The first thoracic endovascular aortic repair (TEVAR) was performed by Dake et al in July 1992.1 Over the past 25 years, TEVAR has become the gold standard for treatment of the majority of thoracic aortic aneurysms (TAAs) and complicated type B thoracic aortic dissections (TADs). TEVAR failure modes vary and are dependent on the type of pathology being treated; TAA failures result in endoleaks, and TAD failures result in persistent false lumen perfusion. The rates of reintervention after TEVAR also vary and are lowest for TAAs (15%–20%), higher for chronic TADs (30%–35%), and highest for acute TADs (46%).2–5

There is little doubt that repair is required for type I endoleaks from proximal or distal stent graft fixation site failures, type III endoleaks from stent graft component separation, and gutter endoleaks in parallel stent graft TEVAR. However, treatment options for type II endoleaks as a result of retrograde flow into the TAA from smaller-caliber arteries (intercostal arteries) and larger-caliber arteries (left subclavian and celiac arteries) remain controversial. Treatment is usually reserved for patients that experience associated aneurysm growth, which is unlikely unless it is the left subclavian and/or celiac artery that was covered with the stent grafts and they are the source of type II endoleaks. Following TEVAR, endoleaks from stent graft porosity (type IV) and endotension (type V) are rare and are usually treated with relining of the previous stent grafts.

Persistent false lumen perfusion after TEVAR for TAD is associated with increased incidence of stent graft failure as well as aortic aneurysm- and dissection-related mortality. Management strategies for persistent false lumen perfusion are evolving and primarily focus on embolization with a variety of agents, occluding dissection septum fenestrations responsible for false lumen perfusion, or a combination of both.

TEVAR REINTERVENTION FOR ENDOLEAKS

Type I endoleaks after TEVAR most likely occur from inadequate proximal or distal seal zones or stent graft oversizing of > 30%. Multiple approaches have been taken to promote both aneurysm sac thrombosis and endoleak flow channel elimination. Placing proximal or distal stent graft extensions into nonaneurysmal and dissection-free aorta is often needed to obtain a seal. This sometimes requires extra-anatomic surgical or endovascular reconstruction of the arch or visceral arteries. When treating complex thoracic aortic pathology, the use of Heli-FX EndoAnchors (Medtronic) has been shown to decrease the rate of stent graft migration and type I endoleaks.6 In select patients with type I endoleaks and narrow channels, catheter-directed embolization techniques have also been described.

The majority of type II endoleaks, particularly when they arise from smaller branch intercostal arteries, are considered benign and do not necessarily mandate intervention. When thoracic stent grafts are placed in zone 2, the left subclavian artery can become the source of type II endoleak, and similarly, when the distal stent graft landing zone is beyond the celiac artery, it too can become the source of type II endoleak. These larger arteries rarely thrombose on their own and often require catheter-directed embolization. The route of catheter insertion (transradial/bra-chial/femoral or translumbar/thoracic) is dependent on the extent of TAA and TAD morphology.

Type III endoleaks resulting from stent graft junctional failures can often be addressed by relining the existing stent grafts with a new device to bridge between the existing components.7 Heli-FX EndoAnchors may be used to seal these junctions by promoting stent-to-stent fixation, although long-term durability has not been established. Endoleaks resulting from endograft fatigue, graft tear, or stent fracture can often be addressed by relining the defective graft. Open repair is rarely necessary.
Gutter endoleaks are observed when a variety of parallel stent graft combinations are used for managing complex TAAs and TADs. The natural history of gutter endoleaks remains ill defined and likely depends on pressure transmission across the gutter endoleak. Studies have shown that in the absence of thrombosis, pressure transmission via endoleak channels is unchanged regardless of its length or diameter. However, when an endoleak has thrombosed, pressure reduction is directly proportional to its length and inversely proportional to the diameter of its channel. Gutter endoleaks that persist may transmit systemic pressure to the aneurysm sac and could be treated by catheter-directed embolization techniques. The success of gutter endoleak embolization in pressure reduction to the aneurysm sac depends on its length and diameter.

**TEVAR REINTERVENTION FOR FALSE LUMEN PERFUSION**

The primary goals of endograft placement in TAD are true lumen expansion and false lumen thrombosis. The success of TEVAR for TAD is dependent not only on coverage of the proximal septal fenestration (entry tear), but also the number and size of distal septal fenestrations (reentry tears). False lumen remodeling is often impaired in patients with persistent false lumen perfusion following TEVAR, and these patients are at the greatest risk for late aortic-related morbidity and mortality.

Distal fenestrations can be sealed by stent graft extension. Shorter stent graft use has an increased rate of persistent false lumen perfusion and thus a need for secondary interventions. However, there is a direct correlation between the length of coverage and risk of spinal cord ischemia and paralysis, which must be taken into consideration when planning repair. Distal extension with a bare-metal stent at the time of TEVAR has been studied. Although it promotes false lumen thrombosis and true lumen expansion, more studies are needed to show definitive benefit over TEVAR alone. In patients with subacute or chronic dissection, fenestrations may be sealed with occlusive plugs through the septum or covered stents into large branch vessels (eg, mesenteric or renal arteries).

Promoting complete thrombosis of the false lumen will change the pressure gradient and flow dynamics of the aorta. Multiple strategies exist to address this issue, and efficacy varies depending on overall aortic diameter and size ratio of the true to false lumen.

**False Lumen Embolization**

Embolization of the false lumen can be achieved with selective catheterization through a septal fenestration from either the antegrade or retrograde approach (Figures 1 and 2). Coil embolization is used with good efficacy in smaller lumens. Coils can be used to create a dense nest of embolic material, with angioplasty balloons used to prevent migration during deployment. Amplatzer vascular plugs (Abbott Vascular, formerly St. Jude Medical) have also been used to occlude the false lumen channel, with and without adjunctive embolic material.

In June 2007, Tsai et al published their findings from the International Registry of Acute Aortic Dissection and identified partial false lumen thrombosis as an independent predictor that significantly increases mortality. To better understand the implications of false lumen embolization (FLE) to induce thrombosis in patients who undergo TEVAR, Mehta et al first performed TEVAR and FLE in July 2007 and analyzed 104 patients with complicated acute and chronic TAD who underwent TEVAR with FLE (n = 40; 37.7%) and compared them to those who underwent TEVAR without FLE (n = 66; 62.3%). The false lumen embolic agents included Amplatzer vascular plugs and detachable coils. Data were prospectively collected and analyzed for 30-day and long-term mortality, spinal cord ischemic complications, and the need for secondary interventions. Results showed that TEVAR with FLE patients experienced a significantly lower 30-day mortality (2.6% vs 9.6%). In TEVAR with FLE patients, there were no spinal cord ischemic complications, and 29% of the patients required one or more additional catheter-directed FLE procedures.
during follow-up. At a mean follow-up of 18 months, 88% of TEVAR with FLE patients had a significant decrease in all false lumen-to-systemic pressure indices, and 65% had significant false lumen remodeling with > 5-mm maximum diameter reduction. There were no thoracic or abdominal aortic ruptures, conversions to open surgical repair, or deaths. These findings were the first to suggest that FLE techniques to promote thrombosis should be considered when managing complex acute and chronic TAD.

Other adjunctive techniques have also been described for creating false lumen thrombosis. In situations of large false lumen channels, thoracic stent grafts can be partially deployed on the backtable, restraining sutures tied in the middle of the stent grafts that would refrain from full deployment and allow for the inner delivery catheter removal, thus creating the candy-wrap plug. This stent graft is delivered and deployed into the false lumen, and an Amplatzer vascular plug is used to occlude the remaining narrow opening, which obliterates retrograde flow into the false lumen. Both techniques have shown short-term efficacy and durability. As with any new technique, deployment of two stent grafts (one within the true lumen and the second within the false lumen) using the candy-wrap plug technique potentially increases the amount of radial force within the dissected aortic wall, which inherently is weakened, and there is a potential of aneurysmal degeneration and possibly rupture. The Knickerbocker technique mitigates some of this risk by limiting expansion to only the midportion of the endograft.

These findings on false lumen thrombosis suggest that total aortic replacement is rarely necessary, unless there is aneurysmal degeneration within the paravisceral or infrarenal segment. Full aortic coverage can be accomplished endovascularly with fenestrated devices or parallel grafts.

SUMMARY

TEVAR remains a durable option for aortic pathology with a low rate of secondary intervention. Elimination of endoleaks in TAAs is necessary to prevent further aneurysmal expansion. Although TADs are also at risk of aneurysmal degeneration from persistent false lumen perfusion, the approach to management should be different than aneurysmal disease. Complete false lumen thrombosis, but not seal, is necessary for mortality reduction and aortic remodeling.