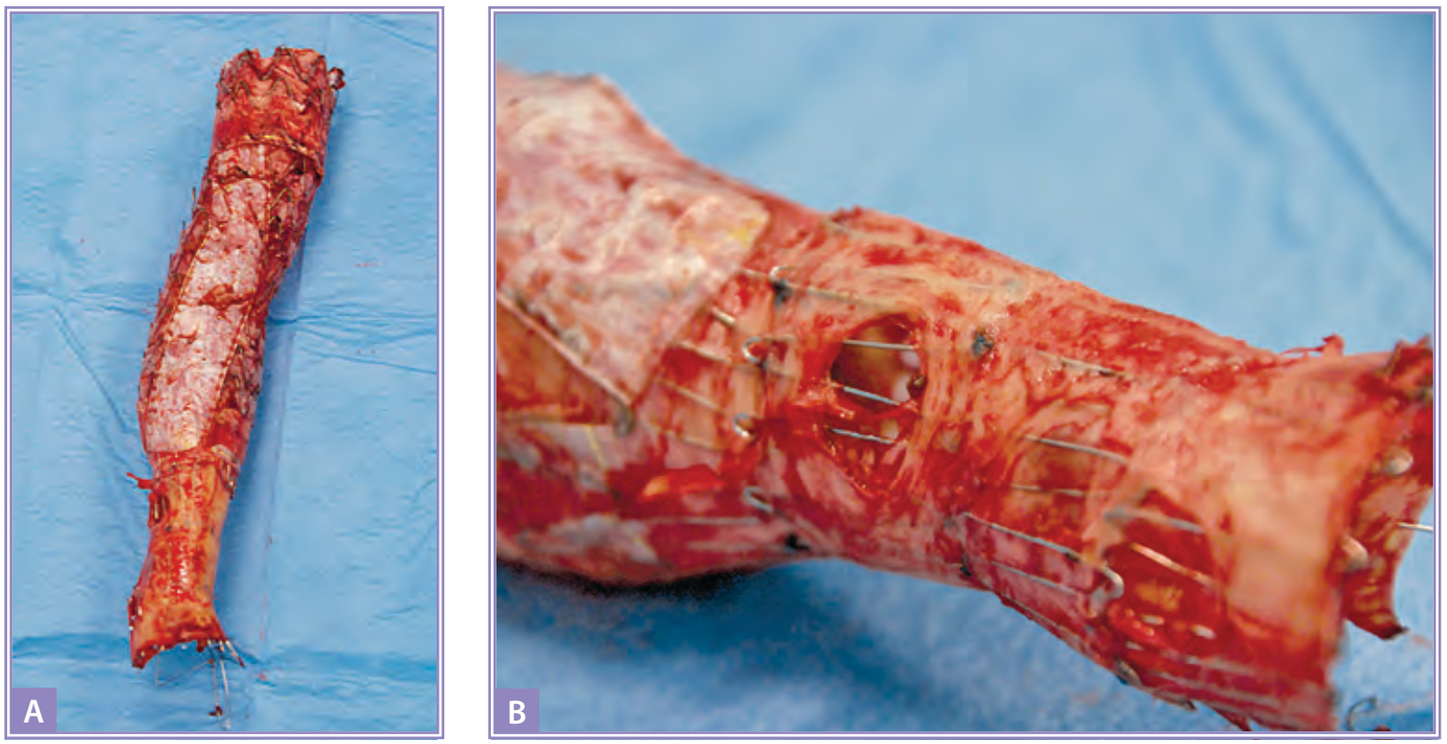

Fig. 23.6 Late open conversion of chronic aneurysmal evolution of distal thoracic and abdominal false lumen in a patient previously treated with the PETTICOAT technique (**A**). The intraoperative examination of the device evidenced full endothelialisation of the distal bare stent with the exception of the ostia of the visceral arteries (**B**).

cated Type B AD has been recently published [28]. Notably, the two groups were largely similar in terms of baseline demographics, medical history, and clinical characteristics. However, there was a trend (though not statistically significant) toward greater stent-graft coverage in the TEVAR group. The overall change in the TL volume was statistically greater in the PETTICOAT group, while the overall change in FL volume was not statistically different (pre-procedure vs. 12 months follow-up). In the abdominal aorta, although the total abdominal aortic size increased similarly in both groups, only the PETTICOAT group had substantial TL expansion postoperatively. In addition, only the PETTICOAT group exhibited a significant reduction in FL volume in the abdominal aorta postoperatively and maintained a net decrease in the average FL volume at 12 months, in contrast to the TEVAR group, although without statistical significance. Therefore, compared with TEVAR alone, the bare-metal dissection stent appeared to improve aortic remodelling in the distal aorta, at least during early follow-up [28]. Recently, Hofferberth et al. [29], in order to enhance the abdominal remodelling, proposed an evolution of the PETTICOAT technique, the So-Called Stent-Assisted Balloon-Induced Intimal Disruption and Relamination STABILISE in AD repair. This procedure aims at the reapposition of the intima lamella to the aortic wall by ballooning the distal bare stent (for details please refer to Chapter 24).

The remodelling data evaluated in this review confirm a clear-cut role of PETTICOAT in acute Type B AD complicated by dynamic malperfusion because this technique enhances the effect of the proximal TEVAR, improving the re-expansion of the TL of the distal thoracoabdominal aorta. However, there is lack of evidence to support a routine employment in Type B AD due to the unproven gain in terms of mid-term survival as well as aortic remodelling of the distal FL.

Moreover, since fenestrated and branched repair has been described with encouraging results in patients with post-dissection thoracoabdominal aneurysms [30-33], the feasibility of

such a complex endovascular aortic repair with a previous PETTICOAT has been questioned. The feasibility of fenestrated endografting through a previous PETTICOAT has been demonstrated successfully once in the literature. No major conflict with the struts of the bare dissection stent during catheterization and bridging stent placement were reported [34]. Interestingly, we performed an open standard conversion in two cases of post PETTICOAT chronic aneurysmal evolution of FL (Fig. 23.6). In both cases we noticed a complete re-endothelialisation of the dissection bare stent with the exception of the ostia of the visceral arteries. On the one hand, the presence of a PETTICOAT would seemingly jeopardize a fenestrated procedure by increasing the procedural complexity: the crossing of visceral vessels ostia through the stent struts might be challenging and, as a general rule, the presence of a previous aortic device could reduce the trackability and the rotability of a second endograft [28]. On the other hand, the greater TL in the distal aorta observed in the PETTICOAT cohorts may increase the anatomical feasibility of fenestrated procedures by reducing the excluded cases due to the excessively narrow TLs [28].

23.4 Conclusions

Acute Type B AD complicated by end-organ malperfusion has a substantial augmented risk of 30-day mortality if left untreated. Stent-graft treatment may improve the prognosis of these challenging cases; however, it may fail to completely address residual distal aortic dynamic malperfusion. The combination of proximal standard stent grafting of the proximal entry tear with the deployment of a bare stent in the distal thoracoabdominal aorta, termed the PETTICOAT technique, might be beneficial in solving residual dynamic malperfusion. Analysing the literature data not only confirms that the PETTICOAT technique is safe and feasible but also that it is able to effectively re-expand the TL of the thoracoabdominal dissected aorta, improving end-organ perfusion. However, since there is no evidence of improved mid-term survival as well as positive remodelling of the FL in the distal aorta, when compared to a simple proximal stent grafting, a wide-spread use of the PETTICOAT technique is not justified and it should be limited to cases complicated by dynamic malperfusion.

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31

Open surgical treatment of chronic Type B aortic dissection (dissecting aneurysms)

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Enrico Rinaldi, Sara Spelta, Alessandro Grandi and Domenico Baccellieri

31.1 Introduction

Chronic Type B aortic dissection (AD) is the second most common cause of thoracoabdominal aortic aneurysm (TAAA) development after degenerative etiology. Among patients undergoing surgery for TAAA, up to 24% suffer from chronic dissection [1]. In approximately 20-40% of patients with AD (both types A and B), the descending thoracic and thoracoabdominal aorta may become aneurysmal within 2 to 5 years [2]. The presence of blood flow in the false lumen is the most significant risk factor for an increase in diameter, with aneurysmal degeneration involving the descending thoracic aorta usually starting from the isthmic region and subsequently extending to the abdominal aortic tract or even to the iliac arteries [2].

A dissecting aneurysm can have a similar or faster growth rate compared to a degenerative thoracic aneurysm [3, 4], but its rupture rate is higher [5]. Hence, the indication to surgery for asymptomatic dissecting TAAA is usually considered a diameter ≥5.5 cm, or even lower in the case of associated connective tissue disorders.

Historically, open treatment of dissecting TAAA consisted of graft replacement with reattachment of the main aortic branches by means of the inclusion technique, which was introduced by S.E. Crawford in the 1970s, and mastered by Houston surgeons in the following decades. As open repair of dissecting and degenerative TAAA is known to be associated with significant morbidity and mortality, great efforts have been made in recent decades to improve surgical outcomes, resulting in the current use of a multimodal approach with several surgical and anesthesiological adjuncts, in order to maximize organ protection and prevent end-organ ischemia [6]. In this chapter, we will describe our current strategies used prior, during, and immediately after open surgical treatment of TAAA following chronic Type B AD.

31.2 Preoperative vascular imaging

An accurate preoperative imaging is required in all patients to precisely assess the aortic anatomy, fully evaluate the aortic main branches, and obtain information about the spinal cord

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G. Melissano - R. Chiesa (eds), Aortic Dissection patients true stories and the innovations that saved their lives

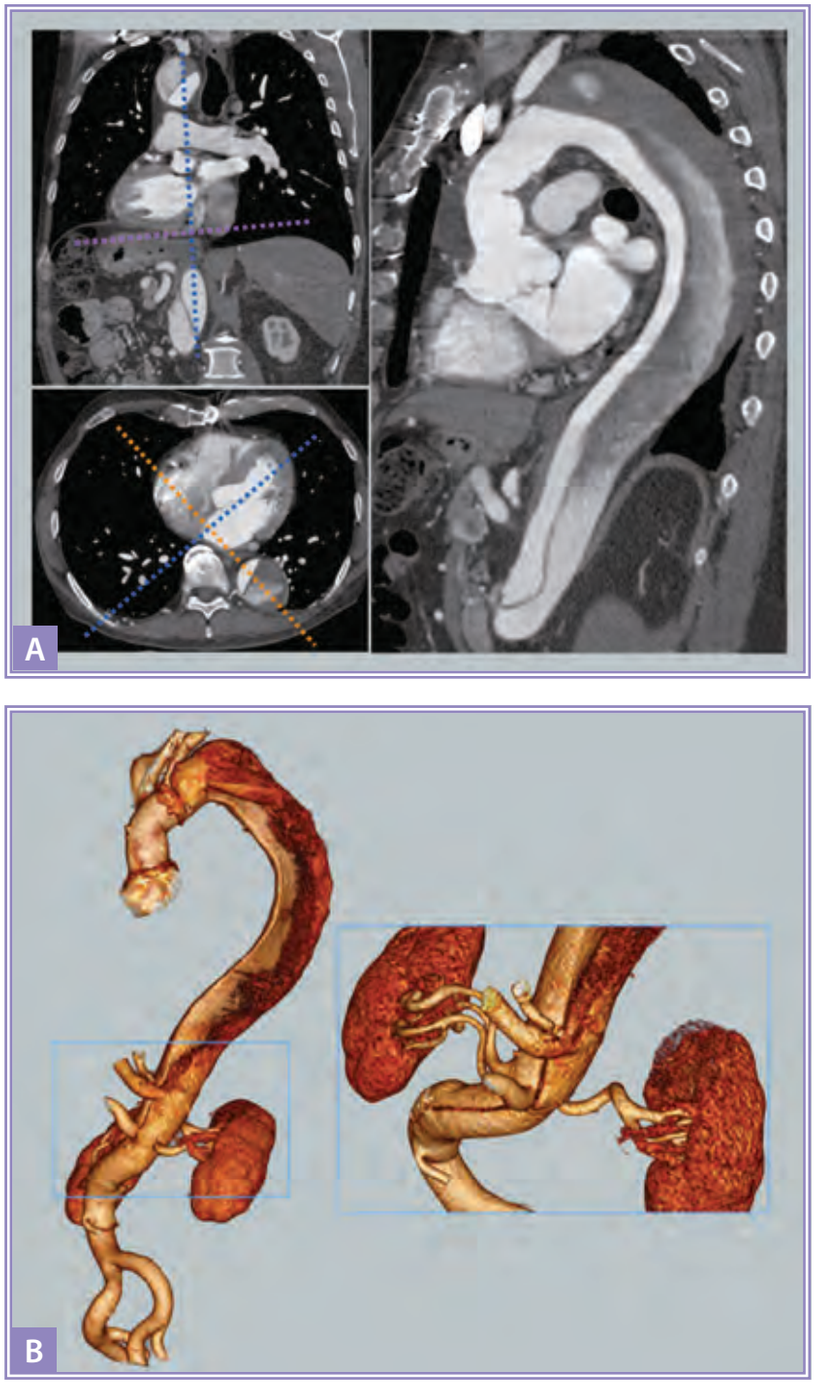


Fig. 31.1 **A**, Thanks to multi-planar reformatting, the curved path of the dilated dissected thoracic aorta is well reproduced and the true and the false lumen are properly visualized. **B**, 3D volume rendering allows to determine the conformation of the entire dissection and the relative position of the visceral and renal vessels, necessary for planning a tailored reimplantation strategy.

vasculature, in order to plan the best surgical strategy tailored to the individual. Provided that there are no specific contraindications, our preoperative imaging protocol includes contrast enhanced computed tomography (CT).

Post-processing of CT images using reformatting software and workstations allows to visualize specific morphologic issues related to AD, needed for a thorough surgical planning. In particular, the aorta and, even more so, the dissecting lamella have a tortuous path, which may be correctly analyzed using oblique multi-planar reformattings (MPRs), as shown in Figure 31.1. Maximum intensity projection (MIP) protocols are usually avoided in the case of dissections, because information on the dissecting lamella and parietal thrombus is lost.

After obtaining an optimal projection of the aorta, the true cross-sectional diameter of the aneurysm is measured and the true and the false lumen are properly visualized, as well as the relationship of the dissection with the major aortic branches, in order to define the extent of the disease. Also, the quality of the aortic wall at the intended clamping sites, the presence, extension, and characteristics of the thrombus, and the number and location of main and secondary tears in the dissecting lamella have to be carefully evaluated.

Special attention must be paid to the quality and relative position of the visceral vessels ostia, in order to plan a tailored preimplantation strategy (e.g. Carrel patch inclusion vs. individual bypass or multi-branched pre-sewn grafts) or prepare associated endovascular maneuvers (e.g. direct stenting or use of sutureless anastomosis).

Finally, preoperative knowledge of the arterial supply to the spinal cord is extremely useful for procedure planning and risk stratification, in order to prevent spinal cord ischemia and subsequent paraparesis or paraplegia (see Chapter 36) [7].

31.3 Patient optimization

An adequate preoperative assessment of the patient's physiologic reserve of cardiac, pulmonary, and renal function, along with an accurate knowledge of the cerebral and spinal cord vascular anatomy are useful for evaluating the operative risk, planning the best operative strategy and taking early additional perioperative precautions.

31.3.1 Cardiac optimization

Preoperative transthoracic echocardiography is a satisfactory non-invasive screening method that evaluates both valvular and biventricular function. Exercise testing or dipyridamole-thallium myocardial scanning identifies regions of the myocardium that are reversibly ischemic, and in patients with a significant history of angina or reduced ejection fraction cardiac catheterization with coronary arteriography is indicated [8].

CT coronary angiography has emerged as a less-invasive method to visualize the coronary arterial anatomy. Multi-detector scanners allow one to obtain images at certain specific phases of the cardiac cycle with the least coronary artery motion, providing information also about the aorta and left ventricular ejection fraction [9].

In patients undergoing thoracic aortic open repair, severe coronary artery occlusive disease is treated with percutaneous transluminal angioplasty prior to aortic surgery. Preoperative coronary stenting is usually accomplished with bare-metal stents, trying to avoid the use of drug-eluting stents requiring prolonged (>6 months) double antiplatelet therapy that in turn increases the risk of perioperative bleeding. Recently, the novel COMBO stent (OrbusNeich, Fort Lauderdale, FL), which combines sirolimus elution with a $CD34^+$ antibody to attract endothelial progenitor cells and thus enhance early endothelialization, is currently under investigation vis-à-vis its potential to reduce double antiplatelet therapy duration from 6 to only 3 months after stent implantation [10]. Its use may be considered in patients needing coronary stenting, who are also affected by large dissecting TAAAs requiring surgical repair in a timely fashion. Nowadays, surgical myocardial revascularization by means of coronary-aortic bypass grafting is limited to selected patients in whom percutaneous transluminal angioplasty would be inappropriate.

31.3.2 Renal optimization

Renal function is a well-established predictor of postoperative outcome [11]. The National Kidney Foundation currently recommends the use of estimated glomerular filtration rate

(eGFR) to assess renal function in order to avoid the misclassification of patients on the basis of serum creatinine levels alone [12]. Based on the GFR assessment, chronic kidney disease has been shown to be a strong predictor of death after thoracic aneurysm repair for both open and endovascular procedures, even in patients without clinical evidence of preoperative renal disease [13]. Renal size, corticomedullary ratio, presence of renal cysts, and renal artery anatomy are determined from angio-CT/angio-magnetic resonance (MR) and by ultrasound. Occlusive disease of the renal arteries may be treated during aortic repair, and patients are not rejected as surgical candidates based on impaired renal function only.

Adequate hydration, diuretics administration and premedication with N-acetylcysteine and endovenous bicarbonates may be used in patients with chronic kidney disease to reduce preoperative serum creatinine levels and optimize renal function [14].

31.3.3 Pulmonary optimization

Evaluation of pulmonary function with arterial blood gases and spirometry is performed for all patients undergoing open surgery of the thoracic/thoracoabdominal aorta. In patients with a forced expiratory volume in 1 sec (FEV_1) lower than 1.0 L and a partial pressure of carbon dioxide (PCO_2) higher than 45 mmHg, pulmonary function can be improved by stopping smoking, progressively treating bronchitis by means of steroids and bronchodilators, losing weight and following a general exercise program for a period of 1 to 6 months before operation. However, in patients with very large or symptomatic aortic aneurysms, the operation often cannot be delayed despite their poor pulmonary function. An endovascular approach may be evaluated in these cases, when feasible.

Otorhinolaryngological evaluation by means of fiberoptic nasopharyngoscopy is routinely performed to assess respiratory space, vocal cord motility and possible paralysis that may be due to recurrent laryngeal nerve compression by the false lumen.

31.3.4 Neuropsychological evaluation

A brain CT scan together with a neuropsychological evaluation by an independent neurologist is obtained for all elective patients. Preoperative brain CT scan is evaluated in order to detect possible contraindications to spinal cord fluid drainage. Optimization of psychiatric disorders, and potential pharmacological improvement of mood tone are useful to enhance the patient's compliance in the perioperative period, increasing adherence to medical prescriptions and physical rehabilitation therapy. Preoperative somatosensory evoked potentials (SSEPs) and motor evoked potentials (MEPs) are evaluated to provide the basal peripheral neurological condition for comparison to the intraoperative one, and can drive consequent surgical and anesthesiological maneuvers to decrease spinal cord ischemia risk, such as increasing the spinal fluid drainage rate, blood pressure, volemia, hematocrit, oxygenation, or providing early revascularization of intercostal and iliac arteries [15].

31.4 Surgical strategy

31.4.1 Multisystemic protection strategies

A multimodal anesthesiological approach and complex multisystemic monitoring prior, during, and after dissecting TAAA open repair are of paramount importance, and involve collaboration with many specialists (Fig. 31.2).

Fig. 31.2 Multidisciplinary approach to TAAA surgery. The cardiovascular anesthesiologist keeps the patient under continuous monitoring and performs transesophageal echocardiography (**A**) in order to evaluate cardiac function during the whole procedure. The anesthesiologist modulates fluid exchange and body temperature with a pressure infusion system and fluid warmer (**B**). Intraoperative blood salvage is performed during TAAA surgery. The cell saver (**C**) is used to recover the blood lost during the procedure and to re-infuse it into the patient. **D**, The perfusionist controls and modulates the left heart bypass performed with Bio-Medicus® Perfusion System (Medtronic, Minneapolis, MN), in agreement with the anesthesiologists. A circulating water mattress with heat exchanger (**E**) is placed under the patient, in order to assist body temperature management. The neurologist checks the motor-evoked and somatosensory-evoked potentials to assess spinal cord integrity (**F**). The LiquoGuard® (Möller Medical, Fulda, Germany) (**G**) allows simultaneous cerebrospinal fluid (CSF) pressure measurements and drainage intraoperatively and during the first 48-72 hours after the surgical procedure.

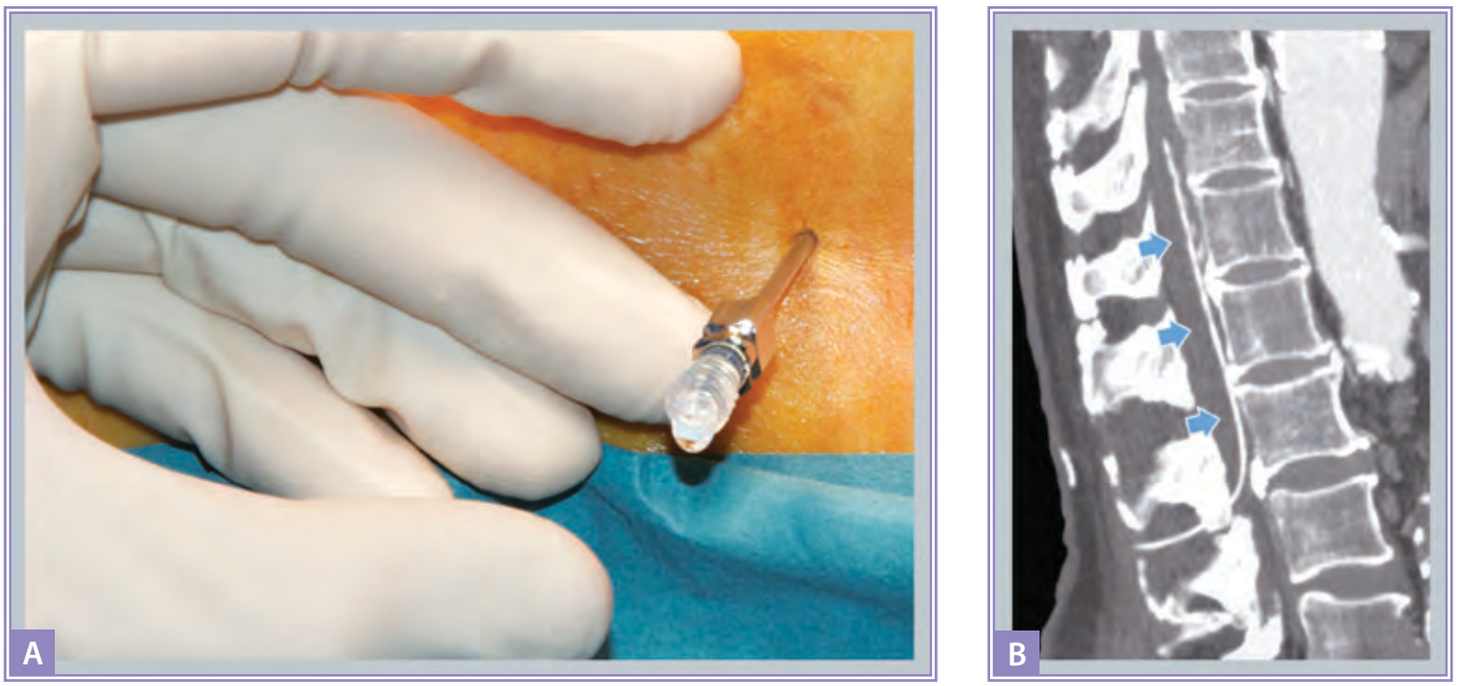


Fig. 31.3 Once the dura mater has been punctured with the introducer needle (**A**), a drainage catheter is inserted 8 to 10 cm beyond the tip of the needle into the subarachnoid space (**B**). After the drain has been secured to the patient, it can be connected to the pressure transducer and baseline measurements can then be made.

Perioperative anesthesiological management is described in detail in Chapter 38. Briefly, blood pressure is monitored invasively through a radial cannula and a femoral cannula that monitors distal perfusion during aortic clamping and left heart bypass (LHBP). In dissecting thoracoabdominal aneurysms, a differential blood pressure between the radial and femoral cannulas is not uncommon: in these cases, it is important to assess the difference at the beginning, and take it into account during the surgical procedure.

Orotracheal intubation is performed using a double-lumen endotracheal tube, in order to maintain a unilateral right ventilation during aortic isolation and reconstruction. Intraoperative transesophageal echocardiography is used to evaluate cardiac function, output and volemia throughout the procedure; in dissections, it is also able to detect if guidewires used for cannulation are correctly placed in the true lumen and can immediately detect a disastrous complication such as retrograde Type A AD after proximal clamping (see Chapter 40).

After anesthesia is induced and prior to surgical incision, the dura mater is punctured with an introducer needle and a standard lumbar CSF drainage catheter (Integra LifeSciences, Plainsboro, NJ) is inserted 8 to 10 cm beyond the tip of the needle into the subarachnoid space for the cerebrospinal fluid (CSF) drainage (Fig. 31.3). The CSF pressure is usually maintained during the first 48-72 hours at 8-10 mmHg using an automatic system (LiquoGuard®, Möller Medical, Fulda, Germany) allowing simultaneously CSF pressure measurements and CSF drainage by a closed system with dual pressure sensors with an electronically controlled peristaltic pump (Fig. 31.2 G).

Intraoperatively, MEP and SSEP monitoring is performed to assess spinal cord integrity, and guide intraoperative strategies to prevent neurologic deficits. Based on decrease or loss of MEPs and SSEPs, intraoperative modifications (spinal fluid drainage, blood pressure, volemia, hematocrit, oxygenation, early revascularization of intercostal and iliac arteries) can be made when necessary.

31.4.2 Surgical technique

The thoracic incision varies in length and level depending on the extent of the aneurysm, but it is usually made between the 5th and 7th intercostal ribs. Section of the rib posteriorly of the rib or, if necessary, its whole resection associated to a gentle and progressive use of the retractor is useful to reduce thoracic wall trauma and fractures; antero-laterally, the incision curves gently as it crosses the costal margin, reducing the risk of tissue necrosis. The pleural space is entered after single right lung ventilation has been initiated. Single-lung ventilation is maintained throughout the aortic replacement.

After the thoracoabdominal incision, a limited circumferential section of the diaphragm is routinely carried out, sparing the phrenic center so as to reduce respiratory weaning time. An involvement of the phrenic center leading to paralysis of the left hemidiaphragm produced by its radial division to the aortic hiatus could contribute significantly to postoperative respiratory failure [16].

Due to the inflammatory reaction in the acute phase of dissection, pleural adhesions are quite common in chronic AD. In these cases it is important to perform a limited tissue dissection to minimize pulmonary bleeding.

Special care must be taken when isolating the proximal aortic neck, which can be encircled using a vessel-loop. The vagus nerve and the origin of the recurrent nerve must be identified, since they can be damaged during isolation of the aortic isthmus and clamping maneuvers. Identification and clipping of some "high" (T1-T6) intercostal arteries can sometimes facilitate the preparation for the proximal anastomosis, thus reducing aortic bleeding. The upper abdominal aortic segment is exposed via a transperitoneal approach; the retroperitoneum is entered lateral to the left colon, and medial visceral rotation is performed so that the left colon, spleen, and left kidney can be retracted anteriorly and to the right. A transperitoneal approach allows direct view of the abdominal organs to evaluate the efficacy of revascularization at the end of aortic repair.

Cross-clamping of the descending thoracic aorta leads to several hemodynamic disturbances, including severe afterload increase and organ ischemia. The technique for distal aortic perfusion with LHBP has proved to be extremely useful during aortic repair [1]. The main rationale for the use of LHBP is the perfusion of distal organs with concomitant reduction of cardiac post-load during clamping of the distal arch/descending thoracic aorta. The basic circuit for LHBP includes an inflow cannula, an inline centrifugal pump without a cardiotomy reservoir or oxygenator, and an outflow cannula. In preparation for LHBP and aortic clamping, to reduce bleeding from the extensive tissue exposure, intravenous heparin is administered (70 IU/kg).

The inflow cannula is placed in the left superior pulmonary vein (20 Fr moldable cannula) to reach the left atrium, and the arterial blood is then reinfused into the femoral artery. In chronic aortic dissections, proximal cannulation of the aorta should be avoided in favor of pulmonary vein cannulation. After left groin cut-down and surgical exposure of the common femoral artery, a percutaneous outflow cannula (14-16 Fr) is placed by means of an over-the-wire technique (.035") and carefully progressed into the non-dissected segment of the left iliac axis (Fig. 31.4). The cannula is then secured with a purse-string suture and the artery is not occluded, allowing distal perfusion of the ipsilateral limb during assisted circulation. A "Y" bifurcation is connected to the circuit and is provided with two occlusion/perfusion catheters (9 Fr) for selective perfusion of the visceral vessels.

In most cases of chronic Type B dissection, proximal aortic clamping is done between the left common carotid artery (LCCA) and the left subclavian artery (LSA), allowing the proximal anastomosis to be performed at a healthy and less fragile aortic site. In a few selected cases, when the location, extent, and severity of disease precludes placement of a proximal aortic clamp distally to the LCCA, hypothermic circulatory arrest is performed to allow proximal anastomosis to be completed.

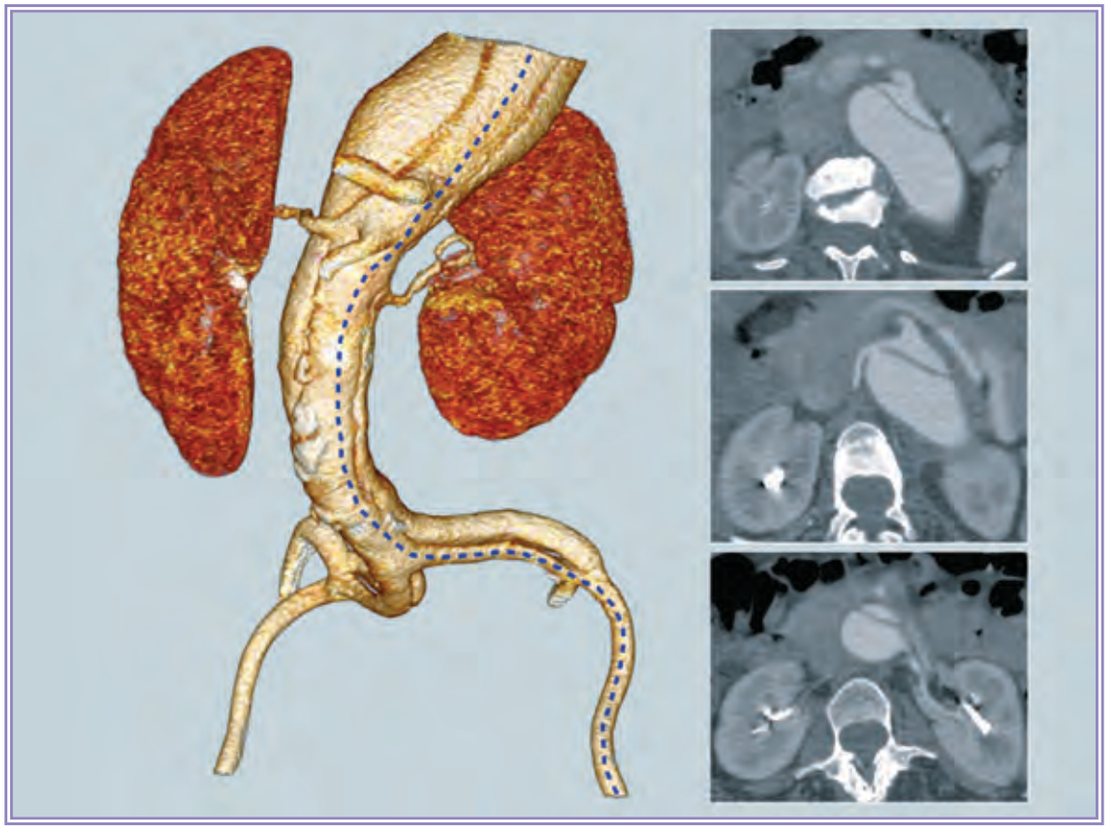


Fig. 31.4 Preoperative evaluation with angio-CT is important to assess the extent to which the dissection has involved the iliac arteries, and the origins of the visceral vessels. The blue line on the 3D volume rendering underlines the partially compressed true lumen; in the axial scans one can distinguish the origins of the visceral and renal vessels from the true and false lumen.

Fig. 31.5 Proximal anastomosis. **A**, The proximal aortic clamp is placed between left common carotid artery (LCCA) and left subclavian artery (LSA) (blue arrow) after the LSA has been selectively clamped (green arrow). The thoracic aorta is completely transected and the false (*) and the true (**) lumen are visualized. **B**, The proximal end of the graft is sutured to the descending thoracic aorta with a 2/0 or 3/0 polypropylene running suture reinforced with a strip of Teflon felt (yellow arrows).

Once the proximal aspect of the TAAA is isolated between clamps, the descending thoracic aorta is transected and separated from the esophagus. The proximal end of the graft is sutured to the descending thoracic aorta, usually using a 2/0 or 3/0 monofilament polypropylene suture in a running fashion. The anastomosis can be reinforced with multiple small Teflon pledgets, with one strip of Teflon felt, or even a double layer of Teflon felt placed inside and outside the aortic wall as a "sandwich" (Fig. 31.5).

The clamp is then removed and reapplied onto the abdominal aorta above the celiac axis (sequential cross-clamping). A longitudinal aortotomy is performed with a complete septotomy. Reimplantation of intercostal arteries to the aortic graft plays a critical role in spinal cord protection. Critical patent segmental arteries from T8 to L2, and/or other specific ones identified by preoperative CT as a candidate source of the Adamkiewicz artery, are temporarily occluded with Pruitt (LeMaitre Vascular, Burlington, MA) catheters to avoid the blood steal phenomenon, then selectively reattached to the graft by means of aortic patch or graft interposition. In dissecting aneurysms, most of the patent intercostal arteries arise from the false lumen. The reimplantation can be technically demanding, and the use of reinforcing Teflon pledgets and strips is often indicated (Fig. 31.6).

When the aneurysmal evolution is limited to the descending thoracic aortic tract, replacement only of the thoracic aorta can be performed with a supradiaphragmatic or supraceliac "double-barrel" distal anastomosis. In this case, the septotomy may be extended distally across the abdominal portion in order to reduce the risk of malperfusion.

In the case of thoracoabdominal dilatation, the distal clamp is moved below the renal arteries and the aneurysm is opened below the diaphragm. Visceral isothermic hematic perfusion

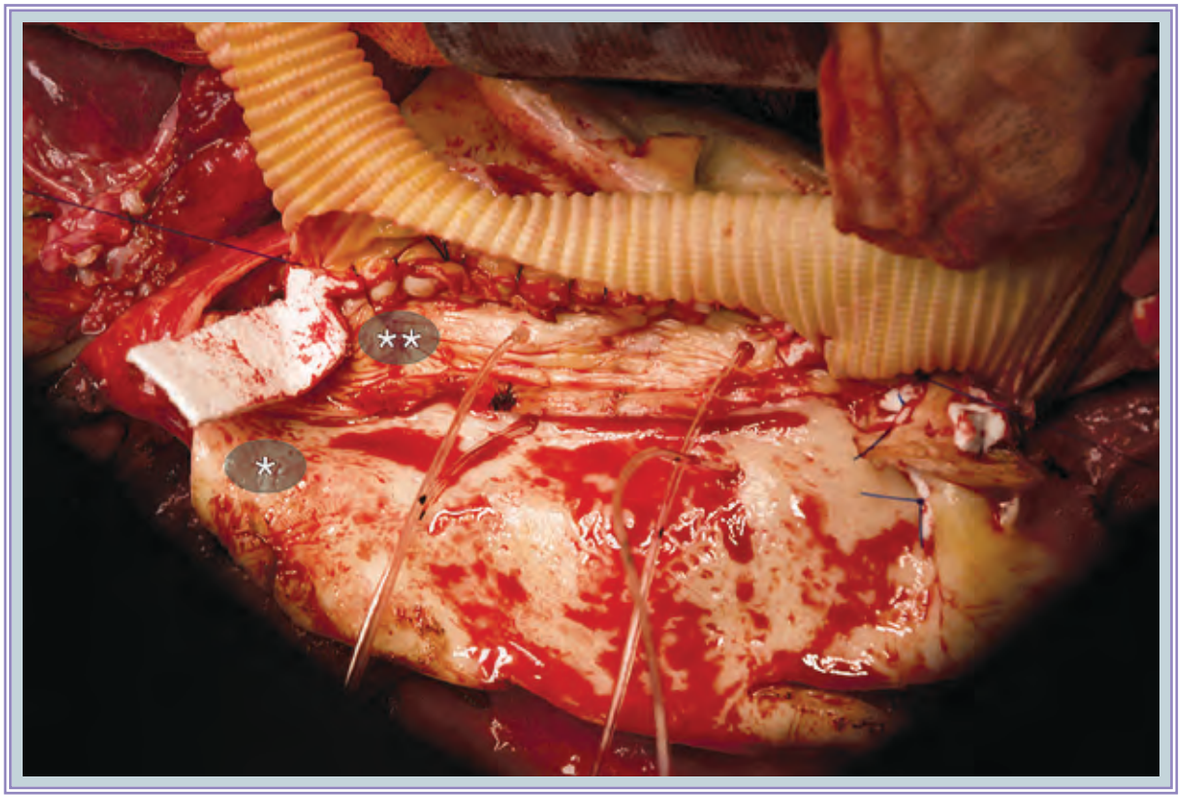


Fig. 31.6 Critical intercostal arteries arising from the false (*) and the true (**) lumen are temporarily occluded with Pruitt catheters and selectively reattached to the graft by an aortic patch or sutured.

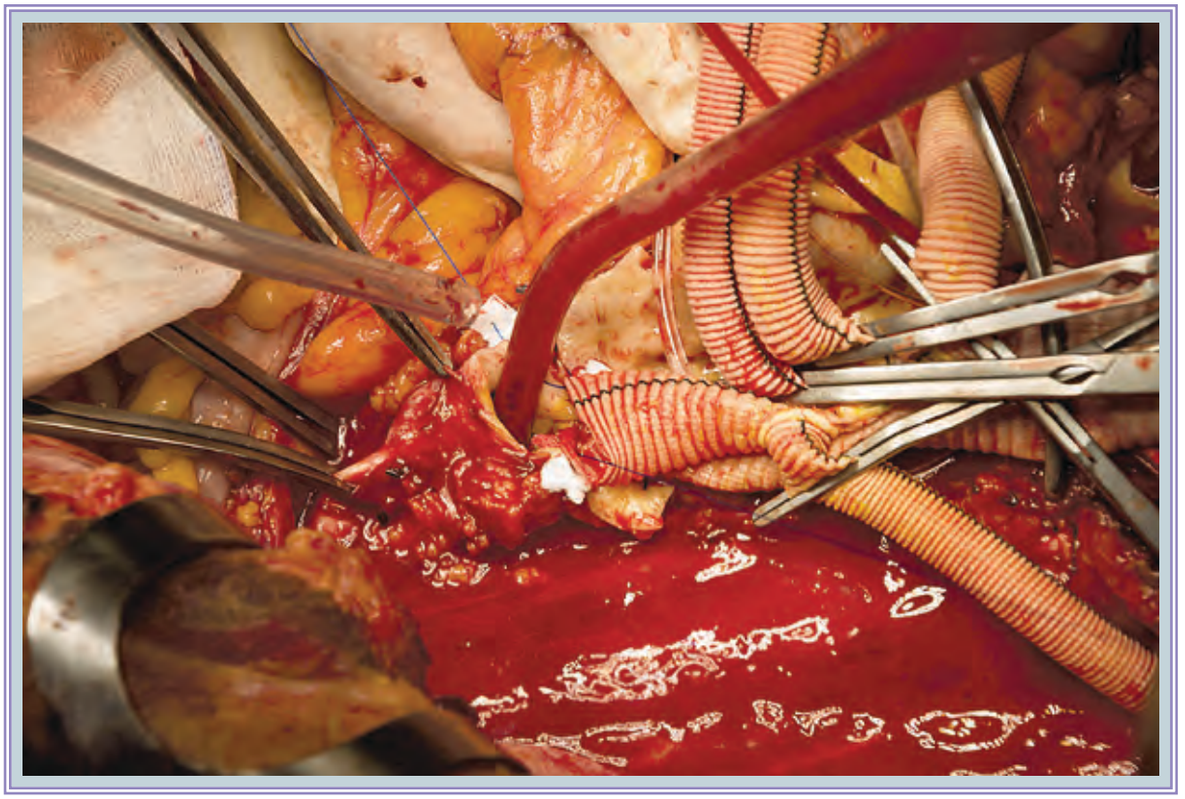


Fig. 31.7 Distal anastomosis. In the case of a multi-branched aortic graft, the distal anastomosis is performed before reattachment of the visceral and renal vessels in order to restore first the antegrade flow to the iliac and hypogastric arteries. During this period, visceral perfusion is maintained by the left heart by pass (LHBP).

is then maintained by the pump with irrigation-perfusion catheters of the same size (9 Fr) inserted selectively into the celiac trunk and the superior mesenteric artery. Selective cold perfusion of the renal arteries is performed with Custodiol (histidine-tryptophan-ketoglutarate, Essential Pharmaceuticals, Ewing, NJ) [17]. Special attention must be paid when inserting the cannula in a visceral vessel that might be involved by the dissection. For reimplantation of the visceral arteries, a side cut is tailored in the graft, and the celiac trunk, superior mesenteric artery, and renal arteries are reattached by means of a Carrel patch. If the dissecting lamella involves the origin of the visceral vessels it must be carefully removed at this time. In some cases, a direct stenting of the vessel ostia could also be useful. The Carrel patch is usually limited to not more than three vessels to avoid large aortic wall islands, which are known to be at risk of further aneurysmal degeneration in time [18].

The remaining vessels to be reimplanted, in particular the left renal artery, are usually reattached separately by a 6-8 mm Dacron graft interposition or using the Gore® Hybrid Vascular Graft (W. L. Gore and Associates, Flagstaff, AZ) for a distal "sutureless anastomosis" (Fig. 31.7). This ePTFE graft is composed of a distal section, radially supported by a nitinol self-expandable stent that allows for a sutureless anastomosis and prevents kink [19].

After reattaching the vessels included in the Carrel patch, we prefer to restore first the antegrade flow to the iliac and hypogastric arteries by completing the distal anastomosis (Fig. 31.8), and then reattach the remaining renovisceral vessels (mainly the left renal artery), provided that an adequate cold perfusion can be maintained throughout the procedure. After unclamping and renal reperfusion, indigotindisulfonate sodium (40 mg) is administered intravenously, and its urine excretion is verified thereafter.

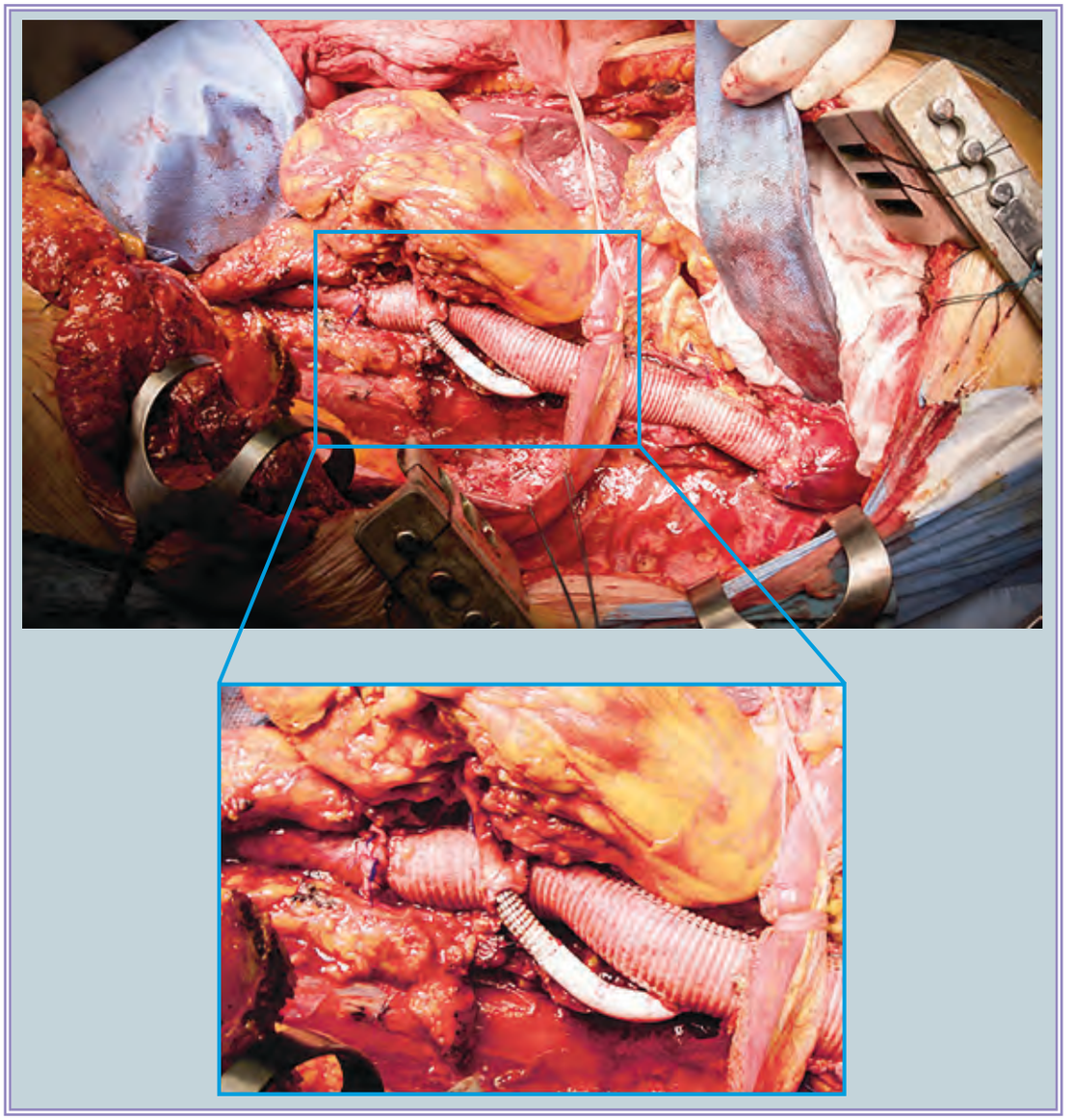


Fig. 31.8 Thoracoabdominal aortic aneurysm surgical repair with reimplantation of the celiac trunk, superior mesenteric artery and right renal artery in a Carrel patch; the left renal artery is subsequently reattached separately by a 8 mm Gore® Hybrid Vascular Graft.

31.4.3 Multi-branched grafts

The reimplantation of a large amount of native aorta in the inclusion technique for TAAA repair carries the risk of subsequent recurrent aneurysm formation [18]. Techniques proposed to reduce the incidence of visceral aortic patch degeneration include routine exclusion of the left renal artery from the Carrel patch, or single-vessel reattachments using commercially available pre-sewn multi-branched aortic grafts. These individual anastomoses can be safely accomplished, especially in the case of increased distance between the visceral vessel ostia (i.e. when the visceral segment of the aorta is involved by severe aneurysmal degeneration). If vessels originate close together, the use of a multi-branched graft may be technically demanding. In this case, a separate extended exposure of the visceral vessel is performed, with transection of each artery at its origin, and end-to-end anastomoses with the graft branches. Additionally, in order to reduce the amount of native aorta included in our repair, a pre-sewn multi-branched graft is always preferred in patients with connective tissue disorders (Fig. 31.9).

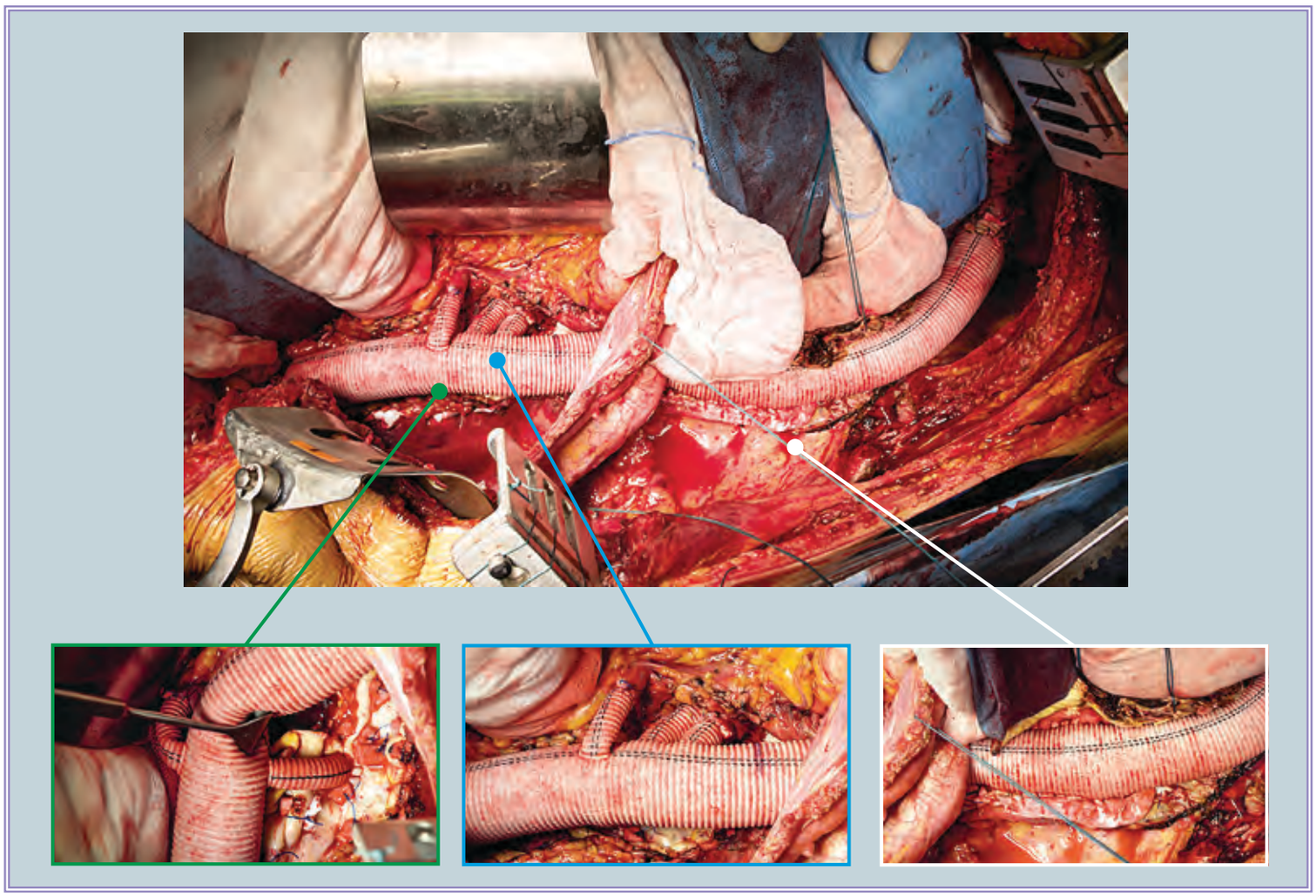


Fig. 31.9 Dissecting TAAA surgical repair. The intercostal arteries are reattached to the graft by means of an aortic patch using a Teflon felt strip to reinforce the suture (white box). The celiac trunk, superior mesenteric artery and left renal artery are reattached separately using the pre-sewn branches (blue box). The right renal artery is also reattached to the graft using a dedicated single-branch graft (green box).

31.4.4 End of procedure

After unclamping, the entire aortic repair is inspected, and all the pulses of the aortic branches exposed are palpated also after de-rotation of the abdominal viscera. Any bleeding, kinking or torsion of the aortic branches is carefully checked. Urine output is monitored regularly.

The atrial and femoral cannula are removed; the purse string sutures are tied and reinforced. Protamine is then given, and lab tests, including thromboelastometry (ROTEM®, TEM group, Basel, Switzerland), are conducted to check for any coagulopathy that has to be reversed (see Chapter 38).

The diaphragmatic pillars are approximated to reshape the aortic hiatus, and the left hemidiaphragm is sutured with a running polypropylene suture. A closed-suction abdominal drain is positioned along the repaired aorta in the left retroperitoneal space and two chest tubes are placed in the postero-apical and basal space. The abdominal and thoracic walls are sutured in standard fashion.

31.5 Specific strategies in patients with previous TEVAR

Due to the increasing number of Thoracic Endovascular Aortic Repair (TEVAR) procedures that have taken place over the last few decades, a specific section must be dedicated to the management of Type B AD in patients with previous TEVAR, who in time often require conversion to open surgical repair of the thoracic or thoracoabdominal aorta.

The most common situation for TEVAR failure requiring conversion to open surgery is that of false lumen enlargement leading to aneurysmal degeneration, and subsequent rupture. This is mainly due to false lumen reperfusion and re-pressurization, caused by unsuccessful exclusion of the proximal entry tear (i.e. Type Ia endoleak, or late migration), or distal reperfusion from abdominal re-entry tears (even after successful coverage of the proximal entry tear).

Typically, the first therapeutic option in this scenario is endovascular relining, by means of proximal or distal extension with covered endografts, false lumen obliteration with custom-made devices, or thoracoabdominal total endovascular repair with fenestrated and branched endografts. However, in selected cases, conversion to open surgery may be chosen, entailing total or partial endograft removal (Fig. 31.10). In addition, surgical conversion is preferred in most cases of endograft infection, in young and fit patients, and in cases of associated connective tissue disorders.

Although open repair techniques for thoracoabdominal dissecting aneurysm repair in the case of previous TEVAR are generally the same as those earlier described in this chapter, the following topics deserve a special attention:

- exposure of the thoracic aorta;
- location and techniques for proximal clamping;

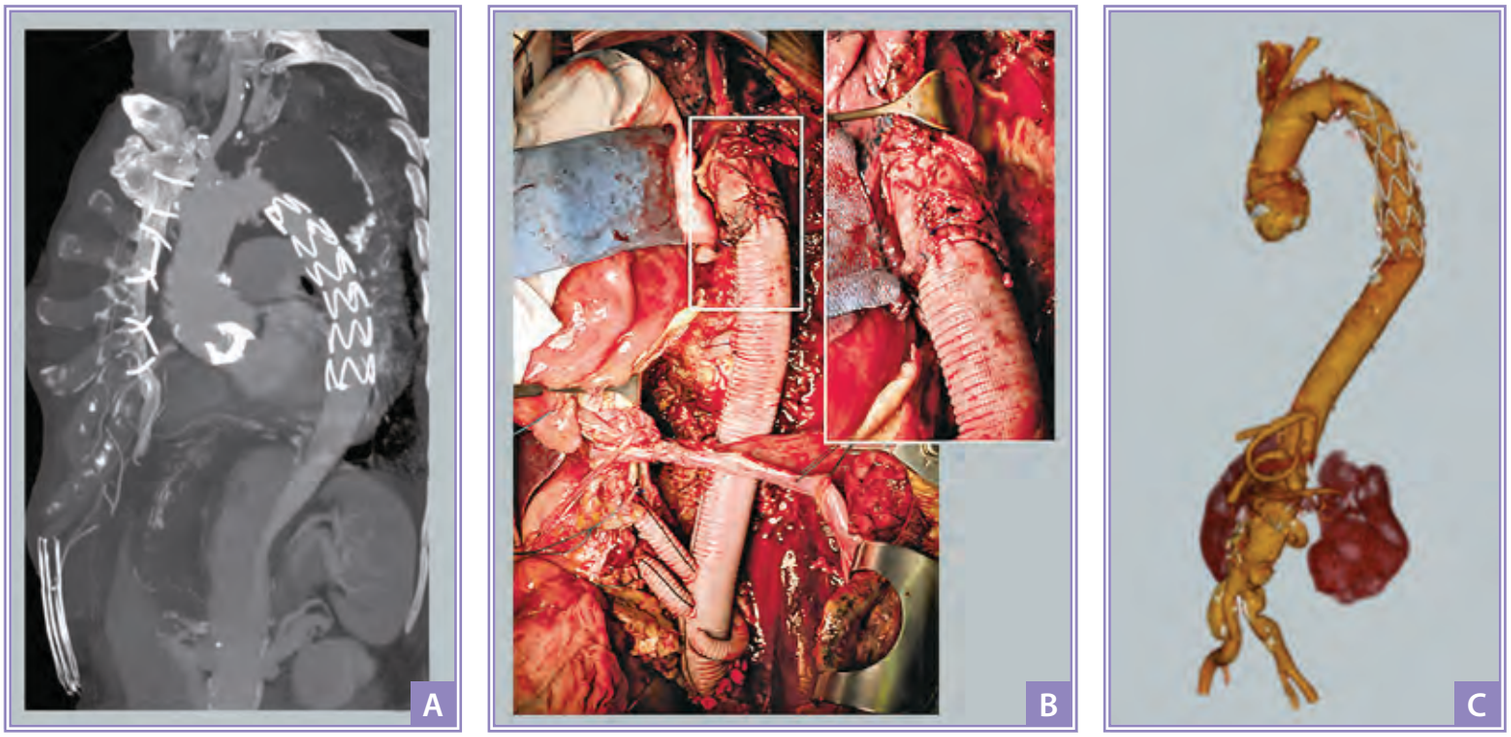


Fig. 31.10 **A**, Preoperative angio-CT. Dissecting TAAA in a Marfan patient with previous ascending aorta and aortic valve replacement and frozen elephant trunk. **B**, Surgical repair with a multi-branched graft and selective reattachment of visceral and renal vessels. In this case, the proximal end of the graft is sutured to the distal end of the previous stent graft using a 2/0 monofilament polypropylene running suture. **C**, Postoperative angio-CT 3D volume rendering.

- techniques for endograft removal;
- graft-to-endograft anastomosis characteristics;
- intercostal and visceral arteries management.

In the case of previous TEVAR, an inflammatory reaction is commonly observed involving the aortic wall and surrounding structures. This is usually associated with intraoperative findings such as tight left pulmonary adhesions, a fibrous reaction involving the heart and left pulmonary veins, vagus nerve, left recurrent laryngeal nerve and/or the thoracic duct. Special care needs to be employed in accessing the left pleural space, in the pulmonary vein cannulation, and in the thoracic aorta exposure, with a cautious progressive dissection of the tissues, which sometimes requires collaboration between the cardiac and the thoracic surgeon.

Proximal aortic cross-clamping is achieved at the level of the previous proximal endovascular neck in most cases. Consequently, the proximal metal struts of the endograft may be clamped together with the aortic wall, possibly requiring the use of atraumatic clamps with sponge rubber inserts. In the case of planned total endograft explantation, a "strut-free" proximal portion of the aorta (usually involving the distal arch) has to be exposed, generally requiring delicate retraction of the vagus and left recurrent laryngeal nerve, resection of the ligamentum arteriosum, exposure of the LCCA and LSA, and aortic cross-clamp placement between these two vessels.

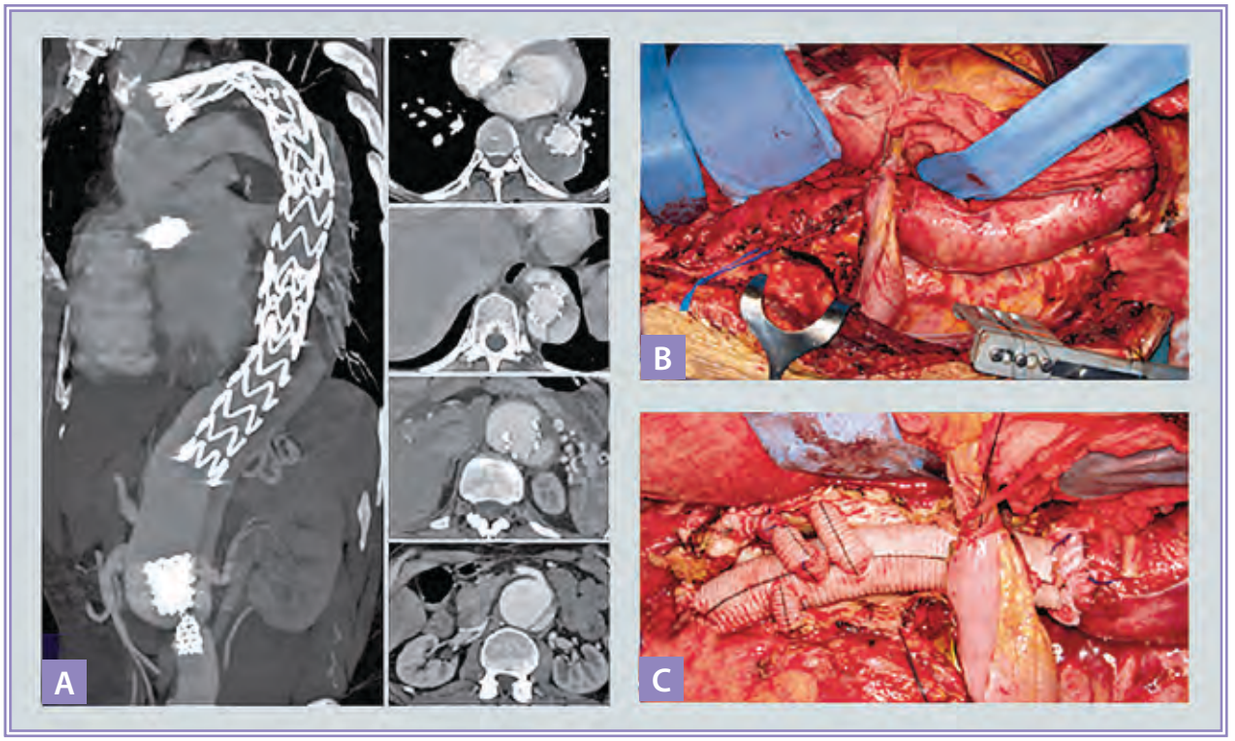
Total endograft removal may be deemed necessary in cases of suspected prosthetic infection, device rupture or collapse, or complete loss of proximal sealing by means of major migration. However, total excision of the endograft is considered a dangerous maneuver in most cases, due to the risk of aortic wall damage by metal struts, hooks and barbs during device rip-out. This may also lead to retrograde dissection of the arch and the ascending aorta. Moreover, the residual aortic wall after endograft removal often appears extremely fragile, perhaps due to the chronic wall ischemia caused by the prolonged radial force exerted by the stent graft. Finally, complete endograft removal requires aortic cross-clamping proximally to the endovascular device, mostly at the level of aortic arch branches, or even hypothermic circulatory arrest.

In the majority of cases, the conversion to open aortic surgical replacement may be accomplished following only a partial explantation of the endograft. In this case, an intraoperative evaluation of the re-entry tears by transesophageal echocardiography can be useful to guide the level of proximal clamp placement. From a technical point of view, after cross-clamping, endograft and aortic transection is performed. This type of incision allows to obtain a section of the aorta that contains both the endograft into the true lumen, and the septum as well as the false lumen. Resection of the stent graft is always accomplished at the level of the fabric to reduce the risk that the cut stent struts may damage the surgical graft or the suture line. If the stent struts have to be cut, special attention has to be paid to avoid direct impingement of the leftover spikes on the new aortic tube-graft.

The "graft-to-endograft" anastomosis is then performed by including within the vascular suture the previous stent graft and the outer aortic wall; in these cases a "three-layer technique" is suggested in order to reinforce the anastomosis using Teflon felt all around the aorta (Fig. 31.11). Particular attention is needed concerning the metal stent spikes at the level of the anastomosis, which must be pushed inside the graft lumen with each running suture stitch.

Intercostal arteries in the stented zone in patients with previous TEVAR are usually totally thrombosed or the aortic wall is inadequate to allow the replanting. Critical intercostal arteries at the level of T8-T12 might be found distally and are thus reimplanted.

The distal clamp is sequentially moved to the distal abdominal aorta or iliac arteries. After extending the aortotomy, septotomy, visceral perfusion and reimplantation of the arteries is performed with the same techniques as previously described. If the dissection has extended into a vessel origin, separate reimplantation (with or without graft interposition) is recommended. If the dissection involves the iliac or femoral arteries, bifurcated reconstructions are suggested.



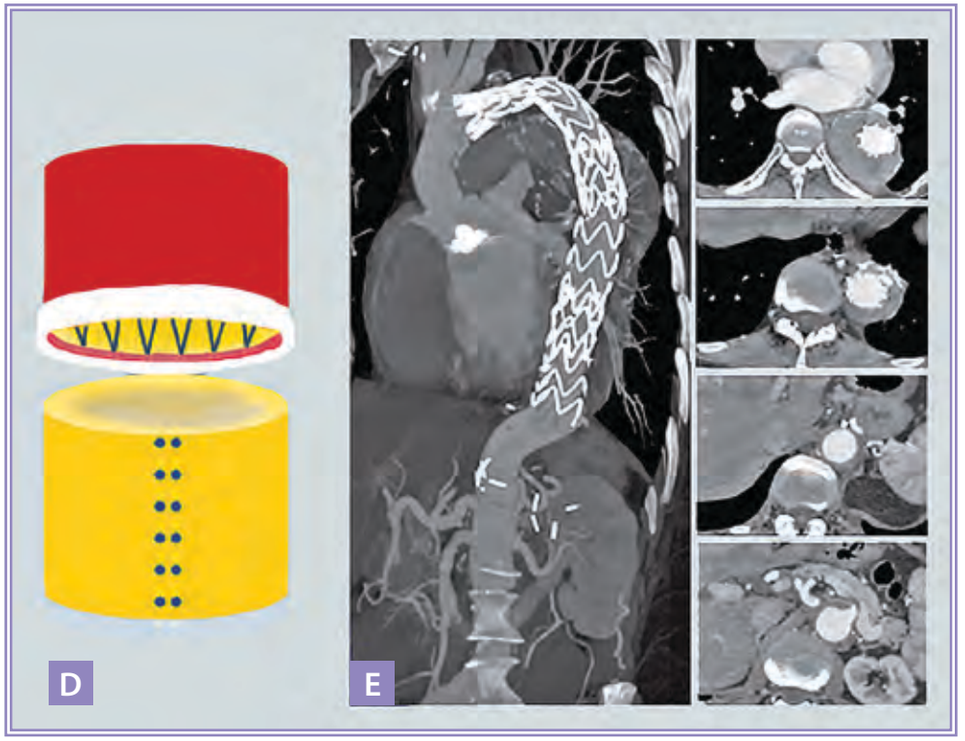


Fig. 31.11 **A**, Preoperative angio-CT. Marfan patient with previous ascending aorta and aortic valve replacement and TEVAR. The distal thoracic and the visceral aorta are progressively enlarged causing a dissecting TAAA. A balloon-expandable stent had also been placed previously at another institution in order to re-expand the true lumen. **B**, Surgical exposure of the dissecting TAAA. **C**, The stent graft and the aorta are transected 5 cm above the diaphragm and a multi-branched graft is used with selective reattachment of the visceral and renal vessels. **D**, Included in the proximal anastomosis are the partially preserved stent graft, the aortic wall and a Teflon felt strip. **E**, Postoperative angio-CT. Complete exclusion of the thoracic part of the aneurysm with surgical replacement of the visceral aorta and selective reimplantation of the visceral and renal vessels.

31.6 Early postoperative management

The main focus of the immediate postoperative management after thoracoabdominal aortic repair in the Intensive Care Unit (ICU) is aimed at early detection of any possible neurological and cardiovascular complication, to promptly institute prophylactic or therapeutic interventions.

As soon as normal body temperature is reached, the patient is allowed to temporarily wake up even if prolonged ventilation support is still required. If spinal cord or brain damage is suspected, CT imaging is immediately performed to exclude intracerebral bleeding or spinal cord compression by an intradural hematoma. Brain MRI is used to detect early any ischemic or embolic lesions; if none are detected, brain CT scan may be repeated 24-48 hours after the event. In the case of paraparesis or paraplegia, mean arterial pressure is kept above 90 mmHg, cerebrospinal fluid is drained in order to lower the cerebrospinal fluid pressure below 10 mmHg, and methylprednisolone (1 g in bolus, then 4 g over 24 hours in continuous perfusion) and 18% mannitol (5 mg/kg four times a day) are administered. MRI of the spinal cord is electively performed to confirm the diagnosis and identify lesion location and extension.

If signs of lower-limb, renal, or visceral malperfusion develop in the postoperative period, immediate diagnostic measures must be performed to assess organ blood supply and to appropriately plan surgical revascularization procedures. For a precise visualization of visceral organ perfusion, emergency digital subtraction angiography or angio-CT scanning is performed.

In uncomplicated cases, drainage tubes are removed at 36 to 48 hours postoperatively, while the intrathecal catheter for cerebrospinal fluid drainage is usually stopped after 48 hours, and then removed after 72 hours if no clinical signs of spinal cord ischemia have occurred.

A prolonged period of several days of mechanical ventilation is not unusual, especially after emergency operations, in patients with intraoperative abundant bleeding, and after longer periods of circulatory arrest. Recent studies suggest the use of noninvasive ventilation (NIV) after TAAA open surgery, also in a prophylactic fashion, in order to reduce postoperative acute respiratory failure (ARF) [20]. The benefit of perioperative NIV was demonstrated in a recent systematic review and meta-analysis of randomized trials, both for patients with established ARF as well as for those without ARF but with a high risk of common postoperative pulmonary complications [21].

In patients with severe chronic kidney disease, transient temporary hemodialysis may be needed early after surgery.

31.7 Conclusions

Open surgical repair is still considered the best therapeutic option in most patients affected by chronic Type B AD with aneurysmal evolution, especially those with associated connective tissue disorders. Recent results from our institution and from the literature confirm that surgical outcomes of dissecting TAAA open repair have improved significantly over the last decades, thanks to technical evolution, especially in the area of organ protection. However, despite adjunctive strategies, morbidity and mortality rates are still not negligible. Patient selection has to be based on a careful preoperative assessment and risk evaluation. Surgical repair of dissecting TAAA requires a multidisciplinary approach, usually in high-volume centers. If all specialists involved constantly update and refine their techniques in order to prevent perioperative complications, the outcomes of open surgery for dissecting TAAA will likely continue to improve still further in the coming future.

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AORTIC DISSECTION

patients true stories
and the innovations that saved their lives

This gem of a book on aortic dissection brings together as authors all the superstars in the field and is edited by two of them, Germano Melissano and Roberto Chiesa. The volume contains the most thorough and up-to date information on this disease entity and its treatment. Interestingly it also includes two chapters which provide perspectives of patients suffering from this serious disease which is so often unrecognized and poorly treated. Accordingly this text is a "must have" for all specialists and generalists interested in the challenging disease process of aortic dissections.

This valuable text is published on the occasion of the highly esteemed "Aortic Surgery – How To Do It" international meeting, which has been running for 14 years and enjoys a superb international reputation. The meeting has a stellar faculty and has been endorsed by many scientific societies, as well as by the Marfan Foundation (a prominent U.S.-based patients association). It is attended annually by more than 1,000 delegates and is further described at www.aorticsurgery.it.

Books that were published for previous "How To Do It" meetings, such as Thoraco Abdominal Aorta: Surgical and Anesthetic Management (2012) and History of Aortic Surgery in the World (2014), were favorably reviewed and remain an important part of most libraries – especially ones that are used by vascular and cardiac surgeons and others interested in these difficult-to-treat entities. This volume is another in this outstanding series.

In the current exemplary volume, Drs. Melissano and Chiesa, have recruited as contributors all the world innovators and leaders who are advancing knowledge in the natural history and exciting new treatments of Types A and B aortic dissections. All the contributors are widely acknowledged to be world leaders and experts in the field. Their research and clinical excellence have led to the rapid advances in the treatment of these aortic dissection disease entities – advances which are slowly being adopted around the world. Clearly the achievements of these experts has been one of the most exciting developments in vascular disease treatment of the last 3 decades. All these achievements are beautifully summarized and illustrated in this outstanding book which is without equal and an indispensable addition to the library of anyone interested in the management of aortic dissections.

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