Combined Aortic and Pulmonic Stenosis

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Although deformities of the aortic or pulmonary valves are among the commonest congenital cardiac malformations, lesions involving both valves have not been frequently reported. Five isolated cases were found after a review of the pertinent literature. In view of the rarity of such lesions and the unusual diagnostic and surgical problems presented by these patients, it seems worth while to report four cases with severe aortic and pulmonic obstruction seen within the past 2 years at the Children's Hospital Medical Center.

Report of Cases

Case 1

M. A., a 4-year-old Belgian girl, was admitted in May 1958 to the Children's Hospital Medical Center for cardiac surgery. A heart murmur was first noted at the age of 6 weeks. The child was retarded both physically and mentally, and was moderately dyspneic on exertion.

On physical examination her poor development was striking. There was no evidence of congestive heart failure. The cardiac impulse was felt maximally in the fifth left interspace in the anterior axillary line and had a heaving character. A grade III to IV stenotic systolic murmur, accompanied by a thrill, was heard best at the second left interspace, but was almost equally intense at the second right interspace. The second sound seemed single, maximal at the fourth left interspace, and decreased in intensity both at the aortic and pulmonic area. The phonocardiogram (fig. 1) showed the systolic murmur to be diamond-shaped with an early peak at the second right intercostal space and with a later maximum and longer duration at the second left interspace. The aortic component of the second sound, which was the only one recorded, was obscured by the murmur at the pulmonic area but was clearly visible at the aortic area. A mid-diastolic rumble was registered at the apex.

The electrocardiogram (fig. 2) showed a mean QRS axis of +180°, a prolonged P-R interval (0.21 second), and marked right ventricular hypertrophy. Radiologically (fig. 3), the heart was slightly enlarged with a predominant right ventricular contour. The pulmonary vasculature was within normal limits. Cardiac catheterization (table 1) indicated severe infundibular and valvular pulmonic stenosis with a small right-to-left shunt at the atrial level. Associated aortic stenosis was suspected clinically on the basis of the cardiac impulse and the loud early systolic murmur at the aortic area.

On May 24, 1958, the child underwent cardiac surgery by means of the pump-oxygenator. The diagnosis of aortic stenosis was confirmed by direct pressure measurements (table 1). The perfusion was started, the pulmonary artery was opened, and the pulmonic valve was found to be deformed and stenosed, leading to a narrow muscular infundibulum. Little could be accomplished by opening the pulmonic valve alone. During a 50-minute perfusion period, a pericardial patch was inserted into the right ventricular outflow tract continuing into the stem of the pulmonary artery. Because of the prolonged perfusion period, it was decided not to attempt relief of the aortic obstruction. The postoperative course was marked by hypotension and spontaneous hypothermia. Shortly after the operation, the patient developed ventricular fibrillation, which responded only temporarily to cardiac massage and electrical countershock. She died 2 hours postoperatively.

Autopsy

The heart weighed 100 Gm.; both ventricles, predominantly the right, were hypertrophied. The thickness of the right ventricle was 1.5 cm., that of the left ventricle 1.1 cm. An atrial septal defect of the secundum type, measuring 1.5 cm. in its greatest diameter, was demonstrated. The pulmonary valve measured only 2.2 cm. in circumference and the three leaflets were hypoplastic, irregular, and thickened. The circumference of the valvular ring had been increased to 3.3 cm. by the insertion of the pericardial patch. Muscular hypertrophy resulted in a narrowing of the infundibulum. The aortic valve was also stenotic and measured 2.0 cm. in circumference. Some

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subvalvular stenosis was noted. The ventricular septum was intact and the ductus arteriosus was closed.

Summary

This 4-year-old girl had a combination of severe aortic and pulmonic stenosis, both being predominantly valvular and to a lesser extent subvalvular in nature. Aortic stenosis was suspected on the basis of the heaving left ventricular impulse and the characteristics of the systolic murmur. At surgery both lesions were identified but only the pulmonic was partially corrected. The patient probably died as a consequence of the incomplete correction of the obstructive lesions.

Case 2

R. M., a 17-year-old girl, was first admitted to the Children's Hospital Medical Center in 1956. She was known to have had a heart murmur since birth. The child was asymptomatic except for slight fatigue on exertion. The findings were thought to be characteristic of severe pulmonic stenosis and cardiac catheterization (table 1) confirmed this clinical impression. A Brock valvotomy, performed in April 1957, resulted in alleviation of her mild symptoms, until the end of 1959. At that time fatigue, dizziness, and palpitations recurred. She was readmitted to the hospital in April 1960.

Physical examination revealed a prominent right ventricular and an even more marked, heaving, left ventricular impulse. The second sound was single and of normal intensity at the second left interspace. A systolic thrill and a grade V stenotic murmur were maximal over the manubrium sterni and the second right interspace, transmitting well into the neck. The murmur was slightly less intense at the second left interspace. A grade II diastolic rumble was audible at the apex. There were no signs of congestive heart failure. The electrocardiogram (fig. 2) revealed digitalis effect with somewhat lower right ventricular and slightly higher left ventricular potentials than 4 years previously. X-ray examination (fig. 4) showed progressive cardiac enlargement, involving mainly the right ventricle, and decreased pulmonary vasculature. At repeat right heart catheterization (table 1) evidence of severe valvular pulmonic stenosis was found. The end-diastolic pressure in the right ventricle was elevated. The cardiac output was significantly lower than in 1956. Because of the left ventricular cardiac impulse and the murmur suggesting aortic stenosis, a retrograde arterial catheterization was performed. A withdrawal tracing from the left ventricle to the aorta demonstrated a large systolic gradient at the supravalvular level. Cine-

Figure 1

Phonocardiogram, patient M. A. Peak intensity of systolic murmur occurred early at the second right interspace and later at the second left interspace. A diastolic rumble was present at the fourth left interspace.

angiograms from the left ventricle showed a constriction 1 to 1.5 cm. above the aortic valve. A left-to-right shunt was ruled out on the basis of dye-dilution studies.

Open-heart surgery was performed on June 8, 1960. Externally, a distinct circular narrowing was seen in the first part of the ascending aorta,
about 1 cm. above the valve ring. The heart was arrested with potassium citrate, and the aorta was opened across the constricted area, just above the noncoronary cusp. The edges of the right and left aortic valve leaflets were partially fused with the aortic wall; the posterior leaflet was non-adherent but puckered up. After an unsuccessful attempt to insert a Teflon patch, the aortic wall was repaired by bringing the vertical margins of the incision together in a transverse way. This only partially relieved the constriction. Subsequently, the pulmonary artery was opened, the commissures of the narrow and deformed valve were split and a pericardial patch was sewn in. After 1 hour of cardiac arrest and 2 hours and 15 minutes of body perfusion, the heart action was poor and blood pressure could not be maintained. The patient died on the table.

Autopsy

The heart weighed 750 Gm., both ventricles being grossly hypertrophied. The thickness of the right ventricle measured 1.5 to 2 cm.; the left, 2 to 2.5 cm. The great arteries were in the normal position, both atria were hypertrophied and dilated. The interatrial and interventricular septa were intact. The tricuspid valve was normal. The pulmonary valve consisted of three thickened and rigid cusps. Before insertion of the patch, it could hardly have admitted an index finger. The first segment of the pulmonary artery showed post-stenotic dilatation. The mitral valve was normal, but since the hypertrophied interventricular septum protruded into the small left ventricular cavity, one could assume that some degree of obstruction to diastolic inflow may have existed. The supravalvular aortic stenosis had a complex architecture, very similar to that described by Morrow et al. in their case 1; the right and left leaflets had a pouch-like aspect as the lateral parts of their free margins were fused with the aortic wall, leaving a central opening at the top. Additional openings in each of these cusps, leading into the coronary sinuses, were noted. The
COMBINED AORTIC AND PULMONIC STENOSIS

Table 1

<table>
<thead>
<tr>
<th>Case no.</th>
<th>RA</th>
<th>RV</th>
<th>PA</th>
<th>PW</th>
<th>LV</th>
<th>Ao</th>
<th>SA</th>
<th>Cardiac index (L/min./M²)</th>
<th>Shunt</th>
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<td>130/65</td>
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<td>B.H.</td>
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<td></td>
<td></td>
<td></td>
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<td>1-to-l atrial shunt (5.2</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>L/min./M²)</td>
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RA, right atrium; RV, right ventricle; PA, pulmonary artery; PW, pulmonary wedge pressure; LV, left ventricle; Ao, aorta; SA, systemic artery; m, mean pressure.

*Pressure taken on the operating table, at which time the systolic pressure in ascending aorta was 70 mm. Hg.
†Pressure just above the aortic valve but below the supravalvular stenosis.
‡A posteriori it became evident that this was the pressure in the single ventricle.
§Desaturation was thought to occur at atrial level, whereas in fact the r-to-l shunt was at ventricular level.

posterior leaflet was larger than normal, flabby, and puckered up; it was not fused with the aortic wall. Although the whole supravalvular region was narrowed, the main site of obstruction was a band of fibrous tissue at the upper rim of the sinuses of Valsalva, where the free margins of the right and left leaflets fused with the aortic wall. The coronary arteries were huge, the left one measuring 8 mm. in diameter, just beyond the ostium.

Summary

In this 17-year-old girl, a combination of severe valvular pulmonic and supravalvular aortic stenosis was found. At the age of 13 years, pulmonic stenosis was diagnosed, and a Brock valvotomy was performed. When she was readmitted in 1960, associated aortic stenosis was suspected clinically. Right and left heart catheterization demonstrated the presence of a double lesion and localized the site of the stenoses. At surgery the obstruction could not be completely relieved and the patient died.

Case 3

R. McM., a boy known to have a heart murmur since the age of 6 months, was first admitted to the Children's Hospital Medical Center in 1952 at the age of 3½ years. The parents had noted cyanosis, dyspnea on exertion, and occasional squatting. The main findings related to the heart were marked cyanosis, clubbing, and a grade IV systolic murmur at the third left interspace. The electrocardiogram (fig. 2) showed P-pulmonale and right ventricular hypertrophy. X-ray examination was reported to show cardiac enlargement involving both ventricles, a prominent main pulmonary artery, and diminished pulmonary vasculature. In spite of the somewhat atypical radiologic findings, the tentative diagnosis of tetralogy of Fallot was made.

Because of increasing symptoms, a Brock valvotomy was performed in 1953 without prior catheterization. At operation the aorta was seen to arise anteriorly and to the left of the pulmonary artery. A catheter inserted through the right ventricular wall could easily be advanced into the pulmonary artery; a systolic pressure gradient between right ventricle and pulmonary artery of approximately 140 mm. Hg was demonstrated. After the operation symptomatic improvement, lasting 2 to 3 years, ensued.

Because of an increase in symptoms, however, the boy was readmitted in 1957. Slight cyanosis at rest and a cardiac impulse suggestive of combined ventricular hypertrophy were noted. A grade IV stenotic murmur, accompanied by a thrill, was maximal at the second left interspace and was heard faintly at the second right interspace. A grade II diastolic rumble was audible at the apex. The electrocardiogram had not changed signifi-
cantly since 1952. X-ray examination (fig. 5) showed considerable cardiac enlargement, a bulging middle left heart border and decreased pulmonary vasculature. At cardiac catheterization (table 1), a systolic pressure of 250 mm. Hg was found in the right ventricle; neither the pulmonary artery nor the aorta could be entered. In view of the very large systolic pressure difference between right ventricle and systemic artery, severe pulmonic stenosis with intact ventricular septum was diagnosed. It was assumed that the arterial unsaturation resulted from a right-to-left shunt at the atrial level.

In May 1958, the child was operated upon by means of extracorporeal circulation. The right ventricle was opened and, in effect, a single ventricle was encountered. A diaphragmatic obstruction in the pulmonary outflow tract was relieved by cutting out fibromuscular tissue. Then a finger could easily be passed upward through a normal valve into the pulmonary artery. An attempt was made to construct an interventricular septum with a pear-shaped piece of Ivalon. Mattress sutures through the anterior and posterior ventricular wall were used to anchor the lower two thirds of the patch into place. The upper third of the patch was fixed to the right side of the upper crescent-like remnant of the septum. After closing the ventriculotomy and stopping the body perfusion, the heart action was unsatisfactory, and oozing from the surfaces was considerable. The patient died a few hours later in a low output state.

**Autopsy**

The aorta arose anteriorly and to the left and the pulmonary artery posteriorly and to the right in a vascular arrangement typical of corrected transposition. The heart was grossly hypertrophied and weighed 425 gm. The right ventricle measured 2.7 cm. in thickness, the left ventricle up to 3 cm. The right atrial wall appeared thickened, and the foramen ovale was probe-patent. The tricuspid valve was normal. The pulmonary valve measured 5.5 cm. in circumference. Its outflow tract had been reamed out, and the degree of the original stenosis could not be accurately assessed. After the Ivalon patch was removed, it became evident that there was no ventricular septum, except for a large band of muscle resembling a crista supraventricularis. This extended from the anterior aspect in a posterior and superior direction and separated the aortic from the mitral valve. The right ventricle had a finer trabeculation than the left ventricle. The mitral valve was normal. The left ventricular outflow tract had an infundibular architecture, was markedly narrowed, and hardly admitted the tip of the index finger. The aortic valve was normal and measured 0.2 cm. in circumference.

**Summary**

In the case of this 9-year-old boy with corrected transposition, single ventricle and severe subvalvular pulmonic and aortic stenosis, the correct diagnosis was not made ante mortem. The systolic murmur was faint at the second right interspace and did not suggest aortic stenosis. The electro-
COMBINED AORTIC AND PULMONIC STENOSIS

Table 2

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Sex</th>
<th>Age (yr.)</th>
<th>Cardiac Impulse</th>
<th>Physical findings</th>
<th>Electrocardiogram</th>
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<td>M.A.</td>
<td>F</td>
<td>4</td>
<td>Combined with LV dominant</td>
<td>Grade IV* at 2LIS† (slightly less at 2RIS)</td>
<td>Grade II, QRS-axis 180° RVH</td>
</tr>
<tr>
<td>R.M.</td>
<td>F</td>
<td>17</td>
<td>Combined with LV dominant</td>
<td>Grade V at 2RIS (slightly less at 2LIS)</td>
<td>Grade II, P. pulmonale PR .24 sec, QRS-axis + 120° RVH, Digitalis effect</td>
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<tr>
<td>R. McM.</td>
<td>M</td>
<td>9</td>
<td>Combined with equal LV and RV</td>
<td>Grade IV at 2LIS (faint at 2RIS)</td>
<td>Grade II, P. pulmonale PR .13 sec, QRS-axis + 105° RVH</td>
</tr>
<tr>
<td>B.H.</td>
<td>F</td>
<td>9</td>
<td>Combined with LV dominant</td>
<td>Grade IV at 2LIS (absent at 2RIS)</td>
<td>Grade II, PR .14 sec, QRS-axis + 50° Combined RVH and LVH, Negative T in V5 and V6</td>
</tr>
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</table>

*All murmurs graded from I to VI.
†2LIS, second left interspace parasternally; RVH, right ventricular hypertrophy; LVH, left ventricular hypertrophy.

cardiogram showed pure right ventricular hypertrophy. The left ventricular component in the cardiac impulse was not fully appreciated. Since the associated aortic stenosis was unsuspected, the large pressure difference between the right ventricle and the systemic artery was attributed to severe pulmonic stenosis with intact ventricular septum. The patient died because the unrecognized aortic stenosis was unrelied, and a ventricular septum could not efficiently be constructed.

Case 4

B. H., a 9-year-old girl, born after a normal pregnancy and delivery, had no cardiac symptoms in infancy and early childhood. On her first physical examination, at the age of 3 years, a heart murmur was noted. She was admitted to the Children's Hospital Medical Center for cardiac evaluation in 1957, at the age of 7 years. The patient appeared to be well developed, without signs of congestive heart failure or cyanosis. The cardiac impulse was predominantly left ventricular in character. A systolic thrill and a grade IV stenotic, systolic murmur were maximal at the second left interspace, poorly transmitted to the second right interspace. The second sound was faint at all areas, but best heard at the lower left sternal border where it was split. A grade II diastolic rumble was heard at the apex.

The electrocardiogram (fig. 2) showed right ventricular hypertrophy and left ventricular hypertrophy with a "strain" pattern. X-ray examination (fig. 6) showed moderate cardiac enlargement with a right ventricular contour and markedly increased pulmonary vasculature. The main pulmonary artery could not be identified at fluoroscopy. Cardiac catheterization was incomplete and revealed the presence of a left superior vena cava, elevated systolic pressure in the right ventricle (87/6 mm. Hg) and an atrial left-to-right shunt. The pulmonary artery was not entered. No definite conclusions were reached, but the clinical diagnosis of aortic stenosis, in addition to pulmonic stenosis and a left-to-right atrial shunt, was entertained.

When the child was readmitted in 1959, the only symptom was mild exertional dyspnea and the clinical findings had not changed significantly. At catheterization (table 1) the catheter could be advanced from the right atrium into the left atrium and the left ventricle where the systolic pressure was very high. The pressure in the right ventricle was also markedly elevated. The pulmonary artery could not be entered. A consistent increase in oxygen saturation of approximately 17 per cent was found at right atrial level. Cineangiograms from the right ventricle and the left atrium outlined the position of the great arteries and showed the pulmonary artery coming off the right ventricle, posteriorly and to the right of the aorta, in a fashion typical of corrected transposition.

At open-heart surgery in April 1960, the predicted great vessel arrangement was found and
an atrial septal defect was palpated from the right atrium. After the heart was arrested with potassium citrate, the ascending aorta was opened and a normal aortic valve was seen. Three quarters of an inch below the annulus, however, a tight stenosis, estimated at 5 to 6 mm. in diameter and consisting of fibromuscular tissue was found. This could be stretched with a clamp and further dilated with the finger. Between the aortic valve and the sub-valvular stenosis, a small ventricular septal defect of about 5 mm. in diameter was seen and readily closed. The pulmonary artery was opened and a moderately severe valvular stenosis was found that was corrected by splitting one partially fused commissure. Repeated attempts to discontinue the perfusion were unsuccessful; every time the cannulae were clamped, aortic pressure dropped after 20 to 30 seconds. It seemed obvious that the upper two thirds of the greatly thickened left ventricle did not participate in systole. The patient died on the table. Autopsy permission was not obtained.

Summary
This 9-year-old girl had corrected transposition of the great vessels, atrial septal defect, small ventricular septal defect, subvalvular aortic stenosis, and valvular pulmonic stenosis. The diagnosis was established preoperatively. The main lesion was the very severe subvalvular aortic stenosis resulting in predominant left ventricular hypertrophy, reflected in the physical and electrocardiographic findings. An apparently satisfactory anatomic repair of both the aortic and pulmonic stenoses was achieved, but adequate heart action was not re-established after stopping the body perfusion.

Discussion
It is obvious that these four cases of combined aortic and pulmonic stenosis do not represent a homogeneous group. In the first two patients the great arteries were in the normal position; the clinical and physiologic picture was that of an obstructive lesion, the pulmonic stenosis being predominant in the first patient, the aortic stenosis in the second. Pathologically, dominant right ventricular hypertrophy was observed in the former and left ventricular hypertrophy in the latter instance. The remaining two patients had corrected transposition of the great arteries and both had other associated anomalies, which altered the clinical and physiologic picture. One (case 3) had a functional single ventricle with tight pulmonic stenosis, resulting in a

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right-to-left shunt. The last patient (case 4) with predominant aortic stenosis, had an atrial septal defect with a moderately large left-to-right shunt. The sites of obstruction and their combinations varied greatly, as shown in table 2. Of the pulmonic stenoses, 3 were valvular and one subvalvular. One aortic stenosis was valvular, one supravalvular, and two were subvalvular.

Notwithstanding these anatomic and physiologic variations, the patients had several features in common, which will be further analyzed in respect to their diagnostic value.

The physical examination proved to be very informative, in that in three patients (cases 1, 2, and 4) it supplied the principal reason to suspect an associated left-sided lesion in the presence of a clinical picture otherwise suggesting pulmonic stenosis. All three had, in addition to the expected right ventricular impulse, a left ventricular heave, incompatible with uncomplicated pulmonic stenosis. In two of these the systolic murmur was very well heard at both the second left and second right interspaces. In case 1 phonocardiography demonstrated that the diamond-shaped murmur was short with an early systolic peak at the aortic area, whereas it was longer with a mid-systolic maximum at the pulmonic area. These are the phonocardiographic patterns usually associated with aortic and pulmonic stenosis, respectively.7 In the two patients with corrected transposition of the great vessels, the characteristics of the systolic murmur were less helpful, probably because of the unusual position of the semilunar valves.8 All patients had an easily audible mid-diastolic rumble at the apex, which only in one instance (case 4) might possibly have been accounted for by a left-to-right shunt. Similar diastolic murmurs are often heard in other conditions with concentric left ventricular hypertrophy, especially in coarctation of the aorta,9 congenital aortic stenosis,10 and obstructive cardiomyopathy.11 It is probable that the hypertrophied interventricular septum, bulging into a small left ventricular cavity, produces some degree of obstruction to diastolic inflow. At autopsy this anatomic arrangement could distinctly be demonstrated in one of our cases (case 2). An apical diastolic rumble is not heard in patients with pure pulmonic stenosis; thus, its presence may suggest an associated left-sided lesion.

The electrocardiograms showed P-pulmonale in two, right axis deviation (+180°, +120°, and +105°) in three, and right ventricular hypertrophy in all four patients. Although marked left ventricular hypertrophy was anatomically verified in every instance, only in one case (case 4) was it clearly reflected in the electrocardiogram, which showed biventricular hypertrophy and a "strain pattern" in the left precordial leads. The other three children had evidence of marked right ventricular hypertrophy, but unlike the electrocardiogram commonly associated with severe isolated pulmonic stenosis, the transitional zone was early and abrupt: a tall R wave in V1 was followed by a deep S wave in V2. This was also noted in previous case reports and the deep S wave in V2 was interpreted as indirect evidence of left ventricular hypertrophy.

Cardiac enlargement was a consistent radiologic finding, and in all patients it was thought to involve predominantly the right ventricle. Suggestive evidence of left ventricular enlargement was found in three cases, but in none was it convincing enough to point conclusively to an associated left-sided lesion. The pulmonary vasculature was within normal limits in one patient, decreased in two, and markedly increased in the last patient (case 4) who had an atrial left-to-right shunt.

In the final diagnosis cardiac catheterization plays an important role. In three patients a double lesion was suspected clinically, and in two of these it was proved by combined right and left heart catheterization. In three patients the presence of aortic stenosis was confirmed at the time of operation by direct pressure measurements.

In one patient with corrected transposition of the great vessels, the diagnosis was not made during life. The right ventricular systolic pressure was so far above the systemic artery pressure that the diagnosis of pulmonic...
stenosis with intact ventricular septum was made. The presence of a single ventricle and the association of aortic stenosis were not recognized at cardiac catheterization, because of the incompleteness of the study (neither of the great vessels nor the left ventricle was entered) and a misinterpretation of the dye-dilution curves. Furthermore, the statistical probability of the association of ventricular septal defect with corrected transposition of the great vessels was not well known in 1957. This stresses the importance of the observation that pulmonic stenosis with an intact ventricular septum should not be diagnosed on the basis of a pressure difference between the right ventricle and a systemic artery alone.\textsuperscript{12}

From the viewpoint of surgical correction there can be little doubt that one is dealing with a complex situation that forbids surgical attack, or at least carries a very high risk. Surgical relief of a pulmonic obstruction alone has an operative mortality approaching zero, and surgical correction of congenital aortic stenosis alone carries a mortality rate of only a few per cent. Such experiences (encountered by many surgeons) would naturally lead one to think that these two manipulations (which are not particularly difficult procedures) could be combined into a single operation for the type of case under discussion, with a reasonable probability of successful outcome. It has been our bitter finding, however, to have a fatal outcome in each of the four instances summarized here.

In retrospect, the surgical handling of our four patients could have been improved in several respects, and might possibly have led to some survivors: 1. The use of elective cardiac arrest by the potassium citrate method is reasonably safe if it is not employed for more than a half hour. Its extension far beyond this time in two cases suggested that myocardial damage was an important element leading to fatal outcome. 2. In one patient the outstanding lesion was a marked constriction just above the aortic valve that we did not relieve sufficiently. Such a narrowing could be appropriately enlarged by the adequate use of an inlay patch of plastic material or aortic homograft. 3. It becomes obvious that it is unsatisfactory to attack only one of the obstructions in the pulmonic and aortic valves. If the aortic valve alone is opened up (and there is no interventricular septal defect), there is very likely to be an insufficient amount of blood entering the left side of the heart; hence, severe peripheral hypotension leads to death. Contrariwise, if only the pulmonic obstruction is relieved, there may be a flooding of blood into the lungs, leading to pulmonary edema. These hearts, obstructed in both outflow tracts, have obviously carried on life with a delicate balance between the two sides. Surgical relief of one side alone will probably lead to disaster; success can only be hoped for by simultaneous relief of both blocks.

It is important to realize that many of these patients have interventricular septal defects, which range from small orifices to enormous openings, giving essentially a "single ventricle." The former can be easily closed. Indeed, the latter can be occasionally successfully corrected by construction of a new septum, but this step frequently leads to fatality because a single ventricle (while seemingly large) is divided into two chambers, each of which is too small to function in a satisfactory manner.

Summary

Combined aortic and pulmonic stenosis is a rare lesion. The clinical, hemodynamic, and pathologic findings in four patients are presented. The presence of a forceful left as well as right ventricular impulse (in four patients), the difference in the character of the ejection murmur at the second left as opposed to the second right interspace (in two patients), and a diastolic rumble at the apex (in four patients) were characteristic. The electrocardiogram showed right ventricular hypertrophy in all patients; additional and definite left ventricular hypertrophy was found in one. Radiologically, there was evidence of right ventricular enlargement in all and suggestive left ventricular enlargement in three.

Simple valvular stenosis of both valves was found in only one patient, and valvular pul-
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monary and supravalvular aortic stenosis in another. The two remaining patients, those with corrected transposition of the great arteries, had complicated lesions including a single ventricle in one and small ventricular septal defect in the other.

Attempts at surgical correction were unsuccessful in all cases.

References

If it is beyond question that the veins of the neck are bilaterally congested and it is equally beyond doubt that the liver is not enlarged, an obstruction of the superior cava must be considered. Its diagnosis will rest upon the discovery of anastomotic veins and upon failure to induce the veins of the neck to pulsate. A second reason for the same discrepancy is atrophic cirrhosis of the liver in a congested patient; the diagnosis of the liver condition will then turn upon the degree of hardness, and perhaps irregularity, of the liver margin.

There is the reverse case: an engorgement of the liver has been present for a long time and the venous spaces within have become permanently dilated and its substance a little or much fibrosed. In such, even if the signs of increased pressure in the veins greatly decline, the size of the liver may not decrease much or proportionately. It is a discrepancy which previous knowledge of the course of the malady explains.—Sir Thomas Lewis. Diseases of the Heart. New York, The MacMillan Company, 1933, p. 17.
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