Dissecting Aneurysm of the Aorta: Diagnosis, Treatment and Prognosis

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Dissecting Aneurysm of the Aorta: Diagnosis, Treatment and Prognosis

Hahn J. Lee, M.D.* and Ellet H. Drake, M.D.**

The records of 13 patients with proven dissecting aortic aneurysm were studied with particular attention to the diagnosis, treatment, and prognosis in each case. The three essential steps in reaching an early correct diagnosis included (1) history and physical examination, (2) serial chest x-rays and (3) aortography. Electrocardiogram and SGOT and LDH measurements seem to be of value only when incompatible with the diagnosis of acute myocardial infarction. Significant differences between Type 1 and Type 3 dissections were observed in their clinical manifestations, results of treatment and natural prognosis.

Dissecting aneurysm of the aorta is a serious condition that poses difficult problems in clinical diagnosis and management. According to the review by Hirst et al., 19 series of dissecting aneurysm of the aorta were reported from 1934 to 1954, Hirst's 339 cases being the largest. The frequency of correct antemortem diagnosis in 17 of those 19 ranged between zero and 47%. Among Hirst's 339 cases, 137 patients or 40% were correctly diagnosed before autopsy.

The treatment of this entity is still far from satisfactory. Numerous patients never reach the hospital, while others succumb in the course of diagnostic studies or sometimes during thoracotomy. Since the introduction of vigorous hypotensive treatment by Wheat and associates in 1965, there has been much controversy as to whether drug therapy or surgical intervention should be used for this disease. Because of the favorable reports and claims for both modes of therapy, a better understanding of the natural history of the disease may help in the selection of therapy. Both approaches are useful in selected patients.

This communication reviews experience at Henry Ford Hospital in 13 proven cases of dissecting thoracic aneurysm and points out problems confronting internists and surgeons in clinical diagnosis and management. All 13 patients were either Type 1 or 3 according to DeBakey's classification.9

Patients Studied

All records indexed at Henry Ford Hospital from January, 1964, to December, 1968, as dissecting aneurysm of the aorta were reviewed. Cases in which there was aortographic, surgical...
Lee and Drake

or autopsy proof of the diagnosis were accepted for the study group. Excluded were several patients with Marfan's syndrome or mediocystic necrosis in the ascending aorta who presented with chronic fusiform aneurysm and who were found at autopsy to have some dissecting process in the aorta. In addition, cases were not included when a chronic saccular or fusiform aneurysm, involving descending thoraco-abdominal aorta, was demonstrated either radiographically or at surgery and only a minor degree of dissection was found or suspected. The records of 13 patients which satisfied our criteria were studied in detail.

Results

Table 1 summarizes some of the clinical features observed in the 13 patients.

1. Extent of the dissecting process (DeBakey's classification): This was determined from the autopsy report, the operative note or the findings on arch aortography. In four patients, the dissecting process began in the ascending aorta and extended distally (Type 1), and in nine patients it extended in the aorta distal to the left subclavian artery (Type 3). In two of the nine patients with Type 3, the process also involved the ascending aorta by retrograde dissection, so that the ascending aorta was involved in six of the 13 cases. The retrograde dissection was confirmed at autopsy in one case and at surgery in the remaining one. Aortic dissection, end to end, was observed only in those four patients with Type 1 dissection.

2. Initial diagnostic impression on admission: Five of the 13 patients were correctly diagnosed as having dissecting aortic aneurysm when they were first seen by a physician in the emergency room or in the patient's room. Correct diagnosis was reached in all but one patient before discharge from the hospital or before autopsy. Acute myocardial infarction was the initial clinical impression in two patients, arterial hypertension in two, acute pancreatitis in one, spinal cord vascular insufficiency in one, cholecystitis in one and gastrointestinal carcinoma in one.

3. Clinical features. Age, sex and race: Age of the 13 patients ranged between 40 and 73 with a mean of 60 years. There were eight males and five females. Eleven patients were white and two patients black.

Hypertension: Blood pressure in these patients on the day of admission ranged between 90/64 and 300/175 mm Hg. Six patients had blood pressures ranging between 170/110 and 300/175 mm Hg, with mean systolic and diastolic pressure of 215 and 129. Five of the six patients had Type 3 dissection and one patient had Type 1.

Location of pain: Pain was the chief complaint in 12 patients but, in one patient, dyspnea was the only complaint. In 10 patients, the pain was located in or radiated to the interscapular area or back. It was retrosternal or in the anterior chest in nine patients, was epigastric or abdominal in four patients, in the legs or groins in three patients, and in the neck in one patient.

Aortic regurgitation murmur: A diastolic blowing murmur at the aortic area or down the left parasternal area, indicating aortic regurgitation, was present in five patients. Four of these patients were Type 1 and one was
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Type 3 with retrograde dissection.

Alteration in peripheral pulses: This was noted in four of the 13 patients while the other nine had normal peripheral pulses bilaterally.

Neurological findings: Nine of the 13 patients had no abnormal neurological findings. Four patients had mental confusion or severe agitation but no localizing neurological deficits. Only one patient had abnormal localizing findings consisting of paresis of the left superior rectus, bilateral triceps, right psoas iliacus, and the left leg, in addition to absence of deep tendon reflexes in the left leg and fasciculations in both lower extremities. At first, this particular patient was thought to have spinal cord vascular insufficiency, but dissecting thoracic aneurysm was found to be the cause of the myelopathy.

4. Laboratory tests. Chest x-ray: In 12 of the 13 patients, serial chest films suggested dissecting thoracic aneurysm. In four, the initial chest x-ray films gave no indication, but follow-up films showed change in size of the aorta. The chest x-ray findings in 12 patients consisted of widening and elongation of the aorta and disparity in size of the ascending and descending aorta, with prominence of the descending portion of the aorta in Type 3 dissection.

Electrocardiogram: ECG was within normal limits in two cases. Six patients showed non-specific ST-T or T wave changes, two patients LVH, one patient marked ST segment elevations but no abnormal Q waves, one patient complete right bundle branch block, and one patient complete heart block. In the case with marked ST segment elevations, diagnosis of acute myocardial infarction was incorrectly made in addition to dissecting aneurysm. The electrocardiogram is shown in Fig. 1. At autopsy a tear measuring 0.8 cm in length was found one cm above the aortic valve, but the coronary sinuses and ostia were intact, and the coronary arteries were patent throughout. The myocardium was normal under microscopic examination. The patient died 23 hours after the electrocardiogram was taken.

Serum enzymes: SGOT and LDH determinations were made for 11 patients during their hospital stay. In ten of these, values were normal for both enzymes while in one LDH was over 2,000 units. SGOT was not done in this latter case.

Arch aortogram: In 11 of the 13 cases, arch aortography was performed successfully and the findings confirmed the dissecting thoracic aneurysm. Two patients developed cardiac arrest during the procedure and died despite resuscitative measures. Another patient developed cardiac tamponade and severe hypotension shortly after the injection of contrast media into the main pulmonary artery. Immediately, pericardiocentesis was performed, and, after an operation three days later, the patient lived 38 months in good health, before dying of pneumonia.

5. Clinical findings related to types of dissection. Significant differences in some of the clinical findings were observed between Type 1 and Type 3 patients. Aortic regurgitation murmur was present in all four patients with Type 1 and in only one of the nine cases with Type 3. Five of the six patients who had blood pressure above 170/110 on admission, were Type 3 and the sixth was Type 1. In four of
<table>
<thead>
<tr>
<th>Cases</th>
<th>1</th>
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<th>3</th>
<th>4</th>
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<td>Yes</td>
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<td>Retrosternal</td>
<td>Left Chest Mid Back</td>
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<td>Epigastrium Mid Back</td>
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<td>90/64</td>
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<td>Present</td>
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<td>Dissecting Aneurysm</td>
<td>Acute MI</td>
<td>Cord Vascular Insufficiency</td>
<td>Dissecting Aneurysm</td>
<td>Acute Pancreatitis</td>
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<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
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<td>LVH</td>
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<td>Attempted</td>
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<td>Type 1</td>
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<td>No</td>
<td>No</td>
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<td>Period of Survival</td>
<td>23 hours</td>
<td>12 hours</td>
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<td>73 months</td>
<td>18 days</td>
<td>7 days</td>
<td>11 months</td>
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<td>Cause of Death</td>
<td>Cardiac Tamponade</td>
<td>Rupture into Atrium</td>
<td>Cardiac Tamponade</td>
<td>Massive Hemotherax</td>
<td>Renal Failure</td>
<td>Massive Hemotherax</td>
<td>No Autopsy</td>
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<td>9</td>
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<td>11</td>
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<td>56 WM</td>
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<td>60 NF</td>
<td>58 WM</td>
<td>65 WF</td>
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<td>Yes</td>
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<td>No</td>
<td>No</td>
<td>No</td>
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<td>Hypertension</td>
<td>Aortic Aneurysm</td>
<td>Cholelithiasis</td>
<td>Malignant Hypertension</td>
<td>G.I. Carcinoma</td>
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<td>Chest X-ray Suggestive</td>
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<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td>ECG</td>
<td>Normal</td>
<td>Nonspecific ST-T changes</td>
<td>Nonspecific ST-T changes</td>
<td>Nonspecific T changes</td>
<td>LVH</td>
<td>Normal</td>
<td></td>
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<tr>
<td>SGOT &amp; LDH</td>
<td>Not done</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
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<td>Aortogram</td>
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<td>No</td>
<td>No</td>
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<td>Period of Survival</td>
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<td>35 months</td>
<td>62 months</td>
<td>31 months</td>
<td>36 months</td>
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<td>Living</td>
<td>Living</td>
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<td>Living</td>
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</table>
the nine patients with Type 3, the only striking features were severe pain and severe hypertension. Three of the four cases with Type 1 were markedly leth-

Figure 1

An electrocardiogram of Case #1, taken on the day of admission, shows marked ST segment elevations in leads 2, 3, a VF and V1 through V3 with reciprocal changes in other leads, simulating acute myocardial infarction. No definite Q waves are seen.
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argic while one of the nine Type 3 cases had mental confusion. Two of the Type 1 cases showed evidence of occlusion of a major branch of the aortic arch, but none of the Type 3 cases had such occlusion.

6. Prognosis related to types of dissection, patency of renal arteries, the presence of re-entry tear and mode of treatment. Three of the four Type 1 patients died within 24 hours after admission. None of Type 3 patients died less than seven days after admission. Seven of the nine Type 3 patients survived from 11 to 62 months. Five of them are still living. Interesting autopsy findings were seen in one Type 1, who lived 73 months and later died of rupture of the aneurysm and massive hemothorax:

The entry tear was approximately 6 cm above the aortic valve and re-entry tears were present in both common iliac arteries. A completely duplicated or double-channeled aorta was present from the ascending aorta to the common iliacs, with one of the channels apparently supplying the kidneys. A completely endothelialized false lumen with atheromatous plaque formation was found. This particular patient was taking chlorothiazide 500 mg and methyldopa 2 grams daily during the 15 months before his sudden death. His blood pressure varied between 140/90 mm Hg and 180/110 mm Hg.

None of the other three cases with Type 1 was treated with antihypertensive agents during the acute stages. Seven of the nine Type 3 cases were long-term survivors. None of these was given vigorous hypotensive drug therapy either during the acute stages or later on.

Two patients with Type 3 (Case #10 and Case #12 in Table I) were found by aortography to have two aortic channels, one supplying the renal arteries. Both subjects are still living, 35 and 31 months later, respectively. Case #10 had blood pressure of 170/110 mm Hg on admission, which remained relatively stable during her 10 days in the hospital. No antihypertensive agent was administered, but she was given chlorthalidone, 100 mg daily, in the outpatient clinic; her blood pressure remained around 165/85 mm Hg.

Blood pressure of Case #12 on admission was 300/175. Reserpine, 2.5 mg, was administered intramuscularly and therapy with methyldopar 2 grams, apracoline 30 mg and hydrochlorothiazide 50 mg daily was initiated. Blood pressure decreased slowly, remaining around 180/100 mm Hg during the latter part of the 30-day hospital stay. Since discharge the patient received daily guanethidine 25 mg, methyldopa 2 grams, chlorothalidone 100 mg and triamterene 100 mg. His blood pressure has ranged between 140/70 mm Hg and 210/110 mm Hg, but he is otherwise in good health 31 months after the onset of the disease.

Two patients with Type 3, Case #8 and Case #9, had resection of the aneurysm and a prosthesis inserted while being maintained by the pump oxygenator. Blood pressure of Case #8 became normal following the operation and she is still living 60 months later. Blood pressure of Case #9 rose to as high as 200/150 mm Hg, and therapy with methyldopa 2 grams and chlorothalidone 100 mg daily was initiated on the twelfth hospital day. He underwent operation on the twenty-second day. The same medications were continued after discharge and the blood pressure became normal. He lived 38 months and when he died of pneumonia, autopsy showed an intact prosthesis.

Patient #5 with Type 3 had blood pressure of 90/60 mm Hg on admission and complete heart block for which a temporary pacemaker was inserted. Blood pressure readings reached 220/140 mm Hg during the 18-day hospital stay. No antihypertensive agent was given, although the patient received digitalis. Brain and kidney function gradually deteriorated and he died of renal failure. There was no evidence of coronary artery disease at autopsy.

Patient #6 with Type 3 had blood pressure of 200/95 mm Hg on admission, and the blood pressure rose to 250/140 mm Hg during his seven days in the hospital. Alser-
oxylon, 2 mg, and hydrochlorothiazide, 50 mg, were administered daily without effect on the blood pressure. He suddenly died of rupture of the aneurysm and massive hemothorax.

Patient #7, with Type 3, had normal blood pressure ranging between 120/60 mm Hg and 150/90 mm Hg during his nine-day hospitalization. This patient was given hydrochlorothiazide 100 mg and guanethidine 20 mg daily on the sixth hospital day. After discharge from the hospital, guanethidine 20 mg daily and reserpine 0.25 mg twice daily were continued. Blood pressure tended to rise slowly reaching 198/100 on several occasions. The patient had not returned to the clinic for eight months when he suddenly died at home, 11 months after discharge from the hospital.

Patient #13, with Type 3, had blood pressure of 178/110 mm Hg on admission, which gradually rose to 210/120 mm Hg during the 34-day hospital stay. Therapy with chlorothiazide 500 mg twice daily and reserpine 0.25 mg daily was started on the twenty-fourth hospital day and apresoline 25 mg t.i.d. was added on the twenty-ninth day. Blood pressure gradually decreased to around 140/90 mm Hg. The left kidney was shown to be non-functioning. After discharge, treatment was continued with chlorthalidone 100 mg daily and apresoline 25 mg t.i.d. Blood pressure has ranged around 160/90 mm Hg and the patient is in good health 36 months after dissection.

In patient #11 (Type 3), blood pressure was 240/140 mm Hg on admission, and remained around 170/100 mm Hg during the 20-day hospitalization. No antihypertensive agent was administered in the hospital and the patient refused surgery. After discharge, she was given hydrochlorothiazide 50 mg and reserpine 0.25 mg daily. Her blood pressure stayed around 170/100 mm Hg and she is living 62 months later.

Summary

The prognostically important findings described above are summarized below.

<table>
<thead>
<tr>
<th>Favorable Factors for Early and Late Prognosis</th>
<th>Cases Best Exemplified</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Type 3 dissection compared to Type 1</td>
<td>All 9 cases with Type 3 dissection</td>
</tr>
<tr>
<td>2. Intact renal blood supply in at least one kidney</td>
<td>Cases #10, 12 and 4</td>
</tr>
<tr>
<td>3. Presence of re-entry tear</td>
<td>Case #4</td>
</tr>
<tr>
<td>4. Surgical treatment especially for Type 3</td>
<td>Cases #8 and 9</td>
</tr>
<tr>
<td>5. Antihypertensive drug Rx especially for Type 3</td>
<td>Cases #7, 12, 4, 9 and 13</td>
</tr>
</tbody>
</table>

All eight long-term survivors in this series of 13 cases had two or more favorable factors as possible explanations. Patients #5 and #6 who died on the eighteenth and seventh hospital days respectively, had only one favorable factor, Type 3 dissection. In retrospect, both might have had a better opportunity for survival if vigorous hypotensive drug therapy with or without surgical intervention had been undertaken.

Those three patients with Type 1 dissection, who died in less than 24 hours after admission, had none of the favorable factors listed. We believe that neither surgery nor drug therapy could have saved them.

Discussion

Dissecting aneurysm of the aorta may simulate many other acute conditions involving the head, chest, abdomen and extremities, so that the differential diagnosis of this condition may be very difficult. The essential points in reaching an early correct diagnosis may be summarized as follows: (1) Careful history taking and physical examination with a high index of suspicion. This should include documenting the characteristic pain and correlating the physical findings with the various types of dissection; (2) serial chest x-rays; (3) aortography. Diagnostic procedures may need to be performed on an emergency basis, considering the serious nature of the condition.

Among the symptoms, pain is the
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most characteristic. Typically, it is sudden in onset, reaching its maximal intensity quickly. This severe pain has been variously described as ripping, tearing, choking, sharp, constricting, stabbing or burning. It frequently involves the interscapular area, and the migration or extension of pain from its initial location in the thorax to the head and neck, back, abdomen, groin or extremities is almost pathognomonic of dissection. The pain is often persistent, sometimes remaining until death, and may be relieved only temporarily by large doses of morphine. Persistent pain was the rule in those patients with a short survival.

Distinguishing the pain of dissecting aneurysm from myocardial infarction may be very difficult. In myocardial infarction, however, pain seldom reaches its maximal intensity at once, being usually dull, aching, oppressive or squeezing, and not knife-like or ripping in quality. It rarely extends to the back or lower extremities. Occasionally, patients with acute myocardial infarction and peripheral arterial occlusion from mural thrombi may complain of pains in the extremities, especially in the legs, closely simulating dissecting aneurysm. Most of these patients, however, experience a significant time delay between the first chest pain from myocardial infarction and the onset of pain in extremities. This is usually not true for dissecting aneurysm.

DeBakey and associates classified dissecting aortic aneurysm into three types based upon the origin and extent. Those of Type 1 begin in the ascending aorta and extend distally for a variable distance. Type 2 includes those cases where the dissection is confined to the ascending aorta. Cases of Type 2 usually include chronic dissecting aneurysms due to mediocystic degeneration, a pathogenetic process different from that characteristic of Type 1 and Type 3. Those cases in which the dissection begins distal to the left subclavian artery make up Type 3. This classification aids the clinician at the bedside in orienting himself anatomically and helps to explain some of the clinical findings. In addition, it is helpful in making decisions about how best to manage the acutely ill patient. The murmur of aortic regurgitation, evidence of involvement of arch vessels and alteration of consciousness may be observed in the majority of Type 1 cases. In contrast, these findings may be rather infrequent for patients with Type 3. Pulse pressure may be abnormally wide in cases with significant aortic insufficiency. The development of the murmur of aortic insufficiency, following an acute episode of chest or abdominal pain, is practically diagnostic of dissecting aneurysm. Many Type 1 cases have normal, low or even shock level blood pressures while severe hypotension may be the only objective finding in nearly half of Type 3 cases.

Serial chest films were suggestive of dissecting aneurysm in 12 of the 13 cases presented. Since the dissecting process may be rapidly progressing, chest films must be repeated frequently enough to detect the changing size of the aorta, especially in cases whose diagnosis is in doubt. Four of 12 cases had a normal-sized aorta shadow initially which became strongly suggestive of dissection on repeat films. McGeachy and Paulin reported an increase in the size of the aorta in
16 of 22 cases while Hirst\textsuperscript{1} described
an increase in the width of the supra-
cardiac shadow due to aortic or medi-
astinal enlargement in 121 of 339

cases.

Arch aortography is the most reli-
able radiologic technic available in
diagnosis of dissecting aneurysm. Two
patients in the present series developed
cardiac arrest and died during the
procedure and another developed card-
iac tamponade following the proce-
dure. The usual diagnostic finding on
arch aortogram is a narrowed central
opaque column representing the true
lumen in a widened less-dense aortic

shadow. Aortography usually makes it
possible to classify the dissection into
one of three types as described above.
This procedure probably should be
performed in every case suspected of
dissecting aneurysm in order to make
accurate diagnosis and to assist in
making proper plans for therapy. Some
of the obvious Type 1 cases which are
rapidly deteriorating clinically may
have a better chance for survival if
operation is performed immediately.

The principal value of the electro-
cardiogram has been to exclude myo-
cardial infarction. In 1934, White et
al\textsuperscript{8} called attention to the fact that
the absence of electrocardiographic
changes, supporting a diagnosis of
myocardial infarction during the first
few days following the onset of symp-
toms, should suggest the possibility of
dissecting aneurysm. However, pa-

tients with dissecting aneurysm may
occasionally show electrocardiographic
changes suggesting acute myocardial
ischemia or injury. Levinson et al\textsuperscript{9}
observed electrocardiographic changes
suggesting acute myocardial infarction,
acute coronary insufficiency and acute
pericarditis in 14 of 40 cases. They
also noted that at autopsy 20 of their
58 cases of dissecting aneurysm were
incorrectly diagnosed as cases of coro-
nary occlusion. The ECG changes, in
the absence of coronary occlusion,
have been attributed to reduction of
coronary blood flow because of shock,
anemia, coronary artery spasm, tachy-

cardia or direct compression on the
coronary ostia, sometimes associated
with retrograde dissection of supra-
valvular tears, and to pericarditis
from recent hemopericardium. Some
of these patients, however, do develop
transmural myocardial infarction sec-
dary to the dissecting process. Baer
and Goldburgh\textsuperscript{10} found patterns of
typical myocardial infarction in 3 of
23 cases. In these three, dissection of
the coronary arteries with resulting
myocardial infarction was found at
autopsy. Hirst\textsuperscript{1} described ECG pat-
terns of myocardial infarction in 18
of 173 cases. At autopsy, dissection
or compression of one or both coro-
nary arteries was noted in 12 of the
cases.

SGOT and LDH, done in 11 pa-
tients in this series, were normal in
10. These serum enzyme studies seem
to be helpful in differentiating dissec-
ting aneurysm from acute myocardial
infarction. Hirst failed to discuss en-
zyme studies in his extensive review\textsuperscript{1}
of dissecting aneurysm, and no study
evaluating these enzymes in diagnosis
of dissecting aneurysm has been pub-
lished to date.

Vigorous hypotensive drug therapy
in dissecting aneurysm has been ad-
voctated by Wheat et al.\textsuperscript{2} The chief
cause of death in dissecting aneurysm
is not related to the initial tear but to
further dissection and ultimate rup-
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ture. The forces favoring dissection are thought to be pulsatile flow and mean aortic pressure. If these forces can be reduced, static healed lesions may result. On the other hand, elective surgery of the chronic dissecting aneurysm may improve the rate of survival. Pulsatile flow is a function of the rate of ventricular fiber shortening and is considered to be more important in extending dissection than is mean aortic pressure. Aortas from diseased as well as normal individuals are amazingly resistant to merely static pressure increases.11

Trimethaphan, reserpine and guanethidine have been demonstrated to decrease the rate and amplitude of myocardial fiber shortening in addition to their hypotensive effect.2, 12

The method of drug therapy generally consists of (1) intravenous infusion of trimethaphan (1 to 2 mg/ml of 5% glucose in water) at a rate sufficient to lower systolic blood pressure below 120 mm Hg, (2) simultaneous reserpine in a dose of 1 to 2 mg given either intramuscularly or intravenously, and (3) guanethidine 50 mg by mouth twice a day started on the same day. Methyldopa may be added to this program. It should be pointed out that after 48 hours, the patient usually becomes refractory to trimethaphan and further effect is due to the other drugs.

In 1965, Palmer and Wheat reported the long survival of four of the six patients treated with drugs and, in 1967, successful drug therapy in 14 of 15 patients.13 Three of the 14 survivors in the latter series had to have elective surgery because of severe aortic insufficiency. One did not survive the operation. They cited 39 cases similarly treated from other hospitals and four failures, giving an overall success rate of 90%. Unfortunately, their reports failed to discuss the results of treatment according to types of dissection. Our series and others indicate that natural prognosis and results of either drug therapy or surgery is much better for Type 3 than Type 1 dissection. Accordingly, the more cases of Type 3 included in any series, the better the overall treatment results will be.

DeBakey et al reported in 1965 an overall mortality rate of 21% in 179 patients treated surgically. The operative mortality rate in 10 patients with acute Type 1 dissection was 40%, and in 48 patients with acute Type 3, 19%.

Austen et al in 1967, reporting their experience in 113 cases, listed eight patients with Type 1 and 23 with Type 3 undergoing surgical resections. Death occurred in three of the Type 1 cases (38%) and five of the Type 3 cases (22%). No late death occurred in their four-year followup. In contrast, 10 cases with acute dissecting aneurysm were treated with drugs consisting of trimethaphan, methyldopa and thiazides; six (60%) died in one year.

Contraindications to drug therapy include bleeding, severe hypotension or shock, severe heart failure and significant compromise of a major branch of the aorta. Palmer and Wheat considered the following criteria as indications for surgery: (1) failure of blood pressure to respond to drugs satisfactorily within four hours, (2) compromise of a major branch of the aorta, (3) failure to control progression of the dissection, (4)
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uncontrollable acute aortic insufficiency, (5) development of a saccular aneurysm, and (6) progressive enlargement of the dissecting aneurysm. From this measurement many Type 1 cases would represent contraindications to drug therapy and indications for surgery. Since most cases of Type 1 are likely to die within a few days,$^\text{15}$ immediate surgical intervention may well be justified. On the other hand, surgical intervention should be used more selectively for those cases of Type 3. Most of Type 3 cases present no contraindications to medical therapy. With the use of drugs, many patients can be safely carried into the subacute or chronic stage and elective surgery be performed with less risk, if indicated. Among the seven long term survivors in our series who were Type 3, five were treated with drugs on a long term basis and did not require surgery while two underwent surgery in the subacute stage and survived. The important factors influencing long term survival in the present series included type of dissection, treatment, status of renal blood supply and the presence of re-entry tear.

Three of our nine long term survivors had evidence of relatively intact blood supply in at least one kidney despite the fact that the dissecting process involved the abdominal aorta. In the autopsied case reported by Cassidy and Pinger,$^\text{16}$ a patient who survived nine years had the right renal artery arising from the aneurysmal sac, having become separated by dissection from the inner wall. Dissection of one or both renal arteries occurred in 63 of the 505 cases in Hirst's series.$^\text{1}$ The presence of re-entry tear was suggested by Shennan$^\text{17}$ as a factor influencing prognosis. He observed sites of re-entry into the true lumen present in 66 of 73 chronic dissections and in only 26 of 218 recent dissections. Hirst$^\text{4}$ observed that 50% of the 53 cases with re-entry survived six weeks or longer, more than triple the 16% of cases surviving at this period in the entire series of 339 cases.

Summary

The records of 13 patients with proven dissecting aortic aneurysm were studied with particular attention to the diagnosis, treatment, and prognosis in each case. Six of the 13 cases were correctly diagnosed as dissecting aneurysm when they were first seen by a physician at Henry Ford Hospital. The three essential steps in reaching an early correct diagnosis included (1) history and physical examination, with strong index of suspicion, including documenting the characteristic pain and correlating the physical findings with types of dissection, (2) serial chest x-rays and (3) aortography. Electrocardiogram and SGOT and LDH measurements seem to be of value in diagnosis only when they are incompatible with the diagnosis of acute myocardial infarction. Significant differences between Type 1 and Type 3 dissections were observed in their clinical manifestations, results of treatment and natural prognosis. The factors favoring a successful outcome include (1) Type 3 dissection compared to Type 1, (2) intact blood supply in at least one kidney, (3) antihypertensive drug therapy, especially for Type 3, (4) surgery, especially for Type 3 and (5) presence of re-entry tear. All eight long-term survivors had two or more
favorable factors, and the three patients who died in less than 24 hours after admission had none of the favorable factors listed here.

REFERENCES


