Pathogenesis of Dissecting Aneurysm

By Herbert Braunstein, M.D.

EARLY pathologic studies of dissecting aneurysm emphasized the roles of sudden stress and of transient or persistent hypertension as precipitating causes.1 These concepts were disputed by Gsell,2 Erdheim,3,4 and Cellina,5 and as a result, their hypothesis that dissecting aneurysm required a pre-existing, underlying weakness in the aortic wall became widely accepted. Erdheim2,3 and Cellina4 had concluded that an elastic tissue defect with mucoid accumulation in the aortic wall was the cause of dissection. However, they utilized no formal controls and the necessary intensity of elastic damage in "mucoid medionecrosis" over that in the "normal" population was unknown. A number of observations antedating the studies of Erdheim and Cellina had indicated that essentially normal arterial walls were characterized by marked variation in chromatric mucopolysaccharide content.6-8 Gsell's demonstration2 of a smooth muscular aortic defect in dissecting aneurysm was likewise recorded without a report of control studies. Subsequently, Rottino9,10 demonstrated that this aortic lesion was common in both the presence and absence of dissection. Nonetheless, Gore11 and de Faria12 suggested that smooth muscle degeneration played a major role in the pathogenesis of dissecting aneurysm.

There is clear evidence that laminar foci of muscle loss10,13,14 and accumulation of mucopolysaccharide occur in a substantial proportion of aortas without dissecting aneurysm. Large quantities of mucopolysaccharide may be seen in essentially normal medias or may appear in association with degenerated or damaged elastica.13 Failure to demonstrate degenerative alterations in most instances of dissecting aneurysm led Hurley14 to postulate a biochemical abnormality in collagen despite a normal histologic appearance.

It was obviously essential that a large number of "normal" aortas be obtained at random and examined histologically and histochemically in order to establish a control baseline. This having been accomplished,13 factors of possible consequence in the pathogenesis of dissecting aneurysm were evaluated by contrasting clinical and morphologic features in cases of dissecting aneurysm with those in the control group. These observations constitute the present report.

Material and Methods

Fifty-six examples of aortic dissecting aneurysm were procured from the necropsy files at the Cincinnati General Hospital; only 35 were found suitable for use in this survey. Required for selection were: (1) Adequacy of gross description or availability of the actual specimens. (2) Available sections of both the dissection tract (at least two through the entire wall) and the area of internal rupture (at least one from the free margin). Three cases were included in which no internal tear was encountered; in these, multiple sections of the dissection tract were available. (3) Adequacy of clinical information (table 1).

All sections, fixed either in buffered 10-per cent formalin or Zenker's acetic acid were examined by conventional histologic and by a battery of histochemical technics.13 The content of elastic tissue, collagen, fibrin, glycogen, and acid and neutral mucopolysaccharides was quantitated and the results were tabulated. The lesions were then graded as to the intensity of alteration in the area of intimal rupture and other morphologic features and clinical data were evaluated.

Results

Although it appeared that dissection could proceed readily in a histologically normal aortic media, one of two seemingly pertinent
lesions were invariably observed at the area of internal rupture; these were "arteriosclerosis" (group I) and "idiopathic medial degeneration" (group II). The designation "arteriosclerosis" (fig. 1) was based upon the presence of severe (3-4+) atherosclerosis with extensive secondary medial damage. It is noteworthy that, in half of the cases, such a plaque was observed extending from the intima into the dissection tract (fig. 1) at the point of internal rupture.

In "idiopathic medial degeneration" (group II) severe medial damage of the type attributable to arteriosclerosis or such disorders as syphilis was not found (fig. 2). Highlighting the process was fragmentation of the elastica and massive accumulation of mucoid substance. Prominent in the area of intimal tear (fig. 3), the lesion varied in severity from segment to segment in the same aorta. Histochemically, the mucopolysaccharide did not differ qualitatively from that seen in controls. Minor degrees of atherosclerosis were observed in this group. Indeed, in one instance, a plaque was detected at the site of intimal rupture.

The results of the survey are tabulated in table 1. There is some evidence to suggest a basic difference between group I and group II although the latter was quite small (five cases). In group II, the patients were younger and three of five were females (usual necropsy ratio, 1.8 males; 1 female). All save one were Negro patients (usual necropsy ratio, 2.1 white patients; 1 Negro patient). Only one patient was known to have had hypertension (average incidence at necropsy, about 42 per cent). In all cases there was dilatation of the aorta or aortic ring by actual measurement; in one, there was clinical evidence of aortic insufficiency. Despite the infrequency of hypertension, marked cardiomegaly was invariable. Internal and external aortic ruptures were present in all cases. Because there were so few cases in the group, a statistical evaluation of these observations would have little significance. Analysis of a larger number of instances of dissecting aneurysm of this variety is required to provide definite confirmation of the age, sex, and racial predilection of the lesion, although it appears to be distinctive histologically.

Group I (30 cases) comprised generally older patients, and the sex ratio paralleled

Figure 1
An area of severe atherosclerotic damage serves as the point of entry for an aortic dissection. Medial elastica is seriously damaged. (Verhoeff elastic-Van Gieson's stain; ×40.)
Table 1

Characteristics of 35 Cases of Dissecting Aneurysm

<table>
<thead>
<tr>
<th>Observation</th>
<th>Arteriosclerotic (I) (30)</th>
<th>Medial degeneration (II) (5)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Incidence*</td>
<td>Incidence (%)</td>
</tr>
<tr>
<td>Age 40 (youngest 28)</td>
<td>0/30</td>
<td>0</td>
</tr>
<tr>
<td>40-59</td>
<td>13/30</td>
<td>43</td>
</tr>
<tr>
<td>60</td>
<td>17/30</td>
<td>57</td>
</tr>
<tr>
<td>Sex M/F (average 1.8/1)</td>
<td>19/11</td>
<td>1.9/1</td>
</tr>
<tr>
<td>Race W/N (average 2.1/1)</td>
<td>12/18</td>
<td>0.6/1</td>
</tr>
<tr>
<td>Hypertension (Average incidence 41.6 per cent)</td>
<td>29/29</td>
<td>100</td>
</tr>
<tr>
<td>Aortic insufficiency</td>
<td>1/30</td>
<td>3</td>
</tr>
<tr>
<td>Cardiomegaly</td>
<td>30/30</td>
<td>100</td>
</tr>
<tr>
<td>Absolute dilatation of aorta</td>
<td>12/23</td>
<td>52</td>
</tr>
<tr>
<td>Internal tear present</td>
<td>27/30</td>
<td>90</td>
</tr>
<tr>
<td>Site, internal tear</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asc. thoracic</td>
<td>18</td>
<td>67</td>
</tr>
<tr>
<td>Dese. thoracic</td>
<td>7</td>
<td>26</td>
</tr>
<tr>
<td>Abdominal</td>
<td>2</td>
<td>7</td>
</tr>
<tr>
<td>Short tract</td>
<td>12/30</td>
<td>40</td>
</tr>
<tr>
<td>Asc. thoracic</td>
<td>7</td>
<td>24</td>
</tr>
<tr>
<td>Dese. thoracic</td>
<td>4</td>
<td>13</td>
</tr>
<tr>
<td>Abdominal</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>External rupture</td>
<td>14/30</td>
<td>47</td>
</tr>
<tr>
<td>Tract in outer one-third media</td>
<td>24/30</td>
<td>80</td>
</tr>
<tr>
<td>Muscle defects (total)†</td>
<td>10/30</td>
<td>33</td>
</tr>
<tr>
<td>(old)</td>
<td>4/30</td>
<td>13</td>
</tr>
<tr>
<td>Tear through plaque</td>
<td>15/30</td>
<td>50</td>
</tr>
<tr>
<td>Age of Tract</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recent</td>
<td>18/30</td>
<td>60</td>
</tr>
<tr>
<td>Early organization (&lt; 7 days)</td>
<td>2/30</td>
<td>7</td>
</tr>
<tr>
<td>Old</td>
<td>10/30</td>
<td>33</td>
</tr>
<tr>
<td>Syphilis</td>
<td>2/30</td>
<td>7</td>
</tr>
</tbody>
</table>

*Where fractions are used, numerator = number of positive observations, denominator = number of cases in which data were available.
†See text; 8 of 13, acute, infarct-like necrosis.

that of the overall necropsy population. There were 12 white and 18 Negro patients. Hypertension had been present in all instances. In about one third, the dissection tract did not involve the ascending aorta, and, in one instance, dissection was restricted to the abdominal aorta. The three examples of dissecting aneurysm without intimal tear fell within this group, and, in nine cases, the intimal tear occurred in an area other than the ascending aorta. In more than half, there was no external rupture. Chronicity of the dissection was a feature in about one third of the cases. In almost half, the aortic valve was not dilated by actual measurement; aortic insufficiency was clearly present in one case with dilatation. In 50 per cent, an arteriosclerotic plaque penetrated deeply into the media at the site of internal rupture. Two aortas revealed the changes of syphilitic aortitis (figs. 4 and 5).

Characteristics common to both groups included cardiomegaly and dilatation of the aorta. Short and long dissection tracts were encountered with equal frequency in both
Marked accumulation of metachromatic material is seen in same section as depicted in figure 2. (Toluidine blue stain.) Black and gray areas represent pink-staining material. (X 80.)

groups. Microscopically, the severity of the alteration encountered varied greatly from area to area in both groups. It was obvious in many sections that the dissection was proceeding through an area of the media without other histologic abnormality (fig. 6). In all but six instances, all in group II, the dissection tract was located in the outer one third of the media.

Pre-existing medial muscle defects ("linear acellular foci") were found with approximately the same frequency (13 per cent) as in controls. These lesions were of linear nature and exhibited loss of smooth muscle nuclei. They were characterized by complete acellularity, loss of acid mucopolysaccharide and compression of otherwise intact elastic lamellae (fig. 7). In eight instances, acute, infarct-like, linear, necrotizing lesions of the media were observed (fig. 8). These were distinguished from the acellular foci by their greater extent and acute character. They tended to occur more frequently in aortas with large intramural hematomas. Furthermore, shadow structures of spindle cells, often with dying nuclear forms, were invariably observed. The lesions were always superficial to the intramural hematoma. Several cell layers adjacent to both the intima and the dissection tract, remained intact, suggesting that they were protected from ischemia by their proximity to intramural and intraluminal columns of blood. Elastic lamellae were not compressed, as in pre-existing muscle defects. Additionally, acid mucopolysaccharides manifested a peculiar patchy distribution resulting from aggregation or clumping. This contrasted sharply with the almost total absence of mucopolysaccharide in the linear acellular foci. The elastic plates remained intact in both lesions.

Discussion

In elucidating the pathogenesis of dissecting aneurysm, this survey appeared to justify the following interpretations:

It was evident that dissection might propagate readily in a histologically normal media (fig. 6). In all 20 dissecting aneurysms of recent onset, this was observed in at least one section. In a majority, aortic dilatation was evident. This was the case in all five instances in group II and in 52 per cent of the cases.
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in group I. Indeed, it is suspected that a much higher incidence would have been observed, if measurements of the circumference of the aorta at the site of internal rupture had been recorded in all protocols. In only eight instances, all with dilatation, was this information recorded; in others, the circumference of the aortic valvular ring was the only such measurement available. Dilatation did not appear to be a reflection of chronicity of dissection; in all but two cases with dilatation, the process was less than 7 days old. Dilatation has the capacity to increase the tension on the intima and underlying media by increasing the lateral hydrostatic pressure against the aortic wall (Bernoulli effect), thus promoting the initial tear. This well-recognized physical principle is related to the slowing of velocity resulting from increasing diameter and has also been invoked as the explanation of the inexorable enlargement of saecular aortic aneurysms. Another physical principle, perhaps equally applicable, is that of Laplace. This law holds that the tension on the wall of a liquid-filled spherical or cylindrical chamber varies directly with the diameter. It is noteworthy that the tear, in most instances, occurs in the ascending segment of the thoracic aorta, ordinarily widest in diameter.

The effects of the two underlying aortic lesions observed in this study are regarded primarily as favoring dilatation, tortuosity, and rigidity of the aorta, thus creating conditions promoting the initial tear. The ability of dissection to propagate through histologically normal media suggests that aortic wall lesions have no further significance.

The cases in group II (figs. 2 and 3) numbered only five. Three of these were females, four were Negro patients, and four were less than 60 years of age. Degeneration of elastica with massive mucopolysaccharide accumulation was present, and dilatation of the aorta was prominent. The larger group (group I) manifested severe arteriosclerosis. Even utilizing routine sections, half of these cases showed severe medial damage attributable to a penetrating atherosclerotic plaque at the site of the internal rupture. It seems reasonable to presume that a higher incidence would have been encountered if additional sections

Figure 5

Dissection tract at the junction of the outer and middle third of an aorta occurs in an area showing inflammation and fibrosis typical of syphilitic aortitis. (Hematoxylin and eosin stain; × 80.)

Figure 6

Dissection proceeds through an area of completely normal media. Elastic fibers are regular and show normal continuity. Note position with respect to adventitia (above). (Verhoeff elastic-Van Gieson's stain; × 80.)
had been available, since the initial tear often extended for several centimeters transversely. It is suggested, therefore, that the column of blood usually enters the wall by penetrating through a deeply seated atheromatous plaque. Although atherosclerosis is generally regarded as intimal in origin, advanced lesions may cause severe damage to the elastica. A previous study showed a high incidence of damage to the media of the ascending aorta resulting from atherosclerosis, often unrecognized on casual gross examination. In cases of dissecting aneurysms, necropsy reports repeatedly characterized the ascending aorta as the seat of minimal or moderate atherosclerosis but microscopic study revealed severe atherosclerosis in the vast majority. Histologically, the process did not differ from the classical picture described in atherosclerosis. In particular, the amount or type of mucopolysaccharide did not differ from that seen in controls with similar degrees of atherosclerosis.13

By virtue of its high incidence, hypertension must be assigned a major role in precipitating dissecting aneurysm. It was present in all examples of arteriosclerosis; in only one of the cases with primary medial degeneration was there blood pressure elevation. The hypertension probably is not the cause of the underlying mural lesion although it may well increase the severity or promote the development of atherosclerosis; a more likely effect is that of the increased intraluminal tension previously noted. Additionally, elevation of blood pressure may promote dissection once the intimal tear has occurred.

That muscular defects are responsible for dissecting aneurysm appears highly unlikely. Their mere presence in individuals without dissecting aneurysms need not rule out an auxiliary role; on the other hand, such an assumption would require a higher incidence in dissecting aneurysm than in the control group, and this was not observed. More im-

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**Figure 7**

*In a plane superficial to a fresh dissection tract (above) is observed a lamina acellular defect, with compressed, but otherwise normal elastica. (Hematoxylin and eosin stain; \( \times 80 \).)*

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**Figure 8**

*A linear area of recent necrosis is seen superficial to a dissection tract (below). A few layers containing persistent spindle cells and neutrophils are present in proximity to the tract. More superficially (above), several layers of cells close to the intima survive. (Hematoxylin and eosin stain; \( \times 80 \).)*
important points may be scored against the muscle defect as a factor in dissecting aneurysm. The defects ordinarily appear in the inner one third of the media, while the dissection characteristically affects the outer one third (fig. 6). Additionally, the amount of muscle in the normal aorta is of such order as to indicate that its function in the maintenance of the integrity of the wall of the aorta is negligible. The success of aortic homografts devoid of viable muscle would tend to confirm this belief. The emphasis by Gsell, Rottino, and Gore on muscle defects as a cause of dissecting aneurysm appears to relate to both acute lesions and older foci of linear muscle loss. The acute necrotizing lesions in our cases resembled those described by de Faria but are considered to result from separation of a portion of the media from its blood supply by the intramural hematoma. Apparently identical lesions have been recorded in experimentally induced dissecting aneurysms in dogs. Certainly the possibility exists that the muscle defects represent an end stage of the acute necrotizing lesion, but there was no specific evidence for this in the histologic sections. Since the incidence of the older muscular defects in cases with dissecting aneurysm was no higher than in controls, it appears unlikely that they are related to previous healed dissections.

In three cases, there was no communication of the intramural hematoma with the lumen. This need not necessarily be regarded as evidence that the dissection in all cases must commence in the wall and rupture into the lumen. Indeed, it is not entirely certain that one or more small perforations either in the aorta or its branches may not be missed in such instances. The stresses and pressures promoting perforation would tend to exert their maximum effect in the opposite direction; it is illogical to presume that rupture would ordinarily occur from an area of lesser (intramural blood vessels) to an area of greater pressure (lumen of the aorta). On the other hand, it is possible that rupture of intramural blood vessels may occasionally lead to dissection confined to the wall without communication with the lumen. In view of the failure to demonstrate a consistent lesion in the media in the area of dissection, there is scant reason to hypothesize that dissection must start here, since this assumption offers no explanation of the initiation of the process.

In the study of Blanton, Muller, and Warren dissecting aneurysms were readily produced in dogs by creating a surgical "intimal tear." The authors were unable to produce proximal dissection by their technic, and concluded that an intramural hematoma usually must precede the intimal tear in naturally occurring dissection. However, they produced their initial tear in the descending thoracic aorta, and, in only three cases did they attempt to make the experimental animals hypertensive, in each after the completion of the procedure. It was noted that dissection proceeded more rapidly and more violently in the hypertensive animals. This study did not accurately simulate the conditions in human dissecting aneurysm, since the intimal rupture in the present series was usually in the ascending aorta and practically all individuals were hypertensive. Nonetheless, it is noteworthy that proximal dissection did not occur in three of the seven instances in our survey in which the initial tear occurred in the descending thoracic aorta.

The presence of two examples of syphilitic aortitis in this and other reports indicates that syphilis, per se, neither limits nor protects against dissecting aneurysm. Indeed, the loose collagenization and mucopolysaccharide accumulation characteristic of syphilitic aortitis in an active phase need offer no barrier to dissection. The severe aortic dilatation and concomitant atherosclerosis are factors that tend to promote dissecting aneurysm.

Dissecting aneurysm without significant medial elastic degeneration has been described repeatedly since the reports of Erdheim. It appears probable that the majority of instances of "mucoid medionecrosis" reported are either within the range of normal variation, or are the result of, rather than the cause of, the dissecting aneurysm. In the latter class may be included the mucopolysac-
charide accumulation seen in the granulation tissue characteristic of dissection tracts in a phase of early organization. In this survey, while arteriosclerosis predominated as an underlying lesion, one seventh of the cases were believed to represent examples of the medial degeneration by virtue of the presence of both severe elastic degeneration and mucopolysaccharide accumulation. In any case, it seems evident that the popular simple hypothesis of Erdheim,\textsuperscript{3-4} of an underlying mucoid medionecrosis predisposing to dissection fails to explain any but a small portion of instances of this disease. Similar alterations are seen in individuals with the Marfan syndrome\textsuperscript{25,26} and in animals given lathyros extract or beta-amino-proprionitrile.\textsuperscript{27-30} However, the formation of small saccular aneurysms, complete and incomplete rupture of the aorta, and short dissections are more common in such circumstances than are the long tracts typically seen in classical examples of dissecting aneurysm. That such is the case is not surprising, since in elastic degeneration the blood in the lumen is ordinarily prevented from entering into the wall by an intact intima, and the blood pressure is not usually elevated.

Observations closely paralleling those in this report were recorded by Hurley.\textsuperscript{14} He was unable, however, to correlate dissection with arteriosclerosis, although he observed a similar incidence of elastic degeneration. As in the present survey, there was no consistent histologic abnormality in the aorta adjacent to the dissection tract. The failure to observe a specific microscopic abnormality associated with the tract caused him to hypothesize that a biochemical abnormality in the collagen without histologic counterpart was present. Hurley's survey did not include any study of the clinical aspects or gross findings in dissecting aneurysm. As a result, the relationship of hypertension and aortic dilatation to dissecting aneurysm was not analyzed. Furthermore, Hurley concluded that the wall of the normal aorta could not readily be dissected while that of aortas with dissecting aneurysm stripped readily. These conclusions were largely based upon the experimental ob-

servations of others that do not simulate conditions in vivo.

Robertson and Smith\textsuperscript{31} found that the minimum pressure required to "dissect" (i.e., produce a bleb) in the wall of the aorta was 230 mm. Hg. Their study, however, was performed by inserting a hypodermic needle into the wall of an aortic strip and exerting steady hydrostatic pressure. Such factors as influence of lateral and pulsatile pressure, size and location of the orifice, shearing force of the motile column of blood, and the effect of coiling and uncoiling were not reflected by this technic. Examples of dissecting aneurysm were not studied. The successful production of dissecting aneurysms in normotensive dogs by creating a luminal communication with the wall\textsuperscript{22} seems to refute the concept that unusually high pressures are required for dissection.

In the study of Cleland,\textsuperscript{32} a mural-stripping technic was utilized to ascertain if there was any specific plane of cleavage in the aorta. The author stated that the greatest tendency to strip was localized in the outer portion of the media in most instances. Milazzo,\textsuperscript{33} using a similar method, measured with a dynamometer the force required to separate layers of the aorta. Although he concluded that there was no predilective site for cleavage, his scattergram clearly indicates that a large majority of instances stripped in the outer media. Obviously, neither study simulates conditions in life, but both do tend to indicate a normal plane of dissection in the outer media. Similarly, in a study of aortas performed for a different purpose,\textsuperscript{34} the outer few lamellae of elastica along with the adventitia could be stripped readily from the remainder of the wall. Examples of spontaneous dissection of the aorta without abnormality in the elastica have been observed both as antemortem\textsuperscript{35} and postmortem\textsuperscript{36,37} phenomena.

In accordance with these observations, the following hypothesis concerning the pathogenesis of dissecting aneurysm is postulated (table 2): The blood usually gains entrance to the aortic wall by passage through the intima. In at least half of the cases, and prob-
### Table 2

**Factors Apparently Influencing Dissecting Aneurysm**

<table>
<thead>
<tr>
<th>Phenomenon</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>b. &quot;Postage-stamp&quot; perforation of vasa vasorum in outer media creates natural plane of cleavage.</td>
</tr>
<tr>
<td>2. Arteriosclerosis</td>
<td>2. a. Weakening of wall results in dilatation.</td>
</tr>
<tr>
<td></td>
<td>b. Diminished flexibility lowers resistance to shearing force.</td>
</tr>
<tr>
<td></td>
<td>c. Plaques form weak points permitting entry of column of blood.</td>
</tr>
<tr>
<td></td>
<td>b. Weakening of elastica results in dilatation.</td>
</tr>
<tr>
<td></td>
<td>c. Weakening of intercellular collagen, increase in ground substance facilitates dissection?</td>
</tr>
<tr>
<td>4. Dilatation of aorta</td>
<td>4. a. Whatever the cause, increases proportion of lateral pressure, predisposes to tear.</td>
</tr>
<tr>
<td>5. Hypertension</td>
<td>5. a. Not related to &quot;medial degeneration.&quot;</td>
</tr>
<tr>
<td></td>
<td>b. May increase severity of atherosclerosis.</td>
</tr>
<tr>
<td></td>
<td>c. Prime effect appears to be purely hydrostatic, increasing force tending to create tear, propagate dissection.</td>
</tr>
<tr>
<td></td>
<td>d. Intramural rupture of vasa vasorum in cases with no intimal tear.</td>
</tr>
</tbody>
</table>

ably more, perforation occurs through an area in which an atherosclerotic plaque is present. The wall of the aorta in the area of laceration has previously been damaged by atherosclerosis with or without syphilis or idiopathic medial degeneration, producing dilatation and rigidity. This results in a rise in lateral pressure and increased tension against the aortic wall. Augmented by hypertension, these forces are adequate to cause a tear through the damaged intima, most often in the ascending thoracic aorta. Once the column of blood enters the wall, it seeks a plane of cleavage, usually the outer one third of the media, at the point where the vasa vasorum ramify. At this point, dissection for greater or lesser distances readily occurs through normal media in a fashion analogous to the tearing at the perforations in a sheet of postage stamps. External rupture may occur at any point, since only a thin layer of adventitia and media separates the blood from the outside.

**Summary**

A group of 35 cases of dissecting aneurysm was studied both clinically and pathologically, in order to evaluate the role of alterations in the aortic wall in the pathogenesis of dissecting aneurysm. It was concluded that the commonly observed dilatation and hypertension promoted the initial tear by increasing the tension on the intima of the aorta. The usual cause of the aortic dilatation in this study was severe atherosclerosis; a much smaller group revealed an idiopathic medial degeneration; muscular lesions were regarded either as insignificant or as a result of the dissection. Although in many instances the initial perforation occurred through an atherosclerotic plaque, dissection proceeded readily through histologically normal media. Accordingly, it was concluded that the two histologic lesions encountered produced their effects largely by provoking dilatation of the aorta rather than by promoting propagation of the dissection.

**References**

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