CHAPTER 135

Aortic Dissection

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Acute aortic dissection is the most common catastrophic event affecting the aorta, with an incidence exceeding that of ruptured abdominal aortic aneurysm. The first report of aortic dissection and the concept of a true and a false lumen are attributed to Shekelton in the early 1800s.[1] The term *anurysme dissequant*, or dissecting aneurysm, introduced by Laennec in 1819,[2] remains a source of confusion because acute dissections can occur in both dilated diseased aortas and aortas of normal diameter in seemingly healthy individuals. Accordingly, the terms *dissection* and *aneurysm* should not be used interchangeably; although dissection can occur in a preexisting degenerative aneurysm and aneurysms can complicate chronic dissections, the presence of one is not dependent on the other. An additional source of diagnostic confusion is the presence of other pathologies of the thoracic aorta such as intramural hematoma and penetrating aortic ulcer, which have clinical and radiographic similarity with acute dissection.

Aortic dissection is a lethal disease. Early studies indicated that without treatment, the majority of patients died within 3 months of diagnosis and few survived the chronic phase more than 5 years because of aneurysmal degeneration and rupture of the outer wall of the false lumen.[3,4] Despite improvements in both medical and surgical therapeutic options, the overall mortality associated with acute dissection remains significant. In a population-based epidemiologic study by Clouse and coworkers, 38% of aortic dissections were diagnosed at autopsy.[5] This substantial mortality rate in undiagnosed patients underscores the importance of early diagnosis and initiation of appropriate therapy. Indeed, Khan and Nair predicted that the mortality rate for acute dissection left untreated will exceed 22.7% within 6 hours, 50% within 24 hours, and 68% within the first week.[6] Death from acute dissection of the ascending aorta is usually secondary to the central cardioaortic complications of aortic rupture into the pericardium, acute aortic regurgitation, and coronary ostial compromise,[7,8] whereas descending aortic dissections are more commonly associated with death from end-organ compromise secondary to obstruction of visceral or extremity vessels.[9,10]

The International Registry of Acute Aortic Dissection (IRAD) is a multinational registry that began enrolling patients in January 1996. It initially included 12 centers in 6 countries and has grown to 24 referral centers in 11 countries. All patients with acute aortic dissection confirmed by diagnostic imaging studies, by direct visualization in the operating room, or at autopsy are included in the registry. Data are entered from a prospectively collected questionnaire consisting of 290 variables that have been externally validated at each site.[11] This project has provided contemporary insight into the short- and now long-term outcomes of acute dissection and proposed therapeutic options and consequently is referenced often herein.[12,13]

In this chapter we review the classification, pathologic anatomy, pathogenesis, clinical findings, and diagnostic and treatment modalities for acute aortic dissection, with emphasis on the role of the vascular/endovascular surgeon. Although a thorough understanding of all components of acute dissection seems requisite for those who propose to treat aortic dissection, the technical principles of graft replacement of the ascending aorta, being the province of cardiac surgeons, is not detailed. Because open graft replacement for the treatment of descending aortic dissection is rarely indicated, it is anticipated that the vascular/endovascular surgeon will become the primary interventionalist in the care of patients with acute dissection of the descending aorta and the complications of peripheral vascular compromise. Finally, treatment of the principal late complication of acute aortic dissection—the development of thoracoabdominal aneurysms—is considered in Chapter 131 (Thoracic and Thoracoabdominal Aneurysms: Evaluation and Decision Making).

CLASSIFICATION

Aortic dissections are classified according to the anatomic location of the entry tear and the time between the onset of symptoms and patient evaluation.

Temporal

A dissection is considered acute when the diagnosis is made within 2 weeks of the initial onset of symptoms and thereafter becomes chronic. Although such designation appears arbitrary, it is based on autopsy studies showing that 74% of patients who die of aortic dissections do so in the first 14 days.[14] Accordingly, such temporal classification combined with the anatomic location of the entry tear can have immediate therapeutic implications.

Anatomic

The anatomic classification of aortic dissection is based on the location of the intimal tear and the extent of the dissection along the aorta. Two classification schemes are used to describe aortic dissections. The original, proposed by DeBakey

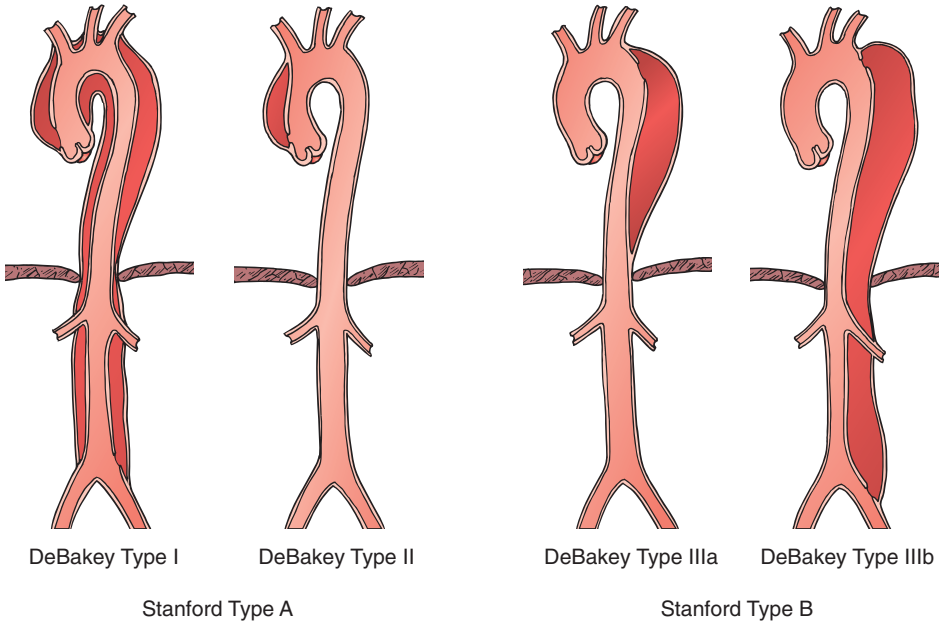


Figure 135-1 Acute aortic dissection can be classified by the original DeBakey system (*darker color*) based on location of the proximal tear and distal extent of the dissection flap or on the Stanford system (*lighter color*), which uses location of the proximal tear alone.

and colleagues in 1965, delineates both the origin of the entry tear and the extent of the descending aortic dissection.[15] The scheme (Fig. 135-1) uses the following classification:

- Type I: The dissection originates in the ascending aorta and extends through the aortic arch and into the descending aorta or abdominal aorta (or both) for a varying distance.
- Type II: The dissection originates in and is confined to the ascending aorta.
- Type IIIa: The dissection originates in the descending aorta and is limited to the same.
- Type IIIb: The dissection involves the descending and variable extents of the abdominal aorta.

The Stanford classification, described by Daily and associates in 1970, simplified the anatomic classification according to the origin of the entry tear alone.[16]

- A Stanford type A dissection originates in the ascending aorta and therefore encompasses DeBakey types I and II dissections.
- A Stanford type B dissection originates in the descending aorta distal to the origin of the left subclavian artery (DeBakey types IIIa and IIIb; see Fig. 135-1).

Because the origin of the entry tear is the key predictor of early outcomes, most patients are now stratified into Stanford type A or B at diagnosis to direct initial therapy. Prompt graft replacement of the ascending aorta is the appropriate treatment for the majority (for exceptions, see "Open Surgical Treatment of Ascending Aortic Dissection") of patients with Stanford type A dissections because they are associated with a high risk for lethal cardioaortic complications (principally, aortic rupture or myocardial ischemia from extension into the coronary arteries) in the hours and days after symptom onset. Alternatively, patients with Stanford type B dissections are managed initially with medical therapy unless one or more complications develop (reviewed under "Pathogenesis of Malperfusion Syndromes").

EPIDEMIOLOGY

Recent population-based studies have estimated the incidence of acute aortic dissection to range from 2.9 to 3.5 per 100,000 person-years.[5,17] Factors that predispose to the development of aortic dissection include older age, hypertension, and structural abnormalities of the aortic wall.[6,11] Men are more frequently affected, with a male-female ratio of 4:1 reported in a recent IRAD series.[18] In our cumulative experience, type A dissections account for 60% of cases, which is consistent with the IRAD data, wherein 62.5% of 1417 patients had type A dissections.[12] The incidence of type A dissection peaks between 50 and 60 years, whereas type B dissections occur more frequently between 60 and 70 years of age.[11] Hypertension is the rule and was present in 70% of patients in the IRAD database (Table 135-1).[18]

Table 135-1 Demographic and Clinical Features of combined MGH and IRAD Experience with Acute Aortic Dissection

Variable	MGH*	IRAD[36]	Total
Patients	512	1078	1590
Age (mean)	62 years	62.4 years	—
Male	347 (67.8%)	732 (67.9%)	1079 (67.9%)
Type A	278 (54.3%)	674 (62.5%)	952 (59.9%)
Hypertension	361 (70.5%)	755 (70%)	1116 (70.2%)
VC/pulse deficit	159 (31%)	259 (24%)	418 (26.3%)

*Unpublished data.
IRAD, International Registry of Acute Aortic Dissection; MGH, Massachusetts General Hospital; VC, vascular complication.

Risk Factors for Dissection

Cardiovascular Conditions

Cardiovascular conditions such as acute myocardial infarction and sudden death have been shown to demonstrate certain chronobiologic patterns of occurrence,[19,20] and the same is true of acute aortic dissection.[21] The onset of dissection occurs most frequently in the morning hours between 6 AM and 12 noon and more often in the winter (28%) than the summer (20%), independent of the endogenous climate.[21,22] Aortic wall structural abnormalities and the presence of a bicuspid aortic valve with or without its accompanying aortic root dilatation are well-established risk factors for ascending aortic dissection. Indeed, the presence of a bicuspid aortic valve has been documented in 7% to 14% of all aortic dissections.[11,23] Other aortic diseases, such as coarctation of the aorta, annuloaortic ectasia, chromosomal abnormalities (Turner's syndrome and Noonan's syndrome), aortic arch hypoplasia, and hereditary conditions (Marfan's syndrome and Ehlers-Danlos syndrome), are also risk factors for the development of acute aortic dissection.[24] Marfan's syndrome accounts for 50% of cases of acute aortic dissection in patients younger than 40 years (see Chapter 134: Aortic Arch Aneurysms and Dissection).[18]

Pregnancy

It has previously been reported that in women younger than 40 years, 50% of aortic dissections occur during pregnancy[25]; however, a recent review of the IRAD data showed an incidence of 13%.[18] Preeclampsia with resultant hypertension is the most common cause of peripartum aortic dissection, but pregnant women with Marfan's syndrome are also at high risk; the presence of a dilated aortic root (>4 cm) is the best predictor of dissection in a pregnant patient with Marfan's syndrome.[26] The most common site of aortic dissection in young (<40 years) patients is the ascending aorta involving the sinuses of Valsalva or the sinotubular junction, whereas type A dissections in older patients are more likely to originate in the ascending aorta.[18]

Cocaine Abuse

Cocaine ingestion, a rare cause of acute aortic dissection in otherwise healthy individuals, has an incidence as high as 37% in an urban setting but was present in less than 1% of the IRAD population.[18,27,28] Recently, Daniel and coauthors reviewed the characteristics of 16 patients who had used cocaine within 24 hours of the onset of acute aortic dissection. They found that the prototypical patient was a young (average age, 47 years), male (75%) smoker (100%) with a history of hypertension (70%) and no particular trend toward dissection type.[28] One possible mechanism is related to catecholamine release, which causes the triad of profound hypertension, vasoconstriction, and increased cardiac output. This creates a dramatic, acute increase in ventricular contraction (shearing) force (dP/dt) on the aortic wall. The intimal tear occurs most often at the ligamentum arteriosum, where the aorta is relatively fixed and unable to tolerate the accelerating aortic pressure wave generated by the profound tachycardia and malignant hypertension.[28,29]

PATHOLOGIC ANATOMY OF ACUTE AORTIC DISSECTION

Intimal Tear

The process of aortic dissection is dynamic, and it can occur anywhere along the course of the aorta and result in a wide spectrum of clinical manifestations. The pathognomonic lesion is an intimal tear followed by blood surging either antegrade (typically) or retrograde (depending on the hemodynamic gradient between the true and false lumina) and cleaving the intimal and medial layers of the aortic wall longitudinally for a variable distance.[6,30] The typical tear is transverse, not circumferential, and the intimomedial layer can be cleaved both longitudinally and circumferentially.[6] The adventitially bound blood-filled space created between the dissected layers of the aortic wall becomes the false lumen. Fenestrations (connections between the true and false lumina) occur within the intimal flap downstream, usually at branch vessel ostia, and are cleaved by the dissection process; they serve as sites of reentry of blood flow into the true lumen and thus maintain false lumen patency. The presence of an "intimal flap," which represents the intimomedial septum between the true and the false lumen, is the most characteristic pathology in acute aortic dissection. The intimal flap/tear originates in the ascending aorta in 65%, in the descending aorta in 25%, and in the arch and abdominal aorta in 10% of patients.[11] In the descending aorta, the intimal tear typically originates within a few centimeters of the left subclavian artery because this segment of the aorta is subject to the greatest pressure fluctuations.[14,31,32] In the usual pattern of a Stanford type B dissection, the cleavage plane will progress with the distal false lumen on the left posterolateral aspect of the aorta (80% of patients); the celiac, superior mesenteric, and right renal arteries typically emanate from the true lumen, and the left renal artery arises from the false lumen.[14] However, variations in this pattern are frequently encountered.

Cystic Medial Necrosis

One pathologic process that is associated with increased risk for aortic dissection is medial degeneration of the aortic wall (cystic medial necrosis), which diminishes the structural integrity of the aortic layers.[32,33] The central lesion appears to be deterioration of medial collagen and elastin fibers through elastolysis; this is considered to be a factor in most cases of aortic dissection.[34] In particular, classic cystic medial necrosis appears to be an essential feature of several hereditary conditions such as Ehlers-Danlos syndrome and Marfan's disease,[24] yet specific connective tissue diseases account for only 10% to 15% of all acute aortic dissections.[11] Even in "normal individuals," in whom dissections occur without any antecedent diagnosis of syndromic diseases, the degree of medial degen-

eration still tends to be greater than expected as part of normal aging. The exact cause of this medial degeneration remains unclear, but advanced age and hypertension appear to be the most important factors.[21,34,35]

Atherosclerosis

Atherosclerosis has not been considered to be an important etiologic feature of acute aortic dissection and was present in only 31% of patients in the IRAD registry.[36] However, Jex and coworkers noted either gross or microscopic atheroma in 83% of patients in their series.[37] Atherosclerotic plaque may, in fact, be protective, serving to terminate the dissection process, because the transmural inflammatory nature of atherosclerosis may fuse the aortic layers.[38] The presence of an atherosclerotic aneurysm with concurrent aortic dissection occurred in only 14% to 15% of patients in recent series.[36,39] The unusual coexistence of an aortic dissection that originates in or involves a preexisting atherosclerotic aneurysm appears to change the natural history, and rupture is the probable scenario. For instance, in a review of 325 patients with aortic dissection, rupture in the abdomen occurred only in the setting of antecedent degenerative, atherosclerotic aneurysm.[38] These findings support the posture of treating such type B dissections as "complicated," and initial surgical priority should be given to the aorta where both entities are present (usually the infrarenal abdominal aorta).

PATHOGENESIS OF MALPERFUSION SYNDROMES

Consistent over several series, aortic branch compromise is present in up to 31% of patients with acute aortic dissection,[10,36,40,41] with progression to malperfusion syndrome correlating with early mortality.[9,10,36] Malperfusion syndrome occurs when there is end-organ ischemia secondary to aortic branch compromise from the dissecting process. It can involve one or more vascular beds simultaneously, with early symptoms often being subtle and of varying severity over the hours and days after initial symptom onset. The terms *aortic branch compromise* and *malperfusion syndrome* should not be used interchangeably because the obstruction is often subtotal and produces variable degrees of end-organ ischemia, and certain affected vessels (e.g., the subclavian and celiac arteries) may not produce critical ischemia, even with total occlusion, as a result of the presence of collateral circulation. Virtually any aortic branch can be affected, and as intuitively suspected, the morbid clinical events will vary as a function of the vascular territory involved. Mesenteric involvement is associated with intestinal infarction, whereas subclavian and lower extremity occlusive events are often well tolerated (Fig. 135-2).

Mechanism

Identifying the mechanisms of branch compromise is a critical step in formulating effective treatment plans. The anatomic and physiologic variables underpinning any compromised

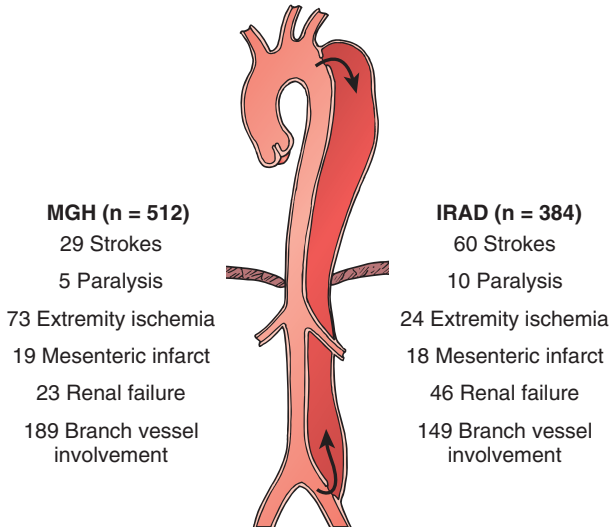


Figure 135-2 Comparison of the Massachusetts General Hospital (MGH) and the International Registry of Acute Aortic Dissection (IRAD) experience with peripheral vascular complications after acute aortic dissection. Differences between the number of branch vessels involved and clinical events represent asymptomatic lesions.

vascular bed include (1) the percentage of aortic circumference dissected, (2) the presence of a distal reentrant focus in false lumen or true lumen outflow, and (3) the topography of branch ostia to the true versus the false lumen.[7] In the minutes after an aortic dissection is initiated, the true lumen (representing the remnant of the original aortic lumen) collapses to a variable degree and the false lumen expands.[42] The adventitially bound outer wall of the false lumen must expand to a larger diameter to accommodate the same wall tension at a given blood pressure, as governed by the law of Laplace. In contrast, the true lumen, which contains the majority of the elastic components of the aortic wall, undergoes radial elastic collapse.[42] Therefore, the degree to which the true lumen recoils and the false lumen expands (i.e., their respective cross-sectional areas) depends on the percentage of the total aortic circumference involved with the dissection. In the presence of a deep proximal tear and the absence of distal fenestrations, mean false lumen pressure increases and results in compression of the true lumen[43] (Fig. 135-3). A compressed true lumen will lead to impaired perfusion of distal structures and should increase the index of suspicion for visceral and renal ischemia. Finally, the topographic relationship of the true and false lumina and the potential for extension of the dissection into the aortic branch itself are the anatomic factors that determine the mechanism of the malperfusion syndrome (see Fig. 135-3).

Two mechanisms for aortic branch vessel compromise have been identified, each of which has specific treatment implications in the management of malperfusion syndromes as originally described by Williams and colleagues.[42]

Dynamic Obstruction

In *dynamic obstruction*, the compressed true lumen is unable to provide adequate volume flow, or the dissection flap may

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