Congenital Aortic Stenosis
Clinical Aspects and Surgical Treatment

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Thirty-seven patients with congenital aortic stenosis have been studied. Only a few were without symptoms referable to the defect. Characteristically, the patients had a loud, harsh systolic murmur, accompanied by a thrill, to the right of the sternum. On x-ray examination the most frequent manifestation was dilatation of the ascending aorta. Electrocardiographic evidence of left ventricular hypertrophy was found in 60 per cent. Nineteen patients have been operated upon with 2 deaths. The procedure has been tolerated amazingly well. Although it is too early to judge the results, all but 2 of the surviving patients appear to have been helped.

During the development of the left ventricular outflow tract and of the aortic valve certain normal processes may be disturbed so that the output of blood is obstructed postnatally. As in similar aberrations of evolution of the right ventricular outflow tract, the obstructing tissue may be proximal to the valve, or the valve cusps may be fused along their margins, leaving a contracted orifice distally. Wherever the site of interference with flow, the physiologic effect is the same: difficulty in the ejection of blood from the left ventricle and the danger of insufficient oxygen supply to the myocardium, to the brain, and to other less vital tissues.

Relatively little attention has been given the malformation recently. Young reported 10 patients believed to have infundibular stenosis, but no physiologic or anatomic studies were performed. In 1947, Grishman, Steinberg, and Sussman presented data on 23 patients, of whom were autopsied. In addition to physical findings, they described pulse tracings, electrocardiograms, and x-ray photographs. Brofman and Feil were convinced, on the basis of their observations in 10 patients, that the diagnosis of infundibular aortic stenosis can be made by reference to arterial pulse tracings. Forty patients believed to have congenital aortic stenosis were studied by Campbell and Kaunte. Four were autopsied; the diagnosis in the remainder was based primarily on clinical data. Smith and Matthews considered 5 cases of aortic stenosis, 3 of which were proved, and referred particularly to bicuspid aortic valves. The phonocardiogram and the arterial pulse were believed diagnostic in the 15 patients of Reinhold, Rudhe and Bonham-Carter. Twenty-eight patients were reviewed by Marquis and Logan: 3 were autopsied; in 6 surgery was performed; and in the remainder clinical observations were believed to be diagnostic.

Because the anomaly can sometimes be helped by surgery and because our observations differ from those presented in the literature, those cases seen here in the past 3 years are analyzed.

Subjects

Thirty-seven individuals have been studied. A diagnosis of congenital heart disease had been made at birth in 9, within the first year of life in 16, and before the fourth year in the remainder. In none was there a history of rheumatic fever nor any illness suggestive of it. There was a definite sex predilection, there being 29 males and only 8 females; while the age range at the time of diagnosis was 4 months to 39 years.

The diagnosis was confirmed by autopsy in 4 patients, 2 of whom had valvar and 2 infundibular
stenosis. An additional 16 patients had valvular stenosis and 1 had infundibular obstruction at surgery.

Associated cardiac and vascular malformations. The most common accompanying abnormality was coarctation of the aorta. It was demonstrated by thoracic aortography in 4 patients, confirmed by surgery in 2 and by autopsy in 1. The fourth was shown by the contrast study to have generalized hypoplasia of the descending aorta from the region of the left subclavian artery to the diaphragm. Four other patients had evidence of obstruction in the distal aorta with weak or absent femoral pulsations and lowered blood pressure in the legs.

Pulmonary stenosis was present in 3 patients, but it was not of physiologic significance. In addition, 1 patient had a patent ductus arteriosus, and a patent ductus was also present in 1 patient with coarctation of the aorta.

Of the patients with infundibular stenosis found at autopsy, a defect in the membranous portion of the interventricular septum was present in 1, and the right coronary artery was absent in the other.

METHODS

A complete history and physical examination, conventional x-ray and fluoroscopic studies, and electrocardiograms with 12 or 14 leads were secured in all patients.

Right heart catheterization was completed in 31 patients, in 1 patient the left side of the heart was simultaneously catheterized, in another, right and left sides were catheterized at different times. Pressures were measured by means of a capacitance-type electromanometer, recorded directly and calibrated with a mercury manometer. Blood samples were analyzed for oxygen content by the method of Van Slyke and Neill.* Cardiac output was determined by use of the Fick principle in those in whom oxygen consumption was measured.

Thoracic aortography was performed in 6 patients. In all a Lehman catheter* was introduced into the brachial artery and guided to the ascending aorta, where radiopaque material was injected.

Brachial artery pulse tracings were secured in all but 3 of the group. Pressure was recorded during the resting state and during the Valsalva maneuver when possible. In young children who were anesthetized, the Valsalva effect was obtained by inflation of a blood pressure cuff about the chest or by manual compression of the thorax while mouth and nares were held closed. In a few, the desired response resulted from induced coughing.

RESULTS

History

Pregnancy. In no instance was a history obtained of an exanthematous disease during gestation. The only abnormalities concerning which definite information was available were nausea and vomiting during the first trimester in 5 cases and nausea and vaginal bleeding in 1. The date of conception and the age of the parents were without significance. No patient was born prematurely.

Growth and Development. Weight gain during infancy was slow in 3 of the 29 for whom figures could be obtained, as was motor development.

Symptoms

No Symptoms. Four patients were considered by parents and physicians to be entirely asymptomatic.

Fatigue. Some degree of exercise intolerance was noted in 30 cases. It was marked in 20, with 1 or 2 flights of stairs or blocks on the level as the limit of activity before obvious fatigue appeared. Six had been restricted in effort since the discovery of a cardiac murmur.

Shortness of Breath. Objective or subjective evidence of respiratory difficulty on moderate exertion was present in 21. Paroxysmal nocturnal dyspnea did not occur.

Profuse Perspiration. Inquiry concerning this phenomenon was made of 26 patients. It was present in 13, with intolerance of bed clothing even during cold weather and the necessity of a change of sleeping garment during the night.

Syncope and Other Central Nervous System Symptoms. Episodes of syncope occurred in 6 individuals. On one occasion after vigorous exercise another exhibited profound muscular weakness, dyspnea, and slight cyanosis. Ten patients complained of headache, dizziness, or visual disturbances.

Cyanosis. Blueness of the lips had been noted in 5 patients, but in only 1 had it been appreciated prior to the discovery of a cardiac abnormality.

Forcible Cardiac Action. Two children were noted by the parents to have prominent precordial heaving.

Cardiac Failure. One child in the series experienced 2 episodes of heart failure before his third birthday. He was then maintained on digitalis for 4 years and had no symptoms in spite of normal activity. Three other children were known to have been decompensated. One
adult entered the hospital in severe failure and responded poorly to medical management.

**Chest Pain.** One patient complained of a sensation of tightness across the chest following exertion. Five had pain or discomfort in the heart area on occasion. It was not severe and did not radiate in any instance.

**Paroxysmal Tachycardia.** Three had one or more episodes of rapid heart action. In 2 it was shown to be paroxysmal atrial tachycardia.

**Epistaxis.** Frequent severe nasal hemorrhage was present in 6 patients, and 1 required carotid artery ligation.

**Paresthesias.** Disturbances of sensation in the lower extremities were present in 3 patients.

**Sudden Death.** Two patients in the group died suddenly some time after discharge from the hospital and another expired suddenly while in the hospital awaiting surgery, all without apparent precipitating cause.

### Physical Manifestations

The physical findings were relatively uniform. Only 3 patients were below normal in development and size; none was superior. Abnormal pulsations of the carotid arteries were present in 3, and thoracic asymmetry in 3.

**Cardiac Enlargement.** As determined by percussion or location of the apical impulse, cardiac enlargement to the left was present in 9, and was marked in only 1.

**Throb.** In only 2 patients was a systolic thrill absent. In the others it was most intense in the region of the systolic murmur and was transmitted to the right side of the neck as a rule.

**Systolic Murmur.** A loud, harsh systolic murmur was audible in each case, best heard in the second, third or fourth right interspaces near the sternal border. Transmission was wide and in nearly every instance the murmur could be heard over the neck vessels and the entire right hemithorax. Three patients had, in addition, a systolic blow localized to the apex region and another a harsh apical systolic murmur. In a fifth, a harsh systolic murmur could be heard in the second and third left interspaces and it differed in character from that heard to the right of the sternum; this boy had mild pulmonary stenosis and a patent ductus arteriosus.

**Diastolic Murmur.** Early, blowing diastolic murmurs were present in the aortic area in 6, the pulmonic area in 2, and at the apex in 2. In the child with a patent ductus there was a faint blow throughout diastole in the second left interspace.

**Aortic Second Sound** This was normal in 17 patients, somewhat accentuated in 6, and inaudible or faint in 14.

**Peripheral Pulses.** There was inequality of radial pulsations and normal femoral pulsations in 1 patient. In 3 the radial pulse was small bilaterally and the femoral barely palpable. Still another had a good right radial pulse, a weak left, and no femoral pulsations. In 7 additional patients the femoral pulsations were absent or markedly decreased in force. Of these 12 individuals, 9 showed some evidence of coarctation of the aorta.

**Blood Pressure.** This was determined both by sphygmomanometer and, in 34 cases, by direct measurement through a cannula in a peripheral artery. In general, the former gave a higher reading, the increase in systolic pressure ranging to 38 mm. Hg, with an average of 17 mm., while that in diastole ranged to 44 mm. (average 12 mm). Three individuals had a higher diastolic pressure on direct measurement than by cuff (10, 30, and 34 mm. Hg). If the direct measurements are accepted, the pulse pressure ranged from 12 to 60 mm. Hg, and in only 4 patients was it less than 25 mm.

The pressure, although normal, was higher in the arms than in the legs in 4 patients and of the same order of magnitude in 3. In 3 children with normal or slightly elevated brachial artery pressure, no reading could be obtained in the legs. Comparison in all these cases was made with cuff pressures.

**Erythema of Digits.** Distinct, chronic redness of the skin over the distal phalanges of the digits was found in 1 child. There appeared to be slight hypertrophy of the tissue in these areas.

**X-ray and Fluoroscopy**

The pulmonary vascular markings were prominent in 3 patients and normal in the rest. The main pulmonary artery and its
FIG. 1. Enlarged heart with prominent rounding of left border.

FIG. 2. More marked enlargement and rounding of left border.

branches were either normal or their contour was ill-defined. Pulsations were not remarkable.

The heart contour varied in the postero-anterior projection. In 3 patients there was a

widened supracardiac vascular shadow with straight borders. The heart was enlarged and there was prominent rounding of the left border (fig. 1). Each had a localized coarctation of the aorta and 1, in addition, a hypoplastic descending thoracic aorta. In a fourth

FIG. 3. Ascending aorta prominent and left border lengthened and rounded.

FIG. 4. Prominent ascending aorta, lengthened and rounded left border, and additional bulge of right atrial appendage.
patient without coarctation, these features were accentuated (fig. 2). The ascending aorta was prominent to the right of the spine and the left border was lengthened and somewhat rounded in 12 patients (fig. 3). The child with pulmonary stenosis and a patent ductus arteriosus had, in addition, a bulge in the region of the right atrial appendage (fig. 4). Four adults had enlarged hearts and prominence of the ascending aorta and of the aortic knob; in 3 the apex was depressed (fig. 5), in the other, the left border was squared (fig. 6). The remaining 17 patients had a normal or slightly narrowed suprachordal shadow, slight or no
cardiac enlargement, and a rounded left border (fig. 7). In 16 patients there was some enlargement of the right ventricle, the level of encroachment of the anterior heart shadow on the retrosternal space in the right anterior oblique projection being used as the criterion.

Some degree of left atrial enlargement was present in 7 patients; it was graded as 1 plus in 4, 2 plus in 1, 3 plus in 2.

The most consistent roentgen abnormality was dilatation and anterior prominence of the ascending aorta in the left anterior oblique view (fig. 8). It was definite in 29, questionable in 1, and absent in 7.

Only 1 patient had an abnormality of the thoracic bony structures; in this case notching of the inferior border of several ribs was associated with coarctation.

Four patients, 3 with coarctation, were demonstrated to have marked posterior indentation of the esophagus at the level of the transverse arch in the left anterior oblique view. The posteroanterior esophagram showed indentation of the left border in this area.

Calcification of the aortic valve was visible in 2 patients.

**Electrocardiogram**

The tracings of 7 patients were normal. Five had nonspecific changes of S-T segment or T wave. Two had left bundle-branch block. One showed the pattern of a septal infarct. The remaining 22 showed left ventricular hypertrophy. The rhythm was normal in all except 1 adult with left bundle-branch block who had 2:1 atrial flutter.

**Cardiac Catheterization**

Cardiac output was found to be within normal limits in all patients in whom oxygen consumption could be measured. In only 1 was it determined both at rest and during exercise; it was found to rise only slightly during the latter phase.

In no case was there evidence of an intracardiac shunt. One child had a significant rise in the oxygen content of pulmonary artery blood.

Four in the series had a definite although slight gradient in systolic pressure between right ventricle and pulmonary artery (6, 10, 12, and 16 mm. Hg), which was constant on repeated withdrawal of the catheter tip from vessel to chamber. One of these was the boy with a left-to-right shunt at the pulmonary artery level; his pulmonary pressure was somewhat elevated (38/10 mm. Hg).

Six additional patients had hypertension in the pulmonary artery, the pressures being 30/20, 45/20, 42/25, 30/10, 110/60, and 80/68 mm. Hg. The pulmonary venous capillary pressure was measured in only 20 patients, among them 3 with pulmonary hypertension. The mean pressure was 10 mm. or more in 9.

**Thoracic Aortography**

Successful visualization of the whole thoracic aorta was secured in 4 patients. In all, the ascending aorta was dilated and a definite area of coarctation distal to the left subclavian artery could be seen in 3. In the fourth the descending aorta was hypoplastic.

In 1 infant the catheter tip could not be manipulated into the aorta. Injection of contrast material was made into the right subclavian artery. The innominate artery was seen to be greatly dilated but opacification of the arch was unsatisfactory. The ascending aorta of another individual could not be entered and the opaque substance was injected into the distal portion of the arch; this section of the vessel was found to be dilated and below it was an area of coarctation; the descending aorta was also dilated for a short distance.

In the section on X-rays it was mentioned that 3 patients had posterior indentation of the barium-filled esophagus. The aortogram in each indicated that this was due to compression by the dilated arch.

**Brachial Artery Tracings**

Of the 34 tracings obtained, not one could be considered normal. A slow rise to the peak of systole was almost universal. Five types of curve were seen: (1) a definite anacrotic notch or slur; (2) a double systolic peak; (3) a notch or slur on the dicrotic limb near the summit, with a distinct dicrotic incisura below; (4) a smooth curve, with a particularly small pulse
pressure; (5) a curve with many vibrations on both limbs and a very small pulse pressure.

The curves were analyzed in relation to various other data: Character of aortic second sound, electrocardiogram, roentgen findings, site of obstruction, degree of stenosis found at operation. The only significant correlation was association of types 4 and 5 with stenosis of the greatest severity.

**Discussion**

The diagnosis of aortic stenosis in only 3 infants (age under 2 years) is somewhat surprising in view of the relatively large number in this age group who underwent cardiac study. One explanation involves the common failure of the physician to place the stethoscope head over the right second and third interspaces. Another is that the physical findings may not suggest the diagnosis until after a period of time. Five of these patients did not have evidence of aortic stenosis when they were first studied. None had the typical murmur and thrill. Each was catheterized and nothing of significance found. At subsequent re-examinations the physical findings changed. Initially, there was a systolic murmur to the left of the sternum in 4 patients and over the xiphoid in 1. As each individual grew older the murmur changed, becoming most prominent in the aortic area, and then louder and harsher. In 3 of these patients, electrocardiographic evidence of left ventricular hypertrophy was present before the typical signs of aortic stenosis developed. The explanation of these changes is not entirely clear. They are probably due to change in position of the aortic valve and to relative increase in severity of the stenosis with increase in body and heart size.

The site of obstruction is known in 21 cases. It was valvular in 18. If these are a fair sample of the entire series, only 1 or 2 additional patients had infundibular stenosis.

The anomalies associated with aortic stenosis in this group are of interest. Pulmonary stenosis of physiologically insignificant degree occurred in 4 patients. One of these also had a patent ductus arteriosus. A ventricular septal defect was present in 1 boy who had infundibular stenosis. In another patient with infundibular stenosis only the left coronary artery was found, with a healed infarct of the interventricular septum. Coarctation of the aorta was the most common accompanying lesion; it was present in 8. A patent ductus was also found in 1 of these 8 patients. All patients with coarctation should therefore be carefully examined for evidence of aortic stenosis.

The greater the degree of obstruction the greater the cardiac burden, and the greater the cardiac reserve the greater the ease with which the burden is carried. Fatigue on moderate exertion indicates an insufficient transport of oxygen to the muscles, because the left ventricle is not able to meet the demands made upon it. Shortness of breath also indicates an insufficient reserve. Increase in rate of cardiac contraction does not meet the need for oxygen, so more air per unit of time is brought to the alveoli by increased respiratory rate. Actual dyspnea can be accounted for by great respiratory demand or by pulmonary edema consequent upon left ventricular failure. Syncope and other central nervous system symptoms may be explained on the basis of a temporary inadequacy of oxygen supply to cerebral tissue, due to a transient failure of the left ventricle to maintain output. The cerebral symptoms may result from other causes, such as aortic or carotid reflexes initiated by dilatation of the ascending aorta or conditions of aortic flow. Chest pain is due to myocardial ischemia because of inadequate coronary flow. Cardiac output may be normal and coronary flow normal, but because of hypertrophy of the left ventricular fibers the demand is greater than normal. In this series, pain was not so frequent or severe as in a similar group with acquired aortic stenosis. Sudden death indicates that left ventricular output can be acutely diminished to a degree that myocardial function is no longer possible. That the circumstance need not be one of severe stress is shown by the sudden collapse and death of one patient while walking across a schoolyard. Another was walking along the beach when he suddenly died. The third was in the hospital awaiting surgery, when she died without warning. A fourth child with undoubted aortic stenosis, but seen only in consultation and not included
in the series, also died suddenly during a period of quiet activity that had not been preceded by violent exertion. Carotid sinus reflexes rather than acute left ventricular failure may at times be the cause of sudden death.

The harsh systolic murmur in the second and third right interspaces must be due to the passage of blood beyond a narrowed outlet. A murmur in this location is found rarely in other congenital heart malformations. The early diastolic murmur in the aortic, pulmonic, or apical regions probably reflects a degree of aortic insufficiency. In one patient with an apical diastolic murmur, resection of the coarcted segment of the aorta was followed by disappearance of the murmur.

The abnormal roentgen features consist, in the main, of prominence of the ascending aorta, rounding or lengthening of the left cardiac border, and right ventricular or left atrial enlargement. Gross cardiac enlargement is not frequent. The dilatation of the ascending aorta may well be due to the same mechanism offered to explain poststenotic dilatation of the pulmonary artery. A jet of blood entering the vessel through a narrow orifice that is eccentrically placed strikes the wall and causes a localized systolic bulge. As time goes on, the constant trauma brings about generalized stretching. Rounding of the left cardiac border is due to hypertrophy of the left ventricular myocardium. Greater degrees of hypertrophy will depress the apex and cause lengthening of the border. Gross enlargement of the chamber appears when there is dilatation as well as hypertrophy. Left atrial enlargement is due to a back-pressure phenomenon. The patients with undoubted right ventricular prominence had pulmonary hypertension.

Pulmonary hypertension in the patient with an accompanying patent ductus might be explained on the basis of narrowed pulmonary vessels and increased resistance as a result of blood flow through the ductus. His pulmonary venous capillary pressure was normal. To account for pulmonary hypertension in the others presents some difficulty. Obviously, left ventricular failure would lead to incomplete emptying of the chamber with consequent overfilling and increased pressure in the left atrium and pulmonary veins. A higher pressure head would then be necessary on the other side of the capillary bed. However, none of the patients with pulmonary hypertension showed evidence of left ventricular failure. Rather than dynamic failure of the left ventricle, a failure in capacity may be considered. Because of obstruction to outflow the chamber wall undergoes concentric hypertrophy, which, in the absence of dilatation, eventually diminishes the capacity due to encroachment of the wall. At some point the capacity decreases minutely, so that ventricular systole occurs before the left atrium has completely emptied. With repetition of the process the residual in the left atrium and pulmonary veins becomes substantial. With resistance to outflow the pressure rises in the left atrium and pulmonary veins and is accompanied by a rise in pressure on the other side of the pulmonary capillary bed. Vascular changes diminish the caliber of small pulmonary vessels; increased resistance results and leads to further hypertension. Thus, in a way, the responsibility for maintaining the circulation is gradually shifted, more and more, to the right ventricle.

We have studied a large number of brachial artery tracings of patients with various types of congenital and acquired heart disease and of many with no heart disease or insignificant lesions. In those with aortic stenosis, whether congenital or acquired, certain abnormalities have been noted in the majority that are not pathognomonic. Some are seen in other congenital and acquired conditions, although not so frequently. Their genesis is not clear. Experimental studies are under way which, it is hoped, will shed some light upon the problem.

Diagnosis

To the present time we have studied several hundred patients, proved at operation or autopsy to have aortic stenosis, either congenital or acquired. As a result of this experience we believe that an individual with a loud, harsh systolic murmur in the region of the second and third right interspaces near the sternum that is transmitted to the neck vessels and is accompanied by a thrill may be considered to have aortic stenosis. The diagnosis is
more certain if the electrocardiogram shows left ventricular hypertrophy, if there is dilatation of the ascending aorta on roentgen examination, and if there is an abnormal brachial artery tracing. Certain proof is established by successful left heart catheterization. This procedure entails the percutaneous puncture of the left atrium by a needle with passage of a small gage catheter through the ventricle and into the aorta. A gradient in systolic pressure from aorta to ventricle is pathognomonic of obstruction at or near the aortic valve. If the aorta is not reached, comparison of pressures in the left ventricle and the brachial artery is significant. Flow across the valve is a determinant in judging severity of stenosis, and simultaneous right heart catheterization with estimation of cardiac output is of great importance in some cases.

The author has had no personal experience with the procedure, but it has been accomplished in our laboratory in 350 individuals. On the basis of stand-by observation it appears unwarranted in those patients that are thought to have aortic stenosis. The diagnosis and the estimation of its severity can be made on data derived from routine studies and right heart catheterization.

The preoperative localization of the exact site of obstruction seems, at present, to be impossible. Absence of the aortic second sound has not been a constant feature in our cases with proved valvular stenosis. The character of the arterial pulse tracing has varied so widely that it appears to be of no diagnostic value. It was hoped that left heart catheterization would be of aid. In 1 patient in this series, however, proved to have infundibular stenosis, combined catheterization of right and left sides was performed. When the catheter tip was withdrawn from aorta to ventricle an abrupt transition was seen from a tracing typical of a great vessel to that of a high pressure ventricle. There was no intermediate zone of low systolic pressure and a ventricular curve as is sometimes seen in pulmonary stenosis.

Treatment

Aortic stenosis is the only congenital cardiac anomaly compatible with a relatively long span of years that requires restriction of activity. The dynamics of other defects, such as pulmonary stenosis, septal defects, coarctation of the aorta, permit the patient’s subjective response to exertion—muscle fatigue and shortness of breath—to insure a margin of cardiac reserve. Aortic stenosis constantly threatens acute coronary insufficiency. The margin between adequate and inadequate coronary flow is small; activity demanding greater cardiac output may quickly exhaust reserve and lead to sudden myocardial failure.

The development of a satisfactory surgical procedure for the relief of aortic stenosis has been described by Bailey.10 It had proved feasible in the correction of the congenital malformation in 7 adult patients. Its application in the child was delayed because the transventricular approach was thought to require a large chamber and because the transaortic approach would require a relatively huge ascending aorta.

The sudden death of 3 children within a short period of time made it obvious, however, that the risk of doing nothing might well be greater than the risk of operation. Surgery was therefore performed in the next patient, a 6-year-old boy in whom the diagnosis of significant aortic stenosis was made. The transventricular approach was used, a dilator being introduced into the left ventricle through an incision near the apex. It was passed into the valve area with considerable difficulty and opened. Immediately, the previously strong thrill over the ascending aorta became feeble. The procedure was tolerated extremely well, with cardiovascular changes no more severe than would be met during a simple thoracotomy.

Since that time operation has been recommended for all children found to have a physiologically significant lesion. A total of 19 patients have been operated upon, 10 of them children. Two were only 3 years of age.

There has been 1 operative death, a 36-year-old man in irreversible failure, who accepted surgery as a desperate measure. Cardiac arrest occurred before any definitive procedure could be carried out.

Another patient, age 18, was improved post-
operatively, in that his exercise tolerance was greater. Eight months following surgery, while dancing, right hemiplegia occurred, and 2 days later he died. At autopsy an area of necrosis was found in the left cerebral hemisphere, with a small particle of calcium in the center. There was no old or recent infection. The aortic valve orifice was adequate in size, but not calcified. Whether or not the calcified particle was an embolus from the valve could not be determined.

One patient, age 19, proved to have infundibular stenosis at operation, was studied by means of combined right and left heart catheterization before and after operation. The systolic gradient from left ventricle to aorta preoperatively was 140 mm. Hg. The cardiac output was 4 L./min. Postoperatively the gradient was 80 mm. Hg and the cardiac output was 2.4 L./min. Although the gradient was smaller, the cardiac output, too, was significantly less. She has shown no clinical improvement. Operation in this patient must be considered a failure.

In the remaining 16 patients, there has been objective evidence of improved flow across the aortic valve. In them the prominent systolic thrill and murmur over the ascending aorta became softer following commissurotomy, a common localized systolic jet-impulse disappeared. In 3 the thrill disappeared.

Symptoms were relieved in 14 of 15 patients. One was asymptomatic although he had been in cardiac failure in early life. Exercise tolerance and respiratory reserve improved, central nervous system manifestations and chest pain stopped, and, as a rule, weight gain has been accelerated in growing children.

The one patient, an 18-year-old girl, who has not improved symptomatically, was operated upon 2 months ago. She has developed signs of aortic insufficiency, and, after temporary improvement, her fatigue and dyspnea are now greater than preoperatively. Her postoperative care, however, has not been well supervised, in that ambulation was allowed too early and her activities following discharge were not controlled.

Aortic insufficiency as a result of operation is of great importance. In 2 additional patients an early blowing diastolic murmur appeared, but without other signs of aortic regurgitation, and symptoms definitely improved. They are not considered to have dynamically significant aortic regurgitation. Surgery was performed on 8 of the 11 patients with early diastolic murmurs on initial examination. One was the girl with infundibular stenosis who showed no improvement in physiologic data; neither systolic nor diastolic murmur changed. In 3 others, the systolic murmur greatly decreased in intensity, but there was no change in the diastolic murmur. In 4, the diastolic murmur could not be heard postoperatively; in them the improved mobility of the stenosed aortic valve seemed to have removed the element of incompetence.

Complications during and after operation have been surprisingly few. Of the living patients, 2 had a stormy postoperative course. One, the girl who now shows aortic insufficiency, was, through error, allowed out of bed in spite of evidence of cardiac failure. A number of thoracenteses were also done, probably unnecessarily. The other, a boy of 14, developed a severe wound infection that was slow to heal. In all, the procedure itself was extremely well tolerated, cardiac arrest or ventricular fibrillation being no source of concern. Ambulation was allowed on the tenth postoperative day in children, and they were allowed to go home on the same day.

It is certainly too early to make any sweeping statements about the operated group, beyond the fact that 14 of the 17 surviving patients have been symptomatically improved. We believe that, in all, the danger of sudden death has been removed. In the children it is possible that residual stenosis may become relatively more severe with increased growth. If so, a second operation may be successful.

The indication for surgical relief of congenital aortic stenosis is the presence of the physiologically significant lesion. It is significant if one or more of the following criteria is demonstrated: (1) troublesome symptoms such as fatigue, shortness of breath, syncope; (2) electrocardiographic evidence of left ventricular hypertrophy; (3) some degree of pulmonary artery hypertension or pulmonary
venous hypertension in the absence of other lesions; (4) a systolic gradient of 50 mm. Hg or more across the aortic valve, with a normal or decreased cardiac output.

The time of election for operation is the time that indications appear, whether it be at 3 months or 30 years. No one can tell when compensation may fail. There is no optimal age; there is only the age of opportunity.

SUMMARY

The clinical and physiologic data of 37 patients with aortic stenosis have been presented.

Fatigue, shortness of breath, and profuse perspiration were the commonest symptoms. Central nervous system manifestations and chest pain were infrequent. A systolic thrill and murmur in the second and third right interspaces were almost universal. Because they are found in this location very rarely in other malformations, they are of great diagnostic significance. Right heart catheterization is very helpful in ruling out other lesions. Left heart catheterization is diagnostic if a gradient in systolic pressure across the valve is demonstrated.

Differentiation of valvular from infundibular stenosis has not been possible in this series.

Because of the danger of sudden death, relief of severe obstruction is mandatory.

A satisfactory operative procedure is available for correction of valvular stenosis. It involves the dilatation of the narrowed orifice by means of an instrument inserted into the left ventricle.

Nineteen patients have been operated upon. There was one operative death and another died 8 months later. One patient, with infundibular stenosis, is unimproved; 1 is symptomatically worse; 1 had no symptoms prior to surgery. The remaining 14 have experienced gratifying relief of symptoms.

SUMMARIO IN INTERLINGUA

Es presentate datos clinic e physiologic ab 37 paciente con stenosis aortic.

Fatiga, dyspnea, e profuse transpiration eseva le symptomas le plus commun. Mani-

festaciones del sistema nervose central e dolores thoracic eseva infrequente. Un systolic fremito e murmure in le secunde e terti interspatios dextere eseva quasi general. Proque illos es rarissime in iste sito in altere malformationes, illos possede un alte grado de signification diagnostic.

Catheterisation dextero-cardiac es de grande adjuta in le exclusion de altere lesiones. Catheterisation sinistro-cardiac es diagnostic per le demonstration de un gradient de pression systolic a transverso le valvula.

Le differentiation inter stenosis valvular e stenosis infundibular non eseva possibile in le presente serie.

A causa del risco de morte subitanee, le alleviation de sever obstructiones es obligatori.

Un satisfacente technica chirurgic es disponibile pro le correction de stenosis valvular. Illo effectua le dilatation del restringite orificio per medio de un instrumento inserite in le ventriculo sinistre.

Dece-nove pacientes eseva operate. Il habeva un morte operatori; un secunde patiente moriva 8 menses plus tarde. Un del patientes—un caso de stenosis infundibular—monstra nulle melioration; in un altere le symptomas es pejor; un caso eseva sin symptomas ante le intervention chirurgic. Le remanente 14 patientes ha experienciate grados gratificante de melioration in lor symptomas.

REFERENCES

6 Reinhold, J., Rudhe, U., and Bonham-Carter,


The study was undertaken to determine whether or not training in the athlete altered the methods of oxygen transport employed during mild to moderately severe exercise.

Cardiac catheterization was carried out on 3 track men, 2 of whom were studied before training and again during training. Cardiac output, A-V oxygen differences, and pulmonary arterial pressures were obtained during rest and mild and moderately severe exercise. In addition, the effect of training on maximum breathing capacity and vital capacity was determined.

The cardiac output and arteriovenous O₂ difference during exercise was no different in the trained than in the untrained individual. Vital capacity was not changed by athletic training; however, the maximum breathing capacity was increased by training. Pulmonary arterial pressure increased in response to all grades of exercise regardless of training.

Training produced no difference in the way the individuals met the tissue demands for an increased supply of oxygen during exercise up to levels requiring 2 liters of oxygen intake a minute. Up to this level of oxygen consumption, the contributions of increased cardiac output and increased oxygen extraction from arterial blood to an increasing rate of oxygen consumption are roughly equal.

The results of this study indicate that athletic training does not change the method by which the athlete meets the tissue demands for an increased supply of oxygen during exercise up to levels requiring 2 liters of oxygen a minute.

Wechsler
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