# Excision of Aneurysm of the Ascending Aorta with Prosthetic Replacement During Cardiopulmonary Bypass *

HENRY T. BAHNSON, M.D., FRANK C. SPENCER, M.D.**

*From the Department of Surgery, The Johns Hopkins Hospital and University*

THE ascending aorta is a common site for the development of aneurysms, but it is also an area which, though easily accessible, is most difficult to treat by excision and replacement, the preferred treatment of fusiform aneurysms. One type of fusiform aneurysm which is frequently localized to the ascending aorta is that due to cystic medial necrosis. An earlier report described our experiences in treating four patients with this condition and commented upon the specificity and localization of these lesions.[1] Since that report we have treated four patients with excision and replacement of the ascending aorta, a method which appears to be significantly better than partial excision and wrapping with a Nylon binder as described earlier. In addition to describing our experiences with what appears to be a better technic we wish to emphasize again the clinical characteristics of this entity, a localized and remediable type of aortic disease.

## Case Reports

**Case 1.** B. L. (JHH 806202), a 35-year-old white steel worker was hospitalized because of lack of energy and twenty-pound weight loss extending over a two-year period, associated with a cardiac murmur and fever. One year before he had had a severe pain in the anterior chest which caused faintness. He recalled five physical examinations prior to this time which he assumed were normal, but following the pain a cardiac murmur was heard on the next examination and his job was changed to a less strenuous one. For the past two weeks he had felt ill with a dull ache in both shoulders and anterior chest, especially noted with deep inspiration.

He recalled sore and swollen joints and being unable to walk between the age of six and eight years. He had been treated for pneumonia and gonorrhea in the past.

The blood pressure was 125/60 mm. Hg. There was no arachnodactyly, and careful ocular examination showed no abnormalities. The heart was enlarged to the anterior axillary line. Loud systolic and diastolic murmurs were heard along the right and left sternal borders. The second sound to the right of the sternum was loud. Peripheral veins were not distended, and there were no signs of cardiac failure.

Serological tests for syphilis and a Reiter Treponema complement fixation test were negative. Chemical studies of the blood were normal except for a two plus cephalin flocculation.

Roentgenograms showed a rounded fullness of the ascending aorta and enlargement of the left ventricle (Fig. 1a). Dilatation of the aorta seemed to stop in the aortic arch as the aortic knob in the upper left silhouette was small. An angiocardiogram confirmed the presence of the localized dilatation (Fig. 1b). A roentgenogram of the chest taken in 1952 showed little if any enlargement of the ascending aorta and a normal cardiac shadow.

At operation on May 14, 1958, the aneurysm extended from the level of the coronary arteries to the origin of the innominate artery. The aortic arch itself appeared to be normal and just proximal to the coronary arteries the aorta tapered strikingly to a nearly normal size. There was a shaggy fibrinous pericarditis, thought to be approximately a month old. The aneurysm was excised during 58 minutes of cardiopulmonary bypass; 14 minutes of anoxic cardiac arrest were used. In the excised aneurysm a dissection extended from the coronary arteries to the upper

* Presented before the Southern Surgical Association, Hot Springs, Virginia, December 8–10, 1959.

** John and Mary R. Markle Scholar.

Aided by Grant H-1326 from the National Heart Institute, U. S. Public Health Service, Bethesda, Md.

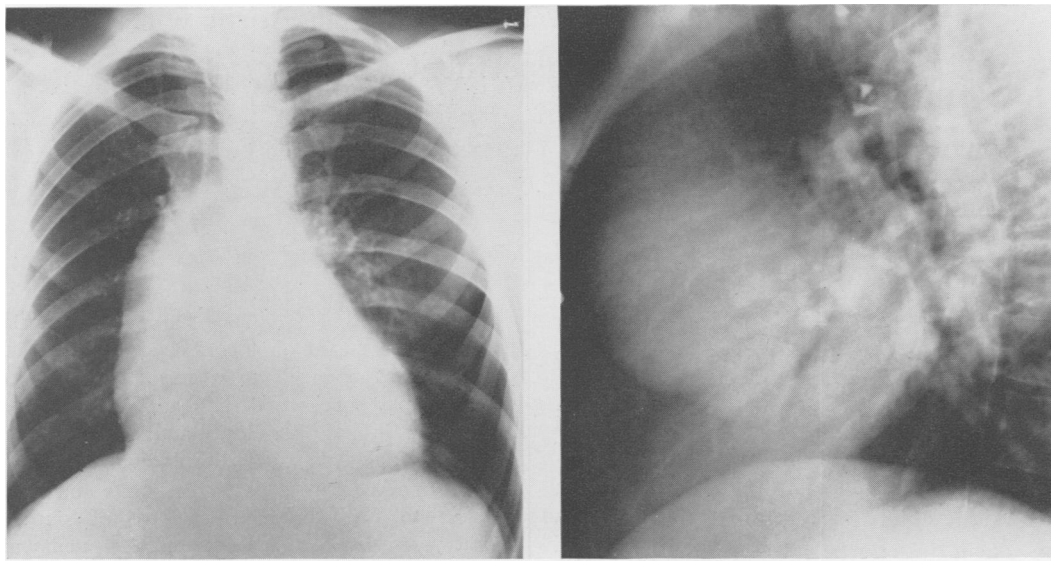

FIG. 1. Case 1. 35-year-old man with chest pain, fever, weight loss, recently noted cardiac murmur. (a) (left) Roentgenogram showing enlargement of ascending aorta with prominent left ventricle. (b) (right) Lateral angiocardiogram demonstrating aneurysm arising just above the sinus of Valsalva. Note normal size of valve ring and sinuses of Valsalva.

portion of the ascending aorta near the line of transection. The aorta was reconstructed with a knitted dacron arterial prosthesis 3 centimeters in diameter. The proximal anastomosis was immediately adjacent to the left coronary artery, some sutures entering the mouth of this artery. Although the prosthesis had been preclotted there was considerable loss of blood through it over the subsequent three hours in spite of neutralization of heparin with protamine and pressure on the area. The patient tolerated the nine-hour and 40-minute procedure satisfactorily but bled an additional 2,700 ml. in the early postoperative period. Histological study of the excised specimen showed striking cystic medial necrosis with fusiform and dissecting aneurysm.

He was discharged on his fourteenth postoperative day but returned five days later with epigastric pain and a gain in weight. Ankle edema, massive proteinuria, and hypo-albuminemia were present. No cause for the apparent nephrotic syndrome could be determined. While in the hospital he lost twenty pounds of weight and had some decrease in his proteinuria on a low salt and high protein diet. Five months after operation the urine showed no albumen and the excretion of phenolsulfonphthalein was 42 per cent in two hours. Grade II systolic and diastolic murmurs were heard adjacent to the sternum. His blood pressure was 142/70 mm. Hg. He was similarly well eighteen months after operation.

**Case 2.** B. B. (JHH 822151), a 23-year-old law student, was admitted for evaluation of a cardiac murmur which had been found in his early childhood. He had no cardiorespiratory symptoms; his life had been active and unrestricted. Dilatation of the ascending aorta had been first noted in 1954 and had increased subsequently. On examination the heart was of normal size with a normal apical beat. There was a thrill over the base of the heart with a rough systolic murmur transmitted into the neck. The second sound on the right of the lower sternum was accentuated.

Roentgenograms and angiocardiograms showed dilatation of the ascending aorta without cardiac enlargement (Fig. 2a, b). Calcium was seen in the region of the aortic annulus. Electrocardiogram was normal. Systolic pressure in the left ventricle was 50 mm. Hg higher than that in the femoral artery on direct puncture of both regions. It was believed that the aortic stenosis was not a threat to him but the dilatation of the aorta was.

At operation on November 28, 1958, a fusiform aneurysm ten cm. in diameter occupied the ascending aorta. Proximal to it, just above the sinuses of Valsalva, the aortic diameter was three cm. and just proximal to the innominate artery it measured two cm. The ascending aorta was excised and replaced with crimped woven Teflon, 25 mm. in diameter. The aorta at either end of the excision was grossly normal. The proximal anastomosis was at the highest point of insertion of the aortic valve cusps and three to four mm. above the orifice of the left coronary artery. The left coronary artery was perfused while the

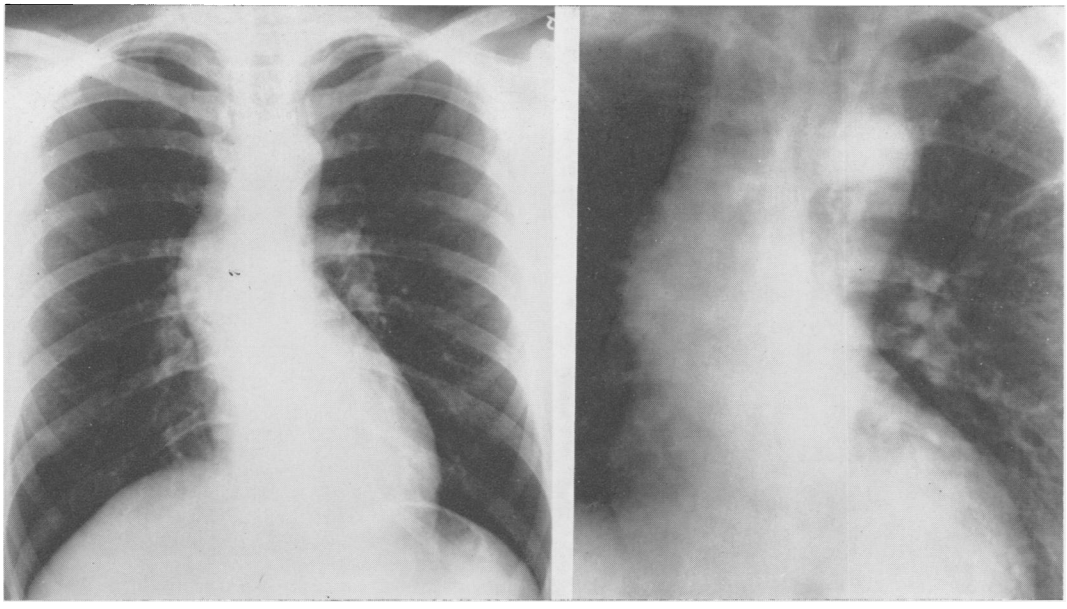

FIG. 2. Case 2. 23-year-old man, asymptomatic. Studied because of murmur and dilated aorta. (a) (left) Roentgenogram showing large ascending aorta but normal heart. (b) (right) Angiocardiogram with dilated ascending aorta but normal valve and aortic arch.

proximal anastomosis was completed. The aortic valve orifice was narrowed because of fusion of the commissures between the right and left coronary cusps and between the right and non-coronary cusps. Both of these were opened for about five mm. and a large opening thus obtained. Direct measurement of pressure showed no gradient across the valve after the repair. Histologic examination of the excised specimen showed typical cystic medial necrosis.

He returned four months later for re-evaluation and removal of troublesome wire sutures in the sternum. He had been asymptomatic. There was a grade II systolic murmur in the right second interspace transmitted to the neck. Pulsations in the right side of the neck were more bounding than on the left, and systolic blood pressure in the right arm was 30 to 40 mm. Hg higher than on the left. By direct puncture arterial pressure was 173/75 mm. Hg on the right, 126/78 mm. Hg on the left, the systolic upstroke being 1,900 and 680 mm. Hg per second respectively. No complete explanation for this discrepancy was found. Roentgenogram of the chest was essentially normal with flattening of the area usually filled by the ascending aorta (Fig. 2c).

**Case 3.** B. S. (JHH 840374), a 24-year-old white housewife, was admitted for treatment of an aneurysm of the ascending aorta known to be present for three years. Three years before, three months following delivery of her second normal child following a normal pregnancy, she noted shortness of breath and a precordial and scapular boring pain associated with exertion, relieved by rest and nitrites. Six months before admission she had been admitted to the Walter Reed Army Hospital and retrograde aortography showed an aneurysmal dilatation of the entire ascending aorta and aortic arch (Fig. 3a, b). Three months before admission she had a tingling sensation in the left arm and pain over the course of the carotid arteries. During the past three and one-half years she had noted fatigue and weakness with exertion, neither being incapacitating.

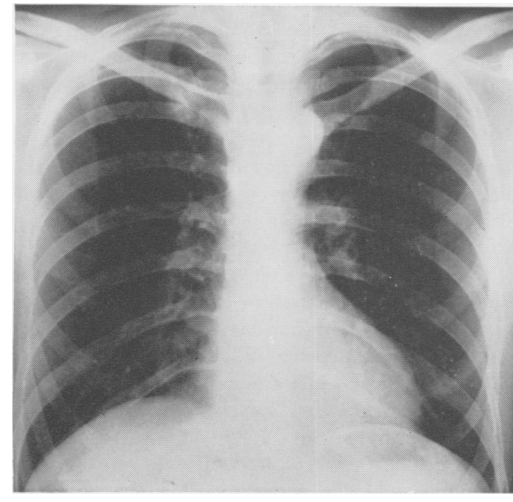

FIG. 2. (c) Postoperative roentgenogram.

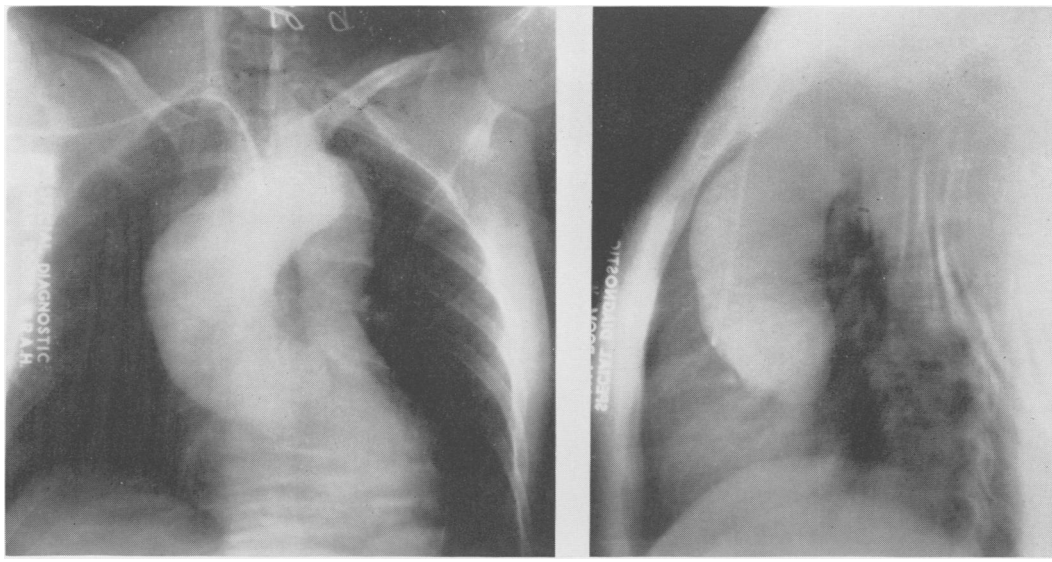

FIG. 3. Case 3. 24-year-old woman with chest pain, some shortness of breath. (a and b) Aortograms showing dilated aorta. The dilatation is not as well localized as in other cases, extends into the arch and beyond.

On examination she was tall but without arachnodactyly. The upper segment of the body measured 87 centimeters, the lower (from symphysis down) 90 centimeters. The eyes were normal. Blood pressure was 130/50 mm. Hg in the right arm, 135/60 in the left. Arterial pulsations were seen in the fundus of the eye and pulsations were prominent throughout the body. There was a forceful apical impulse just outside the midclavicular line. The first sound at the apex was split, thought to be due to an ejection click. The second sound was louder to the right than to the left of the sternum. A grade III diastolic blowing murmur was audible, loudest over the right sternal border but also heard over the apex and the back. A grade II harsh systolic murmur was heard over the right second to third interspace and transmitted to the carotid vessels and to the apex.

Roentgenograms showed an aneurysmal dilatation of the ascending aorta with a tortuous and dilated descending thoracic portion of the aorta (Fig. 3c). The heart was not enlarged. Serological tests for syphilis were negative.

At operation on March 3, 1959, the aorta was dilated to 10 centimeters in the midportion of the ascending aorta and narrowed to 4.5 centimeters at the level of the coronary arteries. The diameter at the level of the innominate artery was about 5 centimeters. The ascending aorta was excised and replaced with woven Teflon 3 centimeters in diameter. The coronary arteries were perfused for 30 minutes during completion of the proximal anastomosis. The heart began to fibrillate after about ten minutes of coronary perfusion but was easily reverted with a single electrical shock when the anastomoses were finished. The aortic valve cusps were normal, although the ring was somewhat dilated. The prosthesis was smaller than the aorta at the proximal end so that some gathering of the aorta was necessary. A prosthesis smaller than the patient's aorta was purposely used in order to reduce the size of the aortic valve ring for its beneficial effect on aortic insufficiency.

Her convalescence was uncomplicated and she was discharged eighteen days after operation. The diastolic murmur heard preoperatively was no longer audible although there was a systolic murmur at the right and left second and third interspaces. Blood pressure was 120/60 mm. Hg. Roentgenograms showed no dilatation of the ascending aorta but the dilatation of the aortic arch persisted (Fig. 3d).

Histological examination showed no evidence of cystic medial necrosis but a marked medial and adventitial scarring. There was extensive destruction of the media with areas of necrosis and numerous giant cells. The changes were morphologically indistinguishable from those due to syphilis.

**Case 4.** L. B. (JHH 857002), a 38-year-old carpenter, had been well until three months before admission when he suddenly experienced sharp, stabbing pain high in the anterior midchest, extending up into the anterior neck. This lasted about a half hour and was gone in eight to ten hours. At an outside hospital it was thought that the electrocardiogram showed evi-

dence of pericarditis. At this time and subsequently he had occasional spells of dizziness. On examination there was a visible pulsation in the second interspace at the right sternal border where a sharp thrill was felt and a harsh high-pitched systolic murmur was heard. The murmur was audible into the neck. Over the base it had a lower pitched musical quality. Pulsation of the right carotid was decreased when compared with the left but other pulses were equal. The electrocardiogram showed nonspecific changes in the T waves. Roentgenogram showed dilatation of the ascending aorta without significant left ventricular enlargement. A dilatation fairly well localized to the ascending aorta was demonstrated by angiocardiography.

On August 26, 1959, the aneurysm was excised and replaced with a Teflon prosthesis. There was a diffuse fibrinous pericarditis. When the aorta was transected it was evident that there was a dissection which began just above the aortic valve and extended into the aortic arch beyond the point of aortic division. The aorta was occluded distal to the innominate artery and it could be seen that dissection extended up along the innominate also. The distal anastomosis was to the outer circumference of the aorta. Both coronary arteries were perfused while the proximal anastomosis was made. Histological examination showed cystic medial necrosis.

A tracheotomy was performed in the early postoperative period because of respiratory embarrassment and his recovery was rapid after this. Ten days after operation, while walking around the ward, he had abrupt onset of dyspnea, caused by a large pulmonary embolus from which he died ten hours later.

Autopsy showed extensive thrombosis of the inferior vena cava and both iliac veins. Emboli were present in both right and left pulmonary arteries. The aortic dissection extended to the distal end of the aortic arch. Histologically slight evidence of cystic medial necrosis was present in the thoracic and abdominal aorta.

## Clinical Picture

In the table are listed data from these four cases (Cases 1 to 4) along with the four cases described previously (Cases 5 to 8) in order to emphasize the features of this disorder. Ages range from 24 to 49. The oldest patient had been aware of the condition and had had cardiac enlargement beginning at 42 years. Young men are predominantly affected as only one of the patients was a woman. In this patient the disease was less well localized to the ascending aorta, and the histological examination showed fewer of the changes of cystic medial necrosis than in the other cases, the sections more nearly resembling the chronic aortitis of syphilis, although it is believed the latter was not present.

Pain has been a prominent feature in most cases. In most instances there is an abrupt onset of precordial pain suggesting

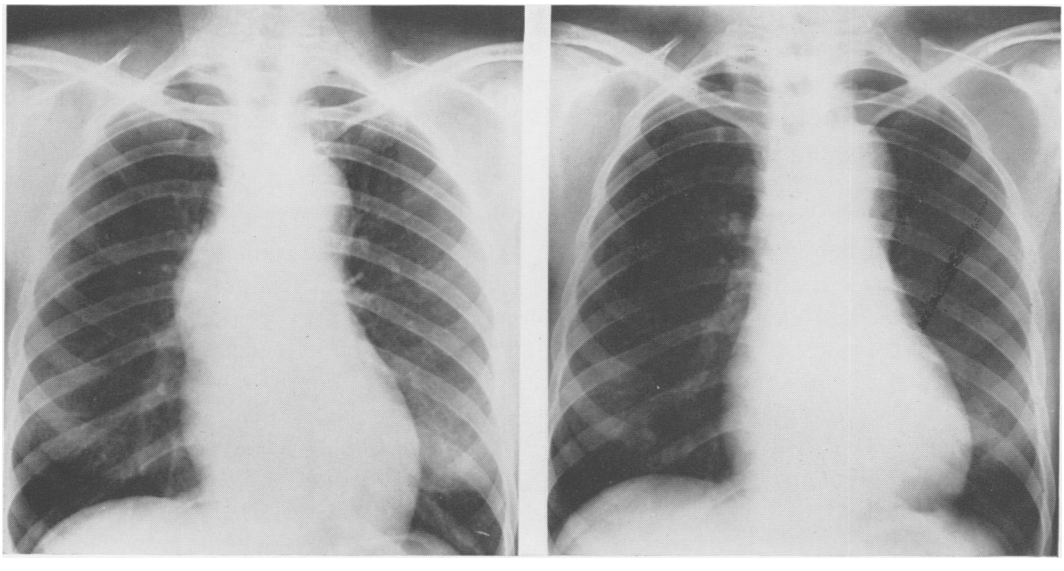

FIG. 3. (c) (left) Preoperative roentgenogram. (d) (right) Postoperative roentgenogram.

TABLE 1. *Cases 1–8*

| Case | Age | Sex | Symptoms | Aortic Valve | Operation | Dissection | Result |
|---|---|---|---|---|---|---|---|
| 1. B. L. | 35 | M | Fatigue, precordial pain | Incompetent | Excision and replacement | Ascending aorta | Good |
| 2. B. B. | 24 | M | None | Congenital stenosis, slight | Excision and replacement | None | Good |
| 3. B. S. | 24 | F | Precordial pain and dyspnea | Incompetent | Excision and replacement | None | Good |
| 4. L. B. | 38 | M | Pain in anterior chest and neck | Slight incompetence | Excision and replacement | Into arch and its branches | Died 2 weeks later of pulmonary embolus |
| 5. S. L. | 39 | M | Congestive heart failure | Badly incompetent | Partial excision and wrapping | None | Satisfactory until death 5 mo. later with myocardial infarct |
| 6. S. T. | 41 | M | Substernal pain | Incompetent | Partial excision and wrapping | None | Improved. Sudden death 4 mo. later. No autopsy |
| 7. A. H. | 31 | M | Substernal pain | Stenosis, prob. congenital; incompetent | Partial excision and wrapping | Ascending aorta | Good |
| 8. T. M. | 49 | M | Congestive failure. Precordial oppression | Calcific aortic stenosis | Attempted partial excision | Ascending aorta and arch | Died during operation |

acute pericarditis, in some instances associated with confirmatory electrocardiographic changes. The abrupt onset of severe pain, on the other hand, in some cases suggested an aortic dissection. Either an old fibrinous pericarditis or aortic dissection has been found at operation in all but one case; we believe this patient was operated upon earlier in the course of his disease than the others.

The aortic valve has been incompetent or stenotic in all patients operated upon. In those with aortic insufficiency without stenosis in whom the aortic valve has been visualized the leaflets were normal but the incompetence was due to dilatation of the valve ring. The truth of this observation is supported by relief of the insufficiency by constriction of the aorta just above the valve either by wrapping or by excision and replacement with a prosthesis. Aortic stenosis was present in three of the eight cases and appeared to be of a congenital type with fusion of the commissure between the right and left coronary cusps.[5]

The left ventricle has been enlarged in all instances, and in two patients progressive congestive heart failure was present. The failure appeared to result from aortic insufficiency. In one instance, Case 5, aortic insufficiency and cardiac failure were strikingly relieved by partial excision and wrapping of the ascending aorta.

In all instances except Case 3 the histological changes described by Gsell in 1928 and Erdheim in 1929 were present. These consist of necrosis and disappearance of muscle cells in the elastic laminae in the media. When the elastic tissue is affected mucoid material is seen to fill cystic spaces. There is little inflammatory reaction and no evidence of reparative process except for collagen deposition in the outer portion of the media.

It is important to emphasize that none of these patients had other stigmata of Marfan's syndrome, although cystic medial necrosis is one of the lesions of this syndrome. The only patient in whom arachnodactyly was suggested was the third

case, the woman who in other respects seems less like the seven male patients. As illustrated earlier the dilatation of the aorta in Marfan's syndrome extends into the sinuses of Valsalva themselves, in striking contrast to the lesion under discussion.[1]

Observations in these eight patients confirm Rottino's earlier study that the aortic abnormality is often localized to the ascending aorta, and for this reason it is potentially amenable to excision and replacement, although this may be part of a generalized disease, the etiology of which is unknown, as suggested by the finding in Case 8 of similar less severe changes in the peripheral vessels. When aortic dissection is present it has been limited to the ascending aorta except in two patients. In these cases the arch was also involved.

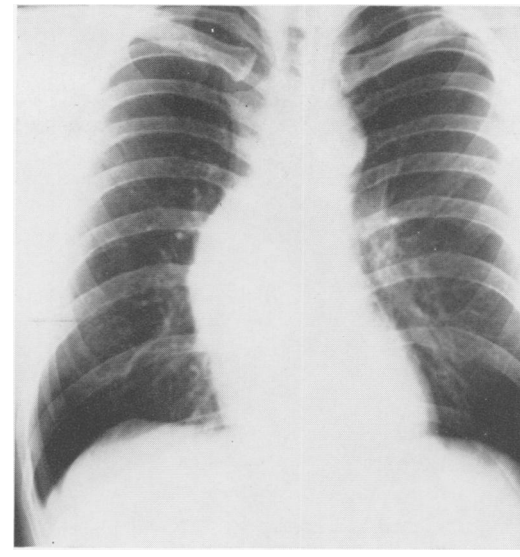

FIG. 4. Case 4. 38-year-old carpenter with pain in anterior chest and neck three months previously. (a) Preoperative roentgenogram showing dilated ascending aorta, limited to this region and proximal arch as seen on angiocardiograms (b and c).

## Surgical Considerations

Cardiopulmonary bypass has allowed excision and replacement of the ascending aorta,[2] treatment which seems to be significantly better than that described earlier for the condition under discussion. The method of cannulation and use of the bypass is illustrated in Fig. 5a. We usually use a single cannula in the right atrium for removal of venous blood but one has better control of the venous return with individual cannulation of the two venae cavae. By the former means and siphon drainage the right heart can be kept fairly empty of blood but the central venous pressure must be kept low. A catheter has been placed in the left atrium through the appendage after the bypass is started and attached to gentle

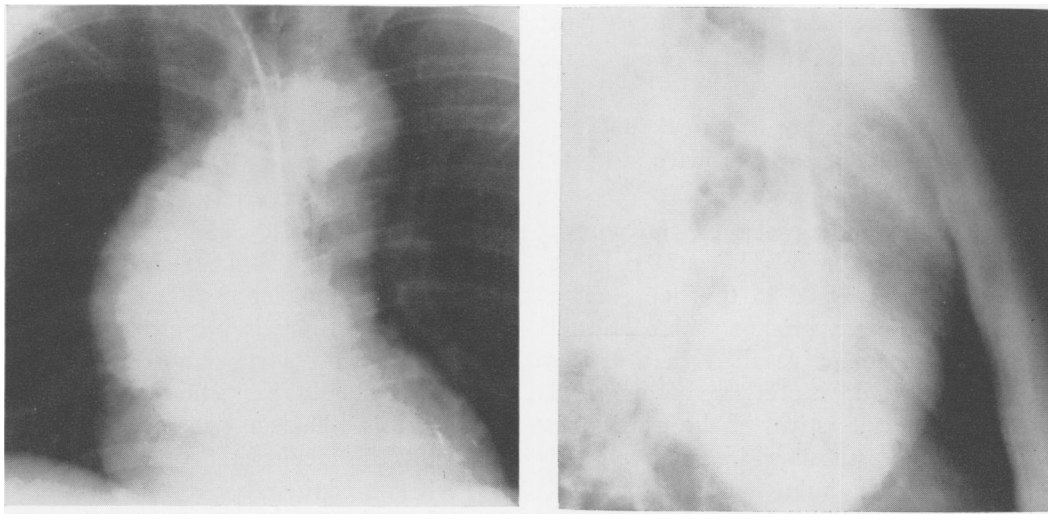

FIG. 4 b, c. The left ventricle was not enlarged significantly. Dissection was found, extending through the arch to the origin of the left subclavian.

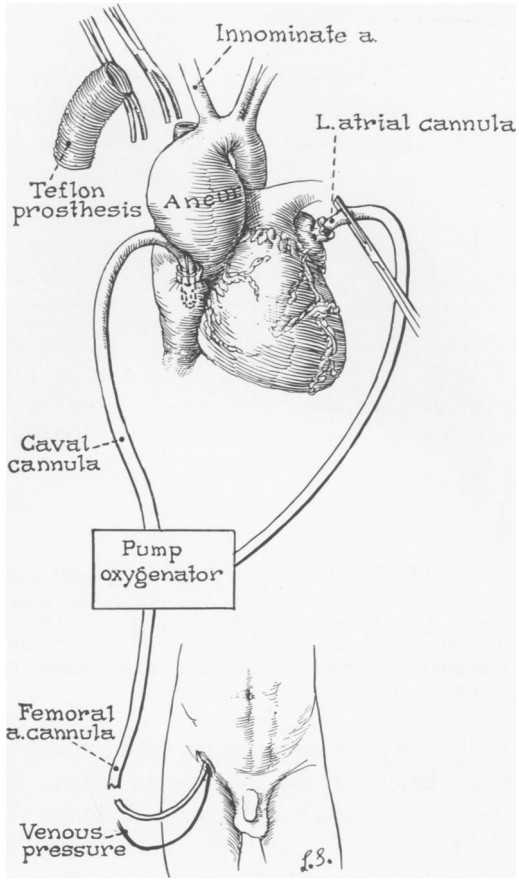


FIG. 5. (a) Plan of bypass.

suction from the bypass apparatus. This is later used to remove blood from the left side of the heart. The femoral artery has been used for arterial return.

Median sternotomy has given good exposure and is probably the ideal incision, possibly except in patients with an unusually large ascending aorta.

We have strongly preferred the woven Teflon prosthesis, largely because there is less tendency to bleed in the heparinized patient. Our one experience with a knitted Dacron replacement resulted in a trying three-hour period awaiting hemostasis and clotting in the interstices of the prosthesis, although the prosthesis had been preclotted before heparin administration. Considerably less bleeding occurs through the woven Teflon.

After the bypass is started the aorta just proximal to the innominate is clamped and the aorta just proximal to this also occluded, sufficient space between being left for anastomosis to the distal cuff after division. A small amount of blood is allowed to return to the right ventricle and thence to the left so that the heart continues to beat and perfuses the coronaries while the distal anastomosis is completed.

One must watch rather carefully the pressure in the ascending aorta at this time, a flabby aorta indicating too complete removal of venous return through the bypass and too high a pressure indicating insufficient removal. In Case 1, insufficient attention was paid to this and the ascending aorta became flabby, following which the heart arrested.

When the distal anastomosis is completed the left atrial cannula is opened and the left atrium kept empty. The aorta is divided just above the coronary arteries and the coronaries perfused. We have tried various methods of perfusion, the best to date being insertion of small plastic catheters with circular balloon tips which are held in place by purse string sutures around the orifice. We have often perfused only the left coronary artery (in treating patients with aortic stenosis) but observation of these patients and particularly their electrocardiograms indicates both arteries should be perfused. This might be intermittent and alternating.

When the aorta is divided just above the coronaries the heart drops away from the operator, a rather harrowing experience on first sight. The prosthesis should be sutured in place then with slight tension to prevent insertion of too long a replacement. If aortic insufficiency is present a prosthesis smaller than the aorta should be used and the aorta should be gathered during the anastomosis. This has been effective in reducing the incompetence.

Perfusion of the coronary arteries is continued until the anastomosis is nearly completed and the cannulae are then removed.

Anoxic cardiac arrest was unintentionally produced in Case 1 but no difficulty was apparent with 14 minutes of arrest. As the proximal anastomosis is completed the left atrial cannula is clamped and the heart allowed to fill with blood. If ventricular fibrillation or poor cardiac action is present after circulation is restored the left atrial suction is again used as the left side of the heart must not become distended.

## Prognosis

The progress of patients in this group can be viewed in retrospect by roentgenograms for periods from two to ten years. During this time the ascending aorta progressively enlarged in all instances. The progressive enlargement, the development of aortic insufficiency, of aortic dissection, and the progressive and severe cardiac failure which occurred in the two patients with the longest duration of untreated disease give convincing evidence that without treatment the condition is a dangerous one. Our initial therapeutic ardor was somewhat dampened by the death of the first two patients, one surely of myocardial infarction (Case 5), and the other suddenly but without autopsy confirmation of the cause of death (Case 6). The continued good health three years after operation of Case 7 and the belief that total excision and replacement will give equally good or better results lead us to the conclusion that operation is indicated when the diagnosis is made. Although this is not urgent with early dilatation of the aorta, dissection or aortic insufficiency or both are almost certain to occur with greater enlargement. The only operative death in the eight patients was the one in whom cardiac failure was severe and of long duration. We do not have sufficient follow up of patients with replacement of the ascending aorta to know the future of the prosthetic material, but experimental observations attest to the retained strength of Teflon over considerable lengths of time.

## Summary

Four cases have been described in which the ascending aorta was successfully ex-

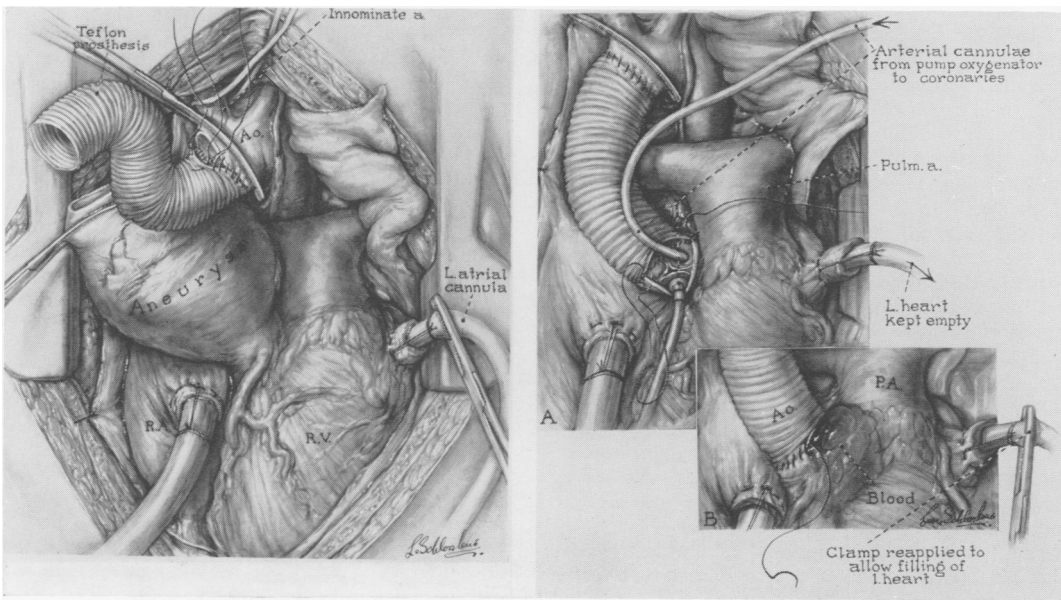


FIG. 5 b. (left) During the distal anastomosis venous blood is not completely removed from the right atrium and the left atrial suction is used only to prevent excessive pressure in the aneurysm. The coronary circulation is thus maintained intact. (c) (right) After the aneurysm is excised all blood is aspirated from both sides of the heart and the coronary arteries are individually perfused while the proximal anastomosis is completed.

cised and replaced with prosthetic material. Aneurysms due to cystic medial necrosis occur selectively in the ascending aorta, occur predominantly in young men, are usually well localized, often are associated with aortic insufficiency, congenital aortic stenosis, or both, and such aneurysms can be treated surgically. Operation is indicated in most instances to remove the threat of the enlarging aneurysm and to treat aortic insufficiency which is frequently present.

## References

1. Bahnson, H. T. and A. R. Nelson: Cystic Medial Necrosis as a Cause of Localized Aortic Aneurysms Amenable to Surgical Treatment. Ann. Surg., **144**:519, 1956.
2. Cooley, D. A. and M. E. DeBakey: Resection of Entire Ascending Aorta in Fusiform Aneurysm Using Cardiac Bypass. J. A. M. A., **162**:1158, 1956.
3. Erdheim, J.: Medionecrosis Aortae Idiopathica. Virchows Arch., **273**:454, 1929.
4. Gsell, O.: Wandnekrosen der Aorta als selbstandige Erkrankung und ihre Beziehung zur Spontanruptur. Virchows Arch., **270**:1, 1928.
5. McKusick, V. A., R. B. Logue and H. T. Bahnson: Association of Aortic Valvular Disease and Cystic Medial Necrosis of the Ascending Aorta. Report of Four Instances. Circulation, **16**:188, 1957.
6. Rottino, A.: Medial Degeneration, Cystic Variety, in Unruptured Aortas. Ann. Heart J., **19**:330, 1940.

---

### Discussion

Dr. W. Sterling Edwards: I have very much enjoyed these two papers and would like to discuss Dr. Scott's presentation. I would agree that the ideal arterial prosthesis is not yet available. However, as Dr. Bahnson and Dr. DeBakey have demonstrated, synthetic prostheses function very satisfactorily in the chest and abdomen. Long grafts in extremities, especially the legs, have been less satisfactory. In 125 synthetic femoral and popliteal bypass grafts for obliterative arteriosclerosis 48 per cent have occluded within three years of insertion. We have diligently studied all patients, both successes and failures, to determine the cause of these late occlusions. Patients with successful long grafts have been studied by serial arteriograms at yearly intervals. An important adjunct of this phase has been visualization of the graft first with the leg straight, then with the knee and hip flexed, (Slide) This slide shows such a study in a patient with a crimped Teflon graft from common femoral to popliteal below the knee 18 months after insertion. The graft appears smooth, without constrictions in the straight leg film. After the second injection with the knee bent one can see buckling of the graft along its entire length since the tube cannot shorten as can an elastic artery. When we developed the crimping principle five years ago, it was hoped that this would allow plastic tubes to remain flexible and able to lengthen and shorten for many years. Unfortunately studies such as these x-rays demonstrate loss of flexibility and elasticity by all porous synthetic grafts after a few months due to fibrous tissue infiltration of the tube.

Another such bent-knee x-ray shows the graft narrowed to a ribbon like lumen behind the knee. This graft thrombosed 6 months later while the patient was squatting to lay a concrete floor.

In regrafting some of these patients with graft occlusion, examination of the old anastomoses have been instructive (Slide). Flexion of the hip or knee will often cause the stiff synthetic tube to invaginate into the adjacent artery, or angulate the anastomosis and partially occlude the lumen.

Results with grafts that terminate above the knee have been almost as poor as those that cross the knee joint. Here flexion of the hip frequently causes invagination of the upper end of the tube into the femoral artery.

These findings lead us to believe that the ideal peripheral graft has yet to be developed.

Dr. Frank Glenn: I rise to discuss very briefly Dr. Scott's presentation and I would like to say that I enjoyed the presentation of Dr. Bahnson very much and I think the coronary perfusion is most important in this type of case.

Some ten years ago when we were using blood vessel grafts Dr. Edward Kiefer in our laboratory took six litter mate pups and placed grafts from older dogs in the thoracic aorta. We have followed these animals very closely since. Dr. Kiefer, as a matter of fact, keeps them in his home.

These animals have developed normally; all were explored, at two years and at that time as had been found by x-ray, there was some calcification in the grafts in all of them. At four years one dog died as a result of a dog fight. There was marked calcium in this animal's graft.

Last year in December almost nine years after the grafts were inserted an animal died and the slide that I would like to show you is a