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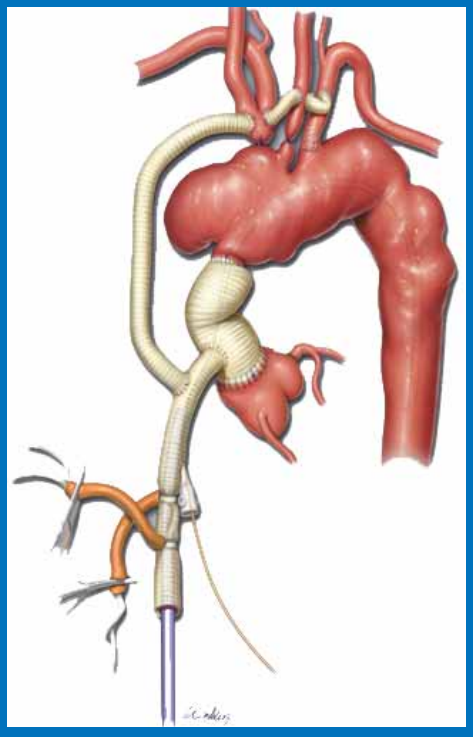

Extra-anatomic debranching of aortic arch vessels with replacement of ascending aorta and antegrade TEVAR via Dacron side branch.
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Correction Notice

Please note the following corrections for Volume 7, Number 2:

In issue 7.2, we mistakenly printed that Dr. Tsung O. Cheng was affiliated with the University School of Medicine and Health Sciences in Washington, D.C. Dr. Cheng is affiliated with the George Washington University Medical Center in Washington, D.C.

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MDCVJ is written for physicians and should be relied upon for medical education purposes only. It does not provide a complete overview of the topics covered and should not replace the independent judgment of a physician about the appropriateness or risks of a procedure or treatment for a given patient.

U.S.News & World Report ranks the Methodist DeBakey Heart & Vascular Center's cardiology, cardiothoracic and vascular surgery programs No. 19 in the nation.

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AORTIC DISEASE – THE EVOLVING THERAPEUTIC CHALLENGE

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Aortic disease remains a significant problem in the population, and there is a concerted effort to identify, define, image, and treat these conditions to ultimately improve outcomes. The rapid development of diagnostic modalities, operative strategies, and endovascular techniques within the realm of aortic disease has transformed the field and broadened the spectrum of patients that can be treated with minimally invasive techniques. There is now a better understanding of the biology of both aortic aneurysms and aortic dissections. Detecting the right set of biomarkers to predict future aortic problems still remains an issue. Several basic science findings are being translated into potential therapeutic agents that may allow early non-surgical treatment of aortic disease. Screening by ultrasound for aortic disease is now available for high-risk patients and, if appropriately applied, may save lives. Coupled with these advances in biology and health policy has been a diffusion of technology into the community, which has resulted in an associated decrease in mortality and morbidity related to aortic interventions. Hybrid procedures, branched and fenestrated endografts, and percutaneous aortic valves have emerged as potent and viable alternatives to traditional surgeries. These new techniques have been made possible through significant advances in biomaterials and bioengineering research and their commercial application by the device industry in concert with academic medical centers and regulatory agencies. Imaging of the aorta is also transforming our understanding of the pathobiology of aortic disease, resulting in a more precise stratification. Better imaging has facilitated better preoperative case planning that allows for the placement of new and more complex devices in more difficult anatomy.

As pioneers in the treatment of aortic aneurysms, The Methodist DeBakey Heart & Vascular Center (MDHVC) developed the Acute Aortic Treatment Center (AATC) to rapidly triage and treat acute aortic disease. Since its inception in 2008, the Center has significantly improved outcomes in this family of diseases. Building on this innovation and strength, MDHVC is also launching the Aortic Center to provide a comprehensive panel of services for the patient with aortic disease. This new center will include preventative screening of abdominal aortic aneurysm (AAA), comprehensive medical management of small aneurysms, non-invasive and advanced imaging of the aorta, and defined therapeutic interventions. Linked to the development of the Aortic Center, Methodist is spearheading a regional quality outcomes registry (Southern Vascular Outcomes Network, or SOVONET) to derive outcomes data for open and endovascular interventions in the south-central United States.

In concert with these advances in technology and imaging, simulation and image manipulation with case rehearsal has allowed physicians to plan, practice, and perfect cases prior to operating on the patient. Open and endovascular simulators are now becoming part of the training paradigm for fellows and residents, and the fidelity of these devices is improving daily. Since practicing physicians can also benefit from these developments, the MDHVC has developed the DeBakey Institute for Cardiovascular Education and Training (DICET) in collaboration with the Methodist Institute for Technology, Innovation and Education (MITIE), creating a national cardiovascular training center to educate, retool, and enhance the skills of practicing physicians and augment current training paradigms for residents, fellows, and auxiliary staff.

This issue of the *Methodist DeBakey Cardiovascular Journal* highlights the progress made in endovascular repair and dissection, grafting, imaging, and hybrid techniques and illustrates the current understanding and capabilities available to diagnose and treat the spectrum of aortic diseases.

DAVIES LENDS EXPERTISE TO THIS ISSUE OF THE *METHODIST DeBAKEY CARDIOVASCULAR JOURNAL*

The editors of the *Methodist DeBakey Cardiovascular Journal* wish to thank Mark G. Davies, M.D., Ph.D., M.B.A., for serving as guest editor of this issue on aortic aneurysm repair. Dr. Davies is a professor of surgery at Weill Cornell Medical College, vice chairman for finance and administration in the Department of Cardiovascular Surgery at The Methodist Hospital, a senior member of The Methodist Hospital Research Institute, and director of research and education for the Methodist DeBakey Heart & Vascular Center. Dr. Davies serves as the founding program director for both the Vascular Surgery Fellowship and the Integrated Vascular Surgery Residency at Methodist and leads the Vascular Biology and Therapeutics and CV Clinical Innovation and Outcomes programs at The Methodist Hospital Research Institute. After receiving his medical training and Ph.D. in vascular biology from Trinity College Medical School in Dublin, Ireland, Dr. Davies completed general surgery residencies at the Royal College of Surgeons in Dublin and at Duke University, followed by a vascular surgery residency at the University of Washington in Seattle. Prior to joining the Methodist faculty in 2008, he held assistant and associate professorships in vascular surgery at the University of Rochester in Rochester, New York, where he also completed his M.B.A. Dr. Davies publishes extensively and continues to receive funding from the National Institutes of Health to support both his clinical and basic science research efforts.

J. S. Fernandez-Moure, M.D.

BIOLOGY OF AORTIC ANEURYSMS AND DISSECTIONS

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Abstract

The biology of aortic aneurysm and dissection has evolved to where we now understand the genetic implications of changes in extracellular matrix proteins, smooth muscle cells, and growth factors and how they affect aortic wall homeostasis. These predeterminants are influenced by smoking, hypertension, and atherosclerosis, and the result is an inflammatory response coupled to an accelerated proteolytic cascade that disrupts both elastin and collagen in the aortic wall.

Biology of Aneurysms

Aortic aneurysms continue to be a significant medical problem with a high associated mortality. In 2000, data from the National Vital Statistics Report on Deaths showed that abdominal aortic aneurysms (AAA) and aortic dissections are the 10th-leading cause of death in men 65–74 years of age (4:1 male to female ratio) and cause more than 12,000 deaths annually.[1] Therefore, understanding the pathogenesis and biology of aneurysms and dissections becomes paramount in the management of these diseases and their associated risk factors.

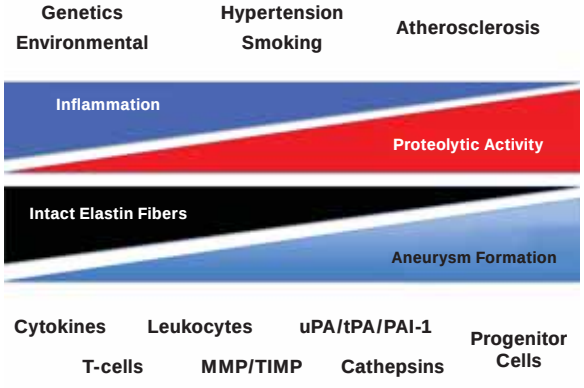

Figure 1. Aortic aneurysm pathobiology. The development of aortic aneurysms is dependent on a combination of predisposing genetic factors and environmental influences that allow for an augmented inflammatory, proteolytic, and cellular response. This milieu of agents induces loss of elasticity and dilatation of the vessel wall.

The pathogenesis of aortic aneurysms is complex and multifactorial (Figure 1). The Committee for Reporting Standards of Arterial Aneurysms defines an aneurysm as a 50% increase in the diameter of a vessel in comparison with its expected normal diameter.[2] Occurrence of extracranial aneurysms has been reported throughout the entire arterial tree, but the predominant location for aneurysms is the infrarenal aorta. In the normal aorta, there is a gradual reduction of the medial elastin fibers, decreasing from 80 layers in the thoracic aorta to 30 in the infrarenal portion. There is also a thinning of collagen within the media and thickening of the intima in the distal aorta. This anatomic difference is the rationale for suturing open aortic grafts as close to the renal arteries as possible to remove all low-collagen-layer infrarenal aorta.

Extent	Origin and Location
I	Distal to L SCA to above the renal arteries
II	Distal to L SCA to below the renal arteries
III	6th IC space to below renal arteries
IV	12th IC space to iliac bifurcation
V	Below 6th IC space to above the renal arteries

Table 1. TAAA classification. L SCA: left subclavian artery; IC: intercostal

Based on their general location (thoracic, thoracoabdominal, and abdominal), each aneurysm is then further classified based on its specific location. Thoracic aneurysms are most commonly located in the descending aorta, arising at the level of the subclavian artery.[3] Aneurysms of the ascending aorta are rare and associated most commonly with Marfan syndrome. The thoracoabdominal aneurysm (TAAA) has a more detailed classification based on origin and extent of dilation caudad. The classification of extent will dictate the tailoring of therapy. Table 1 outlines this TAAA classification.

Abdominal aortic aneurysms are classified primarily based upon how far they extend cephalad. Ninety-five percent of all AAAs are classified as infrarenal AAAs and may or may not extend to the iliac arteries.[4] The management of AAAs associated with common iliac artery aneurysms is the same as the management of the AAA alone; they should be considered a single entity. Juxtarenal aneurysms extend to the level of the renal arteries, and suprarenal aneurysms extend proximally to the level of the superior mesenteric artery and/or the celiac arterial trunk. Despite being found in anatomically distinct locations, the pathogenesis and risk factors associated with the development of aortic aneurysm — whether thoracic, thoracoabdominal, or strictly abdominal — are similar.[5-8]

Risk Factors for and Mechanisms of Aneurysm Formation

Aortic aneurysm formation is associated with advanced age, male gender, cigarette smoking, atherosclerosis, hypertension, and genetic predisposition.[5] The Aneurysm Detection and Management Veterans Affairs Cooperative Study Group (ADAM) identified smoking as the strongest modifiable risk factor for the development of aneurysm. Additionally, elderly age and male

gender were among the three principal risk factors for aneurysmal development and progression.[7, 9] Aortic aneurysm is a disease rarely found in those under the age of 50, and the incidence of death from AAA is 11-fold higher in men aged 60-64 years than in women of the same age.[10] In a recent series, the mean age of patients undergoing repair was 72 ± 7 years.[10] It should be noted that the risk factors for aortic aneurysm development are similar to those of atherosclerosis.[9, 11] Historically, aneurysms were referred to as atherosclerotic aneurysms. This misnomer has been corrected in the medical nomenclature, although atherosclerotic changes are almost universally noted in the aorta at the time of aneurysm repair.[11]

Autoimmune and Genetic Factors

Aortic aneurysm formation has been linked with various autoimmune diseases, including giant cell arteritis, systemic lupus erythematosus (SLE), Takayasu's arteritis, and antiphospholipid syndrome. Similar to these autoimmune diseases, the risk of AAA is perhaps increased by certain genotypes related to human leukocyte antigen class II molecules.[12] Other genetic associations include Marfan and Ehlers Danlos syndromes. The molecular defects in the fibrillin-1 gene, the hallmark of Marfan syndrome, are responsible for impaired structural integrity of the cardiovascular system and specifically that of the aortic root.[13] The defective gene therefore predisposes these individuals to aortic dilation and dissection. These aneurysms are considered to be degenerative. Histologically, fragmentation and degeneration of elastic fibers in the arterial media is commonly observed.

Mycotic Aneurysms

Mycotic aneurysms are rare and can be caused by viral, bacterial, fungal, or spirochetal agents. This is in contrast to the historical prevalence of aneurysms secondary to syphilis in the pre-antibiotic age. Viral and Chlamydia pneumoniae infections may contribute to aneurysm development through the inflammatory cascade. One study on characteristics of the aortic aneurysm wall found Chlamydial antigens in a majority of the AAA tissue when compared to the healthy tissue.[14, 15]

Mycotic aneurysms will typically develop at the focal site of an infected atherosclerotic plaque or an infected long-standing aneurysm. Hemodynamics also play a role in aneurysm formation due to the spatial and temporal variations in hemodynamic forces, the formation of regions of stasis, and the transition to turbulence that facilitate intraluminal thrombus formation, lipid deposition, and various inflammatory mechanisms.[16, 17]

Connective Tissue Degradation and Protease Involvement

Degradation of aortic wall connective tissue has been shown to be a hallmark of AAA formation. Collagen and elastin are the two major connective tissues found in the aorta. Histologically, elastin fragmentation and degeneration are observed in the aneurysm wall.[18] Increased turnover and loss of types I and III fibrillar collagens, as well as excessive elastolysis caused by increased collagenase, elastase, and especially matrix metalloproteinase (MMP) expression, probably underlie aortic dilation and rupture. The major forms of collagen found in the aorta are type I and III.[19] The expression of types I and III is found to be increased in aneurysmal tissue.[20] The increased expression of collagen is localized to adventitial fibroblasts, medial smooth muscle cells, and transformed myofibroblasts found within areas of local inflammation and atherosclerosis.[21] As mentioned previously, there is a gradual reduction of the medial elastin fibers, decreasing from 80 layers in the thoracic aorta to 30 in the infrarenal portion. This reduction plays a critical role in the development of the infrarenal abdominal aortic aneurysm. Elastin, the scaffold of the medial lamellae, is found in a reduced concentration as well as in a fragmented organization within tissues of diseased aorta.[22]

Cytokines regulate matrix metalloproteinase, serine protease, and cathepsin expression. MMPs (MMP-1, -2, -3, -9, -12, and -13), serine proteases (tissue-type plasminogen activator [t-PA]; u-PA; plasmin; and neutrophil elastase), as well as cysteine proteases (cathepsin D, K, L, and S) all localize in aneurysm walls at concentrations higher than those that occur in normal or stenotic atherosclerotic arteries.[23, 24] Endothelial cells, smooth muscle cells, fibroblasts, or macrophages can all produce these proteinases. CD40 ligation on inflammatory and vessel wall cells induces MMPs as well as neutrophil elastase expression and release from human vascular endothelial cells and monocyte/macrophages.

Growth and rupture of aortic aneurysm have been shown to result from increased collagen turnover as evidenced by increased type I collagen degradation products within the wall of aortic aneurysms.[25] Collagen turnover critically depends on specific collagenases that cleave the triple helical region of fibrillar collagen. The study of the pathogenesis of aortic aneurysm has focused on its collagenolytic properties and degradation of the extracellular matrix. The extracellular matrix contains embedded vascular endothelial growth factor (VEGF) and transforming growth factor-β (TGF-β), both of which are responsible for maintaining the extracellular matrix. These factors are downregulated by MMPs. Both MMP-2 and MMP-9 expose a cryptic epitope that inhibits angiogenesis and can control the inflammatory response through the modification of pro-inflammatory cytokines, chemokines, and shedding of membrane receptors.[23, 26]

The activity and expression of these and other proteases is altered in the diseased segments of aortic aneurysmal tissue.[27, 28] These degradative enzymes are secreted from macrophages and aortic smooth muscle cells. MMP activation, specifically, plays a role in the degradation of collagen and elastin. Elastases MMP-2, MMP-9, and MMP-12 are shown to have increased expression and activity in aortic aneurysmal tissue.[23, 24, 26] MMP-2, gelatinase A, is constitutively produced by native mesenchymal cells and has the capacity to degrade not only elastin but also intact fibrillar collagen.[23] High concentrations of MMP-2 are found in small aneurysmal aortas, suggesting a role for the proteinase in early aneurysmal formation. MMP-9, gelatinase B, has the ability to degrade elastin as well as partially hydrolyzed collagen. This inducible enzyme localizes to areas of inflammation in the aortic wall, where infiltrating macrophages can be found. The role for MMP-9 has been supported by the observation that MMP-9 knockout mice do not form aneurysms.[29] Confirmation of this finding was achieved by transplanting wild-type bone marrow into the knockout mice and observing aneurysm formation. Also, when compared to aortic tissue in aorto-occlusive disease, MMP-9 is elevated in the tissue of AAA patients.[30] In addition to the well-investigated MMP-2 and MMP-9, other proteases and their inhibitors have been implicated in the pathogenesis of aneurysms. MMP-12 has been localized to areas of elastin destruction and shown to be elevated in diseased tissues when compared to controls.[24] Unlike other proteases, though, its presence is not absolutely required for the genesis of aneurysm.[29] The endogenous inhibitor of cysteine proteases, Cystatin C, has been shown to be reduced in aneurysmal tissues.[31] Treatment with an MMP-inhibiting tetracycline inhibits the development of experimental AAA in vivo.[32] Prolonged administration of doxycycline is safe and well tolerated by patients with small asymptomatic AAAs and is associated with a gradual reduction in plasma MMP-9 levels.[33, 34]

Inflammation

Inflammation is known to play a large role in the development of aortic aneurysms; as aneurysm size increases, the intensity of the inflammatory cell infiltrate also increases. Chronic transmural inflammation, destructive remodeling of the elastic media, and depletion of medial smooth muscle cells are hallmarks of the degradative process.[28] Immune-mediated processes involving acute phase reactants, IFN-γ-producing T cells, and pro-inflammatory cytokines play an important role, especially in the initiation of aneurysms. They have been associated with aneurysm size and are conceivably produced by the aneurysmal tissue itself. In vitro studies reveal that IL-10, IL-6, and C-reactive protein are present at higher circulating levels in AAAs compared to controls. There is decreased expression of multiple cytokines and chemokines as well as diminished leukocyte trafficking in female aortas compared with male aortas.[35]

Studies have shown how this intense inflammatory infiltrate precedes the development of an enlarged aorta. The elastase infusion model has been used to evaluate the aneurysmal process.[27, 28] In this in vivo model, the infrarenal aorta is infused with porcine pancreatic elastase. Mice infused with elastase with cyclosporine or methylprednisolone did not develop AAAs. The transmural infiltration of inflammatory cells has been hypothesized to be the catalyst for the release of cytokines that in turn trigger many of the previously mentioned proteases. It is also thought that this infiltrate may begin or amplify the digestion process by releasing elastin degradation products that have been shown in vitro to attract mononuclear phagocytes. What has not been shown is the inciting event that leads to the elastin degradation cascade. One thought is that the process of aneurysm formation also has an autoimmune component. This is supported by the extensive lymphocytic and monocytic infiltrate in the adventitia and the immunoglobulin G deposition in the aortic wall.[28] This infiltrate leads to the accumulation of cytokines in the aortic wall; these cytokines then activate the MMPs linked to the collagen and elastin breakdown.

Reactive Oxygen Species

Another area of interest has been the role of reactive oxygen species. Superoxide levels in aneurysmal tissue are 2.5-fold higher than in adjacent non-aneurysmal tissue and 10-fold higher than in control aorta.[36] In the elastase infusion model, more than a 50-fold increase in nitric oxide synthase was observed 2 days after infusion with porcine pancreatic elastase.[37] The reactive oxygen species environment has been shown to increase MMP activity in vitro and, more importantly, increase apoptosis. The histology of aneurysmal tissue shows a decreased density of vascular smooth muscle cells (VSMC). Reactive oxygen species have been shown to induce apoptosis in in-vitro models of vascular smooth muscle cell degradation. The oxidative stress may be another catalyst or propagator of the degenerative cascade of aneurysm formation.[38]

Biomarkers for AAA

A large number of studies have associated different circulating biomarkers with AAA presence or progression. Plasma concentrations of D-dimer reflect the extent of fibrin turnover in the circulation.[38] Recent studies have shown a consistent diagnostic value to the use of D-dimer testing for AAA in addition to clinical exam and other biomarkers. D-dimer has also been shown to be elevated in the aorto-occlusive disease group.[39] Plasma concentrations of D-dimer, though, have been shown to be much greater in patients with AAA than in those with atherothrombosis alone.[39] Recent studies show the diagnostic and prognostic value of D-dimer in disparate population samples, including patients with differing risks and associated arterial diseases.[40]

System	Type	Criteria
DeBakey	Type I	Originates in the ascending aorta, propagates at least to the aortic arch and often beyond it distally
	Type II	Originates in and is confined to the ascending aorta
	Type III	Originates in the descending aorta and extends distally down the aorta or rarely retrograde into the aortic arch and ascending aorta
Stanford	Type A	All dissections involving the ascending aorta, regardless of the site of origin
	Type B	All dissections not involving the ascending aorta

Table 2. Aortic dissection classification.

Biology of Dissection

Thoracic aortic dissection (TAD) is estimated to occur at a rate of 3–4 cases per 100,000 persons per year.[41] Aortic dissections may be classified according to the DeBakey or Stanford classifications (Table 2). Two-thirds of patients with aortic dissection are male, and 62% have a documented type A dissection. Furthermore, patients with a type B dissection are generally older than their type A dissection counterparts (mean of 66 vs. 61 years, respectively).[42, 43] In contrast, patients with penetrating aortic ulcers and intramural hematomas are even older, with a mean age of 77 years (reflecting the increased frequency of atherosclerosis associated with aortic ulcers) and 69 years, respectively.

Nienaber et al. evaluated differences in aortic dissection by sex.[44] Nearly twice as many women as men older than 70 years experienced an aortic dissection, but surprisingly, fewer women were accurately diagnosed as having an aortic dissection within the first 24 hours of hospital presentation compared with men, despite women having a greater frequency of hypertension in the study. With the exception of age (women older than 70 years were more likely to present with aortic dissection), few significant differences existed in the presenting signs and symptoms between men and women.

Chronic acquired conditions, such as systemic arterial hypertension, sometimes in combination with atherosclerosis, cause thickening and fibrosis of the intimal layer and degradation and apoptosis of smooth muscle cells in the media. These processes lead to necrosis and fibrosis of the elastic components of the arterial wall, which in turn produce wall stiffness and weakness from which dissection and rupture may arise. Seventy-five percent of patients with aortic dissection have a history of hypertension.[42] Other acquired conditions that have been associated with acute aortic dissection include direct blunt trauma, tobacco use, hyperlipidemia, cocaine use (including crack cocaine), and pregnancy.[45]

Risk Factors for and Mechanisms of Aortic Dissection Formation

The biology of dissection is different from that of aortic aneurysms (Figure 2). The aortic wall consists of three layers (tunica intima,

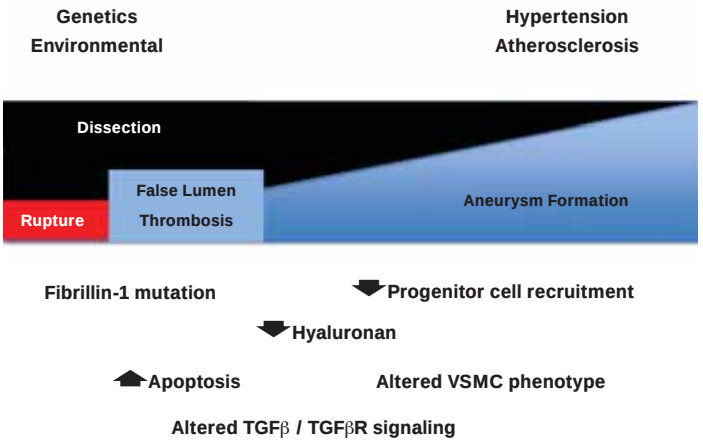

Figure 2. Aortic dissection pathobiology. The development of aortic dissections is dependent on a specific genetic abnormality in extracellular matrix proteins and cytokines, coupled to the presence of chronic hypertension and atherosclerosis. The changes in the vessel wall are associated with loss of vascular smooth muscle cell (VSMC) and a change in VSMC phenotype. Acute dissection can lead to rupture or false lumen thrombosis, and chronic dissections result in aneurysm formation, likely through some of the mechanisms noted in Figure 1.

tunica media, and adventitia). Acute aortic dissection is presumed to occur when an intimal tear develops, permitting entry of blood into a diseased underlying media characterized by elastic degeneration and smooth muscle cell loss. In the normal thoracic aorta, there are peaks in wall stress above the sinotubular junction and distal to the left subclavian artery ostium. This stress distribution may contribute to the pathogenesis of aortic dissections.[46] The dissected aorta is dominated by local, highly disturbed, and possibly turbulent flow with strong recirculation. A significant proportion (about 80%) of the aortic flow enters the false lumen, which may further increase the dilatation of the aorta. High values of wall shear stress have been found around the tear on the true lumen wall, perhaps increasing the likelihood of expanding the tear. Turbulence intensity in the tear region reaches a maximum of 70% at mid-systolic deceleration phase. Incorporating the non-Newtonian behavior of blood into the same transitional flow model has yielded a slightly lower peak wall shear stress and higher maximum turbulence intensity without causing discernible changes to the distribution patterns. In contrast to chronic stable aortic dissection, all acute or acute progressive aortic dissections showed accentuated (18)F- fluorodeoxyglucose uptake at the injured aortic wall or dissection membrane. The maximum standardized uptake values of the dissection membrane or aortic wall were significantly higher in acute aortic dissection than in chronic stable aortic dissection.[47]

The influence of tear location can be examined using three computational fluid dynamics (CFD) configurations. Pressure in the true lumen for all three configurations was similar and varied about 3.4% (largest variation between configuration 1 and 3). Pressure in the false lumen increased by 7.4% for configuration 2 compared to configuration 1 and dropped by 97% for configuration 3 compared to configuration 1.[48] Increased systolic pressure in the false lumen and true lumen was found once the re-entrance tear was occluded (increases by 13%), with the largest intraluminal pressure differences in the distal aorta (2,500 Pa). Occluding the entrance tear, which simulates placement of an EVAR (endovascular aortic repair) device, lowered the false lumen pressure essentially to zero. Removing the intravascular septum to simulate aortic fenestration lowers systolic pressure in the combined lumen by a factor of 3.

Diseases affecting the ascending aorta, such as thoracic aortic aneurysms and type I and II dissections, are primarily associated with medial necrosis on pathologic examination. Medial necrosis is characterized by fragmentation and loss of elastic fibers, loss of smooth muscle cells, and interstitial collections of collagenous tissue and basophilic ground substance. Medial necrosis occurs as part of the normal aging of the aorta but is accelerated by other conditions, including hypertension and genetic alterations that predispose persons to these aortic diseases. In dissecting aneurysms of the thoracic aorta, there is localized expansion of the aortic matrix due to deposition of collagen and other proteins, which decreases the concentration of matrix constituents, including collagen. Comparisons of areas of dissection with corresponding areas of no dissection in aortic specimens showed significant increases in the content of elastin, the content and concentration of proteins other than elastin and collagen, and a decrease in elastin concentration. There were no differences in elastin cross linking. Elastin from dissected aortas had a higher content of aspartate, threonine, serine, glutamate, and lysine and a lower content of glycine, alanine, and valine than elastin from controls. Similar distributions of hyaluronan, versican, decorin, and biglycan were seen in normal and dissected aortas; versican and hyaluronan were more prominent in the external half of the medial layer where the dissection usually occurs.[49]

Type I collagen is the major component of the aortic adventitia, whereas type III collagen comprises the majority of the collagen in the medial layer and is also the major collagen synthesized by smooth muscle cells. In accord with these findings, a number of genetic collagen defects in humans and mice are associated with rupture of the aorta and other large vessels. Patients with type IV Ehlers-Danlos syndrome, caused by mutations in type III collagen, are at risk for rupture of the aorta and other large arteries.[50] Similarly, mice that are null for type III collagen develop dissecting arterial aneurysms that rupture prenatally or in early adulthood. Experimental data has shown that the integrity of the aortic wall depends on an adequate content of type I collagen, and that continued synthesis of collagen in the aorta as a function of age is critically dependent on sequences in the first intron of the COL1a1 gene.[51]

Marfan syndrome (MFS), a relatively common autosomal dominant hereditary disorder of connective tissue with prominent manifestations in the skeletal, ocular, and cardiovascular systems, is caused by mutations in the gene for fibrillin-1 (FBN1). The leading cause of premature death in untreated individuals with MFS is acute aortic dissection, which often follows a period of progressive dilatation of the ascending aorta.[52] There is overexpression of TGF-β in MFS associated with altered hyaluronan synthesis, increased apoptosis, impaired progenitor cell recruitment, and abnormal directional migration. These factors limit tissue repair and are likely to contribute to aneurysm development.[53] Mutations in vascular smooth muscle cell (VSMC)-specific beta-myosin (MYH11) and alpha-actin (ACTA2), two major components of the VSMC contractile unit, cause familial thoracic aortic aneurysms leading to acute aortic dissections.[54] Twenty-two missense mutations in ACTA2, which encodes α-smooth muscle actin, have been identified to cause thoracic aortic aneurysms and dissections.[55]

Studies of the genes that predispose persons without known syndromes to these aortic diseases are focusing on the TAAD1 locus, a major locus for familial thoracic aortic aneurysm and dissection.[56] Familial TAAD demonstrates genetic heterogeneity, and linkage studies have identified three TAAD loci at 5q13-14 (TAAD1), 11q23 (FAA1), and 3p24-25 (TAAD2). The first genes identified to cause TAAD were FBN1, TGFBR2, and TGFBR1. The

underlying genetic heterogeneity of TAAD is reflected in the phenotypic variation associated with familial TAAD with respect to age of onset, progression, penetrance, and association with additional cardiac and vascular features. Recently, mutations in the TGFBR2 gene have been identified as the cause of disease linked to the TAAD2 locus, supporting the hypothesis that dysregulation of TGF-β signaling is a mechanism leading to aneurysms and dissections.[57]

Conclusion

Aortic aneurysm and dissection continues to be a multifactorial disease process with high mortality and morbidity. Both are associated with advanced age, male gender, cigarette smoking, atherosclerosis, hypertension, and genetic predisposition. It is known that inflammatory cell infiltrate and matrix degradation are involved in at least the progression of the aneurysm. Infectious models have been proposed but only substantiated with respect to their involvement in the inflammatory cascade, not the initiating event. Degradation by proteases and under-regulation of proteases by inhibitors have been shown to play a major role in the elastin fragmentation and degradation.

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ACUTE AORTIC TREATMENT CENTER

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Abstract

Acute aortic syndromes are a life-threatening set of conditions that require rapid triage and intervention to obtain satisfactory outcomes. The Methodist Hospital is the first institution to establish an Acute Aortic Treatment Center (AATC) based on a clinical care pathway that expedites the care of acute aortic syndromes. This pathway has resulted in a 64% reduction in time to definitive therapy and a reduction in intensive care unit (ICU) length of stay. Establishment of a multidisciplinary pathway to treat acute aortic syndromes improves efficiency and enhances outcomes.

Introduction

Acute aortic syndromes remain life threatening.[1, 2] The keys to successful treatment of acute aortic disease are early diagnosis, treatment, and disposition.[1-9] Management of acute aortic disease has changed with the increasing realization that endovascular therapies may offer distinct advantages in these situations. Aortic disease represents a unique area in cardiovascular (CV) surgery, where application of the general concept of "door-to-balloon time" would be beneficial. These paradigms have already demonstrated a significant impact on quality, outcomes, and healthcare costs for trauma, myocardial infarction, and stroke.

Concepts of Pathways

The concepts of the Acute Aortic Treatment Center clinical pathway are similar to those used as the foundation of the golden hour of trauma, door-to-balloon time for acute myocardial infarction, and door-to-lysis times in the management of acute stroke.[10-12] Each pathway has benefited patients and improved processes.[13, 14] Hospital and surgeon volume, taken as a surrogate marker for quality, has been directly correlated with lower morbidity and mortality as well as with differences in perioperative complications after aortic surgeries. The presence of a well-prepared and organized trauma service has been shown to have collateral benefit and to improve the outcome in patients treated for ruptured abdominal aortic aneurysms (AAA).[8]

Aortic Centers

The concept of aortic centers, in which a multidisciplinary team is assembled for comprehensive aortic care, has been adopted by many groups with the goal of improving outcomes and increasing clinical volumes.[15-17] These centers are developed to improve patient access and management and to increase patient volume by providing a differentiation factor in the local market.[16, 18] The diffusion of aortic endograft technology into the community and the evolution of cardiovascular skill sets in practitioners have stifled further development of this concept. There is considerable debate on the regionalization of vascular care, with advanced procedures being funneled into high-volume tertiary and quaternary centers of excellence with a comprehensive, experienced faculty and a multidisciplinary approach.[19] The Leapfrog Group healthcare quality initiative has proposed "evidence-based" hospital referral criteria for specific procedures, including elective abdominal aortic aneurysm repair. These criteria include an annual hospital AAA operative volume exceeding 50

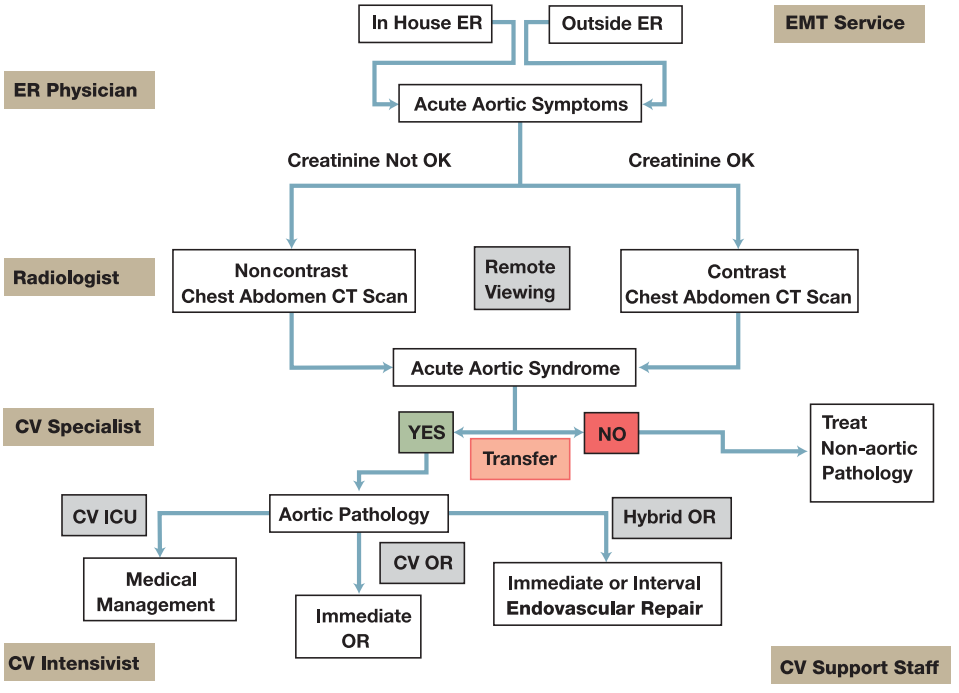

Figure 1. AATC Pathway

cases and provision of ICU care by board-certified intensivists.[19-21] It has been reported that patients transferred with a ruptured AAA had an over twofold increase in ICU days used but no difference in total length of stay.[22] However, acceptance of transfer patients with ruptured aortic aneurysms from community centers did not adversely affect patient survival.[22, 23]

The Acute Aortic Treatment Center (AATC) builds on these foundations but is centered on a single, difficult-to-manage disease process. Implementation of the AATC has resulted in a growth in emergency and after-hours care, emergent and urgent procedures, and acceleration in semi-elective interventions within the inpatient environment.

The AATC Pathway (Figure 1)

The AATC leverages the hospital's existing rapid-response services infrastructure that was created to shorten door-to-balloon times for acute myocardial infarction, rapid intervention at a stroke center, or effective management within the "golden" hour at a trauma center. The AATC requires that five essential physician positions be in place to effectively operate: 1) an emergency room (ER) physician to function as a point person in the ER and act as an interface with emergency medical technician (EMT) service personnel; 2) the medical director of the CVICU to coordinate care for emergent stabilization; 3) a computed tomography (CT) radiologist to provide emergent interpretation of CT imaging; 4) an on-call triaging AATC CV surgeon; and 5) cardiac and vascular surgeons who work in close collaboration as part of a CV therapy team or CV department. In addition to the physician coordination, the center requires development and implementation of a clinical pathway from diagnosis through emergent transportation to the AATC and a clinical pathway from the emergency room through the scanner to the ICU or operating room. Specific protocols are in place for the management of each of the following clinical presentations: Type A dissection (e.g., immediate operating room intervention), Type B dissection (e.g., medical management), malperfusion syndromes, intramural hematoma, ruptured thoracic aorta, ruptured abdominal aortic aneurysms, symptomatic thoracic aneurysms, symptomatic abdominal aneurysms, penetrating ulcers, thoracic aortic injury, and shaggy aortic syndrome. There are two mechanisms for patient entry into the AATC clinical care pathway.

Pathway A

The patient is transported to the AATC, and CT imaging from an outside institution is immediately uploaded to PACS, if available. There is an in-house radiologist on call who reads the CT and immediately contacts the responsible emergency room physician with the findings. The treatment continues according to the Common Pathway.

Pathway B

The emergency room physician examines the patient and, if acute aortic syndrome is suspected, labs are collected and analyzed with i-Stat (turnaround time: 10 minutes) to determine the creatinine concentration. If the patient's creatinine is ≤1.6 mg/dL, a CT of the chest, abdomen, and pelvis with contrast is promptly obtained; however, if the patient's creatinine is >1.6 mg/dL or he has an iodine or contrast allergy, a CT of the chest without contrast is obtained. This scan is then reviewed by the in-house radiologist on call and discussed with the emergency room physician.

Common Pathway

If acute aortic syndrome is suspected or confirmed, a group page is sent to the following personnel: CV anesthesiologist on call, CVICU attendings and staff, operating room (OR) supervisor, nursing supervisor, and security. While this is being done, the CV surgeon reviews CT scans on site or via Web-based viewing and determines patient disposition. If the patient had a non-contrast CT, the AATC surgeon will make a decision about the risk/benefit ratio of proceeding with contrast because it may be necessary to add a contrast scan for diagnosis/decision making. If intervention is necessary, the AATC surgeon decides whether an open surgical intervention (such as for type A dissection), an endovascular repair (which is appropriate for ruptures of thoracic and abdominal aneurysms and dissection with malperfusion syndromes), hybrid procedures, or medical management (such as for type B dissection) will be needed; a cardiac anesthesiologist is paged, and if an endovascular route is taken, the radiology technician is paged. Once the therapeutic decision is made, the AATC surgeon calls the CVICU intensivist with the treatment plan and the patient is transported expeditiously to the appropriate destination (OR or CVICU). Within the protocol, there are standard treatment algorithms for each condition. Type A dissections and acute AAAs are moved to the OR for surgery and Type B dissections moved to the CVICU for aggressive medical therapy. The CV OR staff are in-house 24 hours daily. If there is a lag, the CVICU attending will become the CV anesthesiologist to expedite care and begin the case. If an AAA is considered appropriate for endovascular aneurysm repair, the procedure will be performed in a dedicated OR suite. All postoperative care occurs in one unit of the CVICU and follows approved CV protocols.

Outcomes of the AATC at Methodist

We have recently documented the results of Methodist's AATC.[24] Following the center's initiation, 49% of patients admitted with aortic disease to the CV services line were considered to have acute aortic disease. This represented a 30% increase in the total number of admissions and a 25% increase in acute pathology. Given the mechanisms in the pathway, a greater number of patients are being referred from emergency rooms and outside hospitals. While there was no difference in the number of ruptured aneurysms encountered, there was a twofold increase in thoracic aortic dissections. Initiation of the treatment pathway resulted in a highly significant reduction in time to definitive therapy (64% decrease; $p = 0.0001$). There was an increase in those undergoing medical therapy alone in the AATC cohort that was consistent with the increase in aortic dissections. Despite the increase in acuity, mortality (4% in the pre-AATC cohort vs. 6% in the AATC cohort), and morbidity (41% vs. 45%), rates were unchanged. Furthermore, there was a significant decrease in ICU length of stay (5 vs. 4 days, pre-AATC cohort vs. AATC cohort) but total hospital length of stay was unchanged (11 vs. 10 days), suggesting that rapid intervention preserved resources, allowing the patient to be cared for more efficiently and in a cost-effective manner

Conclusion

Establishment of a multidisciplinary AATC pathway was associated with a marked reduction in time to definitive treatment, improved throughput with reduced ICU time, and maintained clinical efficacy. The concept is a landmark in advancing cardiovascular care.

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THE CURRENT STATUS OF ENDOVASCULAR REPAIR OF ABDOMINAL AORTIC ANEURYSMS (EVAR)

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Abstract

Abdominal aortic aneurysms (AAA) affect close to a quarter of a million people in the United States every year. Intervention is designed to treat the AAA when the patient becomes symptomatic and to prevent the fatality associated with rupture. Physicians and patients should weigh the risks associated with intervention compared to the risk of rupture for the particular size of the aneurysm and the patient's comorbidities. Thus, the decision to intervene, especially in asymptomatic aneurysms, is mostly based on clinical judgment. Endovascular AAA repair (EVAR) is attractive in that it offers a minimally invasive approach that obviates a major abdominal procedure and cross-clamping of the aorta. We report on the current affairs of the major clinical trials evaluating the outcomes of patients undergoing EVAR and describe the current devices available in the United States for endovascular repair.

Randomized Trials and EVAR

The DREAM trial randomized 351 patients to address the 2-year outcome after conventional or endovascular repair of >5 cm AAA and found that the cumulative rates of aneurysm-related perioperative death were 5.7% for open repair and 2.1% for EVAR. This survival advantage was not sustained after the first postoperative year.[1]

Similarly, the OVER trial randomized 444 patients to EVAR and 437 patients to open surgical repair; all were considered to be candidates for both interventions. The 30-day mortality was 0.5% for EVAR compared to 3% for open repair. At 2 years follow-up, mortality was not significantly different for EVAR (7%) compared to open surgical repair (9.8%). Endovascular repair was associated with reduced median procedure time, blood transfusion requirements, duration of mechanical ventilation, and intensive care unit and hospital stays. There were no differences in major morbidity, secondary therapeutic procedures, or health-related quality of life (HRQL) scores. The authors concluded that longer-term data was needed to evaluate the outcomes of the two procedures. Thus, an interim report will follow at the 9-year trial mark.[2]

The EVAR 1 trial compared EVAR to open AAA repair in patients 60 years or older who had an AAA ≥5.5 cm and who were also fit for conventional open surgical repair. Between 1999 and 2003, 543 patients were randomized to EVAR and 539 to open repair. The 30-day mortality significantly differed for EVAR and open repair (1.7% compared to 4.7%, respectively). However, secondary interventions undertaken within 30 days of the procedure were significantly higher in the EVAR group (9.8%) compared to the open group (5.8%).[3] The long-term results of this trial were further investigated past the initial 30-day mortality benefit of endovascular repair. Following 1 year, the HRQL was similar for patients in both groups. After 4 years of follow-up there was a similar 28% mortality for both groups that was associated with a persistent and yet lower aneurysm-related mortality for the EVAR group. At 4 years, EVAR did not offer an advantage in long-term all-cause mortality or HRQL, mandated ongoing surveillance, and led to a greater number of complications and reinterventions, making it more expensive.[4] After 8 years, the aneurysm-related survival was 93% in both groups while graft-related complications and reinterventions remained higher with endovascular repair.[5]

Unlike the previous trial, the EVAR 2 trial evaluated whether EVAR improved survival in similar patients who were not fit for open surgical repair of their AAA. Patients enrolled had significantly worse health than those studied in the EVAR 1 trial. Patients were randomized, and 166 were treated with EVAR while 172 had no intervention. Death from aneurysm rupture at 30 days in the no-intervention group was 9% and was similar to the 30-day mortality in the treatment group. All-cause mortality at 4 years was 64% for both groups without a difference in aneurysm-related mortality. Similar to the EVAR 1 trial, hospital costs for patients treated were higher without a difference in HRQL scores. Obscuring the data, more than a quarter of patients assigned to the no-intervention group underwent repair, one-third of them doing so because of patient preference. However, these crossovers did not alter the main conclusion of the trial that EVAR had a considerable 30-day operative mortality in patients already unfit for open repair of their aneurysm, did not improve survival over no intervention, and was associated with a need for continued surveillance and reinterventions at substantially increased cost.[6] At the 8-year follow-up, the aneurysm-related mortality in the EVAR group was lower. However, this did not result in a difference in overall mortality. A total of 48% of patients had complications related to the EVAR, and 27% of those patients required reintervention, adding to the increased cost of the procedure compared to the no-intervention group.[7]

In essence, these trials demonstrated that for AAAs >5 cm, EVAR reduces 30-day aneurysm-related mortality and is associated with a lower length of hospital stay and related surgical benefits from a less morbid procedure. The data on longer-term benefits of EVAR compared to open repair is less clear, with no obvious survival advantage and an expense of continued surveillance, increased reintervention rates, and additional costs.

Small Aneurysm Trials and EVAR

To determine if EVAR is safe in the treatment of small AAAs, the CAESAR trial randomized patients >50 years of age with an AAA between 4.1–5.4 cm to receive immediate EVAR or surveillance by ultrasound and computed tomography. Repair in the surveillance group was performed if the AAA diameter reached ≥5.5 cm, the diameter increased >1 cm/year, or the patient became symptomatic. Between 2004 and 2008, 182 patients were randomized to EVAR and 178 to surveillance. The authors found that mortality and rupture rates in AAAs <5.5 cm were low and could not demonstrate a clear advantage between the early or delayed strategy. However, approximately 60% of the small aneurysms under surveillance grew to require repair within a 36-month period. Interestingly, the authors found that approximately 17% may lose feasibility for EVAR during this period. The authors conclude that EVAR is safe for small AAAs if close supervision is performed.[8]

In a similar manner, the PIVOTAL trial was organized to determine whether early EVAR reduced the risk of rupture or early aneurysm-related death compared with surveillance in patients between 40–90 years of age and with AAA size between 4–5 cm. This trial had 366 patients randomized to EVAR and 362 to surveillance for a mean follow-up period of 20 ± 12 months. During this study period, 30% of the patients in the surveillance group underwent EVAR — 70.6% for growth of the aneurysm, 11% due to request for repair, and 7.4% for the development of symptoms. The rate of perioperative mortality was 0.6%, and the 3-year rate of rupture was zero, surprisingly. The lower-than-expected rate of rupture prompted early termination but with the commitment to follow currently enrolled patients. Until longer-term data becomes available from this study, close surveillance and treatment with EVAR as clinically indicated appears as safe as EVAR for the small aneurysms in this trial.[9] These studies suggest that in patients with small aneurysms, careful and close monitoring and surveillance is safe and will detect those who become symptomatic and those who demonstrate an increase in aneurysm size and require repair.

EVAR and Advanced Age

Fonseca and colleagues[10] compared the periprocedural and late EVAR outcomes in 117 octogenarians to 205 patients younger than 80 years of age who underwent treatment over a 5-year period. The octogenarians were significantly more likely to have diabetes, coronary artery disease, chronic obstructive pulmonary disease, and renal insufficiency. There were no significant differences in the rates of perioperative myocardial infarction, stroke, death, or arterial ischemic complications between this high-risk medical group and the younger cohort. However, octogenarians had a higher rate of access site hematomas, pulmonary problems, and other perioperative complications. Interestingly, the younger patients were twice as likely to develop a type II endoleak.

In a report by Goldstein and associates,[11] 24 patients >90 years of age who had an AAA were treated with EVAR. Perioperative mortality was 4.2% based on one patient, and there were no aneurysm-related deaths beyond the 30-day period. The authors conclude that with or without symptoms, patients over 90 years of age should be considered for EVAR. Despite their advanced age, patients benefit with a low morbidity and mortality and mean survival exceeding 2.4 years, especially in those with few comorbidities. These studies suggest that elderly patients >80 years of age with AAA, reasonable life expectancy, and adequate anatomy should be considered for EVAR. Though postoperative complications are higher than in younger cohorts, the morbidity of open AAA repair remains higher.

Endograft Devices in the United States

Since their initial design, endograft devices have undergone device redesigns and improvements to address sealing, fixation, migration, kink, and iliac access and to expand their aortic treatment indications. The following constitutes a concise description of the current state of commercially available AAA endografts in the United States (Table 1).

The AneuRx™ (Medtronic, Inc., Minneapolis, MN) modular bifurcated stent graft has a self-expanding nitinol skeleton that supports a woven polyester fabric (Figure 1). The diamond-shaped nitinol rings that comprise the exoskeleton provide the high radial strength required to achieve infrarenal fixation without active anchoring barbs. The delivery device has a hydrophilic coating designed to ease the delivery system's passage through tight and tortuous arteries by reducing friction with the arterial wall. The main body of the device is available in diameters of 20–28 mm and iliac limbs of 12–24 mm. The device is MRI conditional up to 3 Tesla.

The Excluder™ (W.L. Gore & Associates, Inc., Flagstaff, AZ) modular bifurcated stent graft is composed of a self-expanding nitinol wire construct with helical configuration that supports an expanded polytetrafluoroethylene (ePTFE) and fluorinated ethylene propylene (FEP) graft (Figure 2). The endograft carries nitinol anchors at the proximal edge that participate in active infrarenal

Table 1. Characteristics of commercially available AAA endografts in the United States.

Device	Maker	FDA Approval	Bifurcated Design	Stent Material	Fabric	Active Fixation	Suprarenal Fixation	MRI Conditional
AneuRX™	Medtronic, Inc.	1999	Modular	Nitinol	Polyester	No	No	Yes
Excluder™	W.L. Gore & Associates, Inc.	2002	Modular	Nitinol	ePTFE	Yes	No	Yes
Zenith™	Cook Medical, Inc.	2003	Modular	Stainless Steel	Polyester	Yes	Yes	Yes
Powerlink™	Endologix	2004	Unibody	Cobalt-Chromium	ePTFE	No	No	Yes
Talent™	Medtronic, Inc.	2008	Modular	Nitinol	Polyester	No	Yes	Yes
Endurant™	Medtronic, Inc.	2010	Modular	Nitinol	Polyester	Yes	Yes	Yes

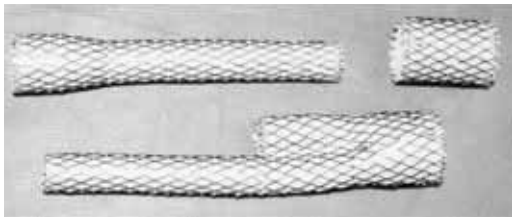

Figure 1. The AneuRx™ (Medtronic, Inc., Minneapolis, MN) is constructed with woven polyester and self-expanding nitinol (nickel-titanium) alloy stent rings. Each ring has a series of diamond-shaped segments and broad proximal and distal sealing areas to accommodate longer aortic bodies, longer and larger straight iliac limbs, and flared iliac limbs and that comprise the exoskeleton. The AneurRx™ device also has the Xcelerant® Hydro Delivery System designed to ease the delivery system's passage through tight and tortuous arteries.

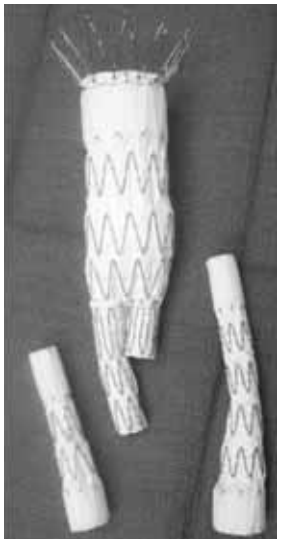

Figure 3. The Zenith Flex™ (Cook Medical, Inc., Bloomington, IN) is a modular bifurcated stent graft constructed with woven polyester Dacron that has a kink-resistant sheath and is composed of stainless steel stents. It uses a trigger-wire release mechanism during deployment and anchoring barbs to stabilize the graft to the aortic wall. The trigger-wire system also allows repositioning even when the device is partially deployed.

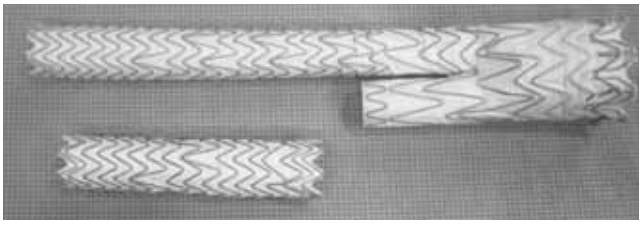

Figure 2. The Excluder™ (W.L. Gore & Associates, Inc., Flagstaff, AZ) is constructed from durable ePTFE bifurcated graft with an outer self-expanding nitinol support structure to combine both device flexibility and material durability. The device is inserted by a catheter-based delivery technique. The endograft carries nitinol anchors at the proximal edge that participate in active infrarenal fixation to resist migration and an ePTFE FEP sealing cuff at the same end.

Figure 4. The Powerlink™ (Endologix, Irvine, CA) is created with self-expanding cobalt-chromium stents that form the inner structure. Aortic seal is achieved by radial force attained by the expanded endografts, while fixation is achieved by the graft sitting on the aortic bifurcation.

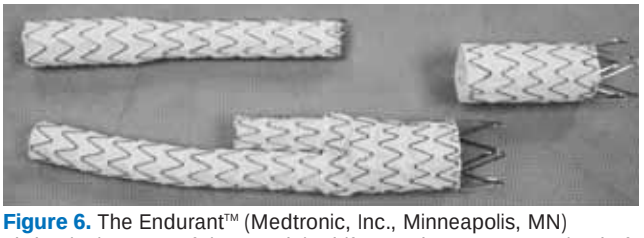

Figure 6. The Endurant™ (Medtronic, Inc., Minneapolis, MN) abdominal stent graft is a modular bifurcated system comprised of nitinol M-shaped stents attached to a high-filament polyester fabric most recently approved for use in the United States. Like other grafts, Endurant™ possesses suprarenal fixation along with stent anchoring pins.

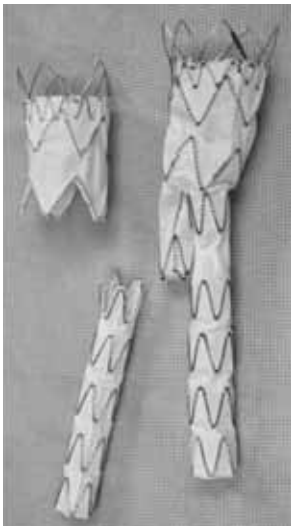

Figure 5. The Talent™ (Medtronic, Inc., Minneapolis, MN) consists of a fabric tube supported by a metal frame, and each graft is compressed into a long, thin, tube-like device to aid in deployment. The proximal end has bare springs allowing for suprarenal fixation that depends on the device's radial force component. The delivery device has a hydrophilic coating designed to ease the delivery system's passage through tight and tortuous arteries by reducing friction with the arterial wall; once deployed, it self-expands to the diameter of the aorta.

fixation to resist migration and an ePTFE FEP sealing cuff at the same end. The main body of the device is available in diameters of 23–31 mm and iliac limbs of 10–20 mm. The device is MRI conditional up to between 1.5–3 Tesla.

The Zenith Flex™ (Cook Medical, Inc., Bloomington, IN) is a modular bifurcated stent graft constructed with a frame of self-expanding stainless steel Z-stents that are sewn to woven polyester Dacron grafts (Figure 3). The endograft has a suprarenal bare stent containing anchoring barbs for active fixation to resist migration. The main body is available in diameters between 22–36 mm and iliac limbs of between 8–24 mm. The device is MRI conditional up to 3 Tesla.

The Powerlink™ (Endologix, Irvine, CA) bifurcated unibody stent graft is created with self-expanding cobalt-chromium stents that form the inner structure of the device that supports an ePTFE graft (Figure 4). Aortic seal is achieved through radial force from the expanded endograft while fixation is achieved by the graft sitting on the aortic bifurcation. The main body of the device is manufactured in sizes ranging from 25–28 mm in diameter. The iliac limbs are 16 mm in diameter with extension limbs available between 16–25 mm in diameter. The device is MRI conditional up to between 1.5–3 Tesla.

The Talent™ (Medtronic, Inc., Minneapolis, MN) abdominal stent graft is a modular bifurcated system comprised of nitinol springs attached to a polyester fabric material (Figure 5). The proximal end has bare springs, allowing for suprarenal fixation that depends on the device's radial force component. The delivery device has a hydrophilic coating designed to ease the delivery system's passage through tight and tortuous arteries by reducing friction with the arterial wall. The main body of the device is available in diameters of 22–36 mm and iliac limbs of 8–24 mm. The device is MRI conditional up to 3 Tesla.

The Endurant™ (Medtronic, Inc., Minneapolis, MN) abdominal stent graft is a modular bifurcated system comprised of nitinol M-shaped stents attached to a high filament polyester fabric most recently approved for use in the United States (Figure 6). Similar to the Zenith Flex endograft, Endurant™ possesses suprarenal fixation along with stent anchoring pins. The main body is available in diameters between 23–36 mm and iliac limbs of 10–28 mm. The device is MRI conditional up to 3 Tesla.

Conclusion

EVAR has rapidly emerged as the preferred treatment for AAAs at many medical centers in the United States, a remarkable shift considering that the first aortic endografts were approved in 1999. The low morbidity, shortened recovery time, and lower perioperative mortality have propelled this treatment to the forefront. Review of the available data suggests that there is an early mortality benefit that fades over time and a higher need for secondary procedures. Nonetheless, when presented with the options of endovascular repair versus the invasiveness of open repair, patients largely will choose the less-invasive option. The endografts continue to evolve to overcome many of their initial difficulties. Current devices vary considerably in their properties and offer lower profile, hydrophilic delivery systems, greater flexibility, improved methods of fixation, and a greater range of sizes. The problems of Type 2 endoleak and need for secondary intervention continue to challenge endograft outcomes, as does the need for lifetime surveillance. As the endografts develop further with next-generation devices, advances in these shortcomings are expected and will almost certainly lead to further widespread adoption of the treatment modality.

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THE CURRENT STATUS OF ENDOVASCULAR REPAIR OF THORACIC AORTIC ANEURYSMS (TEVAR)

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Abstract

Thoracic endovascular aortic repair (TEVAR) has been one of the most important advances in the management of thoracic aortic disease in the past two decades. The procedure was originally developed by Dake and colleagues in 1994 for the treatment of descending thoracic aortic aneurysm (DTAA), using the same principles as EVAR for the treatment of abdominal aortic aneurysm (AAA).[1] The first device approved for this indication was the TAG device (W.L. Gore & Associates, Inc., Flagstaff, AZ) in 2005. Since then there has been a plethora of changes and new developments related to thoracic endovascular aortic repair. This article will summarize the major updates related to TEVAR, focusing on three main aspects: what is new in device technology and future prospects; the expanding indications of TEVAR for the treatment of other pathologies and the newly developed techniques involved; and a glimpse at the expected future direction in the field.

Current Status of TEVAR Devices

Currently, there are three FDA-approved devices in the U.S. market for TEVAR. The first one is the Gore TAG device (W.L. Gore & Associates, Inc., Flagstaff, AZ), which was the first approved device in the country (Figure 1). It is a self-expanding endoprosthesis made of an expanded polytetrafluoroethylene (ePTFE) tube reinforced with ePTFE/fluorinated ethylene (FEP) film and an external nitinol self-expanding stent along the entire graft. A circumferential PTFE sealing cuff is located on the external surface of the endograft at the base of each flared scalloped end. The endoprosthesis size ranges from 26–45 mm in diameter and 10, 15, and 20 cm in length. The device needs a sheath for its introduction that ranges between 20 French (Fr) and 24 Fr depending on the device diameter. Since its original design, the device has had several modifications including removal of the longitudinal wire that had the tendency to fracture, addition of a low permeability film, and modifications of the sealing zone comorbidities and delivery system. Gore also developed a new sheath in 2010 with a pressurized balloon hemostatic valve to reduce blood loss (Figure 2). Currently, Gore is working on a conformable device (C-TAG) that provides better conformability to the arch area, and it received FDA approval to investigate this device in treating thoracic aortic aneurysms and other thoracic aortic pathology including traumatic aortic transection and acute aortic dissection.

The second approved device for TEVAR is the Zenith TX2 endovascular graft (Cook Medical, Inc., Bloomington, IN), a two-piece modular device that has full-thickness woven polyester fabric sewn to self-expanding stainless steel Cook-Z stents (Figure 3). For added fixation, the proximal component has caudally oriented barbs, and the distal component has distal bare stents with cranially oriented barbs. The device comes in diameters between 28–42 mm and lengths between 120–216 mm; it is loaded in its own sheath that is 20 Fr for sizes up to 36 mm and 22 Fr for larger sizes. Some new modifications on the device since its introduction include the creation of a new hemostatic valve that markedly reduces blood loss during the procedure and a modification to the sheath that reduces the risk of kinking

Figure 1. Conformable GORE TAG® Device (C-TAG®).
Image courtesy of W.L. Gore & Associates, Inc.

Figure 2. GORE® DrySeal Sheath. It is comprised of an introducer sheath with a GORE® DrySeal inflatable Valve attached, a dilator, and a 2.5 ml valve inflation syringe. When the valve is inflated, the sheath is completely hemostatic and multiple devices can be introduced through the sheath without compromising the seal.
Images courtesy of W.L. Gore & Associates, Inc.

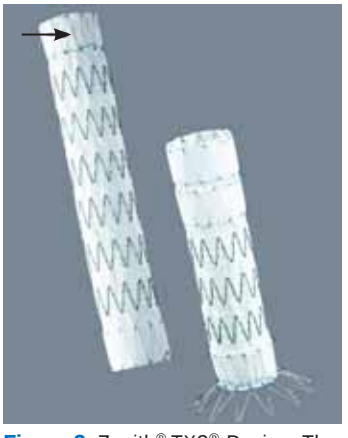

Figure 3. Zenith® TX2® Device. The black arrow points at the proximal caudally oriented barbs on the proximal main body component.
Image courtesy of Cook Medical, Inc.

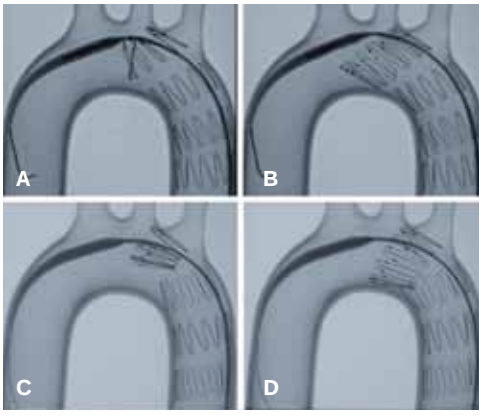

Figure 4. (A) and (B) show the original TX2® Device with "Bird's Beak Effect" in the aortic arch. (C) and (D) show the new TX2® Pro-Form™ Device addressing this problem.
Images courtesy of Cook Medical, Inc.

Figure 5. Talent™ thoracic stent graft device.
Image courtesy of Medtronic, Inc.

Figure 6. Talent™ thoracic stent graft with the Captivia® delivery system. The proximal bare metal stent is constrained in the delivery system to be released independent of device deployment. Image courtesy of Medtronic, Inc.

Graft Type	Graft Diameter (mm)	Access Diameter (mm)
Talent™	22–32	7.3
Gore TAG®	26, 28	7.6
Cook TX2®	28, 30, 32, 34	7.7
Talent™	34, 36, 38, 40	8.0
Talent™	42, 44, 46	8.3
Gore TAG®	31, 34	8.3
Cook TX2®	36, 38, 40, 42	8.7
Gore TAG®	37, 40, 45	9.2

Table 1. Comparison of device graft and access diameters. Device diameters are based on labeled diameters from device instructions for use.

during passage along tight curves, a risk that was present in the older sheath design. Also, the trigger wire conformation now allows the device to be constrained at the proximal stent, which results in better conformity to the aortic wall in the curvature of the arch (Figure 4).

The third device is the Talent Thoracic Stent Graft (Medtronic, Inc., Santa Rosa, CA) composed of a series of shaped, sinusoidal, self-expanding nitinol wire rings that act as springs (Figure 5). A full-length connecting bar connects the proximal and distal springs. The nitinol structure is covered by a monofilament polyester woven graft that is sewn securely to the nitinol structure. The device comes in four different components: proximal main, proximal extension, distal main, and distal extension. The proximal stent grafts and the distal extensions are equipped with a bare spring to improve fixation and allow deployment across the origins of the great vessels proximally and the celiac axis distally. The device comes in a wide range of diameters between 22–46 mm with 2 mm increments and lengths between 112–116 mm. Tapered grafts are available to accommodate the differences in diameter along the length of the aorta. The device comes preloaded in its Medtronic Xcelerant delivery system, which has a 22–25 Fr outer diameter (as opposed to the inner diameter in the TAG and TX2 devices) depending on the diameter of the device. The recently released Captivia delivery system offers complete control of device deployment, with minimal friction during the deployment process, and allows proximal device conformation to acutely angled thoracic aortic arch anatomy (Figure 6). There is currently a fourth device (Relay, Bolton Medical, Inc., Sunrise, FL) that is undergoing a phase II trial in the United States.

The sizing criteria for device choices differ, with the Gore TAG® trial using the inner aortic diameter and the Talent™ and TX2® trials using outer aortic diameter. Also, the access diameters of the devices are reported in different ways: the TX2® and TAG® devices report the sheath diameter needed (which means inner diameter of the delivery sheath required), while the Talent™ device reports the outer diameter size as it is already loaded into a delivery sheath. Which device has the smallest delivery system? It really depends on the graft diameter in its constrained condition within its delivery system (Table 1).[2] Table 2 summarizes a comparison of the three FDA-approved devices, and Table 3 summarizes the 30-day and 1-year outcomes of their regulatory trials.

All the device manufacturers are pursuing fenestrated and branched devices for both abdominal and thoracic cases to expand their use and versatility in dealing with aortic pathologies involving the great arch and visceral vessels. These are complex areas where the inherent curvature of the arch, the closely packed supra-aortic trunk vessels, and the potential for serious adverse consequences have delayed development.[2] (See "Fenestrated and Branched Endographs" in this issue, page 35.)

Table 2. Comparison of the three FDA-approved TAA devices. ePTFE: expanded polytetrafluoroethylene; OD: outer diameter.

Company	Product Name	Stent Material	Graft Material	Stent Expansion	Length (cm)	Diameter (mm)	Sheath required for delivery	Delivery sheath (Fr)	Proximal seal required (mm)
Cook Medical, Inc.	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	20, 22	30
W.L. Gore & Associates, Inc.	Gore TAG® Thoracic Endoprosthesis	Nitinol	ePTFE	Self-expanding	10, 15, 20	26, 28, 31, 34, 37, 40, 45	Yes	20, 22, 24	20
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)	20

	Gore TAG	VALOR	STARZ
30-day results			
Mortality	1.5%	2.1%	1.9%
Paraplegia/Paraparesis	2.8%	8.7%	5.6%
Stroke	3.5%	3.6%	2.5%
Major adverse event	28%	30%	41.9%
Endoleak	3.6%	25.9%	4.8%
Access complications	14%	9.2%	22%
1-year follow up			
Endoleak	3.9%	12.2%	3.9%
Sac size increase >5 mm	9%	8.5%	7.1%
Migration >10 mm	0.7%	3.9%	2.8%
Ruptures	0%	0.5%	0%
Conversion	0.7%	0.5%	0%

Table 3. Results of the three FDA-approved thoracic devices according to their regulatory trials. STARZ: The study of Thoracic Aortic Aneurysm Repair with the Zenith® TX2® TAA Endovascular Graft; VALOR: Vascular Talent™ Thoracic Stent Graft System for the Treatment of Thoracic Aortic Aneurysms

New Techniques and Treatment of Other Aortic Pathology

The introduction of TEVAR almost two decades ago was essentially targeted at the treatment of thoracic aortic aneurysm. Actually, all the current devices have been approved primarily for use in descending thoracic aortic aneurysm repair (DTAA). With the advancement of endovascular technology, those techniques have been applied to other challenging pathologies affecting the thoracic aorta. Acute aortic ulcer and intramural hematoma are now being primarily treated using thoracic endografting techniques. In fact, this is an approved indication for both the TX2 and the Talent devices. Other more complicated pathologies include acute and chronic aortic dissection, traumatic aortic transection, mycotic aneurysms, and aortoesophageal and aortobronchial fistula.

Surgical treatment of aortic dissection is indicated in all type A dissection and acute type B dissection with malperfusion, rupture, or impending rupture and in chronic dissection with progressive aneurysmal dilation. Historically, open aortic repair for aortic dissection has been associated with high morbidity and mortality. The International Registry of Acute Aortic Dissection (IRAD) investigators reported an in-hospital mortality of 34% for those undergoing open surgical intervention for complicated type B dissections. These patients also had a 40% in-hospital complication rate.[3] The use of endovascular techniques for the management of complicated type B dissection has been more promising: the IRAD investigators reported an 11% in-hospital mortality rate and 21% complication rate in patients who received endovascular therapy for complicated type B dissection.[3]

Aortic dissection is a complex pathology and there is no standard endovascular technique that is available for its management. Multiple different techniques have been developed to address aortic dissection including endovascular fenestration, with or without the use of bare metal aortic and branch vessel stenting, and the use of stent grafting to seal the proximal injury point of the aorta. The type of therapy selected depends on the pathologic criteria of the case, the presence of significant aortic occlusion, and the type of branch vessel occlusion whether static or dynamic. Whatever the technique used for endovascular therapy, intravascular ultrasound (IVUS) and high-quality flat plate fluoroscopy are mandatory for the safe and effective performance of the procedure. Compared to the earlier debate regarding the use of endografts for treating aortic dissection, the Society for Vascular Surgery guidelines currently recommend preoperative vascularization of patients whose anatomy requires covering the left subclavian artery with an endograft unless the procedure is done emergently, in which case selective revascularization of the left subclavian artery is suggested.[4] Covering the left subclavian artery without revascularization has been reported to have a four-fold increase in spinal cord ischemia that results in paraplegia, stroke, and upper extremity ischemia.[5]

Despite the lack of level I evidence, open repair is falling out of favor as the gold standard for the treatment of descending thoracic aortic injury (DTAI).[6] Recently, there has been more enthusiasm using TEVAR for the treatment of DTAI. For example, in a recent review by Jonker of all patients in the state of New York treated for DTAI from 2000 to 2007, the number of TEVAR cases exceeded the number of open procedures.[7] In the United States, however, there is no FDA-approved device for the treatment of DTAI. This has led to the off-label use of stent grafts that were not originally designed for this kind of pathology and in turn have had many limitations. As a result, device manufacturers have started designing endografts for that specific indication. A nonrandomized registry for the new C-TAG device (W.L. Gore & Associates, Inc., Flagstaff, AZ), is under way to evaluate the safety and efficacy of the device in DTAI.[6]

Other complicated thoracic aortic pathologies include mycotic and infected aortic aneurysm and aortoesophageal and aortobronchial fistula. These pathologies are all infected pathologies and carry an extremely high mortality rate if treated only medically.[8] The gold standard open surgery is also associated with unacceptably high mortality and morbidity rates.[9, 10] Compared to open surgery, the use of minimally invasive TEVAR to treat these pathologies in those often moribund patients has been reported to have early mortality as low as 0%.[11-13] The late results of the procedure remain poor, but the risk of late mortality is often related to the underlying comorbidities frequently present in this high-risk population. It appears that thoracic endovascular repair is considered a suitable but palliative therapeutic option in patients presenting with infected thoracic aortic pathology.[14]

Future Directions

The use of TEVAR in the treatment of thoracic aortic pathology is an active field that carries many promises for the future on multiple fronts. Devices will definitely become more developed and will be more suited to the specific pathology they are intended to treat. Improvement in the delivery platforms will allow safer and more accurate device deployments. Smaller device calibers will reduce both access-site complications and the use of surgical conduits currently needed in 9–21% of cases.[15] More flexible devices will conform better to the aorta, specifically in the hostile aortic arch environment as well as other tortuous areas. Also, the development of branched devices will expand the utility of the technology in covering more pathologies and treating them in a more effective manner. As a matter of fact, there are multiple attempts at developing such devices by all the major device manufacturing companies.

Imaging is an integral part of the success of TEVAR procedures, and the field is booming with new ideas and technologies. In addition to the liberal use of IVUS and high-quality fluoroscopy,

Figure 7. The modern hybrid suite at The Methodist Hospital. In addition to regular fluoro capabilities, the new Siemens fixed unit (DynaCT) located in the suite features rotational 3D angio/CT capabilities with fusion technology. The monitors can show the patient's vital data, real-time fluoro, IVUS images, previous CT and angio studies, and real-time road mapping.

other newer imaging modalities are being developed and refined to allow a safer and easier performance of complex endovascular procedures. Rotational angiography performed using motorized C-arms can create a three-dimensional (3D) image compared to the 2D image of conventional digital subtraction angiography. Some of the newer machines can also perform intraoperative 3D rotational CT scan. In the United States, three such fluoro CT systems are commercially available: DynaCT (Siemens Medical Solutions, Forchheim, Germany), XperCT (Philips Medical Systems, Eindhoven, The Netherlands), and Innova CT (GE Healthcare, Waukesha, WI) (Figure 7). Both rotational angiography and CT 3D technology allow the operator to have more confidence about vascular and soft tissue anatomy when performing complex endovascular procedures on the thoracic aorta.[16]

Another emerging technology is the ability to fuse past studies on current live fluoroscopically derived images. This is the basic concept behind the so-called roadmapping that can be performed using regular fluoroscopy machines. However, this technique is static in nature; if another fluoro projection is used, the roadmapping capability is lost. To overcome this limitation, a preoperative CT scan is fused over a non-contrast intraoperative rotational CT scan by registering anatomical landmarks in both scans (usually bony landmarks in different projections). Using this technology, the roadmapping becomes dynamic and the C-arm can be moved — maintaining the ability to navigate using the images from the preoperative CT scan fused with the live fluoro image. The data from the preoperative CT can be projected onto the live fluoro either as a full 3D image or in the form of a computer-generated graphic, which will then update in the correct projection depending on the arc-angle of the C-arm (Figure 8).[16] While the efficacy of these new technologies has yet to be determined, imaging technology development will prove to be an integral part of the future of TEVAR.

Another promising area has been the use of medical robotics. Currently, intravascular robots are being developed that may play an important role in the future of endovascular surgery. They can be used to assist endovascular devices in avoiding vascular wall contact with more precise navigation and in cannulation of side branches of the aorta in a more deliberate and predictable manner — which can be extremely helpful in cases of fenestrated and branched endograft.

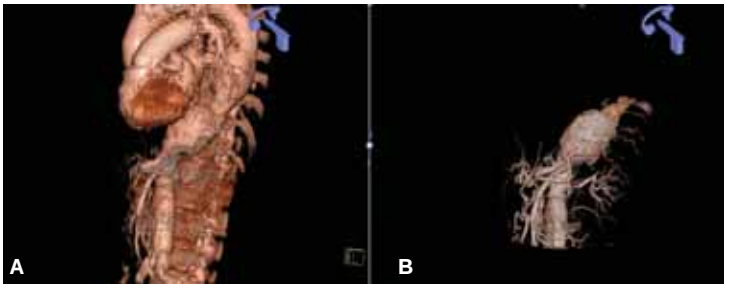

Figure 8. (A) shows a previous 3D CT study superimposed on (B) current 3D angiographic study, which can be fused into the current live fluoro to allow real-time continuous road mapping capability.

The combination of new devices, expanding robotics, and imaging technology development will lead to collective improvement and advancement of the TEVAR technology as a whole.

Summary

Endovascular management of thoracic aortic diseases has made long strides since its introduction in 1994. This modality of therapy has been an important addition in a field of surgery that has typically been associated with high morbidity and mortality. The future definitely carries promise, as there is ongoing development of endovascular devices, techniques, imaging, and technology that will ultimately improve outcomes for patients with aortic disease.

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ENDOVASCULAR REPAIR OF RUPTURED ABDOMINAL AND THORACIC AORTIC ANEURYSMS

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Abstract

Management of acute aortic pathology remains one of the most challenging clinical entities, with a persistently high mortality rate both prior to and upon arrival to a hospital.[1] Responding to the distinct advantages of endovascular approaches to aortic disease, many high-volume cardiovascular centers have focused on endovascular therapies for managing patients with ruptured or leaking aortic aneurysms and other acute aortic syndromes. Nonetheless, similar to outcomes for other surgical emergencies, time and efficiency are critical in managing these conditions. Early diagnosis, transport to an appropriate acute care facility, rapid institution of optimal medical management, availability of cardiovascular anesthesia and intensive care, and appropriate and timely surgical intervention continue to be the keys to success.[2] This article discusses the endovascular approach to ruptured abdominal and thoracic aortic aneurysms.

Endovascular Repair of Ruptured Abdominal Aortic Aneurysms (AAA)

Ruptured AAA

Abdominal aortic aneurysmal disease affects approximately 5% of men and 1% of women over the age of 60 years, and multiple epidemiological studies indicate that the incidence is increasing despite improved medical management of certain risk factors.[3] The mortality rate of a ruptured abdominal aortic aneurysm (rAAA) approaches 90%, and perioperative mortality averages 50% during emergency open repair.[1] Ruptured AAA is the tenth-leading cause of death in the United States, and much of this occurrence is underestimated due to lack of autopsy proof and the default attribution of death to a cardiac event.[1, 3-5]

In the setting of rAAA, patients who survive the initial event and present to an emergency room are often in profound circulatory shock due to hemorrhage and peripheral ischemia-reperfusion injury upon restoration of flow.[6] The subsequent mortality rate of patients who do survive the initial repair approaches 50% due to the "second hit" of systemic inflammatory response syndrome (SIRS), which can later progress to sequential organ failure and multiple organ dysfunction syndrome (MODS).[7-9]

Endovascular Repair for Ruptured AAA

It has been nearly two decades since Parodi and colleagues pioneered the first endovascular aortic aneurysm repair (EVAR) in humans using a Dacron graft introduced retrograde through the femoral artery.[10, 11] Buoyed by device development and refinement of techniques, EVAR has now replaced open aortic repair as the treatment of choice for patients undergoing elective AAA treatment.[12] This evolution stems from large, randomized, controlled trials over the last decade that compared morbidity and mortality rates and favored EVAR over traditional open repair in appropriately selected patients.[13-17]

Several studies have confirmed the feasibility of an endovascular approach to rAAA. Especially when performed under local anesthesia, EVAR provides less of a physiologic challenge compared to traditional open repair.[18, 19] EVAR for rAAA was first reported in 1994 by Marin et al. Since then, worldwide experience from large-volume centers performing emergency EVAR (eEVAR) demonstrates that 30-day mortality rates vary from 7–39%, with an average of 22%.[20] This represents a reduction by nearly half when compared to conventional emergency open repair.[20-23] Variation in these results likely represents the lack of a standard protocol for perioperative care, the steep learning curve for lower-volume centers, and the bias for performing eEVAR in older, sicker patients with pre-existing comorbidities.[24]

To date, there are no large randomized controlled trials comparing the efficacy of eEVAR to traditional open repair, and little long-term outcomes data exists. A recent Cochrane Collaboration review concludes that without randomized controlled trials, the benefit of eEVAR remains undetermined.[6] In response, recruitment is ongoing for three European randomized trials comparing emergency open repair vs. eEVAR for rAAA: Amsterdam's Acute Endovascular Treatment to Improve Outcome of Ruptured Aortoiliac Aneurysms (AJAX) trial, Paris's Ruptured Aorta-Iliac Aneurysms: Endo vs. Surgery (ECAR) trial, and the United Kingdom-based Immediate Management of the Patient with Rupture: Open vs. Endovascular Repair (IMPROVE) trial.[25] Some would argue that despite the lack of randomized controlled trials, the adaptation of eEVAR into current practice is appropriate based on evidence collected worldwide from large-volume and teaching institutions.[26] Even when considered independent of volume, in-hospital mortality is significantly reduced when eEVAR for rAAA is performed in teaching institutions with vascular surgery training programs.[27]

Technical Aspects of eEVAR

The endovascular care of patients with suspected rAAA depends on a comprehensive knowledge of endovascular techniques along with the equipment and facilities to perform intraoperative imaging on an emergent basis. A multidisciplinary approach must be implemented that includes emergency and operating-room personnel, imaging technologists, anesthesiologists, and intensive care physicians. An algorithm for the treatment of patients with suspected rAAA must first

determine hemodynamic stability. Unstable patients, typically defined as those with a systolic blood pressure (SBP) <90 mmHg, are immediately transported to the operating suite and prepared for either open or endovascular repair. Patients who are hemodynamically stable (i.e., SBP >90 mmHg) swiftly undergo computed tomography angiography (CTA); once rAAA is diagnosed, the anatomic suitability for EVAR is determined. Patients with infrarenal neck length ≥10 mm, aortic diameter ≤32 mm, bilateral iliac artery diameter ≥5 mm, and neck angulation ≤75° are generally suitable for eEVAR. Although this expands the current Food and Drug Administration (FDA) anatomical criteria for elective EVAR,[28] the majority of patients who present with rAAA can be managed effectively with eEVAR based on the above criteria.[29, 30]

All patients must receive adequate, but not excessive, resuscitation while definitive treatment is being initiated. Permissive hypotension, in which SBP is maintained around 70 mmHg (between 50 and 100 mmHg), has proven to be an efficacious therapy, and this gentle physiologic condition can be maintained by the administration of local or loco-regional anesthesia during repair.[23, 31, 32]

After the patient is prepped and draped in the operating room, ipsilateral access is obtained either percutaneously or with femoral artery cutdown, and the floppy guidewire is exchanged for a stiff wire in order to pass a large sheath (12–14 ft x 45 cm in length) for an aortic occlusion balloon. In hemodynamically unstable patients, the balloon is inflated in the supraceliac position, while in stable patients the balloon can be placed in the juxtarenal aorta as a precautionary measure. It is important to ensure that the sheath is advanced fully to stabilize the occlusion balloon in the correct position and prevent downward displacement into the AAA. The stent graft, typically a modular device, is then exchanged under fluoroscopic guidance for the aortic occlusion balloon if the patient remains stable. In cases that necessitate deployment of the occlusion device, the aortogram may be performed through the sheath of the occlusion balloon, and the stent graft may be exchanged from the flush catheter in the contralateral limb for deployment.[30] Alternatively, the aortic occlusion balloon may be placed in the contralateral femoral and an aortogram performed through an ipsilateral pigtail catheter that is then exchanged for the main body of the stent graft (Figure 1). Post-deployment arteriogram is performed to ensure adequate seal and exclusion of the rAAA. If a type I endoleak is discovered, adjunctive procedures such as additional ballooning, aortic cuffs, or Palmaz stent placement are performed. Total percutaneous eEVAR can be achieved in experienced hands and in patients with favorable anatomy.[33]

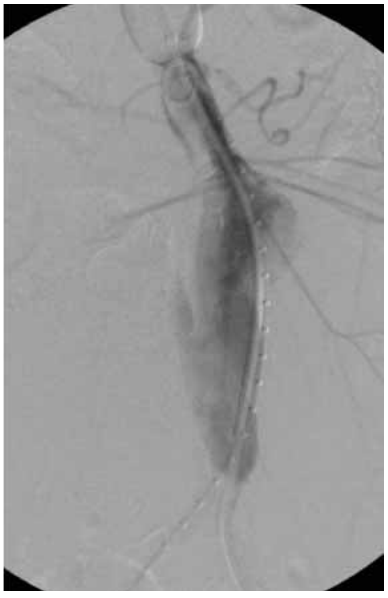

Figure 1. Aortogram performed through an ipsilateral marking pigtail catheter with contralateral aortic occlusion balloon inflated in a hemodynamically unstable patient. The pigtail catheter is replaced with the stent graft for deployment.

Conversion to Open Repair

On occasion, eEVAR fails to provide definitive repair in the setting of rAAA, and open surgical repair is needed. While techniques for open repair will not be discussed here, two important points must be made. First, during conversion to laparotomy, the aortic occlusion balloon and its corresponding sheath are secured in place to maintain aortic occlusion at the appropriate level and prevent dislodgement of the balloon into the rAAA. Additionally, the approach to open repair must be tailored to the type of endograft deployed, including the position of proximal and distal fixation.

Postoperative Care

Postoperative care in the intensive care unit is necessary for continuous hemodynamic monitoring. Vigilant surveillance for signs of abdominal compartment syndrome (ACS) must not be underemphasized as this is a frequent complication. Factors associated with the onset of ACS include hemodynamic instability, massive transfusion requirement, and postoperative coagulopathy.[30] Bladder pressure monitoring and frequent assessment of pulmonary and renal function must not be neglected, and if signs of organ dysfunction ensue, a decompressive laparotomy with temporary abdominal closure is sought.

Complications of eEVAR

Complications of eEVAR are similar to complications from elective EVAR and include endoleak, need for reintervention, stent graft migration, contrast and atheroemboli-induced renal failure, hemorrhage, peripheral ischemia, and local wound complications.[25] Similar to patients after elective EVAR, patients treated with eEVAR undergo postoperative and scheduled surveillance imaging, including contrast-enhanced CT or possibly contrast-enhanced ultrasonography. Long-term outcomes data after eEVAR for rAAA have yet to emerge; however, when compared to open repair, initial trends suggest that the higher secondary intervention rates documented after EVAR[13] could be replicated in patients following eEVAR.[31]

Endovascular Repair of Ruptured Thoracic Aortic Aneurysms

Thoracic aortic aneurysms (TAAs) occur less frequently than AAAs but harbor a 20–54% five-year survival rate due to fatal rupture if left untreated.[34, 35] Elective intervention is generally recommended for TAAs greater than 5.5 cm in diameter due to an annual mortality and rupture risk of 15%.[35-38] Ruptured TAA (rTAA) occurs less frequently than rAAA; however, of the patients who do survive transport to a hospital, overall mortality rates approach 97%.[39, 40]

Open surgical repair of the rTAA, first described in 1951 by Lam and Aram, provides direct inspection of the aneurysm and surrounding branches but requires thoracotomy, aortic cross-clamping, and sometimes cardiopulmonary bypass.[39, 41] First applied in the setting of elective repair, thoracic EVAR (TEVAR) 30-day mortality rates demonstrated an improvement over traditional open repair in several non-randomized trials.[42-44] Emergent thoracic EVAR (eTEVAR) was first attempted for rTAA in 1997 by Semba et al.[45] Since then, eTEVAR has been applied both to rTAA and traumatic thoracic aortic injuries with improvements in perioperative mortality rates.[39, 46-48] In a large, systematic meta-analysis of traditional open repair versus eTEVAR for rTAA, Jonker et al. found a significantly lower 30-day mortality rate (33% vs. 19%, p = 0.016) and incidence of myocardial infarction (11.1% vs. 3.5%, p = 0.047); however, no significant differences in rates of stroke or paraplegia were observed.[39]

Technical Aspects of eTEVAR

Similar to eEVAR, eTEVAR requires appropriate preoperative evaluation with CTA and the facilities available to assemble the eTEVAR team quickly, including having a hybrid operating room immediately available. In cases where conversion to open repair is necessary, the facility must also have immediate access to resources that include thoracic surgery backup.

Local, spinal, or general anesthesia can be selected depending on the patient's clinical condition, providing that SBP remains less than 100 mmHg. Emergent TEVAR can be performed via standard femoral cutdown; alternatively, a total percutaneous approach can also be achieved.[49] In the contralateral femoral artery, a graduated-marked pigtail catheter is introduced and an aortogram is performed. The stent-graft is advanced over a stiff guidewire to the desired position using fluoroscopy. A proximal landing zone of 2 cm prior to the takeoff of the left subclavian artery is generally required; however, successful intentional occlusion has been recorded.[50] An equivalent distal landing zone length of 2 cm is also required, and if two stent-grafts are deployed, overlap must be greater than 5 cm in order to avoid separation in the case of tortuous anatomy.[51] In the setting of TAA, gentle and swift dilation of the proximal and distal landing zones with balloon angioplasty can secure wall apposition of the stent-graft.[51] Stent-graft size selection is based on the diameters of the proximal and distal landing zones, and an oversize factor of 20–30% is generally allowed to facilitate secure anchoring and seal.[52]

Complications of eTEVAR

Complications of both open and eTEVAR include cardiac events, stroke, paraplegia, multiple organ failure, and infection.[39] Concern over graft durability and a relatively high rate of endoleak (>10%) has led to a requirement by the FDA for continuous surveillance with contrast-enhanced CT after repair.[36, 39] Aneurysm-related death after eTEVAR remains higher than after open repair, and estimated aneurysm-related survival after 3 years remains at 71%.[39] The need for improved device technology greatly outshadows the low overall incidence of ruptured TAA, and industry attention to device advancement is necessary.

Paraplegia caused by the interruption of branch vessels to the spinal cord occurs less frequently after TEVAR than open repair; however, the risk remains between 3–6%.[51] Factors that influence spinal cord ischemia include prior AAA repair, length of thoracic aorta coverage, hypogastric artery interruption, subclavian artery coverage, emergency repair, and hypotension.[51] Strategies proposed to reduce this complication include cerebrospinal fluid drainage, intercostal artery reimplantation, hypothermia, and maintenance of normal SBP, though none of these approaches have consistently proven efficacious.

Case Example of eTEVAR

A 94-year-old Caucasian male who previously had an rAAA treated with eEVAR presented with abdominal pain, hemorrhagic shock, and a left hemothorax. An urgent CT scan of his chest, abdomen, and pelvis demonstrated an aneurysm of the descending thoracic aorta, with blood in the left chest consistent with a ruptured thoracic aneurysm. The Methodist Hospital Acute Aortic Treatment Center protocol was initiated,[2] and he immediately underwent eTEVAR (34 mm x 20 cm TAG) and placement of a left chest tube thoracostomy to evacuate the hemothorax. The patient recovered from this event and was eventually discharged from the hospital. He continues to be asymptomatic at 2-year follow-up with no complications on monitoring CTA (Figure 2).

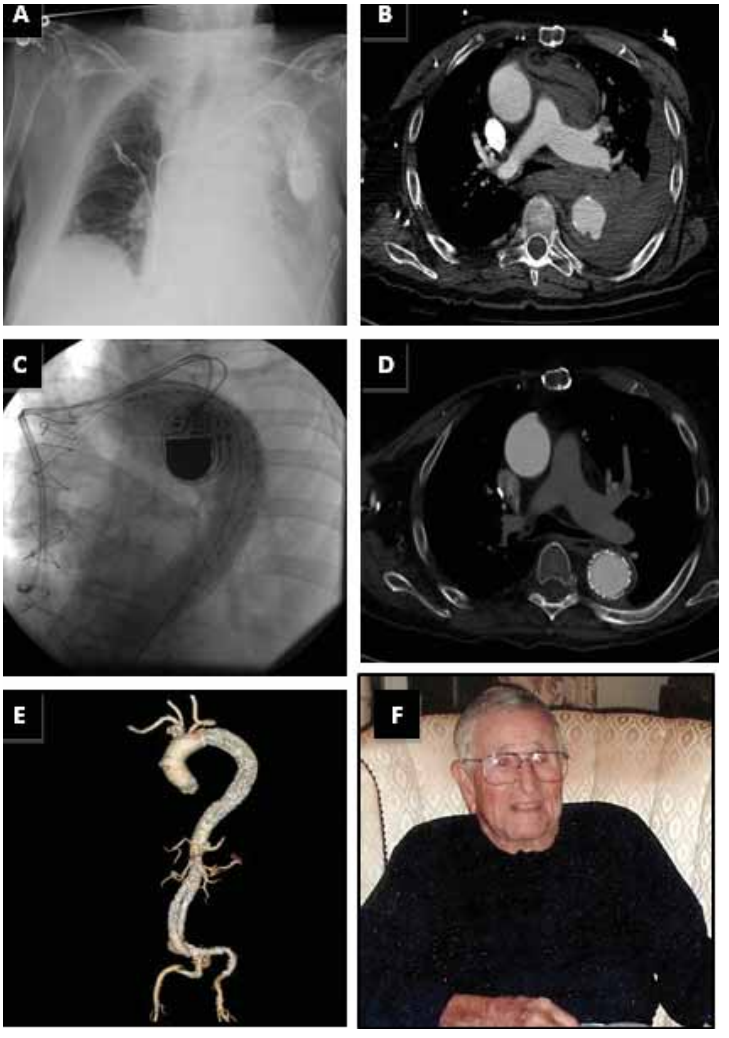

Figure 2. Preoperative, postoperative, and follow-up images of rTAA and rAAA in the same patient. (A) Chest X-ray on presentation demonstrating left hemothorax. (B) CT showing rTAA with hemothorax. (C) Deployment of a TAG® endograft (W.L. Gore & Associates, Inc., Flagstaff, AZ) for repair of ruptured aneurysm. (D) CT at 2 years follow-up. (E) 3D reconstruction of thoracic and abdominal aortic stent graft repair of rTAA and rAAA. (F) Patient with full recovery 2 years after eEVAR of combined rTAA and rAAA.

Conclusion

The evolution of endovascular treatment of AAA and TAA has dramatically changed the approach to these life-threatening diseases. Even in the acute setting of aneurysm rupture, an otherwise fatal condition can now be approached in a minimally invasive manner, providing improved perioperative outcomes and increased options for high-risk patients. Large-volume teaching institutions clearly provide the highest standard of care with this new technology and have consistently demonstrated superior results.[24] In the elective repair of AAA, EVAR is now considered the gold standard in anatomically amenable patients.[12] While long-term outcomes data of eEVAR for rAAA and rTAA remains to be seen, the endovascular approach shows promise in becoming the future standard of care for ruptured aortic aneurysmal disease.

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J.J. Naoum, M.D.

CHANGING PARADIGM IN ENDOVASCULAR TREATMENT OF DESCENDING THORACIC AORTIC DISSECTIONS

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Abstract

Descending thoracic dissections originating distal to the origin of the left subclavian artery carry a significant mortality if left untreated. Past thinking advocated avoiding surgical treatment of acute Stanford type B or DeBakey type III dissections, reserving therapy for chronic dissections over 14 days to a month after presentation.[1] The current evolution of endovascular devices for the treatment of thoracic aneurysms has proven helpful in treating this pathology in a less invasive manner when compared to open surgical repair. The paradigm for treatment has evolved beyond the nature of the timing of the dissection: the current trend for treatment considers clinical findings and the development of complications. Complicated dissections include those that have developed aneurysmal dilatation >5.5 or 6 cm, organ or distal limb malperfusion, aortic rupture, uncontrolled hypertension even after adequate medical therapy, and persistent pain including rapid expansion of the affected aorta, among others (Table 1).[2-5] This article reports on the current paradigm involving thoracic endovascular aortic repair (TEVAR) of Stanford type B or DeBakey type III dissections.

Criteria for complicated descending thoracic aortic dissections
Rupture
Malperfusion
Aneurysmal degeneration
Rapid aortic expansion/dilatation
Uncontrolled hypertension on adequate medical therapy
Persistent pain attributed to the dissection

Table 1. Criteria for complicated descending thoracic aortic dissections.

Uncomplicated Descending Thoracic Aortic Dissection

Xu and associates evaluated their experience in treating 84 patients with chronic aortic dissection with endovascular stent grafting.[1] Only 3 patients were complicated and presenting with rupture. The entry tear was sealed in 91.7% of patients. Three patients died of rupture of the thoracic aorta because of endoleak, and when present, this accounted for 42% of the deaths. At 5 years, 75.2% of patients were alive without an endoleak or the need for any additional endovascular or surgical intervention. The authors suggest that with increased surgical experience and refinement of the stent grafts, results are expected to improve.

The INvestigation of STEnt Grafts in Patients With Type B Aortic Dissection (INSTEAD) trial prospectively enrolled 140 patients with a stable, uncomplicated type B dissection, and subjects were randomly assigned to be treated with stent graft placement and optimal medical therapy or to optimal medical therapy and surveillance.[6] Patients were considered unsuitable for randomization in the presence of acute complications or thoracic aortic diameter ≥6 cm. Notably, from 597 patients screened, only 140 met randomization criteria. Cumulative 1-year survival was 97% ± 3.4% with optimal therapy and 91.3% ± 2.1% with endovascular aortic repair. Aortic expansion >6 cm was more prevalent with medical treatment and was followed by crossover to TEVAR in 11.2% of patients and conversion to open repair in 4.4% of patients, accounting for a 15.6% conversion rate overall. Stent graft placement was associated with a 92.6% complete false lumen thrombosis and morphologic evidence of aortic remodeling that reached significance.

The INSTEAD trial revealed that the structural and morphologic remodeling associated with prophylactic TEVAR in uncomplicated patients did not improve survival compared to optimal medical therapy and careful surveillance at either 1 or 2 years.[6,7] For an

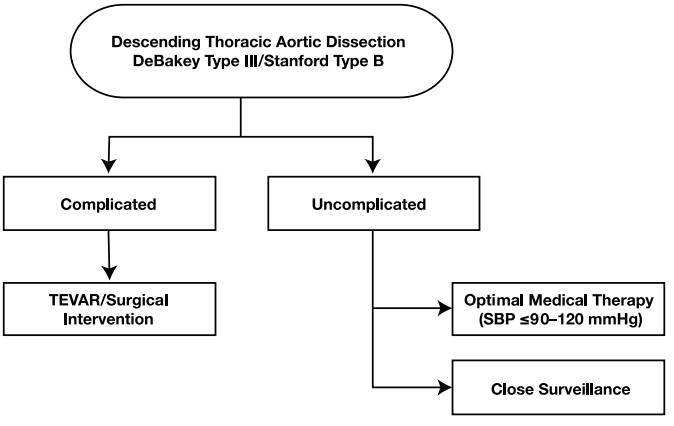

Figure 1. Basic algorithm for the treatment of descending thoracic aortic dissections.

uncomplicated descending aortic dissection, medical management has an excellent outcome as long as surveillance is used to identify progression of aortic disease and any complications that will then necessitate surgical intervention (Figure 1).

This data provides evidence that supports the treatment of uncomplicated Type III aortic dissection with optimal blood pressure control with an SBP ≤90-120 mmHg and with additional careful surveillance at 3- to 6-month intervals to identify potential complications.[7, 8]

Complicated Descending Thoracic Aortic Dissection

In a report by Pearce and colleagues,[9] 127 patients were treated with TEVAR for a type III thoracic aortic dissection; of those, 15 patients had a complicated and acute presentation. Indications for repair in the latter group included malperfusion in 53% of patients, persistent pain in 27%, and aortic failure in 33%. Malperfusion resolved in 80% of patients. Overall 30-day complications occurred in 46.7% and were associated with 13.3% mortality, 13.3% paraplegia, and 13.3% renal failure needing hemodialysis. The authors stressed that the intent of intervention was the stabilization of the true lumen and correction of malperfusion. During follow-up, they observed the successful exclusion of the entry flap and thrombosis of the false lumen. Interestingly, this was not associated with a reduction in the overall aortic size, a finding that is in contrast with a reported 71% decrease in aortic size observed with TEVAR in the treatment of complicated chronic dissections.[10]

Steuer and associates investigated the early and long-term outcomes of TEVAR for complicated type III dissections.[11] A retrospective review was carried out in 50 patients with acute complicated aortic dissection and in an additional 10 patients with complications >14 days after the onset of symptoms. Complications included rupture, end-organ ischemia, and acute dilatation. Within 30 days, these 60 patients had a 3% mortality rate, a 2% incidence of paraplegia, and a 5% stroke rate. Five-year survival and freedom from reintervention were 87% and 65%, respectively. The authors concluded that for acute complicated type B aortic dissection, TEVAR can be performed with excellent survival. However, its morbidity and durability still need further evaluation.

A complicated acute type B aortic dissection requires early intervention. Rakhlin and associates utilized TEVAR to treat 26 patients with malperfusion, 22 patients with rupture, and 17 patients with both complications as a result of an acute aortic Type B dissection.[12] Thoracic endografting alone was successful in treating 95% of patients with thoracic aortic rupture while being effective only in 58% of cases of malperfusion. In the latter group, 42% required additional adjunctive procedures to restore end-organ perfusion, predominantly to the lower extremity in 50%. While TEVAR alone was sufficient to manage aortic disruption in patients with rupture, additional procedures were necessary in cases with malperfusion. This suggests that endovascular therapy must be tailored to each patient presentation in order to achieve improved results.

In a similar study by Szeto and colleagues, 35 patients were treated with TEVAR for acute complicated Type B aortic dissection.[2] Rupture was present in 18 patients and malperfusion in 17. The primary tear site was successfully covered in 97.1% of patients. Coverage of the left subclavian artery was needed in 71.4% of patients. In cases of malperfusion, adjunctive procedures were necessary in 88.2%. Thirty-day mortality was 2.8%, while 1-year survival reached 93.4% ± 4.6%. Renal failure, stroke, and permanent spinal cord ischemia each occurred in 2.8% of patients and vascular access complications in 14.2%. Length of hospital stay was greater in patients with malperfusion compared to patients presenting with rupture. However, another report has shown a 16% 30-day mortality associated with TEVAR in the treatment of acute complicated aortic dissection in which 79% of patients had either malperfusion or rupture.[4]

The indications for treatment of acute aortic dissections usually involve rupture, malperfusion, and in fewer instances persistent and uncontrolled hypertension. Complications in chronic dissections usually involve aneurysmal degeneration or progression, even though rupture and malperfusion can still occur. Parsa and colleagues published their mid-term results using TEVAR for the treatment of complicated acute and chronic type B aortic dissections.[10] Their definition of acuteness was the 2-week mark after onset of symptoms. The 30-day mortality and paraplegia or paresis were both at 2%. Endoleaks were present in 21% of patients, and 23.4% required reintervention with distal endografting extension for downstream dilatation. As a result, the authors recommend that in chronic complicated dissections, the endograft should be extended up to the celiac axis.

The goals of TEVAR in complicated aortic dissection are to achieve coverage of the proximal aortic primary tear to allow expansion of the true lumen and the concomitant thrombosis or obliteration of the false lumen (Figure 2). The end result is to re-establish adequate flow through the aorta and distal end-organ or limb perfusion.[2, 8, 9, 13]

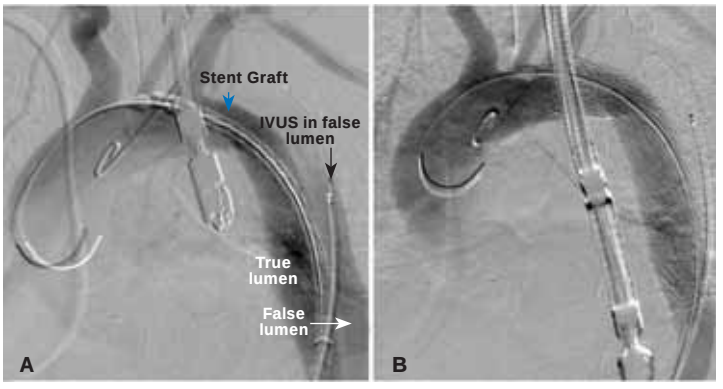

Figure 2. Treatment of a complicated aortic dissection. (A) Arteriogram before stent graft deployment showing perfusion of the false and true lumen. (B) Arteriogram following successful stent graft deployment with coverage of the proximal aortic primary tear, expansion of the true lumen, and lack of perfusion of the false lumen.

Aortic Remodeling Following TEVAR

Thrombosis of the false lumen is expected to lead to its eventual shrinkage and obliteration. Its effects are believed to stabilize the dissected aorta and allow for its remodeling.[13, 14] In the INSTEAD trial,[6] the process of false lumen thrombosis in the aorta was enhanced with TEVAR, achieving 92.6% complete thrombosis and morphologic evidence of aortic remodeling confirmed by radiographic imaging at one year. This intuitive concept does not always hold true, thus highlighting the fact that multiple factors come into play. Pearce and colleagues showed that even with persistent exclusion of the entry flap and consistent thrombosis of the thoracic lumen in their patients, reduction of the overall aortic size was not observed during an 11 ± 6-month follow-up.[9]

Patency of the false lumen is a major factor affecting the natural progression of the aorta, whether treated with TEVAR or medical therapy. With failure to thrombose, the false lumen will maintain blood flow and lead to malperfusion, rupture, aneurysmal dilatation, and compression of the true lumen, even if treated with

a stent graft. Thus, the presence of an endoleak can correlate with increased mortality and rupture.[1]

In contrast to TEVAR treatment of descending aortic dissections, optimal medical treatment alone failed to demonstrate expansion of the true lumen or shrinkage of the false lumen, leading to aortic expansion >6 cm in 15.6% of patients.[6, 7] In addition, it has been associated with 13.7% mortality.[15] However, ruptures have still been observed in the face of false lumen thrombosis in 22.6% of medically treated patients and in 31.6% of patients who experienced partial thrombosis.[15] It is possible that aortic thrombus diminishes nourishment to the vessel wall, thus weakening it in the setting of increasing radial pressure in the former case.[16] Formation of partial thrombus leads to flow obstruction in the false lumen, increasing the existing pressure in that channel and possibly leading to rupture.[2, 13]

Conclusions

Our current understanding of descending aortic dissections should now lead us to shift our thinking from acute and chronic dissections to the concept of complicated and uncomplicated dissections. The timing in which the dissection occurs is important; however, surgical or endovascular treatment should be guided by the presence of symptoms rather than by the existence of the dissection alone. In uncomplicated cases, medical management is optimal. Endovascular treatment of descending aortic dissections has emerged as a less invasive and promising tool to combat this condition in symptomatic patients with an otherwise near-lethal disease.

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A.B. Lumsden, M.D.

ADVANCED AORTIC IMAGING: FUTURE DIRECTIONS

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Abstract

There have been dramatic advances in aortic imaging over the last decade. Some of these capabilities have been driven by the development of aortic endografts, the need for accurate measurement of aortic dimensions, and capabilities for simulating endograft placement. The development of three-dimensional (3D) reconstruction has rapidly moved from being an additional luxury item to a commodity, either packaged into advanced imaging systems or freely available as downloadable, highly advanced software such as OsiriX for the Macintosh computer. Other advances such as dynamic magnetic resonance angiography (MRA) have resulted from continuous improvement in the hardware (acquisition of signal) and software (post-processing capabilities) of these imaging systems. We are particularly intrigued by the ability of these capabilities to improve the diagnosis and treatment of aortic disease. Furthermore, there is a rapidly emerging field of creating a 3D image in the interventional suite, which can potentially be used to steer catheter-based robots in a manner never before conceived. These various components will be described below.

The Basics

Interventional imaging has to include four features in order to be of great promise in the endovascular arena: 1) the equipment must have adequate definition and thereby be able to characterize tissues and define boundaries between anatomic structures; 2) the system must be interactive and intuitive; 3) three-dimensional capabilities are necessary when navigating vascular anatomy; and 4) the system must include a fourth dimension, the ability to evaluate motion. The vascular bed is a dynamic one, and therefore not including motion could allow for misinterpretation. Motion occurs with the cardiac cycle, respiration, and aortic pulsation. Deformation also occurs when stiff devices are advanced through blood vessels. Both motion and deformation can affect the accuracy of procedure performance. The ability to compensate for both motion and deformation are currently being developed.

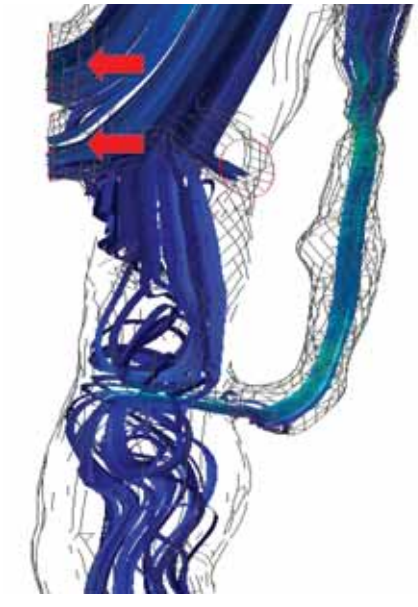

Figure 1. Computational fluid dynamic analysis of re-entry points of a type B dissection distal to the celiac and superior mesenteric arteries (arrows).

Dynamic Magnetic Resonance Angiography

Although the resolution of 3D computed tomography (CT) scans is optimal, magnetic resonance imaging (MRI) has the added advantage of providing additional physiologic data. We utilize dynamic 3D MR reconstruction in all patients with aortic dissections, often with computational fluid dynamics (CFD) overlay (Figure 1). Dynamic MRA has also been very useful in demonstrating mobile aortic thrombi when searching for an embolic source (Figure 2). While computational simulations in general, and of blood flow in human arteries in particular, have been the topic of research in the last decades,[1,2] only recently with the introduction of advanced clinical imaging techniques and progressed computing power has it been possible to tailor these simulations towards the conditions found in a particular individual.[3-6] Initially, CFD simulations were restricted to 2D models and idealized geometries. Solutions even for these simplified geometries could only be obtained after many hours or even days. Continuous technical advances, however, have now made it possible to convert information from images acquired during a routine clinical exam into 3D complex mathematical

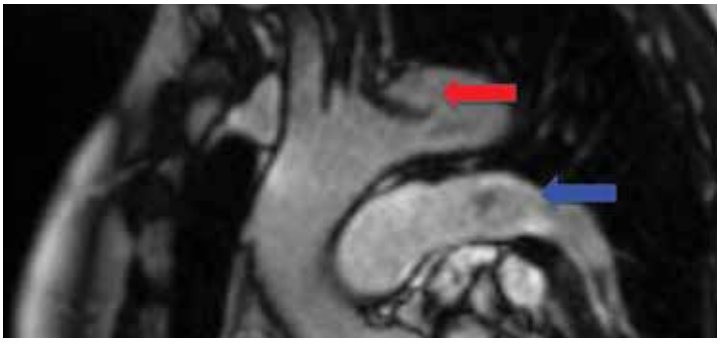

Figure 2. There is a large mobile embolus lodged in the orifice of the left subclavian artery (red arrow) and a second embolism in the left pulmonary artery (blue arrow). This patient had a patent foramen ovale and was suffering from paradoxical emboli. Both these lesions were highly mobile on the dynamic study.

Figure 3. Robotic hybrid room that permits angiography and the acquisition of a fluoro CT scan.

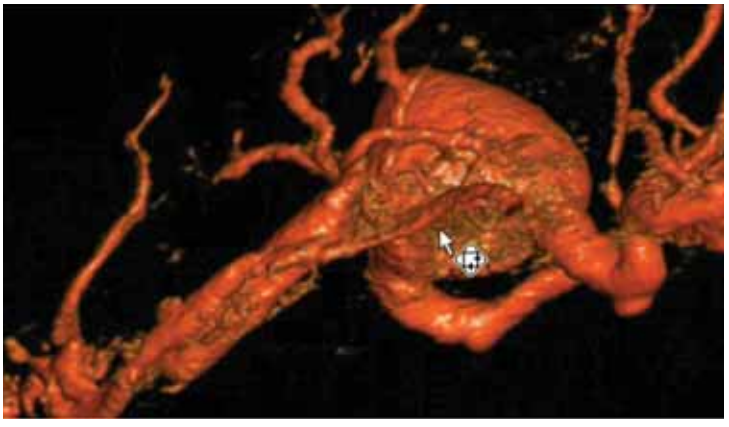

Figure 4. 3D reconstruction of a splenic artery aneurysm showing 2 large branches arising from the aneurysm. In the aortogram, it appeared that this was a simple saccular aneurysm that would have been amenable to stent grafting.

meshes consisting of hundreds of thousands of small-volume elements for transient simulation of the hemodynamics in human artery segments, either in health or in disease.[7] The results of these simulations provide access to hemodynamic parameters that are currently not reliably measurable with clinical imaging methods. Arguably, one of the most important of these parameters is the wall shear stress (WSS) that the flowing blood is exerting onto the arterial wall. Wall shear is an important determinant of dissection. Other parameters include dynamic pressures (dynP) and recirculation patterns; the latter may facilitate the adhesion of material onto the artery wall and promote the creation of atherosclerotic lesions, as in the bulb of the carotid bifurcation, for example.

Hemodynamics may play an important role in type B aortic dissections (TB-AD). A recent flow study of a chronic TB-AD demonstrated a direct dependence of systolic and diastolic pressures in the true and false lumen on TB-AD morphology, emphasizing the need for a better understanding of hemodynamic forces in TB-AD.[8, 9] Towards this goal, we employed CFD simulations to investigate the feasibility of quantifying changes in hemodynamic parameters before and after thoracic endovascular aortic repair (TEVAR) treatment of type B aortic dissections.[10, 11]

Fluoro Computed Tomography

The ability to rapidly spin a radiation source and detector around the patient permits acquisition of a wide-field computerized tomographic image. Imaging companies have developed new combined angiography/CT suites, which use flat-panel detector (FD) technology for improved resolution angiography that is also able to produce improved cone-beam volume CT images (Figure 3). The system permits 3D rotational digital subtraction angiography (DSA) or cone-beam volume CT interchangeably with the same FD C-arm (Figure 4) so that patients do not have to be transferred to a separate unit in order to obtain both imaging modalities. Real-time feedback of endovascular procedures is possible for both DSA and CT.

When comparing fluoro CT to a 16-slice multidetector CT scanner, Irie et al. found that fluoro CT was able to scan a wider area in a shorter period of time while delivering superior quality coronal and sagittal reconstruction images.[12] Fluoro CT allows a contrast resolution of 10 HU as well as a slice thickness and in-plane resolution of <1 mm.[13]

One of the concerns with this cone-beam technology is the amount of radiation exposure to the surgeon/interventionalist and patient. It was found that the total radiation dose is 236 mGy for FD-based fluoro CT, while the dose for 3D DSA using the same system is about 50 mGy.[12] Other authors revealed that the dose of radiation for a conventional head CT was similar to that of fluoro CT, namely 60 mGy.[14]

Fluoro CT can be used to import and overlay previously acquired 64-slice images (Figure 5). This registration process allows interventionalists to intervene in real time using a previously acquired high-resolution image. For the first time, interventionalists will have the ability to rapidly acquire a CT image while performing a procedure and to evaluate the adequacy of an intervention. This CT capability is likely to dramatically affect how many procedures are performed, allowing point-of-service adjustment of an operative plan (Figures 6a, 6b, 7). One of the areas where fluoro CT has the potential to garner the most advantages is as a navigational tool. As devices become more refined and are able to challenge more complex anatomy, fluoro CT will be able to assist in obtaining the indispensable 3D imaging necessary to situate and guide the instrument to its target. This can be a particularly attractive feature when one starts discussing potential applications for flexible robotics.

Figure 5. A 3D angiogram (white) has been fused on top of a previously acquired CT scan (brown), demonstrating how accurate image fusion can be.

Importing and Fusing Images: Registration of Axial Imaging with Angiography

The fusion of axial imaging and angiography in the interventional suite will soon enable endovascular navigation in

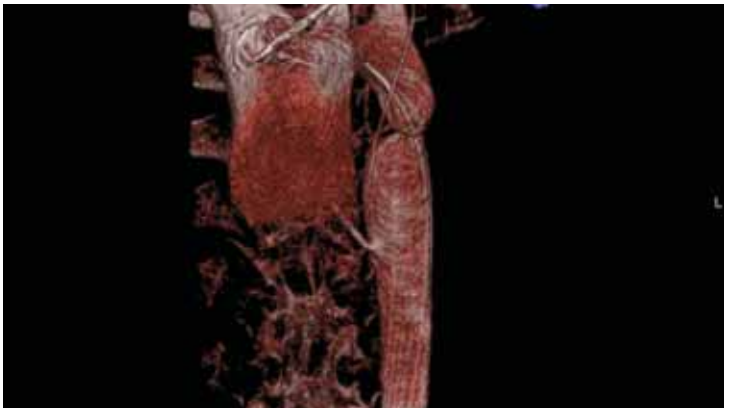

Figure 6A. Fluoro CT scan of an aortic coarctation with a stent graft being positioned within the stenosis.

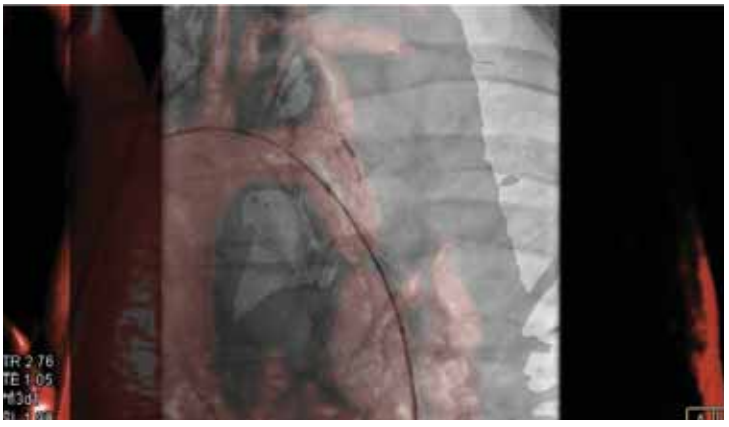

Figure 6B. The fluoro CT image of the aorta has been projected back onto the image intensifier and is being used as a 3D road map for accurate device deployment.

3D-like perspective. Co-registration of multimodality imaging in the angiographic suite overcomes some of the weakness of each separate modality and accentuates the strengths of both. Angiography provides 2D luminal contour detail but does not provide extraluminal tissue information. Combination technology that aligns 2D angiography with 3D images allows for better visualization of vessel tortuosity and the relationship of the lumen to surrounding structures. Since the vessels are 3D structures, visualizing them in 3D during procedures is more intuitive (Figures 6, 7).

Specialized software now allows for co-registration of the angiographic images with the reconstructed axial images. Initially, most applications for fused multimodality imaging were used for cardiac or intracranial interventions.[15-17] In the future, multimodality imaging and processing will likely evolve to become standard tools of vascular specialists. Multimodality image fusion can be achieved in different ways. One option for co-registration of fluoroscopy and axial imaging is real-time MRI or CT in the interventional suite. However, this currently requires specialized endovascular equipment and poses safety concerns for the treating clinicians.

Once the CT or MRI images are co-registered with the angiographic images, a real-time working overlay or roadmapping can be projected for the treating clinician. New software to perform the co-registration with CT and MR is now becoming commercially available, but the quality of the fusion images remains unclear.

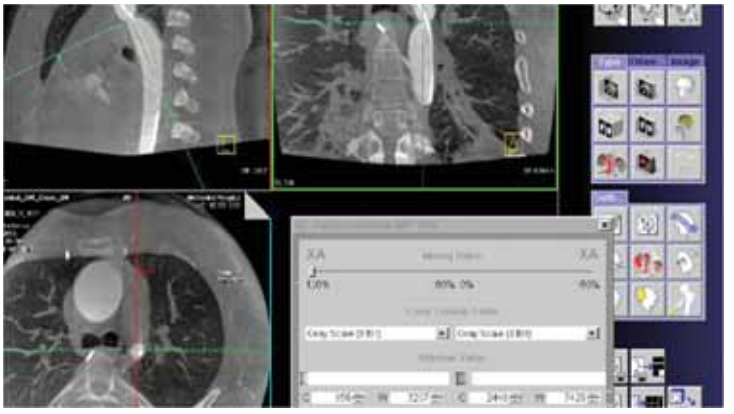

Figure 7. Workstation image of the 3D reconstructed aorta. Center line tracking through the coarctation permits very accurate measurement of the degree of stenosis. The images can be manipulated in multiple different planes and rendered in the format shown in Figure 6A. This patient also has a dilated ascending aorta.

One unsolved problem is the vessel deformity caused by stiff intraluminal wires, catheters, and devices. The deformity of the vessel causes a mismatch between the preoperative imaging and the live angiogram. However, as this exciting technique evolves, more precise and integrated images will become available.

Simulation and Patient-Specific Simulation in Aortic Disease

One real challenge in the evolution of a true simulation experience for aortic endografts has been the disconnect that has existed between simulation companies and the device manufacturers. The device companies have long been uncertain about the real value in simulation and consequently have been reluctant to invest in development of simulation modules. The simulation companies, with fewer cash reserves, have not had the resources or desire to go it alone in the development of these aortic environments. Both have long felt that hospitals should appreciate the potential role of simulation in credentialing and re-credentialing. But, to date, no matrices exist by which hospitals can utilize these expensive simulators to credential, refuse credentialing, or remove credentialing for physicians based on their performance in aortic simulation or in any other endovascular models.

Consequently, it is only recently that aortic endograft simulation models have evolved. Medtronic first developed a thoracic simulation environment in partnership with Medical Simulation Corporation for deployment of their Talent™ endograft. Interestingly, they also developed a dissection module that would have been of immense value but was unavailable for U.S. physicians since this was an off-label indication for their device.

W. L. Gore has partnered with Simbionix to develop an Excluder® (abdominal aortic aneurysm endograft) simulation platform. One potential advantage of the Simbionix platform is the ease of performing patient-specific simulation. CT scans in DICOM format can be loaded into the simulator to provide what is better termed "patient-like" simulation. Why the distinction? Basically, these simulation scenarios are created from a contrast-enhanced CT scan whereas interventionalists deploy devices using angiography. Not only do we deploy using real-time angiography, but the device itself deforms the anatomy and these device-tissue interactions are not yet modeled in simulation scenarios. Consequently, most simulation companies are not quite ready to claim the true fidelity of "patient-specific" scenarios. This claim may need an FDA-

approved clinical trial to demonstrate true fidelity before that claim can be made. Nevertheless, we strongly believe that the capability of continuously modifying the scenarios is absolutely a prerequisite to provide value in simulation. Real value is in simulating tomorrow's case today, provided one is not seduced into performing the procedure because of ease of use in a simulation environment.

Putting It All Together: Illustrative Case

The patient was a 75-year-old male, 4 years post treatment of an abdominal aortic aneurysm with an aortic endograft. Follow-up CT scan showed the presence of an endoleak, thought to be type 2, confirmed by duplex scanning. During the next 2 years there was a progressive increase in aneurysm size. Retrograde embolization of feeding lumbar arteries was performed via the ascending lumbar. The aneurysm continued to increase to 7.3 cm. We decided to proceed with direct sac puncture.

The patient was placed prone on the hybrid table. Noncontrast fluoro CT scan was performed using a Siemens Artis zeego®. The previously performed contrast-enhanced CT scan was imported into the workstation and, using image fusion software, fused with the noncontrast fluoro CT. The procedure was planned on a workstation — where we selected the target endoleak blush on the CT scan, the appropriate skin access location, and placed target markers on both electronically. Length to target was 14 cm. A virtual needle guide was then created and transmitted to the live fluoro image. A 15-cm Chiba needle was advanced along the virtual needle guide directly into the target immediately anterior to the left limb of the device, and a sacogram was performed. This demonstrated a large vessel draining from the sac and several small lumbar arteries. A 4-Fr sheath was advanced into the sac, and selective catheterization of feeding and draining vessels was achieved using standard techniques. Direct embolization was successfully performed. Duplex scanning 24 hours later showed that, for the first time in 4 years, there was no endoleak.

This patient scenario demonstrates the effective use of image fusion software to minimize contrast utilization, real-time case planning in a 3D hybrid environment, and the use of a virtual needle guide to facilitate safe and accurate access to the intra sac leak location.

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C. Duran, M.D.

ROBOTIC AORTIC SURGERY

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Abstract

Surgical robotics was first utilized to facilitate neurosurgical biopsies in 1985, and it has since found application in orthopedics, urology, gynecology, and cardiothoracic, general, and vascular surgery.[1] Surgical assistance systems provide intelligent, versatile tools that augment the physician's ability to treat patients by eliminating hand tremor and enabling dexterous operation inside the patient's body. Surgical robotics systems have enabled surgeons to treat otherwise untreatable conditions while also reducing morbidity and error rates, shortening operative times, reducing radiation exposure, and improving overall workflow.[2] These capabilities have begun to be realized in two important realms of aortic vascular surgery, namely, flexible robotics for exclusion of complex aortic aneurysms using branched endografts, and robot-assisted laparoscopic aortic surgery for occlusive and aneurysmal disease.

Flexible Robotics

Diagnostic and interventional catheters are currently limited by the ability to simply rotate around one axis. One depends on a variety of preformed catheters to fit non-uniform vascular anatomy. Therefore, catheters are often inadequate when performing complex interventions, and surgeons are forced to use a multitude of catheters to get to the site of the intended intervention. Having a catheter with which movement could be controlled in multiple planes would allow for greater precision, confidence, and safety as the surgeon proceeds through the often complex arterial system. Robot-assisted surgery provides such a catheter, enabling fine, predictable, and consistent movements that ultimately increase procedural speed and reliability.

In 2007, Hansen Medical, Inc., the lead developer of robotic technology for endovascular interventions, received FDA approval of their Sensei® Robotic Catheter System for use in cardiac ablation procedures. Vascular surgeons began investigating the value of using the robot to assist in placing endovascular grafts in the aorta with the goals of improving performance, reducing operative time, and overcoming prohibitive obstacles when managing thoracoabdominal aneurysms. The most extensive non-clinical experience in endovascular robotics comes out of work from St. Mary's/Imperial College in London, which demonstrated clear benefits in cannulation times, tool movements, accuracy in cannulation, and performance scores over conventional methods when performing complex endovascular procedures in silicone aortic models.[3, 4] They have also shown clear advantages in terms of minimizing radiation exposure for the operator, with cannulation times reduced from over 17 minutes for conventional methods to less than 3 minutes using the robotic system; this data mirrors studies of ablation for atrial fibrillation, which showed clear reduction in procedure times as well.[5] Valderrabano has also shown decreased radiation times for ablation cases, down to only 5 minutes of fluoroscopy time.[6] In unpublished data, we have recently demonstrated the technical feasibility of robot-assisted antegrade in-situ fenestration of a stent grafting using the Artisan™ catheter (Figure 1), Hansen's first-generation endovascular catheter system, in pigs, which has also previously been shown.[7] Although this technique is far from perfected, it can potentially provide an "off-the-shelf" solution to complex aortic aneurysms. Furthermore, it can provide a solution for troubleshooting when a visceral/renal branch has inadvertently been covered.

Figure 1. The Sensei Robotic Catheter System and Artisan™ Control Catheter from Hansen Medical.

However, the Artisan catheter has notable limitations in practical utility. The 14-French (Fr) sheath is too large for use in a variety of vascular beds, including the infrainguinal, renal, and visceral arteries. The reduced range of motion (1-way fixed bend on sheath, 4-way variable on leader) limits movement of the catheter to a single plane. As such, Hansen has developed a vascular prototype catheter. This new vascular catheter is 9 Fr (outer diameter), while a 6-Fr inner diameter for the sheath accommodates 6-Fr third-party devices. It has steerable inner and outer guidable catheters enabling 6 degrees of freedom and further enhances tactile and visual depiction of tissue deformation. Early work once again shows improvement in the learning curve, and improved cannulation times should further reduce radiation exposure to the patient and operator.

An experienced vascular surgeon and interventional radiologist have compared cannulation times of contralateral iliac, renal, and superior mesenteric arteries, demonstrating improved cannulation times (data submitted for publication). Looking at the incidence of vessel injury in animal models, we have shown superiority of the robotic cannulations with less damage to vessel walls (only 1 event) and intimal thrombus formation (no events) as compared to the manual arm.[8] We anticipate that as these systems continue to advance, the flexible catheters will further enable operators to

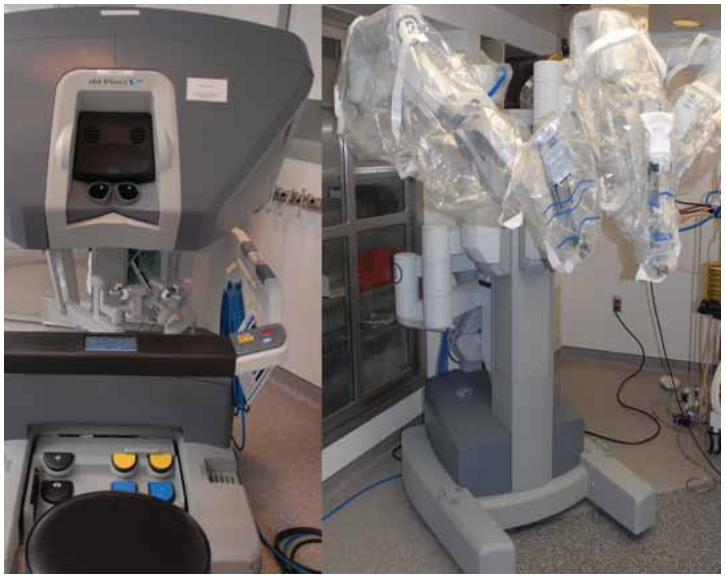

Figure 2. The Intuitive Surgical da Vinci® System.

navigate difficult angles from femoral access points, improve off-wall navigation, and enable successful in-situ fenestration of stent grafts in humans.

Robotic-Assisted Laparoscopic Surgery

Throughout surgical disciplines, the advantages of minimally invasive surgery have been demonstrated and have, in many cases, become the standard (Tables 1, 2). However, the particular difficulty of performing vascular anastomoses has heretofore proved prohibitive for accomplishing timely and safe minimally invasive operations for patients requiring aortic repair. In 1995, Intuitive Surgical, Inc. created the computer-enhanced robotic system known today as the da Vinci Surgical System (Figure 2). The goal of this device was to create familiar hand movements from open surgery while performing operations via a minimally invasive approach. The advent of robotics in cardiovascular surgery made a minimally invasive approach to aortic surgery, a technically challenging procedure, more practicable. Key to the success of the robotic approach was EndoWrist® (Intuitive Surgical, Inc., Sunnyvale, CA). EndoWrist® attachments for da Vinci are modeled after the human wrist, which allows full range of motion, facilitates hand-eye coordination similar to the human brain, and provides dual-channel (3-dimensional) vision necessary for the more dexterous maneuvers required in creating vascular anastomoses.[9]

Animal studies confirmed the benefits of the da Vinci Surgical System by showing that the time required to perform an anastomosis, clamp time, and total operative times were reduced.[5, 9, 10] Wisselink and colleagues pioneered robotic-assisted surgical repair of aortic occlusive disease, publishing reports of the first two cases performed in humans and demonstrating feasibility of the operation.[11] They went on to publish promising results with respect to the steep learning curve of the operation in the initial series of 17 patients, demonstrating a 50% reduction in clamp times for the later 9 patients as compared with the initial 8 patients.[12]

Stadler and colleagues, the group having the largest experience with robot-assisted laparoscopic aortoiliac procedures, recently published results from a series of 150 patients. They reported a 97.3% rate of successful completion, a 2.7% complication rate, and shortened anastomosis and clamp times (27 and 39 minutes, respectively) as compared to a purely laparoscopic approach.[13] Several groups in Europe have now demonstrated not only the feasibility of robot-assisted aortic reconstruction but also safety and shortened anastomosis times.[11-14] Our group has initiated an Investigational Device Exemption (IDE) trial, which we hope will pave the way for introducing robotic vascular surgery in the United States. We have developed and participated in a training program that begins with work on inanimate models, thereafter advancing to pig models and ultimately cadavers. We have shown the effectiveness of this training insofar as having a great degree of preparedness for the cadaver labs, where we were able to perform aortobifemoral bypasses within 2 hours. Inanimate and team training are probably the two elements that played the greatest role in our training paradigm. With the direct involvement and supervision of Dr. Petr Stadler, we plan to perform the first robot-assisted repair of aortic disease in humans in the United States later this year.

Table 1. Advantages and disadvantages of conventional laparoscopic surgery and robot-assisted surgery using a master/slave device (adapted from Lanfranco et al.).[14]

	Advantages	Disadvantages
Conventional laparoscopic surgery	• well-developed technology • affordable and ubiquitous • proven efficacy	• loss of touch sensation • loss of 3D visualization • compromised dexterity • limited degrees of motion • fulcrum effect • amplification of physiologic tremors
Robot-assisted surgery	• 3D visualization • improved dexterity • seven degrees of freedom • elimination of fulcrum effect • elimination of physiologic tremors • ability to scale motions • micro-anastomoses possible • telesurgery possible • ergonomic position	• absence of touch sensation • expensive • high start-up cost • may require extra staff to operate • new technology • unproven benefit • requires square footage (large)

Table 2. Advantages and disadvantages of robotic-assisted and conventional vascular catheterization.

Human strengths	Human limitations
• strong hand-eye coordination • dexterous • flexible and adaptable • can integrate extensive and diverse information • rudimentary haptic abilities • able to use qualitative information • good judgment • easy to instruct and debrief	• limited dexterity outside natural scale • prone to tremor and fatigue • limited geometric accuracy • limited ability to use quantitative information • limited sterility • susceptible to radiation and infection
Robot strengths	**Robot limitations**
• good geometric accuracy • stable and untiring • scale motion • can use diverse sensors in control • may be sterilized • resistant to radiation and infection	• no judgment • unable to use qualitative information • absence of haptic sensation • expensive • technology in flux • more studies needed

Conclusion

Robotic technology is set to revolutionize the manner with which cardiovascular surgery is performed. It has the potential to expand on current surgical treatment modalities in both endovascular and "open" vascular interventions. Some issues such as lack of haptics, tactile feedback, and interface in human-robotic interactions remain a significant safety concern and will add another level of safety when resolved. It remains to be seen whether or not the benefit of its usage overcomes its cost. Although feasibility has largely been shown, more prospective randomized trials evaluating efficacy and safety must be undertaken, and further research must evaluate cost effectiveness or a true benefit over conventional therapy for robotic surgery of the aorta to take full root.

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FENESTRATED AND BRANCHED ENDOGRAFTS

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Abstract

Thoracoabdominal aneurysm repair is undergoing a paradigm shift. The evolution of branched and fenestrated grafts has expanded the use of endografts to the visceral aorta and the arch of the aorta and broadened the spectrum of patients who can now be considered suitable for endografting.

In turn, these advances have lead to a decrease in mortality and morbidity at specialized centers and are propelling the development of a series of modular devices to facilitate wider dispersion of the technology. Preoperative case planning, advanced imaging, and technical experience are keys to successful outcomes. This review examines the current state of fenestrated and branched endografting and the workarounds that have been developed to increase the use of endovascular aortic repair (EVAR).

Current Clinical Issue

Open thoracoabdominal aortic repair is a significant physiological challenge to any patient and is associated with a sobering pattern of outcomes when examining the global experience. However, many individual high-volume centers do report good outcomes, with mortality and morbidity rates dependent on the health of the patient, the extent of the aneurysm, the skill of the surgeon, and the quality of perioperative care.[1] Treatment of many infrarenal and descending thoracic pathologies has shifted to endovascular grafting.[2-5] Endovascular repair of the descending thoracic aorta has demonstrated a marked reduction in the physiological challenge, a reduction in morbidity, and improved long-term outcomes.[6] Currently, the use of endovascular techniques within the aorta is limited by the need for sufficient proximal and distal landing zones and the need to maintain satisfactory flow to the major branches of the arch, the visceral segment, and the internal iliacs. Two endovascular systems have been developed and trialed to overcome these limitations: fenestrated stent-grafts and branched stent-grafts. Fenestrated grafts have holes in the fabric that are positioned adjacent to the aortic branch artery orifices. These fenestrations are secured to the aortic branch artery ostia by deploying smaller, covered stents (side branches) through the branched fenestrations and into the target arterial branches. Grafts may also contain scallops to accommodate branches without compromising the seal zone. Branched grafts, on the other hand, incorporate pre-attached limbs or cuffs targeted for the aortic branches and are directed and deployed into the branch, thus avoiding a junction and the need for a second covered stent. Preoperative case planning, advanced imaging, and technical experience are critical to successful outcomes. Availability of these two designs is limited to specialty centers, and this has spurred the development of other off-label techniques, including parallel stenting (chimney or snorkel techniques), dual bifurcated stent deployments, and on-table modification of commercially available stent grafts.

Abdominal and visceral aorta

Chimneys, Snorkels and Periscopes

The principle of parallel stenting is to place a covered stent in a target aortic branch and extend the stent into the lumen of the aorta at the level of the landing zone, such that the small covered stent is within a gutter of the graft and allows perfusion of the target branch directly from the oncoming flow of blood. This can be used for the main renal arteries, the superior mesenteric artery (SMA), and the celiac artery where there is an insufficient proximal landing zone (Figure 1). The snorkel technique allows the small covered stent to be placed in the gutter of the graft, but in this case the inflow is from the bounce back from the iliac or femoral bifurcation and is indirect flow. This can be used for the visceral vessels, the renals,[7] and the internal iliacs. The secret to a

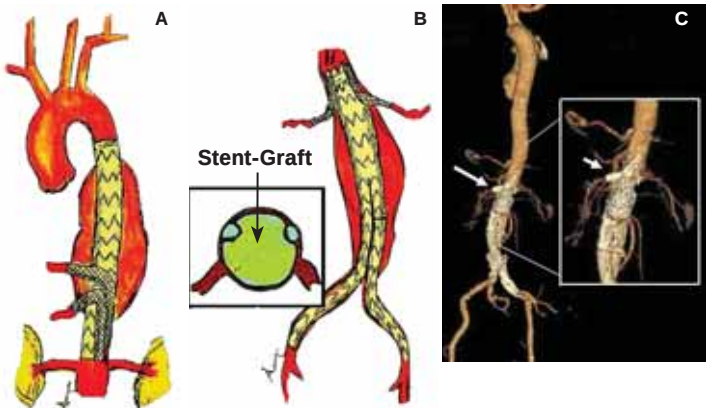

Figure 1. (A) The illustration demonstrates a double periscope technique to maintain patency in the celiac and SMA during thoracic endografting. (B) The illustration demonstrates a double chimney technique to maintain patency in both renals during juxtarenal endografting. (C) The computed tomography (CT) reconstruction shows a juxtarenal endograft with a single chimney graft to preserve the renal artery. The inset is a close-up of this area. Reprinted with permission from the Department of Cardiovascular Surgery, Methodist DeBakey Heart & Vascular Center.

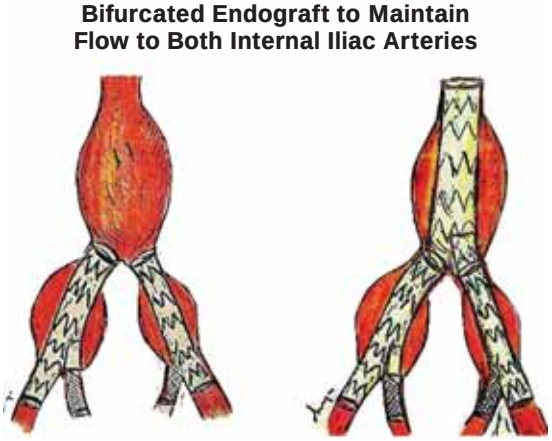

Figure 2. The illustration demonstrates a bilateral double endograft technique to maintain patency in the bilateral internal iliac arteries during abdominal aortic endografting. Reprinted with permission from the Department of Cardiovascular Surgery, Methodist DeBakey Heart & Vascular Center.

successful chimney or snorkel is to spiral the covered stent around the primary device and avoid a direct gutter inline with the blood flow, thus avoiding a type 1 endoleak. Case reports and small case series have suggested that this technique is effective in the short term.[8]

Dual Bifurcated Endograft Deployments

The principle of this technique is to deploy a second bifurcated device within the limb of a primary EVAR device to maintain flow to one or both internal iliac arteries (Figure 2). The limitation of this technique is the size requirement of the primary graft limb and the recipient vessel. It is also technically more difficult, and the risk-benefit ratio is unknown. Case reports suggest that this technique is technically achievable and effective in the short term.

Institutional Off-Label Modification of Commercial Endografts

The final technique is to modify an FDA-approved device on the back table with scallops or fenestrations such that there are one or more ostia or uncovered areas for the visceral and renal vessels in the abdomen. This represents an off-label modification of the device and carries significant legal and regulatory ramifications. A second technique that has emerged is in situ (in vivo) fenestration using a laser, cutting wire, or needle. This approach has been applied in the arch of the aorta with some early success.

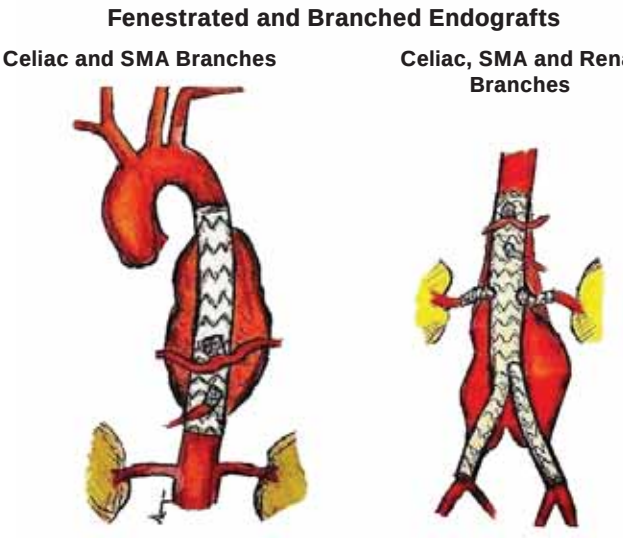

Figure 3. The illustration demonstrates (right) a branched thoracic graft used to maintain flow to the celiac and SMA vessels, and (left) a fenestrated graft used to treat a type IV abdominal aortic aneurysm and maintain flow to the visceral and renal vessels. Reprinted with permission from the Department of Cardiovascular Surgery, Methodist DeBakey Heart & Vascular Center.

Commercial Fenestrated and Branched Endografts

Several commercial companies are developing first- and second-generation fenestrated and branched endografts in both modular and non-modular designs (Figure 3). In selected patients, fenestrated and branched stents appear to be a safe and effective alternative to open surgery for juxtarenal and thoracoabdominal aneurysms, with low complication and mortality rates. Sixty percent of thoracoabdominal aortic aneurysms (TAAAs) would be suitable for branched/fenestrated stent grafting, but 40% are unsuitable due to adverse anatomy.[9]

In a prospective analysis of 119 patients undergoing implantation of a fenestrated endovascular device with short proximal necks (3–10 mm), Greenberg et al. demonstrated 100% technical success without the acute loss of any visceral arteries.[10, 11] The 30-day endoleak rate was 10% (all type II). In-stent stenoses occurred in 12 renal arteries and 1 SMA. The SMA and 6 renal arteries were treated, and 2 renal stenoses are awaiting treatment; 10 of 231 stented renal arteries occluded (3 prior to discharge). Similar results have been reported by other groups.[12-15] The clinical outcomes of the U.S. multicenter study of 30 patients are concordant with previous single-center studies.[16] The recent Association Universitaire de Recherche en Chirurgie Vasculaire study of 80 patients with fenestrated grafts in a total of 237 visceral vessels showed that 99% of the target vessels were patent at completion of the case.[17] Predischarge imaging identified 9 (11%) endoleaks: 3 were type I, 5 were type II, and 1 was type III. No aneurysms ruptured or required open conversion during the follow-up period. One patient had sac enlargement within the first year, associated with a persistent type II endoleak. In-stent stenoses or occlusion affected 4 renal arteries, of which 3 underwent reintervention. A standardized, off-the-shelf, multi-branched stent-graft is applicable in 88% of cases of TAAA.[18] Moderate degrees of cuff-to-artery misalignment had no effect on the efficacy of multi-branched stent-graft insertion.[19]

Preservation of pelvic circulation in high-risk patients treated for bilateral or unilateral common iliac aneurysms combined with or without abdominal aortic aneurysm is a small but significant clinical problem (Figure 4).[20] Nine series have reported the use of iliac branch devices (IBD) in a total of 196 patients.[21] Technical success was 85–100% with no aneurysm-related mortality. Iliac artery occlusion occurred in 24 of 196 patients. When comparing the first- with the second-generation IBD outcomes, cumulative patency rates at 2 years revealed no statistical difference. No endoleak, and particularly no IBD, modular side branch disconnection, late

Figure 4. The illustration demonstrates an iliac branched endograft employed to maintain flow to the internal iliac artery during iliac aneurysm endografting. Reprinted with permission from the Department of Cardiovascular Surgery, Methodist DeBakey Heart & Vascular Center.

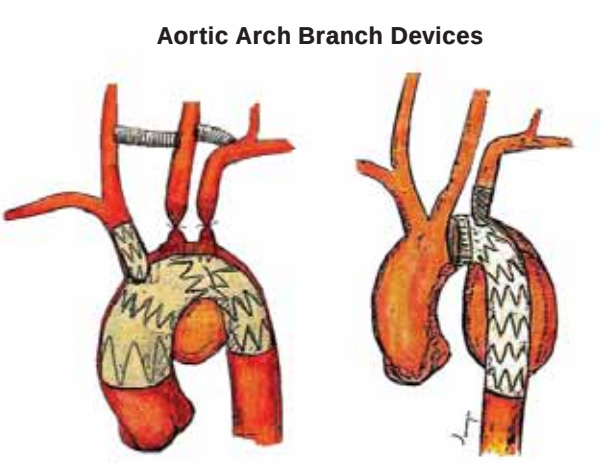

Figure 5. The illustration demonstrates a branched thoracic endograft with and without supra-aortic vessel bypass to treat an arch aneurysm.
Reprinted with permission from the Department of Cardiovascular Surgery, Methodist DeBakey Heart & Vascular Center.

rupture, or deaths have yet been encountered.[22] Other groups have reported similar findings. A recent study of 74 patients showed that there were no significant differences in failures of hypogastric side branch deployment (2 of 32) compared with hypogastric coiling (3 of 42). Reintervention rates were similar, but buttock claudication and impotence were more frequent after hypogastric exclusion.[23] Side branch endografting for iliac aneurysm may be considered a primary choice in younger, active patients with suitable anatomy.

Arch of the Aorta

The arch of the aorta poses unique problems for endografts, with both anatomic and hemodynamic conditions that are much different than those found in the descending visceral and infrarenal aorta. In general, debranching of the aorta has been used to facilitate placement of an endograft (Figure 5).[24-26] A recent report of 11 patients who underwent urgent thoracic endovascular aortic repair (TEVAR) combined with a chimney graft demonstrated immediate technical success and good short-term outcomes.[27] A second report described a double-barrel stent technique used to maintain aortic arch branch vessel patency during TEVAR as technically successful, with maintenance of branch vessel patency and absence of type I endoleak.[28] Single-branch thoracic aortic endografts have been developed as a simpler, safer, and more effective means of treating aortic dissections, with entry tears in proximity to the left subclavian artery. In 16 patients, branched endografts deployed to seal the entry tear of a Stanford type B dissection had a 94% technical success rate. No paraplegia or distal organ or limb ischemia was noted, nor were there any mortalities or complications. By 3 months post-treatment, symptoms had abated, thrombosis had formed in the false lumen, and the true lumen had resumed its normal diameter in 15 of the 16 stent-graft patients.[29]

Conclusion

Fenestrated and branched endografts offer us the bridge to the next frontier of aortic surgery. Once endovascular therapies can successfully negotiate the branches of the aorta and maintain patency, complete endografting of the aorta will be possible, and the need for open surgery will be significantly curtailed.

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WHEN TO REPLACE THE ASCENDING AORTA?

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Abstract

Ascending aortic aneurysm, while usually detected incidentally, is a serious condition that requires close monitoring and timely surgical follow up. Management of patients with thoracic aortic aneurysms (TAA) is optimally performed in a multidisciplinary manner that prevents or delays the need for surgical intervention. Patients with aneurysmal degeneration should be followed in a medical aortic clinic that manages all risk factors in an effort to delay or prevent the need for replacement of the ascending aorta. Symptoms, aortic size, growth rate, and genetic/familial factors are taken into account to develop a treatment plan specific to each patient that is in line with the most recent national guidelines. This article provides an evidence-based overview and key recommendations for intervention on the ascending aorta.

Introduction

Aneurysm of the ascending aorta is a disease that is usually detected in patients between the fifth and seventh decade of life[1] and is the most common thoracic aortic pathology treated in the operating room.[2] Awareness of the clinical significance of TAA disease has increased in recent years. As diagnostic tools have improved our ability to identify aortic pathology, surgical outcomes have also improved, making intervention a life-saving option in the majority of cases. Elective surgical repair of aortic root and ascending aortic aneurysms is a relatively low-risk procedure with mortality risk less than 5% at experienced centers. Isolated ascending aortic aneurysm repair should have an even lower perioperative risk (2–3%).

The thoracic aorta is divided into four parts: the aortic root (which includes the aortic valve annulus, the aortic valve cusps, and the sinuses of Valsalva); the ascending aorta (beginning at the sinotubular junction and extending to the brachiocephalic artery origin); the aortic arch (which begins at the origin of the brachiocephalic artery and is the origin of the head and neck arteries); and the descending aorta (which begins at the isthmus between the origin of the left subclavian artery and extends through the diaphragm into the abdomen). It has been recognized that the "normal aortic diameter" is influenced by a number of factors, including patient age, sex, body size, location of aortic measurement, method of measurement and the robustness and type of imaging methods used.

Aortic aneurysm histology is characterized by medial degeneration with disruption and loss of elastic fibers. These changes differ depending on the etiology of the aneurysm and are particularly pronounced in patients with connective tissue disorders.

Figure 1. MRI of ascending aortic aneurysm.

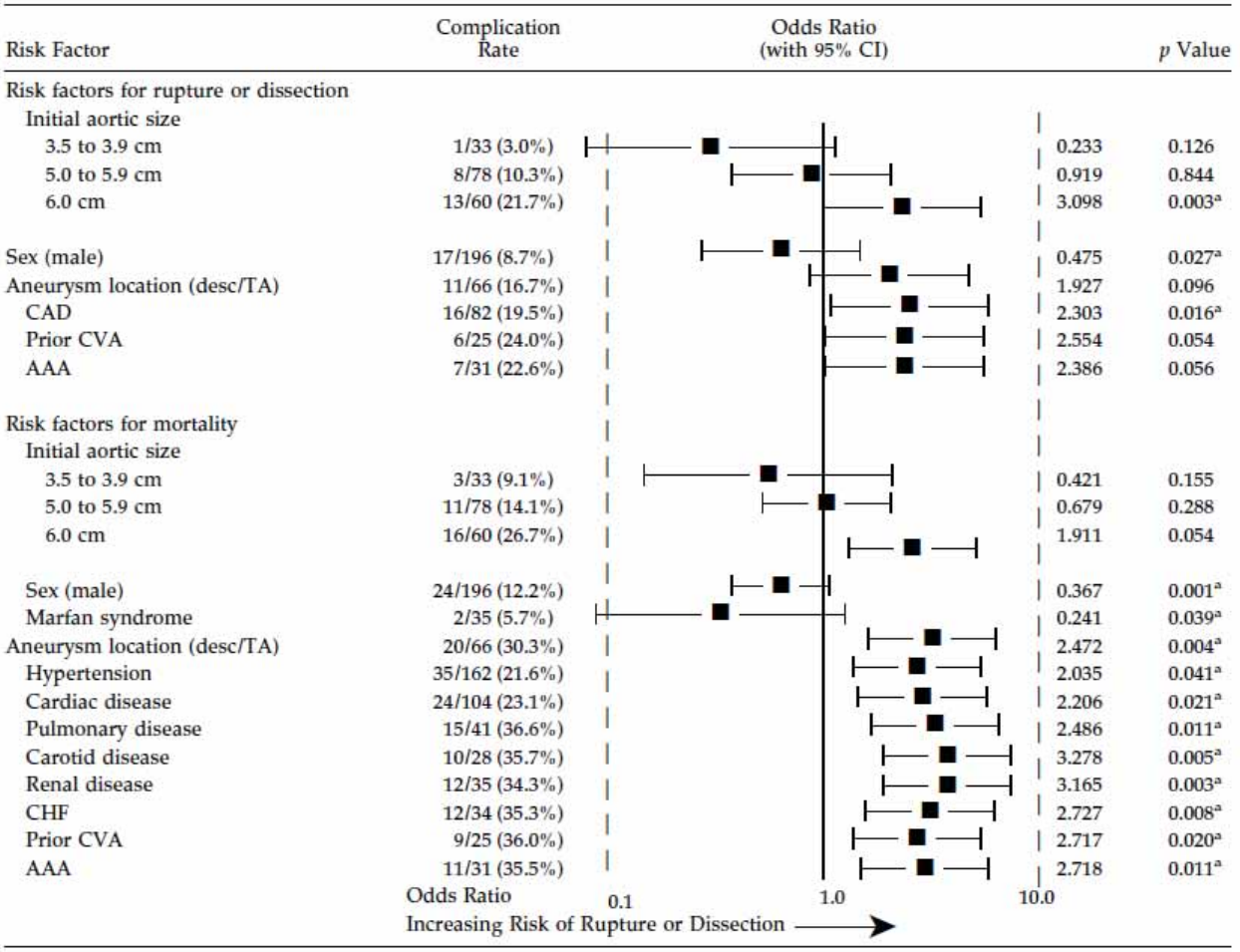
Ascending Aortic Aneurysms

Patients presenting with TAA are most commonly asymptomatic. Aneurysmal aorta is usually detected by an astute primary care physician or cardiologist during routine chest X-ray, computed tomography (CT) scan, or echocardiography (Figure 1).[3] Symptomatic patients often will complain of a sudden chest pain with no prior manifestation. In an ascending aortic aneurysm, the cusps of the aortic valve remain normal until dilatation of the sinotubular junction leads to poor coaptation of the aortic cusps, with a central jet of aortic valve insufficiency.[1]

Prophylactic intervention is the best treatment for patients with aortic aneurysms who meet guideline criteria for intervention. In an aortic emergency situation, surgery is mandatory but carries a significantly increased risk.[4] A supracoronary graft is a viable and simple option when the aneurysm is located just in the ascending aorta without involvement of the aortic root.

Thoracic Aorta	Range of Reported Mean (cm)	Reported SD (cm)	Assessment Method
Root (female)	3.50–3.72	0.38	CT
Root (male)	3.63–3.91	0.38	CT
Ascending (female, male)	2.86	NA	CXR
Mid-descending (female)	2.45–2.64	0.31	CT
Mid-descending (male)	2.39–2.98	0.31	CT
Diaphragmatic (female)	2.40–2.44	0.32	CT
Diaphragmatic (male)	2.43–2.69	0.27– 0.40	CT, arteriography

Table 1. Normal adult thoracic aortic diameters. CT: Computed Tomographic imaging; CXR: Chest X-ray; NA: Not Applicable.



[a] Statistically significant result. All of the following variables were analyzed: initial aortic size, sex, Marfan syndrome, aneurysm location, hypertension, cardiac disease, tobacco history, pulmonary disease, carotid disease, renal disease, coronary artery disease (CAD) congestive heart failure (CHE), prior cerebrovascular accident (CVA), and history of abdominal aortic aneurysm (AAA). Only results for initial aortic size and those where $p < 0.10$ are shown. Bars on graph indicate 95% confidence intervals (CI), odds ratios cannot be calculated when the incidence of disease is zero. desc/TA = descending or thoracoabdominal aorta.

Table 2. Univariate analysis of risk factor predictive of dissection rupture or of mortality. Reprinted with permission from Elsevier.[10]

Aortic Size and Growth Rate

Population studies have provided us with a reliable measure of normal adult thoracic aortic diameters (Table 1).[5] Studies of the natural history of ascending aortic aneurysms indicate that aneurysms exceeding 6 cm in maximum diameter are associated with a particularly high risk of complications. The yearly risk of rupture increased 11-fold for aneurysms 5.0–5.9 cm in diameter compared with those less than 4.0 cm in maximum size, and it increased 27-fold for those over 6.0 cm. The annual risk of the composite endpoint of dissection or death was 15.6% for aneurysms greater than 6.0 cm (Table 2). By the time a patient's ascending aortic size reaches 6 cm, that patient has incurred a cumulative 34% risk of rupture or dissection. An even stronger correlation with risk of rupture is seen when diameter is indexed to body surface area, with less than 2.75 cm/m^2 associated with low risk (4% per year), 2.75–4.25 cm/m^2 associated with intermediate risk (8% per year), and greater than 4.25 cm/m^2 associated with high risk (approximately 20% per year).[6] In a study by Clouse et al., the annual rupture risk was nil for aneurysms less than 4.0 cm in diameter, 16% for 4.0–5.9 cm, and 31% for 6.0 cm or larger.[7]

Intervention on an ascending aortic aneurysm is usually considered when the aortic diameter is ≥5.5 cm since size is the most powerful predictor of complications (Figure 2). Coady et al. suggested that the growth rate is 0.08 cm per year in a small aneurysm and 0.16 cm annually in larger aneurysms.[8] A faster growth rate was found in patients with bicuspid aortic valve (0.19 cm per year) when compared to tricuspid aortic valves (0.13 cm per year).[9]

In the interest of providing some margin of safety, most aortic surgeons would agree that intervention is indicated with an aortic diameter of 5.5 cm. However, for some patients at low surgical risk or for those with known connective tissue disorders, bicuspid aortic valve, or family history of aortic emergency, surgical intervention at an earlier stage (4.5–5.0 cm diameter) may be appropriate. These recommendations for surgical intervention were comprehensively addressed in the *2010 Guidelines for the Diagnosis and Management of Patients With Thoracic Aortic Disease*.[5]

Other Criteria for Intervention

Emergency and Mandatory Intervention

There are well-established situations where surgical intervention is mandatory without symptoms, increased aortic size, or any other criteria. These are urgent situations related to pathology of the ascending aorta: dissection, spontaneous rupture, intramural hematoma, and symptoms related to the aneurysm.[10] While some authors have advocated serial imaging and conservative management of intramural hematoma, these are largely considered a precursor to aortic dissection and should be treated aggressively.

Symptoms

A symptomatic TAA must be resected regardless of size. When a patient presents with symptoms related to a TAA rupture or dissection such as chest pain, the size of the aneurysm has no relevance in the decision to proceed with surgical intervention.[11] Substernal or interscapular pain may be a precursor or a sign of insidious rupture or dissection and should be taken seriously despite benign aortic imaging studies.[12] Patients can be harmed by not being offered surgery despite symptoms because aortic dimensions do not satisfy a particular size criterion. The size criteria are only for asymptomatic patients.

Genetic Factors

There is a strong link between certain genetic factors that predispose patients to aortic pathology — including aneurysmal degeneration, rupture, and/or dissection. There are several syndromic and nonsyndromic genetic conditions that are associated with the development of TAA and present with dissections at smaller diameters than usual. These conditions include Marfan syndrome, Loeys-Dietz syndrome, Turner syndrome, bicuspid aortic valve, and other genetic mutations (TGFBR1, TGFBR2, FBN1, ACTA2, COL3A1, MYH11). If such syndromes are suspected, referring the patient to a center for genetic sequencing and counseling is warranted and may influence therapy.

Some patients who do not meet the phenotypic criteria for Marfan syndrome but present with a characteristic degenerative aneurysm are termed forma frusta of Marfan syndrome. Patients with Marfan syndrome present with aneurysmal disease at an earlier age,[13] with unpredictable manifestations and a faster aneurysmal growth rate compared with the rest of the population.[10] In addition, the rate of aneurysm growth and the risk of dissection are markedly increased in this patient group,[10] as is the degree of elastin fiber disarray seen histologically. Such characteristics have required the aortic size recommendation for surgical intervention be lowered to 4.0–4.5 cm. Intervention should be lowered further to 4.0 cm when there is a rapid increase in aortic size. Similarly, when a patient with Marfan syndrome has a family history of dissection or presents with aortic regurgitation, intervention is warranted at 4.0 cm. Early referral for aortic root and ascending aortic replacement has the added potential benefit of increasing the likelihood that the aortic valve will be spared.

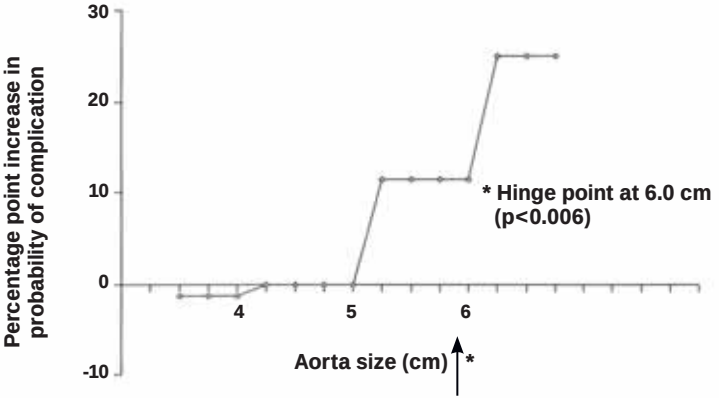


Figure 2. Regression analysis for the ascending aorta.
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Bicuspid Aortic Valve

Bicuspid aortic valve (BAV) is a relatively common heritable condition. Contrasting to Marfan syndrome where there is a genetic link, the genetics of BAV has not been clearly identified.[14] BAV is seen in 1–2% of the population[15] and is associated with aneurysmal aortic dilatation in 30–50%, even in patients with a normal functioning aortic valve.[16] BAV has been associated with genetic mutations involving NOTCH1 — a gene encoding a transmembrane signaling protein involved in the development and maintenance of the aorta. Another genetic mutation involving UFD1L has also been linked to BAV. The most common aortic aneurysm associated with BAV is located just above the sinotubular junction, ends below the innominate artery, and is often associated with aortic stenosis.

Patients with BAV are particularly susceptible to aortic dissection, and their risk of aortic emergencies is increased compared to other patients with degenerative aneurysmal disease. Similar to Marfan syndrome, BAV patients display advanced elastin fragmentation and increased matrix metalloproteinases (MMP) that may weaken the aortic wall and lead to a more unpredictable complication rate. In addition, increased rates of aortic dilatation following aortic valve replacement have been described in BAV patients compared to controls with trileaflet aortic valves.[17] For these reasons, many have advocated earlier aortic intervention for smaller-diameter aortic aneurysms (4.0–5.0 cm).[5, 9, 18]

Family History

If a patient has a family history of aortic dissections, aneurysms, or ruptures, they should be considered for surgery when the aortic size is in the range of 4.5–5.0 cm. Aortic imaging is recommended for first-degree relatives of patients with TAA and/or dissection to identify those with asymptomatic disease. If one or more first-degree relatives of a patient with known TAA and/or dissection are found to have thoracic aortic dilatation, aneurysm, or dissection, then imaging of second-degree relatives is advised.[5]

Medical Therapy for Aneurysms

While surgical resection remains the primary method for treating ascending aortic aneurysms, appropriate medical therapy has an important role in decreasing the rate of enlargement and possibly preventing emergency situations. Studies on Marfan syndrome patients proved that β-blockade will help to decrease the rate of extension.[19] Also, it has been recently shown that a fibrillin 1 (FBN1) gene mutation is associated with Marfan syndrome and leads to dysregulation of TGF-β signaling, which may explain the connective tissue abnormalities seen in these patients. These important findings have prompted investigations of the ability of Losartan, an angiotensin II receptor blocker with TGF-β antagonist properties, to reduce aortic complications in an animal model of Marfan syndrome. These studies may offer an important therapeutic option to improve the connective tissue abnormalities seen in patients.[20]

In general, a reduction in the rate of rise of left ventricular pressure (dP/dt) would reduce wall tension, degeneration, and possibly the risk of acute rupture. Aggressive blood pressure control with beta blockade and risk factor modification (smoking cessation, cholesterol lowering, etc.) are necessary treatment adjuncts to improve short- and long-term outcomes in patients with ascending aortic aneurysms. Such management protocols are optimally done in a multidisciplinary aortic clinic where aortic specialists follow patients in a regular fashion along with their primary care physician.

Summary

Optimal management of ascending aortic aneurysms is performed in an elective manner, usually in asymptomatic patients. Aggressive screening and comprehensive follow-up protocols will reduce the frequency of aortic emergency situations. The risk associated with elective open surgical repair in the current era is low (usually less than 5%), and long-term results are excellent. In summary, the following size criteria should be used for early surgical referral:

- Symptomatic TAA must be resected regardless of size. Symptoms may be due to pain, compression of adjacent organs, or significant aortic insufficiency.
- Asymptomatic patients with degenerative thoracic aneurysm, chronic aortic dissection, intramural hematoma, penetrating atherosclerotic ulcer, mycotic aneurysm, or pseudoaneurysm who are otherwise suitable candidates and for whom the ascending aorta or aortic sinus diameter is 5.5 cm or greater should be evaluated for surgical repair.
- Patients with BAV, genetically mediated disorders, or familial history of thoracic aortic disease should undergo elective operation at smaller diameters (4.0–5.0 cm depending on the condition) to avoid acute dissection or rupture.
- Patients with an aneurysm growth rate of more than 0.5 cm/year in an aorta that is less than 5.5 cm in diameter should be considered for operation.
- Patients undergoing cardiac surgery and who have an ascending aorta or aortic root of greater than 4.5 cm should be considered for concomitant repair of the aortic root or replacement of the ascending aorta.
- Aortic imaging is recommended for first-degree relatives of patients with TAA and/or dissection. If one or more first-degree relatives of a patient with known TAA and/or dissection are found to have TAA, then imaging of second-degree relatives is advised.

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AORTIC ARCH DEBRANCHING: ADVANCED AND HYBRID TECHNIQUES

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Abstract

Aortic arch procedures have traditionally involved complex surgery with increased risk of mortality and morbidity. Throughout the last decade, however, novel and safe surgical approaches aimed at debranching the great vessels with definitive aortic arch repair have been developed. Currently, hybrid arch procedures allow for open or minimally invasive aortic access to be complemented by endovascular stent-graft techniques, which may eliminate the need for prolonged hypothermic circulatory arrest and its associated complications. Hybrid thoracic aortic and arch repairs have become the preferred approach, with open procedures performed only if hybrid approaches are not possible for technical reasons. In the future, aortic pathology may also benefit from the development of branched and fenestrated endografts that would be deployed in a modular fashion. This article describes the rationale, procedural steps, and recent outcomes data of novel aortic arch procedures.

Introduction

Surgical management of patients with aortic pathology involving the arch and its branch vessels are traditionally challenging cases[1] leading to significant morbidity and mortality.[2, 3] Recently, our group at the Methodist DeBakey Heart & Vascular Center (MDHVC), among others, has developed novel surgical approaches aimed at debranching the great vessels with definitive aortic arch repair as the goal. These recent hybrid advances in aortic arch repair are proving to be a safer and viable alternative to conventional open repair. Reducing morbidity of open arch reconstruction and expanding the indications of this life-saving therapy to higher-risk patients have been the goal.

There is now extensive data, as seen in the published consensus guidelines,[4] that aortic aneurysms should be followed closely and repair contemplated when aortic size exceeds 4.5 cm in diameter, or even smaller in certain patient groups with connective tissue disorders or bicuspid aortic valves. The earliest repairs of thoracic aortic arch aneurysm (TAA) required extra-anatomic bypass to maintain cerebral blood flow, cardiopulmonary bypass (CPB) with resection and reconstruction of the thoracic aorta, and subsequent take down of the previously created extra-anatomic bypass. Griepp simplified the procedure by introducing profound hypothermia and circulatory arrest to allow cerebral protection during aortic arch resection.[5] Results from the Mayo group in a series of 95 open aortic arch repairs showed a mortality of 16.8% and a stroke rate of 9.5%.[6] The introduction of axillary cannulation and antegrade cerebral profusion decreased both mortality and stroke rate to 6%, still highlighting the complexity of this disease. For our increasingly elderly and frail patients, open aortic arch repair is associated with an in-hospital mortality of up to 20% and a stroke rate of up to 12%.[7, 8] For patients with significant comorbidities, open surgical repair may not be a viable option due to prohibitive estimated

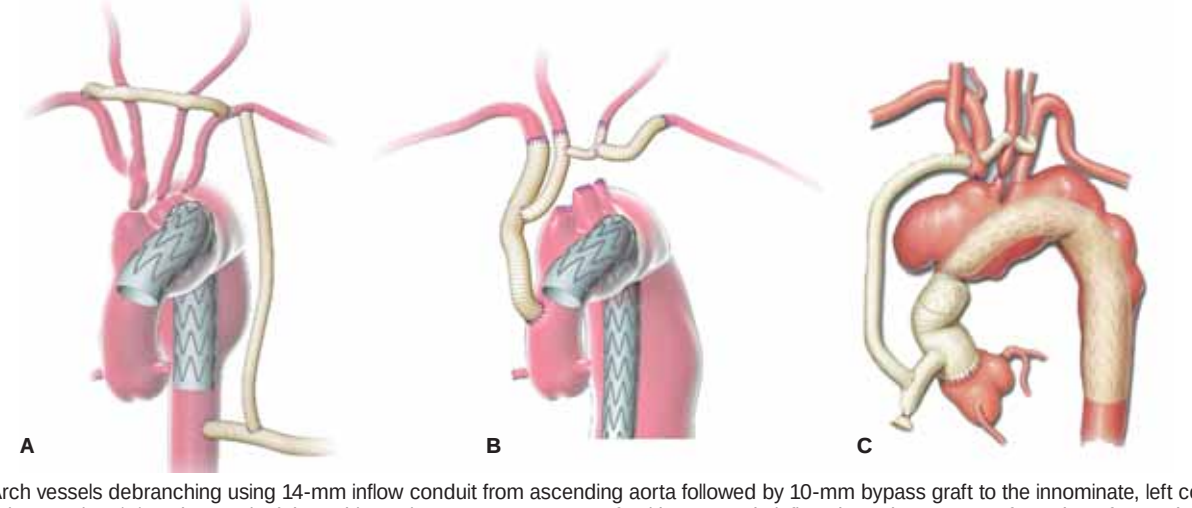

Figure 1. (A) Arch vessels debranching using 14-mm inflow conduit from ascending aorta followed by 10-mm bypass graft to the innominate, left common carotid, and left subclavian arteries. (B) Arch vessels debranching using 10-mm Dacron graft with retrograde inflow through 10-mm graft. Endograft was delivered using 14-mm conduit. (C) Arch vessels showing a 10-mm limb being sutured to a 14-mm trunk. The 10-mm limb is tunneled superiorly to revascularize the supra-aortic trunks, whereas the 14-mm trunk is used as the conduit for antegrade stent-graft placement. The 14-mm stump is oversewn after completion of the stent-graft deployment.[10-11]

mortality and permanent morbidity. Hybrid repair of TAA has been developed to address these high-risk patients without using circulatory arrest and CPB, which can be poorly tolerated.[9-12]

Rationale for Hybrid Approach

The traditional open repair of TAA is heavily influenced by the need for CPB, cardiac arrest, and profound hypothermia with circulatory arrest.[13-18] Thoracic aneurysms that require coverage of the innominate artery or the left subclavian artery for proper graft fixation are not amenable to standard, isolated endovascular techniques.[19-21] TAA can be isolated or involve the ascending and/or descending thoracic aorta, in which case they can be repaired by extra-anatomic bypass of the cerebral vessels and left subclavian when desired, along with antegrade or retrograde endograft deployment with CPB, cardiac arrest, or circulatory arrest, thereby removing many technical risk factors.[22-29] For TAA involving the ascending thoracic aorta, CPB and cardiac arrest are necessary for ascending aortic replacement, but debranching and endograft repair will eliminate the need for profound hypothermia and circulatory arrest (Figure 1).

Technical Issues With Arch Aneurysms

Endograft repair of aneurysms requires anatomically appropriate landing zones for the proper fixation and sealing of the aneurysm at its proximal and distal extent. It also requires that critically important arteries not be covered unless these are revascularized using some method, and it requires access to deliver the graft for deployment. The thoracic aorta has been divided into five zones. Zone 0 includes the ascending aorta to just beyond the origin of the innominate artery. Zone 1 extends from the end of zone 0 to just beyond the origin of the left carotid artery. Zone 2 begins at the end of zone 1 and extends to the origin of the left subclavian artery. Zone 3 represents the proximal descending thoracic aorta from the left subclavian to the mid-descending thoracic aorta. Zone 4 comprises the rest of the descending thoracic aorta (Figure 2). Endograft coverage of zones 0 or 1 will cover vital cerebral blood flow from the innominate and/or left carotid arteries and cannot be done without establishing an extra-anatomic route of blood flow. Endografts that cover zone 2 will cover the left subclavian artery. Although it can be covered without an untoward effect in some patients, there is a belief that revascularization of the left subclavian in these circumstances may improve outcomes.[30] Even when appropriately revascularized, landing an endograft in the aortic arch may pose a technical challenge due to the curvature of the arch and potential "bird beaking" of the graft.[31] Each of the techniques used to allow endograft coverage of the aortic arch are extending the proximal "branchless" section of the descending thoracic aorta to allow an appropriate proximal landing zone. The final technical consideration is the feasibility of delivering the stent graft to the site of deployment in an antegrade or a retrograde fashion.

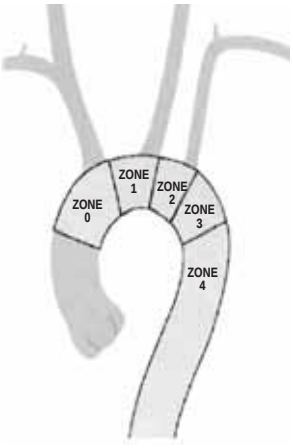

Figure 2. Ishimaru arch map according to the different landing zones.[42]

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Hybrid Aortic Arch Techniques

The technique used for aortic arch repair is largely dependent on the patient's anatomy and the nature of the pathology (dissection vs. aneurysm).[32-35] Aneurysms involving the ascending aorta usually mandate use of CPB and hypothermic circulatory arrest during the open distal anastomosis. However, using the novel hybrid and debranching approaches described below, the time required and morbidity are diminished significantly. Arch aneurysms not involving the ascending aorta can typically be repaired using an arch debranching procedure without CPB, with endovascular repair across the arch and proximal descending thoracic aorta using a landing zone within the normal ascending aorta. The following describes the surgical approach adopted at MDHVC based on the affected anatomy.

Ascending Aortic Involvement

If the ascending aorta is involved and must be replaced, we use a standard median sternotomy for surgical access. CPB, cardioplegic cardiac arrest, and standard distal ascending aortic cross clamp technique are used for ascending aortic replacement. The arch aneurysm is left in place, thus avoiding profound hypothermia and circulatory arrest. A 12-mm graft is attached to the proximal ascending graft, and its separate side arms are constructed and attached end-to-end to the innominate, left carotid, and left subclavian arteries. This allows the proximal stent graft to land in the distal ascending graft to provide a safe seal (Figure 3).

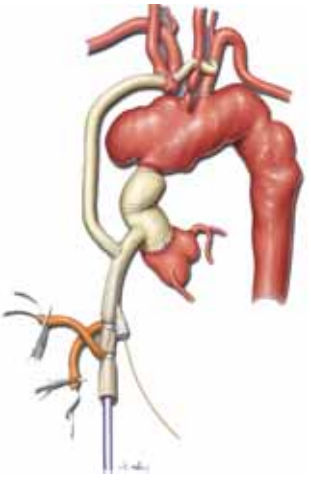

Figure 3. Extra-anatomic debranching of aortic arch vessels with replacement of ascending aorta and antegrade TEVAR via Dacron side branch.[12]

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Ascending Aorta Not Involved but Innominate Artery Involved

When the ascending aorta does not need to be replaced, the entire procedure can be done without stopping the heart. Surgical access may be via a standard median sternotomy or mini-right anterior thoracotomy and cervical incisions. Median sternotomy allows attachment of a 12-mm graft to the proximal ascending aorta using a side-biting clamp. Care must be taken to lower the systemic blood pressure to avoid dissection or premature clamp release and to attach this graft far enough proximally to allow an adequate landing zone in the ascending aorta. Because this graft is used for antegrade stent graft deployment, we bolster the anastomosis with a pledgeted suture at the toe and heel of the ascending aortic attachment to prevent disruption. Side arm grafts are individually constructed and attached to the innominate, left carotid, and left subclavian arteries. After this step, it is easier to attach the left subclavian graft previously sewn to the left subclavian in an end-to-side manner to the existing left carotid graft. Once the arch has been completely reconstructed in this extra-anatomic fashion, the stent graft is deployed in an antegrade manner through the 12-mm graft attached to the ascending aorta. The distal extent of the stent graft depends on the distal extent of the aneurysm and achieving an adequate distal landing zone. This approach for debranching the entire aortic arch

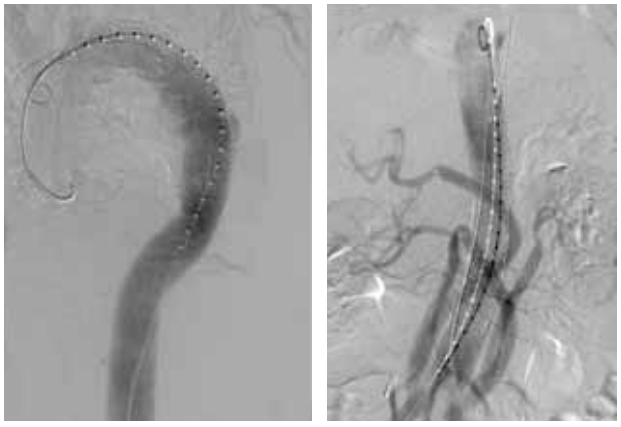

Figure 4. Completion angiography after stent grafting in a patient who underwent arch and abdominal debranching shows the patency of bypass grafts and the exclusion on the aneurysm.[10]
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and celiac and superior mesenteric arteries allows a distal graft landing just above the renal arteries (Figure 4).

Innominate Not Involved

If the endograft has a proximal landing zone that must cover the left carotid and left subclavian arteries, cervical incisions only for right carotid to left carotid bypass and a left carotid subclavian bypass can be carried out with retrograde endograft deployment.

Minimally Invasive Arch Repair

The alternative to a median sternotomy is a small anterior right thoracotomy, as described for minimally invasive access for aortic valve replacement and cervical incisions. Recently, we reported a minimally invasive, non-sternotomy approach to debranching the aortic arch that may prove to be useful in frail and debilitated patients for whom reducing surgical trauma would be of significant benefit.[36] For this approach, a small incision at the lower sternocleidomastoid muscle on each side allows access to the right and left common carotid arteries. A small left supraclavicular incision exposes the left subclavian artery. A 10-mm Dacron graft is pre-sewn onto a 12-mm graft on the back table and tailored to fit the proximal ascending aorta; the 12-mm graft is then attached to the proximal ascending aorta. At the completion of this anastomosis, clamps are placed on the 12-mm and the 10-mm grafts, and the side-biting clamp is removed. Pledgeted sutures are again placed at the toe and heel of the ascending attachment, and the anastomosis is carefully inspected for any bleeding.

At this point the 10-mm graft is within the chest, next to the ascending aorta, and would not be an easy anastomosis to perform if not already done. The 10-mm graft must then be passed through the sternal outlet into the right cervical incision for anastomosis to the right common carotid artery; exit the sternum in the midline and against the posterior table of the sternum to avoid injury to the innominate vein. An 8-mm graft is then attached to this 10-mm graft and passed anterior to the trachea, making a loop low behind the upper sternum; it is then attached to the left common carotid artery leaving the trachea uncovered by graft. A standard left carotid subclavian bypass completes the arch debranching. The proximal left carotid and innominate arteries are ligated to prevent type II endoleaks. The left subclavian is closed proximally with a coil or plug via the left arm to prevent endoleak while preserving the vertebral artery. Antegrade deployment of the stent graft can then be carried out via the original 12-mm graft attached to the ascending aorta.

We treat most aortic arch hybrid cases as a single stage using antegrade deployment, whereas visceral debranching and stent grafting for thoracoabdominal aortic aneurysm are performed as a staged procedure with retrograde deployment (Figure 5).[37]

Results

Hybrid arch procedures provide a safe and viable alternative to traditional open surgical repair. In general, hybrid approaches have a lower mortality and morbidity for high-risk older patients. Recently, such approaches have extended hybrid repair indications for complex arch pathology once thought to be prohibitively high risk for open arch surgical repair.

At our institution, hybrid thoracic aortic and arch repairs have become the preferred approach, with open procedures performed only if hybrid approaches are not possible for technical reasons.

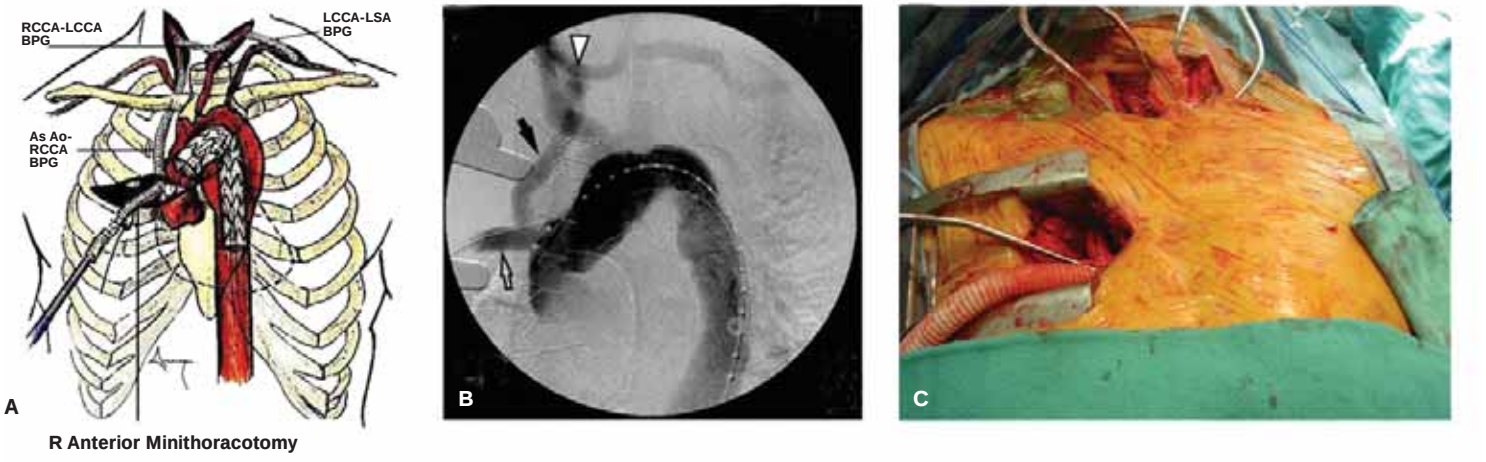

Figure 5. (A) Schematic drawing showing the procedure. Via a 5-cm incision at the third intercostal space to access the ascending arch, a 12-mm–10-mm bifurcated hemashield Dacron graft is created. A partial occluding clamp is used on the ascending aorta to attach the 10-mm arm of the bifurcated 10/12-mm graft to the right common carotid or innominate artery. Remaining arch vessels are bypassed through carotid-carotid and left carotid-subclavian bypass. Antegrade stenting of the aortic arch is carried out through the RAM via the remaining 12-mm limb.[36] (B) Intraoperative angiogram showed the ascending aorta to right common carotid artery (RCCA) bypass graft (black arrow), RCCA to LCCA bypass graft (white arrow), and the stent graft deployment via a 12-mm limb (hollow arrow) through the anterior minithoracotomy and complete exclusion of the aneurysmal sac.[36] (C) Intraoperative photograph.[10]
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Complication	Hybrid Aneurysm (n=33)	Hybrid Dissection (n=7)	P value
MI	6%	0%	1
Respiratory Failure	33%	20%	0.65
Renal failure	15%	20%	1
GI	24%	0%	0.38
SCI	15%	0%	0.56
CVA/TIA	18%	40%	0.61
Death (30 days)	24%	0%	0.31
Composite endpoint	13%	0%	0.07

Table 1. Comparison of the outcome of aneurysm and dissection (%).[10] *CVA*: cerebrovascular accident; *GI*: gastrointestinal; *MI*: myocardial infarction; *SCI*: spinal cord ischemia; *TIA*: transient ischemic attack. Composite endpoint is the combined death and permanent paraplegia rate at 30 days.

In our experience with hybrid aortic repair in patients who were denied open surgery due to preoperative comorbidities and low physiologic reserve, 55% were symptomatic on presentation and 83% were done emergently; 76% underwent debranching of the aortic arch, 17% of the visceral vessels, and 7% required both. Primary technical success was achieved in all cases, and of these, 43% were staged. The 30-day mortality was 5%. Myocardial infarction developed in 6% and respiratory failure in 33% (Table 1). These hybrid approaches, while initially performed mostly for very sick or emergent cases, proved the technical feasibility of the procedure given the medical and anatomical complexity of these patients, with encouraging results.[9-12]

Milewski and coworkers compared a hybrid arch repair cohort with an open aortic arch repair cohort and found a trend for lower incidence of neurologic deficit of 4% compared to 9% per group, while the short-term/in-hospital mortality rate was 11% and 16%, respectively. The only statistically significant difference was the mortality rate between age groups and not among surgical approaches: the older patients (over 75 years old) had a higher mortality rate of 36%.[38]

In another series reported by Hughes and colleagues, 28 patients underwent hybrid arch repair with 30-day in-hospital rates of death, stroke, and permanent paraplegia/paresis of 0%, 0%, and 3.6%, respectively.[39] At a mean follow-up of 14 ± 11 months, there were no late aortic-related events. Two patients (7%) required secondary endovascular reintervention for a type 1 endovascular leak. No patient has shown a type 1 or 3 endovascular leak at latest follow-up.[39-41] Similarly, Canaud reported a 6.8% risk of stroke with an actuarial survival of 70% at a mean follow-up of 29.9 months.[42]

Regardless of the configuration used, hybrid approaches to arch repair are achieving similar or better short- and long-term outcomes compared to the open arch replacement procedures in most reported series.

Conclusion

Regardless of the approach selected, aortic arch pathology remains a challenging surgical undertaking. Open aortic arch repair for aortic arch aneurysm can be carried out at reasonable but not insignificant risk in appropriate patients. The hybrid endovascular stent graft approach has been developed in an attempt to decrease the mortality and morbidity of open arch repair and to allow extension of life-saving therapy to high-risk patients who may not be reasonable candidates for open repair.

In the future, aortic pathology may also benefit from the development of branched and fenestrated endografts that would be deployed in a modular fashion.[43] This technology would allow us to potentially debranch the aortic arch from the inside, further minimizing the surgical insult.

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B. Ramlawi, M.D., M.M.Sc.

ADVANCED IN AORTIC ROOT SURGERY

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Abstract

The aortic root is often affected by aneurysmal degeneration of the ascending aorta or dissection. It is important for the clinician to be familiar with current guidelines and recommendations for detection, monitoring, and intervention of the aneurysmal aortic root. Timely surgical referral to an experienced aortic center allows for close monitoring and possible intervention that may preserve the aortic valve in appropriate cases and avoid disastrous complications such as aortic dissection, rupture, or death. Patients with bicuspid aortic valve syndrome or connective tissue disorders (e.g., Marfan syndrome) are particularly at risk and should be followed aggressively. Whenever possible, attempts should be made to preserve or repair the aortic valve using valve-sparing aortic root replacement techniques. This article provides an overview of recent advances in management of the aortic root, including guidelines for surgical intervention, technical procedures, and outcomes.

Introduction and Anatomy

The anatomic space between the aorta and the left ventricle is called the aortic root and includes, from top to bottom, the sinotubular junction (which separates the aortic root from the ascending aorta), aortic sinuses, aortic cusps, and aortic annulus (aorto-ventricular junction).[1] The three aortic cusps have a crescent shape and often are of different sizes, but the length of the base of a cusp is consistently 1.5 times longer than the length of its free margin. Therefore, a large cusp will have a proportionally longer annulus, free margin, intercommissural distance, and a larger sinus of Valsalva. The non-coronary and right cusps and sinuses of Valsalva are usually larger than the left cusp and sinus. The aortic annulus is a tridimensional structure that, for practical purposes, is measured as the maximal distance along a single horizontal plane at the level of its nadir. In childhood, the diameter of the aortic annulus is approximately 20% larger than the sinotubular junction (STJ). As the elastic fibers of the arterial wall change with increasing age, the STJ dilates and becomes equal in diameter to the aortic annulus and in later life, especially in cases of aortic stenosis, is usually 10% larger than the annulus.

Aortic Root Pathology and Surgical Indications

Ascending aortic aneurysms often cause dilatation of the STJ with consequent aortic insufficiency due to lack of coaptation of the cusps (Figure 1). One or more aortic sinuses may also become dilated in patients with ascending aortic aneurysm, but the aortic annulus often remains unchanged. Patients with ascending aortic aneurysms and aortic insufficiency are usually in their sixth or seventh decade of life. If the aortic cusps are normal or minimally elongated along their free margins, it is possible to reconstruct the aortic root, repair the cusps if necessary, and re-establish aortic valve competence.

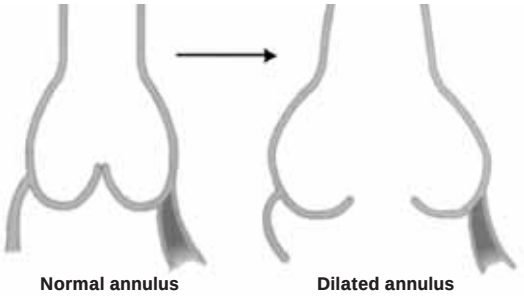

Figure 1. Aortic root dilatation as cause of aortic insufficiency.[29]
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The mechanism of aortic insufficiency in patients with aortic root aneurysm is more complicated. Dilatation of the aortic root often starts at the level of the sinuses of Valsalva (Figure 1). As wall tension increases, the STJ begins to dilate. The aortic annulus may also dilate in these patients, further complicating the mechanism of aortic insufficiency by widening the fibrous subcommissural triangles of the non-coronary aortic cusp. Depending on the rate of expansion of the aneurysm, the cusps may or may not become elongated and overstretched and develop stress fenestrations along the commissural areas, rendering them unsuitable for aortic valve repair.

Figure 2. Annuloaortic ectasia as commonly encountered in Marfan syndrome.

The most frequent indications for aortic root replacement are primarily aortic root aneurysm, aortic dissection, or endocarditis involving the root.[2] Annuloaortic ectasia is a form of aneurysmal dilatation of the proximal ascending aorta and aortic annulus that is often associated with Marfan syndrome (Figure 2). It can also be a complication due to tertiary syphilis. Aortic root aneurysm and aortic dissections, if not treated, can lead to significant morbidity and mortality. Timely surgical

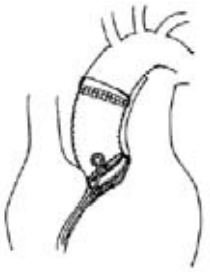

Figure 3. Modified Bentall procedure for aortic root replacement.[30]
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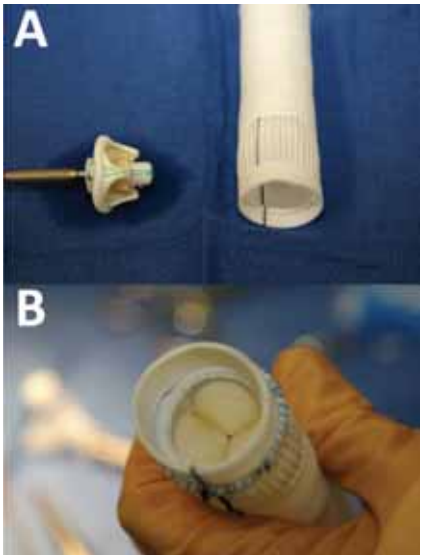

Figure 4. Construction of biologic valve conduit using Gelweave Valsalva™ graft.

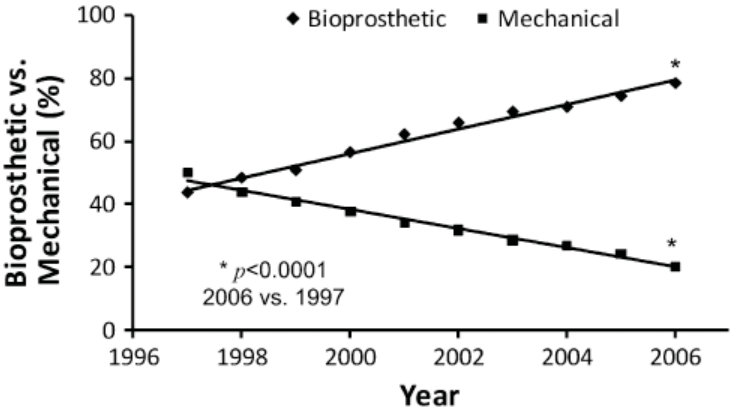

Figure 5. Society of Thoracic Surgeons trend for bioprosthetic vs. mechanical valve implantation.[31] Reprinted with permission from Elsevier/The American Association for Thoracic Surgery.

repair of the diseased root usually takes the form of aortic root replacement with or without a valve-sparing approach. This could be achieved as a composite prosthesis or as separate elements. An ascending aortic aneurysm can be simply fixed by a tube graft replacement of the ascending aorta with satisfactory results.[3] Modified Bentall procedures eliminate the entire aortic root tissue and, with this, the risk of future annular dilatation and associated aortic insufficiency or recurrent aneurysm (Figure 3).[3]

Acute aortic dissection involving the ascending aorta requires emergent surgery. The minimum operation required is to replace the ascending aorta to prevent extension of the tear into the root, which could cause aortic or coronary insufficiency; to prevent rupture of the ascending aorta; and to minimize the likelihood of extension of the tear into the head vessels, causing cerebral ischemia. While replacing the ascending aorta achieves the above goals, this operation may not stabilize the aortic root. In cases where the root is dilated or involved in the dissection, replacement of the aortic sinuses is indicated.

Most patients with aortic root aneurysms are asymptomatic and have no physical signs if they have no aortic insufficiency. Some patients may complain of vague chest pain. Severe chest pain is suggestive of rapid expansion or intimal tear with dissection. Echocardiography establishes the diagnosis and provides information regarding the aortic cusps. Computed tomography scan and magnetic resonance imaging of the chest are also diagnostic and useful in providing information regarding the entire thoracic aorta. Surgery is recommended when the transverse diameter of the aortic root exceeds 55 mm.[4-6] If the aortic valve can be preserved, surgery should be considered when the transverse diameter of the aortic root reaches 45–50 mm, if there is aortic size growth >5 mm per year, or if the patient is symptomatic.[7-8] This size criterion is also used for patients with genetic abnormalities, those with a family history of acute aortic dissection, and in patients with a bicuspid aortic valve.[9]

Operative Strategies

Where the aortic root is extensively destroyed by dissection or endocarditis, or if there is aneurysmal enlargement of the root, then aortic root repair is warranted for improved short- and long-term outcomes.[10] The most common form of aortic root repair is the modified Bentall operation involving replacement of the aortic valve, sinuses of Valsalva, and the ascending aorta with reimplantation of the coronary ostia into the new Dacron aortic graft. This can be performed by using a number of options, such as composite valve-graft conduits (mechanical or biologic), aortic root allografts or xenografts, and valve-sparing root replacements. Axillary artery cannulation, through an 8 mm end-to-side graft, has proven to be an excellent tool for any aortic procedure by allowing the surgeon to maintain all options for repair of the root, ascending, and arch. It is currently our preferred approach.

Modified Bentall Procedures: Composite Valve-Graft Conduit (Mechanical and Tissue)

The most popular prosthesis for aortic root replacement is a pre-manufactured composite mechanical valve and Dacron tube graft conduit. Alternatively, a composite biologic valve-conduit can be constructed at the time of operation (Figure 4). This can be easily performed on the back table by selecting a graft 5 mm larger than the stented valve (e.g., a #23 stented porcine/pericardial valve sutured into a #28 mm Dacron tube graft). These biologic valve conduits are often used in older patients or others who would like to avoid the need for anticoagulation. As demonstrated by Society of Thoracic Surgeons data (Figure 5), there has been a significant decline in the number of mechanical valves used as biologic valve durability has improved over the past 10–15 years. Many active patients in their 40s and 50s or younger are requesting biologic valves to avoid the potential hazards of anticoagulation and the associated lifestyle modification that would be required with mechanical valves.[11]

Some of the more recent advances involving aortic root replacement using composite biologic valve conduits include forward planning for subsequent operations or interventions, particularly on patients presenting to surgery at a young age. In particular, as the use of these composite biologic valve conduits become more popular in younger patients, minor technical adjustments at the time of initial aortic root repair may lead to a significantly simpler subsequent operation. One of these modifications is use of the De Paulis Gelweave Valsalva™ graft conduit (Terumo Cardiovascular Systems Corporation, Ann Arbor, MI) that allows for a more geometric reconstruction of the root that

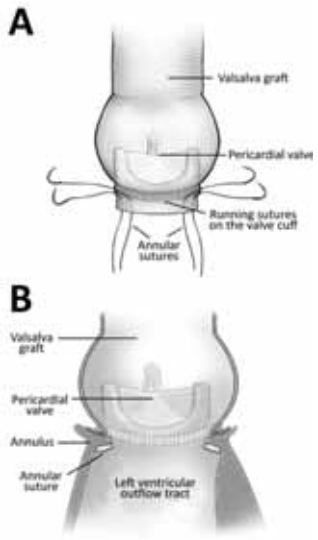

Figure 6. (A) Supra-annular composite biologic valve conduit using the Valsalva graft. (B) Composite graft seated above the annulus using proximal skirt of the graft below valve attachment.[13]

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Figure 7. (A) Mechanical valve conduit (On-X Ascending Aortic Prosthesis). (B) Freestyle® xenograft aortic root.

(A) Image courtesy of On-X Life Technologies, Incorporated™. Used with permission. (B) Copyright Medtronic, Inc. Used with permission

mimics the billowing of sinuses of Valsalva. This conduit has several potential short- and long-term advantages, including decreasing the need for significant coronary ostia mobilization when reimplanting the coronary buttons into the graft — thus rendering this portion of the procedure easier, especially during a redo operation, endocarditis or aortic dissection where adhesions are problematic. This graft may also confer a long-term benefit as it allows for a more physiologic closure of aortic valve cusps and coronary perfusion, which may improve long-term durability of the biologic valve. This also has the potential added benefit of displacing the coronaries away from the aortic annulus, rendering future transcatheter aortic valve interventions (TAVI) less prone to coronary obstruction.

Supra-annular placement of the biologic valve within the conduit can have significant potential benefits. This can be performed by implanting the biologic valve 2–3 mm into the Valsalva graft (using a running 4-0 polypropylene suture) and allowing a 2- to 3-ring skirt at the proximal end of the graft. Non-everting pledgeted 2-0 polyester sutures are placed on the aortic annulus. Those sutures are subsequently passed through the Valsalva graft collar below the sewing cuff of the prosthetic valve (Figure 6). Theoretically, the Valsalva conduit reduces the tension of coronary buttons compared with the conventional tube graft. It also creates more space between the biologic valve struts and coronary buttons and may decrease the risk of coronary button complications.

Passing the annular sutures through the graft collar is the major advantage of this technique. Because the valve is seated above the aortic annulus, this technique allows the surgeon to place a larger-sized valve than the annulus, which is especially helpful for patients with a small aortic annulus. The effective orifice area becomes the left ventricular outflow tract, not the internal diameter of the prosthetic valve. The valve position is slightly higher compared with a technique passing the annular sutures through the valve cuff; however, the coronary buttons do not need to be placed higher because the Valsalva graft creates space between the valve and the graft wall. These techniques may allow the surgeon to reoperate on the valve only, without compromising the root structure if the prosthetic valve has become degenerated.[12] The use of the Valsalva graft allows for a larger space in the root to facilitate both resection of the old valve and replacement of a new prosthesis. Another potential advantage is possible future deployment of percutaneous aortic valve prostheses inside such a biologic valve conduit. Having a larger valve orifice allows a larger-sized percutaneous valve to be deployed, while displacing the coronary buttons away from the biologic valve may decrease the risk of coronary occlusion complications. Mortality rates in the literature for such operations have been reported as 2–7%.[13, 14] Recently, the Columbia group described a technique for aortic root replacement using the 3F stentless equine biologic aortic valve (ATS Medical, Inc., Minneapolis, MN) within the Gelweave Valsalva graft with excellent short-term results and very low mean transvalvular gradients (4.0 ± 1.7 mmHg).

Modified Bentall Procedures: Allografts and Xenografts

Alternatives to biologic or mechanical valve conduits include xenograft (porcine) aortic root replacements (e.g., Medtronic Freestyle®) and cadaveric aortic root allografts (e.g., Cryolife CryoValve® Aortic Valve Allograft) (Figure 7). The stentless design of both these grafts offers a potential for improved hemodynamics over stented tissue valves. Both of these options are used in patients who want to avoid lifelong anticoagulation and have low rates of infection. Allografts are offered as a complete aortic graft from the aortic valve to the arch and often include the anterior leaflet of the mitral valve. This provides the surgeon with flexibility to replace the ascending aorta and use of the anterior leaflet for reconstruction of any defects within the root, as can be seen in endocarditis. However, allografts are occasionally limited in availability, tend to be more difficult to implant, and have suboptimal durability secondary to accelerated calcification. Aortic allograft implantation is currently performed less frequently and is often reserved for patients with aortic root abscess. Porcine aortic roots are treated with glutaraldehyde fixation at low pressures and anti-calcification treatment to maintain structure and potentially improve durability. A recent study by Yacoub and colleagues compared the Freestyle xenograft to allograft root replacement in a randomized fashion. Late survival was similar after homograft versus Freestyle root replacement. However, Freestyle aortic root replacement was associated with significantly less progressive aortic valve dysfunction and a lower need for reoperations.[15]

Aortic Valve-Sparing Root Procedures

Aortic valve-sparing procedures involve replacement of the aortic root and proximal ascending aorta while leaving the native aortic valve in situ. Candidates for this operation are patients who develop aortic root pathology, with or without aortic insufficiency, but with normal aortic valve cusps. Patients with ascending aortic aneurysm and aortic insufficiency often have a dilated STJ but normal or minimally dilated aortic sinuses and normal cusps, and all that is needed to restore aortic valve competence is to reduce the diameter of the STJ at the time the ascending aorta is replaced.[16, 17]

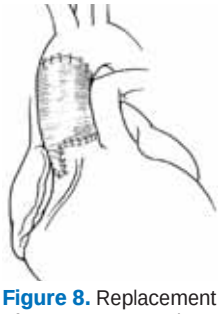

Figure 8. Replacement of one or more aortic sinuses is feasible when necessary.[32]

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In an important paper by Lawrie and DeBakey, a durable repair was presented for a large number of patients with preserved valve function and dissecting aneurysm by placing a tube replacement and conserving the aortic root, excluding patients with Marfan syndrome.[3] Durability of this repair would be increased by performing AV resuspension at the commissural level. Occasionally in patients with severe aortic

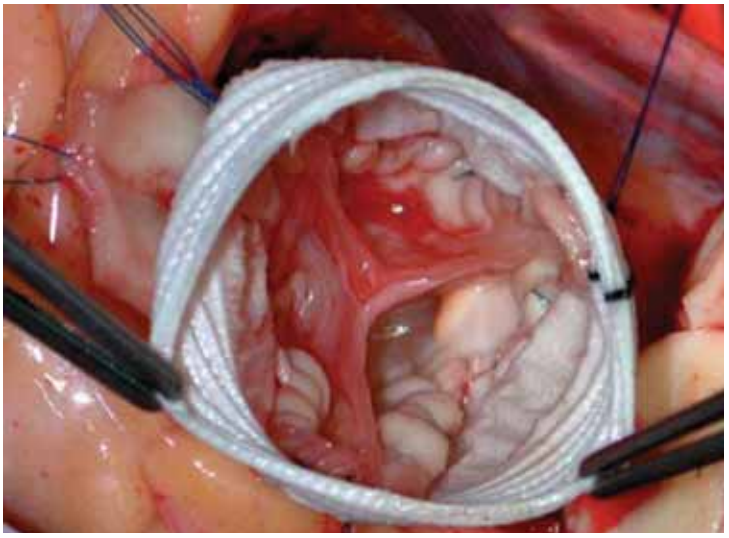

Figure 9. Valve-sparing root replacement using the reimplantation technique.

insufficiency, one or more cusps remain nearly normal because of the asymmetric nature of aortic root dilatation. If the non-coronary aortic sinus is dilated or altered by aortic dissection, a neosinus can be created by tailoring the graft with a tongue of tissue that is sutured directly to the aortic annulus (Figure 8).

There are two types of aortic valve-sparing operations for patients with aortic root aneurysm: remodeling of the aortic root (described by Yacoub[18]) and reimplantation of the aortic valve (described by David[4]) (Figure 9). In the remodeling procedure, the three aortic sinuses are excised, and a properly tailored tubular Dacron graft of diameter equal to the estimated diameter of the STJ is sutured to the aortic annulus. The coronary arteries are then reimplanted into their respective sinuses. Remodeling of the aortic root replaces all three aortic sinuses but does not stabilize the aortic valve annulus and therefore leads to delayed dilatation of the aortic annulus.

Remodeling of the aortic root may be inappropriate for patients with Marfan syndrome or annuloaortic ectasia because the annulus may continue to dilate and cause late aortic insufficiency[7, 19-22] The aortic valve reimplantation technique avoids this potential late complication by excising the aortic sinuses and reimplanting the aortic valve into a Dacron graft, thus stabilizing the annulus. It has been suggested that the presence of the aortic sinuses is important for normal cusp motion and, potentially, cusp durability.[23-25] Several modifications to the reimplantation procedure were introduced to create neoaortic sinuses, including use of the Gelweave Valsalva graft mentioned above.[26, 27] Short- and long-term outcomes for these procedures have been encouraging. David et al. reported that freedom from moderate aortic insufficiency at 10 years was 80 ± 7%, and freedom from severe aortic insufficiency was 98 ± 1%. Preserving the normal anatomic structure of the aortic cusps is the key element in the success of the procedures and serves to highlight the importance of the patient selection process.

Aortic valve-sparing root procedures have acceptable results, with long-term stable outcomes of the aortic valves even in patients with aortic insufficiency, bicuspid valve, and connective tissue disorders such as Marfan syndrome and Loeys-Dietz syndrome.[28] Many of these long-term results require validation as they are mostly reported by the originator of the operative approach.

Conclusions

Aortic root repair is the treatment of choice for patients with aortic root pathology. Improvements in surgical technique, including cardiopulmonary bypass, myocardial protection, cerebral protection, and anesthetic management, have resulted in improved perioperative and long-term outcomes following aortic root repair. With improvements in bioprosthetic valve durability and fewer problems associated with anticoagulation, patients and surgeons are increasingly choosing composite biologic valve grafts with excellent short- and long-term outcomes — typically less than 5% mortality for elective procedures. Advances in technology and techniques include using the Valsalva graft, which re-establishes the anatomic and physiologic function of the root in the form of pseudosinuses. Also, supra-annular implantation of the valve conduit has allowed for improvements in hemodynamic parameters (reduced gradients) and potentially improved valve durability while allowing for easier possible reintervention surgically or via percutaneous valve implantation. In the future, as endovascular technology develops ways to navigate the coronary ostia, branched and fenestrated grafts may play a role in aortic root repair.

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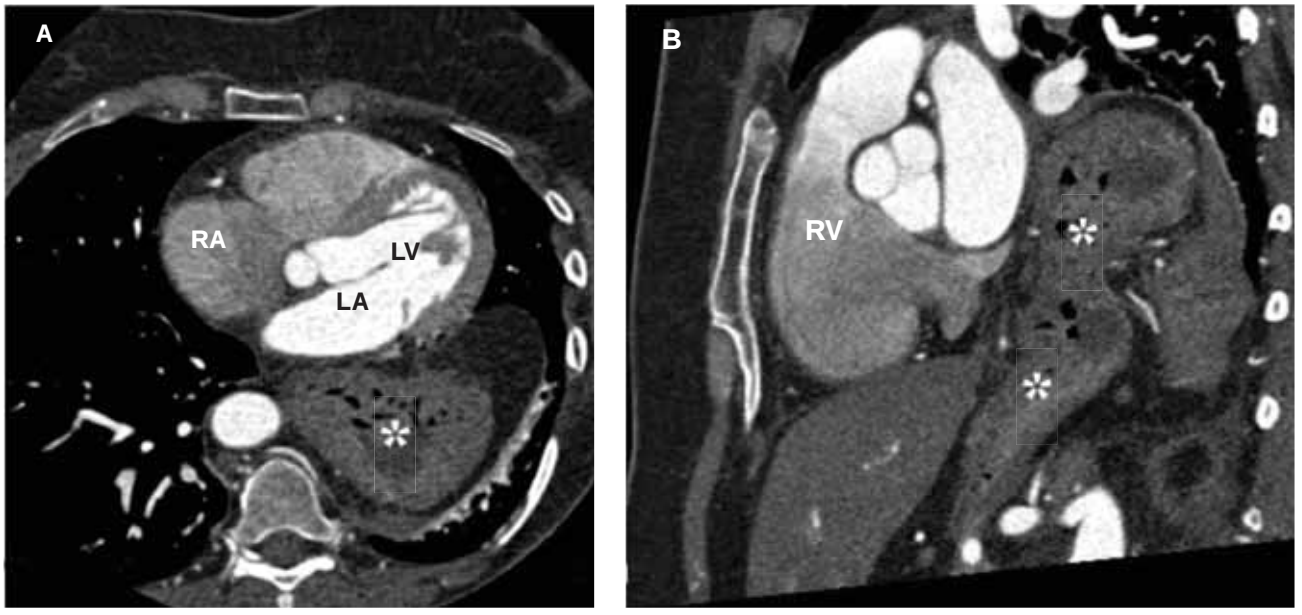

Noncardiac cause of atypical chest pain: a 40-year-old female presented to the emergency department with atypical chest pain. The cardiac CT (A: axial image; B: sagittal image) showed absence of coronary stenosis and plaque but revealed a very large hiatal hernia (*), likely the cause of her symptom. RA: right atrium; LA: left atrium; LV: left ventricle; RV: right ventricle

Image courtesy of Su Min Chang, M.D.

Imaging Vignette: Scimitar Syndrome

Saurabh Rajpal, M.D.; Lucien Abboud, M.D.;
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Methodist DeBakey Heart & Vascular Center, The Methodist Hospital, Houston, Texas

A 51-year-old female with a past medical history of diabetes mellitus, hypertension, and pulmonary hypertension was referred for cardiac magnetic resonance (CMR) evaluation of possible left-to-right shunt due to a suspected increase in oxygen saturation in her right atrium during right heart catheterization. CMR demonstrated partial anomalous pulmonary venous connections (PAPVC) with both right superior and inferior pulmonary veins draining into the inferior vena cava, consistent with scimitar syndrome (Figure 1). There was normal left-sided pulmonary venous return, and no atrial septal defect was detected. Phase contrast CMR demonstrated left-to-right shunt of 6.0 L/min with a Qp:Qs of 1.9:1.0.

Scimitar syndrome is a rare congenital anomaly (3–5% of all PAPVC, incidence of which is 0.4–0.7% of adults at autopsy) characterized by anomalous venous connections from the right lung into the inferior vena cava. The symptoms in the adult form are often mild initially, but chronic right-sided overload and pulmonary hypertension are late complications. The term "scimitar syndrome" derives from the shadow created by the anomalous vein on the chest radiograph that resembles a scimitar — a curved Turkish sword. Gadolinium-enhanced MR angiography is a non-invasive, radiation-free technique that exploits its high spatial resolution to provide three-dimensional anatomically detailed images. Phase-contrast CMR can add functional data sets to allow quantification of shunting and calculation of Qp:Qs ratio.

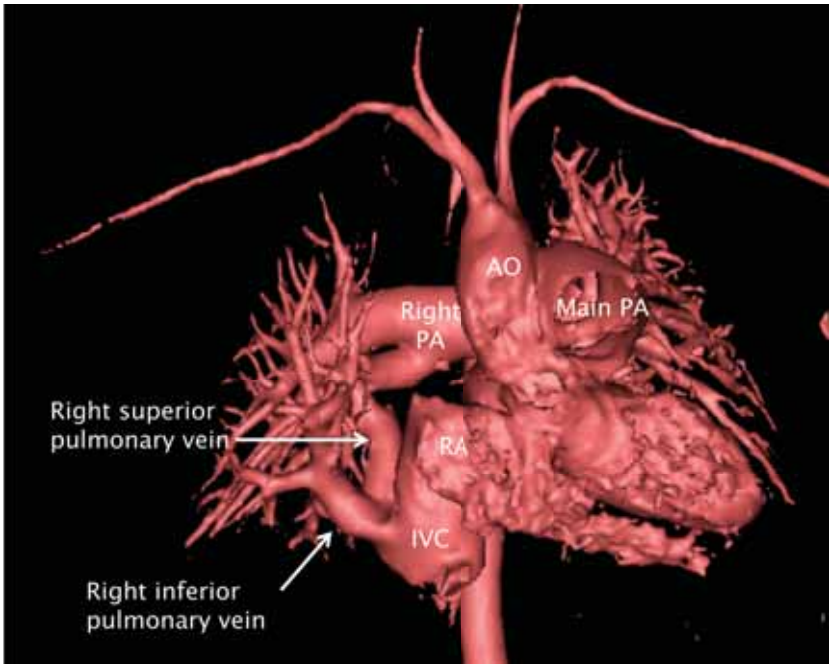


Figure 1. Anterior surface shaded display of gadolinium-enhanced chest magnetic resonance angiography; right superior and inferior pulmonary veins drain into the inferior vena cava (scimitar syndrome). AO: aorta; PA: pulmonary artery; RA: right atrium; IVC: inferior vena cava

Through the generosity of Charles R. Millikan, D. Min., vice president for spiritual care and values integration, an annual award competition was established at The Methodist Hospital among the resident staff. For the inaugural competition, residents submitted a poem or essay of 1,000 words or less for the topic, "On Being a Doctor." A committee of 5 was selected from The Methodist Hospital Education Institute to judge the entries. Criteria for judging were established by this committee. The 2nd place winning essay is herein published. The 3rd place winning entry will be published in the next issue of this journal.

RACECAR

John McFate, M.D.

"Gentlemen, start your engines…" This commonly known phrase will always hold a deeper meaning with me for the rest of my career and life. As physicians, we may not remember every detail about the patients we encounter, but there are some that we can never forget. During residency, we learn that being a doctor is not about the regulated minutes and hours. On the contrary, those minutes, hours, and moments give us opportunities. These opportunities shape our qualities and beliefs that represent the essence of our core as a professional healthcare provider.

"Good morning Racecar, always good to see you." Every day that greeting made me chuckle, because it represented a small symbol of the relationship he and I forged over many weeks as patient and doctor. The path we took to get there is one I will never forget. He had a significant history with multiple comorbidities and previous kidney and pancreas transplants. Over the weekend, the patient came to our hospital for scrotal pain. Initially, he was diagnosed with epididymitis and treated accordingly. As I returned to work the following Monday, our role was to monitor his transplant graft function. Something was not right. This is where I learned a valuable lesson; look beneath the surface. Over the next 24 hours, the patient's clinical exam worsened, and it became apparent that he did not have the initial diagnosis. He was found to have Fournier's gangrene requiring immediate surgical intervention. As I sat there explaining to him the procedure and the reasons for surgery, I could see the fear in his eyes. It was in this instance that I was given one of those moments to make a difference. We had a long and meaningful conversation which put his mind at ease. He said he had faith and trust in me and our team in order to accomplish whatever needed to be done, and he did not feel alone.

As expected, his debridement became more extensive after initial exploration. He was in critical condition post operatively. During this time, I began to converse with his father who soon arrived after hearing of his son's illness. Once again, there were multiple questions that needed answers. As a resident caring for his son, I was given the chance to continually communicate the different aspects of his case and management. It was clear to me the only thing I needed in order to instill hope and compassion was time. During his admission, numerous specialty medical and surgical teams cared for the patient. Yet, with all of our specialists and their individual components to his plan, he always looked to me for understanding. I took this opportunity each day as a privilege, not a burden. No one can make another person "care" for someone. We have to recognize the opportunity that is laid before us and seize that moment. After we discussed the different management strategies, we would also talk about his life. The conversation would range from his home to hobbies to his future plans. As in our discussions of his medical care, these other times further strengthened our relationship and, in turn, his trust in me as his doctor.

As he recovered in the following weeks on the ward floor, he gave me the alias of "Racecar." I was puzzled and a little concerned. I soon learned though, that he was a huge racing fan, and that I reminded him of his favorite famous driver. That title represented more than just a celebrity look alike. It represented many different things. If you have ever seen a race, it appears that the "racecars" just drive around in laps over and over again. They go many miles but never go anywhere. Sometimes as doctors, I think we can feel like this. Rounds in the hospital, same patients every day, slow progress in their recovery, progress notes, and all of our daily tasks can become redundant. But more importantly, I have learned that in the races he watches, the driver wins or loses depending on the pit stops, not the number of laps. This is the lesson he taught me without even realizing it.

Humbly, I accept the name "Racecar" as a personal badge to remind myself of the responsibilities that lay before me. When our lives get stressed and hectic, I remember this simple name to regain perspective. The chances we have to make a difference in the lives of our patients and their families are not taken for granted. Equally as important as the number of laps around the hospital and cases performed each day are the stops we make with our patients and each other. We can all be "racecars" moving fast to provide the best care possible for our patients. We cannot forget though, to stop, park, and take the time to talk with our patients. In those instances, we break down barriers, build relationships, and create hope and healing. This, my fellow colleagues and friends, is how we will win the race!

CRABBY OLD MAN

This poem, "Crabby Old Man," was sent to me by a close friend/patient because of its sentiment but not knowing I would publish it in our journal. Its origin is anonymous but reportedly came from an elderly man who died in the geriatric ward of a nursing home. It was believed he had nothing left of value. But after going through his meager possessions, they found this poem. Copies were made and distributed to every nurse in the nursing home and thence to the world. I am told his sole bequest to prosperity has since appeared in other publications.

It is worth remembering when you next encounter a serious elder who you might brush aside without seeing what was once there. As an elder, it resonated strongly with me. — W.L. Winters Jr., M.D.

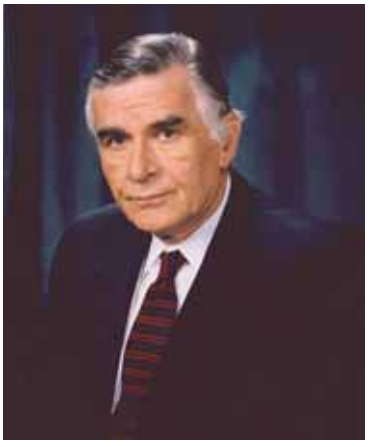
What do you see nurses? … What do you see?
What are you thinking … when you're looking at me?
A crabby old man … not very wise,
Uncertain of habit … with faraway eyes?
Who dribbles his food … and makes no reply.
When you say in a loud voice … 'I do wish you'd try!'
Who seems not to notice … the things that you do.
And forever is losing … A sock or shoe?
Who, resisting or not … lets you do as you will,
With bathing and feeding … The long day to fill?
Is that what you're thinking? … Is that what you see?
Then open your eyes, nurse … you're not looking at me.
I'll tell you who I am … As I sit here so still,
As I do at your bidding, … as I eat at your will.
I'm a small child of Ten … with a father and mother,
Brothers and sisters … who love one another.
A young boy of Sixteen … with wings on his feet.
Dreaming that soon now … a lover he'll meet.
A groom soon at Twenty … my heart gives a leap.
Remembering, the vows … that I promised to keep.
At Twenty-Five, now … I have young of my own.
Who need me to guide … And a secure happy home.
A man of Thirty … My young now grown fast,
Bound to each other … With ties that should last.
At Forty, my young sons … have grown and are gone,
But my woman's beside me … to see I don't mourn.
At Fifty, once more, babies play 'round my knee,
Again, we know children … My loved one and me.
Dark days are upon me … my wife is now dead.
I look at the future … shudder with dread.
For my young are all rearing … young of their own.
And I think of the years … and the love that I've known
I'm now an old man … and nature is cruel.
Tis jest to make old age … look like a fool.
The body, it crumbles … grace and vigor, depart.
There is now a stone … where I once had a heart.
But inside this old carcass … a young guy still dwells,
And now and again … my battered heart swells.
I remember the joys … I remember the pain.
And I'm loving and living … life over again.
I think of the years, all too few … gone too fast.
And accept the stark fact … that nothing can last.
So open your eyes, people … open and see.
Not a crabby old man … Look closer … see ME!!

W.L. Winters Jr., M.D.

MANUS JAMES O'DONNELL, M.D. 1929–2010

William L. Winters Jr., M.D.
Methodist DeBakey Heart & Vascular Center, The Methodist Hospital, Houston, Texas

One of Dr. Michael E. DeBakey's favorite cardiologists, Dr. Manus James O'Donnell, died March 22, 2010 at age 80. Manus, as he was known to his friends, remained an Irishman to the core during his years in Houston, retaining proudly his Irish brogue. Manus was born in Killough County, Ireland, on May 5, 1929. He attended St. Malachy's College (class of 1945) in Belfast, Ireland, before entering medical school in Queens University, Belfast, graduating in 1955. He then served an internship at the Mater Infirmorum Hospital in Belfast, followed by a medical residency at the Royal Victoria Hospital completed in 1958. During Manus's cardiology fellowship at the Royal Victoria Hospital, he engaged in animal experiments using the Lillehei Dewall pump oxygenator with Dr. Frank Partridge, who was preparing to start a human open-heart surgery program. From 1960 to 1965, he served as a principal registrar in medicine and collected numerous scholastic honors on his way to becoming a member of the Royal College of Physicians in 1961.



Manus James O'Donnell, M.D.
1929–2010

In 1965, he accepted a fellowship in cardiology at Baylor College of Medicine and moved his family to Houston, Texas, their permanent residence thereafter. Upon completing the one-year cardiology fellowship, he accepted an appointment as assistant professor in medicine at Baylor with an active staff appointment at The Methodist Hospital, where he remained until his retirement in 2003. His professional career centered around his teaching and clinical responsibilities at Baylor College of Medicine and The Methodist Hospital. Starting in 1975, he directed the activities of the cardiac catheterization laboratories at The Methodist Hospital for many years. He shared with several other cardiologists the responsibilities managing the patients of Dr. Michael E. DeBakey — an intellectual and physical challenge every day of the week. His good humor, ready smile, and Irish brogue endeared him to his colleagues and patients alike. His favorite relaxation was golf played all over the world, wherever there might be found a golf course.

He left behind his wife of 50 years, Patricia, and his daughter, Phiona, plus two sisters and several nieces and nephews. Manus was a delightful colleague and a compassionate physician embodied in a very fine human being. He is one of those upon whose shoulders was built the foundation of The Methodist DeBakey Heart & Vascular Center as it stands today. He is missed, but his spirit remains enshrined within the walls of this Center.

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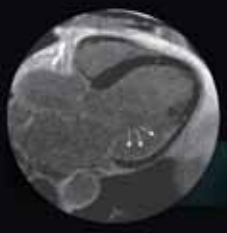

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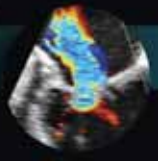

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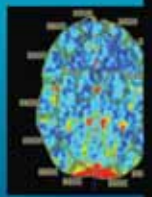

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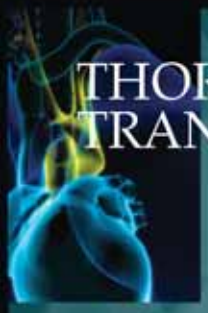

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The Methodist DeBakey Heart & Vascular Center continues the groundbreaking work begun by famed heart care pioneer, Dr. Michael E. DeBakey, and his associates, who developed many of today's life-saving techniques, tools and procedures at The Methodist Hospital. Located in Houston, Texas, the Methodist DeBakey Heart & Vascular Center combines research, prevention, diagnostic care, surgery and rehabilitation services in a coordinated multidisciplinary program with one focus: delivering compassionate, effective care and treatment to patients suffering from heart disease.

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