The TEVAR Landscape in 2012

A status report on thoracic endovascular aortic repair.

BY FRANK J. CRIADO, MD, FACS, FSVM

Endovascular developments have changed the entire field of thoracic aortic surgery in a profound and irreversible manner. The true beginnings can be traced back to the first stent graft procedure for repair of a descending thoracic aortic aneurysm (TAA) performed by Dake et al in 1992.[1] Looking back, it was almost inevitable then that endovascular solutions for the thoracic aorta would emerge after the landmark procedure for treatment of an abdominal aortic aneurysm (AAA) by Parodi and associates in 1990 and Volodos' first-ever endovascular aortic repair in 1986.[2]

When compared historically with their abdominal counterpart, thoracic devices and techniques have lagged behind in terms of caseload and refinement. But, to their advantage, thoracic endovascular approaches have been embraced with enormous enthusiasm and acceptance, almost from inception, because of the widely acknowledged unmet needs in the field where open surgical treatment puts patients through maximally invasive and technically difficult operations that have been mastered only by a select few centers of excellence around the world.

The perception of a poorly served patient population has thus evolved, providing great impetus for the creation of less-invasive therapies. The new era in thoracic aortic surgery is upon us today: it can be defined with a single acronym, TEVAR (thoracic endovascular aortic repair), in which the "A" stands for aortic, reflecting the varied multiple pathologies encountered in the thoracic aorta. This is different from the "A" of EVAR (endovascular aneurysm repair), which signifies *aneurysm*, because AAA is the predominant and almost exclusive target pathology in the abdominal segment.

THORACIC AORTIC ANEURYSM

Although less frequent than AAA, TAA remains a serious problem because affected patients face a rather limited 20% to 54% 5-year survival expectation (due to rupture when
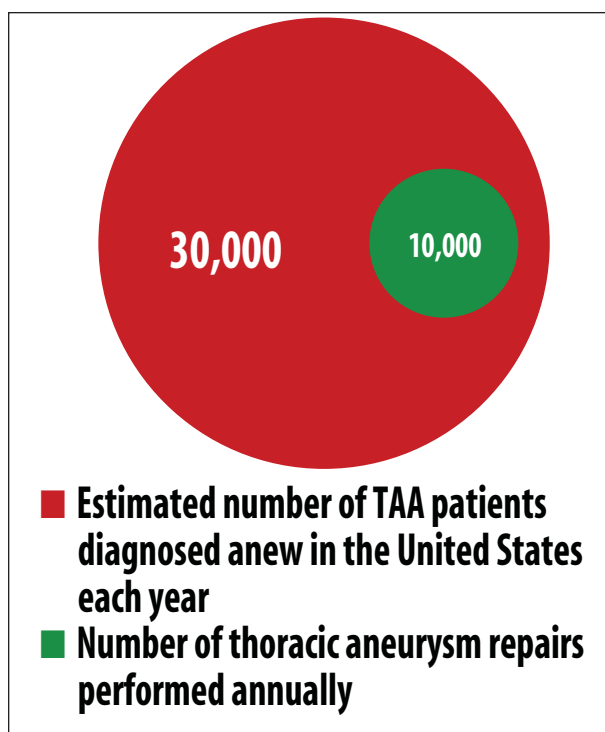

Figure 1. Estimated number of TAA patients diagnosed in the United States each year compared to the number of thoracic aneurysm repairs performed annually.

left untreated).[3,4] The incidence of TAA is said to be 10.4 per 100,000 person-years[5] or approximately 30,000 new cases annually (minimum) in the United States alone; the corresponding figure for AAA is approximately 200,000. The actual numbers are likely to be even higher, given the asymptomatic nature of TAA disease in 95% of cases. The true magnitude of the disease total in the United States (number of patients harboring a TAA) can be approximated from its known incidence and a comparison with the more reliable data available for abdominal aneurysms (Figure 1).[3]
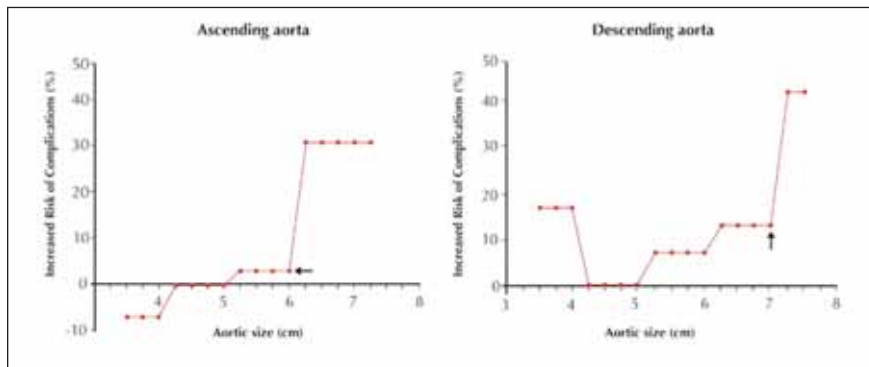
Figure 2. Hinge points of aortic diameter. Reprinted from the *Journal of the American College of Cardiology*, 55/9, Elefteriades JA, Farkas EA, Thoracic aortic aneurysm: clinically pertinent controversies and uncertainties, 841-857, Copyright 2010, with permission from Elsevier.[6]
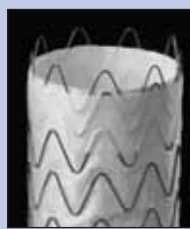
Thoracic aneurysms larger than 5.5 cm in diameter carry a yearly rupture risk of 15% and must be considered for elective repair.[4,7,8] Ruptured TAAs (rTAAs) occur at the rate of 3.5 per 100,000 persons per year—far lower than the number of ruptured AAAs (rAAAs). Intriguingly, the incidence of acute aortic dissection (AD) and rTAA are almost identical.[9] Overall mortality rates for rTAA approach 97% among those who reach the hospital alive,[10] and most experts would agree that a thoracic aneurysm is a more efficient killer than AAA because rupture tends to cause rapid exsanguination with little, if any, anatomical opportunity for the kind of tamponade and temporary containment often seen in rAAA cases.

TAAs are designated by their anatomic location and extent: ascending, arch, descending, and thoracoabdominal aortic aneurysms (TAAAs). The descending thoracic aorta is the most common location (30%–40%). Anatomic segment notwithstanding, all aortic aneurysms share a com-

mon pathogenesis and essentially the same risk factors: advanced age, male gender, cigarette smoking, atherosclerosis, hypertension, and genetic predisposition. Cigarette smoking is unequivocally the most important modifiable risk factor.[11]

Historically, most TAAs (and AAAs) were labeled to be atherosclerotic in nature, a misnomer that has been corrected at present with the more proper term *degenerative*. But it should be noted that both diseases—atherosclerosis and aortic aneurysm—often coexist and tend to be aggravated by the same risk factors. Degradation and loss of collagen and elastin in the aortic wall are the pathogenic hallmarks of aneurysm formation. For reasons that are not yet clear, there is an apparent cross-link between TAA disease and intracranial aneurysms, with some experts suggesting that thoracic aneurysm patients should undergo cerebrovascular imaging to uncover occult and potentially dangerous aneurysms in the head.

The Yale database, with more than 3,000 patients, has produced much valuable information on the nature of TAA disease.[12] Noteworthy facts include: TAA disease is largely genetic in nature, with a predominantly autosomal-dominant inheritance; matrix metalloproteinase enzymes are activated in the pathogenesis of TAA; wall tension approaches the tensile limits of aortic tissue at a diameter of 6 cm; by the time a TAA reaches 6 cm in diameter, 34% of patients have suffered dissection or rupture; and, extreme physical exertion and severe emotion can and do precipi-

March 2005	May 2008	June 2008	April 2011	August 2011
Gore TAG	**Cook** Zenith TX2	**Medtronic** Talent Captivia	**Medtronic** Valiant Captivia	**Gore** Conformable TAG

Figure 3. A timeline of FDA approvals for TEVAR stent grafts.

tate acute aortic dissection.

The incidence of TAA seems to be on the rise, although this could be related to enhanced detection amid the ever-increasing use of diagnostic imaging. However, some evidence appears to suggest an actual bona fide increase in the true incidence.[8,13,14] TAA growth tends to be slow and indolent: approximately 0.3 cm per year in the descending thoracic aorta and 0.1 cm per year for the ascending thoracic aorta. Rapid enlargement is usually associated with an intercurrent aortic dissection.[6]

Lifetime analysis of the TAA's risk of rupture and dissection[11] has uncovered clear-cut "hinge points" in the aortic diameter at which rupture or dissection are likely to occur (Figure 2): 6 cm in the ascending and 7 cm in the descending thoracic aorta. These are the sizes (presumably) where the wall tension approaches (or exceeds) the elastic limits of the aortic wall;[6] it is not at all clear why the descending
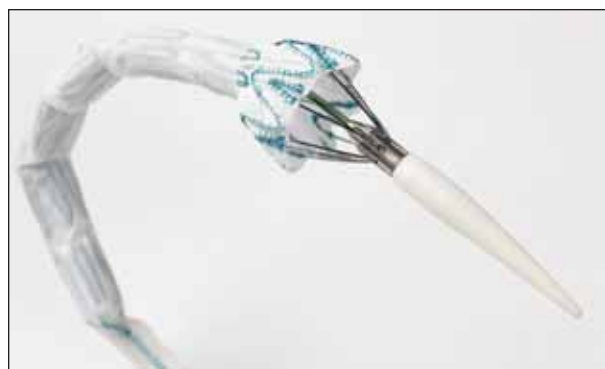
Figure 4. Relay thoracic stent graft (Bolton Medical, Inc., Sunrise, FL).

aorta tends to rupture at a larger size. It is therefore possible, at least conceptually, to prevent death from thoracic aortic

TABLE 1. KEY FEATURES OF THORACIC STENT GRAFTS								
Company Name	Product Name	Stent Material	Graft Material	Stent Expansion	Graft Length (cm)	Diameters (mm)	Introducer Sheath Required	Delivery System (F)
Bolton Medical	Relay Thoracic Stent Graft[a]	Nitinol	Woven polyester	Self-expanding	Straight: 10, 15, 20, 25; tapered: 15, 20, 25	Straight: 22–46; tapered proximal: 28–46; distal 24–42	No	22–25 (OD)
Cook Medical	TX2 With Pro-Form	Stainless steel	Woven polyester	Self-expanding	Proximal: 12–21.6; tapered proximal: 15.2–20.8; distal: 13.6–20.7	Straight proximal: 28–42; tapered proximal: 32–42; distal: 28–42	No	23, 25 (OD)
Gore & Associates	Gore TAG Thoracic Endoprosthesis	Nitinol	ePTFE	Self-expanding	10, 15, 20	26, 28, 31, 34, 37, 40, 45	Yes	20, 22, 24 (ID)
	Gore Conformable TAG Thoracic Endoprosthesis					Straight proximal: 21, 26, 28, 31, 34; tapered proximal: 31, 26; distal: 26, 21		18, 20, 22, 24 (ID)
Medtronic, Inc.	Talent Thoracic Captivia	Nitinol	Dacron polyester	Self-expanding	11, 16, 20	22, 24, 26, 28, 30, 32, 34, 36, 38, 40, 42, 44, 46	No	22, 24, 25 (OD)
	Valiant Thoracic Captivia							
Abbreviations: ePTFE, expanded polytetrafluoroethylene; OD, outer diameter; ID, inner diameter. [a]The Relay Thoracic Stent Graft is not currently FDA approved, but approval is expected in 2012.								

rupture by undertaking repair before the aorta reaches such a dangerous diameter. The 5.5-cm-diameter threshold for intervention emerges as the most reasonable for the majority of patients.

Available evidence on mechanical forces and wall behavior supports the notion that the aorta becomes quite literally "a rigid tube" as it reaches 6 cm in diameter in which the systolic expansion ceases, and the full force of ventricular contraction translates into wall stress, leading to rupture (or dissection). Certain clinical conditions might warrant an even more aggressive attitude, such as patients with Marfan syndrome, bicuspid aortic valve, and those with a family history of aortic dissection. Symptomatic aneurysms (5% of the TAA population), on the other hand, should be repaired regardless of size.

Surgical treatment of TAA was reported as early as 1951.[15] Operative techniques and perioperative care have improved enormously over the past several years, allowing skilled surgeons to perform extensive and complex thoracic aortic surgery with relative safety and excellent outcomes. But, remarkably, such excellence is confined to a few centers worldwide.[16] Furthermore, many patients are deemed non-candidates for such highly invasive operations because of
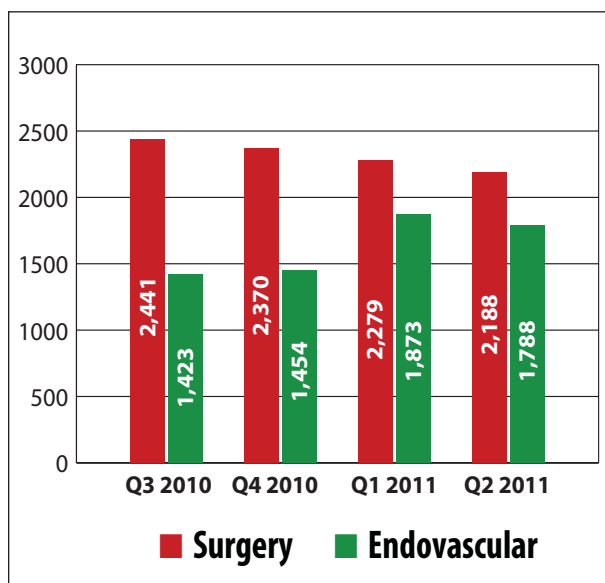

Figure 5. Current estimate of repair procedures performed for TAA repair. Reproduced from the United States Vascular and Endovascular Monitor Panel Report, 2nd Quarter, September 2011.

Figure 6. Bird-beaking occurs from malapposition to the lesser curve when a stent graft is placed at or across the knuckle of the aortic arch.

serious medical comorbidities and the fear of complications and death. This explains better than anything else the rapid rise of TEVAR and its transformational influence.

There are currently four FDA-approved thoracic stent graft devices: Gore TAG (Gore & Associates, Flagstaff, AZ), Zenith TX2 (Cook Medical, Bloomington, IN), Talent (Medtronic, Inc., Minneapolis, MN), and Valiant (Medtronic, Inc.) (Figure 3). Formal commercial launch of the latter is expected in the near future. A fifth thoracic device, the Relay stent graft (Figure 4), has completed pivotal trial enrollment and awaits regulatory approval (anticipated for 2012). The various devices differ in several important aspects (Table 1). They have been approved for endovascular treatment of fusiform and saccular aneurysms of the descending thoracic aorta, as well as penetrating aortic ulcers (PAUs), with the exception of Gore TAG, which received a TAA indication only. FDA approval was based on the 1-year results achieved in the various trials that were designed and conducted to test each device in the clinical arena.[17-20]

TEVAR developments have transformed the thoracic aortic surgery landscape (Figure 5). Advances in aortic imaging have contributed significantly as well. An important retrospective review of rates of TAA repair in the United States from 2000 to 2007 published by Walker et al in 2010 was illuminating in this regard:[21] the open repair rate was observed to increase from 3.3 per million in the 2000 to 2002 time period, up to 5.6 per million in 2003 (when multislice computed tomographic scanners were introduced). The TEVAR repair rate changed dramatically from a low of 1.2 per million in 2005, moving sharply upward to 6.1 repairs per million in 2006 after the first FDA approval of a thoracic stent graft (Gore TAG).

From a technology standpoint, thoracic devices got through the "infancy" phase in the 1990s and early 2000s and are now beginning to reach "early adulthood." Although the list of TEVAR accomplishments is robust and impressive, several important issues remain to be conquered, chiefly:

· Conformability to the bend, or knuckle, of the distal arch to make it possible for endografts to hug the lesser curve without bird-beaking (Figure 6). Such a prob-
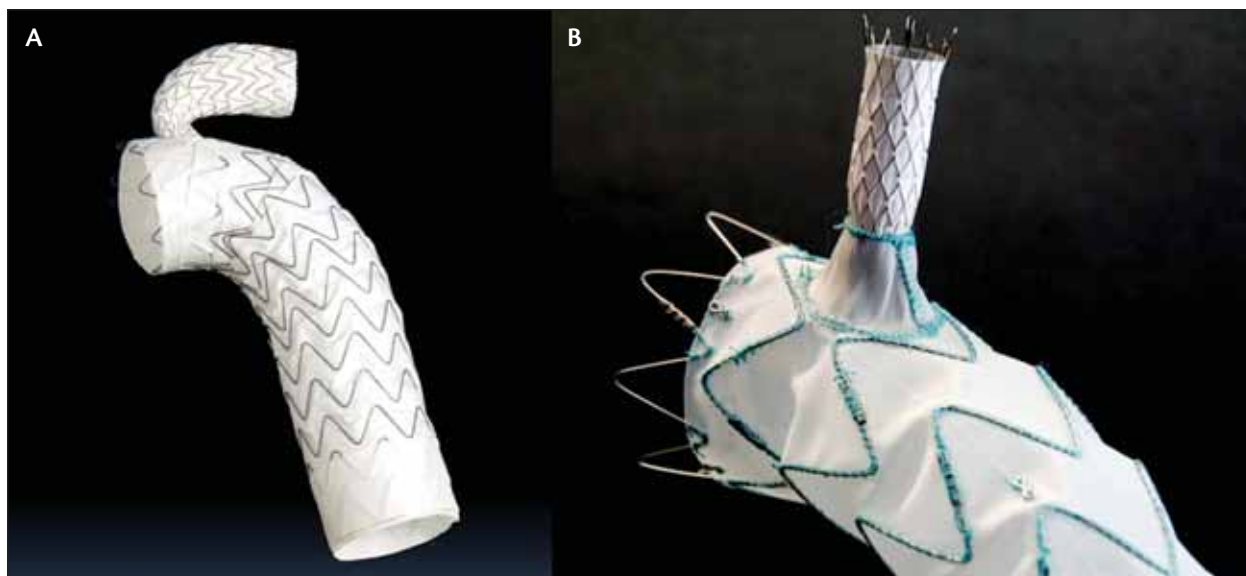
Figure 7. Branched thoracic stent graft development. Gore's branched thoracic stent graft prototype (A). Medtronic's branched thoracic stent graft prototype (B).
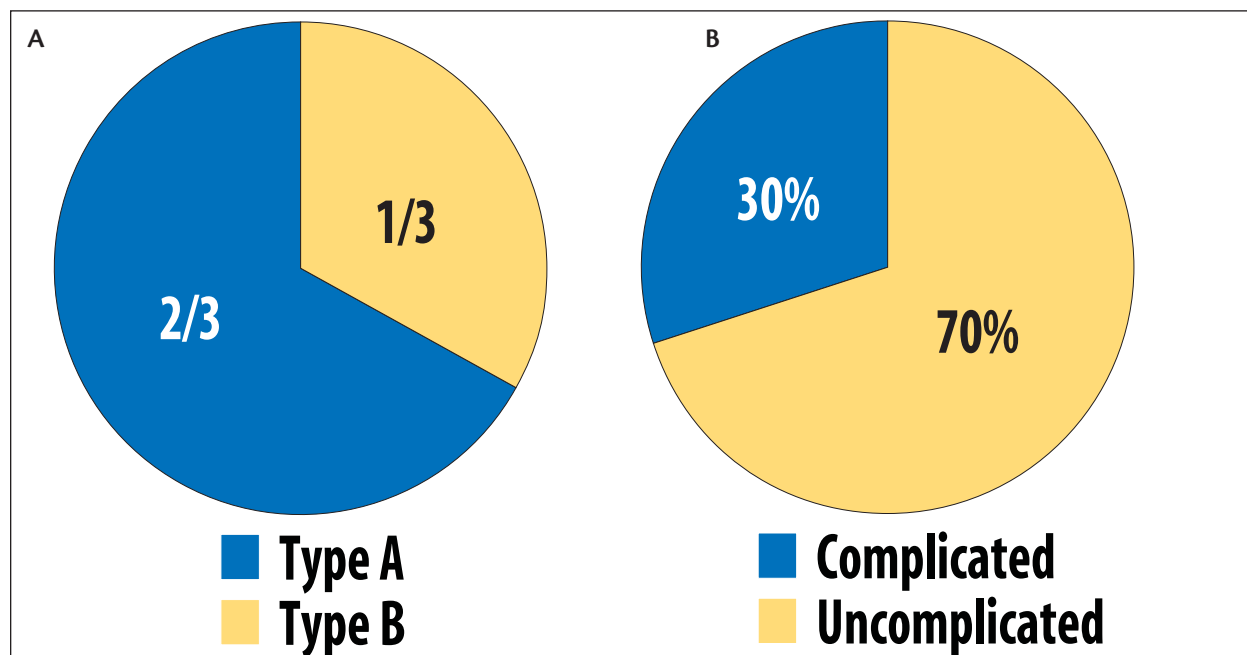
Figure 8. There are an estimated 10,000 total new cases of aortic dissection in the United States each year, two-thirds being type A and one-third being type B (A). There are an estimated 3,000 cases each year of acute type B dissection, 30% of which are complicated, and 70% of which are uncomplicated (B).

lem can result in malapposition and proximal seal failure and type I endoleak, poor fixation, and even graft collapse in some cases. More recent device developments may have already addressed such issues: Medtronic's Valiant Captivia, Cook's Zenith TX2 Pro-Form, and Gore's Conformable TAG devices represent the latest-available design enhancements that would appear to have largely resolved such shortcomings.

- Lower-profile delivery systems with an outer diameter of < 22 F are eagerly awaited. This will be most helpful to minimize access-related complications and expand the applicability of TEVAR as women (who have notoriously small and fragile access arteries) make up > 20% of patients overall.
- Aortic branch management has long been recognized as a critically important next frontier and represents a significant research and development project for every major company in the field. Multiple iterations and designs will likely be developed and become available in the years to come (Figure 7). Meanwhile, the use of adjunctive techniques (chimney technique, etc.) and hybrid combinations (debranching included) continue to gain momentum at the present time.

AORTIC DISSECTION

Acute AD is the most common fatal aortic catastrophe, resulting in more deaths annually than ruptured AAAs. The incidence in the United States is estimated to be relatively low at 10 to 15 cases per 100,000 adults annually, amounting perhaps to 10,000 new cases each year (Figure 8).[22] AD's frequently malignant clinical course justifies its status as a major cause of cardiovascular morbidity and mortality. An intimomedial tear, or entry tear, allowing the powerful aortic blood flow to rip into the wall is the initiating event, resulting in the creation of a secondary flow channel, or false lumen (FL), that propagates distally in a spiraled (more commonly) or straight fashion to involve various extents of the aorta. This frequently occurs all the way down to the bifurcation and into one or both iliac arteries.

The FL can also propagate proximally. The pressurized FL tends to compress the true lumen (TL) in the chest (and beyond), sometimes to the point of near-collapse with impediment of distal blood flow to the viscera, the spinal cord, and the lower extremities below. The entry tear is almost always located in the thoracic aorta—ascending aorta in type A and descending aorta distal to the origin

COMPONENTS OF ACUTE AORTIC SYNDROME

- Aortic dissection
- Intramural hematoma
- Penetrating aortic ulcer
- rTAA

(Permission for use granted by Cook Medical Incorporated, Bloomington, Indiana.)

Figure 9. Zenith TX2 TAA stent graft.

of the left subclavian artery in type B. Secondary, or reentry tears (fenestrations), on the other hand, can occur in the distal thoracic and/or the abdominal aorta.

Medial degeneration of the aortic wall is the underlying anatomic defect that sets the stage for AD to occur. Inherited connective tissue disorders such as Marfan syndrome (and Ehlers-Danlos or Loeys-Dietz syndromes) and other TAA and AD familial syndromes can serve as the root cause in some cases.[23] But it seems that the majority of AD patients develop dissection from severe, often uncontrolled or poorly treated arterial hypertension that induces severe degenerative changes in the aortic wall over time. It is not certain whether hypertension alone can cause AD without a predisposed aorta. The likelihood of dissection occurring is also influenced by diameter because dilatation of the aorta results in increasing wall tension and mechanical stress.[12]

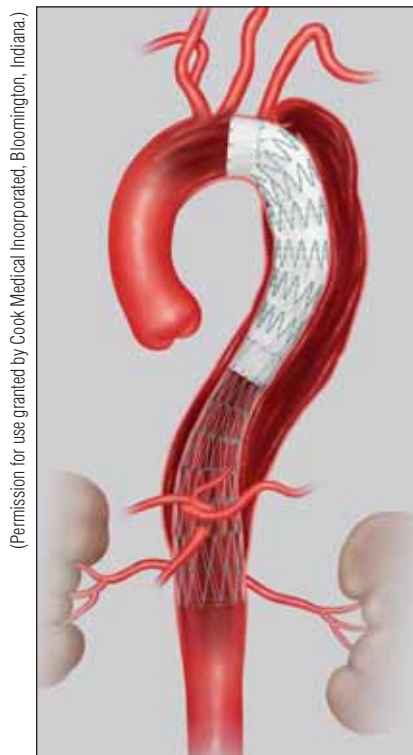Data from the International Registry of Acute Aortic Dissection[24] point with clarity to the most important risk factors for the development of acute AD. The following stand out: male gender (2:1 male-to-female ratio), age (sixth and seventh decade), a history of hypertension, previous cardiac surgery (including aortic valve replacement or repair), bicuspid aortic valve, Marfan syndrome, and crack cocaine use.

Intramural hematoma (IMH) and PAU are two other conditions that often present with similar symptoms as those of acute AD and may be linked etiologically and pathogenically. IMH results from hemorrhage within the aortic wall but without an intimomedial flap or tear. It is thought of as a precursor to dissection in many cases and has been documented to evolve into a classic AD (with a double-barrel lumen) in nearly 20% of afflicted patients.[25] Unlike AD, however, the majority (2/3) are classified as type B because they involve the descending thoracic aorta.

Similarly, most PAU lesions are located in the descending aorta. The condition tends to occur in elderly individuals with severe generalized atherosclerosis. PAU can also be a precursor to AD and be associated with IMH. All three conditions, together with rTAA, are often referred to as components of so-called acute aortic syndrome (see *Components of Acute Aortic Syndrome* sidebar).[26]

Clinically, AD is classified as acute when the symptoms are 14 days in duration or less. Beyond the first 2 weeks, AD is said to be chronic. Such designations evolved at a time (more than 30 years ago) when the vast majority of acute AD patients died within a few hours or days from onset, a fact that may perhaps explain the arbitrary and unhelpful use of the term *chronic* from the 15th day on.[27]

Anatomically, AD is classified according to the extent and location of the dissection process in the aorta: DeBakey's types I and II (or Stanford type A) denote ascending aortic involvement (plus/minus more distal extension), and type IIIa and IIIb (Stanford type B) characterize an AD that begins beyond the origin of the left subclavian artery and propagates distally for various lengths.[27] The mortality rate of untreated acute type A dissection is staggering: approximately one-third of patients die within the first 24 hours, and 50% die by the end of the second day. The 2-week mortality rate approaches 80%. It remains a true cardiac surgical emergency.

The terms *complicated* and *uncomplicated* are used to further characterize acute type B (type III) dissection. Patients presenting with rupture (blood outside the aortic
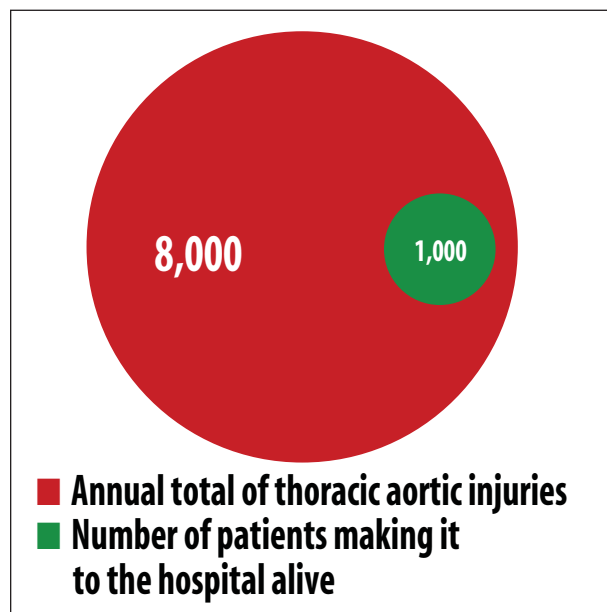

■ **Annual total of thoracic aortic injuries**
■ **Number of patients making it to the hospital alive**

Figure 10. The annual total number of thoracic aortic injuries compared to the amount of patients arriving at the hospital alive.

wall), malperfusion (visceral/renal, spinal cord, and/or the lower extremities), or acute diameter expansion in the distal arch or proximal descending aorta (> 45 mm total aortic diameter) are designated as having a complicated dissection. Unrelenting pain, uncontrolled hypertension, and "image worsening" are also considered components of this definition by some experts.

Approximately 30% of acute type B patients are thus diagnosed as complicated and are known to be in great peril in the absence of urgent intervention. The rest (70%) present with uncomplicated AD and are generally managed medically, mainly through the use of pharmacologic anti-impulse and antihypertensive therapy as well as pain control. Patients with uncomplicated acute AD who are treated with present-day optimal medical therapy face a 30-day mortality rate of 10%. However, they are exposed to possible serious complications over time, including the development of a dissecting TAA (out of the enlarging FL in the chest) in 20% to 30% of cases.[28] Thus, close monitoring and follow-up (with serial imaging) of medically managed patients is absolutely mandatory. AD complicated by overt ischemia (malperfusion) or rupture, on the other hand, requires prompt intervention, which results in a 20% or higher mortality rate within 30 days.[29]

In this realm, open surgical repair continues to disappoint because of excessive rates of morbidity and mortality.[29] This is the same as with other clinical indications in the thoracic space. Dissatisfaction with standard surgical treatment has been the most powerful driving force propelling the development of less-invasive options.

For AD, the endovascular revolution began precisely on May 20, 1999, with the publication of two landmark articles that appeared back-to-back in the same issue of the *New England Journal of Medicine*.[30,31] The pioneering early work of Nienaber et al and Dake et al strongly suggested for the first time that endovascular treatment of acute AD using stent graft devices to reline the TL in the chest and cover the entry tear might offer a safer and more attractive alternative to traditional open surgery. Interest in such strategies peaked a few years later, and a rapidly growing clinical experience has largely substantiated their initial impression.[32,33]

Today, TEVAR is considered to be a first-line therapy for the majority of complicated AD patients presenting with an interventional imperative. This paradigm shift from open surgery to TEVAR has occurred gradually but unmistakably, fueled largely by the notoriously poor outcomes of traditional surgical treatment and the distinct appeal of the less-invasive approach. However, on the downside, the evidence base is not strong, and thoracic devices have not been formally tested in the treatment of AD, and the use of stent grafts remains off label (as of the end of November 2011

when this writing was completed). It is hoped that ongoing and recently completed trials will provide the necessary clinical evidence to support regulatory approval of an on-label stent graft indication for AD in the near future.[34,35] More daunting yet, scientific evidence in favor of TEVAR intervention in the setting of uncomplicated type B AD remains elusive, with the continuing recommendation at present to manage most of these patients medically.

The development of dissection-specific devices is another important goal. Cook pursued such an endeavor before anyone else: the company's TXD stent graft was recently approved for European commercialization. The device consists of a proximal standard endograft that can be extended distally using a variable number of interlinked bare-metal self-expanding stents to stabilize the dissected lamella and re-expand the TL throughout its extent (Figure 9). In truth, a similar strategy could be used with various combinations of currently available devices. The early clinical experience and patient outcomes have been encouraging.[36] Other AD-specific endograft designs will no doubt follow in the next several years, and these are likely not to have proximal bare stents or redesigned bare stents that are short, soft, and with well-rounded smooth peaks.

Lastly, ongoing disappointment with surgical outcomes and continued technological advances will almost inevitably result in the creation of endovascular solutions for type A dissection. Current developments with transcatheter valve devices make such a prediction all but certain.

THORACIC AORTIC INJURY

The term *transection* is misleading and largely inaccurate, because most thoracic aortic injuries (TAIs) do not involve a complete loss of continuity across the aorta. Nonetheless, the term has come to signify blunt traumatic aortic thoracic disruptions that often prove fatal. Most are related to deceleration injuries occurring during automobile accidents, falls from great heights, etc. It accounts for nearly 20% of deaths related to vehicular collisions,[37] and it is the second most frequent cause of traumatic death overall.[38] Eighty percent to 90% of victims die at the site, almost immediately. Among those who make it to the hospital alive, up to 50% will die within 24 hours. There may be as many as 8,000 such injuries occurring annually in the United States and approximately 1,000 victims arriving at the hospital alive (Figure 10).[39]

Historically, surgical treatment has produced less than stellar outcomes, with an average mortality rate of 28% and a 16% paraplegia rate. Endovascular repair is growing in acceptance and adoption, as the results appear to be clearly better than those of open surgery.[39-41] In fact, in many centers, TEVAR has already replaced surgical treatment in the management of most such trauma victims,[42] and it is antici-

pated to become the next FDA-approved indication for thoracic stent graft devices in the very near future. Approval of the Talent thoracic device in June 2008 represented an important enhancement of technical capabilities with the offer of relatively small-size grafts (22- and 24-mm diameter) that are particularly well suited for implantation in small (and healthy) aortas frequently encountered in these cases. That said, endovascular repair of TAI continues to be performed using all commercially available thoracic devices at this time. The current off-label nature of endovascular repair is likely to change soon, as TAI is set to become the next FDA-approved indication for TEVAR.

A new classification that recognizes four different extents of aortic injuries (and their treatment implications) has been another helpful addition to our body of knowledge and is already having an impact at the time of making an informed decision on which lesions to select for prompt or immediate repair versus those that can safely be observed without intervention.[43] But it must also be acknowledged that information on long-term outcomes following stent graft repair of thoracic aortic injury is essentially nonexistent, so proclamations of "total triumph" must be tempered accordingly.

CONCLUSION

In the end, the TEVAR landscape can be described as one of profound transformation, paradigm shifts, and intense ongoing research and evolution. The pace of change will likely continue and almost inevitably accelerate in the foreseeable future. Although it is true that many challenges and unfulfilled promises remain, it is plain to see that we have (as a result) become inarguably better in our capacity to address a large number of complex life-threatening aortic pathologies, bringing enormous benefit to many patients. Stay tuned, for this is just the beginning! ∎

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