Coarctation of Aorta Complicated by Bacterial Endocarditis and an Aneurysm of the Sinus of Valsalva

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Dr. Frank Glenn: The patient for today's discussion presented some particularly interesting and challenging problems when she was first seen at The New York Hospital in November 1954. I am going to ask our panel to review with you the ideas and considerations that confronted us in the diagnosis and management of this patient's heart disease. Dr. Engle, will you please give us the history?

Dr. Mary A. Engle: The patient, a 27-year-old married woman, was admitted for evaluation of coarctation of the aorta. From her and from the physicians who took care of her from infancy we obtained the following information. She was born 1 month prematurely. Because she failed to thrive, she was taken to a pediatrician at the age of 5½ months. He discovered a loud systolic murmur all over the precordium and in the lung fields. The aortic and pulmonic second sounds were distinct, and there was no precordial bulge. Fluoroscopy disclosed enlargement of the left ventricle. These findings remained unchanged throughout infancy and childhood and she was considered to have a ventricular septal defect.

Failure to thrive was most marked during the first year. After the age of 2, however, she began to improve and by the age of 9 years she was average in size (fig. 1). She tired after exercise and became short of breath more quickly than her playmates. Restriction of activities was advised by the physician. At this time she had a murmur of aortic insufficiency. The first recorded blood pressure determination was at age 12, measured at 165-145/65 mm. Hg in the arm.

In 1947 the patient had fever and blood culture was positive for Streptococcus viridans. The blood pressure in the arms was 210/70 and in the legs 122/80 at this time. She received a course of penicillin for 2 weeks. She suffered a second episode of Str. viridans infection in 1954. This was treated with intravenous penicillin and streptomycin for 6 weeks. The diagnosis of coarctation of the aorta was made at this time. The cardiothoracic ratio at this time was 53.4 per cent.

Dr. Glenn: Dr. Engle, is this the usual course of events for a patient with coarctation of the aorta?

Dr. Engle: Yes, it is a common story. The patient with coarctation of the aorta is apt to have difficulty from his lesion at 2 age periods: early infancy and early to mid-adult life. Babies may fail to thrive and even go into heart failure in the first months of life. The early difficulties seem related to the severity of the coarctation as well as to the relationship of this constriction to the ductus arteriosus, which is normally still patent in the newborn period. Medical management of the heart failure is quite successful in tiding those babies over the period of early difficulty.
COMPLICATED COARCTATION OF AORTA

Fig. 1. Growth chart of patient V.W. Failure to grow was most marked during first year. After age 2 it improved and by age 9 was in the average range. Blood pressure in arm at 12 years was 165-145/65 mm. Hg; at 16 years, 150/20; and at 18 years, 150/40. (We are indebted to Drs. Benjamin Kramer and Charles Shook of for supplying the information on this patient's childhood and adolescent years, including growth data.)

until adequate collateral circulation develops. This is usually achieved by the age of 2 years. This particular patient was not in heart failure but she did have cardiac enlargement at least from 5 months of age and her growth was markedly retarded for the first year.

It is interesting that adults may not volunteer such a history even though this was a difficult time in their lives. It is a period beyond their memory, and unless a parent or the physicians' notes are available, as in the present case, this part of the history may be lost.

DR. GLENN: Dr. Stewart, if these children survive childhood, in brief, what may be the course of their disease?

DR. HAROLD J. STEWART: Certain patients live a normal life span and die of some disease not related to the congenital defect. On the other hand during adulthood, patients with coarctation of the aorta are subject to several complications: (1) to subacute bacterial endarteritis or endocarditis, which may occur on the additional lesion of bicuspid aortic valve, (2) to rupture of the aorta, (3) to a cerebral vascular accident, (4) or to the manifestations of hypertensive cardiovascular disease, and perhaps heart failure, (5) heart failure or rupture of the aorta, or of a cerebral aneurysm, may result from the increased burden of pregnancy.

DR. GLENN: Let us return to the case presentation. What were the pertinent findings on examination when this patient came under observation here?

DR. ENGLE: Physical examination revealed strong radial pulses and weak femoral pulses with a perceptible lag behind the radial impulse. Blood pressure in the arms was 190/50-0 and in the legs 95/40 mm. of Hg. A bounding pulsation was visible in the suprasternal notch, and pulsations could be
seen and felt over the chest posteriorly. There was no cyanosis or clubbing of fingers or toes. The heart was enlarged to the anterior axillary line. A systolic thrill was palpable over the aortic area and in the suprasternal notch. A loud, long systolic murmur, maximal in the second right interspace, was transmitted to the neck vessels and the aorta, over the precordium, and to the chest posteriorly. A separate harsh systolic murmur was heard in the fourth and fifth left parasternal spaces. A long, blowing diastolic murmur along the left sternal border originated at Erb's point.

The lungs were clear, the liver and spleen were not enlarged, there was no peripheral edema, and embolic phenomena were not detected.

Laboratory studies showed normal red blood cell count, hemoglobin, and hematocrit level, but the white blood cell count was 12,500 and the sedimentation rate was elevated. Three blood cultures were negative.

X-ray and fluoroscopy of the chest (fig. 2) revealed a cardiothoracic ratio of 60 per cent. The enlargement was predominantly left ventricular, though there was some enlargement of the left atrium as well. The aortic knob was normally prominent. In the left anterior oblique projection the ascending aorta was dilated and widely pulsatile. Barium swallow confirmed the left atrial enlargement and disclosed an anterior displacement of the esophagus by a dilated portion of the ascending aorta. This was thought (and later shown by angiography) to be due to the poststenotic dilatation of the aorta below the coarctation. There were no other retro-esophageal vessels, suggestive of aneurysmally dilated intercostal arteries. Rib notching was present bilaterally.

An electrocardiogram disclosed a pattern of left ventricular hypertrophy (abnormally tall R waves in the left precordial and foot leads with deep S waves in the right precordial leads) and of left ventricular "strain" (depression of S-T segments and inversion of T waves in left precordial and foot leads with reciprocal changes over the right side of the precordium).

DR. GLENN: Dr. Lukas, will you give us the pressure measurements you made?

DR. DANIEL S. LUKAS: Intra-arterial pres-
Complicated Coarctation of Aorta

sure tracings were characteristic of coarctation of the aorta and aortic insufficiency. Measurements were 227/63 (mean 122) mm. Hg in the arm and 110/66 (mean 82) in the leg.

Dr. Glenn: Dr. Stewart, will you comment on the diagnosis at this point?

Dr. Stewart: Dr. Glenn, I think that the diagnosis of coarctation of the aorta was readily made clinically in this patient. The volume of all the peripheral pulses should be estimated clinically in every patient and the diagnosis should be suspected when the volume of the pulses in the legs is less than that in the arms, or may indeed be absent. Moreover, the blood pressure should be taken not only in both arms but also in both legs in every patient in all age groups.

The following findings pointed to this diagnosis: 1. The volume of the radial pulses was greater than the femoral pulses. 2. The blood pressure in the arms was higher than in the legs, indicating an obstruction to the flow of blood through the aorta below the subclavian artery. It may be said parenthetically that the blood pressure may be normal in the arms, but decreased to absent in the legs. The carotid pulsations were exaggerated as were the aortic pulsations in the suprasternal notch. 3. The peripheral signs of collateral circulation shown by pulsations and bruits over intercostal arteries posteriorly and other collateral vessels used to carry arterial blood from above the coarctation back into the aorta below the obstruction. 4. The x-ray of the chest showed notching or scalloping of the lower margins of the ribs by the dilated tortuous intercostal arteries. Both the pulsations and notching may be absent in children. The presence of these latter 2 signs is a valuable guide to the surgeon as to the possibility of a successful operation because of the availability of these vessels to maintain circulation while the aorta is clamped. The x-ray showed left ventricular enlargement and absence of the aortic knob. 5. The exact location of the coarctation and the amount of proximal segment beyond the subclavian available to the surgeon for the repair was demonstrated by angiocardiogram, which Dr. Steinberg will discuss shortly.

Perhaps Dr. Engle has some additional comment to make.

Dr. Engle: Localization of the coarctation was possible from the studies thus far presented. The fact that the pulses in the neck and in both arms were equally strong placed the obstruction distal to the left subclavian artery. There was evidence on barium swallow of a large retroesophageal vessel below the level of the descending aortic arch (fig. 2B). In such a patient this deformity of the esophogram is due to the poststenotic dilatation of the aorta just beneath the coarctation. Thus the narrowed segment of aorta was localized to the area of the aorta where it most commonly occurs: at the level of the ligamentum arteriosum. One additional localizing sign that is useful is the smooth, blowing, well-localized systolic murmur that is heard posteriorly near the midline overlying the narrowed segment. If one listens along the course of the descending aorta, such a murmur can often be detected, even if the coarctation is in the abdominal aorta. This patient, however, had such a loud systolic murmur over her chest anteriorly and posteriorly that this sign was less valuable.

Coarctation of the aorta is much less common in females than in males. When it is present, it is apt to be in an unusual location or complicated by some additional lesion. This patient's coarctation was in the usual location, but the presence of an associated cardiac lesion was suggested by the loud systolic murmur over the heart and chest that were present from infancy. The murmur of a coarctation itself is soft and is heard better posteriorly over the descending aorta than it is over the heart. With enlargement of the left ventricle and dilatation of the mitral valve ring or of the aorta, murmurs of relative mitral insufficiency or aortic stenosis appear, but her murmur was quite loud and unchanging from early infancy, even though her heart was not much enlarged. A more likely ex-
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Left common carotid artery

Internal mammary artery

Internal mammary arterial collaterals

Left subclavian artery

Coarctation

Sinus of Valsalva

Wall of left ventricle

Aorta

Left ventricle

Fig. 3. Left anterior oblique angiogram 7 seconds after beginning the injection. Left. X-ray showing hypertrophy of left ventricle, aneurysm of sinus of Valsalva, coarctation of aorta, and internal mammary arterial collateral vessels. Right. Tracing of x-ray.

Planation is that she had a congenital anomaly of the aortic valve in the form of a bicuspid, slightly stenotic aortic valve or subvalvular ridge. Abnormalities of the region of the aortic valve are commonly associated with coarctation of the aorta.

Dr. Stewart: In my experience the systolic murmur in adults is heard best anteriorly over the base of the heart. If it is very loud, it may be heard posteriorly as well but it is difficult to be certain whether a murmur heard posteriorly may not be bruit over the collateral circulation.

The murmur of aortic insufficiency had been heard in this patient at 12 years of age before she had subacute bacterial endocarditis. It is not uncommon to hear such a murmur in these patients in the absence of any complication. I have thought that the murmur was due to dilatation of the aortic ring by the dilatation of her aorta with the hypertension above the coarctation. Murmurs are not heard in "normal" bicuspid aortic valves.

Dr. Glenn: The history and findings given by Dr. Engle justified the diagnosis of coarctation of the aorta, aortic regurgitation, and bacterial endocarditis. Demonstration of the exact location of the coarctation enables the surgeon to plan his approach as well as to be forewarned of additional possible abnormalities.

Dr. Steinberg, will you review the x-ray findings including the angiograms of the patient?

Dr. Israel Steinberg: The conventional roentgenograms showed left ventricular enlargement. There were absence of the aortic knob and posterior indentation of the esophagus by a dilated descending aorta. Rib notching was present bilaterally from the fourth to seventh ribs. Angiocardiography showed coarctation of the aorta at the usual site just below the origin of the left subclavian artery (fig. 3). There was dilatation of the ascending aorta and brachiocephalic arteries, and large arterial collateral vessels were seen. A
2.2-cm. saccular aneurysm of the right aortic sinus was clearly visualized.

DR. GLENN: Dr. Steinberg, would you tell us something about your experience with aneurysms of the sinus of Valsalva?

DR. STEINBERG: The aortic sinuses are intracardiac and cannot be identified on conventional roentgenography. During angiocardiography they appear as dilatations at the root of the aorta immediately above the aortic valves and are best visualized in the left anterior oblique view. Inconstant filling of the coronary arteries during angiocardiography does not always allow individual identification of the sinuses. However, in the left anterior oblique view the right coronary sinus is regularly anterior to and just behind the sternum. The aortic sinuses are in close relation to all the cardiac chambers, particularly the right atrium and ventricle. The origins of the pulmonary artery, the interventricular septum, and the left atrium are adjacent, whereas the superior vena cava is more distant.

Aneurysms of the aortic sinuses (of Valsalva) are rare and are either congenital or acquired. The acquired types are chiefly due to syphilis or bacterial endocarditis. The congenital aneurysms are thought to be due to a developmental defect in either the aortopulmonary septum or the elastic tissue of the aortic sinuses. Edwards and Burchell attribute congenital aneurysms to lack of continuity between the aortic media and aortic ring. In aortic sinus aneurysms the wall is made up of the atrium instead of aortic media; pressure within the aorta causes bulging of the sinus, producing an aneurysm.

In an 11-year period, during which over 3,000 patients were studied by the intravenous method of angiocardiography, 25 cases of unruptured aneurysms of the aortic sinus have been diagnosed at this center. Ten were acquired; 9 were due to syphilis and 1 was due to arteriosclerotic dilatation of the thoracic aorta. Fifteen were congenital and of these 6, including the case herein presented, were associated with coarctation of the aorta.

One patient had aneurysmal dilatation of the aortic sinuses and pseudocoarctation of the aorta while another had associated aortic and mitral valvular disease. Seven patients had arachnodactyly. Aneurysmal dilatation of the aortic sinuses was found in all the cases except the one reported here; in this, the right aortic sinus alone was aneurysmal.

Aortic regurgitation, which is common in patients with aortic sinus aneurysms, has been attributed to dilatation of the aortic ring and was present in all the acquired cases of aortic sinus aneurysm. In congenital aortic sinus aneurysms, anomalies of the aortic cusps have been frequent at autopsy. In our series, in addition to the case described above, aortic incompetence occurred in a patient with arachnodactyly.

Congenital aortic sinus aneurysms are paper thin and have a tendency to rupture into the structures of the right side of the heart. When perforation occurs, severe overloading of the right cardiac chambers, pulmonary hypertension, and intractable heart failure follow. Sudden onset of dyspnea associated with a machinery murmur suggests rupture of the aortic root with creation of an aorticocardiac fistula. Confirmation of this event may be obtained by cardiac catheterization.

The incidence of aneurysm of the aortic sinuses in coarctation of the aorta is difficult to estimate; it occurred 6 times among 128 patients with coarctation of the aorta studied angiocardiographically at this center. The ages of patients varied from 13 to 27 years with an average of 21 years. Five were males and 3 females. Only 1 was a Negro. Three patients were asymptomatic although a history of previous hypertension or heart murmurs was present in every instance. Headache was present in 2, while 1 had bacterial endocarditis. Systolic murmurs (grade III to IV) transmitted to the axilla and back, especially over the suprascapula area and similar to those usually found in coarctation of the aorta, were common.

In all cases of aneurysmal dilatation of the
aortic sinuses and coarctation there was enlargement of the heart, especially the left ventricle. Deformity of the aortic arch and rib notching were constant findings. Angiocardiology provided the definitive diagnosis of aortic sinus aneurysm and demonstrated the point of coarctation. In the literature there is also a report of a right aortic sinus diagnosed by retrograde aortography.

DR. GLENN: Thank you. That is a beautiful demonstration.

DR. ENGLE: Will you tell us more of her hospital course?

DR. ENGLE: While these studies were being performed, the patient ran a low-grade fever, around 37.6 C., and had a persistently elevated white cell blood count and sedimentation rate. The possibility of incomplete cure of the subacute bacterial endocarditis was raised.

DR. GLENN: Dr. Stewart, what should be done to evaluate such a situation? What therapy would you recommend for bacterial endocarditis?

DR. STEWART: A persistent search should be made for embolic manifestations: peripheral petechiae in nailbeds, fingertips, eye grounds; repeated blood cultures at the height of the fever. Arterial blood cultures have not been more effective in securing positive blood cultures than venous blood. The most common organism that causes subacute bacterial endocarditis in this defect is the Streptococcus viridans. Should the blood culture be positive, an optimal course of therapy should be instituted.

It is the general experience that more relapses occur in patients treated for the shorter periods and with the smaller dosage schedules. Accordingly one starts out on a 6-week program of a total of 4 to 6 million units of sodium penicillin G every day, given in divided doses at 2-hour intervals intramuscularly. If later tests indicate that the strain of the organism is a nonresistant one, the amount of penicillin is increased. On occasion the treatment may be stopped at 4 weeks.

If there is need for attempting to treat for a shorter period, a rapid 2-week "combined" therapy course may be used, namely, a total of 6 million units of sodium penicillin G intramuscularly in divided amounts every 2 hours, every day, and a total of 2 Gm. of dihydrostreptomycin (or 1 Gm. of streptomycin and 1 Gm. of dihydrostreptomycin) in 4 doses intramuscularly as originally described by Robbins and Tompsett.

If repeated blood cultures are negative but subacute bacterial endocarditis is still likely or suspected, a course of therapy should be given. Since 1 out of 5 instances of subacute bacterial endocarditis with positive blood cultures is due to enterococcus, it is best to treat those with negative cultures as though they are of this worst type and give the combined therapy for a period of 6 weeks.

DR. GLENN: Dr. Stewart, will you pull together the diagnosis that finally emerged?

DR. STEWART: It was apparent that the patient had coarctation of the aorta with very high blood pressure above the coarctation, but with good collateral circulation. There was the murmur of aortic insufficiency. The angiocardogram showed the coarctation to be located below the left subclavian artery, and also demonstrated an aneurysm of the sinus of Valsalva. The patient had had 2 episodes of bacterial endocarditis or endarteritis, the last one fairly recently. There was no way of ascertaining whether the sinus of Valsalva was the site of the implantation, or perhaps an associated bicuspid aortic valve, or the coarcted segment.

She was a patient with a complicated background who was presented for correction of the coarctation of the aorta.

DR. GLENN: We anticipated 2 hazards in particular with this patient. The first was that an increase in the blood pressure following the application of occluding clamps prior to resection of the coarcted segment might result in a rupture of the aneurysm of the sinus of Valsalva. The second was the possible requirement of prolonged occlusion of the aorta because of technical difficulties that might arise from the result of a long-standing infection, the bacterial endocarditis.

DR. STEWART, how soon is it safe to operate
after recent subacute bacterial endocarditis?

Dr. Stewart: Dr. Glenn, with this patient with slight fever only, who had coarctation of the aorta with very high blood pressure and an aneurysm of the sinus of Valsalva, the possibility of its rupture indicated that repair of the coarctation, if it could be done, should be carried out as soon as possible.

To this end she was given a 2-week course of penicillin therapy and was discharged afebrile on December 6. Because of nocturnal cough, dyspnea, and orthopnea as evidence of early heart failure, the patient was digitalized. She was readmitted on February 13, approximately 2\(\frac{1}{2}\) months later. This period of time was allowed to permit healing of the endocarditis and formation of strong scar tissue in the aneurysm of the sinus of Valsalva in case this was involved.

Dr. Glenn: Mr. Artusio, what problems does this patient present to the anesthesiologist?

Dr. Joseph Artusio: We have used diethyl ether in this clinic to accomplish a light level of anesthesia, which we have termed analgesia. The level is one in which there is minimal depression to respiration and circulation, and the patient frequently is oriented as to place but is completely analgesic and has no memory of the experience. At this level of anesthesia the patient has optimal reactivity to the stresses of surgery, blood loss, extreme positions on the operating table, and change of position on the operating table.

Premedication consisted of a small dose of atropine, 0.2 mg., and in order that the circulation not be embarrassed by the presence of an opiate, none was used. A 250-mg. dose of thiopental sodium, in a 2.5 per cent solution, was given for the psychic sedation afforded the patient, so that she would not fear the anesthetic mask. Nitrous oxide-oxygen was started, with an 8-L. flow of 75 per cent nitrous oxide and 25 per cent oxygen, and ether was gradually added to the mixture until the patient entered a light level of surgical anesthesia. At that time, under direct vision, the pharynx and larynx were sprayed with 2 per cent Xylocaine for topical anesthesia, a 38-

French cuffed endotracheal tube was inserted into the trachea, and the patient was immediately returned to the analgesic state of anesthesia where she responded to the spoken voice.

Since this patient had an aneurysm of the sinus of Valsalva in association with her coarctation of the aorta, we were most concerned that during the period in which the proximal aorta was clamped the blood pressure should not rise unduly and produce rupture of the aneurysm. It was planned to use Arfonad, a thiophanium derivative, which would induce ganglionic blockade in the periphery and control the height of the blood pressure. However, from previous experience with this compound we knew that the desired hypotension was not always achieved. This resistance to hypotension is particularly true in the young adult group to which this girl belonged. Thus it was considered that if Arfonad did not produce the necessary hypotension, comitant hypothermia might aid to control blood pressure.

Actually Arfonad worked very well and we were able to reduce the blood pressure at will. The blood pressure before the aortic clamps were applied was 230/80 mm. Hg with a pulse rate of 80. Arfonad was started by intermittent injections of 3 mg. followed by a 0.1 per cent adjustable drip. In this manner the blood pressure was maintained within a range of 140 to 200 systolic. Following the anastomosis the blood pressure leveled off to 170/80.

Dr. Glenn: Dr. Holswade, what are the advantages of hypothermia in this situation and how was it used?

Dr. George R. Holswade: It was hoped that by cooling the patient to approximately 32 C. the blood pressure would fall subsequent to a decrease in cardiac output and would not rise precipitously during the occlusion of the aorta. When surface cooling of the body occurs, there is often a short initial rise in blood pressure due in part to peripheral vasoconstriction and possibly to shivering in some cases. But as the cooling progresses, the blood pressure falls in a curve.
that is parallel to that of the temperature and to that of the cardiac output. Cooling in a patient such as this should not be carried to a rectal temperature lower than 31 or 32°C. In this temperature range there is little danger of serious cardiac arrhythmias. This degree of hypothermia might provide just enough slowing of heart action, just enough decrease in cardiac output and just enough hypotensive effect to prevent a rupture of the aneurysm of the sinus of Valsalva when the aorta is cross clamped. For this reason it seemed advisable that the patient be cooled while the chest was being opened and the coarctation was being dissected free.

Cooling of the patient did not delay the operation. It was easily accomplished by pumping a cold alcohol solution through special rubber blankets (Thermo-rite), which were placed beneath the patient and over the lower portion of the body. To further speed up the cooling process plastic bags filled with crushed ice were packed around the patient. The temperature was reduced to 32°C and maintained at that level until the anastomosis was completed. Warm water at 44°C was then pumped through the rubber blankets.

The patient remained on the operating table until her body temperature had returned to 35°C. She withstood the procedure extremely well and was awake at the end of the operation.

Dr. Glenn: The operation was accomplished with little more than the usual effort required for an adult with a coarctation. There was a very luxuriant collateral circulation. The intercostal arteries near their origin from the aorta were almost aneurysmal. Three pairs were divided. The internal mammary was very large.

The heart was enlarged, the left ventricle being quite prominent, its wall hypertrophied. The proximal aorta at its origin was enlarged and a coarse thrill was felt in systole, but perceptible in diastole. A saccular aneurysm of the right aortic sinus measured 2.2 cm. in diameter, and projected 2 cm. above the surrounding cardiac surface. It had an egg-shell consistency and expanded very little on contraction of the left ventricle. Probably it was partially obliterated and, in the belief that it might further decrease following the lowering of the blood pressure after resection of the coarctation, it was left unmolessted. Furthermore, it has been demonstrated that the intracardiac approach seems to be the avenue of preference.

The coarcted segment measured 1 cm. in length, and in its midportion as it was held forward on slight tension, a diaphragm could be palpated. A palpable thrill was quite evident, with a jet that could be felt most distinctly 2 cm. beyond on the left lateral aspect of the dilated distal segment. Here there was a definite "jet plaque." The ligamentum arteriosum entered the coarcted segment just proximal to the diaphragm. It measured 1 cm. in length and 0.3 cm. in diameter. On palpation it seemed tubular and possibly patent but there was no thrill. It was divided, the aortic half was patent but
the pulmonary portion was obliterated (fig. 4A).

Clamps were placed proximal and distal to the coarctation. There was an elevation of the blood pressure from 160/80 to 220/90 mm. Hg. The coarcted segment was resected, and an anastomosis was done. This required almost 30 minutes and during this period the blood pressure, although fluctuating between 210/90 and 130/80, leveled off at 170/80, being controlled with Arfonad. After completion of the anastomosis the distal clamp was removed and then the proximal clamp was gradually released over an interval of 15 minutes. The blood pressure did not fluctuate thereafter and remained at 170/80.

The lumen of the diaphragm in the resected segment was slit-like in shape and measured 2 mm. in its greatest diameter. There were numerous linear bright yellow fibrous plaques in the diaphragm and the adjacent wall of the aorta (fig. 4B).

The wall of the aorta was thickened in the area of coarctation and its pattern was somewhat disrupted (fig. 4C). At the junction of the thickened portion and the more normal vascular wall there was an atheromatous area consisting of a collection of large mononuclear phagocytes with foamy cytoplasm. Connective tissue was abundant in the wall of the aorta and in the area of diaphragmatic narrowing of the coarctation the usual parallel pattern had disappeared and the fibers were tangled, twisted, and turned upon one another.

Her immediate postoperative course was uneventful and without complications. She was discharged 3 weeks later. At that time the blood pressures were right arm 150/55; left arm, 160/60; right leg, 210/110; left leg, 200/100.

Dr. Engle, what has happened to this patient since she left the hospital?

Dr. Engle: After convalescence she returned to her regular occupation as a freelance writer. It is now 2½ years since the patient's operation. She states that she has never been so energetic or untiring. She can climb 3 or 4 flights of stairs before her legs fatigue. She does not experience shortness of breath, and she sleeps flat comfortably. She no longer takes a digitalis preparation.

Her blood pressure is 130/50 - 30 mm. Hg in the arms and 140/50 in the legs. Exaggerated intercostal pulsations are gone. The aortic systolic murmur and the diastolic murmur of the aortic insufficiency persist. The aortic pulsation in the suprasternal notch is still evident. The heart size is less by 1 cm. than preoperatively, and this improvement is due chiefly to a decrease in left ventricular size (fig. 5). There is no longer evidence of left atrial enlargement, and the ascending aorta is smaller. Her electrocardiogram continues to show left ventricular hypertrophy and "strain."

Thus the manifestations of coarctation of the aorta have disappeared. Relief of this cardiac burden has apparently made her better able to withstand the aortic valve involvement. Objectively, as well as symptomatically, she is better. She will continue under medical supervision.

Dr. Stewart: Dr. Glenn, what has been our experience with aortic insufficiency in coarctation of the aorta?

Dr. Glenn: Aortic insufficiency is seen in coarctation under 3 circumstances. The first is believed to be due to prolonged hyperten-
sion that produces enlargement of the proximal aorta and the aortic ring at the level of the valves. Secondly the aortic valve may have only 2 leaflets instead of 3, and slight enlargement of the aorta may result in insufficiency. Then in the third group there are those instances where disease such as bacterial endocarditis or rheumatic heart disease may cause a loss of valve substance that results in insufficiency. Our experience with aortic insufficiency associated with coarctation of the aorta is limited. We have operated upon 4 such patients. All 4 tolerated the operation well. With a return of the blood pressure to normal, 1 patient lost the signs of insufficiency. Another, although experiencing relief from the preoperative hypertension, continued to have signs of aortic insufficiency and increasing left heart failure, and died almost a year later. The other 2 patients have had a persistence of definite evidence of aortic regurgitation but are definitely improved. This patient, our fifth with aortic insufficiency, continues to have a systolic and diastolic murmur along the left sternal border. Evidently she has a definite aortic regurgitation, now 30 months after operation.

Dr. Stewart: During the course of the 30 years of this woman’s life, advances in medicine and surgery have come along just in time to help her out of difficulty: antibiotic therapy for subacute bacterial endocarditis and surgery for removal of the coarctation. If she should later need help because of aortic insufficiency or rupture of an aneurysm of the sinus of Valsalva, these conditions too are now within the ever-widening area of cardiovascular lesions, both congenital and acquired, that are amenable to surgery. Close teamwork of the medical and surgical specialties interested in this field has made possible these great steps forward.


The recent Mayo Clinic experience in the management of aortic and iliac arterial occlusive disease is described. In the present series of 99 patients, intermittent claudication was the chief symptom and the status of the leg pulses was the most important physical finding. Preoperative aortography was done routinely in order to define the extent of the occlusions. Technical aspects of resection of the obstructed vessels and homograft reconstruction are outlined. Excellent operative results were obtained in 71 patients (72 per cent) as indicated by complete relief of symptoms and return of leg pulses. Five deaths occurred, all postoperatively. The most important factor in the success of this surgery is insuring that the distal anastomosis is made to a patent artery, if necessary to the superficial femoral.
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Circulation. 1958;17:432-442
doi: 10.1161/01.CIR.17.3.432

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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