Direct Repair of Dissecting Aneurysms of the Aorta

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Dissecting aneurysm of the aorta has been recognized as a clinical and pathologic entity for 200 years.1 For most of this time it was considered sufficiently uncommon and carried such a hopeless prognosis that it received scant attention except as a medical curiosity. Until recently the majority of cases were diagnosed only post mortem. Renewed interest in the problem has shown that the incidence of the process is much higher than had been recognized in the past, and that it is now possible to make the clinical diagnosis with a high degree of accuracy. The development of first palliative2 and now curative surgical procedures has further stimulated interest in the problem, with the result that more cases are suspected and actively investigated to obtain a positive diagnosis. As late as 1937, when Shennan3 collected 300 cases of dissecting aneurysm, the diagnosis had been made ante mortem in only six of these. In 1958, a collected series of 339 cases had a premortem diagnosis in 40.4 per cent,4 thus, revealing the appreciation by the physicians of this problem. Despite the bizarre variants in symptomatology associated with acute dissection, a well-substantiated clinical diagnosis can be made in the majority of cases.5 In view of the manifestations of the disease the tendency is frequently directed toward multiple diagnoses in an attempt to explain the widespread organ involvement. The onset of the process is usually sudden, with severe pain of a "tearing" quality, most frequently the pain begins subternally or precordially, but may arise elsewhere in the chest. Peripheral extension of the pain may occur or this may be the sole manifestation of pain. As is evident from the anatomy of the aorta and its branches, any organ or systems may be involved to a lesser or greater extent according to its participation in the process. Thus, cerebral, myocardial, pulmonary, renal, or neurologic systems may be involved in the extension of the dissection. The use of contrast angiography has markedly enhanced the accuracy of diagnosis when it was employed to screen those patients in whom the diagnosis was suspected or those in whom the diagnosis was not apparent from simpler studies. When venous angiographic technics or retrograde aortography is added to the clinical features, a satisfactory diagnosis can usually be established with a high degree of accuracy.

In the natural history of the disease it now appears that between 10 and 25 per cent of those manifesting the signs of the acute process will survive for more than two months. Of this group a high proportion live for prolonged periods of time, and may manifest signs of aneurysms, aortic regurgitation, coronary insufficiency, or neurologic changes. Where the process has become chronic, a difficult diagnostic problem arises, since a history of the acute attack is frequently lacking.

Of those who die during the acute phase (75 to 90 per cent) a great majority succumb within the first several days, most of the remainder within 2 weeks, and a final small group within 2 months. It is notable that there are very few deaths within the first 12 hours. It is apparent therefore that to achieve optimal salvage from this disastrous process, definitive surgical intervention should
be advised early after its onset. During the past 3½ years 28 patients have had various procedures for palliation or correction of this lesion, with a 68-per cent survival. One of these survivals had an intrapericardial hemorrhage due to rupture just prior to initiation of the procedure. It should be noted, however, that in relation to the entire problem the number of patients who survived the acute process and lived for prolonged periods would appear to be much larger than previously reported. This opinion has been further substantiated by a group of patients whom we have studied in whom a chronic dissection has been present for months or years. These patients have been treated during this period prior to admission for conditions other than dissecting aneurysm, and the true diagnosis has been made by special studies and confirmed by operation.

Extensive experience with direct repair of aortic insufficiency and diseases of the aortic root has directed our attention to the possible methods for total correction of the fairly large group of dissecting aneurysms that originate in the ascending aorta and the aortic root. Operative procedures, which necessitate isolation of the ascending aorta in order to permit direct visualization of the aortic valve or replacement of the ascending arch, can be accomplished in several ways. Since coronary artery perfusion is interrupted by ascending aortic occlusion, cardiac muscle blood supply must be maintained by direct coronary perfusion or the cardiac metabolism must be reduced by other methods. It has been our experience that normal and diseased cardiac muscle can be adequately protected for periods as long as 110 minutes with total cessation of coronary flow by local cardiac cooling (fig. 1). If the myocardial temperature is maintained at 5 to 10 C., periods of cardiac arrest within these limits are well tolerated without observable impairment of myocardial contractility in the postoperative period. Further investigation has not yet defined the maximal time that safe cardiac arrest can be maintained by local cardiac cooling. We have demonstrated that if myocardial temperature is held in the range of 6 to 8 C. that the safe period of arrest is well over 60 minutes, even in the hearts seriously damaged by valvular disease. Our experience has led us to the conclusion that this is an extremely safe and effective technic. After the experimental use of this method in animals it has been applied clinically in over 150 patients for the correction of lesions of the aortic root and aortic valve, including patients with known chronic dissecting aneurysms who also showed evidence of severe aortic valvular incompetence.
Discussion

Classification of dissecting aneurysms in relation to the anatomic level of the aorta in which it arises is important from both diagnostic and surgical considerations. These sites of origin are (1) the attachments of the aortic valve leaflets, (2) ascending aorta above the aortic valve, (3) transverse arch, (4) at the level of the subclavian artery, and (5) low thoracic or abdominal aorta.

Those dissecting aneurysms arising at the level of the subclavian artery or distally to this point can be directly resected. In some cases a prosthesis must be inserted to restore continuity, after reconstruction of the distal aorta. In others a direct anastomosis can be made after resection of the site of the intimal tear. These form the groups for whom a simple curative procedure has been available for some time.

Those that arise proximal to the innominate artery present the (fig. 2) most challenging problems. The use of a reentry-type operation, in which the distal dissection is obliterated and a window created proximally, has salvaged many patients in this group of proximal dissections who otherwise would not have survived.

After a direct approach to the area of the aortic valve was developed, it appeared possible that a method for the total correction of these lesions of dissection in the root or in the ascending portion of the aorta could be accomplished by means of cardiopulmonary bypass. Even the patient in an acute phase of dissection can be treated by this direct type of repair. In the reentry type of procedure cross-clamping of the aorta has been associated with proximal aortic rupture, cardiac arrest, ventricular fibrillation, or left ventricular overload. These dangers are essentially eliminated when the repair is accomplished with direct closure, since the site of origin within the aorta is not perfused during cardiopulmonary bypass and cardiac arrest, and the myocardium is protected from arrhythmias during the cold arrest.
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Figure 5
Tricuspid valve composed of three Dacron cloth leaflets impregnated with silicone rubber. One or all of these cusps may be utilized in the repair of aortic valvular disease.

Figure 6
In addition to the dissecting process a coronary leaflet has been disrupted. Following the restoration of a normal channel the cusp was found to be irreparable. A silicone rubber leaflet is sutured into position to replace the deficient cusp.

Five patients with dissecting aneurysms and aortic valve leaflet involvement have now been surgically corrected by direct procedures on the aortic root. All of the patients had a complete correction of the dissecting process and aortic insufficiency. Blood pressures in all were normal immediately after the correction. One patient did not survive the postoperative period. Four patients have done well and have maintained their status of cardiac compensation for a period up to 18 months following operation. All have shown a decrease in cardiac size and have continued to show normal blood pressures. There have been no signs or symptoms of recurrence of dissection. The aortic root has remained small in all. The primary intimal tear in the ascending aorta is most frequently of a transverse variety and may involve the entire circumference or only part of the circumference. The formation of the second channel may proceed proximally, in which case the suspension of the cusp or cusps at the commissure is disturbed, thus allowing the cusp to tilt forward and become incompetent (fig. 3). These patients require a complete restoration of the integrity of the suspension of the leaflets (fig. 4). It is in this group that tears of the cusps are also occasionally observed, and these rents may be primarily repaired or prostheses may be required to complete the repair (fig. 5). Secondary involvement of the aortic leaflets frequently occurs in dissections originating low in the root and can be recognized and repaired at the time of the direct approach. In these situations, the incompetence may be produced by detachment of one or more cusps from their attachment to the aortic wall, or a cusp may be directly torn in the course of the dissection (fig. 6). When the dissecting process extends distally, bleeding into the pericardium by sudden or gradual rupture of the aneurysmal mass may lead to death, or the process may continue to form a double channel distal to the ascending aorta. In other cases where the dissecting process originates well above the leaflets and extends distally, the cusps themselves are uninvolved per se; however, dilatation of the ascending aorta occurs and cusps become insufficient during diastole, because of failure of coaptation. Di-
The concept of direct repair of dissecting aneurysm of the aorta has been proposed. The complete repair of the dissection and concomitant repair of aortic insufficiency have been discussed.

References
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