Problems in the Surgical Management of Acute Dissecting Aneurysm of the Aorta *

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Introduction

Dissecting aneurysm of the aorta has been recognized as a distinct entity for over 150 years.[13] Prior to that time, there were glimpses into its true nature but Maunoir, in 1802, was the first to publish a clear description of the disease. As early as 1822 there was a report of a "cured" case by Shekelton of Dublin, at which time he described lucidly the distal opening through the intimal wall of the sac and pointed out the significance of the reopening from the aneurysm into the aorta. This was amply confirmed by Henderson of Edinburgh in 1843. In 1826 the famous French physician, René Laennec, first used the term "aneurysme disséquant" and this name has been generally accepted since that time.

It was about a half a century from Maunoir's important publication until the earliest report of a correct clinical diagnosis made by Swaine *et al.* in 1855. This was a case with three months survival and the diagnosis was confirmed at autopsy. It is both interesting and pertinent to note here that in the next 78 years (until Shennan's very comprehensive monograph in 1933) there were only five additional cases in the world's literature with diagnosis during life. It is not surprising, therefore, that little thought was devoted to the possibility of surgical therapy during that time.

Review of Surgical Treatment of Dissecting Aneurysm. Although the necessary prerequisites for surgical approach to this vascular accident had been available for many years, it was not until 1935 that Gurin, Bulmer and Derby[7] reported the first attempt at a definitive procedure. Their patient was a 43-year-old man who presented with excruciating pain in the epigastrium with extension downward to the right thigh. The right leg was cold, had a weak femoral pulse, and had markedly diminished sensory and motor function. A diagnosis of dissecting aneurysm of the aorta with obstruction to the right iliac artery was made. Operation was decided upon to prevent gangrene of the affected leg. The initial step was to explore the femoral artery to confirm the diagnosis of obstruction. This was done, the abdomen was then entered, and the right iliac artery was dissected out. The aneurysm was quite apparent and the iliac artery was opened longitudinally through a portion of the vessel seemingly unaffected by the dissection. The occlusion of the lumen was found to be due to elevation of the intimal wall of the aneurysm opposite a large atheromatous mass. An incision was made through the internal lamina of the sac with release of dark, unclotted blood. A clamp was inserted through the intimal incision and gentle dissection was done to free up any of the adherent clot. Upon releasing the clamps, brisk bleeding occurred and following closure, the vessel pulsated normally for the first time. The arteriotomy

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was closed with interrupted silk sutures but the incision into the aneurysmal sac through the intimal layer was left open in the hope that complete rechanneling would occur. Postoperatively, there were palpable pedal pulses and there was no evidence of circulatory disturbance in the leg. The patient died on the sixth postoperative day with rising blood urea, cough, bloody purulent sputum, high fever, and with patchy pneumonitis demonstrated on the chest x-ray. Unfortunately, an autopsy could not be obtained.

Following this unsuccessful attempt at operative intervention, the next procedure was not reported until 1953. At that time Johns [8] reported a case of dissecting aneurysm of the abdominal aorta with perforation causing a large retroperitoneal hematoma. Although the nature of the primary lesion was not recognized at operation the bleeding was controlled by closure of the defect with a continuous silk suture. The patient succumbed on the eighth postoperative day of acute renal failure.

Recently, Shaw [12] has reported a case with the finding of acute arterial obstruction at the level of the iliac arteries. At operation the aneurysmal sac was found to be filled with fresh thrombus. This was extruded manually and the obstruction was thought to be relieved. A segment of the internal lamina was then excised to allow the blood in the aneurysmal space to return to the true aortic lumen and the opening into the distal aneurysmal space was closed with sutures. The patient subsequently had to be re-operated upon, however, because of arterial obstruction in the right popliteal and posterior tibial arteries from fresh thrombi. Following the second operative procedure the leg remained viable, but the patient died on the seventh postoperative day of acute renal failure.

The chief stimulus for the growing interest in the surgical management of dissecting aneurysms has come from the work of DeBakey, Cooley, and Creech.[3] Whereas there had been three scattered case reports prior to their initial publication, these authors presented a series of six well-documented cases. In this group were the first successful operative cases, with four of the six patients surviving. Their fundamental operative principles included the creation of a distal internal opening as a re-entry site and closure of the distal aneurysmal space. The actual operative procedures performed were varied and among them were included the local excision and closure of a saccular dissection and the resection of the descending thoracic aorta with replacement by a homograph. The procedure which probably has proved to be the most useful consists of division of the descending thoracic aorta, creation of the re-entry site through the intimal lamina, closure of the distal aneurysmal opening and anastomosis of the ends of the aorta. These authors also stressed for the first time the importance of a trans-thoracic approach rather than the abdominal.

In a subsequent paper DeBakey [4] reported an additional four patients, making a total of ten cases. In this group there were seven survivors, a remarkable record. Several of these patients had the so-called chronic dissection with a survival of several weeks or months. At least two of them, however, were operated on during the first month. The first case underwent surgical exploration on the second day of disease with a sudden fatal ending on the eighth postoperative day from intrapericardial rupture of the aneurysm. The other, operated on after a period of four weeks, had an uneventful recovery and is alive and well. Regarding the handling of these acute cases, DeBakey *et al.*[3] expressed the belief that operative intervention was indicated at the earliest opportunity.

REPORT OF CASES

Case 1. A 49-year-old white man was admitted August 19, 1955, one hour following onset

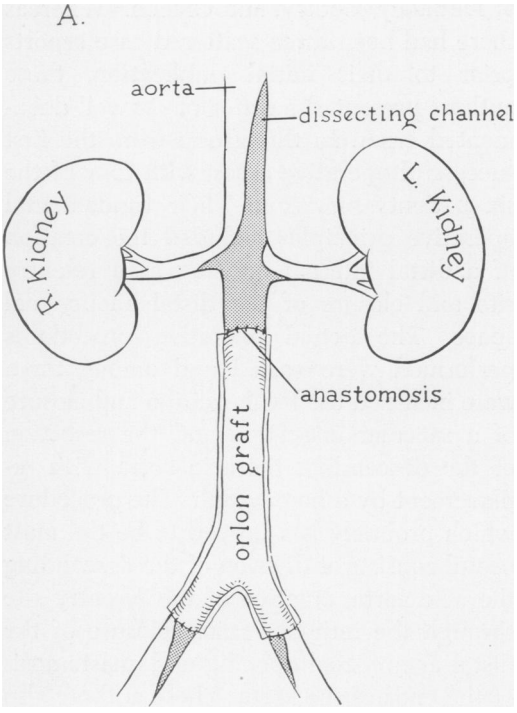

FIG. 1 A. Diagram of the completed operation.

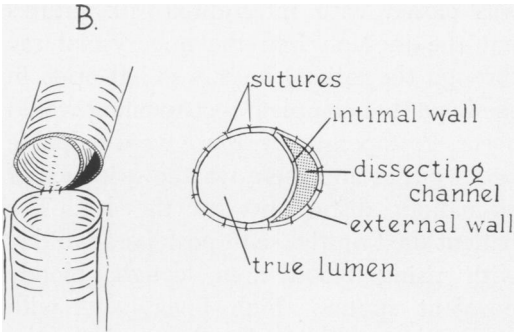

FIG. 1 B. Illustration of the method of anastomosis to maintain the distal re-entry site. The internal wall of the sac is trimmed back and not included in the suture line. This forms the opening through which the blood in the aneurysmal space flows back into the true aortic lumen.

of excruciating substernal pain with radiation between the scapulae. The pain subsequently extended down into the low back and hips and was accompanied by extreme weakness and loss of sensation in the legs.

The patient had been treated for severe hypertension for 3 years, having suffered a cerebrovascular accident and an episode of cardiac failure. Six weeks prior to the present admission he had been found to have a blood pressure of 210/145 mm. Hg, far advanced retinopathy, cardiac hypertrophy and hepatomegaly. Laboratory studies revealed a 3+ albuminuria, blood urea of 60 mg. per cent, and decreased PSP excretion. While in the hospital the patient developed a large hematoma medial to the right scapula, felt to be due to the rupture of an intercostal artery. His response to several anti-hypertensive drugs was not good and he was discharged essentially unimproved, continuing to show an elevated blood urea (72 mgm. per cent).

On admission the patient was cold and clammy and complained bitterly of agonizing pain in the low back and legs. The blood pressure was 300+/150 mm. Hg in each arm. The heart was questionably enlarged and there were no murmurs. The legs were paralyzed and exhibited marked hypesthesia. The abdominal aorta was easily palpated but there were no pulses in either femoral region. An admitting diagnosis of acute dissecting aneurysm of the aorta with bilateral iliac obstruction was made.

In view of the loss of circulation to the lower extremities, it was felt that surgical intervention was mandatory. An exploratory laparotomy was begun 3 hours following the onset of symptoms, revealing a dissecting aneurysm of the aorta extending into the chest proximally and into the external iliac arteries distally. There was hemorrhagic extravasation around the right iliac artery but no perforation was identified. Attempts to reestablish the circulation to the legs were carried out from the aorta above and through bilateral femoral arteriotomies below. These procedures were unsuccessful and resection of the aortic bifurcation and proximal iliac arteries was then carried out. An Orlon prosthesis was used to replace the resected vessels and a routine anastomosis was performed following the excision of a window of intima from the proximal aorta to provide a re-entry site for the aneurysmal channel into the true lumen (Fig. 1, A and B). Following removal of the occlusive clamps the blood pressure fell precipitously and was unobtainable for a short time. Response to transfusions and intravenous norephinephrine was rapid and a bilateral lumbar sympathectomy was then performed.

The postoperative course was characterized by oliguria, a rising blood urea and an increase in restlessness and disorientation. The return of function to the legs was complete, accompanied by excellent pedal pulses bilaterally. The urinary output increased from a total of 18 ml. on the first postoperative day to 360 ml. on the sixth postoperative day. However, the blood urea had

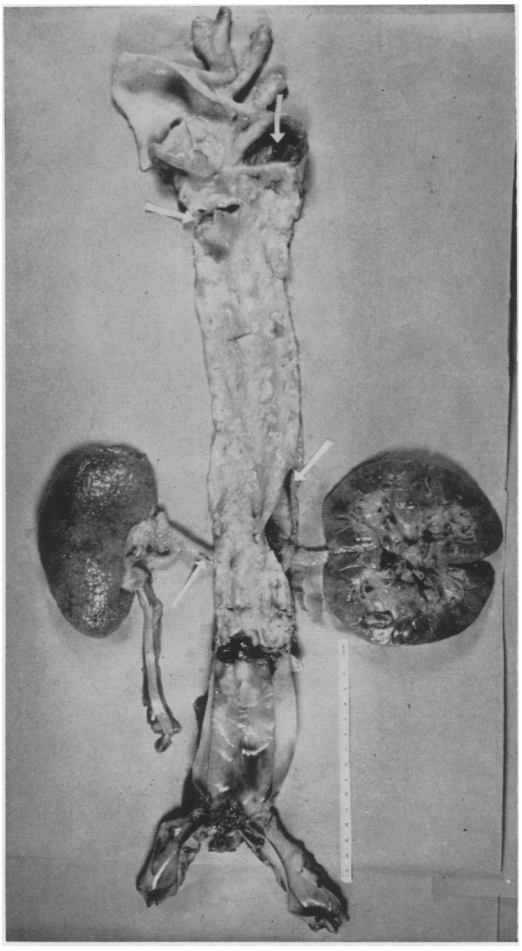

FIG. 2. Photograph of the opened aorta at autopsy. Note, (1) False aneurysm just below the subclavian artery, (2) transverse tear of proximal internal opening, (3) minor involvement of renal arteries, (4) orlon prosthesis with surrounding fibrous sheath, and small thrombus at aortic suture line.

steadily risen to 250 mg. per cent and he died on the sixth postoperative day.

Postmortem examination disclosed that the dissecting aneurysm began at the descending limb of the arch of the aorta (Fig. 2). An additional finding of interest was a separate saccular aneurysm in the transverse arch of the aorta which had apparently been associated with the large hematoma seen on previous admission. There was no renal infarction and the histologic changes in the kidneys were those of extensive arteriolar nephrosclerosis, with no evidence of acute tubular degeneration (so-called lower nephron nephrosis). There was marked cardiomegaly with edema and congestion of the lungs. There was no evidence of bleeding from the aneurysm or

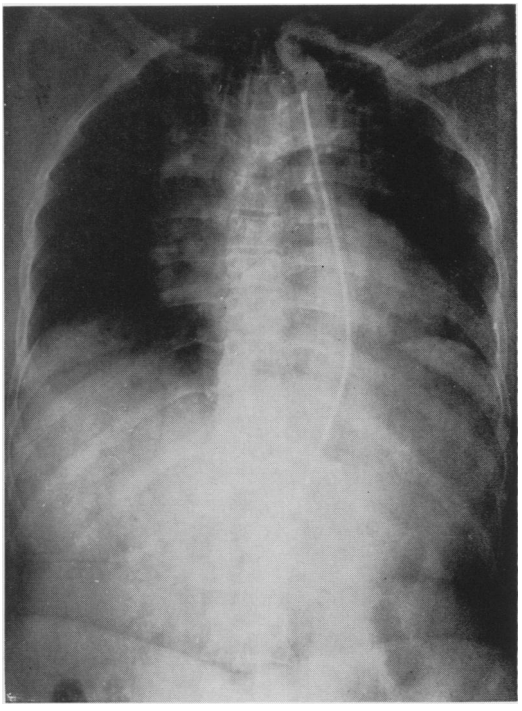

FIG. 3. Photograph of the retrograde aortogram in Case 2, demonstrating the widened superior mediastinum and the double channel in the descending aorta.

continuation of the dissection in the postoperative period. The Orlon prosthesis was patent throughout. The cause of death was designated as advanced arteriolar nephrosclerosis with renal failure and congestive heart failure.

Case 2. A 56-year-old colored woman was admitted November 7, 1956, 3 hours following the sudden onset of very severe pain in the lower thoracic and lumbar regions of the back. There had been no precordial, substernal, or epigastric pain. She had been given an intravenous dose of a narcotic but the pain was unabated. The past history revealed only a persistent hypertension treated by her physician for the past several years.

Physical examination revealed an obese woman in severe pain. Her blood pressure was 220/110 mm. Hg. Examination of the chest was normal. The right femoral pulse was thought to be weaker than that on the left. Urinalysis revealed a 2+ albumin and 25 erythrocytes per HPF, but other laboratory examinations, including a blood urea, were within normal limits.

The patient was originally thought to have a renal calculus, but the finding of a normal intravenous pyelogram and a consistently low blood pressure in the right leg focussed attention on the

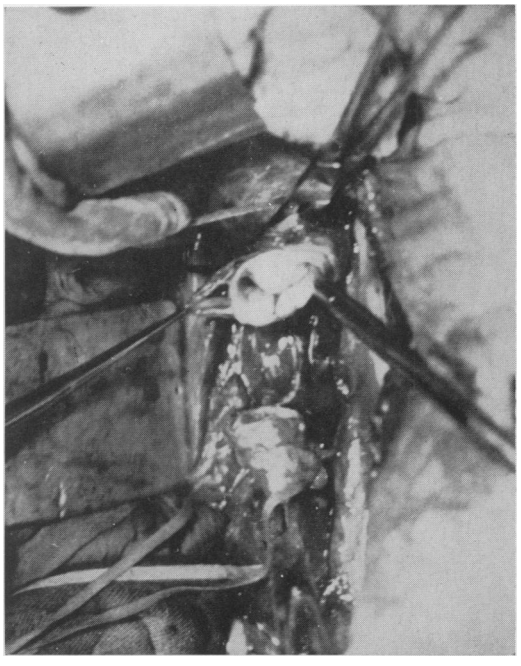

FIG. 4. Photograph made in Case 2 showing the divided descending aorta. The external wall of the aneurysm is being elevated and the clamp is in the true aortic lumen.

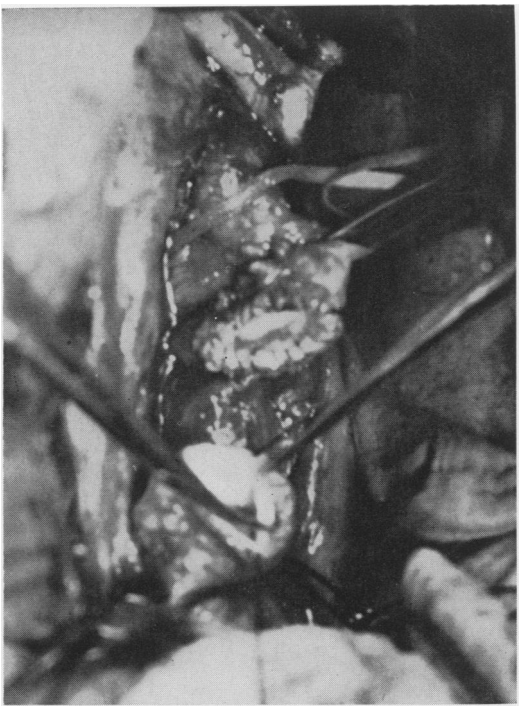

FIG. 5. Photograph of Case 2 with the opening into the distal aneurysmal space closed, and a segment of the proximal internal lamina being excised.

aorta. The pain subsided steadily over a four-day period and a retrograde aortogram was performed on the eighth day (Fig. 3). This established the diagnosis of a dissecting aneurysm involving both the thoracic and abdominal aorta. However, the blood urea rose to 113 mg. per cent following this procedure and it was decided to postpone operation until the renal function returned toward normal.

On the 28th of November a thoracotomy through the left 8th intercostal space was performed. The aneurysm was seen to involve all the visible intra-thoracic aorta; the pericardium was not opened. The pericardial, pleural, and mediastinal spaces were free of hemorrhage. The descending aorta was then divided in its middle third revealing a well-formed aneurysmal channel involving two-thirds of the circumference of the vessel (Figs. 4 and 5). The opening into the distal dissection was closed with a continuous silk suture, a crescent of proximal intima was excised as a re-entry site and the ends of the aorta were anastomosed (Figs. 6 and 7). The blood pressure rose to over 300 mm. Hg during the aortic occlusion, necessitating the use of an intravenous drip of trimethaphan camphorsulfonate (Arfonad®). With this agent the blood pressure could be maintained at a level of about 225 mm. Hg. Following removal of the clamps there was a marked hypotension requiring the use of intravenous norepinephrine for a short period.

The postoperative course was uneventful and the patient was discharged on the fourteenth postoperative day. She has continued to do well since discharge except for a gradual return of the blood pressure toward the preoperative level. This is being controlled at the present time with antihypertensive medications.

Case 3. On November 28, 1955, a 58-year-old white man was admitted to another hospital because of substernal pain radiating to the neck and both shoulders. A tentative diagnosis of myocardial infarction was made but the electrocardiographic changes were non-specific. He was placed on bed rest with some relief of the pain. On December 4, 1955, 7 hours prior to arrival at this hospital, the patient was seized with excruciating pain in the chest, back, and upper abdomen radiating into the legs. He collapsed, was unresponsive and neither pulse nor blood pressure was obtainable. Cardiac stimulating drugs were administered through the jugular vein and the patient slowly improved. For 3 hours after regaining consciousness the patient was unable to move his legs and

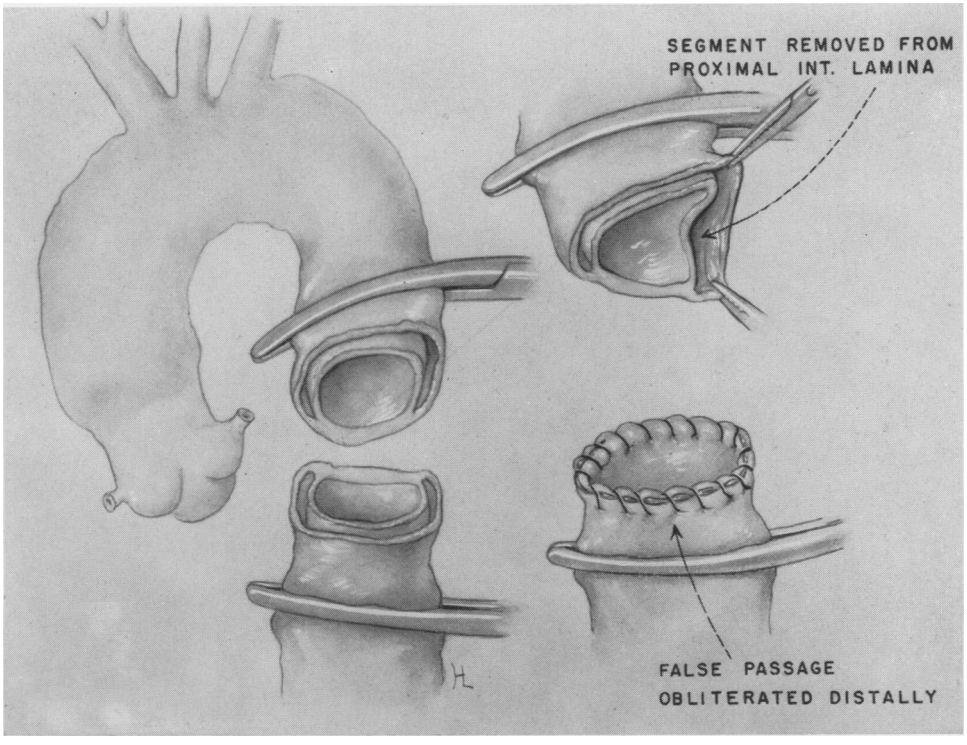

FIG. 6. Drawing made of the operation in Case 2 to portray the basic steps in the procedure.

no femoral pulses were palpable. An electrocardiogram was taken which showed slight non-specific changes, being similar to the one taken several days earlier. The clinical signs of an acute cardiac tamponade appeared and the diagnosis of dissecting aneurysm of the aorta with intrapericardial rupture was made. The patient was transported by ambulance to the University of Virginia Hospital.

On admission the patient was alert but extremely apprehensive. The blood pressure was 90/70 mm. Hg in each arm with equal femoral pulses of fair quality. The veins of the neck and arms were distended but there was no peripheral edema and the breath sounds were normal. The heart appeared to be enlarged with very distant sounds of poor quality. The patient was taken directly to the operating room with a diagnosis of intrapericardial rupture of a dissecting aneurysm.

A thoracotomy was performed through the left 5th interspace. The pericardium was found to be tense and filled with blood. Following release of the cardiac tamponade the patient's general condition improved immediately with a rise in systolic blood pressure from 90 mm. Hg to 120 mm. Hg. The bleeding was not brisk and the point of bleeding could not be seen; it appeared to be coming from the opposite side of the aorta. However, it was easily controlled with mild pressure and a sheet of Orlon cloth was then wrapped around the ascending aorta and sutured in place. Within a few minutes the bleeding stopped and further exploration of the thoracic aorta was carried out. The ascending aorta was erythematous and appeared thickened and a dissecting aneurysm was believed to be present in this area. Although the descending aorta did not appear to be abnormal, an anterior thoracotomy was carried out for inspection of the intima. This was normal but a window of intima was excised in the hope that any extension of the dissection would re-enter the true lumen at this point. During the occlusion of the aorta the blood pressure began to rise and reached the level of 200 mm. Hg in spite of intravenous Arfonad therapy. Just prior to removal of the aortic clamps, rather brisk bleeding appeared in the pericardial cavity with an estimated blood loss of 1,000 ml. in a few minutes. The heart suddenly stopped and required massage for about 10 minutes. At the end of this time effective contractions returned and the bleeding was controlled with pressure over the Orlon fabric. The day following operation the electrocardiogram showed evidence of a lateral myocardial infarction and this was followed shortly by the onset of auricular flutter. The patient was digitalized

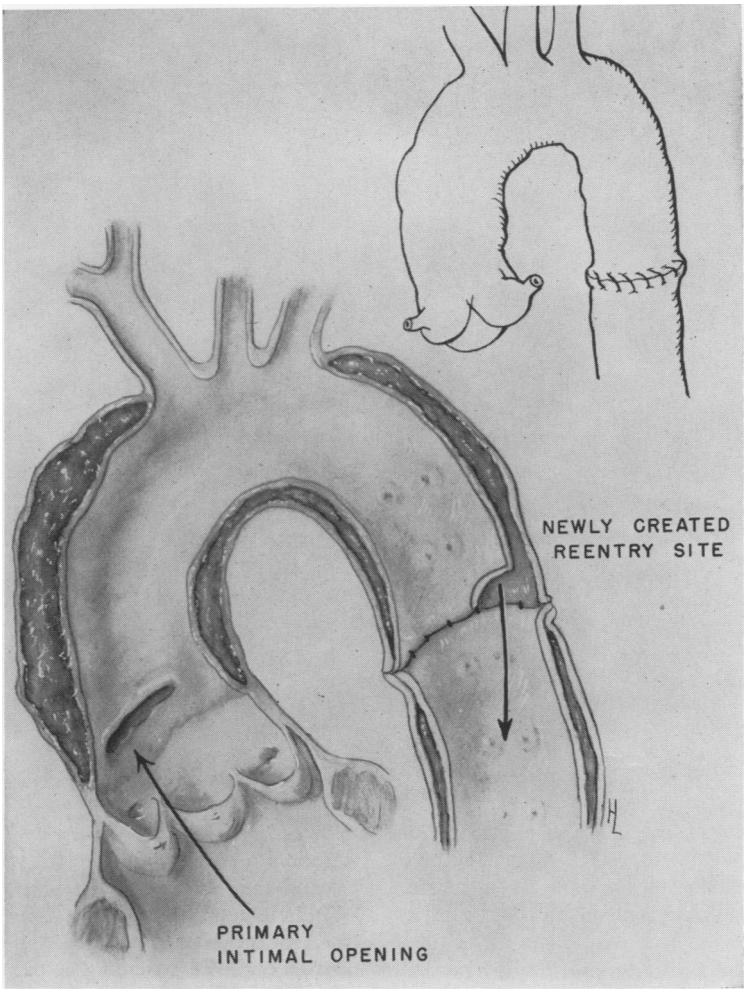

FIG. 7. Drawing made to illustrate the result of such a procedure as performed in Case 2. Note the decompression of the proximal aneurysmal space and closure of the entrance into the distal space.

and improved steadily through the ensuing 10 days, at which time he was alert and feeling well. On the 12th postoperative day the patient suddenly became dyspneic and slightly cyanotic, but denied any pain in the chest. During the next 3 days the respiratory difficulty gradually became worse, peripheral edema developed, and he died on the 15th postoperative day.

Autopsy revealed a large myocardial infarction of the anterolateral aspect of the left ventricular wall (Fig. 8). There was marked coronary sclerosis with a recent thrombus in the anterior descending branch of the left coronary artery. A careful search revealed neither a dissecting aneurysm nor the histologic characteristics of cystic medial necrosis of the aorta. A search for the site of the previous hemorrhage was likewise unrewarding; there had been no further bleeding since operation. The cause of death was considered to be cardiac failure following a large myocardial infarction.

Case 4. On December 12, 1955, a 52-year-old colored man was admitted from the Emergency Room complaining of crushing substernal pain radiating to the shoulders and back. A narcotic administered prior to his arrival here had failed to alleviate the pain.

On admission the patient was semicomatose and there was no obtainable blood pressure. Intravenous norepinephrine was started immediately with return of the systolic blood pressure to 78 mm. Hg and an improvement in the sensorium. The heart sounds were muffled and there was a cardiac arrhythmia. It was initially believed that

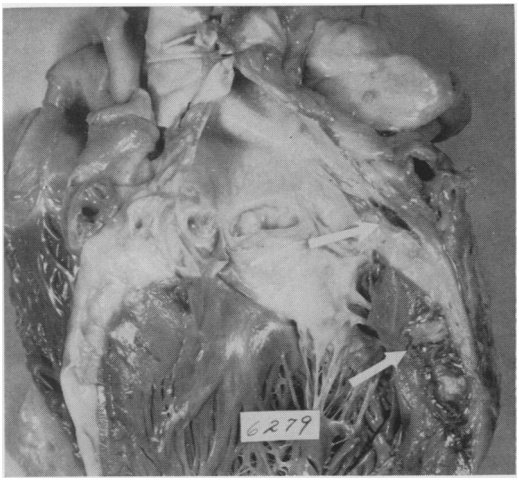

FIG. 8. Photograph of the autopsy specimen in Case 3, with the arrows pointing to the thrombosed coronary artery and the myocardial infarction.

an acute myocardial infarction was the most likely diagnosis, but that a dissecting aneurysm was a possibility. Following admission the intravenous norepinephrine therapy had to be continued for maintenance of blood pressure. About 6 hours following admission an electrocardiogram revealed changes suspicious of an anterior myocardial infarction. The patient developed marked dilatation of the neck veins with distant heart sounds and fluoroscopy revealed an enlarged heart with poor pulsation. The electrocardiogram at the 12-hour period showed regression of the previous abnormalities. A pericardial tap demonstrated dark blood under relatively low pressure and a diagnosis of ruptured dissecting aneurysm was made.

Twenty-one hours following admission a thoractomy through the left 4th interspace was carried out. The ascending aorta was definitely enlarged and there was about 200 ml. of blood in the pericardial cavity. The descending aorta appeared entirely normal, and a diagnosis of dissecting aneurysm limited to the ascending aorta and arch of the aorta was made. A search was carried out for the point of perforation but this was unsuccessful. Insertion of a #26 needle tangentially into the wall of the aneurysm revealed it to be extremely thin. The entire intrapericardial aorta was then wrapped with Orlon fabric which was sutured into proper position. Near the end of the procedure, an electrocardiogram revealed the onset of ventricular rhythm with a widened QRS complex. The patient's condition remained critical during the first 2 postoperative days, requiring intravenous norepinephrine and showing signs of

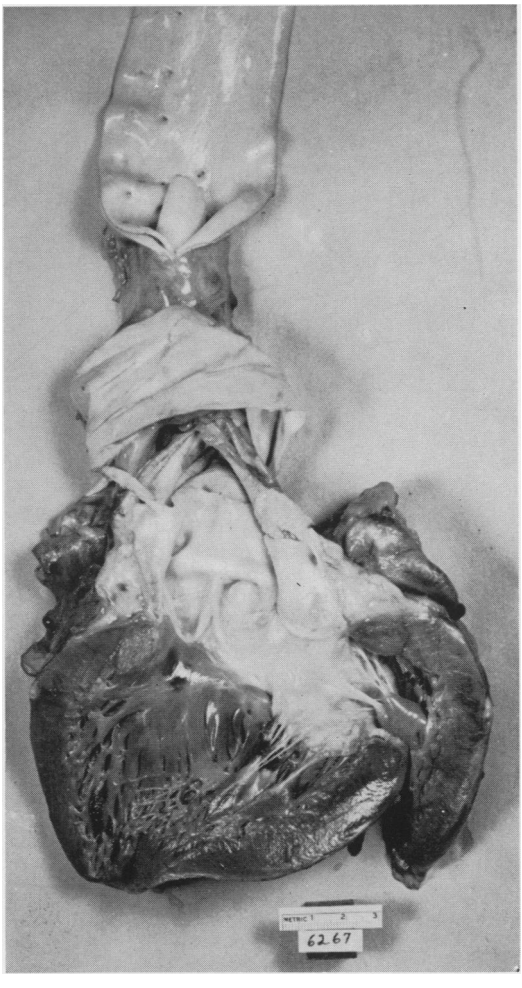

FIG. 9. Photograph of autopsy specimen in Case 4 with the aorta incompletely opened. Note the complete circumferential primary rupture, the band of Orlon fabric around the ascending aorta, and the protrusion of the invaginated internal lamina into the descending aorta.

posterior myocardial ischemia on the electrocardiogram. However, he remained comfortable and alert until the morning of the third postoperative day when he had a generalized convulsion and became comatose. Physical examination at this time revealed the left carotid and radial pulses to be markedly diminished. The femoral pulses were equal and of fair quality. In spite of various resuscitative measures the patient died about one hour following the convulsion. At that time it was believed there had been an extension of the dissecting process which had caused acute cerebral ischemia.

At postmortem examination there was no evidence of recurrence of the external hemorrhage.

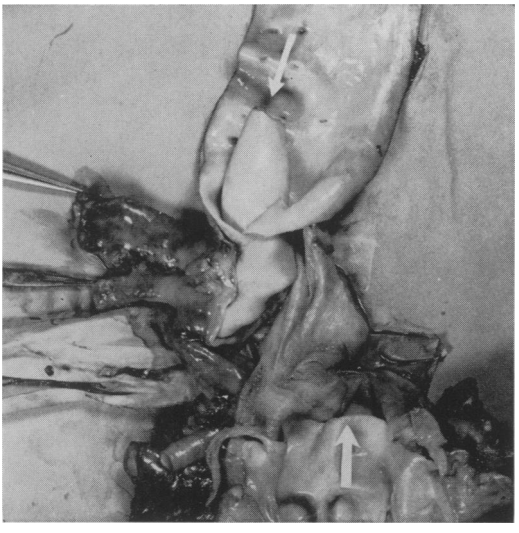

FIG. 10. Photograph of the autopsy specimen in Case 4 with the aorta completely opened, showing the nature of the obstruction to the vessels of the arch, and the very thin, wrinkled external wall of the aneurysmal sac.

The dissecting aneurysm was found to involve the entire circumference of the vessel and was limited to the ascending aorta and arch of the aorta. However, a most unusual complication had occurred with complete invagination of the internal lamina of the aneurysmal sac into the descending aorta (Fig. 9). This invagination seemingly happened suddenly, partially obstructing the vessels of the arch of the aorta, and precipitating the convulsion, coma, and death (Fig. 10). The peripheral flow was maintained through the core of the invagination as evidenced clinically by the good quality of the femoral pulses (Fig. 11). Additional findings included a ligature around a branch of the right coronary artery, but without a myocardial infarction.

Case 5. On January 17, 1956, a 62-year-old white man was admitted with the diagnosis of dissecting aneurysm of the aorta. He had been hospitalized elsewhere 19 days previously with excruciating pain in the interscapular area accompanied by peripheral vascular collapse. He was found to have a hemiparesis, which proved to be transient, and a markedly clouded sensorium. Two days following admission here he developed recurrence of the severe pain and shortly thereafter was found to have a large hemothorax, and 800 ml. of bloody fluid was removed from the left pleural space. The blood urea rose to 90 mg. per cent and the electrocardiogram showed evidence of posterior myocardial ischemia. On admission here the patient was in no acute discomfort but was still markedly lethargic. Peripheral

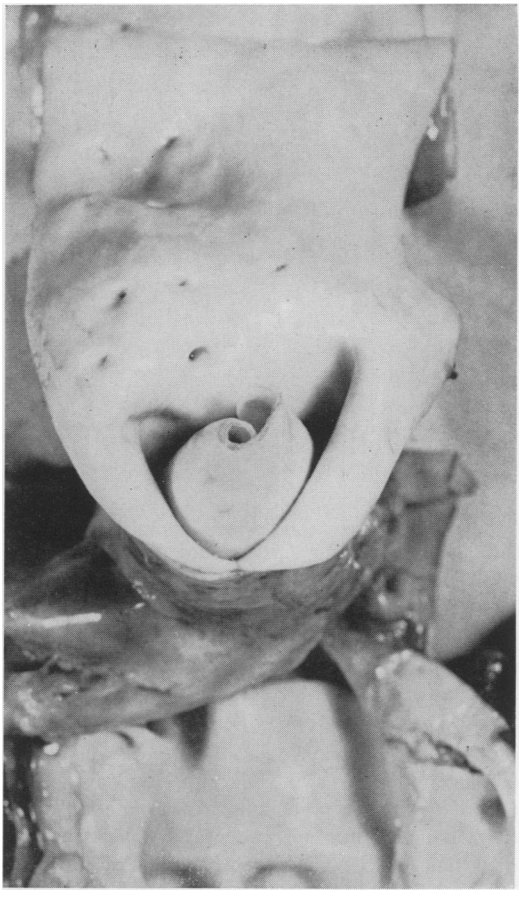

FIG. 11. Photograph of the autopsy specimen in Case 4 showing the invaginated internal lamina from below.

pulses were equal, full and regular with a blood pressure of 160/80 in each arm. The only significant laboratory finding was a blood urea of 57 mg. per cent. A roentgenogram of the chest revealed a large left pleural effusion with slight displacement of the mediastinum to the right. On January 18th a left thoracotomy through the 4th interspace was carried out. About 2000 ml. of bloody fluid was present in the left chest but there was no bleeding from the aneurysm at the time of operation. The heart appeared to be somewhat enlarged but the pericardial space was free of hemorrhage. The aorta was widened and thickened throughout the entire visible area. The descending aorta was divided between clamps, revealing a double lumen. In the aneurysmal space was a large thrombus with an 8 mm. channel in the center (Fig. 12). As much thrombus was removed as possible and the distal aneurysmal orifice was obliterated with a continuous silk suture. A segment of the proximal internal lamina

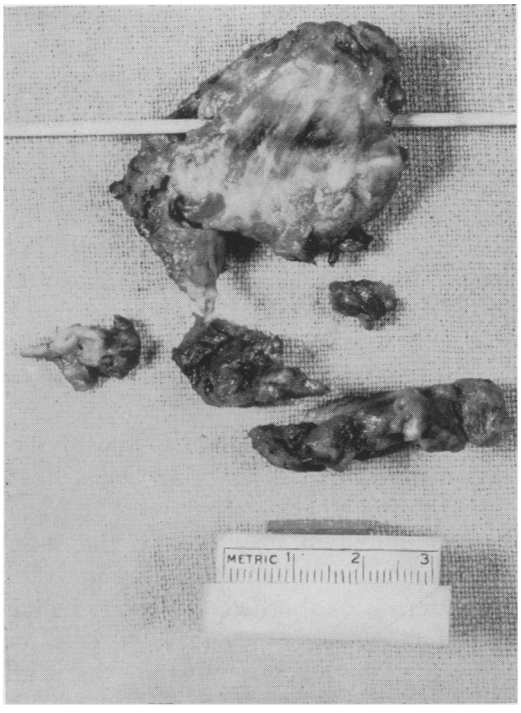

FIG. 12. Photograph of the thrombus removed from the aneurysmal space in Case 5 showing a probe passing through the central channel.

was excised and a routine anastomosis carried out. Following the application of the aortic clamps, the blood pressure began to rise and intravenous Arfonad® was begun by continuous drip. With this therapy the blood pressure rise was limited to a maximum of 250 mm. Hg. Following removal of the clamps a mild hypotension developed which was easily controlled with the vasopressor agent. However, following division of the aorta the anesthesiologist had noted that the plane of anesthesia was deepening. Although no more anesthetic agent was administered the patient showed no signs of response. At the conclusion of the procedure the thoracotomy tube was irrigated but no evidence of recurrent bleeding was obtained. About one hour later the patient became steadily less responsive and respirations ceased, followed shortly by a fall in blood pressure and death.

Permission for autopsy could not be obtained. It was believed the cause of death was cerebral damage following extension of a dissection of one or both of the carotid arteries.

DISCUSSION

In looking at the problem of acute dissecting aneurysm of the aorta from a therapeutic standpoint, there are two principal reasons for considering the operative approach. The first is the very high mortality which has accompanied the non-operative management of these cases in all reported series. This has been variously estimated at 75 to 90 per cent [13] and when one considers the data of Mote and Carr,[11] the true figure is probably nearer the latter value. In analyzing the cases from the San Francisco coroner's office, these investigators found dissecting aneurysms in 1.1 per cent of all non-violent deaths. It seems likely, therefore, with the relatively high proportion of dissecting aneurysms in these cases of rapid and unexplained death, that the true mortality is higher than is generally reported.

The second feature suggesting surgical intervention in this condition is found in the nature of the so-called healed dissection. The great majority of these cases are found to have ruptured back into the true lumen of the aorta distally, forming a reentry site for the aneurysmal channel.[13] On the other hand, the usual cause of death in rapidly fatal cases is hemorrhage from rupture of the aorta externally. From a study of available material it seems to be somewhat of a chance occurrence as to which course a given patient will pursue. In the light of the above considerations, it has seemed warranted to try planned internal decompression of the acute dissections, fully realizing that a small percentage of the cases with spontaneous formation of a distal internal opening have had a subsequent external rupture.

Many patients are not candidates for operation due to the rapidity of the process with an early fatal termination. In evaluating the problem of the duration of the disease, some authors [5, 9] have felt that most of the patients survive for 48 hours or more. However, others [11, 13] disagree with this and it seems more likely that the majority of patients die in the first 24 hours.

When one again considers the fact that in the San Francisco coroner's office there were 60 cases of acute death from dissecting aneurysms in one five-year period, it appears that most published series are from a highly selected group of patients as far as duration of survival is concerned. Nevertheless, the proportion of patients surviving for 12 hours or more in the series from large general hospitals has been quite high, and for this group, surgical treatment should be possible.

In analyzing the problems which arise in the management of these cases, the first encountered is that of establishing the diagnosis. In 1933, at the time of Shennan's monograph,[13] only six cases (or 2 per cent) had been diagnosed preoperatively. By 1950, 10.6 per cent of the 724 cases reviewed by Levinson *et al.*[9] had been correctly diagnosed prior to death and in individual series this had been as high as 30 to 40 per cent.[2, 9] Judging from similar situations in the past, the accuracy of clinical diagnosis will improve still further as an effective therapeutic tool becomes available.

The differential diagnosis of dissecting aneurysm of the aorta most commonly includes either a myocardial infarction[2] or an intra-abdominal emergency.[1] The importance of a correct clinical diagnosis in cases simulating coronary artery disease is obvious, since surgery must be avoided in patients with acute myocardial infarctions, yet is urgently needed when a dissecting aneurysm is present. The differential diagnosis is not so critical in those patients with abdominal symptoms because the primary disease may require surgical therapy and a negative exploration is not nearly as hazardous. As has been said so often before, the greatest factor in aiding a correct diagnosis is to have a high index of suspicion.

The pain in acute dissecting aneurysm is usually very severe and often is described as a tearing sensation. In any patient with pain in the chest or back of such severity that narcotics fail to relieve it, the diagnosis of a dissecting aneurysm should be considered. Usually of great significance is the location of the original pain and the changes in position which may follow. Although it may mimic exactly a myocardial infarction with radiation of the pain to the neck and shoulders, it more commonly radiates through to the back between the scapulae. Should the pain shortly move into the lower thoracic and lumbar areas of the back with radiation to the hips or legs a diagnosis of dissecting aneurysm is greatly strengthened. The pain of myocardial infarction may involve the intrascapular or lower thoracic areas but once its pattern is established, it tends to remain constant.

In the physical examination there are several features which, if present, should make one suspicious of a dissecting aneurysm. An aortic diastolic murmur is frequently found and should there be indication that it is of recent occurrence (many of these patients have been examined regularly for hypertensive cardiovascular disease) it offers strong support to the diagnosis. The difference in peripheral pulses has long been recognized as one of the salient clinical features and in one of our patients (Case 2) provided the real clue to the diagnosis. The findings of unequal pulses should be confirmed by blood pressure determinations in the four extremities. There may be a secondary neurological disorder such as hemiplegia or paraplegia, which may be either permanent or transient. In two of our four patients there were definite indications of central nervous system involvement (Cases 1 and 5). It should be emphasized that such findings, in an illness beginning with severe pain, should bring to mind an acute dissecting aneurysm. Finally, an important characteristic of the disease is hemorrhage into

the pericardial or pleural space. The discovery of a bloody effusion in either of these spaces, accompanied by other features of the disease, is very strong evidence favoring the diagnosis. One might surmise that such findings would be of little practical help due to the probability of death ensuing rapidly. However, such is not necessarily the case. Two of our four cases were diagnosed primarily on this basis (Cases 3 and 5), one from the signs of cardiac tamponade and the other from a massive hemothorax. But this is not an infallible diagnostic aid as is graphically illustrated in our third case. This man had suddenly collapsed and developed a paraplegia which subsequently cleared. An electrocardiogram showed no change from one taken a few days previously and he shortly developed the clinical signs of cardiac tamponade. At operation the pericardium was found to be distended with fresh blood, and a dissecting aneurysm of the ascending aorta was thought to be present. At autopsy examination 15 days later, there was no aneurysm demonstrable, but a large myocardial infarction of the left ventricular wall was present.

The laboratory aids are of occasional value, with the finding of blood in the urine and elevated blood urea being the most useful. Radiologic examination of the chest may be of considerable help in the acute as well as the chronic cases. The widening of the aortic shadow or the presence of intrapericardial or intrapleural fluid may be detected in this manner. In patients in the later course of their disease an angiocardiogram may be performed safely and in most instances this should establish the diagnosis.

The electrocardiogram may follow a variety of patterns as emphasized by Levinson *et al.*[9] However, it is of value to find a normal electrocardiogram, particularly if this persists for several hours. It is unusual to find a repeatedly normal electrocardiogram in a patient with a myocardial infarction large enough to produce severe pain and peripheral vascular collapse. The signs of myocardial ischemia are seen not infrequently with the dissecting aneurysm, as the coronary arteries are sometimes involved in the dissection. Other features seen frequently are those of hypertension and pericarditis. If the electrocardiogram gives clear-cut evidence of an acute myocardial infarction, however, serious consideration should be given to withholding operation, unless a definite external hemorrhage or complete peripheral vascular obstruction is demonstrated.

If all the factors in these cases are carefully evaluated it is probable that the majority of patients living long enough to be hospitalized should have a correct diagnosis made. In the eight-month period since undertaking this new therapeutic approach which demands an accurate early diagnosis, six patients have had the diagnosis of dissecting aneurysm made in this hospital. In five of these the diagnosis was correct while one proved to be a myocardial infarction with bleeding into the pericardial cavity and cardiac tamponade. An additional patient was discovered at autopsy to have died from an intrapericardial rupture of an acute dissecting aneurysm. This patient, who had previously had rheumatic heart disease and a myocardial infarction, was diagnosed as having another infarction and died 12 hours following admission.

Once the diagnosis has been established, the question of the proper time for surgical intervention arises. From the study of autopsy data, it is an inescapable fact that the most critical period in the disease is the first 48 hours. It is in this period, therefore, that one would expect the greatest salvage of cases to occur. On the other hand, there is little doubt that this is the most dangerous period in which to operate and that the surgical mortality will be higher in these early cases. It is apparent

that this increased risk of operation must be balanced against a very considerable risk of temporizing with a rapidly fatal disease. At the present time it appears that the operative risk in these very early cases is outweighed by the extreme gravity of their prognosis and operation should usually be undertaken as an emergency procedure. However, as the duration of the disease progresses, the danger of sudden death decreases. In cases of longer standing, additional time may be taken to obtain diagnostic studies and to improve the general condition of these patients. An example of this approach is found in Case 2. The diagnosis was not established until an aortogram was performed on the eighth hospital day (Fig. 3). Following this procedure, which demonstrated the aneurysm extending below the renal arteries, there was a marked rise in the blood urea. In view of the experience of others, as well as ourselves, with renal failure following surgical intervention in dissecting aneurysm of the abdominal aorta, operation was delayed until renal function again approached normal. This patient made an uneventful postoperative recovery.

At the opposite pole are the cases with survival of months prior to the discovery of their lesions. These may be brought to light by the finding of a widened aortic shadow on chest x-ray, the development of symptoms of congestive failure or pressure on mediastinal structures.

The greatest single cause of death in these chronic cases is congestive heart failure and, together with chronic renal disease and cerebral hemorrhage, it comprises almost two-thirds of the entire group. It is not known whether current operative technics will influence the course of this cardiac and renal disease, but it is doubtful since many patients suffer these complications prior to the development of the dissecting aneurysm. Consequently, the presence of chronic heart disease or renal insufficiency accompanying a dissecting aneurysm of long duration is not in itself indication for operation.

These patients may die of rupture of the aorta years after the initial episode, but the majority of them do not. Of Shennan's series of 75 chronic dissecting aneurysms only 16 (21 per cent) died of external rupture. Of this group four died of a rupture of an entirely new dissecting aneurysm while an additional four died following an extension of a previous dissection. However, in the eight additional cases external hemorrhage followed rupture of a secondary saccular aneurysm. It is doubtful that there is sufficient evidence to justify the routine surgical approach to all chronic dissecting aneurysms from the standpoint of preventing subsequent hemorrhage. The great difficulty and increased hazard in removing the ascending aorta and arch of the aorta, by far the most vulnerable site in this disease, would greatly limit the usefulness of the routine surgical attack upon all of these lesions. However, it is clearly indicated that the development of a secondary saccular aneurysm greatly increases the chance of hemorrhage. For these lesions surgical therapy is practicable and could result in considerable prolongation of life. In addition, the presence of pressure symptoms or the demonstrable localization of the lesion to an area where the proximal portion of the lesion may be excised would seem to offer sufficient indication for surgical intervention.

There is little doubt that the operative approach should be through the chest. Both in Gore's [6] and in Shennan's [13] series, 95 to 98 per cent of all acute dissections began in the thoracic aorta, while the distal limit reached the abdomen in only 35 and 55 per cent of these same large series. Furthermore, although from 85 to 95 per cent of these patients died from hemorrhage, only 2 per cent of these occurred in the abdomen. Both from the standpoint of

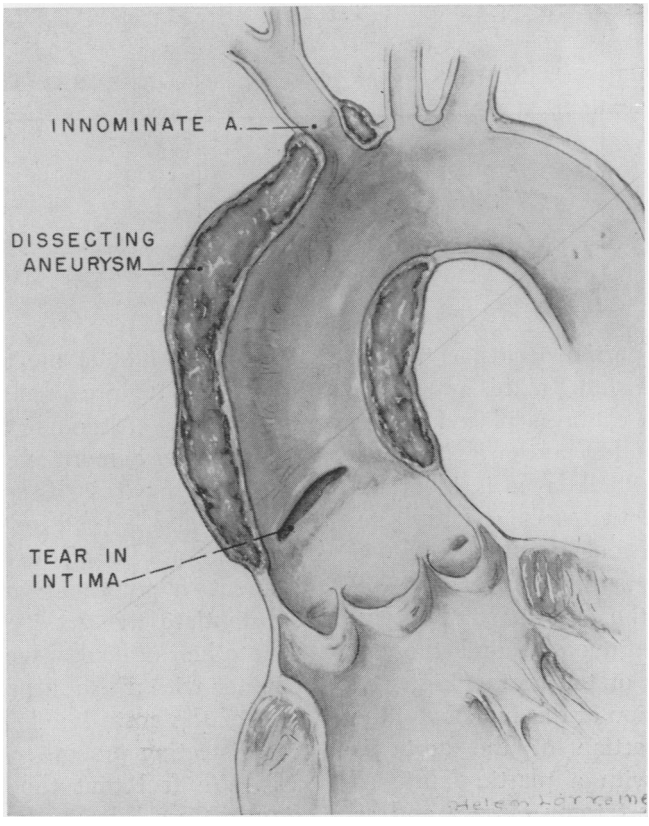

FIG. 13. Drawing of the type of lesion localized to the ascending aorta and arch of the aorta.

finding the aneurysm and controlling an external perforation, a transthoracic exploration should be utilized. Exceptions to this are cases with complete obstruction to the distal aorta or iliac arteries and the occasional case with definite intra-abdominal hemorrhage.

Having localized the lesion, the choice of operative procedure depends to a large extent on the pathologic and anatomic variation encountered. The different procedures which may be utilized have been excellently demonstrated by DeBakey, Cooley and Creech.[3] There are four general aims that must be kept in mind regardless of the type of operation to be performed. They are (1) prevention of distal extension of the aneurysm; (2) creation of a distal re-entry site for the aneurysmal sac; (3) prevention and/or relief of arterial obstruction; (4) control of hemorrhage from the aneurysm (including relief of cardiac tamponade if present). Unfortunately we have been unable to carry out these precepts in all of our cases.

One of the most perplexing problems which we have encountered concerns the management of lesions localized to the ascending aorta or arch of the aorta (Fig. 13). This is a frequent finding as evidenced by analysis of autopsy data (Table I). Whereas only one-seventh of the chronic cases failed to reach the descending aorta, over one-half of the acute cases were of this limited extent. We have been concerned with the technical limitations imposed by lesions of this type which prevent the complete division of the aorta with repair as utilized in the distal portion. We have consequently done some preliminary experiments in ani-

TABLE I.

LOCATION	ACUTE (5 weeks or less)	CHRONIC (More than 5 weeks)
Proximal to descending aorta	54%	14%
Descending aorta or below	46%	86%

mals regarding a partial occlusion technic which seems promising. In this procedure a partial occlusion clamp is placed on the aorta and the occluded portion of the wall is incised. The intimal layer is identified and a segment excised as a re-entry site. The distal aneurysmal opening is closed with a continuous suture (Fig. 14). Following repair of the aortotomy, the original suture is continued around the entire circumference of the aorta (Fig. 15). Following completion of this maneuver, the intrapericardial portion of the aorta is wrapped snugly with a plastic fabric as support for the aortotomy closure and as re-inforcement to the entire intrapericardial aorta to help prevent subsequent external rupture (Fig. 16). This should be positioned as close to the heart as possible, taking care not to injure a coronary artery. This serves not only to lessen the distension on this most vulnerable area, but also acts to promote hemostasis. This technic is untried clinically, but offers an approach to an extremely difficult situation.

A somewhat similar problem is related to blood pressure changes coincident to cross-clamping the descending aorta. The most distressing feature in our hands has been the severe rise in pressure attendant with the occlusion of the aorta. Hypotension subsequent to removal of the clamps may also be difficult to control. There are two potential complications which make the avoidance of hypertension most desirable and we have experienced each of these disasters. The first and probably the more important of the two is that of perforation of the proximal aorta with external hemorrhage. Although the patient (Case 3) in whom hemorrhage occurred was not found to have a dissecting aneurysm at autopsy and is used only by way of analogy, the rapidity of the bleeding with subsequent hypotension and cardiac arrest substantiate the gravity of this situation. The other difficulty which one may encounter with a rise in pressure from occlusion of the aorta is related to extension of the dissecting process in the proximal vessels. We feel that the sudden hypertension in Case 5 led to further dissection of the vessels of the arch and fatal cerebral ischemia. Although no autopsy was obtained in this case the marked central nervous system symptoms following the original dissection, the failure to demonstrate bleeding from a large thoracotomy tube following closure of the chest, and the manner of death all point to cerebral ischemia as the chief factor in this patient's demise. In each of these patients Arfonad® was given intravenously in an attempt to control the pressure rise, but it was not administered until the hypertension was already developing. It is more effective to start the drug shortly before the application of the occluding clamps and maintain the pressure at the desired level by altering the rate of flow during the period of aortic occlusion. As an additional precaution, in cases which have had external hemorrhage or definite symptoms of impairment to

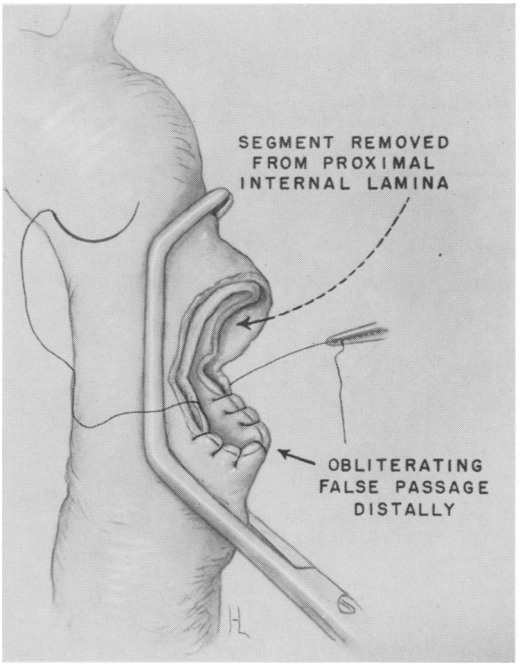

FIG. 14. Drawing of the partial occlusion technic showing creation of the re-entry site and beginning obliteration of the distal space (pictured here in the descending aorta).

cerebral flow, the partial occlusion technic should probably be utilized to minimize the possibility of the development of such a catastrophe.

Prior to the removal of the aortic clamps a vasopressor agent should be prepared for intravenous administration in case the need arises. The distal clamp may be readily removed as a preliminary test of the effectiveness of the anastomosis. However, the proximal clamp should be slowly released over a period of several minutes and reapplied if a sudden drop in pressure occurs. In three of our cases support with an intravenous vasopressor agent following operation was necessary, but only in one of them were we unable to discontinue its use (Case 4). The maintenance of the blood pressure by normal physiological means is desirable in all cases, but particularly in those with advanced renal disease or renal artery involvement by the dissecting aneurysm.

Probably of equal importance is the careful planning of the postoperative regimen. The frequency of fatal rupture in chronic cases, of either the original or a new dissecting aneurysm, serves to emphasize this point. Weight reduction, restriction of activity, sedation, and anti-hypertensive medication should all be used when indicated.

There is now definite evidence that perforation of an aortic aneurysm is not necessarily a rapidly fatal occurrence. This has been well-documented in the case of arteriosclerotic aneurysms of the abdominal aorta and similar evidence is accumulating in the case of dissecting aneurysms of the aorta. Johns [8] and Maier [10] have reported cases with control of hemorrhage in the abdomen and left pleural spaces respectively, although these patients died shortly of other causes. One of the patients presented by DeBakey *et al.*[3] was successfully operated upon following an intrapleural hemorrhage, which had apparently stopped bleeding spontaneously prior to operation. We have had two cases with external hemorrhage from the dissecting aneurysm, one in the pericardial space and one in the left pleural space. It is interesting to note that in neither case were we able to identify the bleeding point. According to Shennan the failure to discover the site of perforation at autopsy is quite common. This constitutes further evidence that the bleeding in these cases may be insidious. In two cases (our Case 2 and that of Maier) active bleeding was controlled by the application of a band of fabric (Fig. 16) and in one (Johns), massive bleeding within the abdomen was controlled by direct suture. This demonstration that the bleeding from a perforation of the aneurysm may be controlled surgically constitutes one of the important bases for the operative treatment of dissecting aneurysms.

Another factor which should be stressed is that the cause of death in many cases from perforation and intrapericardial hem-

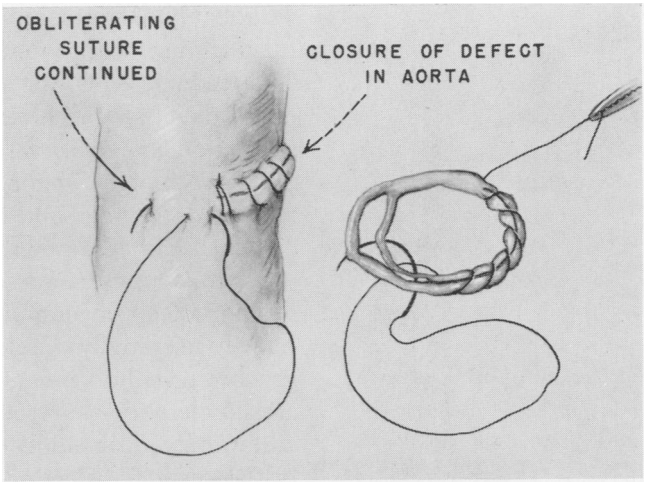

FIG. 15. Drawing showing the suture technic following closure of the aortotomy. The needle must pierce the wall of the aorta behind the previous suture to insure inclusion of the internal lamina with obliteration of the aneurysmal space.

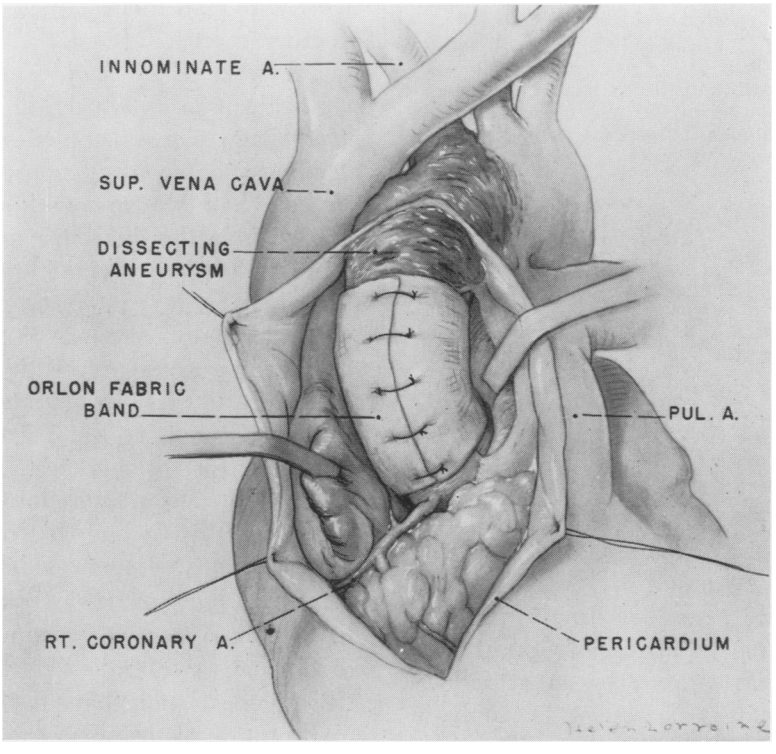

FIG. 16. Drawing illustrating a band of Orlon applied to the intrapericardial aorta.

orrhage is probably not related to the acute blood loss and hypovolemia, but rather to the production of an acute cardiac tamponade. This is emphasized by the small volume of blood which may be found within the pericardial sac in cases of death from intrapericardial hemorrhage. In our case there was a definite tamponade at the time of operation although only about 300 ml. of blood were present within the peri-

cardium. Following evacuation of a pericardial hematoma and control of bleeding in the ascending aorta, the pericardium should not be tightly closed, in case further bleeding should ensue with recurrence of the obstructive symptoms.

There have been several cases reported in which arterial obstruction to the legs was one of the principal features of the disease. This obstruction is usually caused by thrombus developing in the distal end of the aneurysmal space and thus obstructing the true lumen. Simple removal of the clots with creation of a distal internal opening to prevent recurrence of the thrombus will usually satisfactorily relieve the obstruction. However in our first case, despite persistent efforts to avoid resection, we were never able to demonstrate patency through the iliac arteries. This was probably due in some part to the extreme atheromatous changes which were present in these vessels as well as the extensive involvement with the dissection. Another factor which may be of importance is the embolization of distal arteries from the thrombus which formed in the aneurysmal space. Consequently, it is wise to release each of the clamps momentarily before closure of the aorta in an effort to flush out any loose clots which may be present within the aneurysmal sac. In only one reported case [12] has obstruction occurred following operation and that was from thrombi in the popliteal and posterior tibial arteries following relief of obstruction in the iliac artery on that side.

SUMMARY

1) The surgical treatment for acute dissecting aneurysms of the aorta is of recent origin and clinical data relating to these cases is quite limited.

2) Five patients operated on at the University of Virginia Hospital for dissecting aneurysm are presented. One of the patients did not have a dissecting aneurysm but a myocardial infarction with intrapericardial hemorrhage and cardiac tamponade. The difficulties in diagnosis are discussed.

3) Problems relating to the operative handling of these lesions have been discussed with particular reference to (a) the optimum time for operative intervention; (b) the technical limitations imposed by various anatomical factors; (c) control of hemorrhage from rupture of the external aneurysmal wall; (d) the complications associated with blood pressure changes incident to the operative procedure, and (e) the management of peripheral arterial obstruction.

4) With increasing experience in the surgical management of the acute cases, the extremely poor prognosis of these patients should be improved.

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DISCUSSION.—DR. DENTON A. COOLEY, Houston, Texas: Including the cases of dissecting aneurysm of the thoracic aorta which we reported before this association last year we have operated upon a total of 14 patients. In four patients the dissecting process was acute, producing severe pain and vascular collapse. The only death which occurred after operation in this group was probably due to our failure to control hypertension after operation, and intrapericardial rupture with pericardial tamponade resulted eight days later. This experience emphasizes the importance of antihypertensive drugs after such operative procedures particularly where the blood pressure is extremely elevated. The procedure which we proposed for lesions located in the ascending aorta was division of the aorta and creating a re-entrance passage into the true aortic lumen to provide a means of decompressing the false lumen. As yet it is too early to evaluate the results of this method of treatment, but we will continue with this technical approach until something better is available.

The other type of dissecting aneurysm we have treated surgically is the one which originates in the distal portion of the arch at or just beyond the origin of the left subclavian artery. Usually there is a localized fusiform swelling or bulge on the aorta in these cases indicating the site of origin and ultimate perforation. Resection of this area can be accomplished with homograft replacement. If the dissecting process extends below the level of resection then the wall of the true and false lumens should be approximated with sutures to arrest the dissection below the graft and thus protect the vessels located distally which may be compressed by the pressure in the false passage.

In patients with an acute dissecting aneurysm in which the dissection extends from the aortic arch to the iliac arteries producing iliac artery occlusion, it is advisable to interrupt the aorta at some level above the diaphragm to protect the renal arteries since renal artery occlusion may lead to death from uremia if both renal vessels are compressed. Similar treatment in the abdominal aorta above the bifurcation should also be used to relieve the iliac occlusion.

Not all dissecting aneurysms are associated with cystic medionecrosis and hypertension. For example we have operated upon two women both about 40-years-old who were normotensive and both had some of the manifestations of Marfan's syndrome with unusual skeletal development. Moreover, in some arteriosclerotic patients with or without hypertension dissecting aneurysm may occur and the histologic appearance of the aorta will show only findings of arteriosclerosis and the media may be normal in appearance. This paper presented by Dr. Warren was most interesting and should help to clarify some of the pathologic and clinical aspects of acute dissecting aneurysm and serve to place the surgical treatment of these lesions on a sound basis.

DR. WILLIAM H. MULLER, JR., Charlottesville, Virginia: We wish to thank Dr. Cooley for his discussion, and also to compliment him and Dr. DeBakey and their associates for the excellent results they have achieved in their cases, which they reported last year and which they have brought up-to-date this year.

We have tried to emphasize the necessity for early operation in these patients during the first 48 hours or so after the occurrence of the aneurysm because the mortality rate is so exceedingly high. On the other hand, the benefit achieved by our present technics must be balanced against the relatively high operative mortality rate in these acutely and severely ill patients.

The patient with intrapericardial rupture will nearly always have a fatal outcome, and therefore it is in this group that a vigorous effort should be made to control the hemorrhage. The orlon fabric band which we utilized in two patients controlled the bleeding effectively but did nothing for the underlying pathological lesion, as was demonstrated in the patient in whom the intima invaginated through the arch of the aorta.

It is entirely likely that, following control of bleeding, one might replace the arch of the aorta at a later date with a homologous arterial graft, thus rendering a more definitive type of treatment. I believe Dr. DeBakey and Dr. Cooley have done this in patients with sacceform aneurysms.

If it is observed that the dissection has not extended beyond the arch of the aorta, one should probably not cross-clamp the aorta because of the likelihood of reinitiating bleeding and also producing further dissection about the arteries arising from the arch, and which may compromise the blood supply to the central nervous ssytem. In the one patient on whom this was tried, we could find no sac to enter, and therefore could not form a re-entry site.

Finally, many of these patients are hypertensive, and care should be taken after the operation to place them on a regimen to control their hypertension in an effort to prevent further dissection.