#### ROUNDTABLE DISCUSSION

# Type B Aortic Dissection Decisions

Physicians share their approaches to TBAD scenarios, covering treatment considerations, limitations, contraindications, technology, and more.

With Robin H. Heijmen, MD, PhD; Igor Koncar, MD, PhD; Ourania Preventza, MD, MBA, FACS; and Vincent Riambau, MD, PhD



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Most questions about treating dissections seem to be regarding patients with uncomplicated type B aortic dissections (TBADs). INSTEAD-XL has produced pretty convincing evidence to treat these patients with a stent graft once they are medically managed out of

the acute phase. Have there been additional studies that reinforce or contradict this message for you?

**Dr. Heijmen:** It's clear to me that select patients do benefit from thoracic endovascular aortic repair (TEVAR) prior to the chronic phase of dissection to prevent

aneurysmal dilatation (which can be complex to treat by either open or endovascular means). However, it's not definitively clear which exact group of patients and at what interval postdissection. The only other randomized trial is ADSORB, which again demonstrated remodeling (associated with less risk of dilatation) in favor of TEVAR.<sup>1</sup> Many imaging details have been identified so far to be predictive of poor outcome. The evidence is not yet convincing, apart from aortic size at onset, which makes sense with respect to physics. Therefore, we currently meticulously select only certain patients for preemptive TEVAR.

**Dr. Koncar:** It is not easy to differentiate between complicated and uncomplicated dissection in everyday clinical practice, especially when dealing with malperfusion, resistance pain, and uncontrolled hypertension. Fattori et al proposed that not only clinical but also imaging and biochemical parameters can be used to define malperfusion, making the distinction between complicated and uncomplicated dissection very foggy.<sup>2</sup>

The INSTEAD-XL trial showed that endovascular repair covering the entry tear provides good aortic remodeling and prevents long-term complications and death. On the other hand, the study deals with patients with uncomplicated TBAD who reach the subacute phase without any complications. My focus nowadays is on patients with uncomplicated TBAD who develop complications or experience sudden death in the acute phase. Reutersberg et al showed that approximately 30% of patients with uncomplicated TBAD develop complications.<sup>3</sup> More importantly, the majority of these patients developed severe complications such as malperfusion, rupture, and aortic expansion. The limitations of this study were the low number of patients and retrospective design, although the study did present real-world situations that we face in my institution as well. Current literature does not give us enough scientific evidence to determine the subgroup of patients with uncomplicated TBAD who are at risk to develop events in the acute phase and would benefit from intervention.

**Dr. Preventza:** When we talk about uncomplicated TBAD, we should always mention the acuity of the dissection. The INSTEAD-XL trial showed a survival advantage of intervention (TEVAR) versus medical therapy that became evident at 5 years. Most of the patients treated in the INSTEAD-XL trial were in the early chronic or subacute phase; the median number of days from dissection to treatment assignment was 45 days for medical therapy alone and 39 days for medical therapy plus TEVAR. The questions that have been raised since the

results were published are mainly about the timing of intervention: Would earlier intervention have changed the results? Would less variation in the timing have changed the results?

To date, for the treatment of uncomplicated acute TBAD (UATBAD), the only prospective randomized trial is ADSORB, which showed that the stent graft induced remodeling, with thrombosis of the false lumen (FL) and reduction of its diameter. Long-term results are still needed. A study of 2010-2019 data from the Vascular Quality Initiative (VQI) TEVAR and complex endovascular aneurysm repair (EVAR) registry matched patients treated at 1 to 14 days with those treated at 15 to 90 days and found a strong association between TEVAR (versus medical therapy) and a higher risk for reintervention at 30 days and 1 year.

Regarding TBADs of > 3-month duration, the FL is the Achilles' heel. We have various ways to obliterate the FL, but long-term results are still needed, as is a fair comparison between open surgery and TEVAR in these patients. What will be the best long-term (> 10 years) solution for a 50-year-old patient with a chronic TBAD, no significant comorbidities, and with suitable anatomy for TEVAR? We could argue both ways. Depending on the part of the world where the patient is treated, they most likely will have great short-term results with either therapy. But, what will the long-term results be in similar patients 10 to 15 years down the line? Maybe it is time for 10-year follow-up of INSTEAD-XL.

**Prof. Riambau:** UATBAD management is a challenging and tricky subject that deserves more robust evidence to standardize a clear decision-making process. INSTEAD-XL demonstrated positive results for TEVAR versus best medical treatment alone in terms of aortic remodeling and late aortic complications. However, the trial included a combination of TEVAR for both subacute and chronic phases, with a quite modest sample size and only midterm follow-up. We do not know the long-term outcomes for both patient cohorts. Nevertheless, there are no additional studies that reinforce or contradict INSTEAD-XL's conclusions.

Because TEVAR is not always benign, aortic dissection is an evolving pathology and not all UATBADs have the same natural history, it seems much more appealing and fair to identify predictor factors in the early phases and selectively apply TEVAR in the subacute phase. That's the recommendation from the 2017 European Society for Vascular Surgery guidelines. In other words, by identifying "high-risk" patients during the acute phase, we can justify TEVAR and all its potential complications (eg, retrograde type A dissection,

stent graft-induced new entry, stroke, paraplegia). The remaining question is how to define and objectively detect a "high-risk" aorta at the beginning of the UATBAD. We know some of the predicting factors, but there are still important hemodynamic and functional parameters that could play a relevant role in the evolution of UATBAD and must be assessed and properly included in the decision algorithm.

#### What do we know about the differential performance of TEVAR in acute, subacute, and chronic dissections? How does this affect your decision-making?

**Dr. Koncar:** Comparing the performance of TEVAR between acute, subacute, and chronic dissections is not justified except in a randomized trial, which is difficult to design because complicated dissections rarely survive the acute phase. The most frequent comparison in the literature is either registry based (eg, VIRTUE) or systematic review based, in which patients in the acute phase were treated due to complications detected by clinical presentation and those in the subacute phase were uncomplicated or developed less severe

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complications during treatment (eg, aortic expansion, uncontrollable pain, hypertension). For example, the VIRTUE registry investigators reported outcomes of 100 patients with acute, subacute, and chronic dissections. Worse results were seen in the acute phase, where 50% presented with severe complications already indicated for intervention. On the other hand, aortic wall inflammation, fragility, and intimal flap stiffness change with time, affecting TEVAR performance and treatment outcome. Our decision-making is based on clinical presentation and imaging parameters for acute and subacute patients. In patients with chronic disease, we wait for aortic enlargement when open repair is the first choice in young and fit patients, and we reserve TEVAR for other patients.

**Dr. Preventza:** As I said before, the jury is still out regarding long-term results. I mentioned previously the results of the ADSORB trial for UATBAD and the results of the VQI TEVAR and complex EVAR registry. We also know the results of INSTEAD-XL. One of the issues is that even though the dynamic flap between the true lumen (TL) and the FL of the aorta changes and the "plasticity" of the flap varies with the chronicity of the dissection, we use the same endovascular stent grafts to treat dissections of all stages (acute, subacute, and chronic). Is adhering to the notion that "one stent fits all stages" the right way to treat these patients and obtain good long-term results? Precision medicine is important, and customizing treatment to the individual patient is imperative. Additionally, collaboration between physicians and industry is most important when using technology to treat aortic dissections. A multidisciplinary team approach and shared decisionmaking are also key when we are considering treatment for these patients.

**Prof. Riambau:** Multiple studies recommend the subacute phase (sometime between 2 and 12 weeks) as the safer and effective period for TEVAR after UATBAD. In this sense, the aorta still has enough plasticity to be remodeled (compared with chronic dissection). Moreover, TEVAR applied during the subacute phase has been associated with less risk for iatrogenic tears and secondary interventions (compared with acute repair).<sup>4</sup>

**Dr. Heijmen:** Studies have shown that the aorta remodels far better in the early phase of dissection than in its chronic phase. However, the exact cutoff is not yet clearly proven; the timing is a grey zone and it is patient specific, but it seems to range from 6 to 9 months. Preemptive TEVAR should be scheduled



within that early period. In the absence of a clear indication, wait and see, and treat the patient only when aneurysmatic. Do not ignore the benefit of strict blood pressure management in dissection patients. In the acute phase, the aorta may be too fragile, and the risk of procedure-related complications may be increased. Retrograde aortic dissection is an often-lethal complication of TEVAR that should be prevented at all costs in a preemptive procedure. Surgeons will recognize the changing aortic wall and intimal lamella structure over time in various stages of type A aortic dissection. As an example, the aorta is paper thin in the acute phase. In the chronic phase (after years), the FL allows even anastomotic suturing.

## One of the limitations in stent graft treatment is the FL failing to completely thrombose. Are there predictors of FL thrombosis after TEVAR, and can these factors be influenced?

Prof. Riambau: Regarding FL thrombosis, we should consider different scenarios according to the aortic extension. A DeBakey type IIIA dissection that stops above the diaphragm will have different behavior than dissections with abdominal extension. For type IIIA, it is easier to create a complete FL thrombosis if all the intercostals are covered with the stent graft. Type IIIB is a different scenario, with secondary tears below the diaphragm that keep the FL patent. That circumstance is quite common in UATBAD, where distal reentry tears play the role of pressure discharge. Otherwise, if there is not a reentry tear downstream, the TL could collapse with malperfusion complication. The major predictor is the presence of secondary tears below the distal edge of the stent graft. We can influence that by promoting thrombosis using the PETTICOAT, STABILISE, or knickerbocker techniques or FL embolization. A recent paper correlates the "question mark" shape of the arch with the potential FL thrombosis, but it is hard to find a clear rationale for that hypothesis.

**Dr. Preventza:** The Achilles' heel of the endovascular treatment of chronic TBAD is the fate of the FL. Various techniques have been advocated for obliterating the FL, with different results. No one really knows the long-term results of these techniques. In the report of the VQI database study that included 125 repairs for chronic aortic dissection, the authors concluded that TEVAR is safe for these patients.<sup>8</sup> Midterm results showed promising changes of the sac diameter. The extent of stent graft coverage did not affect sac shrinkage, and aneurysms > 5.5 cm were more likely to have shrinkage than aneurysms < 5.5 cm. A FL > 2.2 to 3 mm has also

been associated with lower incidences of aortic remodeling and complete FL thrombosis. One thing is certain: In most studies, there is significant heterogeneity in case selection when we focus on chronic TBAD patients, and thus the benefit of TEVAR versus alternative treatments for these patients remains uncertain.

**Dr. Heijmen:** Persistent retrograde FL perfusion is usually due to distal intimal entries. If present in the lower descending thoracic aorta, the stent graft should be extended distally. Usually, the intimal reentry is at the renal level. It has been proposed to block FL flow with a covered stent into the renal (crossing the membrane), but I have no such experience. Alternatively, an open stent (together with balloon dilation) may be used to align the membrane to the outer FL wall at the visceral level, using the so-called PETTICOAT technique.

Recently, I tend to treat the entire descending thoracic aorta, routinely up to the celiac trunk; the risk of spinal cord ischemia is considered low in the absence of left subclavian artery occlusion and/or previous abdominal aortic aneurysm repair. The further down that is stented, the lower the risk of persistent FL flow. In very select cases, I will carefully balloon dilate the stented segment above the distal end to block retrograde flow, without creating new intimal entry distally. In short, the more visceral vessels that originate from the TL and the less distal intimal (re)entries are present, the better the effect on the FL with respect to thrombosis and remodeling.

Dr. Koncar: Extension of aortic dissection from the left subclavian to iliac arteries makes treatment of this pathology very challenging. Perfusion of the FL versus thrombosis are not of the same importance in all forms of aortic dissection. In the presence of acute dissection complicated with aortic rupture, it is very important to induce FL thrombosis and prevent further bleeding. The flow in the FL may maintain perfusion of the aortic branches originating from the FL and play a protective role for a while. Additionally, partial thrombosis of the FL may induce hypoxia of the aortic wall and aortic enlargement. Likewise, the role of FL thrombosis is changing with time and becoming more important in the chronic phase, where aortic enlargement is expected as a natural history of the disease. Therefore, we should be aware of the factors influencing FL thrombosis and tailor our treatment based on its importance. Because the FL is perfused via TL-FL communications. the number and location of these communications are crucial predictors of partial or complete FL thrombosis. The ability of the stent graft to approximate the intimal flap to the outer aortic wall without causing new

lesions is very important for FL thrombosis. It depends on the stent graft, and we do need improvements of this technology. However, it also depends also on the fragility or stiffness of the intimal flap that is changing with time. The STABILISE technique has shown promising results in removing the intimal flap and thus removing the FL, which is also very beneficial.

#### What is the role of TEVAR in TBAD for patients with connective tissue disorders?

**Dr. Koncar:** The quality of the aortic wall and young age at presentation are the challenges we face when treating patients with connective tissue disorders who develop aortic dissection. Hostile aortic anatomy (especially the aortic arch) and enlargement of the aorta and its branches are aggravating factors for endovascular repair. It is clear that replacing the entire aorta with synthetic grafts would be the optimal solution for this group of patients, but this is not technically possible. Although we have such an expansive application of endovascular technology in clinical practice worldwide, we lack experience in open surgery, and the number of centers performing open repair is decreasing rapidly. Consequently, endovascular options are offered more and more—even to patients with connective tissue disorders. Hopefully, registry-based results (ie, VASCUNET, IRAD) or systematic reviews will give us more information. Regardless of our capability to perform or not, there is a group of patients with connective tissue disorders in whom open repair is not feasible or is at very high risk due to comorbidities, hostile anatomy due to previous procedures, or aortic fragility in the acute phase. In these patients, TEVAR is a very reasonable solution, even if it is only a bridge solution.

**Dr. Preventza:** This is a great topic. We have previously described our experience with these patients in *European Journal of Cardio-Thoracic Surgery*. Recently, we submitted an invited featured article on endovascular therapy for patients with heritable thoracic aortic disease to *Annals of Cardiothoracic Surgery*'s focused issue on thoracic endovascular aortic solutions, scheduled to be published in November 2021. We are also outlining the indications for TEVAR in these patients for another upcoming focused issue.

**Prof. Riambau:** Classically, connective tissue disorders are outside the endovascular scope. Nevertheless, TEVAR can play a positive role in acute complicated TBAD. Another comprehensive indication is for a redo surgery when the thoracic endograft can be deployed proximally and distally in previous vascular grafts. With this, we can avoid secondary iatrogenic intimal tears.

**Dr. Heijmen:** This is not an issue when the proximal and/or distal landing zones are prosthetic after previous open aortic repair. If not, the fragile aorta may increase the risk of retrograde dissection type A proximally and new intimal lesions distally. Patients are usually young, so open surgery might be preferred if it is considered doable. Otherwise, you should carefully balance the pros and cons. Do not exaggerate oversizing, refrain from using bare stents, and follow-up the patients meticulously.

#### In which cases is a dedicated dissection device best suited?

Dr. Preventza: To my knowledge, the only dedicated dissection device approved by the United States FDA is the Zenith TX2 dissection endovascular graft (Cook Medical). In our practice, we have used this stent in cases of acute TBAD with clinical or radiologic malperfusion. Dr. Jayna Patel and colleagues recently analyzed outcomes data with this particular stent at 10 European centers. 10 One hundred twenty patients were treated for TBAD in its acute, subacute, or chronic phase. Stroke and paraplegia rates were 2.5% and 4.2%, respectively; 30-day mortality was 5.8%; and mortality at 30 to 90 days was 1.7%. The overall type la endoleak rate was 6.7% in the perioperative period. Of course, mid- and long-term results are needed when we evaluate this stent to determine how it can help in the long term.

**Dr. Heijmen:** Ideally, in all cases. We currently treat degenerative aneurysms with the same stent grafts as dissections, although the different specifications may favor one over another depending on patient and anatomic details. In the chronic phase, we favor tapered devices to reduce the risk of distal new intimal entries. Gradual distention (over weeks) to a nominal diameter distally is ideal. In gothic arches, there will be a lot of force on the outer curvature of the proximal landing zone. Orthogonally aligning angulated stent grafts may be an option.

**Prof. Riambau:** If we had a dedicated device for each specific aortic pathology, we would apply the dedicated device every time according to the pathologic subtracts. In dissection, we would need a compliant, conformable device that is very smooth and soft on both edges, mimics the biomechanical behavior of the aortic wall, and respects the aortic anatomic shape, avoiding any stress or secondary injury over time. However, current devices are still far away from this ideal device. We hope that the future technologic developments will reach our wishes.

### What strategies do you employ to prevent FL aneurysms? How do you manage existing FL aneurysms?

**Dr. Heijmen:** In most instances, we treat chronic postdissection aneurysms by open surgical means. In recent years, we've performed staged repair, using TEVAR for the descending part in the presence of adequate proximal landing zone, and open surgery for the visceral segment, as it can be very cumbersome to exclude the dilated chronic FL from distal reentries by endovascular means only. We have had some referrals of failed candy plugs (and coils) that complicate open surgical conversion. The same accounts for failed PETTICOATs in which all visceral ostia together with the lumbars are covered with the open stent, and surgical reimplantation is almost impossible.

Therefore, I prefer to focus on preventing the FL from dilating over time—not only with strict blood pressure management but also with preemptive TEVAR in case of early FL dilation (> 4 cm) within the first months postdissection. I do this by treating the entire descending thoracic aorta and balloon dilating in the distal segment to block retrograde FL flow, but only when the procedure is low risk for retrograde dissection and/or spinal cord ischemia.

Prof. Riambau: To prevent FL aneurysm formation, reduce the blood pressure to the FL. Then, cover the main entry tear; this is the first target point but not the only one. Again, aortic remodeling would be more feasible for DeBakey type IIIA dissection—covering and excluding FL in the descending thoracic aorta. In contrast, due to the distal extension, type IIIB could need additional procedures (eg, STABILISE). However, the hemodynamics between TL and FL play a significant role. Not every perfused FL has pressurization. It depends on the flow discharge at the reentries in the distal aspect of the aorta. This information is not always easy to assess. It would need dedicated MRA, software, and future validation. Hopefully, this will be available in the near future to help us identify who needs further therapeutic or preventive maneuvers.

**Dr. Preventza:** We know that approximately 30% of patients with medically managed acute TBAD present for a late intervention (usually 5 years after initial presentation).<sup>11</sup> We also know that strict blood pressure management can benefit these patients in terms of the fate of the proximal part of the descending thoracic dissected aorta.

In our practice, we use open and endovascular repair to treat FL aneurysms, depending on the patient and

the situation. Younger patients with no comorbidities and with thoracoabdominal aneurysms are usually treated with extent I, II, III, or IV open repair according to the extent of the disease. For patients with significant comorbidities and FL aneurysms, we always seek alternative endovascular options. For all patients, regardless of the mode of therapy (open surgery vs endovascular therapy vs conservative treatment), lifelong surveillance is extremely important.

Regarding achieving FL obliteration, we use balloon fracture and fenestration more often than other techniques, but I'm not certain that anyone knows which technique is best for achieving FL obliteration in these cases.

**Dr. Koncar:** In my institution, we base our treatment on covering entry tears because other devices are not available in my country. Besides TEVAR, we frequently use iliac extension to cover distal entry tears in iliac arteries and infrarenal aorta and covered and parallel stents to cover communications in visceral branches (usually seen at the origin of the branch). We hope to start the STABILISE and candy-plug techniques in the future. I find both very promising at inducing FL thrombosis and aortic remodeling.

# Do you feel there is any contraindication to endovascular management for acute TBAD? In which cases does open surgery remain the best option?

**Prof. Riambau:** The major contraindication is related to poor proximal landing zone conditions, meaning when the dissection extends proximally to the landing zone (Z0, Z1, Z2) or when the landing zone is already dilated or not suitable for any thoracic stent graft. In such scenarios, open repair with frozen elephant trunk has been demonstrated to be a good alternative, at least as a first therapeutic step.

**Dr. Preventza:** The current landscape supports endovascular therapy for most patients with acute TBAD. I think we all agree that medical therapy should be always included in the algorithm for treating acute TBAD. TEVAR should be considered for complicated (ie, cases involving rupture or malperfusion) and highrisk cases (ie, cases involving refractory pain, refractory hypertension, bloody pleural effusion, radiographic malperfusion, entry tear on the lesser curvature of the aortic arch, readmission, aortic diameter > 40 mm, or FL diameter > 22 mm). Whether early TEVAR should be performed in the acute or subacute phase is still debatable, but it appears that TEVAR in the subacute

phase may be associated with fewer reinterventions during the first 14 days after primary intervention. Although feasible, open surgical repair for the acute phase of TBAD has fallen out of favor because of the high morbidity and mortality associated with this treatment option. One scenario in which open repair could be considered is chronic TBAD with an aneurysm not suitable to endovascular repair in a patient with acute symptoms and acute-on-chronic dissection. In this case, the open operation will involve the same steps as open repair of thoracoabdominal aneurysm.

**Dr. Heijmen:** I have some doubts about the effectiveness and durability of complete endovascular repair of a chronic postdissection thoracoabdominal aortic aneurysm. Therefore, we prefer an open or hybrid approach with TEVAR first, then finalizing with type IV open repair. In case of acute FL rupture, one must ensure the stent graft completely excludes FL flow, and careful distal balloon dilatation may be necessary. Alternatively, open surgical repair is a valid option in such an emergency to stop acute bleeding.

In case of preemptive TEVAR (to prevent future dilatation), the procedure should be done at virtually no risk. Careful sizing, planning, execution, and follow-up are mandatory and require experience. Such patients should be in regional specialized aortic (endovascular) centers.

**Dr. Koncar:** I would not claim any contraindication for endovascular management of TBAD because it is a life-threatening condition. In clinical practice, we balance solutions and choose the optimal one by considering the early and long-term results, patient quality of life, and cost. In the acute settings, I don't advocate for open surgery in terms of total aortic repair; however, hybrid procedures are very useful in these acute settings. In my experience, covering the left subclavian is frequently needed. This artery is very important for brain and spinal cord perfusion, and thus revascularization is important. If we want to preserve 20 mm of the neck, even in a ortic dissection, then more complex debranching procedures are needed. These patients should be discussed with cardiac colleagues, and an individual decision must be made for every patient. Open repair of the aortic arch with frozen elephant trunk may be reasonable.

### Which are your expectations about the potential usefulness of new technologies like long septotomy?

**Dr. Heijmen:** This may prove to be a valid alternative to all other techniques currently employed to exclude the chronically dilated FL from persistent retrograde

flow. However, at the segment of septotomy, the dissected aorta should not be extensively dilated yet to allow the stent graft to align to the outer (FL) aortic wall.

**Dr. Preventza:** Any new technology addressing aortic dissection, including long septotomy, is welcome. The most important thing is that these patients receive lifelong surveillance to monitor their long-term results.

**Prof. Riambau:** Long septotomy has been associated with unpredictable outcomes. By definition, it is not a reconstructive repair but rather is closer to a destructive maneuver. It could be useful in selective malperfusion syndromes, but not as a routine technique for any TBAD. Other technologies related to new devices and approaches, like focal biomimetic aortic patches to cover focal tears, are now developing. As mentioned previously, dynamic diagnostic techniques will be useful to assess the aortic prognosis and the need for additional interventions.

**Dr. Koncar:** For such a complex pathology, we are far from "one technique fits all." We should be familiar with as many solutions as possible—both open and endovascular. Because intimal flap is one the main features of aortic dissection, techniques that remove it are useful and promising.

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