Society for Vascular Surgery (SVS) and Society of Thoracic Surgeons (STS) reporting standards for type B aortic dissections

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ABSTRACT

This Society for Vascular Surgery/Society of Thoracic Surgeons (SVS/STS) document illustrates and defines the overall nomenclature associated with type B aortic dissection. The contents describe a new classification system for practical use and reporting that includes the aortic arch. Chronicity of aortic dissection is also defined along with nomenclature in patients with prior aortic repair and other aortic pathologic processes, such as intramural hematoma and penetrating atherosclerotic ulcer. Complicated vs uncomplicated dissections are clearly defined with a new high-risk grouping that will undoubtedly grow in reporting and controversy. Follow-up criteria are also discussed with nomenclature for false lumen status in addition to measurement criteria and definitions of aortic remodeling. Overall, the document provides a facile framework of language that will allow more granular discussions and reporting of aortic dissection in the future. (J Vasc Surg 2019;■:1-25.)

Keywords: Aortic dissection; Type B; Classification; Aorta; Dissection; Reporting

SECTION 1. INTRODUCTION

Purpose of the document. Acute aortic dissection is the most common emergency affecting the human aorta, with high mortality and morbidity without appropriate and time-sensitive treatment. Based on data from the International Registry of Acute Aortic Dissection (IRAD),[1,2] patients with acute type B dissection composed approximately 33% of all dissection patients enrolled in the registry across a 17-year period. Management of acute type B dissection has evolved over time and now includes medical, surgical, and endovascular therapies performed by several specialties, including

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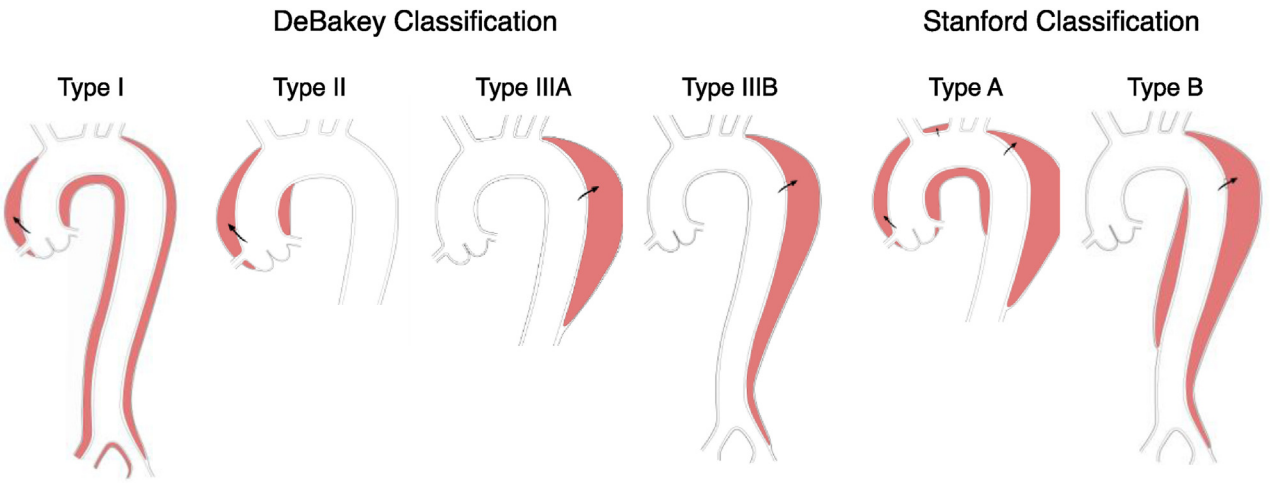

Fig 1. DeBakey and Stanford classification systems for aortic dissection.

vascular surgery, cardiothoracic surgery, interventional radiology, and cardiology.

With the recent blanket U.S. Food and Drug Administration (FDA) approval of endovascular stent grafting for type B aortic dissection (TBAD) as well as our maturing understanding of the anatomy and pathophysiology of the disease, there has been an explosion of literature in multiple specialty journals regarding TBAD presentation, treatment, and outcomes. As such, the purpose of this document is to provide structure to the reporting of TBAD, with particular attention to those attributes of TBAD that, based on the best available evidence to date, would appear to have an impact on outcomes. Prior reporting standards from the Society for Vascular Surgery (SVS) have addressed thoracic endovascular aortic repair (TEVAR) in a more general sense,[3] although these earlier standards did not specifically address aortic dissection. Given the complexity of the topic, it is believed to warrant a separate publication. This combined effort by the SVS and the Society of Thoracic Surgeons (STS) provides a unified consensus on reporting, nomenclature, and classification of TBAD at this point in time.

Organization of the Writing Committee. The committee was headed by two co-chairs, one each from the SVS and STS, with each co-chair responsible for a group of six writers evenly balanced between the societies. Each group was then further broken down into three dyads (one SVS and one STS) who were assigned a specific section of the document, the content of which was further refined by the co-chairs. The completed draft document was then approved by all members of the Writing Committee. The document was subsequently reviewed by the SVS and STS document committees and the FDA, and it was available for societal public comments. The final document was approved by the SVS and

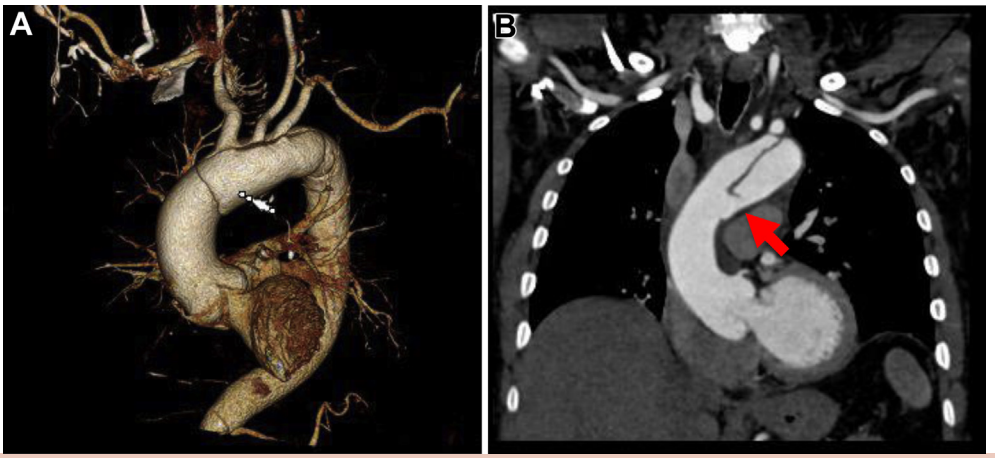

Fig 2. A, Three-dimensional computed tomography angiography (CTA) reconstruction of an aortic dissection (*arrow*) involving the aortic arch. **B,** Coronal CTA image of this same aortic dissection clearly demonstrating the location of the primary tear in the arch (*arrow*).

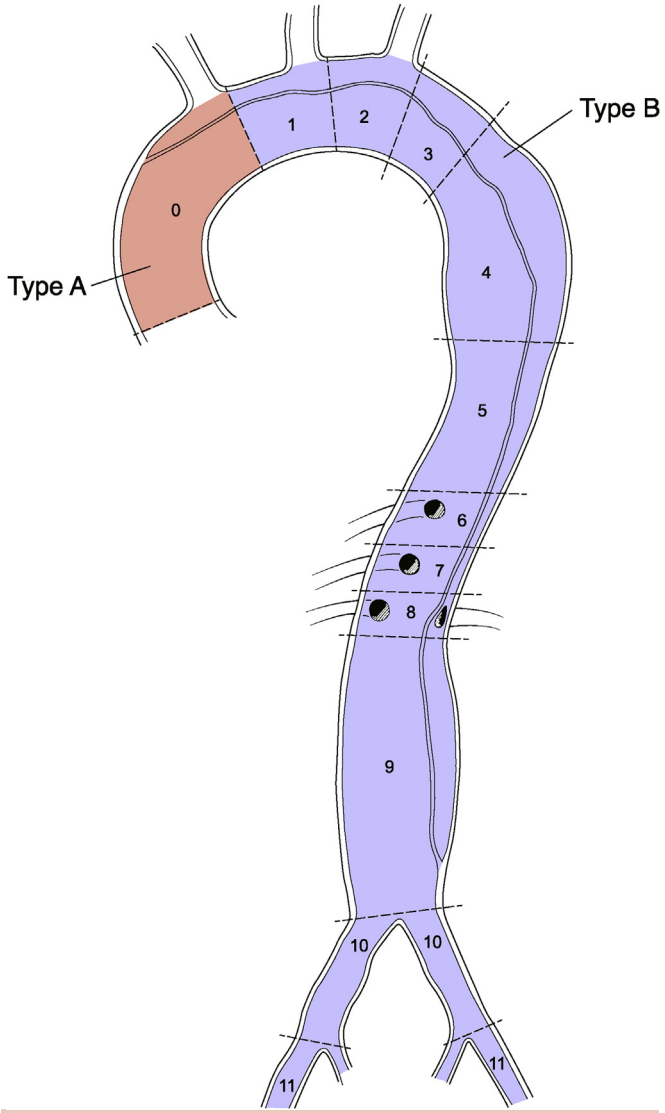

Fig 3. Society for Vascular Surgery/Society of Thoracic Surgeons (SVS/STS) Aortic Dissection Classification System of dissection subtype according to zone location of primary entry tear.

STS document oversight committees after final editing by the Writing Committee's co-chairs based on the feedback received in the review process.

SECTION 2. ANATOMIC CLASSIFICATION OF THORACIC AORTIC DISSECTION

Classification systems for thoracic aortic dissection allow caregivers to communicate accurately when describing aortic disease and are critical for triage, treatment, and prognostic purposes. Historically, classification systems relied on the anatomic location of intimal entry tears and longitudinal extent of the dissection flap. The original DeBakey classification, first described in 1965, defines aortic dissection according to anatomic features. The more widely adopted Stanford classification

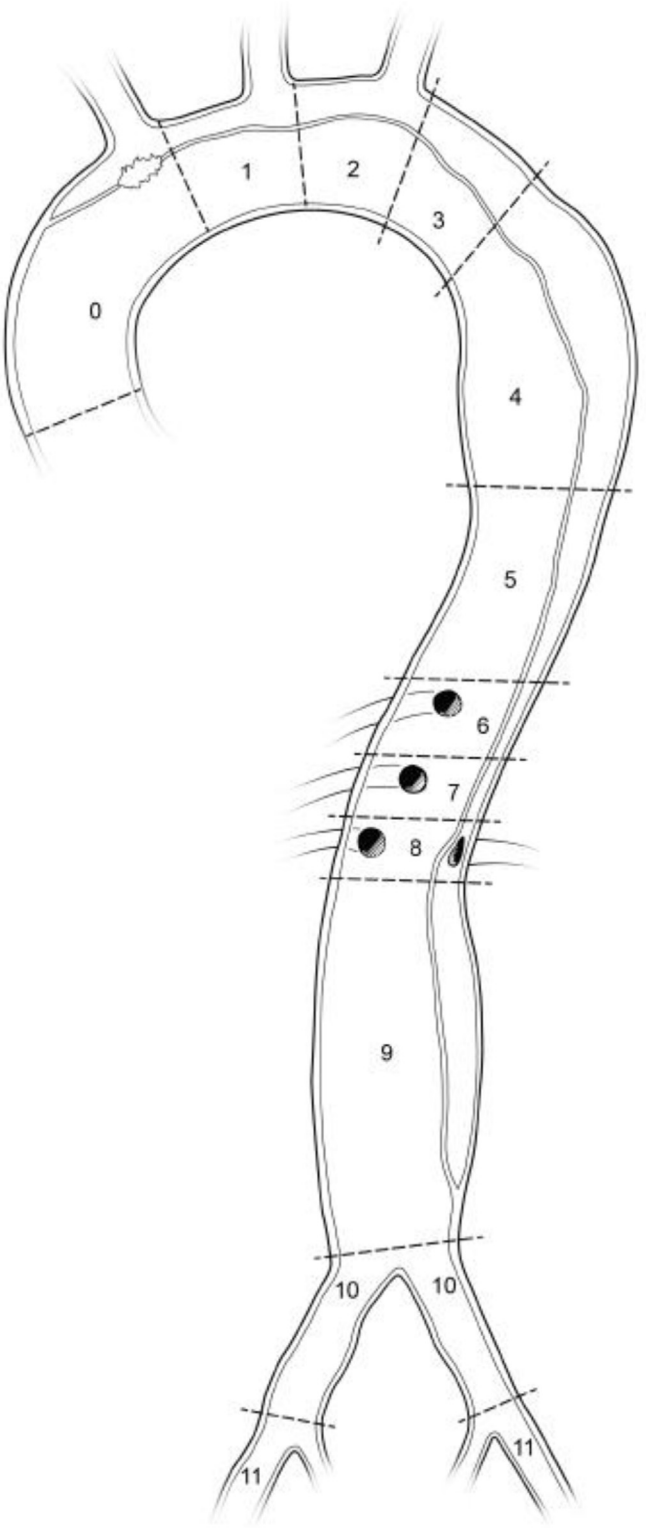

Fig 4. An aortic dissection with an entry tear in zone 0 is classified as type A. In the example illustrated, the dissection process extends distally to zone 9, such that the dissection is fully classified as A_9.

simplified the earlier DeBakey classification and is based on whether the ascending aorta is affected. In type A, the ascending aorta is involved; whereas in type B, the

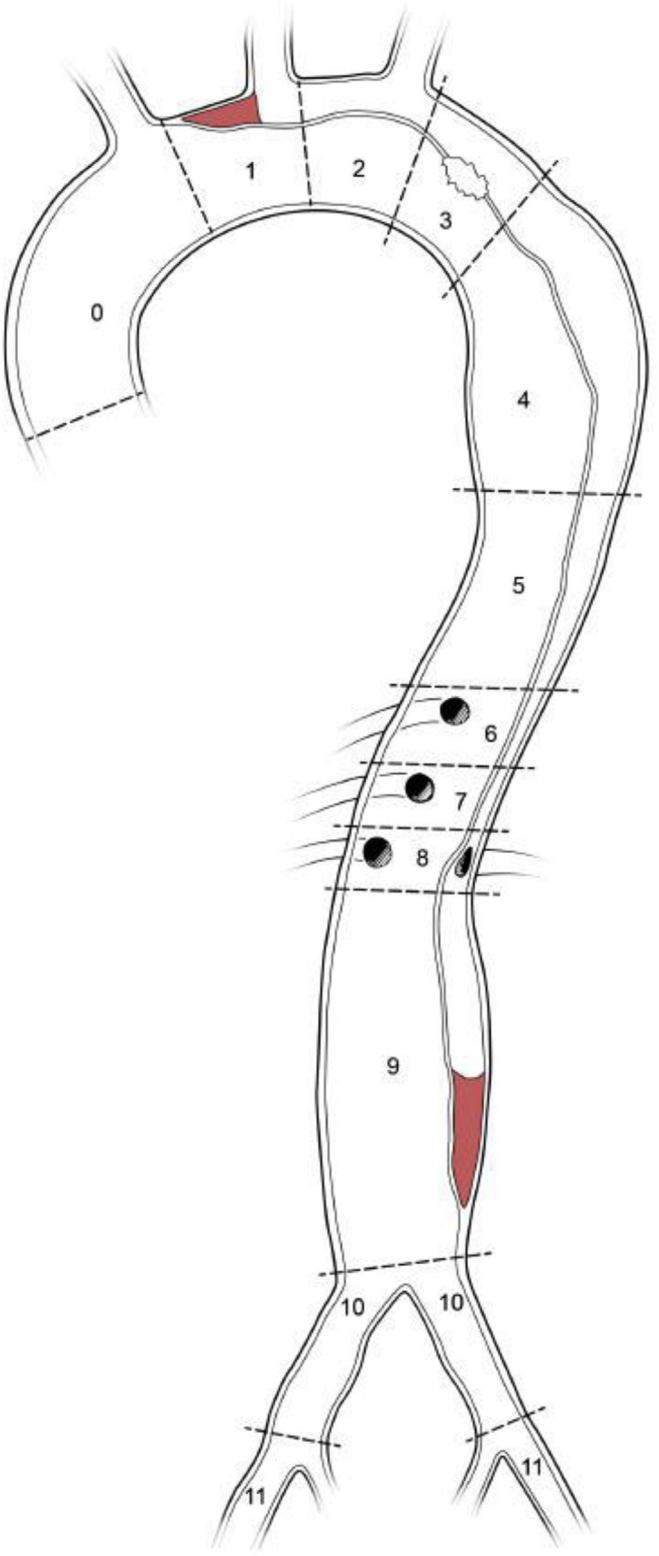

Fig 5. An aortic dissection with an entry tear in zone 1 or beyond is classified as type B. In the example illustrated, the entry tear is in zone 3 and the dissection process involves zone 1 proximally and extends distally to zone 9; the dissection is fully classified as $B_{1,9}$.

ascending aorta is spared, with the entry tear distal to the left subclavian artery (Fig 1). Unlike the DeBakey classification scheme, the Stanford classification does not characterize the distal extent of dissection.[4]

Importantly, neither classification scheme addresses aortic dissections that originate in the arch. In arch dissections, the dissection flap or intramural hematoma (IMH) typically involves the transverse arch and often spares the proximal ascending aorta (Fig 2). The entry tear may originate in the arch itself or distal to the left subclavian artery and extend retrograde to the level of the innominate artery or even the distal ascending aorta. Often, the dissection extends into the great vessels themselves. A recent query of IRAD for all patients presenting with acute type B dissection with an identifiable primary intimal tear found evidence for retrograde arch extension in 16.5%. This finding did not appear to have an impact on management strategy or early and late death, suggesting that retrograde arch extension may be analogous to TBAD.[5] However, without a standardized method of classifying aortic arch dissections, reporting of outcomes in these patients remains cumbersome and haphazard.

Given these limitations, the Writing Committee thought a new classification system relevant to current treatment paradigms was necessary to more precisely describe aortic arch involvement in aortic dissection. Within the new SVS/STS classification scheme for aortic dissection, the distinction between type A and type B is predicated on entry tear location alone. In a type A dissection, the entry tear originates only in zone 0 (Fig 3). The distal extent of a type A aortic dissection is then simply designated by zone. For example, a type A_9 dissection represents a dissection entry tear in zone 0 with distal extension of the dissection into zone 9 (Fig 4). Type B dissections include any aortic dissection with an entry tear originating in zone 1 or beyond. Type B dissections are further characterized by two subscripts ($B_{P,D}$); subscript P describes the proximal zone of involved aorta, and subscript D describes the distal zone of involved aorta. Involved aorta includes both patent and thrombosed false lumen as well as IMH. For example, $B_{1,9}$ denotes a type B dissection with proximal involvement of zone 1 and distal extension to the level of zone 9 (Fig 5), although the primary entry tear may be anywhere between zones 1 and 9. TBAD may also involve the ascending aorta, designated $B_{0,D}$ in that the primary entry tear originated in zone 1 or beyond yet extended proximally to zone 0 (Fig 6). If the entry tear origin is not identifiable, the dissection will remain indeterminate with the designation I. These dissections will always involve zone 0 or otherwise would be sensibly designated type B. Indeterminate dissections will follow the same format for distal extent as type A. Therefore, an indeterminate dissection extending from zone 0 to

zone 9 would be designated I_9 until further imaging or gross anatomic findings at surgery identify the origin of the primary entry tear; thus, the I designation may be impermanent. I dissections that are subsequently determined to be type B (ie, primary entry tear in zone 1 or beyond) may require a different treatment algorithm and may have a natural history that differs from true type A dissection. As such, the Writing Committee thought a separate I classification would allow more precise description of dissection type for future research reporting of outcomes for aortic dissection involving zone 0 with differing entry tear origins.

Using this new SVS/STS classification system will allow clinicians to conceptually project a precise image of entry tear location and proximal and distal dissection extent with one simple designation (Fig 7). It is not necessarily the intent of the Writing Committee that the new classification system should completely replace the current Stanford and DeBakey systems for everyday clinical use, especially for practitioners who are not subject matter experts in aortic disease. This classification system is intended for research reporting, such as comparative effectiveness studies, where it will allow more granular description of study populations and disease processes particularly relating to arch involvement.

Classification of new aortic dissection after prior dissection with or without repair (ie, acute-on-chronic dissection) can be difficult. Patients with a history of prior type A or type B dissection (repaired or unrepaired) now presenting with a new acute dissection should be reported both to historic dissection pathology, type of prior repair (if any), and current "residual" anatomy. For example, patients who present with new acute disease in a medically managed chronic state may be classified as acute-on-chronic A_D or $B_{P,D}$. Similarly, patients with prior aortic surgery managed in the chronic phase and now presenting with acute disease would be described as residual acute-on-chronic A_D or $B_{P,D}$.

Central repair of type A dissection, for instance, can carry multiple surgical solutions, such as aortic valve resuspension with supracoronary ascending tube graft; aortic root replacement (eg, Bentall, valve sparing), with or without concomitant hemiarch or total arch replacement; and total arch replacement, also potentially including conventional or frozen elephant trunk repair. Residual arch and descending aortic disease after central aortic repair (ie, remaining type B after type A repair) is the subject of ongoing investigation.[6] Open reconstruction, endovascular repair, and hybrid procedures should therefore describe zones of residual disease with defined proximal and distal anastomoses and landing zones. For instance, a patient with a prior type A proximal aortic repair now presenting with a symptomatic chronic arch and descending thoracic dissection extending to the iliacs may be referred to as a residual $B_{1,11}$. Classifying this as a residual dissection infers the fact that this patient has undergone prior surgery. We anticipate a significant amount of reporting for management of these situations in the future.

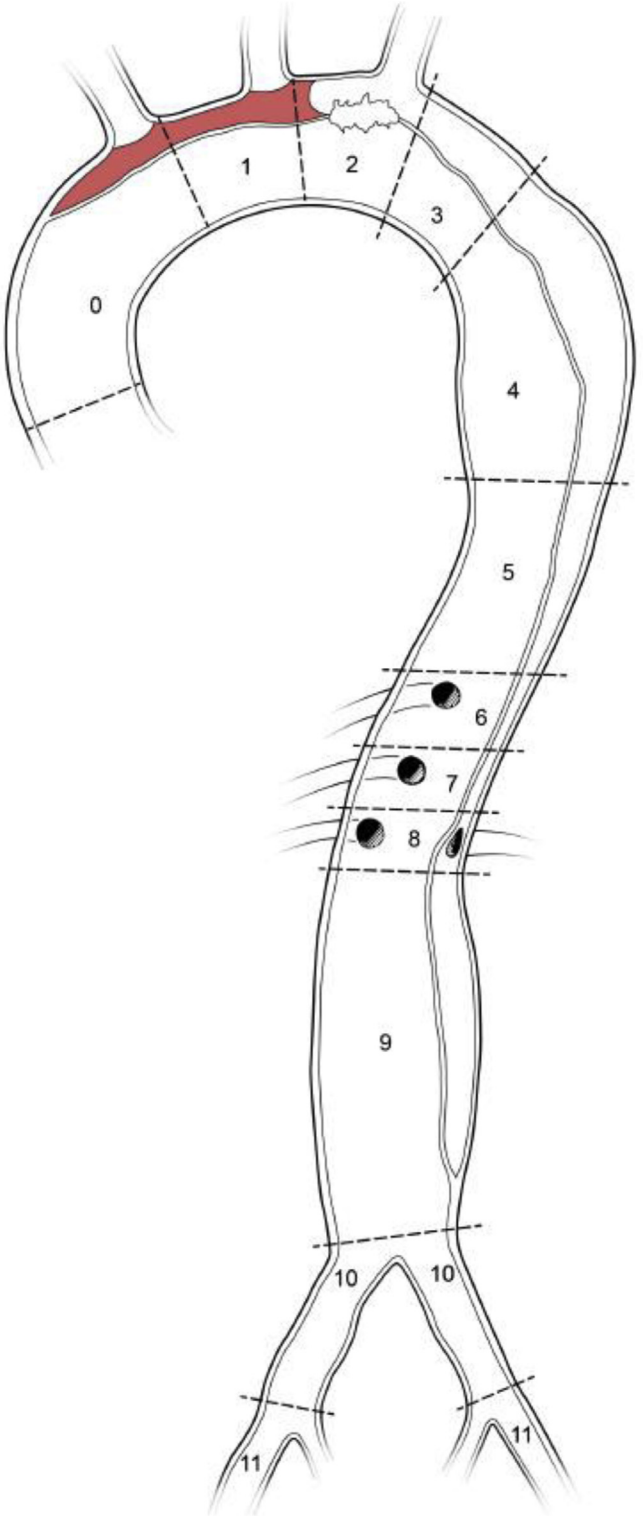

Fig 6. Example of an aortic dissection with an entry tear in zone 2, which classifies it as type B. The dissection process involves zone 0 proximally and extends distally to zone 9. This dissection is fully classified as $B_{0,9}$.

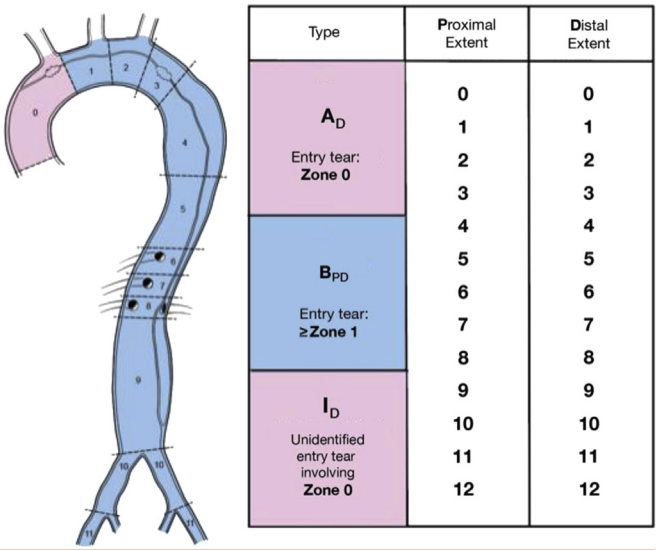

Fig 7. Society for Vascular Surgery/Society of Thoracic Surgeons (SVS/STS) Aortic Dissection Classification System.

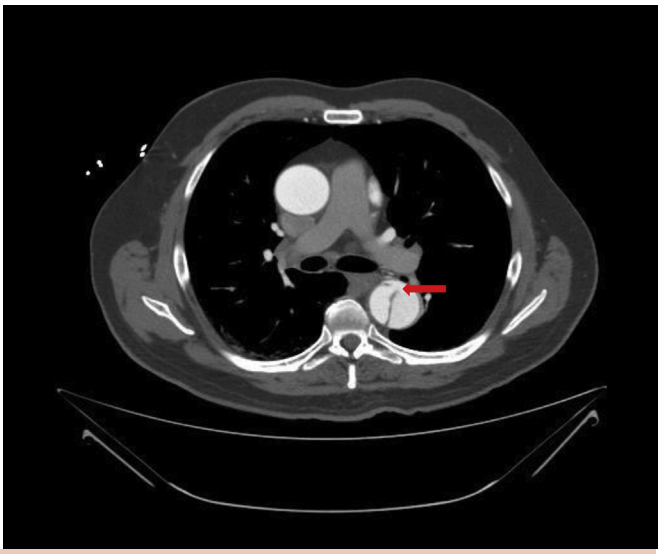

Fig 8. Type B aortic dissection (TBAD) with visualization of entry tear (*arrow*) in the descending thoracic aorta.

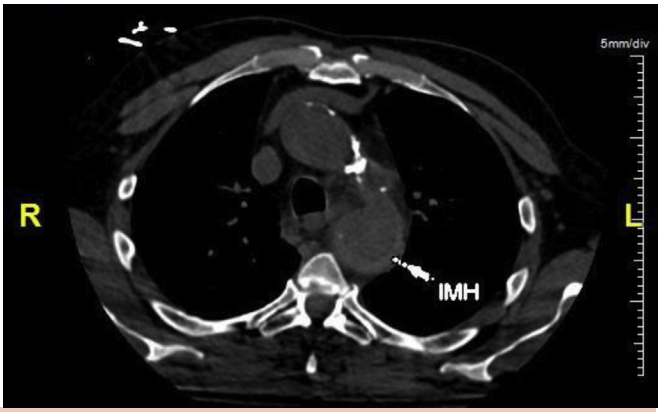

Fig 9. Noncontrast-enhanced computed tomography (CT) scan demonstrating intramural hematoma (*IMH*) of the descending thoracic aorta. Note that the IMH appears bright on nonenhanced imaging.

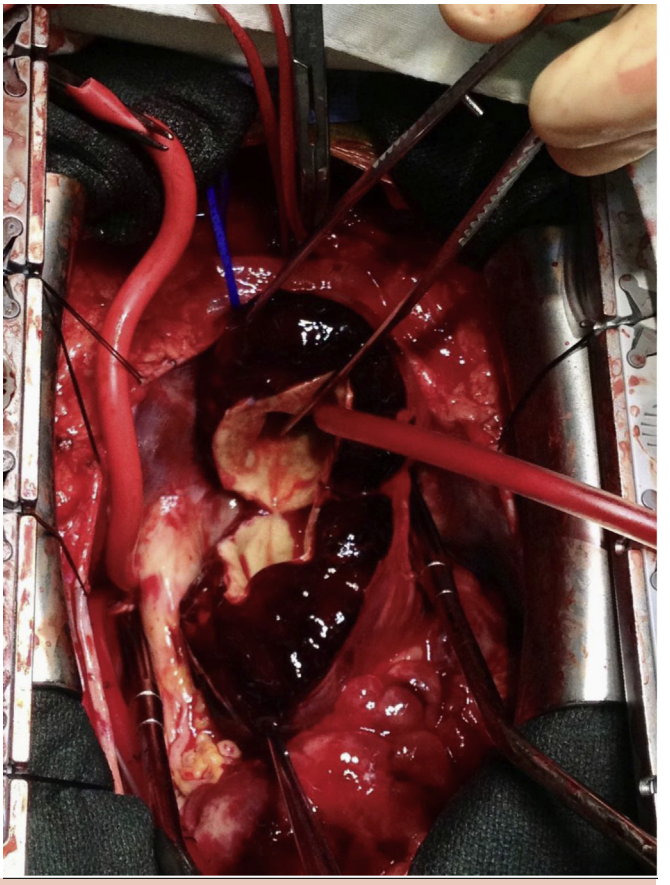

Fig 10. Example of the gross pathologic appearance of an intramural hematoma (IMH), in this case of the ascending aorta, as seen during central repair for acute type A IMH.

Dissection vs IMH. In addition to aortic dissection, two other distinct yet related acute aortic diseases require further description with respect to proper reporting: IMH and penetrating atherosclerotic ulcer (PAU). Aortic dissection is defined by the presence of a tear in the intima that results in a separation of the layers of the media and allows blood to flow through the false lumen (Fig 8). This separate, or false, lumen for blood flow is externally bound only by the outer third of the media and adventitia. IMH, in contrast, lacks an identifiable direct communication between the true and false lumens, a condition that authors in Asia have described as "closed thrombosed false lumen."[7] It is characterized by a hyperdense, crescent-shaped hemorrhage within the aortic wall best seen on noncontrast-enhanced computed tomography (CT) imaging (Fig 9).[8] Several mechanisms for IMH have been proposed, including spontaneous rupture of the vasa vasorum, which causes

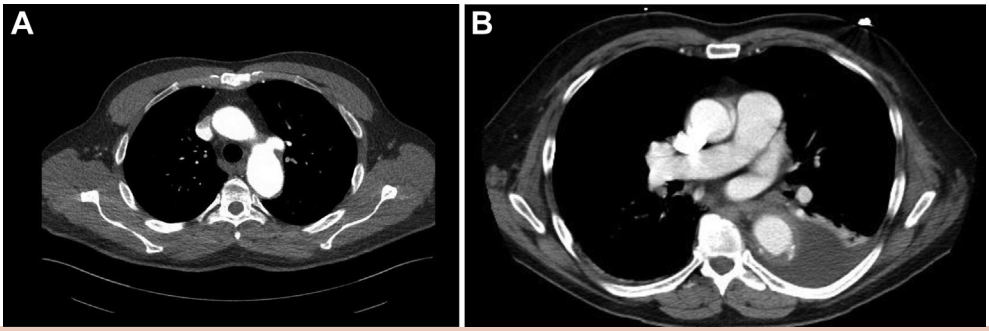

Fig 11. A, Penetrating atherosclerotic ulcer (PAU) without intramural hematoma (IMH). **B,** PAU with associated IMH and left pleural effusion.

bleeding and hematoma formation within the aortic wall (Fig 10).[9] Others have suggested microscopic tears in the intima as the potential cause.[8,9] Differentiating aortic dissection and IMH can be challenging, and the two conditions may coexist in the same patient. IMH can also progress to frank aortic dissection.

PAU is defined as an atherosclerotic lesion that penetrates the internal elastic lamina of the aortic wall and is often diagnosed in the presence of an IMH. PAUs are also referred to as ulcer-like projections, especially in Asia[7] (Fig 11, *A*). Approximately 20% of PAUs have no associated IMH, presumably because of medial fibrosis from chronic atherosclerotic disease.[9,10] PAU rupture risk is directly related to ulcer depth. However, PAU with IMH (Fig 11, *B*) has a higher risk of aortic rupture and portends a worse clinical course compared with a similar sized PAU without IMH.[11,12] It is important to distinguish between true aortic dissection and IMH in reporting as treatment algorithms and outcomes may differ significantly. The extent of IMH should be reported according to zone, as with aortic dissection, and reporting should include the maximal thickness of the aorta in the zone of IMH. PAU is best characterized by ulcer dimensions (saccular depth and diameter of aortic origin) and location by zone. When pathologic processes coexist with one another, we suggest using the predominant disease for classification purposes. Patients with multiple PAUs should be subscripted with their zone locations (eg, for PAU involving zones 3 and 5, $PAU_{3,5}$). If concomitant IMH is present, one would add subscripted proximal and distal extent, $IMH_{P,D}$, as described before. Thus, for a patient with IMH extending from zones 2 to 9 presenting with concomitant PAU in zones 3 and 5, it would be described as $IMH_{2,9}$ with $PAU_{3,5}$.

Chronicity classification of aortic dissection. The historical chronicity classification of aortic dissection originated from the investigations of Hirst et al,[13] who observed that mortality in patients with type A and type B aortic dissection significantly decreased after 14 days. Using this time point, the authors defined acute aortic dissection as ≤14 days from symptom onset and chronic aortic dissection as >14 days from symptom onset. Since this original report, there have been significant advancements in diagnostic imaging, medical treatment, and endovascular and surgical therapy and an improved understanding of the pathophysiologic mechanism of aortic dissection. Therefore, a reappraisal of this classification system is warranted.

A key motivation to re-evaluate the chronicity classification system is the application of endovascular therapy to TBAD. TEVAR was initially described in the treatment of TBAD in 1999 and has transformed the management of this disease during the past 20 years.[14] TEVAR, along with high-resolution CT scans and intravascular ultrasound, has afforded a more sophisticated understanding of dissection flap properties with respect to the chronicity of the dissection. In the acute phase, the dissection flap is thin and highly compliant, with a curvilinear appearance on CT scan. As the dissection flap ages, it becomes thicker and less compliant and has a straightened appearance on CT (Fig 12).[15] This enhanced understanding of dissection flap pathophysiology has implications for the classification of the chronicity of TBAD, and therefore any classification system should incorporate these more recent observations pertaining to intimal flap remodeling.

These lessons learned from contemporary reports in the endovascular era have prompted a reassessment of the traditional chronicity classification system. In a study similar to the initial work of Hirst, Booher et al[16] examined mortality from the time of symptom onset in 1800 patients (TBAD n = 655) from the IRAD database. In this cohort, Kaplan-Meier survival curves demonstrated distinct strata of mortality risk that varied by chronicity following presentation with aortic dissection. In addition, several studies examining various aortic remodeling outcomes in patients with TBAD treated with TEVAR at different time points have suggested the establishment of an additional subacute classification.[17,18] The subacute phase of TBAD was defined as 15 to 30 days by an SVS report on early outcomes after TEVAR for complicated

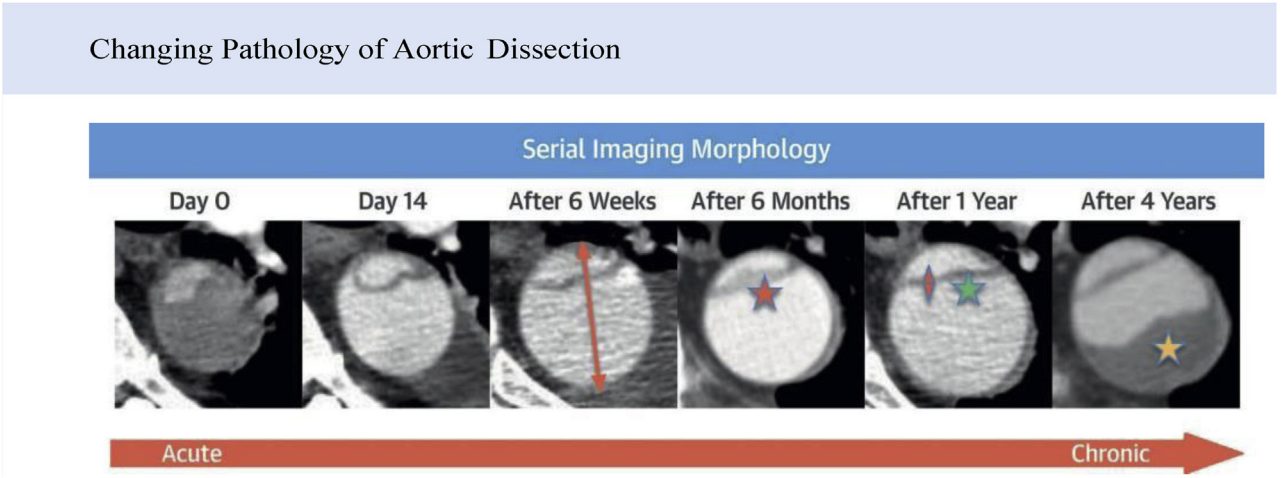

Fig 12. Evolution of aortic dissection flap morphology over time demonstrating transition from acute to subacute to chronic dissection. (From Peterss S, Mansour AM, Ross JA, Vaitkeviciute I, Charilaou P, Dumfarth J, et al. Changing pathology of the thoracic aorta from acute to chronic dissection: literature review and insights. J Am Coll Cardiol 2016;68:1054-65. Reproduced with permission.)

Table I. Society for Vascular Surgery/Society of Thoracic Surgeons (SVS/STS) chronicity classification of aortic dissection

Chronicity	Time from onset of symptoms
Hyperacute	<24 hours
Acute	1-14 days
Subacute	15-90 days
Chronic	>90 days

TBAD.[19] However, the most recent European Society of Cardiology guidelines on the diagnosis and treatment of aortic disease defined the subacute phase as 15 to 90 days.[20] In a study analyzing patients who underwent TEVAR within 3 months of the time of dissection, there was no significant difference in remodeling results of the thoracic aorta between those patients treated at <14 days and those treated between 15 and 90 days.[21] The cumulative data from these reports has led the Writing Committee to develop the following classification system of dissection chronicity, which incorporates both the IRAD and European Society of Cardiology findings: hyperacute, <24 hours; acute, 1 to 14 days; subacute, 15 to 90 days; and chronic, >90 days (Table I).

SECTION 3. CAUSES AND RISK FACTORS

Various causes and risk factors for acute TBAD have been noted in the literature, several of which influence decision-making and affect short- and long-term morbidity and mortality. Included here are the more common causes reported in studies on acute TBAD, the incidence of which among the study population should be described in any reports on TBAD.

Hypertension. Hypertension was present in 80.9% of patients who presented with acute TBAD in the IRAD database.[2] In previous reports summarizing the experience at a tertiary care center, 71% of patients with acute dissection were noted to have a history of hypertension,[22] whereas in more contemporary reports, 62% of patients with acute TBAD were taking antihypertensive medications at presentation.[23] However, the use of antihypertensive medications as a surrogate for the diagnosis of hypertension is almost certain to underestimate the true incidence of this risk factor in the TBAD population, given that TBAD is often a disease of the socioeconomically disadvantaged who are less likely to comply with risk factor modification.[24]

Patients who are reported as having hypertension should meet the definition according to the 2017 Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults,[25] which categorizes hypertension into two stages. Stage 1 is defined as systolic blood pressure of 130 to 139 mm Hg or diastolic blood pressure of 80 to 89 mm Hg. Stage 2 is defined as systolic blood pressure ≥140 mm Hg or diastolic blood pressure ≥90 mm Hg.

Genetically triggered aortic disease. Genetically triggered aortic diseases are due to mutations that affect the aorta and its branches and include disorders affecting mainly the aorta (nonsyndromic) as well as syndromic conditions (associated with abnormalities of other organ systems).[26,27] Many of the syndromic conditions are well-characterized connective tissue disorders (CTDs) with altered phenotypic tissue of multiple organs, including the cardiovascular system, leading to increased risk of aortic aneurysm formation and aortic dissection. Various syndromes have been described, with causative

genetic mutations identified for some. The most well known CTDs include Marfan syndrome (MFS), Loeys-Dietz syndrome (LDS), and Ehlers-Danlos syndrome.

Patients reported as having MFS should meet criteria of the revised Ghent nosology,[28] an expert panel's revision of the Ghent nosology from 1996 that outlines combinations of clinical features, family history, and genetic testing diagnostic of the disorder. Of note, the presence of an *FBN1* gene mutation is not, in and of itself, enough to establish the diagnosis. Reporting patients with CTD is important because these syndromes have been shown to mediate outcomes of aortic dissection. For example, in-hospital mortality of patients with MFS and acute TBAD is lower compared with that of patients without CTD. MFS patients also have better outcomes after open surgery for TBAD, although they have higher reintervention rates.[1]

LDS, as originally described, is characterized by heterozygous mutations in the type 1 or type 2 subunit of the transforming growth factor (TGF) β receptor. Patients may exhibit a clinical triad of hypertelorism, bifid uvula or cleft palate, and arterial tortuosity with ascending aortic aneurysm or dissection.[29] Emerging data suggest significant heterogeneity in the severity of aortic disease among patients with LDS, and patients with *TGFBR2* mutations (LDS type 2) appear to have more severe disease requiring aortic surgery at younger ages.[30] As such, reports of series including LDS patients should include specific details of the mutations present in each patient to allow comparison across studies. An expanded definition of LDS has also been proposed, as follows: type 1, *TGFBR1* mutation; type 2, *TGFBR2* mutation; type 3, *SMAD3* mutation; and type 4, *TGFB2* mutation.[31]

Vascular Ehlers-Danlos syndrome is caused by mutations in *COL3A1* gene encoding type III collagen. Whereas dissection or rupture typically occurs in medium-sized arteries, aortic involvement has also been reported.[29] Outcomes in this population are particularly poor, given severe vessel fragility that complicates any attempt at surgical repair, whether open or endovascular, and the median age of death is 48 years.[32]

Familial thoracic aortic aneurysm and dissection (FTAAD) represents a heterogeneous group with thoracic aortic disease predominating. Genetic mutations in *ACTA2* are most commonly identified (10%-14% of FTAAD), but mutations in *FBN1*, *TGFBR1/2*, and *MYH11* have also been found.[29]

In total, at least 29 genes have been identified to date that are associated with the development of thoracic aortic aneurysm and dissection, with many more likely to be discovered in the future.[27] As such, all reports pertaining to TBAD should include a description of the incidence of these disorders among the study population.

Congenital. Several congenital anomalies are associated with an increased risk of aortic dissection. Bicuspid aortic valve, the most common congenital cardiac malformation, occurs in up to 2% of the population and can be found in conjunction with genetic syndromes (eg, MFS, LDS, FTAAD) or in isolation. An increased prevalence of thoracic aortic aneurysm and dissection in patients with bicuspid aortic valve may have a genetic basis related to mutations in any one of several genes, including *NOTCH1*, *ACTA2*, *KCNJ2*, and *GATA*, but a unifying genetic mutation has not yet been identified.[33]

Coarctation of the aorta, a narrowing near the ductus arteriosus/ligamentum arteriosum, occurs in 6% to 8% of patients with congenital heart disease and is the most frequent congenital anomaly found in patients with Turner syndrome (45,XO). It is also associated with aortic medial degeneration, dilation or aneurysm of the aortic root and ascending aorta, and aortic dissection. Coarctation has been found in 2% of patients with aortic dissection,[34] and there are multiple case reports in the literature dating to the 1960s of coarctation and aortic dissection presenting simultaneously.

Kommerell diverticulum, the term given to aneurysmal dilation of the proximal segment of an aberrant subclavian artery, was first described by the radiologist Burckhard Kommerell in 1936.[35] He observed, on barium swallow study, the delayed passage of contrast material at the aortic knob with esophageal indentation by a pulsatile mass.[36] The diverticulum and aberrant subclavian artery can occur with either a left aortic arch with aberrant right subclavian artery (most common anomaly of the aortic arch with an estimated prevalence of 0.7%-2.0%) or a right aortic arch with aberrant left subclavian artery (estimated prevalence of 0.04%-0.4%). The literature contains multiple case reports and series of dissection occurring at the site of a Kommerell diverticulum.[37-41]

PAU. As noted earlier, PAUs were described by Stanson et al[42] as "an atherosclerotic lesion with ulceration that penetrates the internal elastic lamina and allows hematoma formation within the media of the [aortic] wall." PAUs are generally a manifestation of degenerative aortic disease and accordingly occur in older patients with the typical cardiovascular risk factors of hypertension, hyperlipidemia, and tobacco use.[43] Whereas such patients often present with an acute aortic syndrome, most commonly IMH as noted before, PAU may also be found incidentally on cross-sectional imaging in asymptomatic patients. Although PAUs were initially reported in association with IMH and rupture, PAU as a cause of aortic dissection was first described in 1995.[44]

Trauma. Several grading systems have classified blunt traumatic aortic injury into categories by presence of intimal tear, IMH, pseudoaneurysm, or free rupture.[45,46] Aortic dissection, as classically described with intimal

injury associated with a true and false lumen, may rarely result from blunt traumatic injury.

Iatrogenic aortic dissection as a result of open or endovascular procedures, including cardiac surgery,[47] TEVAR, transcatheter aortic valve replacement, and cardiac catheterization, occurs rarely according to data from the IRAD registry (2.3%).[2] With rare exception,[47] most descriptions of iatrogenic dissection are isolated case reports or small case series.[48-50] Optimal treatment strategies and long-term outcomes of iatrogenic aortic dissection have yet to be determined.

Illicit drugs. Cocaine-related aortic dissection, most commonly TBAD, has been implicated in 1.8% of patients with acute aortic dissection.[51] Amphetamine, methamphetamine, and MDMA/ecstasy use has also been reported in these patients. All reports of aortic dissection should include the demographic of patients with drug-related disease.

Ethnicity. Data also exist on differences in dissection subtype by ethnicity. For example, IRAD data found TBAD to be more common than type A aortic dissection in African Americans (52% vs 48%), which is different from other ethnicities, in which type A predominates (66%-75% type A vs 25%-33% TBAD).[52] As such, data regarding the ethnicity of the patient population should be included in the demographic section of all reports of aortic dissection.

SECTION 4. PRESENTATION

The initial clinical presentation of TBAD patients has major implications for both patient management and subsequent outcomes.[16] Precise definitions are therefore required for accurate documentation of the severity of the dissection, including presenting symptoms, comorbidities, and complications. This documentation is also important to allow comparisons of potential therapies for the various clinical presentations.

The most common presenting symptom of TBAD is pain, which is typically described to be abrupt in onset and tearing in nature. The location should be noted and may include chest, back, abdomen, or lower extremities.[53] The severity of pain should be reported with a verbal numeric rating pain scale (Table II).[54]

Hemodynamics at presentation can predict outcomes and complications from dissection and should be documented accordingly, including systolic and diastolic blood pressure and mean arterial pressure (MAP). The presence or absence of shock should be specified among the study population.

Comorbidities

Comorbidities can have significant implications for the care and outcomes of the dissection patient and should be graded accordingly. Congestive heart failure (new or previously diagnosed), chronic obstructive pulmonary disease, hypertension, chronic kidney disease, peripheral artery disease, cerebrovascular disease, and coronary artery disease should all be documented. Likewise, any surgical history associated with these conditions should be reported in detail, especially as it relates to prior structural cardiac interventions or coronary revascularization, as well as aortic anatomy. As mentioned before, genetically triggered aortic diseases, known or suspected, should be documented in detail along with family history. Supporting genetic testing results (ie, confirmed mutation) among those reported as having genetically triggered aortic diseases should be detailed as well.

Table II. Verbal numeric rating pain scale

Rating	Pain level
0	No pain
1-3	Mild pain (nagging, annoying, interfering little with ADLs)
4-6	Moderate pain (interferes significantly with ADLs)
7-10	Severe pain (disabling; unable to perform ADLs)

ADLs, Activities of daily living.

Uncomplicated dissection

An uncomplicated acute aortic dissection is defined as a dissection with no evidence of rupture or end-organ malperfusion. Uncomplicated dissection is further distinguished by the absence of high-risk features as designated in the following.

High-risk aortic dissection

Although dissections without overt malperfusion or rupture may not be immediately life-threatening, there are patients who fall into a category of high-risk uncomplicated dissection because of a significant risk of subsequent complications. These include both early complications, such as rupture in the subacute period, and late complications including aneurysmal degeneration. The high-risk group includes patients with refractory pain or hypertension and those with high-risk radiographic features.

Refractory pain and hypertension. Refractory pain and hypertension are two of the more difficult high-risk features for which to find consensus definition in the existing literature. Despite this lack of consensus, however, these clinical designations often drive acute intervention for dissection because of their link to poor short- and long-term outcomes.[55] Acute dissection is a painful condition, and alleviation of all pain should not be expected immediately. However, severe pain is concerning, and persistence of severe pain despite adequate blood pressure control, pain medications, and anxiolytic medications should place a patient into the high-risk category. Ongoing pain should be documented using the aforementioned numeric pain scale. Optimal blood

pressure control is defined as a normal blood pressure or whatever pressure is needed both to adequately perfuse the end organs and to resolve pain. Refractory hypertension is defined as hypertension persisting despite more than three different classes of antihypertensive medications at maximal recommended or maximal tolerated doses. Importantly, new-onset hypertension not present in the clinical history before the dissection may be a sign of renal malperfusion and should be investigated accordingly.[56] "Refractory" implies that there is a time limit to achieving the goal of pain or blood pressure control. Although there is an absence of clear outcomes-based data in the literature, the Writing Committee universally agrees that if either of these high-risk features persists for >12 hours despite maximal medical therapy, it may be considered refractory.

High-risk radiographic features. A number of radiographic findings have been associated with late aortic complications or need for intervention. Authors have suggested various diameters that would portend a high risk of late aneurysm formation or high rupture risk, with >40-mm maximal aortic diameter being an often-cited diameter.[57] Others have found that larger (>1 cm) primary tears, location of the tear (inner vs outer aortic curve), certain radiographic findings (eg, high Hounsfield units suggesting a bloody effusion on CT), and radiographic but not clinically apparent malperfusion may portend poor outcomes.[23,58,59] Importantly, this "radiographic malperfusion" of the renal or mesenteric beds is a vague finding that may be related to the CT angiography (CTA) phasing and should be interpreted with some caution. These radiographic findings should be documented in detail, and patients with these features should be described as high risk in reports of TBAD to allow standardized comparison of like groups between studies and to facilitate evaluation of long-term outcomes (Table III).

Table III. Aortic dissection acuity

Uncomplicated
No rupture
No malperfusion
No high-risk features
High risk
Refractory pain
Refractory hypertension
Bloody pleural effusion
Aortic diameter >40 mm
Radiographic only malperfusion
Readmission
Entry tear: lesser curve location
False lumen diameter >22 mm
Complicated
Rupture
Malperfusion

Complicated dissection

Rupture and malperfusion. Rupture is defined as extravasation of blood outside the confines of the adventitia of the aorta, which may be free or contained by the mediastinal pleura surrounding the aorta. Malperfusion is defined as inadequate blood flow to a tissue bed and is the most common reason for emergent intervention in TBAD. The most frequently affected tissue beds in the setting of acute TBAD are the renal, visceral, iliofemoral, and spinal circulations. Malperfusion can vary in severity from mild obstruction to complete occlusion and can be caused by either static or dynamic obstruction of the branch vessels.[60] Static obstruction can be caused by intussusception of the intimal flap into the lumen of the branch, thrombosis of the false lumen of a dissection extending into the branch vessel itself, or continuous pressurization of the false lumen in the aorta throughout the cardiac cycle, causing obstruction of the branch vessel origins (Fig 13, *A*).

Dynamic obstruction is due to changes in blood flow and pressure between the two lumens throughout the cardiac cycle. The dynamic forces lead to intermittent perfusion and obstruction by the intimal septum covering the vessel's orifice (Fig 13, *B*). Dynamic obstruction can be much more difficult to identify, especially on CTA, as the static nature of CTA does not permit evaluation of the septal dynamics or flow throughout the cardiac cycle. A physiologic study, such as gated CTA, intravascular ultrasound, or magnetic resonance imaging (MRI), may help delineate dynamic obstruction, given the ability to evaluate the septum throughout the cardiac cycle (see Section 7). Static or dynamic malperfusion should be documented whenever it is identified.

Renal malperfusion. Renal malperfusion may be clinically evident when both renal arteries are involved, leading to anuria and imaging demonstrating lack of vessel opacification. More often, unilateral radiographic malperfusion may be seen with one kidney appearing less opacified than the other. Importantly, although this may represent true malperfusion, the radiographic finding may simply be reflective of the phasing of CTA, typically when one of the renal arteries arises from the false lumen. Delayed venous- or renal-phase imaging can help delineate whether a kidney is being perfused as contrast material in the collecting system is indicative of at least modest perfusion. Radioisotope renography (ie, MAG3 scan) can also help demonstrate renal perfusion, as can renal ultrasound or dynamic MRI or magnetic resonance angiography (MRA).[61-63] Acute kidney injury alone is not sufficient to define renal malperfusion as a rise in serum creatinine concentration can occur after administration of contrast material, on cardiopulmonary bypass, or even

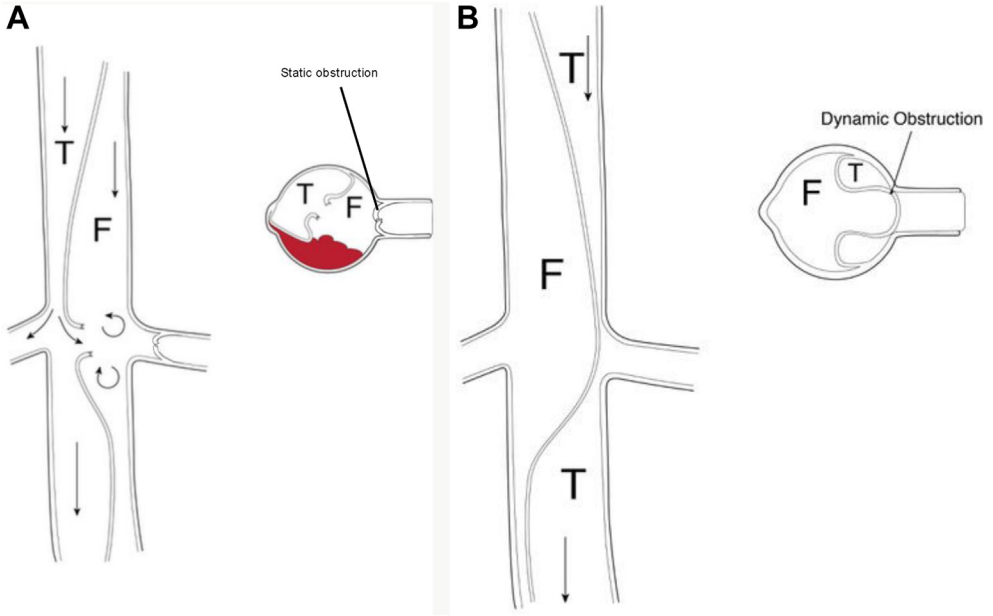

Fig 13. A, Malperfusion by static obstruction in a renal artery. The residual intima within the branch vessel occludes flow to the renal parenchyma. **B,** Malperfusion by dynamic obstruction in a renal artery. The pressurized false lumen (*F*) causes the intimal flap/membrane to cover the orifice of the renal artery, thereby impeding antegrade flow. *T,* True lumen.

with tight blood pressure control in patients with chronic uncontrolled hypertension.[63] Regardless of the true cause of renal impairment, the degree of injury at presentation should be graded in a similar manner to postoperative complications using the Acute Kidney Injury Network (AKIN) grading scheme (Table IV; see Section 6, Outcomes and Complications) if the patient's baseline renal function is known. Oliguria or anuria and elevated serum creatinine concentration are significant findings regardless of grade and should be documented.

Visceral malperfusion. Visceral malperfusion is one of the most dreaded complications of aortic dissection. Patients may present with varying degrees of visceral malperfusion, which should be documented for accurate risk modeling. Early visceral ischemia may be identified by radiographic only malperfusion, with poor opacification of the visceral vessels in the absence of clinical symptoms. Alternatively, early ischemia may have subtle symptoms, such as ileus or abdominal pain, usually out of proportion to physical examination findings, without associated laboratory abnormalities. As ischemia worsens, patients develop overt visceral ischemia with classic signs and symptoms, such as peritoneal signs (acute abdomen), bloody bowel movements, or worsening serum chemistries (such as elevated lactate, base deficit, and acidosis). These patients are deemed to have progressive visceral ischemia. The outcomes after early vs progressive (late) visceral ischemia can be clinically different and therefore should be distinguished in reporting.

Lower extremity malperfusion. As with other forms of distal malperfusion, lower extremity compromise can be manifested in varying degrees ranging from a diminished pulse to overt lower extremity mottling and acute limb-threatening ischemia.[64] As discussed in the physical findings (Section 4), the presence or absence of pulses should be noted. Other markers of malperfusion, such as motor and sensory neurologic deficits, skin mottling, pallor, pain, and temperature difference, should also be documented.

Stroke and spinal cord ischemia (SCI). Neurologic deficits should be described as partial or complete loss of motor or sensory function. Stroke, spinal ischemia, and extremity branch vessel malperfusion should be considered in dissection patients with extremity weakness, all of which may be present on admission, especially in the setting of uncontrolled hypertensive crisis.

When a stroke is identified on presentation, the severity of stroke should be graded with the modified Rankin scale (Table V) and documented before any intervention. The modified Rankin scale is a 7-point scale that has many advantages as a measure of stroke outcome. First, it includes the full spectrum of neurologic outcomes, from no symptoms (0) to death (6). Second, it is intuitive and readily grasped and applied by physicians and patients. Last, its validity is underscored by its agreement with existing stroke scales as well as its correlation with objective stroke data, such as infarct volume.[65] In documenting stroke, anatomic variants such as arch type, aberrant subclavian artery, or an anomalous origin of the left vertebral artery should be noted.

SCI may also be evident on presentation in patients with acute aortic dissection. The spinal cord has a highly

Table IV. Acute Kidney Injury Network (AKIN) classification/staging system for acute kidney injury[a]

Stage	Serum creatinine criteria	Urine output criteria
1	Increase in serum creatinine of ≥0.3 mg/dL (≥26.4 μmol/L) or increase to ≥150% to 200% (1.5- to 2-fold) from baseline	<0.5 mL/kg per hour for >6 hours
2[b]	Increase in serum creatinine to >200% to 300% (>2- to 3-fold) from baseline	<0.5 mL/kg per hour for >12 hours
3[c]	Increase in serum creatinine to >300% (>3-fold) from baseline (or serum creatinine of ≥4.0 mg/dL [≥354 μmol/L] with an acute increase of at least 0.5 mg/dL [44 μmol/L])	<0.3 mL/kg per hour for 24 hours or anuria for 12 hours

[a]Modified from RIFLE (Risk, Injury, Failure, Loss, and End-stage kidney disease) criteria. American Society of Nephrology Renal Research Report. J Am Soc Nephrol 2005;16:1886-1903.
[b]A 200% to 300% increase equals a twofold to threefold increase.
[c]Given wide variation in indications and timing of initiation of renal replacement therapy (RRT), individuals who receive RRT are considered to have met the criteria for stage 3 irrespective of the stage they are in at the time of RRT.
From Mehta RL, Kellum JA, Shah SV, Molitoris BA, Ronco C, Warnock DG, et al. Acute Kidney Injury Network: report of an initiative to improve outcomes in acute kidney injury. Crit Care 2007;11:R31.

variable blood supply, which consists of both extrinsic (derived from the anterior spinal artery, posterior spinal artery, and pial network) and intrinsic (central spinal cord arteries) collateral pathways.[66,67] Branches of the vertebral arteries combine in the foramen magnum and descend to the spinal cord as the anterior spinal artery, which supplies the anterior two-thirds of the spinal cord where the motor neurons reside. As the anterior spinal artery descends caudally away from the cervical portions of the spinal cord, its caliber often diminishes or becomes discontinuous, and there is increased reliance on collateral flow derived from the intercostal and lumbar arteries.[68,69] The majority of intercostal arteries do not supply the spinal cord, however, but rather the nerve roots. The most important extrinsic artery, the arteria radicularis magna, or artery of Adamkiewicz, is the predominant blood supply to the anterior spinal artery at the level of the thoracolumbar spinal cord and usually arises from the left side of the aorta between T8 and L2. The artery may be identified on preoperative CTA imaging. Distally, a robust network is provided by the middle sacral, lumbar, and, most important, internal iliac arteries; the internal iliac arteries are important contributors to the spinal cord collateral network, which may develop in the presence of occlusion of extrinsic segmental arteries.[68]

SCI may result from loss of critical perfusion from the extrinsic pathway, by interruption of the artery of Adamkiewicz, but it can be further exacerbated by the disruption of collateral flow through the spinal cord collateral network by the subclavian and internal iliac arteries. Spinal cord ischemic injury can be lateralizing but usually involves both lower extremities and may include loss of bowel or bladder function. Documentation of the severity of these deficits should be specific. The modified Tarlov scoring system (Table VI) is a useful grading scheme for SCI as it encompasses the full spectrum of injury.[63,70] The scoring system includes varying degrees of paraplegia, including flaccid paraplegia, in which there is no lower extremity movement (score of 0), lower extremity movement without gravity (score of 1), and lower extremity movement with gravity (score of 2). Paraparesis is designated by scores 3 and 4, in which the patient is able to stand with assistance and walk with assistance, respectively. Normal strength is assigned a score of 5. A Tarlov score should be documented for all patients with SCI at presentation, after treatment, on discharge, and in follow-up.

As discussed before, branch vessel malperfusion may cause extremity ischemia, which can be manifested as a neurologic deficit. Malperfusion symptoms are often lateralized, especially in the upper extremities, where left subclavian involvement is more common than right subclavian involvement. In the lower extremities, however, malperfusion causing neurologic deficits may be present in one or both extremities.

Escalation of care. Changes in patient status are important for accurate reporting of outcomes of initial management. Patients with evolution of aortic involvement, such as longitudinal extension of the initial extent of dissection, either retrograde or antegrade, should be reclassified as dissection with progression. The timing of this reclassification, including its relationship to any form of management, should be documented.

Any patient readmitted to the hospital, beyond observation, within 30 days of initial presentation is considered a readmission. The reason for readmission should be detailed along with the need for intensive care unit admission or intervention. All initially uncomplicated patients who are readmitted for recurrent dissection-related reasons (eg, pain, severe hypertension) after institution of medical therapy should be reclassified as high risk. These patients may ultimately be adequately managed with medical therapy; however, documenting readmissions for failed medical therapy will ultimately grow our understanding of the disease process. Only with effective documentation will we be able to discern those patients who can truly be observed from those who should be slated for early intervention.

Table V. Stroke severity: modified Rankin scale

The scale runs from 0-6, running from perfect health without symptoms to death.
0. No symptoms.
1. No significant disability. Able to carry out all usual activities, despite some symptoms.
2. Slight disability. Able to look after own affairs without assistance, but unable to carry out all previous activities.
3. Moderate disability. Requires some help, but able to walk unassisted.
4. Moderately severe disability. Unable to attend to own bodily needs without assistance, and unable to walk unassisted.
5. Severe disability. Requires constant nursing care and attention, bedridden, incontinent.
6. Dead.
From Broderick JP, Adeoye O, Elm J. Evolution of the modified Rankin scale and its use in future stroke trials. Stroke 2017;48:2007-12.

Table VI. Spinal cord injury: modified Tarlov scoring scale

Scale	Motor function	Deficit
0	No lower extremity movement	Paraplegia
1	Lower extremity motion without gravity	Paraplegia
2	Lower extremity motion against gravity	Paraplegia
3	Able to stand with assistance	Paraparesis
4	Able to walk with assistance	Paraparesis
5	Normal	Normal

From Chiesa R, Melissano G, Marrocco-Trischitta MM, Civilini E, Setacci F. Spinal cord ischemia after elective stent-graft repair of the thoracic aorta. J Vasc Surg 2005;42:11-7.

Given the dynamic nature of acute dissection, any and all complications that might be seen at initial presentation of TBAD can likewise subsequently occur at any time during the early hospitalization or after discharge. Any changes from presentation and escalations in care should be noted. This is particularly important for patients with a stable uncomplicated presentation who later develop a transition to a high-risk or complicated scenario. Patients returning after any initial treatment strategy with a high-risk or complicated presentation should be documented as a treatment failure.

SECTION 5. INITIAL MANAGEMENT STRATEGY

Medical management. The institution of prompt medical therapy is critically important and should be a commonality among all presentations. Criteria for optimum medical management (eg, blood pressure and heart rate targets, urine output) should be outlined in the Methods section of any reports. Strategies for achieving these goals, including anti-impulse medical regimens and pain control, should be outlined. As discussed previously, failure to achieve the predefined parameters of medical optimization within 12 hours should be considered initial treatment failure and escalate the patient to a high-risk category.

TEVAR. When TEVAR is used as a management strategy, reporting is performed in cohorts of indications, usually centered around patient status (uncomplicated vs complicated vs high risk). The specifics of procedural planning, including endograft diameter, percentage device oversizing, length of coverage, and use of tapered devices, must be documented. Adequacy of landing zones should likewise be documented as all currently FDA-approved dissection devices call for normal undissected aorta at the proximal seal zone. Unhealthy proximal landing zone has been shown to affect long-term outcomes, and the extent of proximal disease should be documented according to zone.[71] The mode of fixation should be documented as well in terms of radial force fixation, proximal barbs, or open proximal or distal bare spring stent design. Finally, access site, conduit use, and device deployment direction (proximal to distal or distal-first technique) are important details that should be documented.

Other procedural data should include the use of intravascular ultrasound, postimplantation balloon dilation, adjunctive bare-metal stents, intentional (Knickerbocker and stent-assisted balloon-induced intimal disruption and relamination in aortic dissection repair [STABILISE] techniques) or nonintentional (stent graft-induced new entry [SINE]) septal disruption, and use of false lumen embolization or occlusion techniques (eg, candy-plug technique, coils, occluder devices). Procedural complications, such as aortic rupture, vascular injury, retrograde dissection, dissection of branch vessels, or occlusions and septal tears, are not infrequent and require documentation.

Branch vessel management. In reports of TEVAR-based management of TBAD, authors should include details of management of the arch branch vessels if zone 2 or more proximal aortic arch coverage is required. Methods of revascularization should be delineated and include extra-anatomic bypass (eg, carotid-carotid bypass, carotid-subclavian bypass, subclavian-carotid transposition, ascending aorta-based debranching), use of commercially available or investigational branched or fenestrated endografts, and parallel stent grafts. One should also document the use of physician-modified grafts. Timing relative to TEVAR is also useful in correlating neurologic outcomes.

Adjuncts vs reintervention vs planned reintervention. Other revascularization procedures may be adjunctive to TEVAR for TBAD, such as mesenteric artery revascularization, iliac stenting, or femorofemoral bypass. It should be made clear whether these were done pre-emptively (ie, the decision to perform the procedure was made before aortic repair) or after aortic repair for persistent malperfusion. Additional procedures during the index TEVAR operation should be labeled as adjunctive procedures, such as distal bare-metal stenting with or without ballooning, visceral stenting, or any other open revascularization. Reoperation separate from the index TEVAR procedure should be described as either unplanned reintervention or planned reintervention. Unplanned reintervention implies repairing a complication of the index procedure, failure of the devices, or progression of the dissection process resulting in malperfusion, growth, or recurrent symptoms. Planned reintervention, on the contrary, is either a strategic delay or completion of the index procedure in a staged fashion.

Open surgical repair. Reporting for patients receiving open repair should be centered around the indications, specifics of the dissection process relative to chronicity, surgical approach, and anatomic extent of repair. Specifically, management of major aortic branch vessels, such as the arch vessels, visceral branches, iliac arteries, and hypogastric vessels, should be documented, such as use of an island patch, individual branch grafts, or extra-anatomic bypass. The extent of open aortic replacement should also be specified using the defined zones of the aorta. Types and diameters of used surgical grafts should likewise be specified. Organ protection techniques should be detailed, including cannulation sites, use of full cardiopulmonary bypass with hypothermic circulatory arrest and details of systemic temperature management, partial left-sided heart bypass with distal aortic perfusion, adjunctive visceral organ perfusion techniques (crystalloid, blood, flow rates, temperature of perfusate, techniques of delivery), and reimplantation of intercostal and lumbar arteries (location and numbers of vessels reimplanted or revascularized). Perioperative hemodynamic management protocols with specifics of target blood pressures should also be detailed.

Spinal ischemia. Authors should report any maneuvers used to mitigate SCI. As noted before, for open repairs, this will include whether intercostal or lumbar arteries were reimplanted as well as details of number and location of any reimplanted vessels. Placement of cerebrospinal fluid (CSF) drains should be documented along with timing, including emergency or elective placement status, and drainage protocols. Authors should describe whether intraoperative neurophysiologic monitoring, including electroencephalography, somatosensory and motor evoked potentials, and cerebral oximetry, was used. Any changes in neurophysiologic monitoring as well as management provoked by those changes should be documented. Specific blood pressure protocols for intraoperative and postoperative spinal cord protection should also be documented in the Methods section of any reports. Likewise, any adjunctive medical management used to mitigate SCI, such as steroids or naloxone, for example, should be detailed.

Metachronous disease. Lesions that are not related to the initial aortic dissection and are separated by any length of normal aorta should be considered metachronous. Subsequent treatment of such lesions should not be considered a complication of the initial dissection management. One example of this could be a survivor of acute TBAD with an ascending aortic aneurysm. However, any new dissection that is continuous with the initial dissection is considered synchronous and should be listed as progression of disease (ie, new acute TBAD superimposed on a chronic medically managed dissection; see Section 2) or a failure of the initial management strategy (ie, retrograde extension of a dissection after TEVAR).

SECTION 6. OUTCOMES AND COMPLICATIONS

Thirty-day/in-hospital mortality. All deaths occurring within 30 days of symptom onset, 30 days of the index procedure, or during the index hospitalization should be referred to as early dissection-related death. These data should be gleaned from the hospital chart, outpatient follow-up record, or publicly available records including the National Death Index and Social Security Death Index. Additional attempts, including patient family member or medical provider contact, can also be considered if reasonable. Details about determination of the mode of death including autopsy, operative, or radiographic findings consistent with aorta- or non-aorta-related death should be noted. Likewise, the cause of death should be listed as indeterminate for cases in which cause cannot be determined.

Stroke and major adverse cardiovascular events. Stroke is defined as a focal or global neurologic deficit lasting for >24 hours. The Writing Committee recommends use of an objective scoring system to quantitate the degree of functional limitation associated with any stroke and to enable a more accurate representation of how this complication has affected the patient. The modified Rankin scale (Table V), as discussed earlier, should be used to document stroke severity. Strokes occurring within 30 days of surgical intervention (TEVAR or open repair) will be considered procedure related. Strokes occurring within 30 days of medical management will be considered dissection related. It is also important to note deficits that resolve vs those that

persist, at which time a modified Rankin scale score should be repeated to quantitate the degree of recovery.

Other major adverse cardiovascular events occurring within 30 days of dissection onset in the case of medical management or 30 days of surgical intervention should likewise be reported, as should a composite of major adverse cardiovascular events, which includes myocardial infarction, stroke, and death.

Imaging for stroke. In cases in which imaging is obtained, evidence of new cerebral infarct or hemorrhage on CT or MRI, even in the absence of discernible neurologic deficit, also indicates the presence of acute stroke.[65,72] Efforts should be made to separate ischemic from hemorrhagic strokes as subsequent treatment schemes will differ.[73,74] Ischemic strokes are due to infarction of central nervous system tissue, whereas hemorrhagic infarcts are characterized by intraparenchymal, intraventricular, or subarachnoid hemorrhage. Further characterization of strokes radiographically can lend insight into the mechanism of stroke. This holds importance in attributing stroke to an embolic or watershed etiology. The delineation of anterior vs posterior strokes as well as unilateral vs bilateral also lends insight into the pathophysiologic mechanism of stroke as the vascular distribution implicates different vessels (carotid vs vertebral artery) and causes. For example, occurrence of a unilateral posterior circulation stroke after left subclavian coverage without revascularization during TEVAR may indicate posterior circulation hypoperfusion. However, bilateral posterior circulation stroke in the same setting may indicate an embolic cause related to arch manipulation and not affected by the presence or absence of subclavian revascularization.[75] Stroke distribution has also been shown to have an impact on mortality. Previous studies have shown that posterior strokes have higher rates of morbidity and early mortality than anterior strokes.[76] As noted before, laterality should be noted as well, and as branched devices are being developed, studied, and used, these anatomic details will become increasingly important.

After the diagnosis of stroke, any additional imaging performed to rule out potential causes of stroke, such as imaging of the carotid arteries or echocardiography to rule out a cardiac source (eg, intracardiac thrombus or patent foramen ovale with or without atrial septal aneurysm), should be documented.

Acute kidney injury. The definition and classification scheme for acute kidney injury should encompass any decline in kidney function as well as the need for dialysis, whether transient or permanent. As noted before (Section 4, Renal Malperfusion), the etiology of renal dysfunction in patients with aortic dissection is multifactorial and can include renal malperfusion, contrast nephropathy, the sequelae of medical anti-impulse therapy in patients with chronic hypertension, and possible embolization. The aforementioned AKIN grading scheme,[77] a consensus classification developed by intersocietal collaborations between nephrology and critical care, consists of three stages that take into account changes in serum creatinine concentration as well as urine output: stage 1, increase in serum creatinine concentration 1.5- to 2-fold; stage 2, increase in serum creatinine concentration 2- to 3-fold; and stage 3, serum creatinine concentration increase >3-fold or anuria for ≥12 hours (Table IV). In instances in which serum creatinine concentration is unknown at baseline, urine output should be the primary determinant of grade using AKIN. In addition, it should be noted whether there is a need for dialysis and whether this need is temporary in-house only, present at discharge, or permanent on follow-up.

SCI. Multiple causes of SCI have been identified after aortic dissection (see Section 4, Stroke and Spinal Cord Ischemia), including sequelae of the dissection itself and its treatment. Because of the variability of the collateral network in the thoracolumbar spinal cord, endovascular coverage or surgical sacrifice of intercostal or lumbar arteries can cause SCI by compromised flow in watershed areas. Because SCI has also been noted in individuals with patent radicular arteries, the importance of perfusion pressure in the development of SCI should be noted as well.

Spinal cord perfusion pressure (SCPP) is defined by the equation SCPP = MAP − CSF pressure (or central venous pressure, whichever is higher). SCPP in patients suffering SCI should be documented, if feasible, as well as any attempts to manipulate SCPP by raising the MAP or lowering the CSF pressure or central venous pressure. Another cause of SCI is embolization to segmental arteries supplying the spinal cord. Patients with SCI can have varying presentations as described in Section 4, with variable severity, onset, and potential for recovery. Time at onset should be noted as this could implicate a dissection-related vs early perioperative (extent of endovascular coverage, surgical ligation, or embolization) vs delayed perioperative cause, where hemodynamic instability in a patient initially neurologically intact after surgical repair could be the cause.

The modified Tarlov scoring system as discussed in Section 4 (Table VI) is a useful grading scheme for SCI and includes scores of 0 to 5, which encompass the full spectrum of SCI.[78,79] Any degree of SCI related to either the index dissection or its management should be documented using this system. It is also important to note whether there is improvement in functional status at the time of discharge or in late follow-up. Patients with SCI with subsequent improvement at discharge have been shown to have lasting neurologic recovery.[80] Consequently, a Tarlov score should also be documented for all patients with SCI at the time of discharge and in late follow-up.

Bowel ischemia. Bowel ischemia encompasses both mesenteric ischemia and ischemic colitis. Mesenteric ischemia is a clinical diagnosis marked by symptoms, rising lactic acidosis, and radiographic or intraoperative findings consistent with bowel ischemia or necrosis.[19] A high index of suspicion for persistent malperfusion after treatment should be entertained in the presence of ongoing clinical symptoms, continued true lumen compression, or lack of opacification of the mesenteric vessels on postoperative imaging. Unlike mesenteric ischemia noted on presentation, which is categorized temporally as early or progressive (see Section 4, Visceral Malperfusion), continued mesenteric ischemia after the procedure can be categorized according to required intervention. In mild cases, no intervention is necessary, although the patient may have an ileus or difficulty in tolerating oral intake. In severe cases, bowel resection may be required. If a mesenteric stent or further adjunctive procedures are required, these should be documented.

Colonic ischemia should similarly be diagnosed on the basis of the presence of symptoms in addition to lactic acidosis corroborated with radiographic or intraoperative findings consistent with colonic ischemia or necrosis. In addition, because endoscopic evaluation is available to interrogate the colon, a grading scale has been developed to denote the severity of ischemia.[81] Grade I is defined as mucosal ischemia; grade II includes extension to the muscularis propria; and grade III includes transmural ischemia, gangrene, or perforation. The need for colon resection and the extent (segmental colectomy vs total colectomy) should also be noted.

Rupture. As noted in Section 4, aortic rupture is defined as hemorrhage outside of the boundaries of the aorta and thus is diagnosed on the basis of radiographic imaging. Rupture may be contained by surrounding structures or present as free rupture, marked by hemodynamic instability. Aortic rupture occurring early, within 24 hours of index dissection presentation or surgical repair, should be distinguished from that occurring in a more delayed fashion. Therefore, the Writing Committee recommends a classification of peridissection or periprocedural rupture (<24 hours from index presentation in medically managed patients or <24 hours from endovascular or surgical repair), early rupture (1-30 days from index presentation or repair), and delayed rupture (>30 days from index presentation or repair).

Dissection propagation. A retrograde dissection is defined as any new ascending, arch, or descending dissection contiguous with and proximal to the original presenting anatomy. Because multiple causes have been implicated, including poor control of blood pressure in medically managed patients and procedure-related causes such as wire manipulation, radial force from an excessively oversized stent graft, ballooning, or trauma from a proximal bare stent,[82,83] it is important to note the time at onset and likely cause. Unless intraoperative transesophageal echocardiography reveals unexpected proximal (zone 0) disease immediately before a procedure, any proximal change noted thereafter must be considered procedure related. Proximal propagation or retrograde dissection is an anatomic change compared with the presenting anatomy, and the classification should be described as disease related, early (<30 days from procedure or presentation) or late (>30 days from procedure or presentation).

An antegrade dissection is one that is not present on baseline imaging and extends distally after medical management or endovascular or surgical repair. Antegrade dissection should also be described as disease related, early (<30 days from procedure or presentation) or late (>30 days from procedure or presentation).

SINE. SINE is a new tear caused by the stent graft itself, excluding those created by natural disease progression or any iatrogenic injury from endovascular manipulation.[84] These tears may be proximal, leading to pseudoaneurysm formation or retrograde dissection, or distal, resulting in false lumen pressurization and expansion by type IB entry flow to the false lumen (see Section 7). The reported incidence of SINE after TEVAR varies but may be as high as 25%,[84,85] with distal SINE being more common and representing up to 80% or more of SINE in some series.[86] SINE tears are typically delayed in occurrence, with most developing approximately 12 to 36 months after TEVAR. They are usually asymptomatic and discovered on routine postoperative surveillance imaging.[87,88] SINE tears appear to be most common after TEVAR performed for a chronic dissection indication, and the most important risk factor for distal SINE appears to be excessive oversizing of the distal stent graft relative to the smaller true lumen in the chronic setting[85,89] (Fig 14). Determining the location of the aortic injury in relation to the thoracic endograft is paramount in attributing the new entry tear to the device. Distal extension of the dissection or development of an entry tear that is separated in space from the endograft should not be attributed to the device. The timing of the SINE should be noted and reported as either early (≤30 days) or late (>30 days).

Long-term outcomes. Consistent with the reporting standards for TEVAR developed by the SVS,[3] all deaths occurring after 30 days and after the index hospitalization should be classified as late deaths. The cause of late death and its relationship to the dissection event, device, or procedure should be noted (dissection related, nondissection related, or unknown as described in Section 6, Thirty-Day/In-Hospital Mortality) whenever possible. Not all aorta-related deaths resulting from retrograde dissection propagation and aortic rupture are related to the initial dissection. The cause of death may be verified by autopsy or intraoperative findings or based on radiographic imaging. When this information

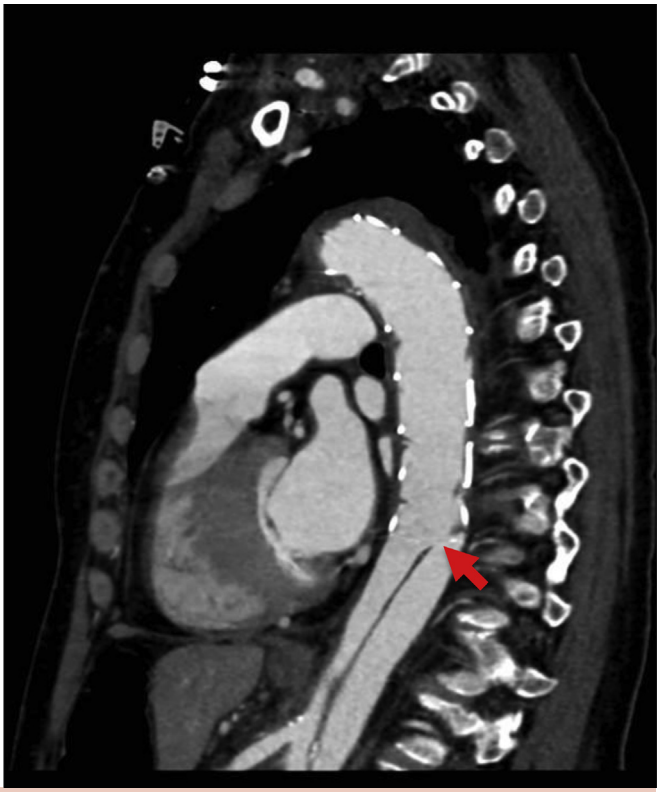

Fig 14. Stent graft-induced new entry (SINE) tear. Note the tear (*arrow*) in the intimal flap just beyond the distal aspect of the stent graft. The SINE creates a fenestration perfusing the false lumen. This is also described as type IB entry flow.

is unavailable, the cause should be denoted as probable if the clinical picture is suggestive of and consistent with a device- or procedure-related cause. The cause of death is labeled indeterminate if these criteria are not met. Accordingly, Kaplan-Meier and life-table analyses should distinguish between all-cause and dissection-related mortality.

SECTION 7. FOLLOW-UP IMAGING

More than 60% of patients with aortic dissection, regardless of initial treatment modality, will develop aneurysmal growth during the next 5 years.[90] The importance of surveillance in this population of patients therefore cannot be overstated. Patients with aortic dissection should be treated as any patient with a chronic disease is treated and require lifelong follow-up by specialists with expertise in the management of aortic disease. Radiology studies obtained should report the result of the initial management chosen, describe any changes in relation to baseline and subsequent scans, and report the presence of any new pathologic change (ie, new malperfusion, entry flow, or aneurysm).

In the absence of contraindications, CTA is the imaging modality of choice, given its sensitivity for the detection of false lumen entry flow and changes in aortic or aneurysm diameter, evaluation of false lumen thrombosis, and assessment for device migration and integrity.[91,92] All follow-up imaging studies should be performed as multiphasic CTA, which includes nonenhanced, arterial-phase, and delayed images.[92-95] The initial nonenhanced images permit the identification of high-attenuation material, such as calcification or Teflon felt from prior open surgical repair, which appears bright and may mimic entry flow on contrast-enhanced images; nonenhanced imaging is also superior for the detection of IMH.[94] Delayed-phase images are necessary to identify stagnant yet persistent false lumen flow. A delayed phase can depict sluggish entry flow that is not visualized during the initial arterial phase,[95-97] suggesting that a triple-phase protocol CT may be a better assessment of the patency of false lumen[94]; delayed imaging may also detect some entry flow not apparent on arterial-phase imaging in patients status post TEVAR. Some have suggested that a delayed phase of 300 seconds is optimal for detection of false lumen flow.[94-96]

Follow-up CTA imaging should report the status of the true and false lumens, abnormal zones of the aorta, any aneurysmal dilation, and presence of entry flow relative to any implanted endografts. It is necessary to document the status of the entire aorta as the dissected aorta may remain stable, show progression of dissection, dilate, or heal. The changes that occur within the aorta should be routinely evaluated on a schedule that may differ from patient to patient but can coincide with a 30-day, 3- or 6-month, and yearly schedule after the index dissection event or surgical intervention; in addition, follow-up imaging may be extended to 18- to 24-month intervals in some patients with stable findings >5 years removed from the index dissection event or repair.[98] Regardless of the interval, all follow-up aortic imaging should include observations on dissection extent, false lumen patency status, presence or absence of aortic growth with specific diameter measurements, aortic remodeling, and source of any persistent entry flow into the false lumen.

Dissection extent: Healing or progression of dissection. The zones of aorta involved with the dissection process may contain a combination of patent, partially thrombosed, or completely thrombosed false lumen. The appearance of hematoma contiguous with the dissection process should be considered anatomically part of the dissection. A comparison of the longitudinal extent of dissection should be made with the initial presenting imaging, and any additional propagation or resolution of dissected aorta should be noted. For example, using the SVS/STS classification, a patient may have presented with a $B_{1\text{-}8}$ dissection, but on follow-up imaging, the proximal involvement no longer involves zone 1 because of interval resolution of associated contiguous aortic hematoma in this segment. This reduction in extent should now be described as $B_{2\text{-}8}$.

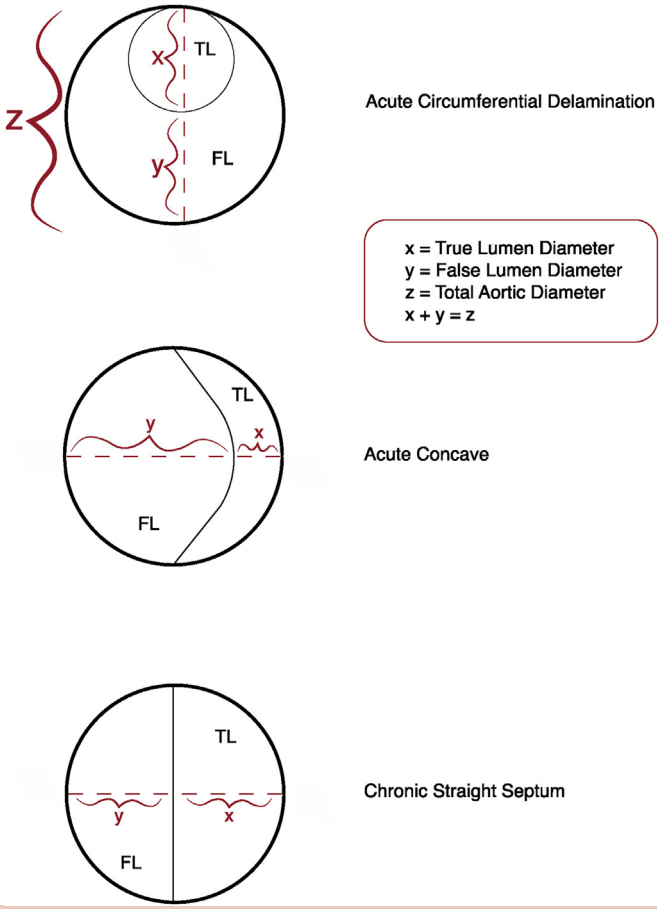

Fig 15. Luminal configurations of aortic dissection. Measurements of true lumen (*TL*) and false lumen (*FL*) diameters, as shown, should be based on a single line bisecting the center of the intimal flap perpendicular to flow. TL (*x*) and FL (*y*) measurements should add up to the total (*z*) aortic diameter.

False lumen patency status. False lumen patency or thrombosis is an important predictor of regional luminal growth and reintervention rate.[99,100] When describing the status of the false lumen, one should document as follows:

Patent: defined as flow present throughout the *entire* aortic false lumen on arterial-phase or delayed contrast-enhanced imaging.
Partial thrombosis: defined as clot within the aortic false lumen but with a residual patent flow channel on arterial-phase or delayed contrast-enhanced imaging.
Complete thrombosis: defined as complete thrombosis of the aortic false lumen on arterial- and delayed-phase imaging.

In describing false lumen changes in published reports, the entirety of the dissection process must be documented, including the thoracic and abdominal aorta and iliofemoral arteries. Representative zones may be described; however, reporting describing only the status of the false lumen within treated segments does not take into account how TEVAR influences downstream untreated segments of the aorta and can be misleading. Therefore, one must be specific in describing which zones fall under the aforementioned descriptions, and reporting must be complete with respect to the entire aorta or dissection extent. When reported end points include the status of false lumen flow after TEVAR, the Methods section of such reports should contain details of the imaging protocols used to ensure that the proper observations have been made.

Aortic growth. Follow-up total aortic diameter should be reported on the basis of CTA or MRA imaging studies. In select cases, such as patients >5 years removed from the index dissection event and with aortic diameters well below the thresholds recommended for intervention, plain CT without contrast enhancement may be adequate for assessing total aortic diameter, although the lack of administration of contrast material limits the precision of obtained measurements and precludes any assessment of false lumen status. In reporting aortic growth, the percentage of patients with total aortic growth, defined as an increase ≥5 mm in maximal aortic diameter in *any* segment after any form of management (eg, medical, surgical, TEVAR), must be described. Reporting changes in mean aortic diameter across a population of patients is often misleading as it allows positive aortic remodeling with diameter regression in one segment (eg, in the treated thoracic segment after TEVAR) to offset aortic growth in other segments, thereby masking the actual rate of aortic growth within the population.[101] Reporting of aortic growth should thus accurately describe the number and percentage of patients in a population who demonstrate growth and the distribution of zones where growth is observed.

Aortic measurements. Diameters should be measured from outer aortic wall to outer aortic wall, such that the measurements are consistent throughout the study population. Aortic diameters should be measured perpendicular to the angle of blood flow (centerline technique). Likewise, aortic diameters should be recorded at levels of interest, such as zones adjacent to the primary tear, zones containing the largest aortic diameters in both the thoracic and abdominal aorta, and treated and downstream segments in patients status post TEVAR. It can be difficult to obtain accurate measurements of the dissected aorta as the aortic shape is often noncircular. To obtain accurate and reproducible total aortic diameter measurements, one should use a straight line bisecting the center of the intimal flap and perpendicular to the plane of blood flow such that the combined true and false lumen diameters will add up to the total aortic diameter (Fig 15). In studying complete aortic morphology, a more

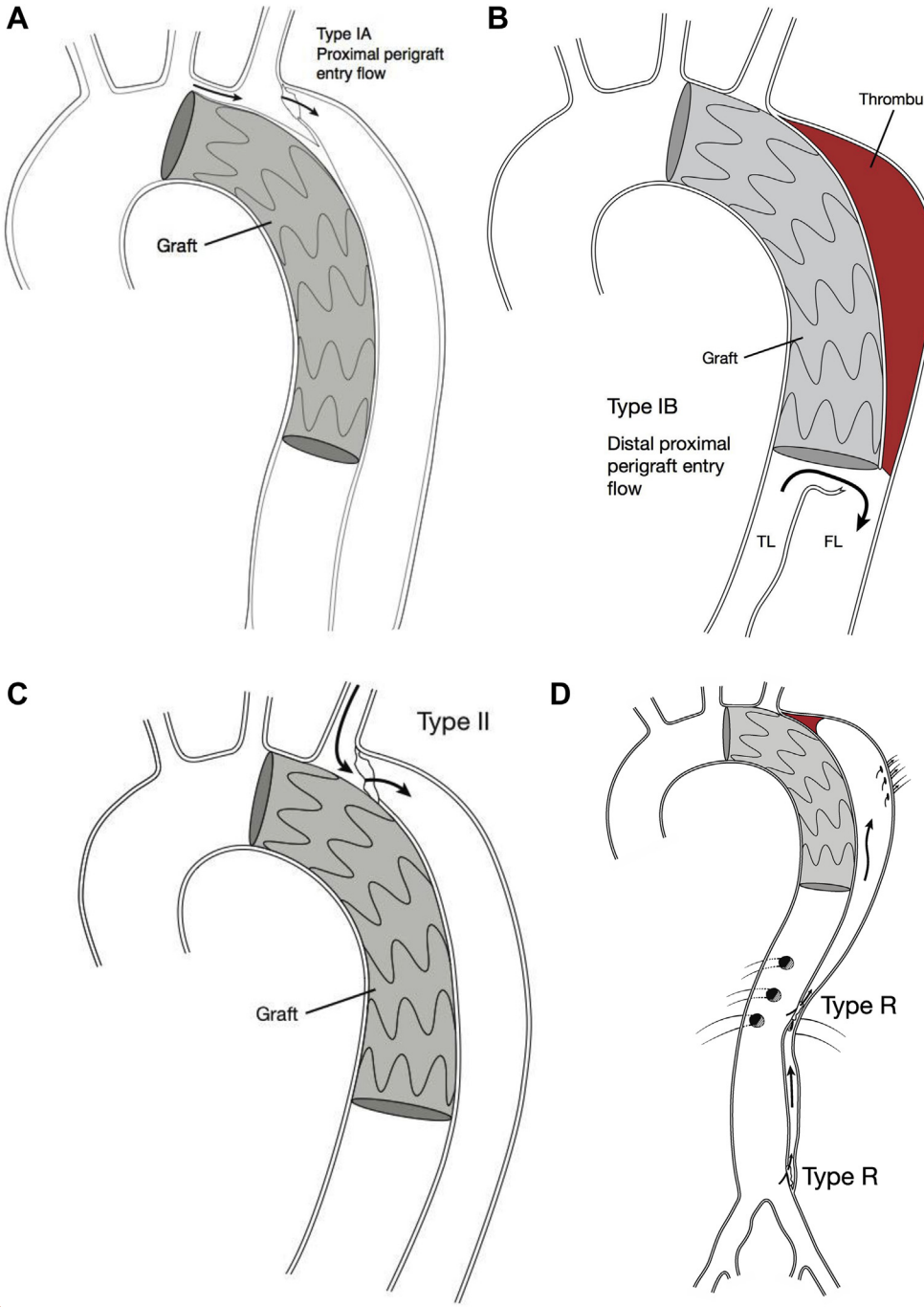

Fig 16. A, Type IA entry flow is a perigraft leak at the proximal edge of the stent graft that allows continued antegrade flow into the false lumen through the primary entry tear. **B,** Type IB entry flow is a distal perigraft leak caused by a tear in the intimal membrane adjacent to the distal edge of the endograft (distal stent graft-induced new entry [SINE]). *FL,* False lumen; *TL,* true lumen. **C,** Type II entry flow is continued retrograde false lumen perfusion through an arch branch (eg, left subclavian artery as demonstrated in the illustration) or intercostal or bronchial artery. **D,** Type R entry flow is antegrade flow from the true lumen to the false lumen through septal, visceral, or distal fenestrations.

comprehensive approach measuring maximal diameter within each individual zone (Fig 3) may be necessary. Studies may choose to report on volume measurements of the true lumen, false lumen, and entire aorta at specific levels in addition to the aforementioned measurements of maximal aortic diameter. However, normal and pathologic volume measurements are not standardized, and therefore sequential CTA or MRA comparisons denoting changes are necessary. In using volume measurements, consider the true and false lumens separately according

to zone as well as the entire aortic volume when reporting.[102]

Aortic remodeling. Aortic remodeling reflects the diameter or volume changes of the true and false lumens over the length of the dissection that take place after TEVAR or best medical therapy. These observations (maximal diameters or volumes) must be detailed for the thoracic and abdominal aorta involved in the dissection process. One should not misconstrue the early changes (eg, proximal false lumen thrombosis along some segment of the endograft) that occur between the preoperative images and those taken after TEVAR as "remodeling." The term *positive aortic remodeling* may be used only to describe an aorta that demonstrates at least one of the following:

False lumen reduction in maximal diameter or volume and no growth in total aortic diameter or volume
True lumen expansion in maximal diameter or volume and no growth in total aortic diameter or volume
Total aortic maximal diameter reduction with variable changes in true and false lumen diameters

Negative aortic remodeling would represent the opposite behaviors or a failure to demonstrate any of these descriptions. This proposed classification scheme addresses a common mistake in assessing remodeling, whereby one may observe select favorable changes yet have an expanding aorta. Remodeling of the entire dissected aorta after TEVAR should be reported according to zones of both the thoracic and abdominal aorta, as one may observe positive changes within segments of the proximal thoracic aorta (ie, treated segments) yet negative changes within the downstream distal thoracic, visceral, abdominal aortic, or iliac zones.

Entry flow into the false lumen. Presence and locations of any entry flow should be reported in initial and any subsequent follow-up studies. In patients status post TEVAR, persistent perfusion of the false lumen adjacent to the stent graft is of importance as it signifies that the treated segment is still perfused and potentially at risk of growth or rupture; the same applies to downstream untreated segments. Reporting of the source of such entry flow after TEVAR can be particularly challenging and may occasionally require arteriography to truly understand the flow dynamics.

Given that endografts reside entirely within the true lumen and do not typically appose to the outer aortic wall distally, the proposed description of false lumen entry flow after TEVAR for dissection differs somewhat from the typical endoleak classification scheme used after TEVAR for aneurysm indications.[3] In describing persistent entry flow after TEVAR for dissection, one should use the following classification system for describing flow to the false lumen:

Type IA entry flow: proximal perigraft entry flow; flow between the proximal endograft and aortic wall allowing systemic pressure antegrade flow into the primary entry tear and proximal false lumen (Fig 16, *A*).
Type IB entry flow: distal perigraft entry flow; distal entry tear adjacent to endograft due to septal fenestration or a new intimal tear at the distal aspect of the stent graft (SINE) allowing systemic pressure direct flow into the false lumen (Fig 16, *B*).
Type II entry flow: retrograde entry flow through arch vessel branches (innominate, carotid, subclavian) or thoracic bronchial and intercostal arteries into the false lumen (Fig 16, *C*).
Type R entry flow: antegrade entry flow from the true lumen into the false lumen through distal branch fenestrations (uncovered intercostal arteries, visceral or renal arteries, lumbar arteries, iliac branches) or septal fenestrations (not including SINE adjacent to the distal endograft, which is type IB; Fig 16, *D*).

Finally, follow-up CTA imaging should report any changes in the morphologic appearance of thoracic stent grafts, such as "bird-beaking" of the proximal segment, collapse, or stent fracture, or any migration of stent grafts from the last documented position.

CONCLUSIONS

The nomenclature and anatomic descriptions provided in this manuscript represent clarification and structure to ongoing variance in reporting with respect to TBAD. Use of these reporting standards will, it is hoped, provoke specifics questions, more granular in nature, that lead to a greater understanding of this disease and its management.

AUTHOR CONTRIBUTIONS

Conception and design: JL, GH, JB, AB, RC, KCO, ME, KK, TM, TR
Analysis and interpretation: JL, GH, JA, JB, BL, GW
Data collection: JL, GH, JB, JA, GW
Writing the article: JL, GH, JA, JB, AB, RC, KCO, ME, KK, BL, TM, TR, GW
Critical revision of the article: JL, GH, JB, AB, RC, KCO, ME, KK, BL, TM, TR, GW
Final approval of the article: JL, GH, JA, JB, AB, RC, KCO, ME, KK, BL, TM, TR, GW
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Overall responsibility: JL

REFERENCES

1. de Beaufort HW, Trimarchi S, Korach A, Di Eusanio M, Gilon D, Montgomery DG, et al. Aortic dissection in patients with Marfan syndrome based on the IRAD data. Ann Cardiothorac Surg 2017;6:633-41.
2. Pape LA, Awais M, Woznicki EM, Suzuki T, Trimarchi S, Evangelista A, et al. Presentation, diagnosis, and outcomes of acute aortic dissection: 17-year trends from the International Registry of Acute Aortic Dissection. J Am Coll Cardiol 2015;66:350-8.

3. Fillinger MF, Greenberg RK, McKinsey JF, Chaikof EL; Society for Vascular Surgery Ad Hoc Committee on TEVAR Reporting Standards. Reporting standards for thoracic endovascular aortic repair (TEVAR). J Vasc Surg 2010;52: 1022-33. 1033.e15.
4. Daily PO, Trueblood HW, Stinson EB, Wuerflein RD, Shumway NE. Management of acute aortic dissections. Ann Thorac Surg 1970;10:237-47.
5. Nauta FJ, Tolenaar JL, Patel HJ, Appoo JJ, Tsai TT, Desai ND, et al. Impact of retrograde arch extension in acute type B aortic dissection on management and outcomes. Ann Thorac Surg 2016;102:2036-43.
6. Pan XD, Li B, Ma WG, Zheng J, Liu YM, Zhu JM, et al. Endovascular repair of residual intimal tear or distal new entry after frozen elephant trunk for type A aortic dissection. J Thorac Dis 2017;9:529-36.
7. Kitai T, Kaji S, Yamamuro A, Tani T, Kinoshita M, Ehara N, et al. Impact of new development of ulcer-like projection on clinical outcomes in patients with type B aortic dissection with closed and thrombosed false lumen. Circulation 2010;122(Suppl):S74-80.
8. Husainy MA, Sayyed F, Puppala S. Acute aortic syndrome—pitfalls on gated and non-gated CT scan. Emerg Radiol 2016;23:397-403.
9. Sundt TM. Intramural hematoma and penetrating atherosclerotic ulcer of the aorta. Ann Thorac Surg 2007;83: S835-41; discussion: S846-50.
10. Cho KR, Stanson AW, Potter DD, Cherry KJ, Schaff HV, Sundt TM 3rd. Penetrating atherosclerotic ulcer of the descending thoracic aorta and arch. J Thorac Cardiovasc Surg 2004;127:1393-9; discussion: 1399-401.
11. Ganaha F, Miller DC, Sugimoto K, Do YS, Minamiguchi H, Saito H, et al. Prognosis of aortic intramural hematoma with and without penetrating atherosclerotic ulcer: a clinical and radiological analysis. Circulation 2002;106:342-8.
12. Evangelista A, Czerny M, Nienaber C, Schepens M, Rousseau H, Cao P, et al. Interdisciplinary expert consensus on management of type B intramural haematoma and penetrating aortic ulcer. Eur J Cardiothorac Surg 2015;47:209-17.
13. Hirst AE Jr, Johns VJ Jr, Kime SW Jr. Dissecting aneurysm of the aorta: a review of 505 cases. Medicine (Baltimore) 1958;37:217-79.
14. Dake MD, Kato N, Mitchell RS, Semba CP, Razavi MK, Shimono T, et al. Endovascular stent-graft placement for the treatment of acute aortic dissection. N Engl J Med 1999;340:1546-52.
15. Peterss S, Mansour AM, Ross JA, Vaitkeviciute I, Charilaou P, Dumfarth J, et al. Changing pathology of the thoracic aorta from acute to chronic dissection: literature review and insights. J Am Coll Cardiol 2016;68:1054-65.
16. Booher AM, Isselbacher EM, Nienaber CA, Trimarchi S, Evangelista A, Montgomery DG, et al. The IRAD classification system for characterizing survival after aortic dissection. Am J Med 2013;126:730.e19-24.
17. Nienaber CA, Kische S, Rousseau H, Eggebrecht H, Rehders TC, Kundt G, et al. Endovascular repair of type B aortic dissection: long-term results of the randomized investigation of stent grafts in aortic dissection trial. Circ Cardiovasc Interv 2013;6:407-16.
18. Rodriguez JA, Olsen DM, Lucas L, Wheatley G, Ramaiah V, Diethrich EB. Aortic remodeling after endografting of thoracoabdominal aortic dissection. J Vasc Surg 2008;47: 1188-94.
19. White RA, Miller DC, Criado FJ, Dake MD, Diethrich EB, Greenberg RK, et al. 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.
20. Erbel R, Aboyans V, Boileau C, Bossone E, Bartolomeo RD, Eggebrecht H, et al. 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases: document covering acute and chronic aortic diseases of the thoracic and abdominal aorta of the adult. The Task Force for the Diagnosis and Treatment of Aortic Diseases of the European Society of Cardiology (ESC). Eur Heart J 2014;35:2873-926.
21. Lombardi JV, Cambria RP, Nienaber CA, Chiesa R, Mossop P, Haulon S, et al. Aortic remodeling after endovascular treatment of complicated type B aortic dissection with the use of a composite device design. J Vasc Surg 2014;59:1544-54.
22. Lauterbach SR, Cambria RP, Brewster DC, Gertler JP, Lamuraglia GM, Isselbacher EM, et al. Contemporary management of aortic branch compromise resulting from acute aortic dissection. J Vasc Surg 2001;33:1185-92.
23. Schwartz SI, Durham C, Clouse WD, Patel VI, Lancaster RT, Cambria RP, et al. Predictors of late aortic intervention in patients with medically treated type B aortic dissection. J Vasc Surg 2018;67:78-84.
24. Andersen ND, Brennan JM, Zhao Y, Williams JB, Williams ML, Smith PK, et al. Insurance status is associated with acuity of presentation and outcomes for thoracic aortic operations. Circ Cardiovasc Qual Outcomes 2014;7:398-406.
25. Whelton PK, Carey RM, Aronow WS, Casey DE Jr, Collins KJ, Dennison Himmelfarb C, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. J Am Coll Cardiol 2018;71:e127-248.
26. Braverman AC. Heritable thoracic aortic aneurysm disease: recognizing phenotypes, exploring genotypes. J Am Coll Cardiol 2015;65:1337-9.
27. Brownstein AJ, Ziganshin BA, Kuivaniemi H, Body SC, Bale AE, Elefteriades JA. Genes associated with thoracic aortic aneurysm and dissection: an update and clinical implications. Aorta (Stamford) 2017;5:11-20.
28. Loeys BL, Dietz HC, Braverman AC, Callewaert BL, De Backer J, Devereux RB, et al. The revised Ghent nosology for the Marfan syndrome. J Med Genet 2010;47:476-85.
29. Goyal A, Keramati AR, Czarny MJ, Resar JR, Mani A. The genetics of aortopathies in clinical cardiology. Clin Med Insights Cardiol 2017;11:1179546817709787.
30. Teixido-Tura G, Franken R, Galuppo V, Gutierrez Garcia-Moreno L, Borregan M, Mulder BJ, et al. Heterogeneity of aortic disease severity in patients with Loeys-Dietz syndrome. Heart 2016;102:626-32.
31. MacCarrick G, Black JH 3rd, Bowdin S, El-Hamamsy I, Frischmeyer-Guerrerio PA, Guerrerio AL, et al. Loeys-Dietz syndrome: a primer for diagnosis and management. Genet Med 2014;16:576-87.
32. Bradley TJ, Bowdin SC, Morel CF, Pyeritz RE. The expanding clinical spectrum of extracardiovascular and cardiovascular manifestations of heritable thoracic aortic aneurysm and dissection. Can J Cardiol 2016;32:86-99.
33. Prakash SK, Bosse Y, Muehlschlegel JD, Michelena HI, Limongelli G, Della Corte A, et al. A roadmap to investigate the genetic basis of bicuspid aortic valve and its complications: insights from the International BAVCon (Bicuspid Aortic Valve Consortium). J Am Coll Cardiol 2014;64:832-9.
34. Jeresaty RM, Basu SK, Franco J. Dissecting aneurysm of the aorta in Turner's syndrome. JAMA 1972;222:574-6.

35. Andersen ND, Barfield ME, Hanna JM, Shah AA, Shortell CK, McCann RL, et al. Intrathoracic subclavian artery aneurysm repair in the thoracic endovascular aortic repair era. J Vasc Surg 2013;57:915-25.
36. van Son JA, Konstantinov IE, Burckhard F. Kommerell and Kommerell's diverticulum. Tex Heart Inst J 2002;29:109-12.
37. Kim JB, Yang DH, Kang JW. Right aortic arch and an aberrant left subclavian artery arising from a Kommerell diverticulum complicated by acute aortic dissection. J Thorac Cardiovasc Surg 2012;144:978-9.
38. Luty C, Laroia S, Burr M. Spontaneous Kommerell diverticulum dissection. J Vasc Interv Radiol 2012;23:1443.
39. Idrees J, Keshavamurthy S, Subramanian S, Clair DG, Svensson LG, Roselli EE. Hybrid repair of Kommerell diverticulum. J Thorac Cardiovasc Surg 2014;147:973-6.
40. van Bogerijen GH, Patel HJ, Eliason JL, Criado E, Williams DM, Knepper J, et al. Evolution in the management of aberrant subclavian arteries and related Kommerell diverticulum. Ann Thorac Surg 2015;100:47-53.
41. Tanaka A, Milner R, Ota T. Kommerell's diverticulum in the current era: a comprehensive review. Gen Thorac Cardiovasc Surg 2015;63:245-59.
42. Stanson AW, Kazmier FJ, Hollier LH, Edwards WD, Pairolero PC, Sheedy PF, et al. Penetrating atherosclerotic ulcers of the thoracic aorta: natural history and clinicopathologic correlations. Ann Vasc Surg 1986;1:15-23.
43. Wada H, Sakata N, Tashiro T. Clinicopathological study on penetrating atherosclerotic ulcers and aortic dissection: distinct pattern of development of initial event. Heart Vessels 2016;31:1855-61.
44. Benitez RM, Gurbel PA, Chong H, Rajasingh MC. Penetrating atherosclerotic ulcer of the aortic arch resulting in extensive and fatal dissection. Am Heart J 1995;129:821-3.
45. Azizzadeh A, Keyhani K, Miller CC 3rd, Coogan SM, Safi HJ, Estrera AL. Blunt traumatic aortic injury: initial experience with endovascular repair. J Vasc Surg 2009;49:1403-8.
46. Rabin J, DuBose J, Sliker CW, O'Connor JV, Scalea TM, Griffith BP. Parameters for successful nonoperative management of traumatic aortic injury. J Thorac Cardiovasc Surg 2014;147:143-9.
47. Williams ML, Sheng S, Gammie JS, Rankin JS, Smith PK, Hughes GC, et al. Aortic dissection as a complication of cardiac surgery: report from the Society of Thoracic Surgeons database. Ann Thorac Surg 2010;90:1812-6; discussion: 1816-7.
48. Shrikhande GV, Khan SZ, Meltzer AJ, Gallagher K, Morrissey NJ. Endovascular management of type B aortic dissection after attempted central venous catheterization. Ann Vasc Surg 2011;25:979.e13-5.
49. Schrimpf C, Teebken OE, Wilhelmi M. Thoracic endovascular aortic repair after iatrogenic aortic dissection and false lumen stent grafting. Ann Thorac Surg 2015;99:1447-9.
50. Kilic A, Siki MA, Szeto WY, Bavaria JE, Anwaruddin S, Desai ND. Concomitant endografting of a type B aortic dissection during transfemoral aortic valve replacement. Ann Thorac Surg 2017;103:e223-4.
51. Dean JH, Woznicki EM, O'Gara P, Montgomery DG, Trimarchi S, Myrmel T, et al. Cocaine-related aortic dissection: lessons from the International Registry of Acute Aortic Dissection. Am J Med 2014;127:878-85.
52. Berretta P, Patel HJ, Gleason TG, Sundt TM, Myrmel T, Desai N, et al. IRAD experience on surgical type A acute dissection patients: results and predictors of mortality. Ann Cardiothorac Surg 2016;5:346-51.
53. Hagan PG, Nienaber CA, Isselbacher EM, Bruckman D, Karavite DJ, Russman PL, et al. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. JAMA 2000;283:897-903.
54. Holdgate A, Asha S, Craig J, Thompson J. Comparison of a verbal numeric rating scale with the visual analogue scale for the measurement of acute pain. Emerg Med (Fremantle) 2003;15:441-6.
55. Trimarchi S, Eagle KA, Nienaber CA, Pyeritz RE, Jonker FH, Suzuki T, et al. Importance of refractory pain and hypertension in acute type B aortic dissection: insights from the International Registry of Acute Aortic Dissection (IRAD). Circulation 2010;122:1283-9.
56. Hiratzka LF, Bakris GL, Beckman JA, Bersin RM, Carr VF, Casey DE Jr, 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. Circulation 2010;121:e266-369.
57. Song C, Lu Q, Zhou J, Yu G, Feng X, Zhao Z, et al. The new indication of TEVAR for uncomplicated type B aortic dissection. Medicine (Baltimore) 2016;95:e3919.
58. Evangelista A, Salas A, Ribera A, Ferreira-Gonzalez I, Cuellar H, Pineda V, et al. Long-term outcome of aortic dissection with patent false lumen: predictive role of entry tear size and location. Circulation 2012;125:3133-41.
59. Evangelista A, Galuppo V, Gruosso D, Cuellar H, Teixido G, Rodriguez-Palomares J. Role of entry tear size in type B aortic dissection. Ann Cardiothorac Surg 2014;3:403-5.
60. Williams DM, Lee DY, Hamilton BH, Marx MV, Narasimham DL, Kazanjian SN, et al. The dissected aorta: part III. Anatomy and radiologic diagnosis of branch-vessel compromise. Radiology 1997;203:37-44.
61. Jonker FH, Patel HJ, Upchurch GR, Williams DM, Montgomery DG, Gleason TG, et al. Acute type B aortic dissection complicated by visceral ischemia. J Thorac Cardiovasc Surg 2015;149:1081-6.e1.
62. Jain A, Tracci MC, Coleman DM, Cherry KJ, Upchurch GR Jr. Renal malperfusion: spontaneous renal artery dissection and with aortic dissection. Semin Vasc Surg 2013;26:178-88.
63. Tsai HS, Tsai FC, Chen YC, Wu LS, Chen SW, Chu JJ, et al. Impact of acute kidney injury on one-year survival after surgery for aortic dissection. Ann Thorac Surg 2012;94:1407-12.
64. Gargiulo M, Bianchini Massoni C, Gallitto E, Freyrie A, Trimarchi S, Faggioli G, et al. Lower limb malperfusion in type B aortic dissection: a systematic review. Ann Cardiothorac Surg 2014;3:351-67.
65. Broderick JP, Adeoye O, Elm J. Evolution of the modified Rankin scale and its use in future stroke trials. Stroke 2017;48:2007-12.
66. Ullery BW, Wang GJ, Low D, Cheung AT. Neurological complications of thoracic endovascular aortic repair. Semin Cardiothorac Vasc Anesth 2011;15:123-40.
67. Miyasaka K, Asano T, Ushikoshi S, Hida K, Koyanagi I. Vascular anatomy of the spinal cord and classification of spinal arteriovenous malformations. Interv Neuroradiol 2000;6(Suppl 1):195-8.
68. Biglioli P, Roberto M, Cannata A, Parolari A, Fumero A, Grillo F, et al. Upper and lower spinal cord blood supply: the continuity of the anterior spinal artery and the relevance of the lumbar arteries. J Thorac Cardiovasc Surg 2004;127:1188-92.

69. Mazensky D, Flesarova S, Sulla I. Arterial blood supply to the spinal cord in animal models of spinal cord injury. A review. Anat Rec (Hoboken) 2017;300:2091-106.
70. Trimarchi S, Segreti S, Grassi V, Lomazzi C, Cova M, Piffaretti G, et al. Open fenestration for complicated acute aortic B dissection. Ann Cardiothorac Surg 2014;3:418-22.
71. Kuo EC, Veranyan N, Johnson CE, Weaver FA, Ham SW, Rowe VL, et al. Impact of proximal seal zone length and intramural hematoma on clinical outcomes and aortic remodeling after thoracic endovascular aortic repair for aortic dissections. J Vasc Surg 2019;69:987-95.
72. Wintermark M, Sanelli PC, Albers GW, Bello JA, Derdeyn CP, Hetts SW, et al. Imaging recommendations for acute stroke and transient ischemic attack patients: a joint statement by the American Society of Neuroradiology, the American College of Radiology and the Society of NeuroInterventional Surgery. J Am Coll Radiol 2013;10:828-32.
73. Powers WJ, Rabinstein AA, Ackerson T, Adeoye OM, Bambakidis NC, Becker K, et al. 2018 Guidelines for the Early Management of Patients With Acute Ischemic Stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 2018;49:e46-110.
74. Powers WJ, Derdeyn CP, Biller J, Coffey CS, Hoh BL, Jauch EC, et al. 2015 American Heart Association/American Stroke Association Focused Update of the 2013 Guidelines for the Early Management of Patients With Acute Ischemic Stroke Regarding Endovascular Treatment: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 2015;46:3020-35.
75. Lee TC, Andersen ND, Williams JB, Bhattacharya SD, McCann RL, Hughes GC. Results with a selective revascularization strategy for left subclavian artery coverage during thoracic endovascular aortic repair. Ann Thorac Surg 2011;92:97-102; discussion: 102-3.
76. Ullery BW, McGarvey M, Cheung AT, Fairman RM, Jackson BM, Woo EY, et al. Vascular distribution of stroke and its relationship to perioperative mortality and neurologic outcome after thoracic endovascular aortic repair. J Vasc Surg 2012;56:1510-7.
77. Mehta RL, Kellum JA, Shah SV, Molitoris BA, Ronco C, Warnock DG, et al. Acute Kidney Injury Network: report of an initiative to improve outcomes in acute kidney injury. Crit Care 2007;11:R31.
78. Huynh TT, Miller CC 3rd, Safi HJ. Delayed onset of neurologic deficit: significance and management. Semin Vasc Surg 2000;13:340-4.
79. Chiesa R, Melissano G, Marrocco-Trischitta MM, Civilini E, Setacci F. Spinal cord ischemia after elective stent-graft repair of the thoracic aorta. J Vasc Surg 2005;42:11-7.
80. Ullery BW, Cheung AT, Fairman RM, Jackson BM, Woo EY, Bavaria J, et al. Risk factors, outcomes, and clinical manifestations of spinal cord ischemia following thoracic endovascular aortic repair. J Vasc Surg 2011;54:677-84.
81. Champagne BJ, Lee EC, Valerian B, Mulhotra N, Mehta M. Incidence of colonic ischemia after repair of ruptured abdominal aortic aneurysm with endograft. J Am Coll Surg 2007;204:597-602.
82. Canaud L, Ozdemir BA, Patterson BO, Holt PJ, Loftus IM, Thompson MM. Retrograde aortic dissection after thoracic endovascular aortic repair. Ann Surg 2014;260:389-95.
83. Khoynezhad A, White RA. Pathogenesis and management of retrograde type A aortic dissection after thoracic endovascular aortic repair. Ann Vasc Surg 2013;27:1201-6.
84. Dong Z, Fu W, Wang Y, Wang C, Yan Z, Guo D, et al. Stent graft-induced new entry after endovascular repair for Stanford type B aortic dissection. J Vasc Surg 2010;52:1450-7.
85. Weng SH, Weng CF, Chen WY, Huang CY, Chen IM, Chen CK, et al. Reintervention for distal stent graft-induced new entry after endovascular repair with a stainless steel-based device in aortic dissection. J Vasc Surg 2013;57:64-71.
86. Jang H, Kim MD, Kim GM, Won JY, Ko YG, Choi D, et al. Risk factors for stent graft-induced new entry after thoracic endovascular aortic repair for Stanford type B aortic dissection. J Vasc Surg 2017;65:676-85.
87. Huang CY, Hsu HL, Chen PL, Chen IM, Hsu CP, Shih CC. The impact of distal stent graft-induced new entry on aortic remodeling of chronic type B dissection. Ann Thorac Surg 2018;105:785-93.
88. Janosi RA, Tsagakis K, Bettin M, Kahlert P, Horacek M, Al-Rashid F, et al. Thoracic aortic aneurysm expansion due to late distal stent graft-induced new entry. Catheter Cardiovasc Interv 2015;85:E43-53.
89. Huang CY, Weng SH, Weng CF, Chen WY, Chen IM, Hsu CP, et al. Factors predictive of distal stent graft-induced new entry after hybrid arch elephant trunk repair with stainless steel-based device in aortic dissection. J Thorac Cardiovasc Surg 2013;146:623-30.
90. Fattori R, Cao P, De Rango P, Czerny M, Evangelista A, Nienaber C, et al. Interdisciplinary expert consensus document on management of type B aortic dissection. J Am Coll Cardiol 2013;61:1661-78.
91. Vardhanabhuti V, Nicol E, Morgan-Hughes G, Roobottom CA, Roditi G, Hamilton MC, et al. Recommendations for accurate CT diagnosis of suspected acute aortic syndrome (AAS)—on behalf of the British Society of Cardiovascular Imaging (BSCI)/British Society of Cardiovascular CT (BSCCT). Br J Radiol 2016;89:20150705.
92. Bonci G, Steigner ML, Hanley M, Braun AR, Desjardins B, Gaba RC, et al. ACR Appropriateness Criteria thoracic aorta interventional planning and follow-up. J Am Coll Radiol 2017;14(Suppl):S570-83.
93. Nauta FJ, Trimarchi S, Kamman AV, Moll FL, van Herwaarden JA, Patel HJ, et al. Update in the management of type B aortic dissection. Vasc Med 2016;21:251-63.
94. Flors L, Leiva-Salinas C, Norton PT, Patrie JT, Hagspiel KD. Imaging follow-up of endovascular repair of type B aortic dissection with dual-source, dual-energy CT and late delayed-phase scans. J Vasc Interv Radiol 2014;25:435-42.
95. Flors L, Leiva-Salinas C, Norton PT, Patrie JT, Hagspiel KD. Endoleak detection after endovascular repair of thoracic aortic aneurysm using dual-source dual-energy CT: suitable scanning protocols and potential radiation dose reduction. AJR Am J Roentgenol 2013;200:451-60.
96. Iezzi R, Cotroneo AR, Filippone A, Santoro M, Basilico R, Storto ML. Multidetector-row computed tomography angiography in abdominal aortic aneurysm treated with endovascular repair: evaluation of optimal timing of delayed phase imaging for the detection of low-flow endoleaks. J Comput Assist Tomogr 2008;32:609-15.
97. Macari M, Chandarana H, Schmidt B, Lee J, Lamparello P, Babb J. Abdominal aortic aneurysm: can the arterial phase at CT evaluation after endovascular repair be eliminated to reduce radiation dose? Radiology 2006;241:908-14.
98. Iribarne A, Keenan J, Benrashid E, Wang H, Meza JM, Ganapathi A, et al. Imaging surveillance after proximal aortic operations: is it necessary? Ann Thorac Surg 2017;103:734-41.

99. van Bogerijen GH, Tolenaar JL, Rampoldi V, Moll FL, van Herwaarden JA, Jonker FH, et al. Predictors of aortic growth in uncomplicated type B aortic dissection. J Vasc Surg 2014;59:1134-43.
100. Patterson BO, Cobb RJ, Karthikesalingam A, Holt PJ, Hinchliffe RJ, Loftus IM, et al. A systematic review of aortic remodeling after endovascular repair of type B aortic dissection: methods and outcomes. Ann Thorac Surg 2014;97:588-95.
101. Famularo M, Meyermann K, Lombardi JV. Aneurysmal degeneration of type B aortic dissections after thoracic endovascular aortic repair: a systematic review. J Vasc Surg 2017;66:924-30.
102. Sobocinski J, Lombardi JV, Dias NV, Berger L, Zhou Q, Jia F, et al. Volume analysis of true and false lumens in acute complicated type B aortic dissections after thoracic endovascular aortic repair with stent grafts alone or with a composite device design. J Vasc Surg 2016;63:1216-24.

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