DISSECTING ANEURYSM of the aorta is a serious condition, which until recently has proved fatal in more than 75 per cent of cases. The disease was clearly recorded by Morgagni\(^5\) as early as 1761, and Laennec\(^6\) in 1819 designated it as "aneurysme dissequant." In spite of this early recognition and the subsequent increasing interest in the condition, the natural course of the disease remained unaltered until about 6 years ago, when methods of surgical treatment were devised.\(^7\)

Spontaneous healing of a dissecting aneurysm has been recognized for more than a century. Shekelton\(^8\) of Dublin, in 1822, and Henderson\(^9\) of Edinburgh, in 1843, reported cases in which the aneurysms ruptured into the lumen of the aorta or an iliac artery at some distal point. Endothelial growth at the sites of rupture was so complete in these "healed" cases that Hope\(^10\) referred to the double aorta as a congenital anomaly. Even though this method of nature to promote healing was apparently well recognized, it was not until 1935 that Gurin, Bulmer, and Derby\(^11\) utilized it to treat a patient with right iliac block produced by dissection. They were able to restore circulation to the extremity by creating a re-entry passage at the site of the iliac obstruction, but death resulted from renal failure. In 1948 Paullin and James\(^12\) attempted to strengthen the wall by wrapping cellophane about the dissected aorta, but the method proved unsatisfactory. Five years later, Johns\(^13\) reported suture repair of a rather unusual form of ruptured dissecting abdominal aneurysm, but the patient died of renal failure. In 1955 Shaw\(^14\) reported a typical case of dissecting aneurysm associated with acute arterial insufficiency of the lower extremities in which aortic obstruction was relieved by a procedure somewhat similar to that employed by Gurin and associates,\(^11\) consisting essentially of an abdominal aortic fenestration with repair of the dissection distally, but the patient also died from renal failure.

Since our first successful operation approximately 6 years ago, we have treated 72 patients with dissecting aneurysms of the aorta. Although the basic principles underlying the surgical procedures employed in these cases are essentially similar, certain variations have been utilized, depending upon the location and extent of the lesion. Some form of excisional therapy with replacement by homograft or prosthesis has been the most frequent procedure. This report is concerned with certain significant observations derived from analysis of this experience.
DISSECTING ANEURYSM OF AORTA

Incidence

Dissecting aneurysm of the aorta has been encountered in from 0.1 to 4.0 per cent of postmortem cases\(^2, 4, 15-20\) comprising from 12 to 25 per cent of all aneurysms of the aorta.\(^15, 16, 19, 21, 22\) Men are affected two or three times as often as women except in persons older than 80 years of age when this relationship is reversed.\(^2, 23\)

In our experience with 1,281 cases of aneurysm of the aorta treated surgically, dissecting aneurysm comprised 6.0 per cent of the total number (table 1) and 20 per cent of the thoracic aneurysms. Of these 72 patients 88 per cent were men, and approximately 90 per cent were in the fifth, sixth, or seventh decade of life (fig. 1). Seven patients were younger than 40 years of age, and two were older than 70. Six of the younger patients had definite characteristics of Marfan's disease. The age range was from 14 to 74 years, with an average age of 54 years.

Although no age group is exempt from this condition, the highest peak reported by most authors has been between the fourth and seventh decades. Commenting on dissecting aneurysms encountered in young persons, Schnitker and Bayer\(^24\) reported almost 25 per cent of 580 cases in patients younger than 40 years of age, and 38 per cent of Gore's\(^25\) cases were in this group. The high incidence of young individuals in these series is undoubtedly due to the selective nature of cases studied. A more likely incidence of 15 per cent of 505 cases has been reported by Hirst and co-workers.\(^23\)

Clinical Manifestations

Several recent reports have emphasized the characteristic clinical manifestations of this condition.\(^1-3, 26-28\) The most common reported symptom is sudden moderate to severe pain, which was initially present in 90 per cent of our patients. Pain was frequently the only symptom (table 2) and in many instances was minimal by the time the patient sought help. Usually substernal, the pain may be associated with pain in the back or epigastrium, occasionally extending to the neck, shoulders, or legs. Substernal or epigastric pain was the

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**Table 1**

Incidence of Dissecting Aneurysms among All Surgically Treated Aortic Aneurysms

<table>
<thead>
<tr>
<th>Location</th>
<th>No. cases</th>
<th>Per cent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic arch</td>
<td>60</td>
<td>5</td>
</tr>
<tr>
<td>Descending thoracic</td>
<td>134</td>
<td>10</td>
</tr>
<tr>
<td>Dissecting aneurysm</td>
<td>72</td>
<td>6</td>
</tr>
<tr>
<td>Thoracoabdominal</td>
<td>29</td>
<td>2</td>
</tr>
<tr>
<td>Abdominal</td>
<td>986</td>
<td>77</td>
</tr>
<tr>
<td>Total</td>
<td>1281</td>
<td>100</td>
</tr>
</tbody>
</table>

**Table 2**

Dissecting Aneurysm of the Aorta. Relative Frequency of Symptoms—72 Cases

<table>
<thead>
<tr>
<th>Symptom</th>
<th>No. of patients</th>
<th>Symptom</th>
<th>No. of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Substernal or</td>
<td></td>
<td>Neurologic manifestations</td>
<td>7</td>
</tr>
<tr>
<td>precardial pain</td>
<td>48</td>
<td>Only symptom</td>
<td>6</td>
</tr>
<tr>
<td>Only symptom</td>
<td>6</td>
<td>Hemoptysis</td>
<td>2</td>
</tr>
<tr>
<td>Back pain</td>
<td>35</td>
<td>Only symptom</td>
<td>1</td>
</tr>
<tr>
<td>Only symptom</td>
<td>1</td>
<td>Impaired leg circulation</td>
<td>2</td>
</tr>
<tr>
<td>Epigastric or</td>
<td></td>
<td>Hematuria</td>
<td>1</td>
</tr>
<tr>
<td>abdominal pain</td>
<td>20</td>
<td>Only symptom</td>
<td>4</td>
</tr>
<tr>
<td>Only symptom</td>
<td>4</td>
<td>Shock</td>
<td>1</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>11</td>
<td>Asymptomatic</td>
<td>2</td>
</tr>
</tbody>
</table>
Serial roentgenograms of the chest demonstrating progressive enlargement during a 3-year period of dissecting aneurysm of the aorta in a 50-year-old physician.

Circulation, Volume XXIV, August 1961
only symptom in 10 patients. Three of these had been treated for myocardial infarction before the correct diagnosis was obvious. The possibility of a dissecting aneurysm should always be considered in patients being treated for coronary occlusion. During the past 5 years studies of postmortem cases at the Methodist Hospital revealed that three individuals thought to have had myocardial infarction had died suddenly from rupture of a dissecting aneurysm.

Initial shock, reported in about one third of cases, is usually out of proportion to the drop in systolic blood pressure. It may be associated with cyanosis, tachypnea, and tachycardia.

The foregoing are usually the manifestations of acute dissection. Only about one fourth of our patients were treated during this early phase of the dissecting process, i.e., within the first few days to a week after onset of symptoms. The interval between onset of symptoms and operation in the remaining cases ranged from several weeks to 2 years.

The reported incidence of neurologic manifestations ranges from 15 per cent to 46 per cent of cases. Three of our patients had suffered from transient paraplegia, two complained of numbness of arm or leg, and two were unresponsive or aphonic for several hours. Neurologic signs or symptoms in patients with thoracic or abdominal pain may be a clue to the early diagnosis of dissecting aneurysm. Certainly neurologic deficits may be easily overlooked in the usual examination of a critically ill patient.

Shortness of breath was a complaint of 15 per cent of the patients. Two patients had transient circulatory disturbance of the legs, and hematuria occurred in one instance.

Only two patients were asymptomatic. In one the lesion was discovered by routine roentgenography of the chest and in the other it was discovered incidentally during pneumonectomy.

Two thirds of the patients had associated cardiovascular or other disease. Of these, moderate to severe hypertension of several years' duration was by far the most frequent, being present in 74 per cent of cases. Other complicating conditions included congestive heart failure, arteriosclerotic heart disease, active duodenal ulcer, portal cirrhosis, and gallbladder disease.

The physical manifestations of dissecting aneurysm were not significant in making a
diagnosis. A precordial apical or basal systolic murmur was present in 40 per cent of cases. A diastolic murmur over the aortic area, which has been emphasized as of diagnostic significance, was present in only eight cases in our series. This probably reflects the small number of patients in our series with involvement of the ascending aorta or aortic annulus. Brachial blood pressure differential, tracheal deviation, cervical venous distention, abdominal aneurysm, and alteration in peripheral pulses were occasional findings. Fifteen per cent of patients had minimally elevated blood urea nitrogen levels. Myocardial ischemia, left ventricular strain, or rhythm disturbance was demonstrable elec-

Table 3

Dissecting Aneurysm of the Aorta. Relationship of Extent of Dissection to Operative Mortality

<table>
<thead>
<tr>
<th>AORTIC SEGMENT INVOLVED</th>
<th>Combined Series of Hirst and Shennan</th>
<th>PRESENT SERIES</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cases</td>
<td>Per Cent</td>
</tr>
<tr>
<td>Ascending</td>
<td>342</td>
<td>47</td>
</tr>
<tr>
<td>Arch</td>
<td>212</td>
<td>29</td>
</tr>
<tr>
<td>Descending</td>
<td>115</td>
<td>16</td>
</tr>
<tr>
<td>Abdominal</td>
<td>54</td>
<td>8</td>
</tr>
<tr>
<td>TOTAL</td>
<td>723</td>
<td>100</td>
</tr>
</tbody>
</table>
trocadiographically in 75 per cent of the cases.

Roentgenograms of the chest usually revealed widening of the supracardiac mediastinum and radiolucency of the arch and descending aorta in the region of the false passage. In some instances superior mediastinal widening may be initially insignificant. Progressive enlargement of the aneurysm during a period of several days or months may be observed (figs. 2 A-J). In such instances the risk of death by sudden rupture of the aneurysm is definitely increased. Angioaortogram made with the patient in an oblique position accentuates the "double-barrelled" appearance of the lesion (fig. 3). Angioaortography has been of utmost value in determining the nature and extent of the dissecting process. Mediastinal motion may occasionally produce a similar appearance, as will an organized thrombus lining a long, fusiform arteriosclerotic aneurysm. Calcification of the intima and wall of the false passage producing a double lumen appearance on plain roentgenography of the chest is rarely seen.

Table 4
Dissecting Aneurysms of Thoracic Aorta. Mortality According to Blood Pressure

<table>
<thead>
<tr>
<th>Blood pressure</th>
<th>No. cases</th>
<th>No. cases</th>
<th>Per cent</th>
</tr>
</thead>
<tbody>
<tr>
<td>149/89 or less</td>
<td>19</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>150/90 or more</td>
<td>53</td>
<td>19</td>
<td>36</td>
</tr>
<tr>
<td>Total</td>
<td>72</td>
<td>19</td>
<td>26</td>
</tr>
</tbody>
</table>

Pathogenesis and Gross Pathology

The basic lesion of dissecting aneurysm appears to be degeneration of the supporting tissue of the media of the aorta and is apparently unrelated to known pathologic processes, such as arteriosclerosis or aortitis, that commonly involve the aorta.16, 34, 35 The condition is frequently associated with Marfan's syndrome and hypertension. Medial degeneration with dissection of the aortic wall has been produced experimentally by a diet of lathyrus odoratus (sweet pea) meal,36, 37 prolonged injection of epinephrine,38 vitamin E deficiency,39 methonium intoxication,40, 41 and other means. Many investigators6, 11, 16, 42-53 believe that the dissecting process is initiated by intimal laceration, whereas others3, 20, 34, 54-59 are of the opinion that dissection follows rupture of a vasa vasorum, the break in the intima occurring secondarily. This is a more plausible explanation for cases of dissecting aneurysm that develop without intimal tear. An intriguing concept of pathogenesis has been offered by Bauer and Hirsch,60 who stated that repeated expansion and contraction of an aortic wall in which there is considerable difference in elasticity of the media and adventitia would result in easy separation of the two layers. Because of unequal elasticity, the shearing force in the wall of the aorta at points of greatest mechanical stress produce dissection without intimal laceration. This incorporates all theories of pathogenesis and explains established observations. It is well known that in the presence of medial degeneration the layers of the aorta can be easily separated.42 This plane of dissection usually lies between the outer two thirds and inner third of the aortic wall (figs. 4A and 4B). The role of external trauma in the pathogenesis of dissecting aneurysm is uncertain.

Of greater practical importance are the location and extent of the disease. Shennan,4 Hirst and co-workers23 divided their cases into four major and 18 minor categories on an anatomic basis. Comparison of our series with theirs indicates the more selective na-
ture of our cases by the greater incidence of the chronic type of lesion (table 3). On the basis of our surgical experience we have classified these lesions into four general categories, since this provides a guide to surgical approach and prognosis (fig. 5).

Type I (12 per cent) includes patients in whom the dissecting process extends distally from the aortic annulus or the aortic arch usually to a point well below the diaphragm (fig. 6). The dissection may involve the carotid, iliac, renal, or mesenteric arteries. Unless there is a localized area where rupture is imminent, resection with graft replacement is of little value. Creation of a re-entry passage is usually preferable and may be done with or without the aid of hypothermia or atriofemoral bypass perfusion.

In type II (6 per cent) the process is localized to the ascending aorta and proximal transverse arch (figs. 7A and 7B). Operative correction requires utilization of the extracorporeal pump-oxygenator and coronary perfusion to permit excision of the lesion and aortic replacement by graft. In some cases because of dilatation of the aortic annulus and consequent aortic insufficiency, it is necessary to perform an annuloplasty either by wedge resection and suture repair with bicuspidization of the aortic valves or by circumferential suture annuloplasty with graft replacement.

In type III (60 per cent) the dissecting process begins immediately distal to the left subclavian artery and continues well below the diaphragm (figs. 2, 3, and 4). A distal false lumen must be dealt with surgically after the thoracic portion has been excised.

Type IV (22 per cent) is similar to type III except that the dissecting process remains localized to the descending aorta (fig. 8). The entire diseased segment may be excised and replaced by graft.
DISSECTING ANEURYSM OF AORTA

Operative correction of the last three types requires use of hypothermia or, preferably, the atriofemoral pump bypass to prevent spinal cord ischemia.

Results of Surgical Treatment

Depending on the nature of the lesion three general types of surgical procedure were employed in this series of 72 patients. In only one patient was the dissection so well localized that excision with aneurysmorrhaphy was possible. In five of the earlier patients a re-entry passage was created into the true lumen of the descending thoracic aorta and the distal false lumen was obliterated by suture. The remaining 66 patients had some form of excisional therapy with aortic replacement with the use of left atriofemoral bypass perfusion, external bypass graft, or hypothermia to prevent the cardiac, neurologic, or renal disturbances that follow prolonged occlusion of the descending thoracic aorta. Special technical problems arose in the five patients with involvement of the entire aorta, the one patient who required temporary carotid perfusion, and the six in whom the true lumen was not apparent. The usual procedure was resection of the descending thoracic aorta so as to include the site of origin of the dissection, obliteration of the false lumen distally, and insertion of a homograft or prosthesis (44 patients) (fig. 9). In 16 patients the entire pathologic process could be excised and replaced by a vascular graft or prosthesis. Of the 59 patients with types III and IV in whom the dissecting process involved the descending aorta, the operative mortality rate was 20 per cent as compared with 26 per cent for the entire group (table 3).

Various materials were used to replace the excised aortic segments. These included aortic homografts in 13 and such synthetic prostheses as Ivalon in four, Nylon-Dacron in six, Daeron in 24, Dacron-Ivalon in 1, and Teflon in 18 cases. Although each prosthesis has its own special characteristics, closely woven fabrics have proved most satisfactory for aortic replacement in patients requiring heparinization. Only one death was attributable to failure of the graft; this death occurred 2 years after operation from rupture of a dissecting aneurysm of the homograft. Homografts have functioned satisfactorily in the thoracic aorta for more than 7 years after insertion. Our first synthetic prosthesis in this series has functioned satisfactorily for more than 4 years.

Under hypothermic conditions the diseased segment in 11 patients was resected and the

Figure 5
Surgical classification of dissecting aneurysms of the aorta based on location and extent of lesion.

Figure 6
Angioaortogram and drawing demonstrating appearance after creation of re-entry site in descending thoracic aorta. Patient remained active 4½ years after operation with no evidence of progression of lesion.
defect replaced by a graft. The duration of aortic occlusion ranged from 19 to 87 minutes. Permanent paraplegia developed in only one patient, cooled to 90 F. with interruption of aortic flow for 63 minutes.

With use of left atriofemoral bypass perfusion, 42 lesions were resected with aortic replacement (fig. 9). The duration of occlusion ranged from 28 to 102 minutes, averaging 49 minutes. Two patients were paraplegic immediately after operation; however, one whose occlusion time was 42 minutes completely recovered after 3 weeks. The other, whose occlusion time was 102 minutes, had permanent neurologic changes.

In two instances localized difficulties caused grafts to be inserted without benefit of bypass or hypothermia. Transitory paresthesias developed in the legs of one of these after 34 minutes of aortic occlusion, whereas the other after 37 minutes of occlusion, had no complication.

In five re-entry procedures no sequelae occurred that could be attributed to temporary occlusion of the descending aorta during the anastomosis, which averaged less than 30 minutes. Interruption of blood flow in the aorta at the midportion of the descending thoracic aorta for such a short period carries a low risk of neurologic damage.

Three patients had a permanent bypass and obliterative endoaneurysmorrhaphy. One patient with a large tortuous dissecting aneurysm extending from the left subclavian artery to the midportion of the abdominal aorta had a permanent external Dacron by-pass from the distal aortic arch to the lower abdominal aorta (fig. 10). After the bypass was functioning satisfactorily, the entire descending thoracic aorta was excised and the two lumens were oversewn at the diaphragm. This patient did well until massive upper gastrointestinal hemorrhage from acute gastric ulcerations required emergency gastrectomy on the eighth postoperative day. He died several days later from myocardial infarction. A second patient died from renal failure following a transfusion reaction 9 days after operation, and a third patient is living and well after obliteration of the aneurysm, which extended from the left subclavian to the celiac artery.

A brief analysis of the 19 operative deaths reveals an average postoperative survival time of 10 days. Six patients died of rupture of the aneurysm, three rupturing into the pericardial sac producing tamponade. Four patients died in congestive heart failure, and three died of renal insufficiency. In three instances cardiac arrest occurred at operation or immediately thereafter. The remaining three patients died of myocardial infarction, pulmonary embolism, and cerebral infarction, respectively.

Three of the 11 patients who had resection
under hypothermic conditions died (27 per cent). Eleven deaths occurred in the bypass perfusion group (23 per cent), and three of four patients who had creation of a re-entry passage died. Advanced age per se did not alter the prognosis. Hypertension appears to be the most significant factor in relation to operative risk as there were no deaths in the normotensive group (table 4).

With few exceptions the results in the 53 patients surviving operation, in all of whom follow-up studies have been made, have been gratifying. Follow-up observations for more than 5 years reveal that most of these patients have resumed normal activities. Many are retired but active, but others engage in hard manual labor. One patient is a semi-invalid, and two are paraplegic. Five patients have subsequently had aneurysms of the abdominal aorta resected successfully with graft replacement (fig. 11).

Only six patients have died since discharge from the hospital. In one fulminating hepatitis developed, presumably from blood transfusions, and the patient died 1 month after operation. Two succumbed to intracranial hemorrhage or hypertensive cardiovascular disease 10 months and 12 months after operation, respectively. One man committed suicide 1 year later. Two patients died of internal hemorrhage after 2 years, one from rupture of an aneurysm of the innominate artery and the other from a rupture of a dissecting aneurysm of the homograft.
Follow-up roentgenograms of the chest and angiocardiograms have failed to demonstrate progression of the disease or unsatisfactory appearance of the homografts or prostheses for periods up to 5 years. One physician, alive more than 5 years after creation of a re-entry passage, has had no further progression of the lesion (fig. 6).

Comment

Until recently, treatment of dissecting aneurysms of the aorta was largely symptomatic and had little or no effect upon the highly fatal course of the disease. This is well illustrated by the reports of a number of observers. In the classic study of Shennan,4 for example, 65 per cent of the patients died within 24 hours after onset, and an additional 26 per cent died within 1 day to 1 week. More recently, Hirst and associates23 in a study of the survival period of 425 patients found that 74 per cent died within 2 weeks after onset and 89 per cent within 3 months. Other investigators have had somewhat similar experience. This indicates that in general only about 10 to 15 per cent of patients have the chronic "healed" type of dissection. Even in this group, however, further progression of the dissecting aneurysm frequently takes place and ultimately leads to fatal termination.

In light of these observations emphasizing the extremely grave nature of this disease, more aggressive therapy directed toward altering the natural highly fatal course of the disease is definitely indicated. That this can be done by application of surgical treatment is evidenced by comparing the significantly greater survival rate in our operative series.
with that of Hirst and associates in their non-operative series (fig. 12).

Many variations exist in the pathologic features of the disease, particularly in regard to location and extent of the dissecting process. Since the surgical approach to these various types of lesions may require certain modifications, it is essential to determine preoperatively as precisely as possible the origin, location, and extent of the dissecting process. In most instances this can be done by special angiographic studies. Our experience would suggest that the most favorable lesions for operative treatment are those in which the dissecting process originates in the descending thoracic aorta distal to the left subclavian artery. Fortunately these are the types of lesions that tend to occur in patients who survive the initial episode for more than a few days and thus permit sufficient time for application of surgical treatment.

Summary

Dissecting aneurysm of the aorta is an extremely serious condition that pursues a rapidly fatal course in more than 75 per cent of cases. Follow-up data on 72 patients surgi-
cally treated indicate that effective surgical treatment significantly alters the course of the disease and in the majority of instances removes the threat of death by rupture. The operative mortality rate in these 72 patients was 26 per cent. For lesions occurring distal to the left subclavian artery, resection of the descending thoracic aorta with replacement by aortic graft utilizing hypothermia or the bypass pump was successful in 80 per cent of cases.

Most patients surviving operation have been able to resume previous activities with minimal risk of sudden death from rupture of the aneurysm or failure of the aortic graft.

The importance of recognizing the characteristic clinical manifestations, of making precise roentgenographic diagnosis, and of instituting effective surgical treatment in the management of dissecting aortic aneurysm has been stressed.

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DISSECTING ANEURYSM OF AORTA

Surgical Treatment of Dissecting Aneurysm of the Aorta Analysis of Seventy-Two Cases
MICHAEL E. DEBAKEY, WALTER S. HENLY, DENTON A. COOLEY, E. STANLEY CRAWFORD and GEORGE C. MORRIS, JR.

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