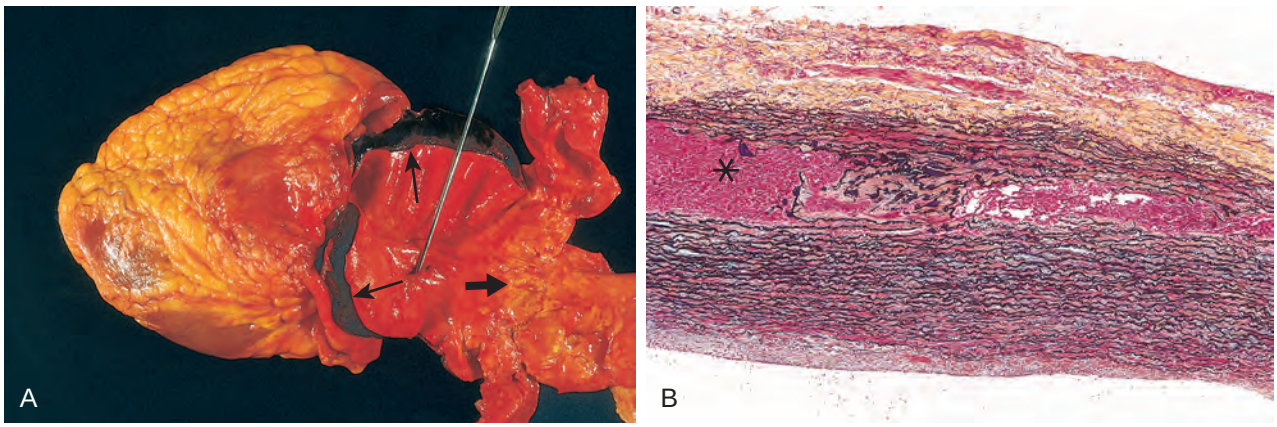

Figure 11-21 Aortic dissection. **A,** Gross photograph of an opened aorta with proximal dissection originating from a small, oblique intimal tear (probe), allowing blood to enter the media and creating a retrograde intramural hematoma *(narrow arrows).* Note that the intimal tear has occurred in a region largely free of atherosclerotic plaque and that propagation of the intramural hematoma distally is arrested where atherosclerosis begins *(broad arrow).* **B,** Histologic view of the dissection demonstrating an aortic intramural hematoma *(asterisk).* Aortic elastic layers are black and blood is red (Movat stain).

the coronary ostia causing myocardial ischemia, and (6) rupture. Most patients with syphilitic aneurysms die of heart failure secondary to aortic valvular incompetence.

Aortic Dissection

Aortic dissection occurs when blood separates the laminar planes of the media to form a blood-filled channel within the aortic wall (Fig. 11-21); this can be catastrophic if the dissection then ruptures through the adventitia and hemorrhages into adjacent spaces. Aortic dissection may or may not be associated with radiologically detectable aortic dilation.

Aortic dissection occurs principally in two groups of patients: (1) men aged 40 to 60 years with antecedent hypertension (more than 90% of cases) and (2) younger adults with systemic or localized abnormalities of connective tissue affecting the aorta (e.g., Marfan syndrome). Dissections can be iatrogenic, for example, following arterial cannulations during coronary catheterization procedures or cardiopulmonary bypass. Rarely, pregnancy is associated with aortic (or other vessel) dissection (roughly 10 to 20 cases per million births). This typically occurs during or after the third trimester, and may be related to hormone-induced vascular remodeling and the hemodynamic stresses of the perinatal period. Dissection is unusual in the presence of substantial atherosclerosis or other cause of medial scarring such as syphilis, presumably because the medial fibrosis inhibits propagation of the dissecting hematoma.

Pathogenesis. **Hypertension is the major risk factor for aortic dissection.** Aortas of hypertensive patients have medial hypertrophy of vasa vasorum associated with degenerative changes such as loss of medial smooth muscle cells and disorganized extracellular matrix, suggesting that ischemic injury (due to diminished flow through the vasa vasorum, possibly exacerbated by high wall pressures) is contributory. Other dissections occur in the setting of inherited or acquired connective tissue disorders with defective vascular extracellular matrix (e.g., Marfan syndrome, Ehlers-Danlos syndrome, defects in copper metabolism). However, recognizable medial damage appears to be neither a prerequisite for dissection nor a reliable predictor of its occurrence. Regardless of the underlying etiology, the trigger for the intimal tear and initial intramural aortic hemorrhage is not known in most cases. Once a tear has occurred, blood flow under systemic pressure dissects through the media, leading to progression of the hematoma. Accordingly, aggressive pressure-reducing therapy may be effective in limiting an evolving dissection. In some cases, disruption of penetrating vessels of the vasa vasorum can give rise to an intramural hematoma *without* an intimal tear.

MORPHOLOGY

In most cases, no specific underlying causal pathology is identified in the aortic wall. The most frequent preexisting histologically detectable lesion is **cystic medial degeneration** (Fig. 11-19), and inflammation is characteristically absent. However, since dissections can occur in the setting of rather trivial medial degeneration, and marked degenerative changes are frequently seen at autopsies of patients who are completely free from dissection, the relationship of the structural changes to the pathogenesis of dissection is uncertain.

An aortic dissection usually initiates with an intimal tear. In the vast majority of spontaneous dissections, the tear occurs in the ascending aorta, usually within 10 cm of the aortic valve (Fig. 11-21*A*). Such tears are typically transverse with sharp, jagged edges up to 1 to 5 cm in length. The dissection can extend retrograde toward the heart as well as distally, sometimes into the iliac and femoral arteries. The dissecting hematoma spreads characteristically along the laminar planes of the aorta, usually between the middle and outer thirds (Fig. 11-21*B*). It can rupture through the adventitia causing massive hemorrhage (e.g., into the thoracic or abdominal cavities) or cardiac tamponade (hemorrhage into the pericardial sac). In some (lucky) instances, the dissecting hematoma reenters the lumen of the aorta through a second distal intimal tear, creating a new false vascular channel ("double-barreled aorta"). This averts a fatal extraaortic hemorrhage, and over time, such false channels can be endothelialized to become recognizable **chronic dissections**.