CLINICAL PROGRESS

Dissecting Aneurysm of the Aorta
The Clinical Features of Thirty Autopsied Cases

By BLAIR D. EBB, M.D., AND I. FRANK TULLIS, M.D.

IN RECENT years the diagnosis of dissecting aneurysm of the aorta has undergone a transition in significance. Formerly, this entity was considered a rather hopeless but academically interesting phenomenon. Today, however, with the growth of experimental and clinical vascular surgery, the diagnosis of dissecting aneurysm of the aorta has become a necessity from a practical and therapeutic standpoint. Already the successful surgical management has been reported from several centers.1–4 A distinct increase in the natural survival rate of 10 per cent5 cannot be expected, however, unless the frequency of antemortem diagnosis is increased. The incidence of antemortem diagnosis has been reported from Domzalski's 0 per cent6 to Logue's 83 1/3 per cent,7 but in major series the average incidence appears to be between 30 and 50 per cent, and this rate appeared only after the monograph by Shennan in 1934.8 Only 27 per cent of the 30 autopsied cases at the John Gaston Hospital in the last 10 years were diagnosed ante mortem. It is the purpose of this paper to review the clinical experiences with dissecting aneurysm of the aorta in autopsied cases at the John Gaston Hospital in the last 10 years, with emphasis on the appearance of the patient and associated conditions.

Historically Morgagni9 in 1760, Maunoir in 1802,10 and Laennec in 182611 described the lesion in the aorta, and Pennock12 in 1839, and Peacock13 in 1843 gave our knowledge the foundation for the first correct antemortem diagnosis reported in 1855 by Swaine and Latham.14 Further progress in clinical concepts was slow, however, until Erdheim's description of medionecrosis.15 Only 6 cases had been diagnosed prior to Shennan's analysis of 300 cases in 1934, but since that time the entity has been recognized with increasing frequency. Interestingly, King George II was reported16,17 to have died of aortic dissection while straining at stool.

Incidence

The actual incidence of dissecting aneurysm has not been determined but it appears to be a more common process than originally thought. The literature through 1947 was reviewed by Levinson and co-workers,18 who could find only 734 cases. In the literature through 1957, we found an additional 682 cases, and with the 30 presented here the total in the literature is approximately 1,416 cases.

The autopsy incidence has varied between 1 per 74519 and 1 per 70.20 The latter series, however, was a report of coroner's cases in which the incidence of sudden unexpected death is higher than in the usual hospital series. In the past 10 years at John Gaston Hospital 30 cases have been found among 9,280 autopsies, an incidence of 1 per 309. In a collection of series in which the figures were given18,21–25 there were 151 cases in 46,869 autopsies, an incidence of 1 per 310 autopsies.

The age range is variable but many aortic dissections have been reported in children and 1 was found in a 14-month-old child.26 One case was reported at 100 years of age8 and 3 of our patients were 85 years old. It appears

From the Department of Medicine, University of Tennessee, and the City of Memphis Hospitals, Memphis, Tenn.
to be most common, however, in the fifth and sixth decades, but as many as 24 per cent have been reported under the age of 40. In our series the age ranged from 36 years to 85 years, with an average age of 56 years. Only 3 were under 40 years of age. Men generally predominate 2 to 1.

Pathology
A dissecting aneurysm of the aorta is a hemorrhage in the aortic media with separation of the layers of the aortic wall. This may or may not be associated with an intimal tear, which most commonly occurs in the ascending aorta just distal to the aortic valves. The tear may occur at the great vessels in the aortic arch or in the descending or the abdominal aorta. Grossly the aorta presents a definite but variable increase in external size and is usually saccular or fusiform in shape. The clinical manifestations usually are a function of the site of the aortic tear and the extent of the dissection. Although the medial hemorrhage usually extends peripherally, proximal dissection may occur, often terminating, as is reported in 78 per cent of one series, with rupture into the pericardial sac and cardiac tamponade as the cause of death. Ruptures into the pleural space (usually the left), into the retroperitoneal space, or into the esophagus have been reported as causes of death, but "re-entry" of the dissecting hematoma into the original aortic lumen may occur, giving rise to the "double-barrelled" aorta, which often is compatible with many years of useful life. Occlusion of branches of the aorta may cause death secondary to the affected organ or system.

Histologically a focal degenerative process, cystic medionecrosis, is seen within the media. Gore described 2 types of medionecrosis, 1 of which usually occurs under the age of 40, consisting predominantly of elastic fiber degeneration, and the other occurs in the older age group in which muscle fiber degeneration predominates. Erdheim's original description reported rarefaction of the media, absence of external elastic laminae, muscle atrophy with cystic spaces between irregularly branching fibers, and slight thickening of the intima.

Cystic medionecrosis may be due to disease of the vasa vasorum and Gore thought that rupture of the thin-walled friable vasa vasorum caused intramural hemorrhage that ruptured the intima. The pressure of the systolic blood flow then may cause extension of the dissection down the loosely connected media.

Etiology and Associated Conditions
There are certain conditions clinically associated with dissecting aneurysm of the aorta that may aid in establishing the diagnosis. Most outstanding is the Marfan's syndrome, characterized by abnormally long and thin extremities, fingers, and toes, with relaxation of the ligaments, hypotonic musculature, dolichocephalic head, kyphosis, funnel-chest, subluxation of the lenses, high-arched and sometimes cleft palate, and prominent ears. Pulmonary anomalies are reported. Other congenital cardiovascular anomalies are seen in the Marfan's syndrome. The familial incidence of Marfan's syndrome suggests some hereditary aspect of dissecting aneurysm. Variants of the Marfan's syndrome occur and any of the many characteristics may be present. Two of the patients in this series exhibited the clinical findings of the Marfan's syndrome.

An increased frequency of dissecting aneurysm has been reported with pregnancy. Of the 49 women reported in 1 series under 40 years of age, 24 (or 50 per cent) were associated with pregnancy. This appears to be more common in the last trimester but occurrence during the postpartum period is noted. There was 1 woman, a Negro aged 38, in this series who was 2 weeks postpartum.

The frequent association of hypertension with dissecting aneurysm is well known but its exact role remains open to question. It may
provide the driving force for medial dissection once the intimal tear occurs. The role of atherosclerosis is even more nebulous, and the earlier concept of disturbed nutrition of the media through atherosclerosis has not been well substantiated.

Coarctation of the aorta enhances the possibility of medionecrosis and dissecting aneurysm. Medionecrosis is usually found proximal to the coarcted segment, presumably due to increased intraluminal pressures. One case of complete aortic atresia at the usual site of adult coarctation with dissection has been reported.

Recently McKusick reported 4 cases of dissecting aneurysm associated with aortic stenosis, and cystic medionecrosis was present in all cases. Other cases have subsequently placed increased emphasis on this associated lesion, and Heath, Edwards, and Smith offered the explanation that the high velocity jet of blood passing the aortic valves strikes the more slowly moving blood distally, causing a lateral displacement of force which then results in structural fatigue and possibly medionecrosis.

The role of trauma has been suggested but is unclear. Trauma even in the form of a fishbone in the esophagus has been considered responsible for dissection. Hypothyroidism has been suggested as a preexisting causative factor, and it has even been proposed that the high incidence of dissecting aneurysm during pregnancy may be due to failure of the thyroid gland to undergo its physiologic hyperplasia. Although 1 of our cases had had a thyroidectomy 17 years before her dissection, a relationship of thyroid function to aortic dissection has not been established.

It is generally agreed that syphilis is not an etiologic factor in aortic dissection. It has been suggested that syphilis hinders the development of aortic dissection by scarring of the media. A history of syphilis or positive serology was found in 10 of our 30 cases but no lesions of cardiovascular syphilis were seen.

Aortic dissection also has been reported in giant cell arteritis, in tuberculous aortitis, due to translumbar aortography, and associated with ganglionic blocking drugs, ergotamine tartrate, and even cortisone acetate in hamsters. The role of an experimental diet of Lathyrus odoratus (sweet pea) in the production of dissecting aneurysm in rats has been found, and the toxic principle appears to be B-aminopropionitrile.

**Clinical Features**

An entity which has frustrated so many physicians obviously would be the subject of many different schemes of classification. One such classification is based on duration of survival. The acute type terminates fatally within 24 to 48 hours; the subacute type may be more gradual, persisting for a period of days or weeks, and the chronic or "healed" type may survive for months or years. We prefer, however, a classification based on the clinical features and the appearance of the patient, as modified from Domzalski, Warren and McGowan, or Baer. We include 6 groups of manifestations: cardiac, peripheral arterial, pulmonary, abdominal, renal, and neurologic. The involvement of several systems may be the first diagnostic lead.

**Presenting Complaint**

Pain was the presenting complaint in 21 or 70 per cent of our patients, in the chest in 12, abdomen in 5, and extremities in 4. Baer reported that 51 per cent of 86 patients had pain and Levinson and associates reported pain in 78 per cent of 58 cases, but in only 29 per cent was it in the chest. The chest pain characteristically was described as sudden, substernal, radiating through to the back, penetrating "gas-like," sharp, squeezing, crushing, tearing, and smothering. It occasionally radiated to the jaw, the shoulders, or the arms. The onset characteristically was sudden and in several instances was related to exertion, e.g., chopping wood, painting from a ladder, and so forth. In several instances the pain was described as being at the peak of intensity at the onset. This has been considered of major importance in differentiating this entity from acute myocardial infarction, in which pain gradually becomes more severe. The pain also frequently radiates into the abdomen and
midepigastrium. It must be remembered that silent dissection may occur without any pain or, on the other hand, the general condition of the patient is such that an adequate history cannot be obtained.

Abdominal or epigastric pain was also of sudden onset, severe, radiating to the back, colicky, constant or intermittent, vice-like, tearing; and the patient characteristically had difficulty in localizing the exact point within the epigastrium.

Pain in the extremities represented a symptom of arterial occlusion. Coldness, numbness, tingling, and weakness in the affected extremity also were mentioned.

Five patients (17 per cent) were admitted as neurologic or psychiatric problems. One patient, a 47-year-old man with Marfan’s syndrome, was admitted to the psychiatric service for despondency and depression of recent onset. A murmur had been described approximately 1 year previously. The course was progressively downhill, with development of congestive failure, renal failure, and recurrent hemotherax. At postmortem examination a chronic “healed” dissecting aneurysm was found. Four patients (13 per cent) were admitted with strokes, 2 with right and 2 with left hemiplegia.

Three patients (10 per cent) were admitted with longstanding congestive heart failure and died of intractable failure. This is not uncommon in the chronic dissection when re-entry has taken place.

One patient was admitted to the orthopedic service after a fall. A small dissection of the abdominal aorta was found at autopsy but death was due to pulmonary embolus.

Secondary complaints included dyspnea, vomiting, hemoptysis, edema, apprehension, pleuritic pain, epigastric distress, sweating, syncope, polyuria, hematuria, cough, and weakness.

The general appearance of the patient as one who is suffering a catastrophe may give the initial lead. Twenty-three of our patients (77 per cent) were acutely ill; 12 presented as acute cardiovascular episodes (see table 1). Four were admitted to the surgical service as problems of peripheral arterial occlusion, and 3 were considered to be patients with acute surgical abdomens.

Cardiac Manifestations

Hypertension or heart disease existed in 21 patients (70 per cent) and hypertension by history or examination was present in 16. Tachycardia was noted in 19 cases. Not infrequently the patient appeared to be in clinical shock but the blood pressure was elevated. Cardiomegaly was found in 19 cases (63 per cent).

Cardiac murmurs, particularly if known to be of recent origin, are of diagnostic importance (table 2). An aortic diastolic murmur has been classically found in dissecting aneurysm when the ascending aorta is involved. This murmur has occurred in from none6 to 56.2 per cent72 in different series, but in a larger series 28 per cent had aortic diastolic murmurs,18 similar to our incidence of 27 per cent (table 3). We wish to call attention to the high incidence of aortic systolic murmurs found both in Levinson’s and in our series. Aortic murmurs, systolic, diastolic, or both, occurred in 41 per cent of the 2 series.

The cause of these murmurs has been the subject of much dispute. It has been suggested that leakage of blood through the intimal tear produces the murmur.73 Others considered that the medial hematoma distorts the aortic valve ring, resulting in insufficiency of the valve.18, 74, 75 It is also possible that a transverse tear in the intima near the aortic commissure loosens the intimal support for the cusp, allowing sagging of the cusp in the direction of the ventricle and insufficiency of the valve.76 It is likely that several mechanisms may produce the murmur and that they may vary in different cases. The existence of murmurs is very important for the diagnosis and in establishing the extent of the process. Our percentage of antemortem diagnosis closely parallels the percentage of murmurs of aortic insufficiency.

Friction rubs of pericardial origin usually are considered rare in dissecting aneurysm and none was encountered in our series. How-
ever, in 505 autopsied cases of Hirst, Johns, and Kime77 friction rubs were found in 22, or 4 per cent.

Cardiac tamponade often occurs from rupture of the aneurysm into the pericardial sac. As most dissections begin in the ascending aorta, the proximity of the tear to the pericardial reflection would explain the high incidence of rupture into the pericardial sac (78 per cent) in 1 series.27 Pericardial aspiration may be diagnostic as well as therapeutic.77

Peripheral Arterial Manifestations

Oclusion of the peripheral arteries often results from the engorged dissecting hematoma and the peripheral arteries may give a number of interesting signs. A significant difference in blood pressure between 2 extremities is characteristic. In our series pulses in some peripheral arteries were absent in 13, not remarkable in 5, and were not examined in 12. "Migratory"77 or transient arterial occlusion may occur78,79 and delay in the pulse beat in the peripheral arteries8 and reduplication of the carotid pulse have been seen.80 Pulsion in either sternoclavicular joint has been described81 and tenderness below a dissecting aneurysm is known as the Weiss-Sosman sign.82

Extracardiac Manifestations in the Chest

These include dyspnea, cough, hemoptyis, pleural pain, and pleural effusion or hemothorax. Most commonly the pleural effusion is on the left. The mistaken diagnosis of pneumonia, malignant disease, or pulmonary infarction is not uncommon. One of the patients in this series with hemothorax had 9 thoracenteses, 4 of which contained atypical cells, but at autopsy no malignant disease was found.

Abdominal Manifestations

The most common abdominal manifestation is the history of pain, but hematemesis,83 melena,84 distention of the sigmoid suggesting obstruction,83 and volvulus85 may be seen. Cholecystitis86 and pancreatitis84 are often suspected. Three of our patients were admitted to the surgical service for suspected acute surgical conditions of the abdomen, suggestive of cholecystitis. No abdominal operations were done, however.

Renal Manifestations

Involvement of the kidneys may result from aortic obstruction proximal to the renal arteries or by dissection of the renal arteries,87 and is manifested by flank pain, hematuria, and uremia. Occasionally ureteral calculus is suspected, and the distribution of flank pain

Table 1

<table>
<thead>
<tr>
<th>Apparent system involved</th>
<th>Number of cases</th>
<th>Per cent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute catastrophic C-V episode A. With peripheral artery occlusion</td>
<td>12</td>
<td>40</td>
</tr>
<tr>
<td>Acute surgical abdomen</td>
<td>3</td>
<td>10</td>
</tr>
<tr>
<td>Acute peripheral artery embolism</td>
<td>4</td>
<td>13</td>
</tr>
<tr>
<td>Chronic congestive heart failure</td>
<td>2</td>
<td>7</td>
</tr>
<tr>
<td>Chronic renal disease with uremia</td>
<td>3</td>
<td>10</td>
</tr>
<tr>
<td>Psychiatric problem</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Orthopedic problem (trauma)</td>
<td>1</td>
<td>3</td>
</tr>
</tbody>
</table>

Table 2

<table>
<thead>
<tr>
<th>Type of murmur</th>
<th>This series (30 cases)</th>
<th>Levinson's series (59 cases)</th>
<th>Total (88 cases)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic systolic only</td>
<td>6</td>
<td>3</td>
<td>9</td>
</tr>
<tr>
<td>Aortic diastolic only</td>
<td>3</td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td>Apical systolic only</td>
<td>4</td>
<td>9</td>
<td>13</td>
</tr>
<tr>
<td>Apical diastolic only</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Aortic systolic and aortic diastolic</td>
<td>3</td>
<td>8</td>
<td>11</td>
</tr>
<tr>
<td>Apical systolic and aortic diastolic</td>
<td>2</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Aortic diastolic and apical systolic</td>
<td>0</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Apical systolic and apical diastolic</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Aortic systolic and apical systolic</td>
<td>0</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>No murmurs heard</td>
<td>9</td>
<td>26</td>
<td>35</td>
</tr>
<tr>
<td>Not evaluated</td>
<td>2</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Total in which murmurs were heard</td>
<td>19</td>
<td>32</td>
<td>51</td>
</tr>
</tbody>
</table>
Table 3
Summary of Aortic Murmurs Described in Thirty Cases in This Series and in Levinson’s Series88 of Fifty-eight Cases. Nineteen of All Cases Had Both Aortic Systolic and Aortic Diastolic Murmurs

<table>
<thead>
<tr>
<th>Murmurs</th>
<th>This series (30 cases)</th>
<th>Levinson’s series (68 cases)</th>
<th>Total (98 cases)</th>
<th>Per cent of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic murmur</td>
<td>14</td>
<td>22</td>
<td>36</td>
<td>41</td>
</tr>
<tr>
<td>Systolic</td>
<td>9</td>
<td>17</td>
<td>26</td>
<td>30</td>
</tr>
<tr>
<td>Diastolic</td>
<td>8</td>
<td>16</td>
<td>24</td>
<td>28</td>
</tr>
</tbody>
</table>

may be especially confusing when hematuria exists. Four of our patients had hematuria but only 1 had gross hematuria.

Neurologic Manifestations

These interesting findings have been classified as arising from ischemic necrosis of the peripheral nerves, ischemic necrosis of the spinal cord, and ischemic necrosis of the brain.88 Ischemic necrosis of the peripheral nerves is the most common neurologic finding and is manifested by numbness, coldness, or tingling. Peripheral sensory loss and areflexia may exist. When the spinal cord is involved, flaccid areflexic paraplegia, hypesthesia, and atony of the bladder may occur, with sphincter paralysis.89 The most striking neurologic finding, however, is ischemic necrosis of the brain due to carotid artery occlusion, with flaccid hemiplegia, hemianesthesia, and often coma. Four of our patients presented this picture. Neurologic changes may be transient.

Laboratory, Electrocardiographic, and Roentgenographic Findings

Laboratory data were obtained in 27 of our 30 patients. The hematocrit levels varied between 30 and 59 volumes per cent and a mild anemia was present in 10 patients. Leukocytosis was found in 21 patients, usually of a moderate degree, and in all but 2 it was less than 20,000 white blood cells per cubic millimeter. Hematuria was noted in 4 patients and was gross in 1 instance. Azotemia was present in 16 patients but only 3 had nonprotein nitrogen values above 100 mg. per cent. Serum amylase was normal in both instances in which it was determined. The serum glutamic oxaloacetic transaminase was examined in 1 case and was elevated (88 units) but there was electrocardiographic evidence of acute posterior wall myocardial infarction.

Electrocardiographic Features

The electrocardiographic findings are variable but in 20 patients on whom tracings were obtained, 16 were abnormal; however, non-specific changes in the ST-T segments were frequently seen. Changes suggestive of acute myocardial infarction might well be overlooked in these patients. The pattern of acute myocardial infarction might be the result of extension of the process into and involving the coronary arteries86 or due to occlusion of the coronary ostia by the dissecting hematoma.91

Other abnormal electrocardiographic findings in our series included disturbances in rhythm (atrial fibrillation, wandering atrial pacemaker, and extrasystoles), disturbances in conduction (first-degree ativoventricular block, complete and incomplete left bundle-branch block) and left ventricular hypertrophy.

Roentgenographic Features

The roentgenographic appearance of a dissecting aneurysm depends upon the location of the dissecting process. The most common features have been thoroughly outlined by Lodwick.92 Wood, Pendergrass, and Ostrum in 1932 reported widening of the suprarecine shadow, with deformity, and thought that a shadow visible along the innominate artery indicated extension along that vessel.93 They also described tracheal deviation, due either to the aneurysm itself or to fluid in the left pleural space. As a rule, however, the most common finding is straightening of the aortic curvature, occasionally with rough irregularities.94 Repeated films may indicate daily extension of the process, and a chest film prior to the dissection is important for comparison.92 Lodwick also described different density in the aorta, particularly when the dissection in-
volved only the lateral portion of the aorta. The widened aortic wall may appear more radiolucent than the dense central core of the aorta. Not infrequently, in the age group most commonly associated with dissecting aneurysm, calcified intimal plaques are found. When dissection occurs, the calcified intima may be pushed medially and the adventitia may be widened laterally. Widening of the space between calcified intima and lateral aortic shadow may be diagnostic. The normal aortic wall measures 2 to 3 mm. in width and measurements of greater than 3 to 5 mm. are suggestive, and of more than 1.0 cm. are practically diagnostic of this condition.92, 95

Left ventricular hypertrophy is frequently present and signs of pericardial effusion may be noted. Pleural fluid is frequently seen on x-ray, most commonly in the left pleural space.

Special roentgenographic procedures have been recommended. The right posterior oblique position has been suggested as most satisfactory on conventional x-ray studies.94 Fluoroscopy may reveal absence of aortic pulsations92 or in the case of hemopericardium, absent or diminished cardiac pulsations. Roentgenograms with a barium-filled esophagus may be helpful.92 Angiocardiography has been reported to be useful.96, 97 One case was diagnosed by translumbar aortography when the radiopaque medium extended an unusual distance cephalad, and was associated with paradoxical visualization of the iliac vessels.98 This procedure, however, is not recommended. Laminograms have been said to be most helpful in the older age groups with the most arteriosclerotic changes.99 For evaluation of abdominal dissecting aneurysms, Lodwick recommended retroperitoneal and intraperitoneal air injections to furnish a radiolucent background.92

Of our 30 cases 19 had roentgenographic studies. Only 1, however, was thought to be diagnostic of dissecting aneurysm but 6 others (32 per cent) were suggestive of aortic aneurysm. In an additional 6 the heart revealed varying degrees of enlargement and the aorta was reported as "elongated and tortuous." Pleural effusion was seen in 3 patients.

Treatment

With the advent of newer surgical technics dissecting aneurysm of the aorta has become a "surgical disease." Since the prognosis is related to re-entry of the dissecting sac into the original aortic lumen.100 the surgical approach is directed toward producing a natural "cure" and has been described as the "re-entry procedure."14, 101, 102 The surgical management was first attempted by Gurin et al.103 in 1935, and later by Shaw in 1955.104 Both were unsuccessful in that the patients died with renal failure, but re-entry was accomplished. DeBakey et al.4, 102 devised a successful approach which attempts to prevent further dissection, restore blood flow, and either to remove or to repair the lesion. The type of repair depends on the site of the intimal tear. When the aortic arch is involved, re-entry with obliteration of the false passage below is chosen. When the intimal tear is distal to the arch, the lesion is excised and replacement graft is installed. Some modifications have been suggested by others.105

The preoperative management may be lifesaving; absolute bedrest with relief of pain by opiates is mandatory. Oxygen should be used for impending shock or dyspnea. Careful regulation of severe hypertension with ganglionic-blocking drugs may help prevent rupture of the aneurysm.106 When signs of peripheral vascular occlusions are present, the limb should be kept in a horizontal position at room temperature, and a sympathetic block may be helpful.107

Summary

The clinical diagnosis of dissecting aneurysm of the aorta depends on awareness of the entity, keen clinical suspicion, and an understanding of the varied manifestations. Clinical suspicion is sharpened by association of certain preexisting conditions found to be related to an increased incidence of aortic dissection. Recognition of the Marfan's syndrome, pregnancy, or preexisting hypertension in a patient presenting as an acute catastrophe
may lead to the diagnosis. A family history of aortic dissection is similarly helpful.

The variable manifestations call for careful reasoning. Multiple system involvement in many cases is the initial lead in establishing the diagnosis. The many systems frequently involved in the symptomatology of aortic dissection have in common the origin of the blood supply. When the aorta is damaged, branches to various organs may be affected so that the symptoms depend on the branches involved. Because any portion of the aorta may be involved in the dissecting process, no single clinical picture suffices for its recognition. Aortic valvular findings often are seen with involvement of the ascending aorta, central neurologic findings may result from occlusion of the carotid arteries, spinal cord manifestations may result from occlusion of the intercostal arteries, renal manifestations may be seen with renal artery involvement, and so on down the aorta. However, the pattern of a catastrophic episode—whether cardiovascular, neurologic, or abdominal—calls for consideration of aortic dissection. All of the finesse of the most careful physical examination may be required to make the diagnosis but the coexistence of chest pain, aortic murmurs, and peripheral artery occlusion is practically diagnostic. Laboratory adjuncts including roentgenographic evidence of mediastinal widening, evidence of hematuria, and the absence of electrocardiographic evidence of myocardial infarction may be helpful. Electrocardiographic evidence suggestive of myocardial infarction does not, however, exclude aortic dissection.

Dissecting aneurysm of the aorta now is a surgical emergency that requires early accurate diagnosis and early surgical treatment, if the natural survival rate of 10 per cent is to be improved.

**Summario in Interlingua**

Le diagnose clinic de aneurysma dissecante del aorta depende de un alerte e acute suspicione clinic e del precise comprehension del varie manifestaciones que charateriza iste entitate. Le suspicione clinic es augmentate per le presentia de un complexo de pre-existent conditiones pro le quales un augmentate incidentia de dissection aortic ha essite constatate. Le recognition del syndrome de Marfan, de pregnancia, o de hypertension pre-existent de un patiente presentate con un catastrophe acut pot esser le base del diagnose. Un historia familial de dissection aortic es similemente de valor.

Le variabilitate del manifestaciones impone le necessitate de un precise rationamento. Afectiones plurisystemal es frequentemente le prime indicio in supporto del diagnose. Le varie systemas que es frequentemente concernite in le symptomatologia de dissection aortic es interrelationale per le comunitate de lor provision de sanguine. Quando le aorta es lesionate, su branca ducente verso varie organos pot esser implicate a varie grados, de manera que le symptomas depende de qual branca del aorta es afficite. Viste que non importa qual portion del aorta pot esser le victim del processo de dissection, nulle specific e unico tableau clinic suffice como base de su recognition. Symptomas in le valvula aortic es frequente quando le aorta ascendente es implicate; manifestaciones in le sistema nervose central pot esser le resultato de un occlusion del arterias carotidica; occlusion del arterias intercostal pot esser responsable pro symptomas referibile al medulla dor-sal; manifestaciones renal occurre in consequentia de un affection del arterias renal; e assi successivamente usque al fin del aorta. In omne, caso, le occurrence del un episódio catastrofiche—sia cardiovascular, sia neurologica, sia abdominal—demanda que dissection aortic es prendite in consideration. Omne le raffinamento del plus meticuloze examine physis es frequentemente necessari pro le obtenion del diagnose, sed le co-existence de dolores thoracice, murmures aortic, e occlusion de arteria peripherie es practicamente infallibile como prova de dissection aortic. Constatations laboratoriale qu es a vices de valor include le observation roentgenologica de un allargamento mediastinal, le estabilimento de hematuria, e le prova que le electrocardiogramma exhibe nulle indicio de infarcimento myocardial. Tamen, le presentia de indicios electrocardiographique de infarcimento myocardial non exclude le possibilitate de dissection aortic.

In nostre dies, aneurysma dissecante del aorta representa un situation de urgentia chirurgica que require un prompte e accurate diagnose e le precoce intervention operatori si nos desira melliorar le cifra del supervivientia natural que amonta a 10 pro cento.

**References**


2. Shumaker, H. B., Jr., and Harris, J.: Cure
DISSECTING ANEURYSM OF AORTA

of dissecting aneurysm of thoracic aorta by
3. Swann, W. K., and Bradsher, J. T., Jr.: Acute
dissecting aneurysm of the aorta; operation
and recovery. New England J. Med. 255: 36,
1956.
4. De Bakey, M. E., Cooley, D. A., and Creech,
O. J., Jr.: Surgical considerations of dissec-
ting aneurysm of the aorta. Ann. Surg. 142:
5. Weiss, S., Kinney, I. D., and Maher, M. M.: 
Dissecting aneurysm of the aorta with expe-
mental atherosclerosis. Am. J. M. Sc. 200:
192, 1940.
6. Domzalski, C. A., Jr.: Diagnosis of dissecting
8. Shennan, T.: Dissecting aneurysm. Medical
London, His Majesty's Stationery Office, 1934.
26, Art. 17, F. 50, 1760. (Cited by Shennan.8)
10. Maunoir: Memoires physiques et pratiques sur
l'aneurisme. Geneva, 1802. (Cited by Shen-
nan.8)
11. Laennec, R. T. H.: Traite de l'auscultation
mediane et des maladies des poumons et du
(Cited by Shennan.8)
12. Pennock, C. W.: Case of anomalous
aneurysm of the aorta resulting from the effusion
of blood between the laminae composing the mid-
dle coat of that vessel. Am. J. M. Sc. 23: 13,
1839.
13. Peacock, T. B.: Cases of dissecting aneurism,
or that form of aneurismal affection in which
the sac is situated between the coats of the
vessel. Edinburg M. J. 60: 276, 1843.
14. Swain, and Latham, P. M.: Case of dissecting
aneurysm of the aorta. Tr. Path. Soc. Lon-
don 7: 106, 1855-1856.
Virchows Arch. 273: 454, 1929.
16. Nicholls, F.: Observations concerning the body
of His Late Majesty. Phil. Tr. Roy. Soc. Lon-
don 52 (Pt. 1): 265, 1761.
18. Levinson, D. C., Edmeades, D. T., and Gri-
ffith, G. C.: Dissecting aneurysm of the aorta:
itself, electrocardiographic and laboratory
features: Report of 58 autopsied cases. Cir-
culation 1: 360, 1950.
E.: Dissecting aneurysm. Quart. Bull. North-
22. Rider, J. A., Chriess, J. W., and Hermann,
G. R.: Dissecting aneurysms; 10 year study.
23. Halfpenny, B., and Brown, C. A.: Dissecting
aneurysms of the aorta: A study of 12 cases.
24. Gutmann, G. E.: Analysis of dissecting aener-
ysm over a 7-year period at Mercy Hospital,
Jonesville, Wisconsin. Wisconsin M. J. 56:
327, 1957.
(Cited by Sailer, S.: Arch. Path. 33: 704, 1942.)
27. Schnitke, M. A., and Batey, C. A.: Dissect-
ing aneurysm of the aorta in young individuals,
particularly in association with pregnancy,
with report of a case. Ann. Int. Med. 20: 486,
1944.
28. Gittleman, S. E., and Leichtling, M.: Dis-
secting aneurysm with perforation into esopha-
29. Gore, I., and Seiwerth, V. J.: Dissecting ane-
urysm of the aorta: Pathologic aspects; an
30. —: Dissecting aneurysms in persons under 40
31. Schlichter, J. G., Amromin, G. D., and Sol-
way, A. J. L.: Dissecting aneurysms of the aorta.
32. Amromin, G. D., Schlichter, J. G., and Sol-
way, A. J. L.: Medionecrosis. Arch. Path. 46:
380, 1948.
33. Schlichter, J. G.: Experimental medionecrosis
34. Gore, I.: Pathogenesis of dissecting aneurysm
35. Neilson, G. H., and Sullivan, J. J.: Dissect-
ing aneurysm of the aorta associated with
Marfan's syndrome. M. J. Australia 43: 923,
1956.
36. Pappas, E. G., Mason, D., and Denton, D.: 
Marfan's syndrome: A report of 3 patients
with aneurysm of the aorta. Am. J. Med. 23:
426, 1957.
37. Thomas, J., Brothers, G. B., Anderson, R. S.,
and Cuff, J. R.: Marfan’s syndrome: 3 cases
with aneurysm of aorta. Am. J. Med. 12: 613,
1952.
38. McKusick, V. A.: Heritable disorders of con-
nective tissue. III. The Marfan's syndrome.
39. Marvel, R. J., and Genovese, P. D.: Cardio-

Circulation, Volume XXII, August 1960
DISSECTING ANEURYSM OF AORTA

Dissecting Aneurysm of the Aorta The Clinical Features of Thirty Autopsied Cases
BLAIR D. ERB and I. FRANK TULLIS

doi: 10.1161/01.CIR.22.2.315
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1960 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/22/2/315

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/