VENTRICULAR SEPTAL DEFECT WITH SHUNT FROM LEFT VENTRICLE TO RIGHT ATRIUM

BACTERIAL ENDOCARDITIS AS A COMPLICATION*

BY

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During a two-year period we have seen six patients with malformations of the ventricular septum resulting in the shunting of blood between the left ventricle and the right atrium. Surgical closure of the defect has been successful in all. A history of bacterial endocarditis was established in two patients and strongly suspected in two others before operation. The surprisingly large number of patients with a malformation previously considered rare and the unusually high incidence of endocarditis as a complication prompted a review of the anatomical and clinical features of this anomaly.

There have been 26 patients reported with left ventricular to right atrial shunts† on whom relatively complete clinical studies are available (Perry, Burchell, and Edwards, 1949; Stahlman *et al.*, 1955; Kirby, Johnson, and Zinsser, 1957; Ferencz, 1957; Lynch *et al.*, 1958; Gerbode *et al.*, 1958; Kjellberg *et al.*, 1959; Braunwald and Morrow, 1960; Levy and Lillehei, 1962). Symptoms were directly related to the amount of left-to-right shunting of blood with pulmonary vascular engorgement. These consisted of poor feeding in infancy and easy fatigue; tachypnoea, pneumonia, and congestive heart failure were often noted. The usual findings on examination were a præcordial thrill and loud systolic murmur along the lower left sternal border; a mid-diastolic apical rumble was heard in some patients.

Chest radiography generally demonstrated cardiomegaly, pulmonary vascular engorgement, and in 15 of the 26 patients specific enlargement of the right atrium. The electrocardiogram was not diagnostic; an abnormal P wave was present in a few of the tracings. Except for one patient with a mean electrical axis of -90° , there were no reports of the axis being to the left or superiorly oriented. Cardiac catheterization demonstrated a left-to-right shunt at the level of the right atrium in all but 2 patients. The right atrial pressure was raised in 5 of the 9 patients in whom it was recorded.

Descriptions of the cardiac defects at operation or necropsy are available in 24 patients: in 6 there was a defect high in the membranous ventricular septum with an opening in the floor of the right atrium just above the insertion of the septal leaflet of the tricuspid valve. One of these also had an abnormal mitral valve with obstruction to the left ventricular outflow tract (Ferencz, 1957). In the remaining patients a defect in the membranous ventricular septum occurred below the level of the tricuspid ring and was associated with a defect in the tricuspid valve. In 11 of these it appears that

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† The term left ventricular-right atrial shunt used in this report does not include those defects with a common atrio-ventricular valve.

the edges of the tricuspid defect fused with those of the ventricular septal defect so that arterialized blood shunted directly from the left ventricle to the right atrium without traversing the right ventricle. In 15 patients surgical closure of the defect was successful and in 8 patients it ended fatally.

Bacterial endocarditis was reported in only one instance (Perry *et al.*, 1949) and was the cause of death in this patient. No operation was performed and the infecting organism was an alpha hæmolytic streptococcus. Although the margins of the tricuspid and ventricular septal defects showed no evidence of an inflammatory process, there were vegetations on the free edges of the anterior and septal leaflets of the tricuspid valve and the chordæ of the anterior leaflet. The most extensive endocarditis occurred on the anterior wall of the right ventricle. The microscopical appearance of the margins of the communication between left ventricle and right atrium showed no evidence of healed bacterial endocarditis and was believed to be consistent with the reaction to the trauma of blood passing through the channel.

CASE REPORTS

Case 1. A 13-year-old girl was first noted to have a cardiac murmur soon after birth. She was asymptomatic during childhood. At 9½ years she developed fever, lethargy, anorexia, cough, pallor, and shaking chill. *Streptococcus viridans* was cultured from 5 separate specimens of blood. She received penicillin and erythromycin for one month, and, in addition, chloramphenicol for the last two weeks. At 10 years of age a thrill and grade 3/6 systolic murmur were noted along the lower left sternal border. Chest radiography showed a slightly enlarged heart with disproportionate right atrial enlargement and suggestive enlargement of the left ventricle. There was moderate pulmonary plethora. A venous angiocardigram revealed large arteries and persistent opacification of the right heart and pulmonary vessels consistent with a left-to-right shunt. Cardiac catheterization demonstrated an increased oxygen content at the level of the right atrium and an additional increment of 1.6 volumes per cent in the right ventricle. The pressure in the pulmonary artery was 44/18, right ventricle 80/3, and right atrium 3 mm. Hg. At 13½ she was admitted for surgical repair of the cardiac defect. Clinical and radiological signs were as before. The cardiogram showed a mean QRS axis of +87°, and an rsR' pattern in lead V3R, but was otherwise unremarkable. At operation a defect measuring 1.5 cm. was noted high in the membranous ventricular septum. The septal leaflet and the posterior third of the anterior leaflet of the tricuspid valve were densely adherent along the inferior edge of the ventricular septal defect. There was no evidence of pulmonary stenosis. The ventricular septal defect was closed with a "teflon" prosthesis. The anterior tricuspid leaflet was sutured to the septal leaflet over an area of 1 cm. that had previously been the site of a cleft.

Case 2. A 17-year-old girl, known to have had a cardiac murmur since early infancy, remained asymptomatic throughout childhood. A faint thrill and grade 3-4/6 blowing systolic murmur were noted along the lower left sternal border at 16 years of age. Chest radiography showed a normal-sized heart and normal lung fields. The mean QRS axis was +47° and the electrocardiogram was unremarkable. At cardiac catheterization a left-to-right shunt was detected at the atrial level by analysis of blood oxygen content, and at the level of the right ventricle and pulmonary artery by inhalation of hydrogen. The pressure in the pulmonary artery was 16/6, the right ventricle 16/3, and the right atrium 2 mm. Hg. At operation a defect of 0.4 cm. was noted high in the membranous ventricular septum. The edges of the defect were fused with thickened chordæ tendineæ arising from the anterior tricuspid leaflet. When the valve was open blood shunted from the left ventricle to the right atrium; when the valve was closed blood shunted to the right ventricle. Closure of the ventricular septal defect and reconstruction of the tricuspid valve were accomplished by direct suture.

Case 3. This 9-year-old boy was noted to have a cardiac murmur at 3 weeks of age. Poor weight gain and laboured respiration characterized infancy. At 6 months of age, a thrill and systolic murmur were found along the lower left sternal border. Although there were frequent respiratory illnesses during early life he became progressively less dyspnoeic. At 2½ years of age he developed persistent fever of three weeks' duration. Scattered petechiæ, splenomegaly, and a harsh systolic murmur in the third left intercostal space were noted. Pneumococcus type 14 was cultured from two separate specimens of blood. He was treated for six weeks with penicillin. At 4½ years of age he was asymptomatic. A grade 3-4/6 harsh systolic murmur was heard along the lower left sternal border, and P2 was widely split. Cardiac enlargement and pulmonary overcirculation were seen on the chest radiograph: the mean electrical axis was zero; there was an rSR' pattern in chest lead V3R; and P waves were 2.5 mv. high. Cardiac catheterization at 5½ years of age revealed an increment in oxygen content at the level of the right atrium. The pressure in the pulmonary

artery was 21/12, the right ventricle 22/2, and right atrium 5 mm. Hg. The course of the catheter during a repeat catheterization at 8 years of age was from right atrium to right ventricle to left ventricle to aorta. Angiocardiography performed with injection into the right ventricular outflow tract demonstrated retrograde flow into an enlarged right atrium and provided indirect evidence of a left-to-right shunt at the ventricular level. He remained asymptomatic; the systolic murmur was unchanged, and a short mid-diastolic murmur was heard at the apex. The mean QRS axis was zero but there was now electrocardiographic evidence of left ventricular enlargement ($Q=8$, $R=45$, $S=10$ mv., and tall-peaked T wave in chest lead V6). At operation a defect measuring 1.5 cm. was found high in the membranous ventricular septum: a portion of the tricuspid valve was adherent resulting in some blood shunting to the right atrium and some to the right ventricle. Closure of the ventricular septal defect and reconstruction of the tricuspid valve were accomplished by direct suture.

Case 4. A 15-year-old youth was known to have had a cardiac murmur since birth. At 13 months of age a systolic murmur was heard over the entire præcordium and cardiac enlargement was seen on the chest radiograph but he remained asymptomatic during childhood. At 13½ years of age a thrill and grade 3-4/6 systolic murmur were noted along the lower left sternal border. Moderate cardiac enlargement was seen radiologically: the left ventricle was relatively enlarged as was the main pulmonary artery and there was a slight increase in pulmonary vascularity. The mean QRS axis was -36° and the electrocardiogram was otherwise normal. Cardiac catheterization revealed an increment in oxygen content at the level of the right atrium and an additional increment of 0.6 volumes per cent at the level of the right ventricle. The pressure in the pulmonary artery was 22/13, the right ventricle 33/5, and the right atrium 4 mm. Hg. At the age of 14 years 10 months he developed persistent fever and skin eruption. *Streptococcus viridans* was cultured from two separate specimens of blood. He received penicillin and streptomycin for four weeks. At 15 years of age, surgical correction of the cardiac defect was performed by Dr. John Kirklin at the Mayo Clinic who reported as follows: "After opening the right atrium we saw a defect that measured about 6×9 mm. in size. It was in the typical high position forming almost a true left ventricular-right atrial shunt. The edges of the septal leaflet of the tricuspid valve were adherent to the edges of the defect. Just anterior to the defect there was a bulge in what must be the membranous septum. This area was quite intact. . . . There was no evidence of the previous bacterial endocarditis. . . . The non-coronary sinus of Valsalva seems almost to be forming an incipient aneurysm. . . . That portion of the aortic root which could be palpated through the right atrium seemed in some ways quite thin." The ventricular septal defect was closed with multiple interrupted sutures.

Case 5. A 6-year-old girl was first found to have a harsh systolic murmur in the fourth and fifth intercostal spaces at 1 year of age. Weight gain was slow in early infancy; she was otherwise asymptomatic. The heart was enlarged radiologically. The electrocardiogram showed left axis deviation and an rR' pattern in chest lead V1. At 2 years of age an apical diastolic murmur was first detected and x-ray examination of the chest showed pulmonary vascular engorgement. She continued to be asymptomatic. At 5½ years of age a thrill was noted over the lower left sternal border. An increment in oxygen content of right atrial blood was found during cardiac catheterization: the catheter was thought to traverse from right atrium to left ventricle. The pressure in the pulmonary artery was 31/12, right ventricle 34/1, and right atrium 5 mm. Hg. Pre-operative examination at 6½ years of age revealed a prominent thrill and grade 4/6 systolic murmur at the lower left sternal border and a grade 2/6 apical mid-diastolic murmur. Chest radiography showed only slight cardiac enlargement and pulmonary vascularity was at the upper limits of normal. The mean QRS axis was -49° , P waves were 3 mv. in height, and there was evidence of left ventricular enlargement ($Q=8$, $R=35$, $S=2$ mv. in V6). At operation a left superior vena cava was seen to enter the left atrium, there was enlargement of the left atrium and right ventricle, and there was a thrill at the junction of the right atrium and left ventricle. A small anomalous vein drained into the right atrium just anterior to the superior vena cava. The margins of a defect in the tricuspid valve were fused with the lower margins of a defect high in the membranous ventricular septum. The ventricular septal defect was closed with the aid of a "teflon" prosthesis. The tricuspid valve was reconstructed by direct suture.

Case 6. This 17½-year-old girl was first noted to have a murmur at 1 month of age. At 23 months of age she developed spiking fever of 3 weeks' duration. Examination revealed a harsh systolic murmur along the lower left sternal border, and splenomegaly. Pneumococcus type 6 was found in blood cultures on two successive days. She was treated with penicillin and sulfadiazine for three weeks. By 15 years of age she suffered from palpitation and dyspnoea on exertion. A thrill and grade 4/6 systolic murmur were noted over the lower left sternal border; there was a low-pitched mid-diastolic apical rumble. Slight enlargement of the heart and overcirculation of the lungs were seen on the chest radiograph. The mean QRS axis was $+30^\circ$ and the electrocardiogram was otherwise unremarkable. There was an increase in oxygen content of right atrial

TABLE I

VENTRICULAR SEPTAL DEFECT WITH SHUNT INTO RIGHT ATRIUM: SUMMARY OF FINDINGS IN 6 PATIENTS

Case No., sex, and age (yr.)	Symptoms	Thrill	Murmurs		Chest radiography			Electrocardiogram				Cardiac catheterization pressures (mm. Hg)			Bacterial endocarditis, infecting organism, and age
			Syst. LLSB†	Diast. at apex	Cardiac enlargement‡	Right atrial enlargement	Increased pulmonary vascularity‡	Mean QRS axis	rR' in V3R	LV enlargement	P > 2.5 mv.	Mean RA	RV	PA	
1 F 13 4/12	0	+	+	0	Slight	+	Mod.	+87°	+	0	0	3	80/3	44/18	<i>Strep. viridans</i> 9 yr.
2 F 17 4/12	0	+	+	0	0	0	0	+47°	0	0	0	2	16/3	16/6	0
3 M 9 4/12	0*	+	+	+	Mod.	+	Mod.	0	+	+	+	5	22/2	21/12	<i>Pneumococcus</i> Type 14 2½ yr.
4 M 15 4/12	0	+	+	0	Mod.	0	Slight	-36°	0	0	0	4	33/5	22/13	<i>Strep. viridans</i> 15 yr.
5 F 6	0*	+	+	+	Slight	0	Slight	-49°	0	+	+	5	34/1	31/12	0
6 F 17 6/12	Dyspnoea and fatigue	+	+	+	Slight	0	Slight	+30°	0	0	0	3	25/3	20/10	<i>Pneumococcus</i> Type 6 2 yr.

* Poor weight gain during infancy.

† Systolic murmur of grade 3/6 or louder over the lower left sternal border.

‡ Graded slight, moderate, or severe.

blood during cardiac catheterization with an additional increase of 0.9 volumes per cent in the right ventricle. The pressure in the pulmonary artery was 20/10, right ventricle 25/3, and the right atrium 3 mm. Hg. During operation at 17½ years of age, a thrill was noted over the right ventricle close to the tricuspid valve. A defect was found in the commissure between the anterior and septal leaflets of the tricuspid valve. Just beneath this there was a defect measuring 1 × 1.5 cm. in the membranous ventricular septum. There was an additional defect in the anterior leaflet of the tricuspid valve through which there was direct regurgitation from the right ventricle to the right atrium. The ventricular septal defect was closed and the tricuspid valve reconstructed by direct suture.

RESULTS

A shunt from the left ventricle to the right atrium was demonstrated at operation in each of 6 patients in all of whom the defect was successfully closed. A summary of the findings in these patients is given in Table I. The age at the time of operation ranged from 6 to 17½ years; 4 were female and 2 were male; 5 were asymptomatic while 1 fatigued easily and was short of breath on exercise. A murmur was noted during the neonatal period in 4 and by 1 year of age in the remaining 2. A præcordial thrill was noted in all 6, as was a long, systolic murmur of grade 3/6 or louder along the lower left sternal border. An apical low-pitched mid-diastolic murmur was detected in 3. The second sound in the second left intercostal space was unremarkable in 3, widely split in 3, and increased in intensity in 2 of the latter.

Chest radiography revealed abnormal findings in 5 of the 6 patients, who had slight to moderate cardiac enlargement: in 2 of these disproportionate enlargement of the right atrium was recognized. Mild increases in central and peripheral vascularity were noted in 3 and moderate increases in 2 others. Typically the aorta was inconspicuous relative to the pulmonary artery.

The mean QRS axis of the electrocardiogram was normal in 4 and oriented superiorly and to the left in 2 patients. In 2 of those with normal axes, right-sided chest leads revealed rR' patterns. Peaked P waves of 2.5 mv. or more and left ventricular enlargement were recorded in 2 patients.

An increase in the oxygen content of blood from the right atrium was noted in all 6 patients during cardiac catheterization. In no instance was there evidence of arterial unsaturation. Mean atrial pressures were 5 mm. Hg or less in all the patients. There were no unusual a, c, or v waves on right atrial pressure tracings. Pulmonary arterial pressures were normal in 4 and raised in 2.

An earlier diagnosis of bacterial endocarditis had been established in 2 patients by the presence of *Streptococcus viridans* on bacterial culture of two or more specimens of blood. In 2 other patients the diagnosis of bacterial endocarditis was strongly suggested by the prolonged course of fever, the presence of a cardiac murmur, and the presence of pneumococci in two successive blood cultures from each patient; in one patient scattered petechiæ were also present. Recovery following intensive antibacterial therapy was complete in all 4.

Surgical correction of the cardiac malformation was successfully performed in each case with the aid of extracorporeal circulation. In 5 of the 6 patients, the edges of the tricuspid valve were partially fused to the rim of a defect in the membranous portion of the ventricular septum, thus allowing the direct shunting of blood from left ventricle to right atrium. In the sixth patient, the defect in the membranous ventricular septum was located just beneath a defect in the commissure between the anterior and septal leaflets of the tricuspid valve. In addition, there was a second defect in the anterior leaflet of the tricuspid valve allowing direct regurgitation of blood from right ventricle to right atrium. In no instance were vegetations detected. However, thickening and distortion of the tricuspid valve were present in each case. The defect in the ventricular septum was closed by direct suture in 4 patients and with the aid of a "teflon" prosthesis in 2. All 6 patients are asymptomatic and living normally after operation for periods of one and one half years or longer. None has been re-catheterized.

DISCUSSION

Anatomy. A portion of the membranous ventricular septum lies cephalad to the attachment of the septal leaflet of the tricuspid valve and forms a part of the floor of the right atrium (Gould, 1960). In the absence of a common atrio-ventricular canal, defects in the upper part of the membranous ventricular septum with direct communication between the left ventricle and right atrium are not common. Since the right tubercles of the atrio-ventricular canal cushions contribute to the completion of the upper part of the membranous ventricular septum (Kramer, 1942; McCullough and Wilbur, 1944), malformations of this area probably represent defects of the endocardial cushion and may be associated with defects of the mitral valve, for example the patient reported by Ferencz (1957).

Defects in the lower part of the membranous ventricular septum will lie just beneath the attachment of the septal leaflet of the tricuspid valve. When the edges of a defect in the tricuspid valve are in approximation with or fused to the margins of the ventricular septal defect, a communication between left ventricle and right atrium will also exist.

Pathogenesis. Anatomical considerations of left ventricular to right atrial shunts have led to an appreciation of the distinction between those less common defects of the upper membranous ventricular septum, which are above the tricuspid ring, and the more common variety exemplified by our 6 patients in which the defect lies beneath and adjacent to defects in the tricuspid valve. In the latter situation, the impact of a jet stream of blood through a high ventricular septal defect leads to thickening and distortion of the leaflets and chordæ tendineæ of the tricuspid valve (Edwards and Burchell, 1958), and may also predispose to infection (Gould, 1960). In time the damaged valve may fuse with the margins of the ventricular septal defect. Although it is not clear whether the tricuspid valve is developmentally normal or not, in either event hæmodynamic trauma would lead to thickening and distortion of the valve. Fusion of the edges of the damaged tricuspid valve to the rim of the ventricular septal defect would be enhanced by the superimposition of bacterial endocarditis.

Perry *et al.* (1949) concluded that the tricuspid defect in their patient was congenital in nature, and that while hæmodynamic trauma might have predisposed to bacterial endocarditis the microscopical appearance of the rim of the defects argued against including infection in the pathogenesis of the tricuspid anomaly. In 3 of the 4 patients with bacterial endocarditis whom we have reported the episode of infection occurred several years before cardiac catheterization and operation. Although there was no gross evidence of endocarditis at the time of operation we can presume only that

the infection had occurred at the site of hæmodynamic trauma. There is no way of knowing whether the tricuspid valve defect was developmental in origin, whether it was acquired secondarily to hæmodynamic trauma, or whether it was a combination of the two.

Diagnosis. The diagnosis of a left ventricular to right atrial shunt is inferred from physical findings suggesting a ventricular septal defect and laboratory findings indicating a left-to-right shunt at the atrial level. Although one would expect to find evidence of increase in size of the right atrium on chest radiography and in the electrocardiogram, and increase in right atrial pressure during cardiac catheterization, these are not always present. The differential diagnosis has been thoroughly discussed by others (Stahlman *et al.*, 1955; Ferencz, 1957; Gerbode *et al.*, 1958; Braunwald and Morrow, 1960), and includes defects of the atrial septum of the ostium primum and secundum types, anomalous drainage of pulmonary veins, ventricular septal defect associated with either tricuspid insufficiency or an atrial septal defect, rupture of a congenital aneurysm of the sinus of Valsalva into the right atrium, and fistula of coronary artery to right atrium.

Radiologically cardiac enlargement and various degrees of pulmonary vascular engorgement are present. In about half the patients disproportionate enlargement of the right atrium can be recognized. This finding, as pointed out by Kramer and Abrams (1962), together with a murmur suggesting ventricular septal defect and catheter evidence of a left-to-right shunt at the atrial level should raise the possibility of a left ventricular to right atrial shunt, but certain endocardial cushion defects may produce similar findings.

Although cardiac catheterization may be helpful in localizing the level of the shunt to the right atrium, the usual finding of normal right atrial pressures and pulse tracing may mask the fact that the left-to-right shunt originates from the high-pressured left ventricle. Pre-operative localization of the defect by angiocardiology has been reported, using injection of contrast media into the left ventricle (Nordenström and Ovenfors, 1960), and this has been combined with indicator dilution studies in order to delineate further the anatomical defects (Braunwald, Morrow, and Cooper, 1959; Braunwald and Morrow, 1960).

The differentiation of patients with left ventricular to right atrial communications of the types described in this paper from patients with common atrio-ventricular canals or ostium primum defects associated with mitral clefts may be very difficult. The findings on physical examination, chest radiography, and cardiac catheterization may be identical. While the electrocardiographic findings of a superior axis and rR' pattern in right-sided chest leads may suggest an endocardial cushion type defect, 2 of our patients had superior axes and 2 others had rR' patterns. Angiocardiology studies with injection of contrast material into the left ventricle may be helpful in delineating the defect. The importance of correlating the findings on physical and laboratory examination, in order to make an accurate anatomical diagnosis, is clear.

The finding of *Streptococcus viridans* in repeated cultures of the blood from 2 of our patients appears to establish the diagnosis of bacterial endocarditis. The presence of pneumococci in more than one blood culture from each of 2 other patients while strongly suggestive of bacterial endocarditis must be interpreted with some caution, since pneumococcal bacteraemia may occur in young children in association with infections of the ears or respiratory tract.

Surgical Considerations. Total correction can be performed utilizing extracorporeal circulation through a simple right atriotomy. When the opening of the ventricular septal defect is located above the level of the tricuspid ring, this may be closed by simple suture. It is more common, however, to observe a defect in the tricuspid valve at the junction of the septal and anterior leaflets. Figure 1A shows a characteristic view of the tricuspid defect as seen from the right atrium in one of our patients. The edges were thickened and probing afforded direct access to the left ventricle. Although it is possible to mistake the tricuspid defect for the total anomaly, careful inspection reveals fused chordæ tendineae and valve leaflets below the tricuspid valve adherent to the margin of a larger defect in the membranous ventricular septum. When the leaflets of the tricuspid valve are detached the underlying ventricular septal defect and its relation to the aortic root are revealed (Fig. 1B). Simple closure of the defect in the tricuspid valve will usually not be sufficient because fusion of the valve

FIG. 1.—A. Tricuspid valve defect seen from the right atrium. The interrupted line indicates the line of incision to reflect the leaflets. B. Ventricular septal defect and aortic root as seen from right atrium after the tricuspid valve is detached.

FIG. 2.—A. Mattress sutures placed in from the margin of the ventricular septal defect to avoid the conduction bundle. B. "Teflon" prosthesis sutured into position completely closing the ventricular septal defect. C. Reconstruction of tricuspid valve by direct suture.

about the ventricular septal defect is incomplete. Closure of the ventricular septal defect is essential for a satisfactory anatomical repair. When the defect in the ventricular septum is large, repair utilizing an intracardiac prosthesis is preferred (Fig. 2A and 2B). The tricuspid valve is reconstructed by simple suture (Fig. 2C).

SUMMARY

Intracardiac shunting of blood from left ventricle to right atrium resulting from a defect in the membranous ventricular septum has been observed in 6 patients. The ages ranged from 6 to 17½ years. A thrill and systolic murmur along the lower left sternal border characteristic of a ventricular septal defect, and catheterization data indicating a left-to-right shunt at the level of the right atrium, were present in all. Chest radiography revealed slight to moderate cardiac enlargement and pulmonary vascular engorgement in 5, with specific enlargement of the right atrium in 2. The electrocardiogram was not diagnostic: the mean QRS axis was deviated to the left and superiorly in 2. *Streptococcus viridans* endocarditis was present in 2 and pneumococcal endocarditis strongly suspected in 2 others before operation.

Surgical exploration revealed a defect in the tricuspid valve, the thickened margins of which were fused to a defect in the membranous ventricular septum. No vegetations were seen. Closure of the ventricular septal defect before reconstruction of the tricuspid valve provided complete correction in each patient.

It is not clear whether the tricuspid defect is acquired secondarily to hæmodynamic trauma or whether the trauma is superimposed on developmentally abnormal tricuspid valve leaflets. In either event hæmodynamic trauma predisposes to bacterial infection and the combination enhances fusion of the tricuspid defect to the margins of the ventricular septal defect.

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