Cystic Medial Necrosis as a Cause of Localized Aortic Aneurysms Amenable to Surgical Treatment *

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WE WISH to recount recent experiences with five cases of cystic medionecrosis of the aorta treated surgically. This pathologic condition, eponymically called Erdheim's cystic medionecrosis, has been previously recognized as a cause of aortic weakness and has been demonstrated in four groups of patients: it is often present in cases of dissecting aneurysm and thought to be the principal cause of dissection in young individuals; it is commonly described as the cause of aortic weakness in cases of spontaneous rupture of the aorta; it is one of the characteristic features of Marfan's syndrome (with, in addition, arachnodactyly, dislocated lenses, and a generalized defect in ground substance); finally it is sometimes seen in slight degree in normal aortas. Our attention was strikingly focused on an additional manifestation of this disease, namely its production of localized aneurysms, by the first such patient we encountered whose case history is as follows:

Case 1. J.H.H. 293069. S. L., a 39-year-old Negro, was admitted in March, 1955, for treatment of congestive heart failure due to aortic insufficiency. He had been admitted to this hospital in 1943 with meningococcemia, treated with sulfamerazine, and again in 1945 with pneumococcal pneumonia. During these admissions his blood pressure was 115/60, and there were no cardiac murmurs. Serological tests for syphilis were negative. Roentgenograms of the chest showed no abnormality of the heart or aorta. Recovery was uneventful from each illness.

In 1947 during an examination for employment it was noted that his blood pressure was 115/74 and that he had a soft, aortic, diastolic murmur. Roentgenography at that time showed slight cardiac enlargement with some prominence of the left ventricle (Fig. 1, a). In 1951 his blood pressure was 140/60, a similar diastolic murmur was heard, and for the first time a loud, aortic, systolic murmur was noted. Chest roentgenogram showed definite enlargement of the proximal ascending aorta (Fig. 1, b). In 1952 he noted mild exertional dyspnea, and when the plant physician was consulted, he was told there was "a bad tube in his heart." In 1954 he developed nocturnal dyspnea, followed by frank pulmonary edema, and was admitted to the hospital for treatment of his cardiac failure. He responded nicely to the therapeutic regimen and mobilized 18 pounds of edema fluid. At that time he had both aortic systolic and diastolic murmurs, and a thrill was associated with the latter. Blood pressure was 128/40. On this and subsequent admissions his serological tests for syphilis and Treponema immobilization tests were negative. Electrocardiogram showed left axis deviation and abnormal T-waves. Roentgenologic studies showed a pulsatile aneurysm of the ascending aorta and left ventricular enlargement (Fig. 1, c). Symptoms of exertional dyspnea were never completely relieved in spite of a low sodium diet, digitalis, and diuretics.

One month before his final admission hospitalization was again required for treatment of severe heart failure and anginal pain. Examination showed no evidence of dislocated lenses and there was no arachnodactyly; no family history of Marfan's syndrome could be elicited. Blood pressure was 140/30. He was acutely ill and orthopneic. The cardiac apical impulse was diffuse and extended to the midaxillary line. There was a gallop but regular rhythm. An apical systolic murmur was transmitted to the axilla. A systolic murmur at the base was transmitted into the neck. A loud, high-pitched, decrescendo murmur was audible in diastole along the left sternal border and maximal in the third and fourth interspaces on the right where a thrill was palpable. The liver was not felt

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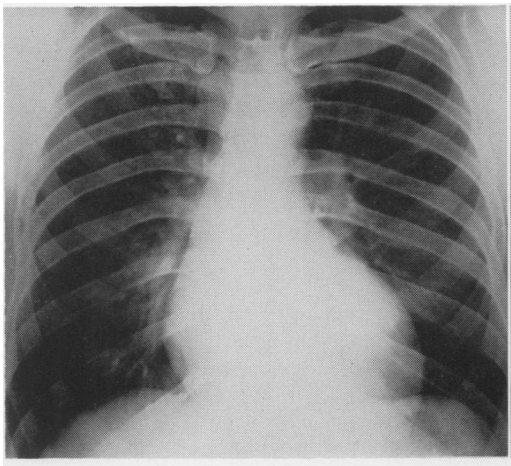

FIG. 1A.

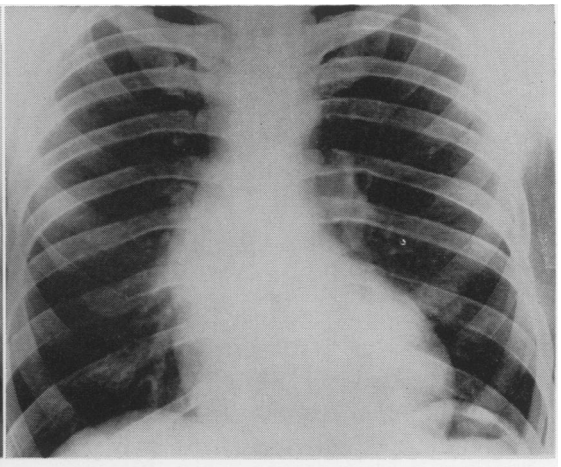

FIG. 1B.

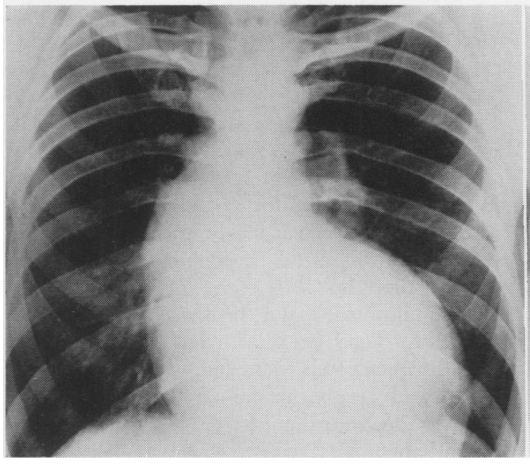

FIG. 1C.

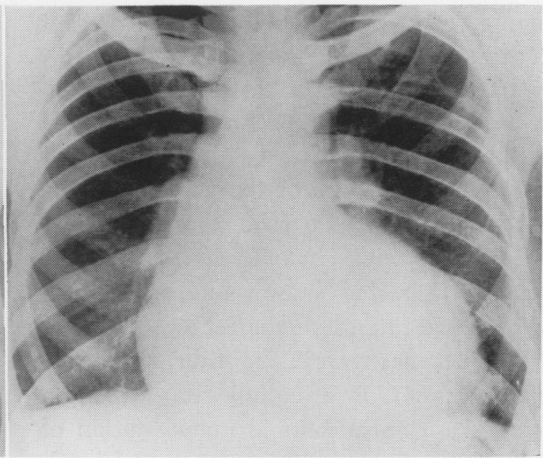

FIG. 1D.

FIG. 1. Case 1. A. Roentgenogram in 1947 showing slight prominence of left ventricle. B. Roentgenograms in 1951 showing beginning prominence of proximal ascending aorta. C. Preoperative roentgenogram showing aneurysm of ascending aorta and enlargement of left ventricle. D. Postoperative film showing reduction in ascending aorta. (Permission for reproduction of Fig. 1, A through D, has been obtained from the authors and editor, reference #7.)

and there was no peripheral edema. A diagnosis of medial necrosis of the aorta, possibly with a healed dissecting aneurysm, was made by Drs. W. E. Mattison and L. E. Cluff.[7] He responded poorly to treatment and was felt to be near the terminal stage of his disease. After losing 10 pounds with an induced diuresis, he was readmitted 2 weeks later for surgical treatment of aortic insufficiency.

A left posterolateral thoracotomy was performed on March 20, 1955 through the bed of the fifth rib, the initial plan being to insert a Hufnagel valve. When the ascending aorta was felt, a large aneurysm of the ascending aorta was noted which stopped abruptly just proximal to the origin of the innominate artery. Dilatation extended to the base of the heart, although at the base itself the dilatation appeared to be less marked and the diameter nearly normal. A diastolic thrill, felt over the heart, was readily obliterated by slightly constricting the first portion of the ascending aorta. Hence, the incision was extended anteriorly to the sternum and the ascending aorta mobilized. The aneurysm could be separated from the pulmonary artery with surprising ease. Dilatation was diffuse with none of the localized bosses usually seen with syphilitic or arteriosclerotic aneurysms. The right ventricle was separated from the aorta to the beginning of the sinuses of Valsalva. The coronary arteries were not visualized. A clamp was placed longitudinally across the mid-

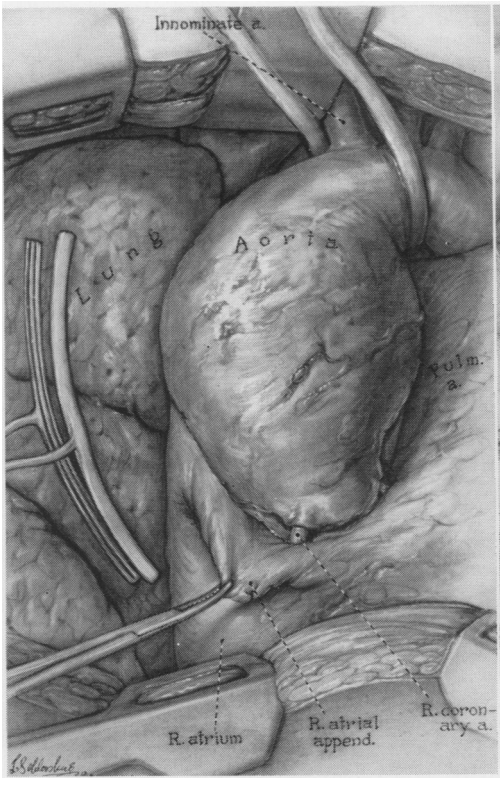

FIG. 1E.

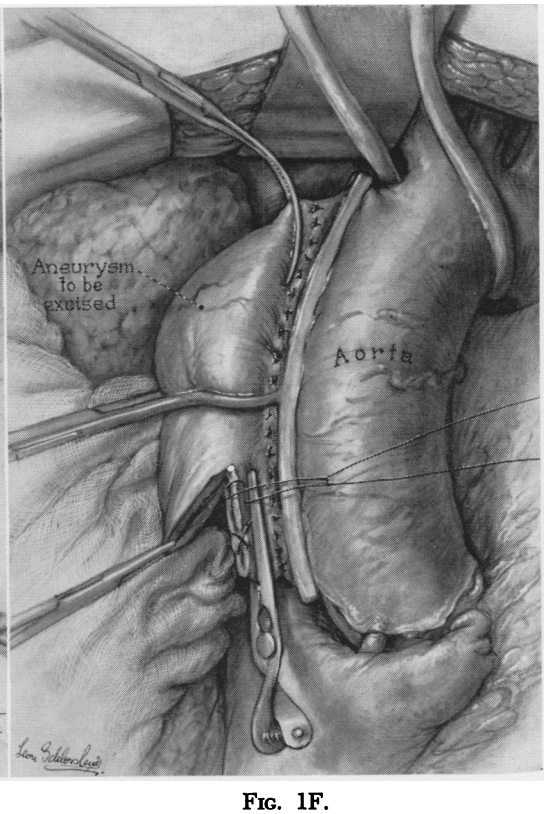

FIG. 1F.

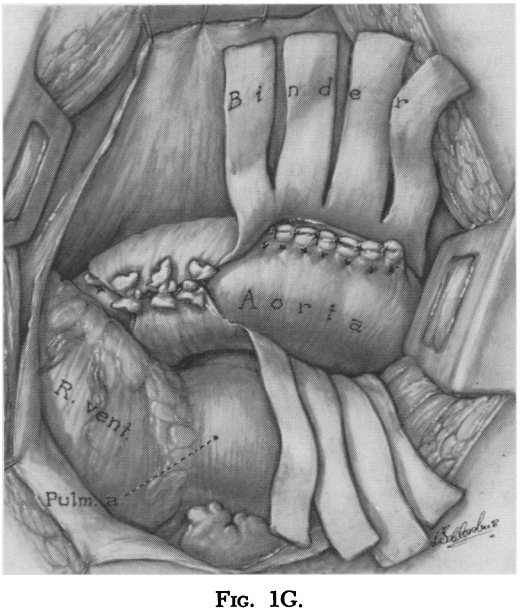

FIG. 1G.

FIG. 1. E. Operative exposure of ascending aorta as carried out in cases 2, 3, 4. In case 1 exposure was initially made for insertion of Hufnagel valve. The ascending aorta was readily dissected from the pulmonary artery and adjacent vena cava. Dilatation began just above the coronary arteries and stopped proximal to the innominate. F. The ascending aorta has been partially occluded with a special instrument * and approximately half the circumference excised. An additional portion of aorta may be later removed by exclusion with a Potts clamp, excision, and suture. G. Nylon Scultetus binder tied around the ascending aorta.

portion of the aneurysm, and about half the circumference was excised (Fig. 1, e). The closure was with two rows of 0000 silk, an inner row of mattress sutures reinforced with figures-of-eight (Fig. 1, f). An additional small bit of aorta was removed adjacent to the heart by grasping the aorta with a curved Potts coarctation clamp and excising the enclosed portion. When the excision was completed the closure extended to the base of the aorta which had been dissected out of the heart. The circumference of the entire ascending aorta was reduced from 30 to about 13 to 15 centimeters. The thrill over the heart had disappeared following reduction of the diameter of the aorta adjacent to the valve. Because it was felt that the entire circumference of the aorta was dis-

* Bahnson aortic clamp available through George P. Pilling & Son Co., 3451 Walnut St., Philadelphia, Pa.

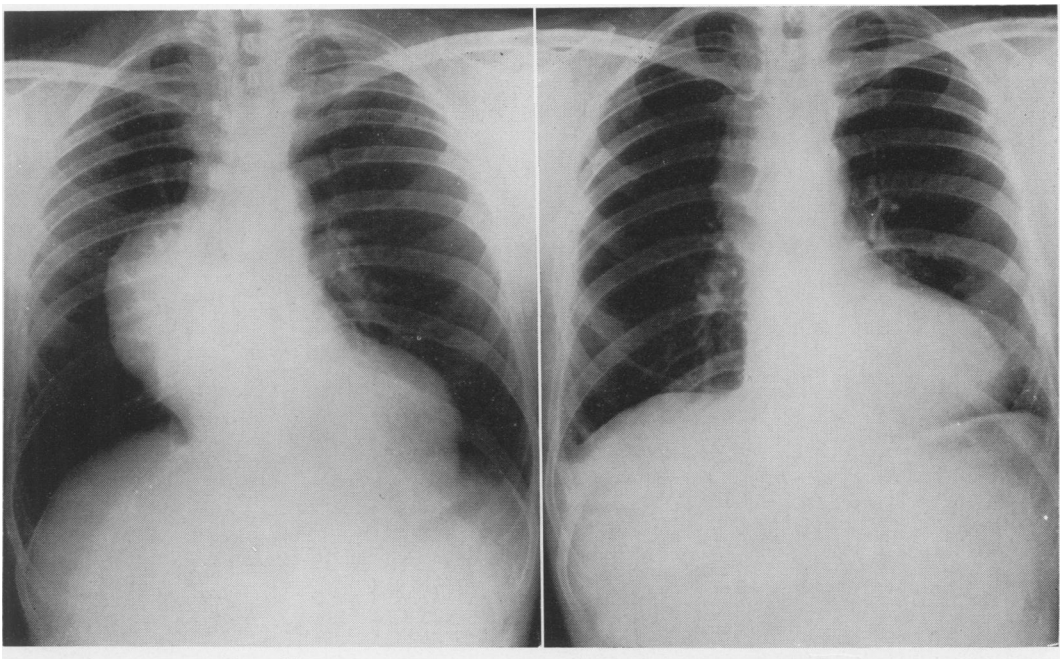

FIG. 2A. FIG. 2B.

FIG. 2. Case 2., A. Preoperative roentgenogram showing aneurysm of ascending aorta and enlarged left ventricle. B. Roentgenogram 2 months after operation.

eased, a nylon Scultetus binder, which had been previously fashioned, was tied around the aorta for support (Fig. 1, g). The excised specimen showed histological changes of cystic medionecrosis.

His postoperative course was uneventful and his blood pressure stabilized at 140/90. The diastolic murmur could no longer be heard along the right sternal border, although a soft, blowing, diastolic murmur appeared 2 months later. A soft systolic murmur at the base persisted postoperatively (Fig. 1, d). His improvement was considerable, and he was able to resume light work and was asymptomatic until 5 months after operation when he died suddenly.

Autopsy:* The repair of the aorta was intact, and there was no evidence of aortic dissection or hemorrhage. The heart weighed 900 Gms., due almost entirely to hypertrophy of the left ventricle. The cusps of the aortic valve were normal. The coronary arteries were patent but there was a small healing subendocardial infarction of the left ventricle. Histologic sections of the ascending aorta were similar to those taken at operation and showed changes of cystic medionecrosis. The transverse portion of the aortic arch was grossly and histologically normal. Examination of the brain was not obtained.

* We are indebted to the Baltimore City medical examiners for this information and tissue specimens.

In summary, a 39-year-old male Negro with a progressively enlarging fusiform aneurysm of the ascending aorta had been followed over an eight year period. Chronic congestive heart failure occurred which appeared to be rapidly and irreversibly progressive. In the absence of syphilis and infection, a diagnosis of cystic medionecrosis was made and this was confirmed by examination of the surgical specimen. Approximately half the aneurysm of the ascending aorta was excised and the remaining vessel wrapped with a nylon binder. Slight constriction of the first portion of the aorta apparently relieved the previously incapacitating aortic insufficiency. Initially improved, he died suddenly five months later, his death being attributed to a myocardial infarction, although the visible infarct was a healing one.

In the past year we have seen three additional patients with similar involvement of the ascending aorta and one of the distal aortic arch, all with histological evidence of cystic medionecrosis.

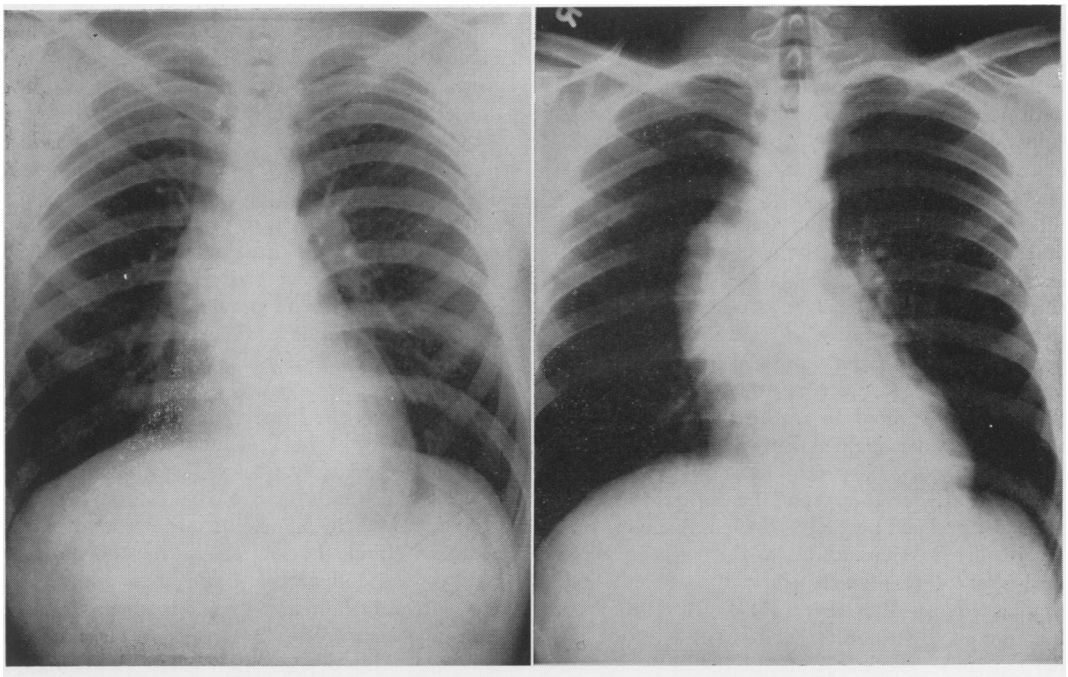

FIG. 3A.

FIG. 3B.

FIG. 3. Case 3., A. Essentially normal chest film in 1953. B. Roentgenogram in 1955 showing scending aortic aneurysm and enlarged left ventricle.

Case 2. J.H.H. 691351. S. T., a 41-year-old man, had been essentially free of symptoms. When discharged from the Army in 1946 his chest roentgenogram was normal. In 1949 he was told he had a small thoracic aortic aneurysm. Shortly thereafter he had a dark field positive, penile, syphilitic lesion and was treated with 51,000,000 units of penicillin. In 1953 a repeat chest film showed the ascending aorta to be considerably enlarged. He was again treated with penicillin and advised to limit his activity. In June 1954 he had a sudden oppressive substernal pain associated with a temperature of 38.8° C. (102° F.). It was thought that he had pericarditis and pleurisy. Chest roentgenograms revealed that the mass had enlarged. He had no other episodes of pain or symptoms. Examination showed a blood pressure of 140/60. There were no stigmata of Marfan's syndrome. A harsh systolic murmur was audible to the right of the sternum in the third interspace, and a diastolic murmur was audible in the second and third left intercostal spaces. The heart was enlarged to the left. Roentgenograms showed a large mass filling much of the right upper and anterior chest (Fig. 2, a). On May 19, 1955 the aneurysm was exposed through a bilateral fourth interspace incision, the sternum being transected. The operative procedure performed was essentially similar to that described above. Postoperatively he had a blood pressure of 180/100 with bloody urine and some costovertebral angle tenderness. This subsided and his blood pressure stabilized at 140/90. A faint systolic murmur persisted, but no diastolic murmur could be heard. His improvement was rapid and he returned to work without symptoms (Fig. 2, b). He was found dead at home 4 months after operation. Two weeks before death roentgenograms showed the left ventricle to be smaller than before operation. Autopsy was not obtained.

Case 3. J.H.H. 714094. A. H., a 31-year-old man, had been told he had a heart murmur at the age of 16 and was rejected by the Army in 1943 because of this. In 1953 he had a dull substernal pain which was aggravated by inspiration. A chest film was essentially normal (Fig. 3, a). The pain disappeared after 4 or 5 days. A similar pain appeared in the summer of 1955. There had been some recent increased fatigability and shortness of breath. On admission, examination showed no stigmata of Marfan's syndrome. Blood pressure was 120/80. The apical impulse was near the anterior axillary line, and there was enlargement to the right. There was a loud systolic murmur in the right second interspace transmitted up to the neck vessels. The aortic second sound was distinct,

and there was an early, decrescendo, diastolic murmur. In the neck there was a high-pitched, cooing, musical murmur. Radiologic studies showed a large diffuse dilatation of the ascending aorta with active pulsation. The left ventricle was considerably enlarged (Fig. 3, b). There was calcification in the region of the aortic valve. At operation on August 31, 1955, a localized fusiform dilatation of the ascending aorta was encountered and approximately half the circumference was excised. When the aorta was incised the central partition of an endothelialized aortic dissection was seen in the portion grasped in the clamp and was secured in the suture. Because of the striking murmur of aortic stenosis an operating tunnel of Dacron was sutured to the ascending aorta and the aortic valve palpated with the finger. The posterior, noncoronary cusp was soft and pliable without evidence of disease. The other two cusps seemed fused and partially calcified. A finger could be readily inserted through the orifice. Direct measurement of pressure showed a 20 millimeter gradient across the aortic valve. The valve was not treated. By compressing the aorta the central partition of a dissection could be felt in the ascending aorta. The extent of it could not be determined. As in the other patients the aneurysmal dilatation stopped proximal to the innominate artery. His convalescence was uncomplicated and he was well 6 months following operation.

Case 4. J.H.H. 726673. T. M., a 49-year-old white man, was admitted because of heart failure and an ascending aortic aneurysm. He had chorea as a child and was told in 1931 that he had a murmur and rheumatic heart disease. In 1945 he was rejected by the Armed Services because of the murmur, although he was asymptomatic. In 1948 he noted dyspnea on exertion and easy fatigability. He was found to have cardiac enlargement and apical and basal systolic murmurs. In spite of an apparently adequate cardiac regimen his condition worsened, and prior to admission he had dyspnea on exertion and occasionally nocturnally. He had a constant sense of precordial oppression without severe pain.

Examination showed a blood pressure of 200/90; peripheral pulses were collapsing. There was no arachnodactyly or dislocated lens to suggest Marfan's syndrome. Auricular fibrillation was present. He appeared chronically ill and dsypneic on slight exertion. He was slender but had no arachnodactyly or dislocated lens. Peripheral veins were distended; the heart was enlarged; liver and spleen were palpable. Loud systolic and diastolic murmurs were heard in the right second and third intercostal spaces. Electrocardiography showed auricular fibrillation and left ventricular strain. Arm to tongue circulation time was 30 seconds. Fluoroscopy showed a large aneurysm of the ascending aorta and considerable enlargement of the left ventricle (Fig. 4).

On January 12, 1956 an attempt was made to excise the aneurysm as in the preceding cases. As the clamp was removed after partial excision of the aneurysm and suture of the aorta, the heart slowed and stopped. Efforts at resuscitation were not successful. The operation had proceeded with less difficulty than on previous occasions. It was thought that chronic cardiac decompensation was largely responsible for his failure to withstand the procedure.

At autopsy the heart weighed 900 Grams, the increased weight being mainly due to left ventricular hypertrophy. There was calcific aortic stenosis with fusion of one commissure. The valve also appeared incompetent. An aortic dissection extended from 4 cm. above the aortic valve through the end of the aortic arch, where there was a reentry, and into the innominate and subclavian arteries. The thoracic aorta was of normal size. Microscopic study showed changes of cystic medionecrosis in the ascending aorta and to much less extent in the subclavian, mesenteric, and pulmonary arteries and the abdominal aorta.

Several distinguishing features of these four cases might be noted. All were men aged 31 to 49 years. Although suggestive histologic abnormalities were noted in the distal aorta and smaller arteries of the one complete autopsy, the gross dilatation was sharply localized to the ascending aorta. The dilatation did not significantly involve the aortic valve ring or the sinuses of Valsalva, although in the first two patients slight constriction of the aorta adjacent to the valve sufficed to relieve aortic insufficiency. Dissection of the ascending aortic aneurysm was present in the last two patients but did not extend beyond the aortic arch. In contrast to those due to syphilis and arteriosclerosis, the aneurysm could be separated readily from the pulmonary artery and adjacent structures. The presence of aortic stenosis in two of the patients suggests an unusual propensity to dilatation of the aorta distal to the valvular stenosis.

An additional patient has been treated with a localized aneurysm due to cystic

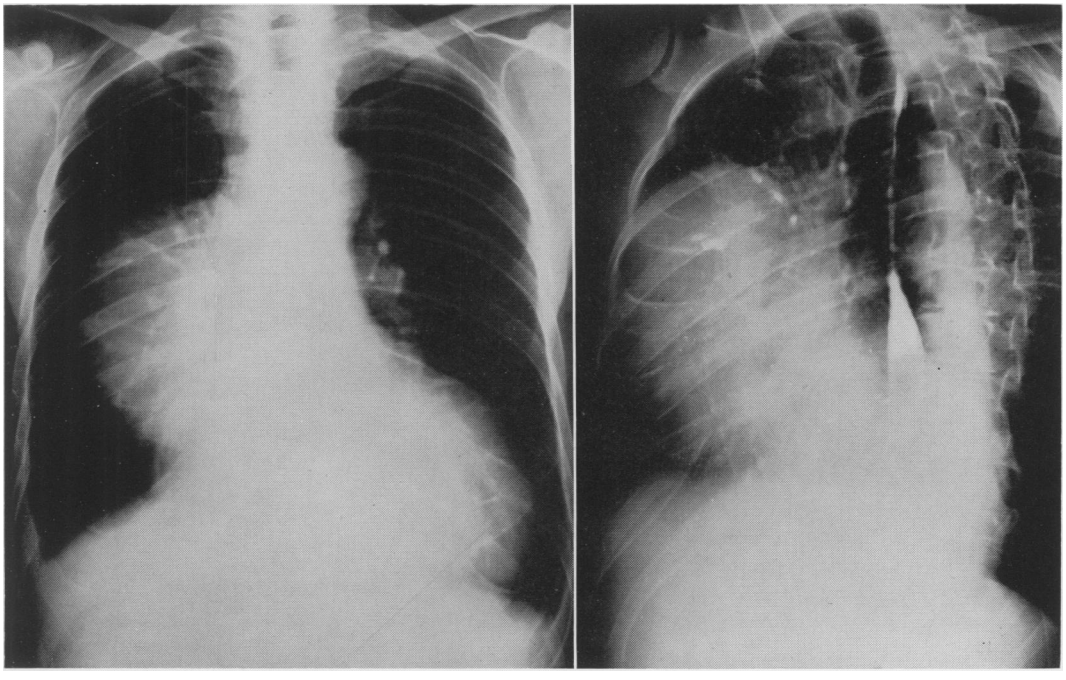

FIG. 4A. FIG. 4B.

FIG. 4. Case 4. Roentgenograms showing the fusiform aneurysm of the ascending aorta and enlargement of the left ventricle.

medionecrosis in the distal portion of the aortic arch.

Case 5. J.H.H. 712364. W. G., a 42-year-old white man, was admited without symptoms because of a mediastinal mass. In 1932 he had been rejected for military service because of a heart murmur, and was told he had a lesion the "size of a quarter" in his mediastinum. In 1954, after an elective chest film, he was told this was the "size of an orange."

On examination there were none of the stigmata of Marfan's syndrome. Blood pressure in the right arm was 156/98, in the left 122/98, and there was a palpable discrepancy in the radial pulses. Blood pressure in the leg was 155/90. There was a grade 3 apical systolic murmur. Roentgenologic studies showed a large, rounded, left anterior mediastinal mass (Fig. 5).

At left anterior thoracotomy on October 5, 1955 an aneurysm of the distal aortic arch, about 8 cm. in diameter, was seen. It began just distal to the left common carotid and involved the origin of the left subclavian artery. An area of minimal coarctation could be felt at the lower end of the aneurysm beyond which was a slight post-stenotic dilatation. The left subclavian artery was ligated and the aorta occluded proximally and distally. The aneurysm was excised and an end-to-end anastomosis done. Approximation was difficult but probably facilitated by elongation of the aorta and a slight indentation in the region of the ligamentum arteriosum such as is often seen with coarctation of the aorta. Total time of aortic occlusion was 30 minutes, during 20 minutes of which the aorta was occluded proximal to the left common carotid because of tearing of the aorta. He had no sequelae of the aortic occlusion, and his convalescence was not remarkable. He was working in March, 1956.

Study of the specimen showed a slight coarctation on the lower end of the aneurysm with little reduction in aortic lumen. Characteristic cystic medionecrosis was seen microscopically.

DISCUSSION

The pathologic changes in the aortic wall, now termed cystic medionecrosis, described by Gsell [6] in 1928 and Erdheim [4] in 1929 have received considerable attention, especially in the German literature. The alterations are frequently seen in the aorta of patients dying of spontaneous rupture,[10] in cases of dissecting aneurysm,[5] as

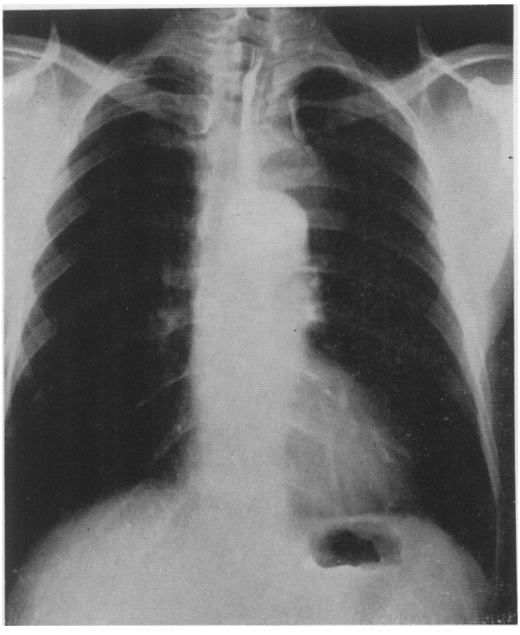

FIG. 5. Case 5. Roentgenogram showing left upper mediastinal mass.

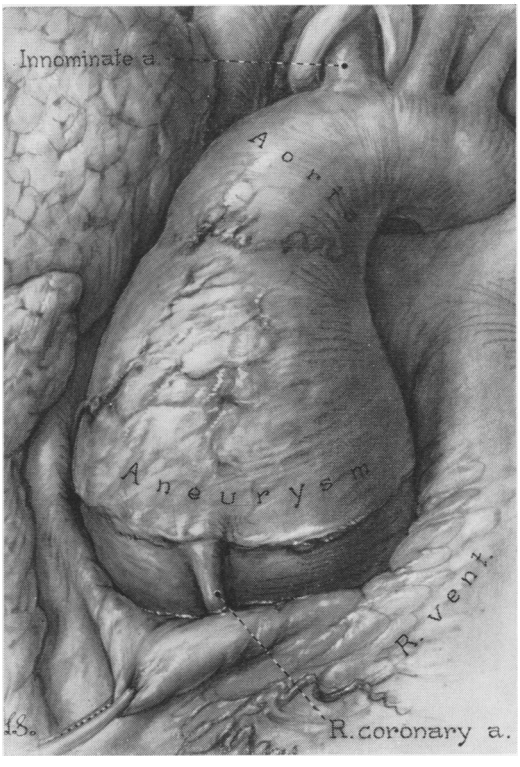

FIG. 6. Aneurysm of the ascending aorta in a patient with Marfan's syndrome. The sinuses of Valsalva are most prominently involved. This is in contrast to the picture of case 1–4 when the sinuses were spared and the disease was most marked in the midportion of the ascending aorta.

one of the stigmata of Marfan's syndrome,[8] and in some otherwise normal aortas.[11] Histologically, the lesions consist of necrosis and disappearance of muscle cells and elastic laminae in the media. When the elastic tissue is affected, mucoid material is seen to fill cystic spaces. There is no inflammatory reaction and no certain evidence of a reparative process except for collagen deposition in the outer portion of the media. Gore has demonstrated the preponderance of elastic degeneration in young individuals in contrast to the predominantly muscular necrosis in older patients dying with dissecting aneurysms. Both seem to be part of the same general disease process.

The etiology is unknown, although considerable speculation and some investigation have been done related to its cause. Toxins have been suggested because degenerative lesions of the aorta can be produced by diphtheria toxin,[3] and in rats by a diet of sweet peas [1] in which the toxic agent appears to be beta-aminonitrile.[9] In the patients seen with cystic medionecrosis, however, no history suggesting toxins can be obtained, severe infections have not always been present, and no explanation can be given of why only a few individuals might be affected. A frequent suggestion is that this is a degenerative disease. The muscular degeneration cannot be distinguished from the histologic changes of senility in the aorta. On the other hand, frequent association of congenital defects suggests a congenital abnormality of the aorta. In Marfan's syndrome there appears to be a congenital and inheritable defect in the ground substance, one manifestation of this being cystic medionecrosis.

Regardless of the etiology, it seems likely that a biochemical or metabolic defect precedes the morphologic one and that more extensive involvement of the ascending

aorta, as seen in our first four cases, is due to greater stress in this region. Rottino found localization of these histological changes to the ascending aorta in seven apparently normal aortas of 210 he examined.[11] In most of the reported instances of spontaneous rupture of the aorta or dissecting aneurysm, the disease has usually but not always been most marked in the ascending aorta. In Marfan's syndrome the immediate begining of the aorta, and especially the sinuses of Valsalva, seem to be most severely affected. In many instances demonstrable histologic changes have been localized to the ascending aorta with no abnormality discernible elsewhere. In spite of the apparent localization the process is probably a diffuse one, and, as in case four, peripheral vessels may be affected. Localization of the dilatation in our first four patients and the reported pathologic data support a surgical attack upon this area of greatest weakness in what is probably a generalized disease. In this respect excision of a portion of the aortic circumference and bolstering the remainder with a binder is comparable to excision of syphilitic and arteriosclerotic aneurysms. Reduction of diameter alone reduces strain on the wall and the tendency to rupture. One patient with Marfan's syndrome and dissecting aneurysm has been operated upon unsuccessfully with an attempt to excise partially the ascending aorta. In this condition dilatation is most prominent in the region of the aortic valve ring and sinuses of Valsalva (Fig. 6), in contrast to cases 1 to 4, where the sinuses of Valsalva were spared,[12] and a valve cusp was caught with the occluding clamp. This condition, ascending aortic aneurysm with Marfan's syndrome, is more difficult to treat surgically and the disease is also more generalized.

Relief of the incapacitating aortic insufficiency in the first patient and the diastolic murmur in the second is encouraging although both later died, the first probably of myocardial infarction and the second of unknown causes. Aortic insufficiency in these patients is caused by dilatation of the valve ring, and the valve cusps are usually essentially normal. It is believed that reduction of the diameter of the proximal aorta by excision of a portion of it sufficed to make the valve competent. Mattison and Cluff were able to find five previously reported cases of aneurysm of the ascending aorta due to cystic medionecrosis associated with aortic insufficiency and chronic cardiac failure. In these patients, as in our first case, incompetence was due to dilatation of the valve ring with otherwise normal aortic cusps. Prior to their diagnosis of our first case, no antemortem diagnosis of this condition had been reported.[7]

A congenital defect is probably the best explanation for the localized aneurysm in the distal aortic arch and slight coarctation of case 5. A similar case, treated by excision, was reported by Monod [9] and thought to be a congenital aneurysm. The patient reported by Woods and Kenny [13] probably had a similar lesion although no tissue was removed for histologic study.

SUMMARY

Cystic medionecrosis may be the cause of a localized aortic aneurysm. Such aneurysms have been seen principally in middle aged patients in the ascending aorta but also at the distal end of the aortic arch. In the latter location the lesion may be treated by excision and aortic anastomosis, possibly with an interposed graft. When the ascending aorta is involved the aortic valve may become incompetent as a result of dilatation of the valve ring. The aneurysm as well as the valvular incompetence has been relieved by excision of part of the circumference of the aorta and restoration of an essentially normal diameter. The structurally weakened aortic wall was reinforced with a nylon binder. The condition

can be recognized clinically and should be treated before great dilatation of the aorta, aortic dissection, or chronic heart failure occur.

Cystic medionecrosis is a poorly understood cause of aortic disease. Results of surgical treatment as well as the underlying disease require further investigation.

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DISCUSSION.—DR. JOHN H. GIBBON, JR., Philadelphia, Pennsylvania: I hope you will pardon me for arising a second time to discuss a paper this afternoon. I think this is a beautiful piece of work by Dr. Bahnson, and it shows what surgery is now able to do for patients who were formerly regarded as hopeless, and to whom we could offer nothing for prolongation of life.

I was partciularly interested in Dr. Bahnson's discussion of aneurysms of the sinus of Valsalva. About two weeks ago I operated upon a patient who had had a cardiac catheterization performed elsewhere, and in whom a diagnosis of an atrial septal defect was made. The patient was in cardiac failure with edema of his ankles. Upon exploration there was no atrial septal defect, but there was a left to right shunt into the right ventricle. The aorta was very large, about the size of some of those that Dr. Bahnson has presented today. There had been a rupture and a false aneurysm, I suppose, of the aortic wall, with erosion of the aneurysm into the right ventricle.

The patient had enormous hypertrophy of his right ventricle and a thrill over the right ventricular outflow tract. Pressure over the area, where the aneurysmal dilation of the aorta abutted the right ventricle, stopped the thrill. A simple mattress suture of silk was passed under the guidance of an overlying finger so as to obliterate the small opening that could be felt. When the mattress suture was tied, the thrill could no longer be felt.

The day after operation the edema of the ankles disappeared. He felt a great deal better. Just before I left Philadelphia my associate, Dr. Templeton, called me and told me that the patient had had a recurrence of his murmur and thrill and that he was once again in bad shape. Presumably the single mattress suture of silk, which I had used to close the defect, had cut through.

Apparently these lesions are infrequently recognized, but I believe they are susceptible to surgical management. I think Dr. Bahnson is to be congratulated upon the beautiful results he has achieved in patients with this type of lesion of the ascending aorta.

DR. ARTHUR H. BLAKEMORE, New York, New York: I have been greatly impressed with this presentation. It recalls to my mind a 43-year-old patient wtih aortic insufficiency and a wide-mouth, saccular aneurysm of the ascending arch of the