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Guidelines for the management of thoracic aortic disease in 2017

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Abstract This review provides a general overview of the consensus statement from the 2010 more recent updates AHA/ACC *Guidelines for the Diagnosis and Management of Patients with Thoracic Aortic Disease*, and highlights current practice patterns. The suggestions herein are intended to facilitate clinical decision making in the management of thoracic aortic disease. The main intent of this report is to highlight screening, surveillance, initial and definitive management of thoracic aortic disease, and special populations that should be considered.

Keywords Thoracic aortic dissection · Thoracic aortic aneurysm · Thoracic aortic disease · Guidelines · Management

Introduction

The management of thoracic aortic disease historically is based on changes to the aorta that are not symptomatic or easily detectable until an acute complication develops with rapid demise thereafter. The relatively unique nature of this situation presents an urgent need to provide insight into identifying patients at risk for acute and catastrophic complications and candidates for potentially curative surgical treatment. Over the past several years, however, marked improvements in outcomes have been observed. Several factors underpin modern management. For example, preoperative patient selection, advances in brain perfusion management and endovascular approaches, and regionalization of referrals to high-volume, tertiary care centers. Most recently, endovascular approaches to the descending thoracic aorta have often appeared to replace open repair.

Standardizing management of thoracic aortic disease is important to improve patient care and ensure the delivery of consistent outcomes. A national expert panel from American Heart Association (AHA) and American College of Cardiology (ACC) and others has addressed the topic of the management of thoracic aortic disease in 2010 [1–6]. The design, development, analyses, and results of the consensus process were released as the *Guidelines for the Diagnosis and Management of Patients with Thoracic Aortic Disease*, and were reviewed here to provide a framework to guide management [1–6].

Throughout the text, the references associated with each indication denote whether the indication is included in current ACC/AHA guidelines [1–6]. Comments lacking a reference represent the opinion of the authors. Our herein review is intended to compliment the consensus guidelines developed in 2010 and updates and aims to provide an overview and expert clinical opinion of the critical issues and considerations of the management of the thoracic aortic disease.

Epidemiology

Thoracic aortic disease affects 3–6 per 100,000 people/year [1–6]. Internationally, population-based studies have shown an annual incidence ranging from 6 cases per 100,000 (British study), to 9.1 per 100,000 in women or 16.3 per 100,000 in men (Swedish study) [7, 8].

It is increasingly evident that thoracic aortic disease is a heterogenous disorder with variable clinicopathologic

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phenotypes and natural history. The diameter of the normal thoracic aorta varies by age, gender, and imaging modality [1–6]. Subtypes of thoracic aortic disease have emerged, with patients classified according to distinct clinical features. Traditionally, thoracic aortic disease subtypes have been identified as either an aneurysm or a dissection. Aneurysm refers to a dilation of the aorta greater than 50% of normal diameter. Dissection involves disruption of the intima and tracking of blood which propagates within the aortic media [1–6]. A dissection is not always clinically distinct from an aneurysm, but overall the most devastating complication of aortic disease other than rupture.

The two major classifications of aortic dissection recognize and emphasize either the site of origin tear (Debakey) or the involvement of the ascending aorta (Stanford) [9–11]. The Debakey classification includes dissections that originate in the ascending aorta and either propagate to the aortic arch (type I), stay confined to the ascending aorta (type II), or originate in the descending aorta (type III) [1]. The Stanford scheme highlights involvement of the ascending aorta (type A) or not (type B), irrespective of point of origin [1]. The Debakey and Stanford schemes both remain relevant in the modern era and provide useful clinical and prognostic information.

Conditions associated with medial degeneration—including Marfan syndrome, Loeys–Dietz syndrome, Ehler–Danlos syndrome, inflammatory diseases of the aorta, Turner syndrome, bicuspid aortic valve, and familial thoracic aortic aneurysm and dissection syndrome—are associated with early onset aortic dissection [1].

Risk-based screening

The main objective in safely managing thoracic aortic disease is preventing future complications. When an aortic dissection, aneurysm, traumatic injury, and/or aortic rupture are recognized, it is paramount that this is immediately communicated to appropriate physician (Class I, Level of Evidence: C) [3]. Screening patients with thoracic aortic aneurysms may enable earlier identification of patients possibly at an increased risk for life-threatening complications.

In asymptomatic patients, the main determinant of the need for intervention is size, whereas any symptomatic patient must be referred for surgery. The AHA/ACC guidelines suggest that 5.5 cm is the threshold for surgical referral for patients with dilated portion of their thoracic aorta (from root to descending), on the basis of the relative risks of surgical complications, rupture, dissection, and death [3]. Physicians evaluating patients with known thoracic aneurysm and any constellation of symptoms (chest or back or extremity pain, vision changes, or shortness of breath) must be cognizant of the possibility that aneurysm pathology may be causing these symptoms. These patients must be emergently referred to a physician.

Recommended workup

Thoracic aortic disease is primarily a surgical disease, but optimal management depends greatly on appropriate workup. Given the presentation may be variable, all patients should be evaluated preoperatively by the cardiac surgeon and a cardiologist.

The initial evaluation includes a complete medical history and physical examination. Classic symptoms of sudden severe onset chest or back pain are a hallmark of thoracic dissection. Thorough questioning regarding the onset, duration, extent, and characterization of the pain is important, as is history of any aneurysms. Patients may also present with neurologic deficits (vision change, headache, and stroke), and abdominal pain caused by mesenteric compromise or extremity pain due to femoral artery dissection. Constellation of several of these symptoms strongly suggests thoracic aortic disease. Because the clinical presentation is not always distinguishable from coronary artery disease, myocardial infarction must be considered. A detailed family history should also be obtained.

A careful physical examination may provide clues to the extent of the aneurysm or dissection. This must include assessment of the brain, heart, mesentery, and extremities. Next, an electrocardiogram, chest X-ray, and appropriate laboratory studies are performed (Class I, Level of Evidence: B) [3]. In patients determined to be high risk for an aortic dissection, a negative chest X-ray should not delay further cross-sectional imaging (Class III, Level of Evidence: C) [3]. Computed tomography (CT) has been the mainstay of evaluation, providing detail and longitudinal monitoring and evaluation of therapeutic response. To obtain maximal benefit, computed tomography angiography (CTA) ideally should be obtained, which may not be possible in all patients. In CTA, intravenous contrast is delivered at rate of 3–5 mL/s, creating 3 mm thick image slices of the aorta from the thoracic inlet to groin. It is critical to evaluate the aortic diameter perpendicular to the axis of blood flow at multiple levels (Class I, Level of Evidence: C). At the level of the aortic root, the maximum diameter is typically the mid-sinus level, where the root is widest. In addition, on the CT scan, the presence of intramural hematoma (IMH) and penetrating atherosclerotic ulcer (PAU) should be noted. Magnetic Resonance Imaging (MRI) can provide superior delineation of the aorta, but its use is limited by accessibility. Once the diagnosis has been made, transthoracic ultrasound remains a useful adjunct to determine the presence of a pericardial or pleural effusion and competency of the aortic valve.

Surveillance of thoracic aortic aneurysm

Although asymptomatic patients with known thoracic aortic aneurysms often have an indolent course, the standard management for surgically fit patients is entry into a surveillance program. Once a critical size is reached, the patients are referred for surgery [1]. Symptomatic patients from an expanding thoracic aneurysm should be evaluated for prompt surgical intervention (Class I, Level of Evidence: C) [3]. However, surgery is not indicated with limited life expectancy or substantially impaired quality of life.

The ACC/AHA advocates that an aortic root or ascending aorta diameter 5.5 cm or greater without risk factors for dissection should be referred for surgery (Class I, Level of Evidence: B) [3]. For patients isolated aortic arch aneurysms, surgery indicated when the arch diameter exceeds 5.5 cm (Class I, Level of Evidence: B) [3]. For arch diameter less than 5.5 cm, it is reasonable to reimage using computed tomography scan or magnetic resonance imaging to detect enlargement of the aneurysm [3]. Twelve-month interval screening for aneurysms less than 4.0-cm diameter and 6-month intervals for 4.0–5.4-cm arch diameter [3].

Once the descending thoracic aortic diameter exceeds 5.5 cm, then intervention is indicated, a Class Ib indication in the ACC/AHA guidelines [3]. For patients with chronic dissection, particularly if associated with a connective tissue disorder and without significant comorbid disease, open repair is recommended. In the setting of degenerative or traumatic aneurysms, saccular aneurysms, or postoperative pseudoaneurysms, it seems worthwhile to consider endovascular stent grafting.

For patients with thoracoabdominal aneurysms, elective surgery is recommended if the aortic diameter exceeds 6.0 cm, or less if a connective tissue disorder is present [3]. Endograft stent graft should be considered if surgical morbidity is elevated. Moreover, thoracoabdominal aneurysm with end-organ ischemia or visceral artery disease may require additional revascularization.

Special populations and circumstances

Bicuspid aortic valve

Similarly, AHA/ACC guidelines recommend surgery in patients with bicuspid aortic valve and an aortic root diameter of 5.5 cm or larger (Class I, Level of Evidence: B) [3]. The threshold for surgical referral is 5.0 cm with bicuspid valve and additional risk factor for dissection (family history or rapid growth) (Class IIa, Level of Evidence: C) [3]. A more recent update has clarified the timing of surgery [12, 14].

Marfan syndrome

According to AHA/ACC guidelines, at the time of the initial diagnosis of Marfan syndrome, an echocardiogram is recommended to determine aortic root and ascending aorta size [3]. Six months thereafter, a repeat echo is recommended to determine rate of enlargement of the aorta (Class I, Level of Evidence: C) [3]. If the aorta is stable, then imaging can be done annually. Once the aortic diameter is 4.5 cm or greater, or if there is significant growth from baseline, the imaging should be more frequent. Surgical repair is reasonable if the maximal cross-sectional area (square centimeters) of the ascending aorta or root divided by the patient's height (meters) exceeds a ratio of 10. This is given the fact that shorter patients have dissection at a smaller size and 15% of patients with Marfan syndrome have dissection at a size smaller than 5.0 cm.

Loeys–Dietz syndrome

Similar to Marfan syndrome patients, according to AHA/ACC guidelines, Loeys–Dietz patients should undergo aortic imaging at the initial diagnosis and 6 months thereafter to establish rate of enlargement (Class I, Level of Evidence: C) [3]. Unlike with Marfan patients, Loeys–Dietz patients should have yearly MRI from head to pelvis (Class I, Level of Evidence: B) [3]. The absolute size at which to recommend surgery is over 5 cm.

Turner syndrome

As above, patients with Turner syndrome should undergo imaging of heart and aorta at the initial diagnosis. If, however, the initial imaging is normal and without risk factors for aortic dissection, repeat imaging is performed every 5–10 years (Class I, Level of Evidence: C) [3]. Annual imaging is recommended if abnormalities exist. According to AHA/ACC guidelines, patients with Turner syndrome, and additional risk factors (bicuspid aortic valve, coarctation of the aorta, pregnant or desiring pregnancy, and/or hypertension), it may be reasonable to perform imaging of the heart and aorta to help determine the risk of aortic dissection (Class I, Level of Evidence: C) [3, 12].

Relatives and genetic mutations

First-degree relatives of patients with thoracic aortic aneurysm or dissection should undergo aortic imaging (Class I, Level of Evidence: B) [3]. If one or more first-degree relatives are found to have thoracic aortic aneurysm or dissection, then imaging of second-degree relatives is reasonable (Class IIa, Level of Evidence: B) [3]. Moreover, then referral to a geneticist may be considered reasonable (Class IIa,

Level of Evidence: B) [3]. The geneticist should screen for the following aneurysm/dissection-associated genes: FBN1, TGFBR1, TGFBR2, COL3A1, ACTA2, and MYH11 reasonable (Class I, Level of Evidence: C) [3]. Sequencing of the ACTA2 gene will determine if ACTA2 mutations are responsible for the inherited predisposition reasonable (Class IIa, Level of Evidence: B) [3]. Furthermore, additional genes may be sequenced: TGFBR1, TGFBR2, and MYH11 (Class IIa, Level of Evidence: B) [3].

Per AHA/ACC recommendations, first-degree relatives of patients with a bicuspid aortic valve, premature onset of thoracic aortic disease with minimal risk factors, and/or a familial form of thoracic aortic aneurysm and dissection should be evaluated for the presence of a bicuspid aortic valve and asymptomatic thoracic aortic disease (Class I, Level of Evidence: C) [3]. Moreover, in patients with bicuspid aortic vales, both the root and ascending aorta need to be evaluated (Class I, Level of Evidence: B) [3].

Thoracic aortic dissection

While no single algorithm is applicable to each case of thoracic aortic diseases, Fig. 1 illustrates the treatment algorithm from which some generalities can be gleaned.

Involvement of the ascending aorta serves as an impetus to approach ascending aortic dissection as a surgical emergency; diagnosis and treatment must be accurate and prompt. The initial evaluation should be straightforward and aimed at preventing associated life-threatening complications such as rupture, myocardial infarction, or tamponade. Until definite surgical repair is underway, the blood pressure should be titrated to MAP of 70 mmHG using anti-hypertensives or intravenous fluids/vasopressor agents. An echocardiogram should be obtained to rule-out pericardial tamponade, contained rupture, and severe aortic insufficiency (Class I, Level of Evidence: B) [3]. Other ascending aorta variants such as intramural hematoma or penetrating ulcer generally are treated similarly to type A dissection.

The descending aorta usually is managed medically unless life-threatening complications develop (Class I, Level of Evidence: B) [3]. These included the following: ascending aorta involvement, limb of mesenteric ischemia, aneurysm expansion, progression of dissection, ongoing pain, or uncontrolled hypertension (Class I, Level of Evidence: B) [3].

Most individuals with thoracic aortic dissection will receive their initial treatment in a community setting. There should be an established plan aimed at medical management and to rapidly address any sustained alterations in cardiovascular function. Moreover, it is recommended that patients are transferred to a facility that is appropriately equipped and staffed to manage thoracic aortic disease. These specialty facilities should have the requisite personnel and provide sufficient means to handle catastrophic circumstances if necessary. It should be expected that patient transfer occurs in a timely and efficient manner. The successful transition of clinical care between the transferring and receiving facility requires proper assessment and communication by both facilities. Key points to discuss are: blood pressure and heart rate control for hypertension and tachycardia; cardiac, neurologic, renal, or mesenteric ischemia; peripheral pulse and perfusion on exam; and all relevant images and reports.

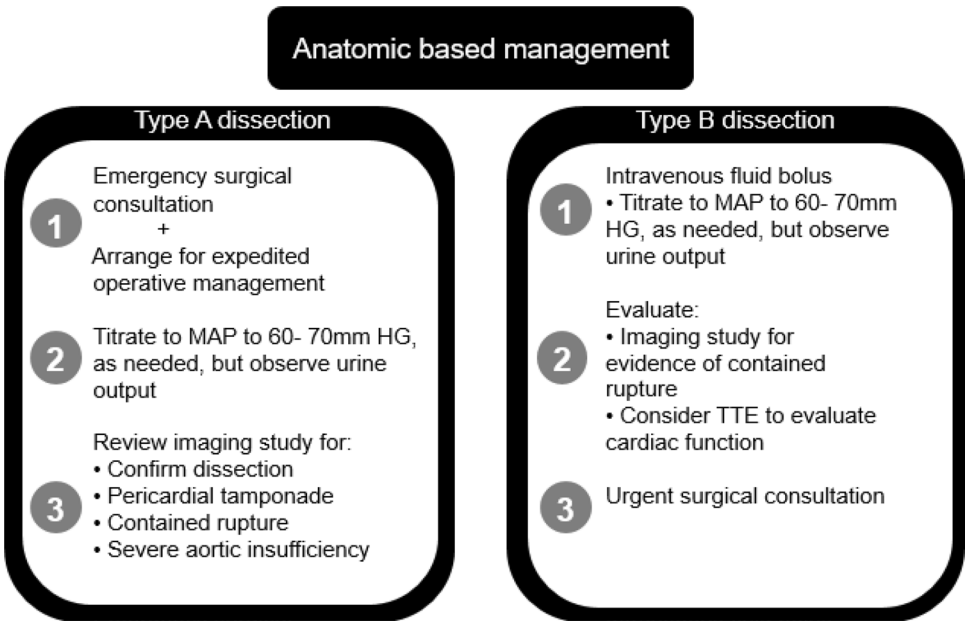

Fig. 1 Management of acute aortic dissection is based on the Stanford classification—involvement of the ascending aorta (type A dissection) or not (type B dissection). Adapted from the AHA/ACC guidelines for the diagnosis and management of patients with thoracic aortic disease [3]

Initial management of aortic aneurysm and dissection

After confirmation of a thoracic aortic aneurysm or dissection, clinicians should be vigilant about decreasing aortic wall stress by controlling heart rate and blood pressure. The blood pressure should be measured in both arms and treatment based on the highest reading. In the absence of contraindications, beta blockade can offer effective rate control in many cases. Beta-blockers are highly effective delivered intravenously and act in part by decreasing wall stress and preventing a hypertensive response [13, 14]. The most recent guidelines have recommended titrating to a target heart rate of 60 beats per minute or less (Class I, Level of Evidence: C) [3]. Beta-blockers should be used cautiously in the setting of acute aortic regurgitation, as a potential for blocking the compensatory tachycardia may exist (Class I, Level of Evidence: C) [3]. If beta-blockers are contraindicated, non-dihydropyridine calcium channel–blocking agents are recommended for second-line use (Class I, Level of Evidence: C) [3]. When adequate rate control has been obtained and systolic pressures remain greater than 120 mmHg, a course of angiotensin-converting enzyme inhibitors and/or other vasodilators is advisable to further reduce blood pressure (Class I, Level of Evidence: C) [3].

Definitive management of thoracic aortic aneurysm and dissection

The surgical management of thoracic aortic aneurysm and dissection needs to be tailored to the anatomy of the underlying aorta and aortic valve. It is strongly advised that patients with an ascending aortic dissection should be referred for emergency surgical repair. The window for effective treatment is optimally within a few hours after onset of symptoms. Starting treatment as soon as possible is and will continue to be a driving force in dissection management. However, surgeons may be reluctant to operate on patients older than age 80 years, with neurological injuries, presenting late after the onset of dissection, or with prior cardiac surgery [1].

For type A dissection, the primary goal of surgery is to eliminate the entry tear. The standard operating procedure includes replacing the ascending aorta from the sinotubular junction to the undersurface of the aortic arch. Although in most patients, there will be persistent downstream dissection with false lumen in the descending and abdominal aorta, the concept is not to entirely eliminate the dissection [1]. In patients without significant aortic root dilation, elderly patients, or young patients with minimal dilation/aortic valve disease—separate valve and ascending aortic replacement is recommended. If the aortic root is involved, this may be repaired (via valve re-implantation) or replaced (composite valve graft), depending on the pathologic or aneurysmal aortic sinuses. Patients with Marfan, Loeys–Dietz, and Ehlers–Danlos syndromes and other patients with dilatation of the aortic root and sinuses of Valsalva require excision of the sinuses. An extensive root dissection or aneurysmal root is generally treated with a modified composite valve and graft to replace the entire aortic root, or valve-sparing root replacement (Class I, Level of Evidence: C) [3]. The authors recommend valve-sparing root replacement *only for surgeons who routinely perform this procedure with proven technical success*. If the root is only partially dissected, then repair is possible with aortic valve leaflet resuspension and sparing of the aortic sinuses (Class I, Level of Evidence: C) [3].

According to AHA/ACC guidelines, for acute dissection with aneurysmal arch and/or extensive arch destruction; for chronic dissection with arch enlargement; and for distal arch aneurysms that involve the proximal descending thoracic aorta, replacement of the entire arch is reasonable (Class IIa, Level of Evidence: B) [3]. Moreover, arch replacement is recommended for arch diameter > 5.5 cm in asymptomatic, low-risk patients with isolated degenerative or atherosclerotic aneurysms (Class IIa, Level of Evidence: B) [3].

The ACC/AHA guidelines recommend open surgical repair for chronic dissection in the setting of a connective tissue disorder and a descending thoracic aortic diameter > 5.5 cm. (Class I, Level of Evidence: B) [3]. Usually, an elephant trunk procedure is required also. In aortic arch and proximal descending aortic surgery, brain and systemic protection is achieved using right axillary/subclavian artery inflow and hypothermic circulatory arrest. In 2012, the EACTS/ESC/EAPCI recommended TEVAR only for complicated type B dissections [1, 15]. The EACTS/ESC/EAPCI divided complicated into two categories: acute or subacute/chronic. Acute complication includes: persistent pain, refractory hypertension, malperfusion, and signs of rupture, hypotension, or shock [1, 15]. Chronic complications include: aortic diameter greater than 5.5 cm, diameter increase > 4 mm, refractory hypertension, recurrent malperfusion, or recurrent symptoms [1, 15]. The FDA and a multidisciplinary subcommittee that included the Society for Vascular Surgery, American Association for Thoracic Surgery, Society of Thoracic Surgeons, and the Society for Interventional Radiology, however, have agreed to limit the definition of "complicated" dissection solely to rupture, impending rupture, or distal malperfusion [1, 16].

The ACC/AHA guidelines recommend open surgical repair of thoracoabdominal aneurysms when diameter > 6.0 cm, or less with Marfan syndrome or other connective tissue disorders (Class I, Level of Evidence: C) [1, 3, 12]. Furthermore, surgical repair is also recommended in setting of end-organ ischemia or significant celiac, superior

mesenteric, or renal artery atherosclerosis (Class I, Level of Evidence: B) [1, 3, 12].

Brain protection during ascending aortic and transverse aortic arch surgery

For repair of the distal ascending aorta and transverse arch, a brain protection strategy is needed to prevent stroke and preserve cognition function (Class I, Level of Evidence: B) [3]. Deep hypothermic circulatory arrest, selective antegrade brain perfusion, and retrograde brain perfusion are possible techniques to minimize brain injury during surgical repairs of the ascending aorta and transverse aortic arch (Class IIa, Level of Evidence: B) [3].

Spinal cord protection during descending aortic open surgical and endovascular repairs

Patients at high risk of spinal cord ischemic injury during open and endovascular thoracic aortic repair need a planned spinal cord protection strategy. These strategies include: cerebrospinal fluid drainage, intrathecal papaverine, and proximal and distal aortic perfusion maintenance (Class I, Level of Evidence: B) [3].

Renal protection during descending aortic open surgical and endovascular repairs

To preserve renal function during open repair of the descending aorta, preoperative hydration and intraoperative mannitol administration are reasonable strategies (Class IIb, Level of Evidence: C) [3]. If the repair involves exposure of the renal arteries, renal protection by either cold crystalloid or blood perfusion may be considered (Class IIb, Level of Evidence: C) [3]. However, per AHA/ACC guidelines, furosemide, mannitol, or dopamine should not be the sole method of renal protection (Class III, Level of Evidence: B) [3].

Long-term surveillance of thoracic aortic disease

The patient with thoracic aortic disease is at risk for aneurysmal degeneration of the diseased aorta and for additional dissection. The risk of additional aortic pathology is primarily dependent on the aortic diameter, underlying tissue quality and blood pressure management. The cornerstone for clinical follow-up is lifetime surveillance with imaging.

Current clinical guidelines recommend that after a Type A or B aortic dissection, computed tomographic imaging (CT) or magnetic resonance imaging (MRI) of the aorta is reasonable at a schedule of 1-, 3-, 6-, and 12-month post-dissection (Class IIa, Level of Evidence: C) [3]. Then, if stable, the same imaging modality can be obtained annually thereafter (Class IIa, Level of Evidence: C) [3]. Similarly, patients with intramural hematoma (IMH) undergo same guidelines as aortic dissections (Class IIa, Level of Evidence: C) [3]. For patients with moderate and relatively stable thoracic aortic aneurysm, MRI instead of CT can be used to minimize radiation exposure [3].

Conclusion

Management of thoracic aortic disease continues to evolve. Although the initial management of thoracic aortic disease is similar, long-term management depends on anatomic involvement. Optimal outcomes are achieved by team-based approach and definite management at referral centers.

Compliance with ethical standards

Conflict of interest Authors declare that they have no competing interests.

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References

1. Goldfinger JZ, Halperin JL, Marin ML, Stewart AS, Eagle KA. Valentin fuster. Thoracic aortic aneurysm and dissection. Review article. J Am Coll Cardiol. 2014;64(16):1725–39.
2. Peterss S, Mansour AM, Ross JA, Vaitkeviciute I, Charilaou P, Dumfarth J, Fang H, Ziganshin BA, Rizzo JA, Adeniran AJ, Elefteriades JA. Changing pathology of the thoracic aorta from acute to chronic dissection: literature review and insights. J Am Coll Cardiol. 68(10):1054–65. doi:10.1016/j.jacc.2016.05.091 (**Review**).
3. Hiratzka LF, Bakris GL, Beckman JA, et al. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for the diagnosis and management of patients with thoracic aortic disease: a report of the. American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. American Association for Thoracic Surgery, American College of Radiology, American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons, and Society for Vascular Medicine. J Am Coll Cardiol. 2010;55:e27–e129.
4. Elefteriades JA, Farkas EA. Thoracic aortic aneurysm clinically pertinent controversies and uncertainties. J Am Coll Cardiol. 2010;55:841–57.
5. Mészáros J, Mórocz J, Szlávi, et al. Epidemiology and clinicopathology of aortic dissection. Chest. 2000;117:1271–8.
6. Olsson C, Thelin S, Ståhle E, et al. Thoracic aortic aneurysm and dissection: increasing prevalence and improved outcomes reported in a nationwide population-based study of more than 14,000 cases from 1987 to 2002. Circulation. 2006;114:2611–8.
7. Howard DP, Banerjee A, Fairhead JF, et al. Population-based study of incidence and outcome of acute aortic dissection and premorbid risk factor control: 10-year results from the Oxford Vascular Study. Circulation. 2013;127:2031–7.

8. Olsson C, Thelin S, Stahle E, et al Olsson C, Thelin S, Stahle E, et al. Thoracic aortic aneurysm and dissection: increasing prevalence and improved outcomes reported in a nationwide population-based study of more than 14,000 cases from 1987 to 2002. Circulation. 2006;114:2611–8.
9. Debakey ME, Henly WS, Cooley DA, et al. Surgical management of dissecting aneurysms of the aorta. J Thorac Cardiovasc Surg. 1965;49:130–49.
10. Peacock TB. Cases of dissecting aneurysm, or that form of aneurysmal affection in which the sac is situated between the coats of the vessels. Edin Med Surg J. 1843;60:276.
11. Daily PO, Trueblood HW, Stinson EB, et al. Management of acute aortic dissections. Ann Thorac Surg. 1970;10:237–47.
12. Hiratzka LF, Creager MA, Isselbacher EM, Svensson LG. 2014 AHA/ACC guideline for the management of patients with valvular heart disease representative members, Nishimura RA, Bonow RO, Guyton RA, Sundt TM 3rd. J Thorac Cardiovasc Surg 2016;151(4):959–66. doi: 10.1016/j.jtcvs.2015.12.001 **(Epub 2015 Dec 9)**.
13. Prokop EK, Palmer RF, Wheat MW Jr. Hydrodynamic forces in dissecting aneurysms. In-vitro studies in a Tygon model and in dog aortas. Circ Res. 1970;27:121–7.
14. Wojnarski CM, Svensson LG, Roselli EE, Idrees JJ, Lowry AM, Ehrlinger J, Pettersson GB, Gillinov AM, Johnston DR, Soltesz EG, Navia JL, Hammer DF, Griffin B, Thamilarasan M, Kalahasti V, Sabik JF 3rd, Blackstone EH, Lytle BW. Aortic dissection in patients with bicuspid aortic valve-associated aneurysms. Ann Thorac Surg. 2015;100(5):1666–73. doi:10.1016/j.athoracsur.2015.04.126 **(Epub 2015 Jul 22; discussion 1673–4)**.
15. Fattori R, Cao P, De Rango P, et al. Interdisciplinary expert consensus document on management of type B aortic dissection. J Am Coll Cardiol. 2013;61:1661–78.
16. White RA, Miller DC, Criado FJ, et al. Multidisciplinary Society for Vascular Surgery Outcomes Committee. Report on the results of thoracic endovascular aortic repair for acute, complicated, type B aortic dissection at 30 days and 1 year from a multidisciplinary subcommittee of the Society for Vascular Surgery Outcomes Committee. J Vasc Surg 2011;53:1082–90.