Dissecting Aneurysm of the Aorta: Its Clinical, Electrocardiographic and Laboratory Features

A Report of Fifty-eight Autopsied Cases

By David C. Levinson, M.D., Donald T. Edmeades, M.D., and George C. Griffith, M.D.

The literature has been reviewed, and 58 autopsied cases of dissecting aneurysm of the aorta observed at the Los Angeles County Hospital over a ten year period have been carefully studied. Clinical-pathologic correlation has been attempted whenever possible, with emphasis on diagnostic features. The increasing incidence of accurate diagnosis of dissecting aneurysm has been noted, and it is hoped that this review will help solidify the clinical syndrome or syndromes of the condition and thus facilitate diagnosis in dubious instances.

Despite the recent excellent reviews and publication of case reports of dissecting aneurysm by David, McPeak, Vivas-Salas, and White,1 Leitch,2 Sailer,3 and others, it was felt that by reviewing a relatively large series of cases some valuable data might be uncovered. The diversity of symptoms in the clinical syndromes presented by dissecting aneurysm cannot be overemphasized if the percentage of correct antemortem diagnosis is to be increased. The importance of a correct diagnosis may grow if surgical treatment should prove effective. Secondly, the mechanism behind the occurrence of the diastolic murmur needs further study. Finally, the electrocardiogram hitherto has been said to show either normal or nonspecific tracings, and it was felt that these were statements which needed further clarification in view of the known courses of dissection in this disease.

Historical Background

Dissecting aneurysm is the lesion produced by the penetration of circulating blood into the wall of the aorta, and subsequent extension for a varying distance resulting in separation of the layers of the vessel wall. An intravascular hematoma may arise from hemorrhage of the vasa vasorum, and have no communication with the lumen of the vessel, or the hematoma may communicate with the lumen of the vessel through one or more intimal tears. Commonly, rupture occurs either back into the lumen via an intimal tear, or into the exterior by perforation of the adventitia.

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Vesalius,4 in 1557, was the first to diagnose an aneurysm. This aneurysm occurred in a man who developed a pulsating abdominal tumor after falling off a horse. Sennertus,5 in 1628, maintained that the immediate cause of an aneurysm was the rupture of the internal coat with elevation and distention of the external coat. This was supported by Scarpa4 in 1804. Nicholls,6 in 1763, in reporting the cause of death of King George II, which occurred while straining at stool, described an aneurysm within the coats of the aorta, accompanied by a large intimal tear and rupture into the right ventricle. He attributed this to the greatly increased intravascular pressure at the time of death. Morgagni,7 in 1760, cited the above case, and reported several cases of his own in which the blood had made its way by degrees through the vessel wall. Maunoir,8 in 1802, first clearly described dissection of the arterial coats by blood. Laennec,9 in 1819, seventeen years after Maunoir, also described dissection of the aorta, and was the first to use the term “aneurysme dis- sequent.” Unfortunately, much of the credit belonging to Maunoir has been given to Laennec. Shekelton,10 in 1822, was the first to describe “healed” dissecting aneurysm in which there was re-entry at a lower level in the aorta, allowing circulation of blood through the false sac. Pennock,11 in 1839, described the first case recorded in the American literature, and demonstrated that dissection takes place in the laminae of the media. As early as 1855, Swaine, Keyworth, and Latham12 reported the first case of dissecting aneurysm to be diagnosed correctly antemortem.

Incidence

The recent literature was reviewed, and records of approximately 734 cases of dissecting aneurysm of the aorta were found. In 88 (10.6 per cent) of these cases a correct antemortem diagnosis had been made. Shennan,13 who reviewed the literature up to 1933, was able to collect 317 cases, of which only 6 (1.8 per cent)
were correctly diagnosed antemortem. The present report is based on the study of 58 cases of dissecting aneurysm demonstrated at autopsy at the Los Angeles County Hospital from 1935 to 1947, inclusive. Of this group, a correct antemortem diagnosis was made in 16 instances (27.5 per cent). During the same period, there were 18 cases in which dissection localized to the abdominal aorta was found at necropsy. These cases will be the subject of a future report.

Autopsy Incidence. The autopsy incidence of dissecting aneurysm at the Los Angeles County Hospital from 1935 to 1947, inclusive, was one in 447 subjects. David, McPeak, Vivas-Salas, and White1 reported an incidence of one in 128 autopsy subjects at the Massachusetts General Hospital from 1937 to 1946. A recent review by Warren and McQuown15 disclosed an autopsy incidence of one in 450 subjects at the Charity Hospital. Their findings were almost identical in this respect to those of the present study. McGeechay and Paulin,16 in 1937, reported an autopsy incidence of one in 500 subjects. Flaxman,17 in 1942, found one dissecting aneurysm in every 714 autopsy subjects at the Cook County Hospital. That these figures may be misleading was evidenced by the excellent report of Mote and Carr,18 who reported 60 cases of dissecting aneurysm during autopsy examinations by the San Francisco Coroner's Office over a five-year period (1933–1937). This greatly exceeded the incidence of dissecting aneurysms in other hospital examinations in San Francisco over the same period. This report confirms the suspicion that dissection often occurs without antecedent history or warning, resulting in death within minutes to hours, and also, that the actual incidence of this entity is much more frequent than one is led to believe from the various autopsy statistics, most of which come from large general hospitals.

Age Incidence. The greatest frequency of dissecting aneurysms (table 1) occurred in persons between the fifth and seventh decades of life. Thirty-six of the 58 cases occurred during this period. The oldest patient was a 90 year old Negro, and the youngest, a 14 month old infant. Schnitker and Bayer19 were impressed with the frequency of this condition among young people. They reviewed the literature to 1943, and found that of the total number of cases reported, 24 per cent occurred in individuals under 40 years of age. In the present series, there were only 6 cases (10 per cent) in persons under the age of 40 years. This discrepancy can be explained in part by the large older-age population found in the Los Angeles area.

Sex Incidence. The ratio of men to women is usually reported as men predominating two to three times as frequently as women. In the group being reported, the incidence in men was found to be about 2.6 times as frequent as that in women. In the 60 subjects seen at the Coroner's Office in San Francisco,18 there were 52 men and only 8 women, the incidence in the men being 6.5 times as frequent as that in the women. Whether this implies that men are more apt to die suddenly than women who have this condition is purely a matter of speculation.

The average age for women was found to be 58 years, and that of men 59.9 years. This is not the usual or, rather, expected finding. The women usually dominate the older-age brackets. The average age in David, McPeak, Vivas-Salas, and White's report was women—63 years, and men—58 years. The delayed onset of hypertension in women would lead one to

<table>
<thead>
<tr>
<th>Age (Years)</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–10</td>
<td>White Negro Chinese</td>
<td>White Negro</td>
</tr>
<tr>
<td>11–20</td>
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<tr>
<td>21–30</td>
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<td>31–40</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>41–50</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>51–60</td>
<td>8</td>
<td>5</td>
</tr>
<tr>
<td>61–70</td>
<td>12</td>
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<td>71–80</td>
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<td>81–90</td>
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<tr>
<td>Total</td>
<td>30</td>
<td>11</td>
</tr>
<tr>
<td></td>
<td>42</td>
<td>12</td>
</tr>
</tbody>
</table>

TABLE 1.—Age, Sex and Race Distribution of Fifty-Eight Cases of Dissecting Aneurysm (1935–47 Inclusive)
suspect that the average age of women with dissecting aneurysms should exceed that of men. An interesting observation by Schnitker and Bayer was that in those cases occurring in women under the age of 40, 50 per cent were found to be in association with pregnancy.

**Race Incidence.** Of the 58 cases, 15 (25.8 per cent) occurred in Negroes. There was no unusual distribution of sex within the races, the men predominating in both races 2.6 to 1. The high percentage of Negroes is not remarkable in light of the fact that hypertension is known to occur twice as frequently among Negroes as among white persons. There was one case worthy of special mention which was found in a 50 year old Chinese, hypertension being reported as unusual in the Chinese race. We were unable to find any other reports in the literature of a dissecting aneurysm occurring in a Chinese.

**CLINICAL CLASSIFICATION**

In 1929, Gager proposed a classification of dissecting aneurysms, concerned primarily with the duration of survival. Such a classification is particularly applicable to this entity, and with modifications, it is presented as follows:

**Acute Type.** Rupture of the intima, dissection of the media, and terminal perforation of the adventitia into a viscus such as the pericardial cavity, pleural cavity, and mediastinum, takes place within minutes to hours, resulting in death within twenty-four to forty-eight hours after the initial onset. Case 1 has been chosen as being illustrative of this type.

**Case 1.** Two hours prior to hospital admission on November 12, 1945, this 46 year old white man had a sudden onset of “constriction” about his chest which lasted about fifteen minutes, and which was soon replaced by an intense, sharp, epigastric pain. The patient was well developed. He was tossing about in bed and complained of pain. His extremities were cold and clammy. His temperature was 97 F. The pulse rate was 88 per minute and respiratory rate 34 per minute. The lungs were clear. There was no cardiac enlargement or heart murmurs. The blood pressure was 175/100. Epigastric and left upper-quadrant tenderness was present.

The urinalysis showed no abnormality. The Wassermann and Kahn reactions were negative. A flat plate of the abdomen showed no renal calculi.

The patient was discharged from the hospital on Nov. 14, 1945.

On Nov. 20, 1945, seven days after discharge, the patient was readmitted to the hospital. He complained of severe epigastric pain and vomiting. The pain had come on while he was asleep, and was severe, steady, and localized to the epigastrium.

At the time of examination the patient was thrashing about and groaning loudly. His temperature was 97.5 F.; the pulse rate was 100 per minute and respiratory rate 34 per minute. The lungs were clear. The heart did not appear enlarged, and the sounds were distant. The blood pressure was 90/50. Epigastric tenderness was present.

The electrocardiogram showed abnormal tracings in that the RS-T segments in Leads I, II, and III were depressed.

Five hours after being admitted in shock, the patient suddenly expired.

The postmortem findings were as follows: The pericardium was normal. The heart weighed 500 grams. The coronary vessels were markedly atherosclerotic but showed no occlusion. The valves were thin and measured: aortic, 8 cm.; pulmonic, 7.5 cm.; tricuspid, 14 cm.; mitral, 12 centimeters. The aorta had a few atherosclerotic plaques, and in the first portion of the arch of the aorta there was a transverse intimal tear which communicated with a dissecting aneurysm. The aneurysm extended distally to the level of the diaphragm, and was filled with recently clotted blood. There were several areas of rupture in the adventitia of the thoracic aorta with resultant bleeding into the mediastinum and left pleural cavity. There was a massive blood clot in the left pleural cavity which weighed 1200 grams.

**Subacute Type.** In this group, the process of intimal rupture and mural dissection is protracted, occurring gradually over a period of days or weeks, during which time symptoms and signs appear which are readily localized and of value in making or confirming the diagnosis. In this group, after a period of slow but progressive dissection, almost always a terminal adventitial rupture occurs similar to that of the acute type, which invariably results in a fatal outcome. Case 2 is illustrative of this type of patient.

**Case 2.** The patient, a 65 year old known hypertensive man, admitted to the hospital on January 25, 1947, and died February 4, 1947. Four days prior to admission, he noticed the onset of severe, sharp pain in the lower portion of his chest, which came on at rest. It lasted about twelve hours, and migrated down into his abdomen. Since that time he had had intermittent pain in the lower part of his back and in his right flank, which was accompanied by frequency of urination, dysuria, and vomiting. In addition, he had been constipated for four days prior to admission.
On examination, the patient chiefly complained of pain in his right flank. His temperature was 100.2 F.; the pulse rate was 88 per minute and respiratory rate 18 per minute. There were a few scattered râles at both lung bases. The heart appeared enlarged to the left on percussion. There was a soft, systolic murmur present over the entire precordium, best heard at the apex. The blood pressure was 190/100. There was deep tenderness in both upper quadrants of the abdomen. Bilateral costovertebral-angle tenderness was present. A bruit was heard just to the right of the umbilicus anteriorly.

The hemoglobin value was 16 grams, and there were 9,500 white blood cells per cu. mm. of blood. The urinalysis showed no trace of albumin. The nonprotein nitrogen was 70 mg. per 100 cc. of blood. Wassermann and Kahn reactions were negative. At fluoroscopy, the aorta was found to be dilated in the ascending and transverse portions of the arch, and the left border of the aorta was shaggy and irregular with diminished pulsations. The left ventricle was moderately enlarged. The findings were thought to be consistent with the diagnosis of a dissecting aneurysm. The electrocardiogram showed abnormality. The RS-T segment in Lead I was sagging and depressed. The Q-T interval was 0.44 seconds.

The course was febrile with daily elevation of temperature to 100 F. Intermittent pain in the right flank, and deep abdominal tenderness persisted. An aortic diastolic murmur appeared. During his ninth hospital day the patient became distended, anuric, and finally expired.

The antemortem diagnosis was dissecting aneurysm.

The postmortem findings were as follows: The pericardium was normal and contained 250 cc. of a straw-colored fluid. The heart was enlarged and weighed 570 grams. The valves were normal; the aortic measured 7 cm. and the pulmonic, 7.5 centimeters. The left ventricle measured 16 mm., and the right 2 to 4 millimeters. There was a dissecting aneurysm which originated at the level of the innominate artery and extended distally for about 2 cm. into both common iliacs. There was a large intimal tear 4 cm. proximal to the celiac axis. In the region of the renal arteries, the dilated aneurysmal sac partially occluded the left renal artery and completely occluded the right. There were large antemortem thrombi which occluded both renal stoma and entered both renal arteries for a distance of 7 millimeters.

The right kidney was a small, soft, pale organ approximately half the size of the left (combined weight of both kidneys—250 grams). The cut surface of the right kidney revealed markedly pale cortical and pyramidal structures. There was a large, yellow area which extended through the entire cortex to the pelvis. The left kidney was also pale, but appeared more normal than the right. The capsules of both kidneys striped with ease.

Microscopic examination of the aorta showed that medial degeneration and cyst formation were present.

The right kidney showed necrosis and fatty degeneration of the tubules, and interstitial hemorrhage typical of infarction.

Chronic or "Healed" Type. This group includes patients who survive the initial dissection. Healing is made possible by either re-entry of the dissected passage into the lumen of the aorta or into the common iliacs, with subsequent endothelialization of the false passage and the establishment of a "double-barrelled" aorta. Or, less frequently, healing may take place by obliteration of the false passage by clot formation with subsequent organization and replacement fibrosis. Patients with "healed" dissecting aneurysms may survive for months or years, and death usually occurs in one of several manners. Most commonly, it is due to congestive failure, a cerebral accident, coronary artery disease, or other incidental illnesses. In a small percentage a second dissection occurs followed by fatal perforation. Case 3 has been chosen as best illustrating this type of course.

Case 3. The patient, a 62 year old white man, a confectioner, was admitted to the hospital on December 11, 1933, complaining of severe pain in his back, radiating to his arms and legs, and accompanied by dyspnea and vomiting.

On examination he appeared to be in acute distress. His temperature was 98.6 F. His pulse rate was 100 per minute and his respiratory rate 24 per minute. There were moist râles at the base of the left lung. The heart was not enlarged. A mitral systolic murmur was present. The blood pressure was 175/100.

The electrocardiogram showed left ventricular hypertrophy. The Wassermann and Kahn reactions were negative.

Shortly after admission, the patient became free of pain, and on his seventh hospital day, December 18, 1933, he was discharged to the Outpatient Department with the diagnosis of hypertension and coronary artery disease.

He was readmitted to the hospital on September 29, 1939, in congestive heart failure, complaining of dyspnea on exertion, paroxysmal nocturnal dyspnea, and precordial pain of three months' duration.

The patient was well developed. He was dyspneic, orthopneic, and slightly cyanotic. His temperature was 97.6 F. The pulse rate was 100 per minute, and
respiratory rate 22 per minute. The lungs were clear. The heart was enlarged, and the point of maximal impulse was felt in the fifth intercostal space at the anterior axillary line. Aortic systolic and diastolic murmurs were present. The blood pressure in the right arm was 200/100, and in the left arm, 190/100. The physical examination revealed no abnormality other than that pertaining to the heart.

The Wassermann and Kahn reactions were negative. The electrocardiogram again showed left ventricular hypertrophy. The orthodiagram showed marked fusiform dilatation of the ascending aorta, with moderate enlargement of the left ventricle. The innominate artery also appeared dilated.

The patient was treated with bed rest, restricted fluids, and digitalis. He improved gradually. Nine days after admission he was discharged to the Outpatient Department.

The significant findings on this admission were the detection of an aortic diastolic murmur, with aneurysmal dilatation of the ascending aorta, and negative serologic reactions. Both the third and fourth admissions (July 28 to August 5, 1940, and January 1 to May 16, 1941) were because of congestive heart failure, and in both instances the response to digitalis and bed rest was satisfactory.

On the final admission, March 5, 1941, he complained of dyspnea, orthopnea and ankle edema of six days' duration.

At this time he was extremely dyspneic and orthopneic. His temperature was 98.5 F., the pulse rate was 80 per minute, and the respiratory rate 20 per minute. Dullness, diminished breath sounds, and tactile fremitus were found over the right lower lobe posteriorly. Moist râles were heard at the base of the left lung. The heart was enlarged, and the point of maximal impulse was in the sixth intercostal space at the anterior axillary line. Systolic and diastolic aortic murmurs were present. The blood pressure was 184/80. The liver was enlarged down to the level of the umbilicus. There was +4 pitting ankle edema. There were 4,000,000 erythrocytes per cu. mm. of blood. The hemoglobin value was 86 per cent. The white blood cells numbered 6,600 per cu. mm. of blood. The urinalysis showed no abnormality, and the nonprotein nitrogen value was 51 mg. per 100 cc. of blood. The electrocardiogram showed auricular fibrillation, left ventricular hypertrophy, and digitalis effect.

The patient was treated with bed rest, digitalis, diuretics, a low-salt diet, and allied measures. Despite vigorous therapy his condition became progressively worse, and he died May 16, 1941.

The postmortem findings were as follows: The visceral and parietal pericardium were adherent by easily separated adhesions. The heart weighed 700 grams. The valves were normal, except for fusion over an extent of 4 mm. between the posterior and right aortic valve cusps. There was no evidence of any rheumatic involvement. The aortic valve measured 8 cm. and the pulmonic, 7.5 centimeters. The left ventricle measured 17 mm., the right 6 millimeters. Approximately 5 cm. above the aortic ring there was a transverse intimal tear which communicated with the false sac of a dissecting aneurysm. The aneurysm extended proximally down into the aortic ring, and distally to the common iliacs, where re-entry occurred. The lining of the true passage was wrinkled and had some lipid plaques. The false passage was lined with intima, was wrinkled and rough, and had almost a tree-bark appearance. The common innominate artery and the left subclavian artery had also been involved in the dissection.

The microscopic examination of the aorta showed medial necrosis with cyst formation.

**Duration of Survival**

The 58 cases being reported consisted of 43 acute and subacute types, and 15 of the chronic or "healed" type. The duration of survival of the patients of each group has been tabulated in table 2.

<table>
<thead>
<tr>
<th>Type</th>
<th>Duration of Survival</th>
<th>Number of Patients</th>
<th>Percent of Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>&quot;Acute&quot; type</td>
<td>1-18 hours</td>
<td>21</td>
<td>36.2</td>
</tr>
<tr>
<td>&quot;Subacute&quot; type</td>
<td>3-10 days</td>
<td>13</td>
<td>22.4</td>
</tr>
<tr>
<td>&quot;Chronic&quot; or &quot;healed&quot; type</td>
<td>3 months to 8 years</td>
<td>15</td>
<td>25.9</td>
</tr>
</tbody>
</table>

About one-third of the total number of patients died within the first forty-eight hours, and about one-half within the first ten days. The fact that one-fourth of the patients had the chronic or "healed" type, surviving three months to eight years, is a much more encouraging finding than that of Weiss, who found that healing occurred in about 10 per cent of the patients, chiefly by means of re-entry of the false passage into the true lumen at a lower level.

**Clinical Symptoms**

*Presenting Symptoms or Mode of Onset.* Pain was the presenting symptom in 45 (77.6 per cent) of the 58 cases. It was located in the chest in 17 patients (29.3 per cent), and in the epigastrium in 14 (24.1 per cent). In the remaining patients the pain was described as being in both the epigastrium and lower chest.
(4 patients), the interscapular region (4 patients), the neck (3 patients), the midback (2 patients), and the sacrum (1 patient). (See table 3.)

The sudden onset of pain, the type of pain, its location initially, and subsequent radiation, are all of utmost value in arriving at a correct diagnosis, and will all be discussed in some detail.

Onset of Pain: The pain was described as having had a sudden, dramatic, and acute onset, usually with no definite relation to effort or activity, except for several instances in which physical exertion may have been a precipitating factor. One case, interestingly, occurred in a 34 year old Negro during sexual intercourse. In this group of 58 cases, physical exertion was not an important factor in initiating the dissection, but, contrariwise, many of these cases came on while the patient was sleeping. Cherry and Cherry\(^2\) carefully reviewed the antecedent activity and occupations of 77 patients with dissecting aneurysm, and arrived at the conclusion that physical exertion is not an important factor in the initiation of dissection, but rather that its occurrence is merely coincidental.

Type of Pain: The pain was most commonly described as being sudden, severe, tearing, ripping, sharp, exerting, cramplike, or burning. Not infrequently it was so severe as to cause the patient to double up in bed, or to lie on the floor in an attempt to obtain relief. In some instances, the pain was so intense that the patient was unable to lie still, and tossed about in bed. It was not infrequently described as a sensation of “something having torn loose” in the chest, or as the feeling of a knife being stuck into the chest and then twisted. In only 2 cases was the pain described as being oppressive in nature, resembling that of acute myocardial infarction. Kilgore\(^3\) described the pain as sometimes “throbbing,” in synchrony with the heart beat, owing to successive pulse waves splitting the media or distending the newly formed wall.

Site of Pain: The most frequent location of pain in this series of patients was in the chest, where it was present in 17 patients (29.3 per cent of the total). David, McPeak, Vivas-Salas, and White\(^1\) recorded the initial pain as being in the chest in 10 of 17 cases (58 per cent). Logue\(^2\) reviewed a group of 12 cases, all diagnosed antemortem, and found the pain substernal in 6 (50 per cent). Because the chest is the commonest site of the initial pain, and because the error most frequently encountered is in mistaking dissecting aneurysms for acute myocardial infarction, the chest pain in the above 17 cases was closely scrutinized. It was described as being in the anterior midchest in 9 of these cases, substernal in 6, and merely in the “left chest” in the remaining 2. Of the 6 patients in whom it was described as being substernal, it was definitely aggravated by deep breathing in 2 patients (associated hemothorax present), in 2 others it was said to be sharp and knife-like, and in only the remaining 2 was it described as being oppressive and crushing. So actually, in only 2 of the 17 cases, in which the pain originated in the chest, was there any real resemblance to the pain of myocardial infarction.

The second commonest location of the initial pain was in the epigastrium or abdomen. Here the pain was described as being ripping, tearing, or cramplike, and was actually usually quite severe. The pain was present in the epigastrium initially in 14 (21.1 per cent) of the patients. The fact that approximately one-fourth of the dissecting aneurysms in this series had their onset with epigastric pain is most important, and recognition of this incidence should be of

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**Table 3.—Mode of Onset in Fifty-Eight Cases of Dissecting Aneurysm**

<table>
<thead>
<tr>
<th>Mode of Onset</th>
<th>Incidence</th>
<th>Percentage of Total</th>
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<tbody>
<tr>
<td>I. Pain</td>
<td>45</td>
<td>77.6</td>
</tr>
<tr>
<td>A. Chest</td>
<td>17</td>
<td>29.3</td>
</tr>
<tr>
<td>1. Anterior mid-chest</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>2. Substernal</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>3. Left side of chest</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>B. Epigastrum</td>
<td>14</td>
<td>24.1</td>
</tr>
<tr>
<td>C. Epigastrum and low in chest</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>D. Interscapular</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>E. Neck</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>F. Midback</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>G. Sacrum</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>II. Syncope</td>
<td>8</td>
<td>13.2</td>
</tr>
<tr>
<td>III. No history of pain or syncope</td>
<td>6</td>
<td>8.6</td>
</tr>
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</table>
value in making the correct diagnosis. Every patient with acute, severe epigastric pain and associated hypertension should be suspected of having a dissecting aneurysm as well as an acute abdomen. The readiness with which the diagnosis of dissecting aneurysm can be confused with that of an acute abdomen is well illustrated in the literature. Finkelstein and Jacobi reported a case of dissecting aneurysm associated with epigastric pain radiating through to the back, in which the antemortem diagnosis had been that of a perforated peptic ulcer. Other cases have been misdiagnosed as acute peritonitis, mesenteric thrombosis, and acute pancreatitis. Further confusion is added to the picture by the fact that the dissection may involve the superior mesenteric artery, resulting in its occlusion and subsequent infarction of the intestine. Kennedy described just such a case, in which the antemortem diagnosis had been mesenteric thrombosis. At necropsy a dissecting aneurysm involving the superior mesenteric artery was discovered.

The difficulty in this differential diagnosis is so marked that not infrequently these patients are subjected to abdominal surgery because they are thought to have an acute abdomen. Levitt, Levy, and Cole tell of a patient sent to surgery for abdominal exploration, and a similar incident followed in one of our patients.

Case 4. The patient, a 59 year old white man, a laborer and a known hypertensive, entered the Los Angeles County Hospital on October 7, 1944, because of severe epigastric pain of several hours' duration. The pain radiated to the back and to both shoulders. He was acutely ill, and tossed about in bed because of severe abdominal pain. His temperature was 98 °F. The pulse rate was 84 per minute.

The lungs were clear. The heart was enlarged and the point of maximal impulse was in the fifth intercostal space at the anterior axillary line. There was an apical systolic murmur and a diastolic murmur over the entire precordium. The blood pressure was 280/150. The abdomen was flat and did not move with respiration. There was boardlike rigidity in the upper quadrants and peristalsis was absent.

There were 10,000 white blood cells (polymorphonuclear neutrophils, 93 per cent; lymphocytes, 7 per cent) per cu. mm. of blood. The urinalysis showed +2 albumin reaction, one to seven erythrocytes per high-powered field, and several hyaline and granular casts. The blood amylase was 300 units (normal 80 to 150 units).

The admitting diagnosis was that of a perforated peptic ulcer with the secondary possibility of an acute pancreatitis. The patient was taken to the operating room several hours after admission and an exploratory laparotomy was performed. The findings at the operation were all negative except for dilatation of the small intestine. One hour after the operation, the patient suddenly expired.

The postmortem findings were as follows: The heart weighed 650 grams. The valves were normal; the aortic measured 9 cm., the pulmonic 8 centimeters. The wall of the left ventricle measured 20 mm., and that of the right ventricle 2 to 3 centimeters. Tree-barking and stellate scars were found in the first portion of the ascending aorta. At the level of the innominate artery there was a dissecting aneurysm which dissected distally down into both common iliacs. There were no intimal tears and no sites of external rupture. The dissection involved the innominate and superior mesenteric vessels.

There was marked dilatation of the stomach, and of both the large and small intestine. There were some areas of the small bowel in which the wall appeared somewhat hemorrhagic. There was also hemorrhage about the head of the pancreas. On sectioning, the pancreas did not appear abnormal.

The aorta showed medial necrosis and cyst formation on microscopic examination. Changes in the pancreas were minimal and were thought mostly to represent postmortem autolysis.

In 16 cases, the pain radiated to the back. Radiation was present in the chest in 7 patients in whom the initial pain occurred in the neck, chest, or intrascapular or similar areas.

Radiation into the flanks was described in 4 cases, and at necropsy this was found to have been due to dissection of one or both renal arteries, resulting in partial or complete occlusion and renal infarction. Blain, Glynn, and Hiratzka noted flank pain simulating that of renal colic in 2 patients, in both of whom the renal arteries were involved in the dissection. They reviewed the literature and found 15 cases with symptoms referable to the urologic system. Of these 15, the correct antemortem diagnosis was made in 9, indicating that a history of radiation of pain to the flanks seems to facilitate the diagnosis.

Radiation of pain to the extremities does not occur with as great frequency as might be expected. In 8 instances the pain was noted to radiate down either the upper or lower limbs.

Recurrent Pain. Of the 45 patients with initial pain as the presenting symptom, recur-
rent pain occurred in 29. This pain was described variously as a dull ache resembling the initial pain but less severe, an intermittent aching, a pleuritic pain, a tearing pain, or a sharp, needle-like pain.

Recurrent pain was present in the chest in 9 patients, and in several of these it had a pleuritic character, being aggravated by respiration. Of the 11 patients having recurrent pain in the abdomen the discomfort was most frequently described as a full aching pain. In the remaining patients the pain centered in the back in 4, in the flanks in 3, in the shoulders in one, and in the legs in one.

Recurrence of pain was, therefore, quite common, and indicated either progressive dissection or ischemia resulting from interruption of the blood supply to such locations as the intestinal tract, kidneys, spinal cord, or extremities.

**Onset of Syncope.** A history of an attack of syncope with recovery of consciousness and no history of pain occurred in only one patient. In another patient a syncopal attack occurred initially, but this was subsequently followed by epigastric pain. In 3 additional patients, syncope occurred, but it was preceded by chest pain in 2 of these, and neck pain in the third. The occurrence of syncope in association with dissecting aneurysm was stressed by Hamburger and Ferris,28 who described 6 patients in whom the outstanding symptom at the onset was syncope or weakness. Four of their 6 patients fainted at the onset or shortly thereafter, and the other 2 suffered from dizziness and weakness, but did not lose consciousness. They point out the fact that syncope is uncommon in association with myocardial infarction, but not with dissecting aneurysm. The suggestion was offered that the syncope was due to involvement of the depressor nerve endings in the aortic arch. The more likely explanation is cerebral ischemia either resulting from the pooling of large quantities of blood in the false passage, or due to blockage or dissection of the innominate and common carotid arteries.

Six patients, 2 of whom had hemiplegia, were admitted to the hospital while unconscious, with no antecedent history of pain. The dissection in these patients had involved one or more of the major blood vessels to the brain.

**Other Symptoms.** In addition to the major presenting symptomatology which has just been discussed, a tabulation of secondary symptoms may be found in table 4. Dyspnea predominated, being recorded in 25 patients. Next in frequency was vomiting, found in 12 patients. In 8 of these with vomiting the dissection had extended down into the abdominal aorta. Oliguria was reported in 5, and in 4 of these the renal arteries were involved in the dissection. In the 3 patients with hemoptyysis, necropsy disclosed dissection along the pulmonary arteries and into the roots of both lungs. Melena occurred in 2 patients, in one of whom the dissection had involved the superior mesenteric artery. The clinical-pathologic correlation of these symptoms has been de-

<table>
<thead>
<tr>
<th>TABLE 4.—Secondary Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Symptom</strong></td>
</tr>
<tr>
<td>Dyspnea</td>
</tr>
<tr>
<td>Vomiting</td>
</tr>
<tr>
<td>Nausea</td>
</tr>
<tr>
<td>Orthopnea</td>
</tr>
<tr>
<td>Oliguria</td>
</tr>
<tr>
<td>Hemoptyysis</td>
</tr>
<tr>
<td>Ankle edema</td>
</tr>
<tr>
<td>Melena</td>
</tr>
<tr>
<td>Hematemesis</td>
</tr>
<tr>
<td>Hematuria</td>
</tr>
</tbody>
</table>

scribed so that their occurrence can be properly evaluated and interpreted.

**CLINICAL FINDINGS**

**General Appearance of the Patient.** The patient was usually in acute distress, writhing and tossing about, complaining bitterly of excruciating pain in his chest or abdomen. The pain was often so intense as to cause him to double up or lie on the floor in an attempt to obtain relief. He frequently presented the picture of shock, with pallor, perspiration, and cold, clammy extremities.

**Pulse Rate and Respiration.** In the majority of cases, the pulse rate at the time of the dissection was recorded as ranging between 80 to 100 per minute. The highest rate was 120 per minute, and this was found in 4 patients. In 5 instances, a bradycardia was present with
rates of 50 per minute in 3, and 40 per minute in the other 2 subjects. Both patients with rates of 40 per minute were thought to have a complete heart block. At necropsy, in one of these, the dissection had descended into the interauricular septum to approach the region of the A-V node. Similar findings were not present in the second case. Davis described a patient in whom the dissection had descended inferiorly into the interventricular septum, and finally ruptured into the right ventricle.

In 4 of the “chronic” cases, cardiac arrhythmias were present. These consisted of complete heart block, auricular fibrillation in 2 cases, and a ventricular tachycardia complicating a recent posterior myocardial infarction.

<table>
<thead>
<tr>
<th>Table 5.—Previous History of Hypertension, and Presence of Hypertension, Normal Blood Pressure, and Shock on Admission</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension on Admission</td>
</tr>
<tr>
<td>---------------------------</td>
</tr>
<tr>
<td>Previous History of Hypertension (31 cases)…</td>
</tr>
<tr>
<td>No Previous History of Hypertension (24 cases) …</td>
</tr>
<tr>
<td><strong>Total</strong> …</td>
</tr>
</tbody>
</table>

The absence of arrhythmias in the acute cases is worthy of attention. There were none, except in the two instances of suspected complete heart block described above, and in an additional case in a patient with auricular premature contractions. In the 17 cases reported by David, McPeak, Vivas-Salas, and White, the maximum pulse rate was also 120, and likewise there were no cardiac arrhythmias.

The changes in the respiratory rate are worthy only of brief mention. An increase in the respiratory rate at the onset was universal except in those patients who had been given large doses of morphine. The sudden development of dyspnea after the onset was usually caused by external rupture associated with bleeding into the pleural cavity or mediastinum, or by dissection of the pulmonary artery.

Temperature. Of 43 patients admitted during or shortly after the onset of dissection, an elevated temperature varying from 99 to 103 F. was present in 20 instances. Thirteen patients were admitted in shock with subnormal blood pressures, and in all of these the temperature was normal or subnormal. There were 10 others who appeared to be in shock despite elevated blood pressures. Members of this latter group had pallor, perspiration, cold and clammy extremities, and similar symptoms, and their temperatures were either normal or subnormal.

All patients in the hospital who survived forty-eight or more hours were noted to have a febrile course with their temperatures fluctuating irregularly from 99 to 102 F. In those patients who went into shock after admission the temperature became subnormal.

Finally, in the absence of shock, fever was common during the acute and subacute stages of dissecting aneurysm.

Observations on Blood Pressure. It is well established that antecedent hypertension is a major factor in the evolution of a dissecting aneurysm. David, McPeak, Vivas-Salas, and White were able to establish a previous history of hypertension in each of their 17 patients. Such was not the case in the present series of patients, composed of persons of the low-income group seen in a large general hospital. Because of the absence of a previous history in many of them, and the not infrequent finding of a normal or subnormal blood pressure on admission, the findings were carefully reviewed (table 5). Values above 140 mm. of mercury systolic and 90 mm. of mercury diastolic were considered abnormal.

A previous history of hypertension (abnormal elevation of both systolic and diastolic) was present in 34 patients (58 per cent), and absent in 24 (42 per cent). In most of these the hypertension was noted to have been of long standing.

Hypertension on admission was present in 23 of the 34 patients with a previous history, and in 10 of the 24 with no antecedent history. The total, therefore, of patients with elevated blood pressures on admission was 33 (56 per cent) of the entire group of 58.

There were 5 patients with systolic hypertension only on admission, and 7 with normal
blood pressures. The group admitted in shock, with subnormal blood pressure, consisted of 13 patients (22 per cent of total). In addition, there were, as previously stated, many patients who clinically appeared to be in shock, in whom the blood pressure was found to be abnormally elevated, having frequently fallen from an even higher original pressure. We have followed the conventional pattern of shock and listed only those patients having subnormal blood pressures in addition to the clinical pattern of shock. The subnormal pressures in some instances is attributed to severe blood loss, and in others, to cardiac tamponade resulting from hemo-pericardium.

Persistence of hypertension in these patients has been thought by some to be of diagnostic value in the differentiation between dissecting aneurysm and coronary occlusion, in that the blood pressure would be more apt to drop in the latter condition than in the former. Actually, those patients with hypertension who develop a myocardial infarction do have a drop in blood pressure, but the blood pressure may remain above the normal range. Nine of the patients with elevated blood pressures on admission showed a significant drop in pressure during their hospitalization. Two of this group developed aortic diastolic murmurs and their blood pressures fell from 210/120 to 132/72, and from 190/110 to 140/75 respectively. Careful observation should disclose that this type of change occurs frequently, and when it does its diagnostic inference is far more important than that of the maintenance of an elevated pressure. The remaining 7 patients developed shock with marked reduction in their blood pressures some time after admission. Actually, there were 34 (53 per cent) of the patients in whom shock, normal blood pressure, and systolic hypertension were present on admission, or whose blood pressure following admission was not sustained at the admission level.

The 11 patients without previous history of hypertension, who were admitted in shock or with normal blood pressures, all had left ventricular hypertrophy post mortem in the absence of valvular disease. The left ventricular wall measured 14 to 21 mm. in these subjects. Because of this, it was felt that pre-existing hypertension has been established either directly or indirectly in all of the cases reviewed.

**Pleural Effusion.** Pleural fluid was detected on physical examination in 6 patients; in 4 it was in the left side of the chest, and in the remaining 2 it was bilateral in one and on the right in the other. The increased incidence of left-sided hemothorax over hemothorax of the right side in dissecting aneurysms is well known. Hemorrhage into the left pleural cavity at post-mortem examination was present in 11 of the 58 subjects (19 per cent). In 2 of these there was an accompanying hemo-pericardium. Rupture into the right pleural cavity was present at autopsy in 2 instances. The increased incidence of hemorrhage into the left pleural cavity as contrasted to the right can be explained by the close proximity of the aorta to the left pleural cavity, and by the fact that when dissection of the pulmonary artery occurs it apparently follows the line of least resistance along the left main branch and into the hilum of the left lung.

**Cardiac Enlargement.** Initial physical examination detected cardiac enlargement as being present in 38 of the 58 patients. In 5 additional patients, fluid in the left side of the chest interfered with accurate estimation of the heart size. At autopsy there were only ten hearts weighing less than 400 grams. In each of these cases there was either a history of hypertension or hypertension had been present on admission. Twenty-two per cent of the hearts weighed between 400 and 500 grams, and 55 per cent of the hearts were heavier than 500 grams. There were only 2 subjects in whom the left ventricle measured less than 14 mm. in thickness.

**Cardiac Tamponade.** Hemorrhage into the pericardium was suspected in only one patient prior to death. At autopsy it was found in 20 of the 58 subjects (34 per cent). The pericardium proved to be the commonest site of rupture.

**Pericardial Friction Rub.** A to-and-fro pericardial friction rub was detected in 3 patients. This finding in association with a dissecting aneurysm may be due to slow seepage of blood into the pericardial sac through an adventitial perforation, to a uremic pericarditis from dis-
section of the renal arteries and the resultant uremia, or, finally, to a pericarditis accompany-
ing myocardial infarction which has resulted from involvement of the coronary arteries by dissection. If the friction rub is due to seepage of blood through an adventitial perforation it is a particularly ominous sign in regard to the immediate prognosis, and should be looked upon as a herald of incipient cardiac tamponade.

Cardiac Murmurs. Resnik and Keefer,29 in 1925, reported the presence of aortic insufficiency in a 57 year old Negro with negative serologic reactions, who at postmortem ex-

TABLE 6.—Distribution of Cardiac Murmurs in Fifty-Eight Patients With Dissecting Aneurysm of the Aorta

<table>
<thead>
<tr>
<th>Type of Murmur</th>
<th>Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic diastolic (alone)</td>
<td>5</td>
</tr>
<tr>
<td>Aortic diastolic and aortic systolic</td>
<td>8</td>
</tr>
<tr>
<td>Aortic diastolic and systolic, and mitral systolic</td>
<td>3</td>
</tr>
<tr>
<td>Aortic systolic (alone)</td>
<td>3</td>
</tr>
<tr>
<td>Aortic systolic and mitral systolic</td>
<td>3</td>
</tr>
<tr>
<td>Mitral systolic and mitral diastolic</td>
<td>1</td>
</tr>
<tr>
<td>Mitral systolic (alone)</td>
<td>9</td>
</tr>
<tr>
<td>Total</td>
<td>32</td>
</tr>
</tbody>
</table>

pathologic process. The explanation of the peripheral signs of aortic insufficiency was that the drop in blood pressure was due to the leakage of blood from the main channel into the aneu-

There are numerous case reports31–37 of patients admitted with the findings of aortic insufficiency and aneurysmal dilatation of the ascending aorta, in whom, despite negative serologic reactions, the diagnosis of syphilitic heart disease was made. At autopsy a dissecting aneurysm was found and the aortic valve appeared normal. Gouley and Anderson,38 in 1940, reported 6 chronic “healed” dissecting aneu-

ysms of the aorta, all associated with aortic diastolic murmurs with peripheral signs of aortic insufficiency. Five out of the 6 patients had negative serologic reactions. The antemor-

tem diagnosis in each case had been that of syphilitic heart disease. The presence of negative serologic reactions is important as approximately 90 per cent of patients with syphilitic aortitis have positive serologic reactions.39 Consequently, an aortic diastolic murmur accom-
panied by aneurysmal dilatation of the ascending aorta and negative serologic reactions should make the clinician consider the diagnosis of a dissecting aneurysm.

Incidence of Aortic Diastolic Murmurs in Fifty-Eight Cases of Dissecting Aneurysm of the Aorta Proved at Autopsy. In table 6 there is tabulated the incidence of cardiac murmurs found in 32 of the 58 patients. Aortic diastolic murmurs were present in 16 patients (27.5 per cent of the total). In 2 instances the di-

astolic murmurs had a musical quality. In 4 subjects the heart tones were inaudible because of cardiac tamponade, so that the incidence of aortic insufficiency could have been higher. David, McPeak, Vivas-Salas, and White41 found basal diastolic murmurs in 9 of 17 patients (56 per cent). They felt that this incidence was higher than that expected among hypertensive patients, but that any attempt to correlate the incidence of aortic dissection on such data might prove misleading. Flaxman42 found aortic diastolic murmurs in 10 of 19 persons with dissecting aneurysm at the Cook County Hos-

pital. Recently Baer and Goldburgh43 reported 44 patients, 6 of whom had aortic diastolic murmurs.

One hundred consecutive autopsy cases of hypertensive heart disease were reviewed to determine the incidence of aortic diastolic murmurs accompanying hypertension in the ab-
sence of aortic valve disease. Aortic diastolic murmurs were present in 2 cases (2 per cent). Garvin\textsuperscript{40} reviewed 200 consecutive cases of hypertension and found aortic diastolic murmurs in 7 per cent.

The total number of mitral systolic murmurs in the present series was 16, exactly equal to the incidence of aortic diastolic murmurs (table 4). This certainly is not the usual finding in hypertension.

Five of the 16 instances of aortic diastolic murmurs were found in a group of 15 patients with chronic or "healed" dissecting aneurysms. The remaining 11 were distributed among 43 individuals with acute dissections. In several of the patients among the acute cases the aortic diastolic murmur was not present upon admission, but appeared during progressive dissection after hospitalization.

<table>
<thead>
<tr>
<th>54 Cases of Dissecting Aneurysm of the Aorta</th>
<th>No. of Cases</th>
<th>%</th>
<th>100 Cases of Hypertension Without Dissection</th>
<th>No. of Cases</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I: Pulmonic valve 0.5 to 2.0 cm. greater than aortic valve</td>
<td>20</td>
<td>37</td>
<td>Group I: Pulmonic valve 0.5 to 1.5 cm. greater than aortic valve</td>
<td>38</td>
<td>38</td>
</tr>
<tr>
<td>Group II: Aortic and pulmonic valves equal</td>
<td>13</td>
<td>24.2</td>
<td>Group II: Aortic and pulmonic valves equal</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Group III: Aortic valve 0.5 to 4.2 cm. greater than pulmonic</td>
<td>21</td>
<td>38.8</td>
<td>Group III: Aortic valve 0.5 to 1.5 cm. greater than pulmonic</td>
<td>42</td>
<td>42</td>
</tr>
</tbody>
</table>

**Explanations of the Mechanism of the Development of Aortic Insufficiency in Dissecting Aneurysms of the Aorta.** Numerous explanations have been offered for the mechanism of the appearance of aortic insufficiency in association with dissecting aneurysms. Two of these have already been discussed. The first was that of Resnik and Keefer,\textsuperscript{29} who felt that the false sac functioned similarly to an arteriovenous shunt, and that regurgitation through the intimal tear above the aortic valves produced a diastolic murmur. The second was that of Hamman and Apperly,\textsuperscript{30} who felt that stretching of the aortic ring resulted in incompetence. Peery\textsuperscript{41} reported 6 cases of intimal tears without dissection localized in the proximal portion of the ascending aorta. He stated that if the intimal tear was at or just above an aortic commissure and transverse to the commissure, aortic insufficiency resulted. His explanation was that, owing to the gaping of the tear, the commissure loosened, and the corresponding cusp dropped to a lower level in the aortic ring. Another proposed explanation was that the dissection extended inferiorly into the aortic ring and that the resultant hematoma distorted the valve ring, or displaced one of the cusps inferiorly, and resulted in valvular incompetence. One of our patients was found at necropsy to have the right cusp displaced inferiorly by a hematoma which had dissected down into the aortic ring.

In the 16 patients with aortic insufficiency the dissection had descended down into the aortic ring in 10. In 4 others it had extended proximally to within 1, 2, 3, and 5 cm. of the aortic ring. In the remaining 2 patients, the dissection had extended proximally in the arch of the aorta to the level of the left subclavian artery.

Minimal pathologic changes of the aortic valve were found in 3 of the 16 patients with aortic insufficiency. In one patient the edges of the valve were thickened and rolled, with fusion between the right and posterior cusps. Two other patients had slight fusion of the edges of the aortic valve leaflets which were not sufficient to result in valvular incompetence.

Measurements of the aortic and pulmonary valve rings were recorded in 54 of the 58 patients. These were compared with similar measurements in 100 individuals with hypertensive heart disease without dissection. This was done to determine whether or not cystic medial necrosis found in the aortas of patients with dissecting aneurysms resulted in greater stretching of the aortic ring than in that due to hypertension not associated with medial necrosis. It was found (table 7) that there was no marked difference in these measurements.
in the two groups of patients. Actually, the incidence of aortic valve ring measurements exceeding that of the pulmonic valve ring was 38.8 per cent in the patients with dissecting aneurysms, and 42 per cent in the hypertensive subjects. (Normally, the pulmonic valve ring measurement exceeds the aortic by about 1 centimeter.)

An analysis was also made of the valve ring measurements of the 16 patients with dissecting aneurysm associated with aortic insufficiency (table 8). Here it was found that in only 2 patients in whom the aortic valve exceeded the pulmonic valve by 3.5 and 4.2 cm., could stretching of the valve ring be inferred as being the mechanism for the production of aortic insufficiency.

**Table 8.—Valve Measurements in Sixteen Patients**

**With Dissecting Aneurysm With Aortic Insufficiency**

<table>
<thead>
<tr>
<th>Measurements</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I: Pulmonic valve 0.5 to 2.0 cm. greater than aortic valve</td>
<td>6</td>
</tr>
<tr>
<td>Group II: Aortic and pulmonic valves equal</td>
<td>2</td>
</tr>
<tr>
<td>Group III: Aortic valve 0.5 to 4.2 cm. greater than pulmonic valve. Of this group in only 2 patients did the aortic exceed the pulmonic by more than 2 cm. (3.5 and 4.2 cm.)</td>
<td>8</td>
</tr>
</tbody>
</table>

**Significance of Aortic Diastolic Murmurs in Dissecting Aneurysm.** The sudden appearance of a basal aortic diastolic murmur in a patient with hypertension complaining of severe chest or abdominal pain is almost pathognomonic of a dissecting aneurysm of the aorta. To be considered in the differential diagnosis of the sudden appearance of an aortic insufficiency is rupture of an aortic valve cusp. The pain and shock of a dissecting aneurysm are usually absent in association with a ruptured cusp. A drop in the diastolic pressure occurs in both conditions. Ruptured cusps are usually accompanied by a tachycardia, whereas in dissecting aneurysms the pulse rate tends to be normal or that of a bradycardia. Finally, but of extreme importance, is the fact that when an aortic cusp ruptures the valve usually has been damaged by prior disease such as syphilis, arteriosclerosis, rheumatic valvulitis, or bacterial endocarditis.

**Pulsating Masses.** Abnormal pulsating masses in the neck were present in 3 patients, and in the abdomen of one. Correlation with postmortem findings showed that those in the neck were due to dissection of the common carotid artery, and that the one in the abdomen was due to dissection of the abdominal aorta.

**Tenderness of Blood Vessels Involved by Dissection.** Dissection of peripheral arteries is often accompanied by localized tenderness over the involved vessel. This was only observed in 2 cases, and both represented dissection of the common carotid arteries.

**Pulsations in the Major Peripheral Arteries.** Diminished or absent peripheral arterial pulsation in association with dissecting aneurysm of the aorta is caused by the vessel being involved in the dissection so that the false sac becomes distended with blood and partially or completely occludes the lumen, or, in addition, thrombosis may occur within the true lumen. Dissection of the major vessels of the aortic arch results in diminished or absent pulsations of those arteries going to the head or upper extremities, such as the carotid, brachial, or radial. When the dissection has extended inferiorly into the common iliacs it may cause diminished or absent pulsations in such vessels as the femorals, popliteals, and dorsalis pedis.

Eleven patients (19 per cent) were found to have unequal or absent pulsations of major peripheral arteries. This was accompanied by comparable significant differences in blood pressure in the involved extremities in all instances (table 9). The carotid, brachial, or radial artery as the site of unequal or absent pulsations in 5 instances, and the femorals in the remaining 6. These clinical observations were correlated with the postmortem findings, and, except for 2 instances where the findings were inadequately described, the vessels involved by dissection corresponded to those in which the abnormal pulsations had been detected.

In addition to the decreased or absent pulse, the involved extremity was not infrequently found to be cold and pale, with impairment of motor power and diminished sensory perception. These changes, when present, were tem-
porary, and lasted from minutes to hours, depending upon establishment of collateral circulation or re-entry of the false sac into the true lumen by intimal rupture. The inequality of pulse and blood pressure usually persisted despite the disappearance of the other signs of arterial occlusion.

The presence of significant differences in pulse or blood pressure in either upper or lower extremities should render the diagnosis obvious in suspected cases. Of course, an arterial embolus could be responsible for these differences.

Nissim recently reported a new sign in dissecting aneurysm found in a major peripheral vessel which had undergone dissection. This consisted of a reduplication of the pulse in the involved vessel, with a single pulse on the uninvolved side. The reduplication was thought to be attributable to the difference in the rate of flow in the true and false sacs.

**Neurologic Manifestations.** The neurologic disturbances encountered in persons with dissecting aneurysm of the aorta have not received proper emphasis in the literature. The recogni-

**Table 9.—Diminished or Absent Pulsations in Major Peripheral Arteries**

<table>
<thead>
<tr>
<th>Vessels and Findings</th>
<th>B. P.</th>
<th>Postmortem Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Left c. carotid—diminished pulsation</td>
<td>R.A. 185/130</td>
<td>Dissection of vessels of aortic arch not described</td>
</tr>
<tr>
<td></td>
<td>L.A. 150/130</td>
<td></td>
</tr>
<tr>
<td>2. Right c. carotid and right radial—diminished pulsation</td>
<td>R.A. 140/100</td>
<td>Innominate artery involved in the dissection</td>
</tr>
<tr>
<td></td>
<td>L.A. 180/100</td>
<td></td>
</tr>
<tr>
<td>3. Right radial and right brachial—absent pulsation</td>
<td>R.A. 0/0</td>
<td>Innominate artery, and right subclavian artery involved in dissection</td>
</tr>
<tr>
<td></td>
<td>L.A. 104/80</td>
<td></td>
</tr>
<tr>
<td>4. Both right and left radials and brachials—absent pulsations</td>
<td>R.A. 0/0</td>
<td>Dissection of vessels of aortic arch not described</td>
</tr>
<tr>
<td></td>
<td>L.A. 0/0</td>
<td></td>
</tr>
<tr>
<td>5. Right radial—diminished pulsation</td>
<td>R.A. 60/20</td>
<td>Innominate artery involved in the dissection</td>
</tr>
<tr>
<td></td>
<td>L.A. 175/125</td>
<td></td>
</tr>
<tr>
<td>6. Right femoral—diminished pulsation</td>
<td>R.L. 185/140</td>
<td>Common iliacs dissected</td>
</tr>
<tr>
<td></td>
<td>L.L. 270/145</td>
<td></td>
</tr>
<tr>
<td>7. Right femoral—diminished pulsation</td>
<td>R.L. 195/140</td>
<td>Common iliacs dissected</td>
</tr>
<tr>
<td></td>
<td>L.L. 225/140</td>
<td></td>
</tr>
<tr>
<td>8. Right femoral—diminished pulsation</td>
<td>No record</td>
<td>Common iliacs dissected</td>
</tr>
<tr>
<td>9. Left femoral—absent pulsation</td>
<td>R.L. 290/140</td>
<td>Common iliacs dissected</td>
</tr>
<tr>
<td></td>
<td>L.L. 0/0</td>
<td></td>
</tr>
<tr>
<td>10. Left femoral—absent pulsation</td>
<td>R.L. 190/140</td>
<td>Common iliacs dissected</td>
</tr>
<tr>
<td></td>
<td>L.L. 0/0</td>
<td></td>
</tr>
<tr>
<td>11. Both femorals—absent pulsation</td>
<td>R.L. 0/0</td>
<td>Common iliacs dissected</td>
</tr>
<tr>
<td></td>
<td>L.L. 0/0</td>
<td></td>
</tr>
</tbody>
</table>

R.A. = right arm; L.A. = left arm; R.L. = right leg; L.L. = left leg.

Differentiation between it and dissecting aneurysm is not always easy, but because of the therapeutic implications (use of heparin, dicumarol, and allied drugs) its importance is apparent.

Ellis described the presence of a pulsus paradoxus in a patient with dissecting aneurysm associated with hemopericardium. Its occurrence in conjunction with acute or chronic constrictive pericarditis is well known, and it should most certainly be anticipated in patients with dissecting aneurysm in whom hemopericardium occurs so frequently.

tion of such changes is of importance if only for the fact that when they are present in suspected cases the incidence of correct diagnosis is greatly increased. Weisman and Adams reported 38 patients with dissecting aneurysm seen at the Boston City Hospital over a ten-year period. These investigators found neurologic symptoms resulting from the dissection in 30 per cent of these patients, and in the patients with such symptoms the correct diagnosis was made in 80 per cent. This was about twice the incidence of correct diagnosis in the patients without neurologic manifestations.
Depending upon the locale of vascular interference, the resultant symptomatology may be found to fall into one of the following three groups: (1) Blockage of the iliac or axillary arteries may result in an ischemic necrosis of the peripheral nerves. The examiner is confronted with a pulseless, cold, weak extremity, with anesthesia and loss of tendon reflexes. These changes are often temporary, lasting from minutes to hours, followed by reappearance of the pulse and return of motor power and other functions, usually due to re-entry of the dissection into the lumen of the involved vessel. (2) Impairment of blood flow through the intercostal arteries is said to cause an ischemic necrosis of the spinal cord. Clinically, there is a flaccid paralysis, urinary retention, and loss of sensation below the involved level. Unfortunately, there is a paucity of pathologic studies of this mechanism. It is known that there is an abundant collateral circulation to the spinal cord; this is so ample that it appears improbable that interference with the lumbar and intercostal arteries alone would result in ischemic necrosis unless the flow through the spinal arteries was simultaneously impaired. (3) Finally, obstruction to the carotids or the innominate artery will result in cerebral ischemia. The clinical picture is variable and may be that of dizziness, confusion, stupor, syncope, hemiplegia, coma, convulsions, blindness, and the like.

Bowman and his collaborators stressed the diagnostic import of “wandering paralysis and vacillating sensory disturbances” occurring below the level of the umbilicus. These were attributed to spinal cord ischemia resulting from interruption of the lumbar and intercostal arteries.

Nine of the 58 patients of this report had definite neurologic symptomatology. Two of these had a left-sided hemiplegia with dissection of the right common carotid artery, found at necropsy. In 5 instances there was a transient weakness of one or both legs, accompanied by numbness and loss of sensation. The dissection was found to have extended down into the iliacs in each of the subjects. One patient had a flaccid paralysis of his right upper extremity due to dissection of the corresponding subclavian artery. Finally, one patient with a flaccid paralysis of both lower extremities and loss of reflexes and sensation was observed. Gross examination of the spinal cord seemed to reveal interruption of its vascular supply, but microscopic examination failed to reveal any pathologic changes. Both common iliacs had also been involved by dissection in this case.

In 5 of these 9 patients (55 per cent) with neurologic symptoms a correct diagnosis of dissecting aneurysm was made, corroborating the findings of Weisman and Adams, who found that the presence of neurologic changes tended to facilitate the correct diagnosis.

**Electrocardiogram in Dissecting Aneurysm of the Aorta**

The most difficult problem in the diagnosis of dissecting aneurysm of the aorta has been the differentiation between it and coronary occlusion. Of the 58 autopsied cases of dissecting aneurysm, 20 (34.5 per cent) were incorrectly diagnosed as coronary occlusion. This difficulty was first stressed in 1934 by White, Badger, and Castleman, who described a case of dissecting aneurysm in which the antemortem diagnosis had been that of a coronary occlusion despite an electrocardiogram showing no abnormality. Recently David, McPeak, Vivas-Salas, and White reported that none of the patterns of the electrocardiograms of 17 patients with dissecting aneurysm could be considered normal. Patterns of left ventricular strain or of changes suggesting coronary disease were found to predominate, but none of the patterns were described. The pattern of acute myocardial infarction was not found in any of their cases, and they felt that the absence of this pattern should lend support to the diagnosis of dissecting aneurysm in equivocal cases. Hargrove, discussing this problem, made the statement that in equivocal cases, an electrocardiogram demonstrating no abnormality favored dissecting aneurysm and that one showing abnormality did not rule it out.

Electrocardiograms were made in 40 of the 58 autopsied cases, and have been correlated with the pertinent clinical and pathologic findings (table 10).
TABLE 10.—Electrocardiograms in Forty Patients With Dissecting Aneurysm of the Aorta

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Electrocardiograph Findings and Interpretation</th>
<th>Clinical and Postmortem Data</th>
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Group I: Patterns of myocardial infarction, acute coronary insufficiency, etc.

1. RS-T₁, RS-T₂, and RS-T₃ depressed. Impression: abnormal electrocardiogram
   B. P. 90/50. Post mortem: coronary sclerosis, old anteroseptal infarct, and left ventricular hypertrophy

2. 5/13/44: T₁ inverted; RS-T₂ and RS-T₃ sagging. 5/14/44: T₁ now upright; T₂ diphasic; RS-T₂ and RS-T₃ depressed. Impression: acute coronary insufficiency
   B. P. 200/140, yet clinical findings of shock present

3. RS-T₂ and RS-T₃ depressed. Impression: abnormal electrocardiogram
   B. P. 290/100, yet clinical findings of shock present

4. RS-T₁ depressed. Small Q₂; RS-T₂ and RS-T₃ elevated. Impression: abnormal electrocardiogram
   B. P. 70/50 (in shock). Post mortem: coronary sclerosis and left ventricular hypertrophy

5. 3/12/41: Low-voltage, flat T waves in Leads I, II, III. 3/14/41: Q₃, deep Q₃; RS-T₂ and RS-T₃ elevated. Impression: recent posterior myocardial infarction
   B. P. 110/60 (in shock). Post mortem: right coronary artery stenotic, with no evidence of occlusion. Left ventricular hypertrophy present

6. T₁ diphasic; T₂ and T₃ flattened. Impression: abnormal electrocardiogram
   B. P. 100/80 (in shock). Post mortem: the dissection had extended down into the interauricular septum and subendocardially into both ventricles

7. T₁, T₂, and T₃ flattened. Impression: abnormal electrocardiogram
   B. P. 190/128 (clinical findings of shock). Post mortem: left ventricular hypertrophy

8. T₁, T₂, and T₃ low; low voltage. Impression: abnormal electrocardiogram
   B. P. 70/50 (in shock). Post mortem: left ventricular hypertrophy

9. 4/29/44: Left ventricular hypertrophy. 5/6/44: Recent posterior myocardial infarct
   B. P. 150/100 (clinical findings of shock). Later B. P. rose to 210/110. Post mortem: coronary sclerosis and recent and old myocardial infarcts

10. 3/7/43: Acute posterior myocardial infarct; pericarditis. 7/26/43: Impression: old posterior myocardial infarct
    Post mortem 7/27/43: healed dissecting aneurysm which had compressed orifice of the right coronary. Old posterior infarct. Left ventricular hypertrophy

11. 9/14/43: T₂ and T₃ inverted; RS-T₁ depressed; RS-T₂ and RS-T₃ elevated; Q₂ and Q₃ present. 12/10/43: RS-T₂ and RS-T₃ more elevated. Impression: old posterior infarct with recent acute insufficiency
    In shock at time of final ECG. Post mortem: the dissection involved the pulmonary artery and extended into the roots of both lungs. Left ventricular hypertrophy

Group II: Acute pericarditis

12. RS-T₁ and RS-T₂ elevated; RS-T₃ iso-electric; T₁ flat; T₂ and T₃ inverted. Impression: acute pericarditis
    Post mortem: fibrinous pericarditis with recent hemopericardium. Left ventricular hypertrophy

13. 1936: RS-T₁, RS-T₂, and RS-T₃ elevated. Impression: acute pericarditis. 1941: Left ventricular hypertrophy
    1936: Time of original dissection. 1941: Post mortem: healed dissecting aneurysm. Pericardium normal

14. RS-T₁, RS-T₂, and RS-T₃ elevated. Impression: acute pericarditis
    Post mortem: fibrinous pericarditis with recent hemopericardium

375
A classification of the various electrocardiographic patterns encountered in persons with dissecting aneurysm has been formulated after a careful study of the present series along with a review of the literature.

**Group I: Electrocardiographic Patterns of Myocardial Infarction and Acute Coronary Insufficiency.**

A. Myocardial Infarction: Infarction may result from dissection of the media of a coronary artery with resulting thrombosis and occlusion, or, the dissection may extend inferiorly in the aortic wall and compress a coronary vessel from without. Wainwright described a patient with serial electrocardiographic changes characteristic of both anterior and posterior myocardial infarction. The dissection in the aorta was noted to have descended inferiorly to involve the wall of the left coronary artery. The resultant aneurysm in the coronary wall

**Group II: Left ventricular hypertrophy.**

17-28 (inclusive) Left ventricular hypertrophy. One patient had an old posterior myocardial infarction. Post mortem: left ventricular hypertrophy. Heart weights: 400 to 700 grams

**Group III: Uremia**

15. T1 and T2 diphasic; T3 flat. Q-T interval 0.44 second. Impression: uremia, pericarditis

16. RS-T sagging. Q-T interval 0.44 second. Impression: uremia, prolonged Q-T interval

**Group IV: Left ventricular hypertrophy (tracings taken at time of dissection)**

29-34 (inclusive) Left ventricular hypertrophy

Post mortem: left ventricular hypertrophy. Heart weights 450 to 850 grams

**Group V: Left ventricular hypertrophy (all these tracings made antecedent to dissection, none being made at time of dissection)**

35. Auricular fibrillation

36. Prolonged A-V conduction. Right bundle branch block

37. Left ventricular hypertrophy. Complete heart block

38. Left bundle branch block

39. Recent coronary occlusion. Ventricular tachycardia. Left ventricular hypertrophy

40. Left ventricular hypertrophy. Acute posterior myocardial infarction

Post mortem: left ventricular hypertrophy. Heart weights: 400 to 700 grams

**Group VI: Miscellaneous—"chronic or healed" dissecting aneurysms**

35. Auricular fibrillation

36. Prolonged A-V conduction. Right bundle branch block

37. Left ventricular hypertrophy. Complete heart block

38. Left bundle branch block

39. Recent coronary occlusion. Ventricular tachycardia. Left ventricular hypertrophy

40. Left ventricular hypertrophy. Acute posterior myocardial infarction

Post mortem: left ventricular hypertrophy. Heart weights: 400 to 700 grams

**Clinical and Postmortem Data**

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<tr>
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<tbody>
<tr>
<td>15</td>
<td>T1 and T2 diphasic; T3 flat. Q-T interval 0.44 second. Impression: uremia, pericarditis</td>
<td>N.P.N. 74 mg.%. Post mortem: malignant nephrosclerosis with uremic pericarditis and uremic colitis</td>
</tr>
<tr>
<td>16</td>
<td>RS-T sagging. Q-T interval 0.44 second. Impression: uremia, prolonged Q-T interval</td>
<td>N.P.N. 80 mg.%. Post mortem: both renal arteries dissected resulting in thrombosis and renal infarction</td>
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**Table 10.—Concluded**

<table>
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<tr>
<th>Group III: Uremia</th>
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<td>N.P.N. 80 mg.%. Post mortem: both renal arteries dissected resulting in thrombosis and renal infarction</td>
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**Group IV: Left ventricular hypertrophy (tracings taken at time of dissection)**

17-28 (inclusive) Left ventricular hypertrophy. One patient had an old posterior myocardial infarction. Post mortem: left ventricular hypertrophy. Heart weights: 400 to 700 grams

**Group V: Left ventricular hypertrophy (all these tracings made antecedent to dissection, none being made at time of dissection)**

29-34 (inclusive) Left ventricular hypertrophy

Post mortem: left ventricular hypertrophy. Heart weights 450 to 850 grams

**Group VI: Miscellaneous—"chronic or healed" dissecting aneurysms**

35. Auricular fibrillation

36. Prolonged A-V conduction. Right bundle branch block

37. Left ventricular hypertrophy. Complete heart block

38. Left bundle branch block

39. Recent coronary occlusion. Ventricular tachycardia. Left ventricular hypertrophy

40. Left ventricular hypertrophy. Acute posterior myocardial infarction

Post mortem: left ventricular hypertrophy. Heart weights: 400 to 700 grams
was filled with a thrombus which occluded the vessel by compressing its lumen. Patient 10, whose history and electrocardiographic tracings are presented, was found at autopsy to have had the orifice of the right coronary artery occluded by a hematoma dissecting down into the aortic ring. An acute posterior myocardial infarction resulted. A case with similar associated electrocardiographic tracings has been reported by Bayley and Monte.49 In Cabot Case 3139149 at the Massachusetts General Hospital, both coronaries were involved in a like manner, but electrocardiographic changes typical of myocardial infarction were absent.

Case 10. The patient, a 28 year old white woman with a known history of hypertension of ten years' duration, entered the Los Angeles County Hospital on March 28, 1943, complaining of the sudden onset of severe, crushing substernal pain, which radiated to her left shoulder and down her left arm. The pain was unrelated to effort and came on about 2:00 A.M. while in bed. It was accompanied by dyspnea, nausea, and vomiting.

The patient was in acute distress and complained of severe substernal pain. Her skin was pale, moist, and clammy. Her temperature was 97 F., her pulse rate 110 per minute, and respiration rate 30 per minute. The blood pressure was 210/150. The retinal vessels had a copper-wire appearance; they were tortuous and arteriovenous nicking was present. The lungs were clear. The heart was enlarged on percussion. The point of maximal impulse was in the sixth intercostal space 2 cm. to the left of the midclavicular line. A diastolic murmur was heard over the primary and secondary aortic areas. The remainder of the physical examination revealed no abnormalities.
acute coronary insufficiency was made. On March 5, 1943, the RS-T segments in Leads I and II were elevated; in Lead III they were isoelectric. The diagnosis of possible pericarditis was made. On March 7, 1943, the RS-T segments in Lead I were depressed; in Leads II and III they were elevated; there was a prominent Q wave in Lead III. The diagnosis of acute posterior myocardial infarction was made. On March 28, 1943, the RS-T segment in Lead I was isoelectric, in Lead II depressed; the T waves in Leads II and III were inverted. On April 4, 1943, T₁ and T₂ were further inverted and coved. The diagnosis of healing posterior myocardial infarct was made.

During the first four weeks of hospitalization the patient's course was febrile with the temperature fluctuating between 100 and 101°F. On her third hospital day she had a convulsion, her blood pressure dropped to 80/0, and she remained stuporous for the ensuing twenty-four hours. An electrocardiogram at this time showed an acute posterior myocardial infarct. Following this, the patient complained of precordial pain for the next three and one-half weeks. A pericardial friction rub was first heard on March 20, 1943. On May 25, 1943, the patient was sent home.

On July 26, 1943, two months after discharge, she was readmitted to the hospital, and complained of severe precordial pain radiating through her chest to her back and up into her neck. She was dyspeptic, orthopneic, pale, cold, and tossing about with pain. She was afebrile. The blood pressure was 185/90. There were a few moist râles at both lung bases. The point of maximal impulse was in the sixth intercostal space 2 cm. outside the midepaximalic line. A mitral systolic and an aortic diastolic murmur were present. The findings of the remainder of the physical examination were noncontributory.

The electrocardiogram was that of an old posterior myocardial infarct.

Shortly after admission the patient became pulseless, comatose, and respiratory ceased.

On postmortem examination, the pericardial sac was found to be obliterated by adhesions and the visceral pericardium to be distended by a recent subepicardial hemorrhage. The heart weighed 500 grams. The left ventricular wall was hypertrophied and measured 16 millimeters. The aortic and pulmonic valves both measured 7.5 centimeters. The aorta had an old intimal tear 1.5 cm. above the aortic valve which communicated with an aneurysmal sac measuring 3 by 3 by 8 centimeters. On the left anterolateral aspect of the sac there was a recent tear which had ruptured into the adventitia and dissected down into the subepicardial space. This sac distended with blood occluded the ostium of the already narrowed right coronary artery. There was a healed myocardial infarct on the posterior wall of the left ventricle. Microscopic examination of the aorta showed medial necrosis with cyst formation.

Case 5 illustrates myocardial infarction without coronary occlusion. The patient, on admission, was in shock, and the electrocardiogram showed tracings characteristic of a recent posterior myocardial infarct. At autopsy there was no evidence of coronary occlusion, but the right coronary artery was markedly stenotic. Similar cases have been described by Kenny and by Bourne and Mills.

In addition, it is obvious that myocardial infarction may occur independently of the dissection. Electrocardiographic patterns of old myocardial infarction, posterior type, were found in two instances. In one of these (Case 32), coronary thrombosis and myocardial infarction had occurred antece dent to the dissection. In the other (Case 40), a chronic or "healed" dissection, it had occurred subsequent to the dissection. Because of the almost universal coexistence of hypertension in patients with dissecting aneurysms, and the known frequency with which coronary artery disease complicates hypertension, all three may be encountered in the same patient, and the diagnosis is made with difficulty.

B. Acute Coronary Insufficiency, due to Reduction of Coronary Blood Flow because of Shock, Tachycardia, Anemia, "Coronary Spasm": Moderate to severe coronary atherosclerosis present in many of these patients predisposes to the development of this type of pattern, namely, depression of the RS-T segments, with lowering to inversion of the T waves. No previous reports of this pattern in dissecting aneurysm were found in the literature. One reason is that this is a difficult, if not impossible diagnosis to make in the presence of left ventricular hypertrophy. Some feel that the pattern of left ventricular hypertrophy actually reflects left ventricular ischemia. Not infrequently these patients have been receiving digitalis which will also cause RS-T segment shifts and T-wave inversion. Patterns thought typical of acute coronary insufficiency were present in 3 cases (Cases 1, 2, and 3).

C. Acute Ventricular Strain Superimposed upon Old Posterior Myocardial Infarct: One
patient (Patient 11) was of particular interest in that the electrocardiographic pattern was that of acute right ventricular strain superimposed upon the pattern of an old posterior myocardial infarct. At autopsy the dissection was found to involve the pulmonary artery and its branches, extending into the hilar regions of both lungs.

*Group II: Pericarditis.* Weiss discussed the occurrence of pericarditis in association with dissecting aneurysm, and commented upon its infrequency. At least 5 cases were found which had been previously reported. All of these patients had pericardial friction rubs and electrocardiographic tracings typical of pericarditis. The mechanisms of the production of pericarditis in dissecting aneurysm are multiple. Most frequently, there is a slow leak of blood into the pericardial sac, and deposition of fibrin upon the lining of the pericardial cavity. This usually heralds an imminent cardiac tamponade. Secondly, a uremic pericarditis may be present, due to either dissection and resultant thrombosis of one or both renal arteries, or primary renal failure of malignant nephrosclerosis, chronic nephritis, and similar conditions. Still another cause would be pericarditis accompanying a myocardial infarction. Patterns of acute pericarditis were present in 4 of the patients in the present series. In one it was thought to be due to a uremic pericarditis. In 2 others (Patients 12 and 14) it was caused by a slow leakage of blood into the pericardial sac. Case 12 has been described in detail with illustrative electrocardiograms. In the remaining case the pericarditis accompanied a myocardial infarction caused by the dissection.

**Case 12.** The patient, a 35 year old Negro garage attendant, was admitted to the Los Angeles County Hospital on February 10, 1941, complaining of severe, crushing, substernal pain which radiated through to the back, and came on while he was engaging in sexual intercourse. The pain was accompanied by dyspnea, nausea, and vomiting. Hyper tension had been present since the patient was 12 years of age.

On admission, the patient was heavily sedated with morphine and had obvious respiratory depression. The temperature was 100.2° F., the pulse rate 120 per minute, and respiratory rate 12 per minute. Both pupils were constricted, and reacted poorly to light. The lungs were clear. Cardiac enlargement was present, the point of maximal impulse being in the fifth intercostal space near the anterior axillary line. Gallop rhythm was present. The blood pressure was 185/145 in the right arm, and 180/150 in the left arm. The remainder of the physical examination revealed no abnormality.

The hemoglobin was 60 per cent, and there were 3,7 million red blood cells and 8,200 white blood cells per cu. mm. of blood. The sedimentation rate was 28 mm. per hour. In the phenolsulfonphthalein test, the dye appeared in five minutes; 30 per cent was present in the first sample and 10 per cent in the second. The nonprotein nitrogen value was 32 mg. per 100 cc. of blood. The icteric index was 19 units. The Wassermann and Kahn reactions were negative. X-ray examination of the chest showed that the left ventricle was enlarged and that there was marked widening of the ascending aorta.

Several electrocardiograms were made (fig. 2). In the tracings made on January 13, 1941, the RS-T segments in Leads I and II were elevated; in Lead III they were isoelectric. Left axis deviation was manifest. The diagnosis was acute pericarditis. On January 16, 1941, elevation of the RS-T segments in Leads I and II was increased. On January 20, 1941, the RS-T segments in Leads I and II had returned to the base line; T\textsubscript{1} was flat and T\textsubscript{3} inverted. On February 5, 1941, there was evidence of electrical alternans in Leads II and III. The RS-T segments were now isoelectric. T\textsubscript{1} was low, T\textsubscript{2} and T\textsubscript{3} inverted.

During the hospitalization, the patient's temperature fluctuated between 100 and 101 F. Pain in the left side of his chest, aggravated by deep breathing, persisted. A week after admission a pericardial friction rub was detected, and the presence of pericarditis was substantiated by electrocardiograms. Later, a to-and-fro basal murmur appeared, and the diagnosis of a dissecting aneurysm was made. The patient appeared to be improving when he died suddenly on February 19, his ninth hospital day.

At autopsy, 600 cc. of clotted blood were found in the pericardial sac. The epicardium was shaggy, reddish gray in appearance, with tags of both fibrinous exudate and organized fibrous tissue. On the anterior surface of the aorta there was a small tear in the adventitia which is the site of rupture into the pericardial sac. When the aorta was opened, about 1 cm. above the valve, there were noted two transverse intimal tears which communicated with a dissecting aneurysm, which extended distally to the level of the right renal artery. At this point there was another intimal tear which marked the site of re-entry. The false sac was filled with a large ante-mortem blood clot. The heart weighed 800 grams. The left ventricular wall was 20 mm. in thickness, the right 7 millimeters. The aortic valve measured 5 cm., and the pulmonic 8 centimeters.
DISSECTING ANEURYSM OF AORTA

The microscopic examination of the aorta showed medial degeneration with cyst formation. The pericardium showed a subacute fibrinous pericarditis.

**Group III: Uremia.** Tracings consistent with uremia were found in 2 instances (Cases 15 and 16). In Patient 15 it was due to primary renal involvement in the form of malignant nephrosclerosis. At autopsy a uremic pericarditis and colitis were also found. In the other patient pattern of uremia occurring in association with dissecting aneurysms of the aorta has not been previously described.

**Case 15.** The patient, a 32 year old Mexican woman, entered the Los Angeles County Hospital on April 1, 1945, complaining of blindness, vomiting, and urinary frequency of two months' duration. At the age of 13 years she had had "kidney trouble." Five years ago, after the birth of her fifth baby, she began to have severe occipital headaches, and

![Fig. 2. Electrocardiograms of Patient 12 (see text)](http://circ.ahajournals.org/)

the uremia resulted from the dissection involving the renal arteries with resultant thrombosis and renal infarction. The electrocardiographic changes characteristic of uremia are prolongation of the Q-T interval, sagging of the RS-T segments, and T-wave flattening and inversion. These changes may be specifically related to the disturbances of calcium and phosphorus or potassium metabolism occurring in conjunction with uremia. The electrocardiographic pattern of hypertension was observed for the first time. Her sixth and seventh pregnancies were complicated by severe headaches and ankle edema. Progressive loss of vision began just prior to the birth of her eighth child (December, 1942).

The patient appeared well nourished and in no acute distress. The temperature was 99 F., the pulse rate 90 per minute, and respiration rate 20 per minute. The blood pressure was 222/180. Light perception was diminished in the right eye, and absent in the left. Examination of the fundi showed narrowed arterioles, arteriovenous nicking, hemor-
rhages, and exudates. The discs were pale. The lungs were clear. The heart was enlarged and the point of maximal impulse was in the fifth intercostal space at the anterior axillary line. The sounds were of fair quality. The remainder of the physical examination revealed no abnormality.

The red blood count was 4.9 million, and the hemoglobin was 92 per cent. There were 15,050 white blood cells per 100 cu. mm. of blood. The urine examination showed a specific gravity fixed between 1.010 and 1.013, +3 albumin reaction, 75 to 100 red blood cells and 30 to 40 white blood cells per high-powered field. The nonprotein nitrogen was 74 mg. per 100 cc. of blood. The Wassermann and Kahn reactions were negative.

The electrocardiogram showed a small Q3 and a prominent Q4. T1 and T2 were diphasic, and T3 was flat. The T wave in CF4 was diphasic. The Q-T interval was 0.44 second (fig. 3). These changes were thought consistent with those due to uremia.

On her third hospital day the patient complained of severe epigastric pain radiating to the precordium. The pain persisted for about a week and was localized in the epigastrium. Her course was progressively downhill, and after three months of hospitalization, she became disoriented and expired on July 6, 1945. The antemortem diagnosis was malignant nephrosclerosis.

On postmortem examination the pericardial sac was found to contain no free fluid. There were recent adhesions between the visceral and parietal pericardium, and a recent fibrous pericarditis was found over the epicardium. The heart weighed 500 grams. The left ventricle measured 15 mm., the right 3 millimeters. In the aorta there was an intimal tear just distal to the origin of the left subclavian artery. This communicated with a dissecting aneurysm which extended distally to a point midway between the superior and inferior mesenteric arteries. The false sac was filled with two large organized blood clots.

The kidneys were normal in size and their surfaces were covered with numerous petechial hemorrhages. Microscopically, arteriolonecrosis was present accompanied by hemorrhage into the glomeruli and the interstitial tissue. The findings were thought to be typical of malignant nephrosclerosis.

**Group IV: Left Ventricular Hypertrophy.** There were 12 patients in this group whose electrocardiograms were made at the time of the dissection. All these patients were found at autopsy to have left ventricular hypertrophy.

In addition there were 6 patients whose electrocardiograms were made prior to the onset of dissection. In none of these had electrocardiograms been made at the time of the dissection. All of the electrocardiographic patterns were characteristic of left ventricular hypertrophy, the presence of which was corroborated at the postmortem examination.

**Group V: Miscellaneous.** This included the patients with “healed” or chronic cases who re-entered the hospital finally to die of congestive heart failure, coronary artery disease, cerebral hemorrhage, or similar catastrophe. Electrocardiograms in 6 of these patients were chiefly the patterns of left ventricular hypertrophy, bundle branch block, coronary occlusion, or cardiac arrhythmias (such as auricular fibrillation, ventricular tachycardia, and complete heart block with ventricular extrasystoles).

![Fig. 3. Electrocardiograms of Patient 15 made on April 2, 1943, showing prolonged Q-T interval (see text).](http://circ.ahajournals.org/)

**Comment on the Electrocardiographic Patterns Found in Association with Dissecting Aneurysm:**

1. Review of the electrocardiograms in 40 patients with dissecting aneurysm revealed them all to be definitely abnormal.

2. The most common pattern was that of left ventricular hypertrophy. This was present in 25 cases (62 per cent).

3. The patterns of myocardial infarction, acute coronary insufficiency, and the like, were found in 13 cases (22.7 per cent).
4. Patterns of acute pericarditis, due to seepage of blood into the pericardial sac, were found in 4 cases (10 per cent).

5. Electrocardiographic patterns of uremia occurred in 2 patients (3.4 per cent), secondary to dissection of the renal arteries, or owing to primary renal disease.

6. Absence of electrocardiographic changes of myocardial infarction should support the diagnosis of dissecting aneurysm in equivocal cases. The presence of such changes does not rule out the possibility of a dissecting aneurysm, as it has been shown that a dissecting aneurysm may result in or be accompanied by a myocardial infarction. There is no specific electrocardiographic pattern in dissecting aneurysm. The patterns may be as varied as the clinical syndromes encountered.

**ROENTGENOLOGIC FINDINGS**

Since the patient in the acute stage of suspected dissecting aneurysm should not be moved and should be spared any unnecessary activity the x-ray study at this time should be limited to a bedside examination. Also the x-ray findings will hardly affect the therapeutic approach. If the initial dissection is survived, thorough roentgen-ray study can be performed at a later date.

Davy and Gates, in 1922, reported the first case of dissecting aneurysm diagnosed ante-mortem in which the diagnosis had been confirmed by the x-ray findings. The x-ray film was reported as showing a widened and somewhat radiolucent shadow of the aortic arch and descending aorta.

Ritvo and Votta felt that the most significant finding was the presence of a widened supracardiac shadow. If the dissection was localized a circumscribed or saccular deformity of the aorta resulted. If the dissection was diffuse and widespread a generalized widening of the supracardiac shadow was expected. Wood, Pendergrass, and Ostrum felt that the increase in width of the innominate or other large vessels originating from the aortic arch was the most pathognomonic roentgen-ray sign.

X-ray examination of the chest was performed in 20 of the 58 patients of this report. Ten, or 50 per cent of these, showed widening and dilatation of the ascending and transverse portions of the arch of the aorta. In 2 additional patients the widening was limited to the descending thoracic aorta. In 3 of these the findings were reported as being consistent with a dissecting aneurysm, and in 2 more as being consistent with aneurysm.

On fluoroscopic examination the aortic pulsations are usually diminished. However, they may be normal or even increased, depending upon the location and the extent of the dissection. As in other aneurysms, the esophagus and trachea may be displaced.

A most significant finding is the sudden appearance of an increase in the width of the aorta, in the region of the arch or in its descending portion. Roesler, Gifford, and Betts reported a case in which this was detected by serial roentgenograms, and thus the correct diagnosis was facilitated.

Golden and Weens reported a dissecting aneurysm involving the arch and descending aorta in which angiocardiography using 70 per cent diodrast demonstrated a double-barrelled contour. The diagnosis of dissecting aneurysm was subsequently confirmed at operation.

Cardiac enlargement, chiefly left ventricular in type, was found in 11 of the 20 patients, and in an additional 4 its presence was obscured by a coexisting left pleural effusion.

If external rupture into the pericardial sac has occurred at the time the x-ray examination of the chest is made the findings will be those of a pericardial effusion. Rupture into the left pleural cavity, almost always left sided, can also be demonstrated by roentgenograms. A left pleural effusion was present in 4 of the present series of cases (20 per cent).

Weiss, Kinney, and Maher described a chronic "healed" dissecting aneurysm in which extensive atherosclerosis developed in the false sac. The roentgenogram of the chest disclosed a double-barrelled aorta with calcification in both the true and false sac. No similar case has been described in the literature.

**LABORATORY FINDINGS**

**Serologic Studies.** Wassermann and Kahn tests were performed in 48 of the 58 patients. Of these, the reaction was reported positive
in 6 instances. Three of the 6 patients were found at autopsy to have syphilitic aortitis. A fourth patient was inadequately studied in that a microscopic study was not performed, and the diagnosis of syphilitic aortitis was made on gross appearance alone. The gross appearance of medial necrosis is often not unlike that of syphilitic aortitis, so the diagnosis in this case is certainly open to question. The remaining 2 patients with positive reactions had no evidence of syphilitic aortitis.

In addition, there were 2 other patients with negative reactions who had syphilitic aortitis at autopsy, making a total of 5 definite cases of syphilitic aortitis.

The significance of negative serologic reactions in the presence of aortic insufficiency associated with aneurysmal dilatation of the ascending aorta has been discussed under the heading of cardiac murmurs. Of the 16 patients in whom aortic diastolic murmurs were observed the serologic reaction was positive in 2 (12 per cent). At autopsy both of these had syphilitic aortitis, but the aortic valves were uninvolved. This low percentage of positive serologic reactions encountered (in association with diastolic murmurs, and the like) is certainly in sharp contrast to the known 80 to 90 per cent positive reactions in patients with syphilitic aneurysms and coexisting aortic insufficiency. Because of this, it is worthy of re-emphasis that dissecting aneurysm be considered in the diagnosis of those patients with aortic insufficiency, aneurysmal dilatation of the ascending aorta, and negative serologic reactions.

**Blood Count.** Anemia: A normochromic normocytic anemia was often present shortly after the onset of dissection. It was probably caused by acute blood loss from hemorrhage into the mediastinum, pleural cavity, peritoneal space, or elsewhere. A progressive drop in the red blood cell count and hemoglobin value was frequently observed in those patients who survived the initial insult and advanced into the "subacute" or "chronic" stages. One patient who survived fifteen days had an initial blood count of 4.5 million red blood cells and hemoglobin value of 10.4 grams. Shortly before death his count had dropped to 3.5 million red blood cells and the hemoglobin value was 8 grams. Another patient with a "chronic or healed" case had a red blood cell count of 5.8 million with 17 grams of hemoglobin initially. Two months after admission there were 3.6 million red blood cells and 10 grams of hemoglobin.

**Leukocytosis:** Most patients at the onset showed a moderate to a marked leukocytosis with a corresponding increase in the polymorphonuclear leukocytes. The white blood cell count varied between 9,600 and 29,500, and most commonly was about 15,000 with a marked polymnucleosis. The elevated white blood cell count probably was caused by hemorrhage into the false sac or one of the serous cavities.

**Urinalysis.** Albuminuria varying from a trace to 4 plus was found in many of the cases.

Hematuria was present in 5 instances on microscopic study; in 3 additional ones the urine had been observed as being grossly bloody. One of these with gross hematuria encountered at autopsy had a necrotizing arteriolitis of the kidney, and the clinical diagnosis had been malignant nephrosclerosis. The other 2 patients with gross hematuria were found to have had dissection of the renal arteries with resultant thrombosis and renal infarction. Two of these patients with only microscopic hematuria were likewise observed to have dissection involving the renal arteries.

In 1945, Blain, Glynn, and Hiratzka reviewed the literature and were only able to find 15 patients with urologic symptoms such as flank pain and hematuria. Hematuria was only present in 5 (33 per cent). Of the 15 patients with urologic findings the correct diagnosis was made in 9 (60 per cent), which is well above the average. In 2 of the 3 patients with gross hematuria in the present series, the correct diagnosis was made.

Halprin reported a case of dissecting aneurysm associated with bilateral occlusion of both renal arteries, which presented the picture of uremia. Buckley reported 2 patients with involvement of the renal arteries, both of whom had gross hematuria. Davis and his associates described a patient with dissection of both renal arteries, who was thought to have acute nephritis.
Hematuria apparently is not a common finding in persons with dissecting aneurysm, but when it occurs it probably indicates dissection of one or more renal arteries with thrombosis and infarction. It may also be due to a concomitant malignant nephrosclerosis.

Nonprotein Nitrogen. Nonprotein nitrogen determinations were made in 9 patients, 7 of whom were found to have elevated values ranging from 47 to 85 mg. per 100 cc. of blood. In 3 of these with elevated values the renal arteries had been involved in the dissection; a fourth patient had malignant nephrosclerosis. Elevation of the blood nonprotein nitrogen may have a multiple pathogenic basis, resulting from a prerenal azotemia secondary to shock, caused by severe blood loss or dissection of the renal arteries with associated renal infarction, or, finally, from destruction and absorption of blood at the site of hemorrhage or external rupture.

Pleural Fluid. In those patients with pleural fluid, a diagnostic tap may reveal a bloody fluid, most frequently present in the left pleural cavity.

Icteric Index. Osgood, Gourley, and Baker in 1936 first called attention to the presence of icterus in a patient with a dissecting aneurysm. The elevation was thought most likely a result of increased bilirubin production from the site of hemorrhage. Clinical icterus is usually absent, but occasionally it may be detected, especially when there is slow hemorrhage into the mediastinum or retroperitoneal space. Clinical icterus was detected in one of the patients in our series. The icteric index was 18. Unfortunately, the test was not performed in any of the other patients, so its value in the diagnosis of dissecting aneurysm cannot be properly determined.

Serum Amylase. In 2 patients an elevated serum amylase was detected. Both of these had abdominal pain and were suspected of having acute pancreatitis. One of these whose serum amylase was 300 units was subjected to an exploratory operation with the preoperative diagnosis of a perforated peptic ulcer with a contiguous pancreatitis. The operative findings were not remarkable, except for small-bowel distension. At autopsy there was found a dissecting aneurysm involving the superior mesenteric artery with hemorrhage about the head of the pancreas. In the other patient the serum amylase value was 500 units. In this patient the dissection of the aorta had only extended inferiorly to the beginning of the lower third of the thoracic aorta. The pancreas appeared grossly normal. No other reports of elevation of serum amylase in association with dissecting aneurysm were found in the literature.

Diagnosis

Willis stated that the diagnosis of dissecting aneurysm was not made often because of the infrequency of its occurrence, the variations in the clinical manifestations with the absence of a characteristic syndrome, and limited usefulness of special diagnostic adjuncts. It is our belief that the incidence of correct diagnosis can be and is being improved, and that from the study of large series of patients, such as the present group, the entity no longer appears bizarre, but falls into various syndromes which can often be readily recognized.

Of the 58 patients in this series who were seen from 1935 to 1947, the correct diagnosis was made in 16 (28.5 per cent). Obviously, this is not a startlingly high percentage of correct diagnosis. David, McPeak, Vivas-Salas, and White reported recently an incidence of correct diagnosis in 7 of 17 cases (41 per cent), these cases having been observed at the Massachusetts General Hospital from 1935 to 1946. Shannon was only able to report a correct antemortem diagnosis in 2 per cent of some 300 cases reviewed up to 1934. Emery recently has reported 24 cases from Australia, none of which were correctly diagnosed. Reich in a recent review from a large general hospital, reported 19 cases, only 2 of which had been correctly diagnosed antemortem.

In the present series, in cases studied between 1935 and 1940 the correct diagnosis was made in 18.7 per cent; in those studied between 1941 and 1947, a correct diagnosis was made in 30.9 per cent. Although this is not statistically significant it seems to indicate a trend of increasing accuracy in diagnosis with respect to dissecting aneurysm.

Summary of the Diagnostic Features of Dissecting Aneurysm. The various clinical features
of dissecting aneurysm have been described in some detail. These may be summarized as:

1. A previous history of hypertension is frequently elicited.

2. The onset is usually sudden and dramatic, with a history of severe, agonizing pain in the chest or abdomen, radiating most frequently to the back or down into the abdomen. Radiation to the extremities is not common, but of diagnostic value when present. Less often, the onset may be marked by syncope or unconsciousness.

3. Clinical shock, with either lowered or elevated blood pressure, is usually present at the onset.

4. Neurologic symptoms were present in 19 per cent of the patients, and varied from onset with syncope, dizziness, unconsciousness, or hemiplegia, all due to interruption of the cerebral blood supply, to transient weakness and anesthesia of one or more extremities due to ischemia of peripheral nerves or to interference with the blood supply to the spinal cord.

5. Aortic diastolic murmurs were present in 27.5 per cent of this series of patients. The appearance of a diastolic aortic murmur with aneurysmal dilatation of the ascending aorta, severe chest or abdominal pain, and negative serologic reactions is almost pathognomonic of dissecting aneurysm. Other investigators report the incidence of aortic diastolic murmurs as high as 56 per cent.

6. Inequalities of pulse and blood pressure recordings were found in 19 per cent of the patients.

7. Renal symptoms, such as oliguria, flank pain, and hematuria, when present, facilitate the diagnosis. They are usually due to dissection of the renal arteries, but may also be due to primary renal disease.

8. The most common electrocardiographic tracing encountered is that characteristic of left ventricular strain. In equivocal cases the absence of the pattern of myocardial infarction is in favor of the diagnosis of dissecting aneurysms in preference to that of coronary occlusion. The pattern of acute coronary occlusion does not rule out the possibility of dissecting aneurysm. The occurrence of varying patterns of myocardial ischemia, acute pericarditis, and uremia in association with dissecting aneurysm has been discussed. All the electrocardiograms (40 patients) showed abnormality. A normal pattern is unlikely in persons with dissecting aneurysm.

9. Chest x-ray examinations were performed on 20 patients. The value of the finding of a widened supracardiac shadow, progressive enlargement of the ascending or descending aorta, the double-barreled shadow, and similar changes has been discussed. Twenty per cent of the patients had left pleural effusions. Cardiac enlargement, which is left ventricular in type, is often present.

10. Other laboratory data, such as the findings of anemia, leucocytosis, hematuria, elevated icteric index, and elevated serum amylase, have value in ascertaining the correct diagnosis.

Prognosis

The duration of survival has already been discussed (table 2). In our series of 58 patients, 21 (36.2 per cent) died within forty-eight hours of the onset, 22 (35 per cent) survived three to sixty days, and 15 (25.9 per cent) survived three months to eight years. These statistics indicate a much better outlook than those usually given. Weiss, Kinney, and Maher felt that healing takes place in about 10 per cent of cases, whereas we found it in 25.9 per cent. Actually, if the work of Mote and Carr is considered, the actual number of patients with dissecting aneurysm dying within the first twenty-four to twenty-eight hours would be greatly increased and correspondingly the percentage surviving into the "healed" type decreased. Mote and Carr reported 60 deaths due to acute dissecting aneurysms recorded at the San Francisco Coroner's Office over a five-year period.

Treatment

The management of these patients in many ways is not unlike that of patients with coronary occlusion. Absolute bed rest with feeding of the patient should be enforced for a long period of time. The pain should be relieved by the liberal use of morphine, employing large doses if necessary. Oxygen should be used continuously for dyspnea and shock, or if signs of occlusion of vessels persists. In profound shock, transfusions of whole blood may be life-saving. Fluids should be administered slowly and the
diet should be light and bland, as in coronary occlusion. Regulation of diet and bowel movements should be such that the patient is spared undue muscular effort. Sedation, preferably that of chloral hydrate, should be used as indicated. The patient should not be subjected to repeated x-ray examination and fluoroscopy in an effort to find out what is going on in the mediastinum. A bedside plate is indicated. Operative re-entry in those cases which have resulted in occlusive disease of the iliacs has been reported by Gurin, Bulmer, and Derby. This may prove not only beneficial in relieving the local obstruction but may prolong life and result in a greater percentage of "chronic or healed" dissecting aneurysms. Anticoagulant therapy (such as heparin or dicumarol treatment) is definitely contraindicated.

**Summary**

The literature has been reviewed, and the findings in 58 cases of dissecting aneurysm from the Los Angeles County Hospital from 1935 to 1947 have been presented, with emphasis on distinguishing clinical features and an attempt at clinicopathologic correlation. Particular stress has been placed on the frequency of the aortic diastolic murmur and the mechanism of its production. Electrocardiograms were made in 40 of the patients, and these have been carefully reviewed and classified in an effort properly to evaluate the electrocardiogram in the diagnosis of dissecting aneurysm.

**REFERENCES**

4. **Vesalius:** Bonetus Sepulchri, Anatom., 1700, Bk. 4, Sect. 2, p. 209. (Cited by Shennan.)
6. **Scarpa:** Treatise on Aneurysm, 1804; translated by Wishart, 1808.
12. **Pennock, C. W.:** Case of anomalous aneurysm of the aorta resulting from ejection of blood composing the middle coat of that vessel. Am. J. M. Sc. 23: 13, 1838.
22. **Kilgoe, E. S.:** Dissecting aneurysm. California & Western Med. 53: 66, 1940.
40 Case Records of the Massachusetts General Hospital: Case 531931. 233: 386, 1945.
58 Burin, D., Bulmer, J. W., and Derby, R.: Diagnosis and operative relief of acute arterial obstruction due to this cause. New York State J. Med. 35: 1200, 1935.
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