Spontaneous Closure of Ventricular Septal Defects

By J. R. Evans, M.D., R. D. Rowe, M.B., and J. D. Keith, M.D.

SYSTOLIC MURMURS heard during the first year of life in apparently healthy children are commonly noted to have disappeared at a later examination.\(^1\)\(^2\) The exact origin of such murmurs is not known but it has been suggested that some are accounted for by exaggeration of the physiologic pulmona

ny systolic murmur and a few by late closure of the ductus arteriosus. Of particular interest is a small group of patients in whom a confident clinical diagnosis of ventricular septal defect was made at the initial examination on the basis of the site and quality of the murmur, yet as the child grew the murmur diminished and ultimately disappeared completely. The results of special investigations in a few of the patients support the clinical impression that these murmurs arise from a ventricular septal defect that closes spontaneously.

Method

All patients were seen in the cardiace department of the Hospital for Sick Children, Toronto, during the period 1949 to 1959. They were divided into 3 groups.

In the first group were 37 apparently healthy children in whom a diagnosis of ventricular septal defect had been made at the first examination but in whom disappearance of the murmur was confirmed by subsequent examinations. Thirty-one of the patients were seen during the first year of life. In each case the diagnosis was based on the clinical assessment including electrocardiographic and radiologic examination but in 4 of the patients cardiac catheterization was performed while the murmur was present and in 1 it was repeated after the murmur had disappeared.

The second group consisted of 9 patients with clinical features typical of the preceding group but in whom the murmur had not yet disappeared at the time of writing. In 5, the murmur was recorded on a Sanborn phonocardiogram with a photographic recorder. Cardiac catheterization was done in each case. In 1 patient the catheter passed through the foramen ovale and cineangiography was performed with injections of 5 ml. of 90 per cent Hypaque (sodium diatrizoate) into the left ventricle. In 3 others, sound tracings were made from the pulmonary arteries, right ventricle, and right atrium with an intracardiac phonocather and the Sanborn recorder. The tracings were compared with external phonocardiograms taken at the same time in the third and fourth intercostal spaces at the left sternal border.

The third group included 1 patient seen during the first 6 months of life with the clinical picture of a large ventricular septal defect and congestive heart failure. Cardiac catheterization was done when the patient was first seen and repeated at 7 years of age when the clinical signs of heart disease had disappeared. This patient was not included in the first group because of the serious nature of the heart disease with which she presented initially.

Observations

Patients with Murmurs That Disappeared

Clinical Features

A diagnosis of ventricular septal defect based on clinical findings was made in 37 children in whom the murmur ultimately disappeared. None of the patients had cardiac symptoms and all but 2 were referred for assessment of a systolic murmur noted on routine examination. One patient experienced frequent respiratory infections and another had unexplained cyanotic attacks. Three patients weighed 2.5 Kg. or less at birth; 1 was a discordant twin. Transient respiratory distress in the neonatal period was recorded in 1 patient and denied in 30 others; in 6 information was not available.

On examination all the patients appeared healthy without cyanosis, respiratory distress, or signs of congestive heart failure. The systolic murmur was moderately loud, usually grade II (of 4 grades), and was of uniform intensity throughout systole beginning with the first sound and extending to or stopping short of the second heart sound. No midsystolic accentuation was evident. A consistent

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Aided by a grant from the Ontario Heart Foundation.
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feature was the superficial blowing quality of the murmur, giving the impression of high-frequency vibrations arising immediately under the diaphragm of the stethoscope. The site of maximum intensity was always in the third and fourth intercostal spaces at the left sternal border. There was no radiation to the axilla or neck vessels. The murmur did not vary with respiration but its duration and intensity decreased markedly in a few patients during the Valsalva maneuver. A faint systolic thrill accompanied the murmur in 6 infants, all of whom were under 6 months of age. In these cases the murmur was usually of grade III intensity. At a second examination under 1 year of age the thrill had disappeared in every instance. The heart sounds, when described, were normal except for 2 infants in whom mild accentuation of pulmonary valve closure was heard. In no instance was an ejection click or diastolic murmur noted. A third heart sound was heard in some patients and a venous hum was present in 2 of the children.

The electrocardiogram never revealed striking abnormalities, and in 24 of the patients it was completely normal. Incomplete right bundle-branch block occurred in 5 patients and in 1 other right ventricular hypertrophy was indicated by an upright T-wave in lead V1, where the ratio of R/S was greater than 1. Left ventricular hypertrophy was present in 4 patients and evidence of combined left and right ventricular hypertrophy was noted in 3 others: in each instance the abnormal tracing was taken during the first year of life and subsequent electrocardiograms after 1 year of age were normal.

Radiologic examination of the heart and pulmonary vascular markings was normal in all patients over 1 year of age. In 6 infants, age 1 to 7 months, there was some cardiac enlargement with a cardiothoracic ratio ranging from 0.55 to 0.61; in 2, the left atrium was enlarged, and in 4 the hilar shadows were increased. Prominent vascular markings without apparent cardiac enlargement were seen in 2 other patients. All radiologic changes had reverted to normal by 1 year of age.

The arterial oxygen saturation was assessed by ear oximetry in 13 patients at rest and with crying and was found to be over 90 per cent in every case.

At subsequent examinations over the years the systolic murmur tended to decrease in intensity and become shorter. With some murmurs a decrescendo quality about midsystole was recognized. Shortly before the murmur disappeared, it occupied only the first third of systole. The age at which the murmur finally disappeared varied greatly but in the majority of cases it was gone by 2 years of age. In 7 children it persisted beyond 3 years, and in 1 the murmur did not go until the child was between 9 and 10 years of age. After the murmur disappeared no abnormal physical signs could be detected. In 10 patients a soft ejection systolic murmur with low-frequency vibrations was heard in the pulmonary area. The site and quality of this murmur, often heard in normal children, clearly differentiated it from the disappearing type of murmur.

**Cardiac Catheterization**

Four of the 37 patients in whom the systolic murmur disappeared were submitted to cardiac catheterization while the murmur was still present. The results are shown in table 1. There was evidence of a left-to-right shunt in each case. In 1 patient, D.L., the over-all rise in oxygen saturation was only 2 per cent but 1 sample of 85 per cent in the right ventricle helped to confirm the presence of the shunt. In the other 3, the increase ranged from 7 to 18 per cent, indicating a mild to moderate shunt. The intracardiac pressures were normal in 3 patients, but in D.B., who had the largest shunt, moderate elevation of right ventricular, pulmonary artery, and mean "wedge" pressures was noted.

At 6 months of age patient F.S. had a long systolic murmur and a rise in oxygen saturation of about 10 per cent at right ventricular level. When he was seen at 3 years of age, the murmur was very short and early in systole. The patient was catheterized again 3 months later but at that time the murmur
had disappeared completely and could not be demonstrated with the intracardiac phonocatheter. No sign of left-to-right shunt persisted and the right ventricular and pulmonary artery pressures were normal (table 1).

**Patients with Probable Disappearing Murmurs**

The results of special investigations in 9 additional patients with typical disappearing murmurs are shown in table 2. It should be stressed that in this group the murmur had not disappeared at the time of writing but, from the clinical standpoint, it was considered in each case to be of the type that would eventually disappear. The murmur was not associated with a thrill, and in all patients the electrocardiographic and radiologic findings were within normal limits.

At cardiac catheterization a left-to-right shunt was demonstrated at ventricular level in 5 patients. It is noteworthy that in 1 patient, M.W., the over-all rise in oxygen saturation was negligible but 3 samples taken from the apical region of the right ventricle were 65, 65, and 85 per cent, considerably higher values than in the right atrium or the pulmonary artery. In 4 patients the presence of a ventricular septal defect could not be established by oxygen studies: in patients R.C. and T.G. the rise in oxygen saturation was 2 to 3 per cent at most, and in J.L. and S.B. no increase was noted. No other abnormality was found, however, to explain the systolic murmur in these cases. The right atrial and "wedge" pressure tracings did not suggest tricuspid or mitral incompetence, and no systolic gradient was present between the right ventricle and the pulmonary artery.

Phonocardiographic studies in the 5 patients in whom a left-to-right shunt was demonstrated suggested a correlation between the length and intensity of the systolic murmur and the size of the shunt. The duration of the murmur is represented diagrammatically in table 2. Patients D.M. and R.M. with the largest shunts had grade-II pansystolic murmurs extending from the first sound through to the second sound. On the other hand, in patients J.F. and C.K. the rise in oxygen saturation was less than 5 per cent and the murmur was softer and shorter, terminating in early or midsystole. A soft, short, early systolic murmur with decrescendo quality was present in patient M.W., in whom no increase in pulmonary blood flow was detected (fig. 1).

More precise localization of the site of the defect was attempted with special technics in 4 of the 5 patients in whom a left-to-right shunt had been shown with oxygen studies. In patient M.W., a selective cineangiogram from the left ventricle revealed a small jet of contrast medium passing through the lower interventricular septum into the right ventricle (fig. 2). There was no reflux of dye into the left atrium to suggest mitral incom-
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Patient with Large Ventricular Septal Defect

The patient presented at 5 months of age with the signs of a large ventricular septal defect in congestive cardiac failure. The systolic murmur was harsh and of grade-III intensity; it was loudest in the fourth left interspace near the sternum, where a soft thrill was palpable. The electrocardiogram showed evidence of both left and right ventricular hypertrophy. On radiologic examination the heart was moderately enlarged with a prominent left atrium and dense pulsatile hilar shadows. With digitalis therapy the rapid respirations, jugular venous engorgement, and hepatic enlargement all disappeared. Cardiac catheterization at 6 months of age showed a marked rise in oxygen saturation at ventricular level with moderate elevation of right ventricular pressure (table 3.) The pulmonary artery was not entered. At this time, there was no diastolic murmur to suggest patent ductus arteriosus or pulmonary incompetence.

Over the next 2 years the signs of left-to-right shunt gradually diminished. The electrocardiogram and chest x-ray returned to normal. Digitalis was stopped, and the patient remained without symptoms. When the patient was 7 years old no abnormality could be detected on examination, except for a very soft short systolic murmur along the left sternal border. Catheterization was repeated and showed normal intracardiac pressures with no significant left-to-right shunt (table 3).

Figure 1

Phonocardiogram of the short, early systolic murmur of patient M.W., in whom a very small defect of the muscular interventricular septum was demonstrated. In this and subsequent tracings the time intervals are 0.04 sec. per small division; S₁, S₂, and S₃ indicate first, second, and third heart sounds respectively; SM indicates systolic murmur.
Figure 2

Selective angiocardiogram from the left ventricle (lateral view) in patient M.W., showing small jet of contrast medium in body of right ventricle. Cine-films demonstrated that this jet originated near the apex of the right ventricle.

Table 2

The Results of Cardiac Catheterization in Nine Patients with the Typical Systolic Murmur of a Ventricular Septal Defect that will Disappear Spontaneously

<table>
<thead>
<tr>
<th>Patient (age)</th>
<th>Systolic murmur</th>
<th>Oxygen Saturation (%)</th>
<th>Pressure (mm. Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>S1</td>
<td>S2 (1-4)</td>
<td>S.V.C.</td>
</tr>
<tr>
<td>D. M. (2 yr.)</td>
<td>2</td>
<td>58</td>
<td>68</td>
</tr>
<tr>
<td>R. M. (1 mo.)</td>
<td>2</td>
<td>60</td>
<td>52</td>
</tr>
<tr>
<td>J. F. (4 yr.)</td>
<td>1</td>
<td>44</td>
<td>57</td>
</tr>
<tr>
<td>C. K. (6 yr.)</td>
<td>1</td>
<td>59</td>
<td>66</td>
</tr>
<tr>
<td>M. W. (3 yr.)</td>
<td>1</td>
<td>54</td>
<td>72</td>
</tr>
<tr>
<td>R. C. (9 yr.)</td>
<td>short</td>
<td>1</td>
<td>79</td>
</tr>
<tr>
<td>T. G. (5 yr.)</td>
<td>short</td>
<td>1</td>
<td>70</td>
</tr>
<tr>
<td>J. L. (14 yr.)</td>
<td>short</td>
<td>1</td>
<td>81</td>
</tr>
<tr>
<td>S. B. (7 yr.)</td>
<td>short</td>
<td>1</td>
<td>77</td>
</tr>
</tbody>
</table>

*Sample drawn from apex of right ventricle.

The duration and intensity of the murmur may be compared with the size of the shunt. In 5 patients, the murmur is represented diagrammatically as recorded at the lower left sternal border with the phonocardiogram.
a thrill. A thrill, however, was noted in 6 infants but in these cases the murmur was louder, pansystolic in length, and electrocardiographic and radiologic evidence of heart disease was present. In each case the abnormal findings including the thrill were gone by the end of the first year and all that remained was the superficial high frequency systolic murmur.

As the children grew older, the systolic murmur became softer and shorter. In some a decrescendo quality was recognized and the murmur terminated about midsystole. The ultimate time of disappearance of the murmur varied greatly. Most were gone by the end of the second year but in one fifth of the present series the murmur persisted beyond 3 years, and in 1 patient it did not disappear until 10 years of age. It is not yet known whether similar murmurs heard in older patients will disappear during adult life.

When the patients were first seen several possibilities were offered to explain the origin of the "disappearing systolic murmur." The site of maximum intensity and the timing of the murmur made ventricular septal defect

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Figure 3

Phonocardiograms of patient D.M. with a moderate left-to-right shunt showing the pansystolic murmur. Lower tracing: Murmur recorded with intracardiac phonocatheter in right ventricle only. Upper tracing: Murmur recorded externally in fourth interspace at left sternal border.
the most probable diagnosis but the superficial blowing quality of the murmur, its termination before the second sound, and the absence of a thrill were all unusual features. Incompetence of the atrioventricular valves was suggested by the blowing quality of the murmur. The site of maximum intensity of the murmur, its lack of radiation to the axilla, and the effect of the Valsalva maneuver militated against mitral incompetence. No inspiratory accentuation of the murmur was recognized nor was there a prominent systolic wave in the jugular venous pulse to suggest tricuspid incompetence. The disappearing murmur lacked the harsh quality, midsystolic accentuation, and palpable thrill found with pulmonary stenosis although it was of similar timing and location to the infundibular type of obstruction. As the disappearing murmur became shorter, its timing resembled the systolic murmur that may arise normally in the pulmonary artery. The latter murmur, however, has a distinctive coarse vibratory quality and is loudest at the upper left sternal border. Originally a broad differential diagnosis was considered in patients who showed the disappearing murmur but in recent years the murmur has been specifically identified without difficulty at the first examination in most cases.

The pathogenesis of the disappearing murmur was clarified by special investigation. A total of 13 patients were catheterized and, of these, 9 were shown to have a ventricular septal defect by oxygen studies, including 4 in whom the systolic murmur subsequently dis-

**Figure 4**
Phonocardograms of patient J.F. with small left-to-right shunt showing the systolic murmur in the first half of systole. Lower tracing: Murmur recorded best with intracardiac phonocatheter at apex of right ventricular cavity. Upper tracing: Murmur recorded externally in fourth interspace at left sternal border.
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Figure 5

Phonocardiograms of patient C.K. with small left-to-right shunt showing early systolic murmur with decrescendo quality. Lower tracing: Murmur recorded with intracardiac phonocatheter at apex of right ventricle only. Upper tracing: Murmur recorded externally in fourth interspace at left sternal border.

appeared. The left-to-right shunt was usually small and in a few instances was not apparent, except in samples drawn from the apical region of the right ventricle. In 4 patients no significant rise in oxygen saturation was demonstrated with the routine method of sampling. No evidence of tricuspid incompetence, mitral incompetence, or pulmonary stenosis was found in the pressure tracings of any of the patients. Sound tracings from the right side of the heart in 3 patients with small to moderate left-to-right shunts revealed a systolic murmur confined to the right ventricle that was similar in quality and timing to the murmur recorded externally at the lower left sternal border. The murmur was loudest at the apex of the right ventricle and became softer or imperceptible in the outflow tract and tricuspid valve region. In another patient the interventricular communication was shown by cineangiocardiography to be a small defect in the lower portion of the septum. No significant left-to-right shunt was detected by oxygen studies in this case but samples from the apical region of the right ventricle were consistently higher than those taken elsewhere in the right side of the heart. From these investigations it is apparent that the disappearing systolic murmur arises from a ventricular septal defect although in some patients the left-to-right shunt may be so small that it is not detected on routine sampling. The location of the defect was established in 4 patients and in each case was found to be near the apex of the right ventricle, corresponding to the muscular portion of the interventricular septum.

The natural history of the disappearing murmur appears to follow a characteristic pattern as judged by repeated clinical obser-
vations. In infancy the systolic murmur is long and in a few cases the clinical signs may suggest a moderate-sized ventricular septal defect with a pansystolic murmur and thrill, electrocardiographic changes, and radiologic evidence of increased pulmonary blood flow. By the end of the first year all the abnormal signs disappear except the systolic murmur. The murmur becomes progressively shorter and softer and eventually it disappears. A comparison of the character of the murmur in the phonocardiogram and the size of the shunt at cardiac catheterization in 5 patients suggests that the gradual reduction in the intensity and duration of the systolic murmur reflects the decreasing magnitude of the left-to-right shunt. Disappearance of the murmur, therefore, might represent balancing of pressures in the right and left ventricle or a reduction in size of the ventricular septal defect. There is no clinical evidence for the former concept, since the physical, electrocardiographic, and radiologic signs do not reflect the development of right ventricular hypertrophy or pulmonary hypertension. Furthermore, in 1 patient with a proved left-to-right shunt who was catheterized a second time after the murmur had gone, there was no elevation of right ventricular pressure and no residual shunt. It would seem, therefore, that disappearance of the murmur represents closure of the ventricular septal defect. If the defect opened in the inflow region of the right ventricle it could be sealed off by adherence of the septal leaflet and chordae of the tricuspid valve, but the gradual sequence of events and the probable location of the defect in the muscular portion of the septum make this unlikely. Alternatively, the defect could be progressively narrowed by hypertrophy of the muscle of the interventricular septum. This, in our opinion, is the probable mechanism of closure. In the newly born infant the septum is very thin between the trabeculae, particularly in the region near the apex of the heart. With growth of the heart and hypertrophy of its musculature the septum is gradually thickened, and small interventricular communications might be expected to be narrowed and ultimately obliterated. In support of such a hypothesis are the observations of Edwards that defects of the muscular septum in infants are ovoid in shape, whereas in adults they are small and linear with the upper and lower margins almost in apposition. In one 65-year-old man the margins of a small defect in the muscular septum appeared to have fused.

Spontaneous closure of a ventricular septal defect is not a new concept. This phenomenon was suspected on clinical grounds by several physicians but in no instance was the diagnosis proved. The first and best documented case was that described by French in 1918.

Some years ago I saw a small boy, 14 months old, and on examination of the heart there was a loud systolic bruit, with its maximum intensity over the fourth intercostal space, close to the sternum, but audible over the whole precordial region, and indeed over most of the chest, both back and front. It was accompanied by a thrill near the sternum, and those of you who have seen congenital heart cases in the wards will realize the kind of case it was when I say that

**Table 3**

<table>
<thead>
<tr>
<th>Age</th>
<th>S.V.C.</th>
<th>I.V.C.</th>
<th>R.A.</th>
<th>R.V.</th>
<th>P.A. Systemic artery (mean)</th>
<th>R.A.</th>
<th>R.V.</th>
<th>P.A. &quot;Wedge&quot; (mean)</th>
<th>Systemic artery</th>
</tr>
</thead>
<tbody>
<tr>
<td>6 mos.</td>
<td></td>
<td>31</td>
<td>23</td>
<td>70</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>60/0</td>
<td>—</td>
</tr>
<tr>
<td>7 yr.</td>
<td>58</td>
<td>—</td>
<td>63</td>
<td>63</td>
<td>66</td>
<td>*</td>
<td>93**</td>
<td>22/0</td>
<td>19/9</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>6</td>
<td></td>
<td>100/60</td>
<td></td>
</tr>
</tbody>
</table>

**Measured by ear oximetry**

*Sphygmomanometer.
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Circulation, Volume XXII, December 1960

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the diagnosis made by myself and others was congenital perforation of the interventricular septum. The father of the boy was an officer in the Navy, and this was his only child. He had always made up his mind that any boy of his should follow him in the service, and it was a tremendous blow to him to find that the child had a bruit which was certain to cause his rejection at the medical examination.

I saw the boy again when he was two years old, and the bruit was about the same as before, very loud and universal. I saw him again when he was five, and there was then absolutely no bruit at all! I saw him again at the age of ten; and he was still without a bruit and had no objective evidence of any heart lesion. The father's keen desire that the boy should follow him in the service has since been gratified and the case has taught me that the bruit of a congenital malformation of the heart may disappear as a child grows up.

French considered that the murmur disappeared because the defect did not enlarge in proportion to the growth of the heart. Other probable examples of disappearance of a ventricular septal defect are cited by Stamm, Weber, Still, Perry, and Muir. Postulated that contraction of the fibrous tissue around an aperture in the interventricular septum might diminish the size of the defect or close it off completely.

The paucity of case reports in the literature would suggest that spontaneous closure of a ventricular septal defect is rare. The present series of 37 patients was culled from over 120 patients seen in a 10-year period in whom the disappearing type of murmur was present. The remainder of the clinical cases were not included, since the murmur was not followed to complete disappearance. Probably other examples of this condition in older children were dismissed as functional murmurs, since no abnormality was detected apart from the murmur. With our current awareness of the syndrome an increasing number of cases have come to our attention. During the last 5 years in the practice of one of us (R.D.R.) 217 cases of ventricular septal defect have been seen, and 54 of these would seem to be the disappearing type of defect. Furthermore, in a recent survey of 80 "normal" newly born, term infants the typical superficial blowing systolic murmur, beginning with the first sound and stopping before the second sound, was heard in 3. If the proposed concept of the pathogenesis and disappearance of the murmur is correct these data suggest that small defects in the muscular ventricular septum that close with growth of the heart are relatively common.

Spontaneous closure is not confined to small defects. The patient who presented with congestive failure had the clinical signs of a major left-to-right shunt in keeping with a large ventricular septal defect. The exact site of the defect was not demonstrated. The mechanism of closure is uncertain but the clinical and hemodynamic evidence of a gradual reduction in the left-to-right shunt over several years is in keeping with the concept that muscular hypertrophy of the septum, perhaps augmented by the abnormal cardiac workload, narrows the septal defect and eventually obliterates it.

Recognition of the fact that small defects of the muscular ventricular septum will close without treatment is of practical importance. These lesions are not uncommon; they can be identified by clinical assessment with reasonable certainty; and, the prognosis is uniformly good. Spontaneous closure of large ventricular septal defects, however, must be rare and unfortunately the clinical signs are not sufficiently distinctive to permit prediction of a favorable outcome.

Summary

A group of 37 children is described in whom a systolic murmur heard early in life gradually diminished and eventually disappeared. When the patients were first seen, the clinical findings suggested a small ventricular septal defect but no thrill was present and the systolic murmur had a superficial blowing quality with high-frequency vibrations and tended to stop before the second heart sound. Cardiac catheterization demonstrated a small left-to-right shunt at ventricular level in 4 of the patients while the murmur was present; in 1 this was repeated after the murmur had gone and no abnormality could be demonstrated.
Cardiac catheterization in other patients with typical disappearing systolic murmurs showed a left-to-right shunt in some but in others this was too small to be detected by routine oxygen studies. A rough correlation was established between the length and intensity of the murmur and the size of the shunt. With angiocardiography and intracardiac phonocardiography the exact site of the ventricular septal defect was localized to the muscular portion of the septum in 4 of the patients.

In 1 patient who presented with congestive heart failure, clinical and hemodynamic findings of a large ventricular septal defect diminished over several years and finally disappeared.

Children with the specific type of systolic murmur described may be recognized as having a small defect in the muscular ventricular septum. The defect is thought to be gradually reduced in size and ultimately closed by hypertrophy of septal muscle. Spontaneous closure appears to be not uncommon with small ventricular septal defects and may rarely occur with lesions large enough to present with congestive heart failure.

Summario in Interlingua
Es descriti un gruppo de 37 juveniles in qui un murmure systolic, audite tosto in le curso de lor vitas, se reduciva gradually e dispareva in le curso del tempore. Quando le pacientes esseva primo vidite, le constatationes clinicae suggereva un miere defecto ventriculo-septal, sed nulle fremito esseva presente, et le murmure systolic habeva un qualitato de sullation superficial con vibrationes ali-frequential e tendeva a arrestar se ante le secunde sono cardiac. Catheterismo cardiae demonstrava un miere shunting sinistro-dextere al nivello ventricular in 4 del paciente durante le tempore quando le murmure esseva presente. In un, le catheterismo esseva repetite post le disparition del murmure, e nulle anormalitate poteva esser demonstrate.

In altere casos typic de disparente murmures systolic, le catheterismo cardiae mostrava, in plures, un shunting sinistro-dextere, sed frequentemente isto esseva troppo miere pro esser detegite per routinari studios de oxygeno. Un crude correlazione esseva establishe inter le longor e le intensitate del murmure e le magnitude del shunting. Per medio de angiocardiographia e phonocardiographia intracardiae le exete sito del defecto ventriculo-septal esseva fixate in le portion muscular del septo in 4 del pacientes.

In 1 caso, presentate con congestive disfallimento cardiae, le constatation clinica e hemodynamic de un grande defecto ventriculo-septal se attenuava in le curso de plure annos e finally dispareva.

Juvenes con le typo specific del hic-describite murmure systolic pote esser recognoscite como suffrante de un miere defecto in le portion muscular del septo. Es opinate que le defecto reducite gradualmente e claudite ultimemente per hypertrophia del muscolo septal. Il pare que le clauditione spontanea de miere defectos ventriculo-septal non es incommun. In rar casos, clauditione spontanea pote occurrer quando le lesion es satis marente pro presentar le symptomas de congestive disfallimento cardiae.

References
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Circulation. 1960;22:1044-1054
doi: 10.1161/01.CIR.22.6.1044

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1960 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

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