Auscultatory and Phonocardiographic Findings in Ventricular Septal Defect
A Study of 93 Surgically Treated Patients

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The auscultatory features of ventricular septal defect have aroused much interest since Roger’s original description of the murmur in 1879.

Wood et al. noted that the systolic murmur began early and spilled over the second sound. A mitral diastolic murmur was heard in 90 per cent of their severe cases and they attributed it to a torrential mitral blood flow. Mannheimer et al. showed that the systolic murmur was faint and was followed by a loud single second sound in patients with a high pulmonary resistance and this was confirmed by Bleifer et al., who also noted that the majority of such patients had a pulmonary ejection sound. Lessof found a pulmonary regurgitant murmur in 40 per cent of patients with a pulmonary artery pressure over 30 mm. Hg. Using intracardiac phonocardiography, Furgiolo and Gunton showed that the mid-diastolic murmur was recorded only in the left ventricle, thus confirming its mitral origin. Craig correlated the catheterization findings with the phonocardiogram and found that those with large left-to-right shunts, or smaller shunts with only slight pulmonary hypertension, had pansystolic diamond-shaped murmurs. When severe pulmonary hypertension was present, the murmur was short, being completed in the first third of systole, the second sound was loud and narrowly split, and an ejection click was always present. In patients with very large shunts, Leatham and Segal showed that a pulmonary ejection murmur was superimposed on the pansystolic murmur and that the wide splitting of the second sound was associated with delay in contraction of the right ventricle.

Material and Methods

This study is based on 93 patients whose ventricular septal defect had been completely closed at open-heart surgery and in whom a phonocardiogram was available. All patients had a moderate or large defect without a right-to-left shunt; thus this paper includes no examples of maladie de Roger or of the Eisenmenger syndrome.

In all patients the presence and severity of pulmonary vascular disease were judged from the extent of residual pulmonary hypertension after operation. The mean pulmonary artery pressure was measured in the operating room after closure of the defect and expressed as a percentage of the mean systemic pressure. Because the defect is closed, this percentage is in fact the ratio of pulmonary-to-systemic resistance and we use it as the best available index of pulmonary vascular disease.

In the following analysis our chief concern is to show how the murmurs and heart sounds serve as a guide to pulmonary vascular disease, since this is the chief determinant of success or failure in operation in ventricular septal defect.

Systolic Murmur

After unsuccessful attempts to measure the area of the murmur or its length as a percentage of mechanical systole, we divided the murmurs on the basis of the phonocardiogram into three groups: pansystolic, long ejection, and short ejection (fig. 1). These groups were then correlated with the pulmonary artery pressure after operation (the index of pulmonary vascular disease) (table 1).

The results are striking and show that a pansystolic murmur rarely occurred in patients who were left with severe pulmonary hypertension after operation. Conversely, patients with a short ejection murmur never had a low pulmonary pressure postoperatively. When a long ejection bruit was found, only one eighth had a normal pressure, while the majority had moderate or severe postoperative pulmonary hypertension.

The systolic murmur therefore provides a very

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good guide to the presence and severity of pulmonary vascular disease.

The varying shape and length of the murmur are probably related to the different pattern of flow through the defect as the right and left ventricular pressures become more nearly equal. Figure 2 is a schematic diagram showing how the murmur might be expected to alter with increasingly severe right ventricular hypertension. The actual amount of blood passing through the defect is obviously of importance too, although in this series of patients selected for surgery, the shunt naturally was usually fairly large. In table 2 the preoperative pulmonary hemodynamics are related to the three varieties of systolic murmur, and the rise in pulmonary pressure and resistance as the murmur shortens is well shown.

**Varying Types of Pansystolic Murmur**

The shape of the murmur was not uniform in the pansystolic group. The chief patterns are shown in figure 3 and are related to the hemodynamic data. It can be seen that when the bruit is rectangular in shape, the pulmonary hypertension is always trivial. Otherwise the variations in shape seem to have little significance.

**Intensity of Systolic Murmur**

Reduction of flow through the defect by severe pulmonary vascular disease would be expected to lessen the intensity of the bruit, and the following data show this to be so. There were three patients with only moderately loud (grade II/IV) systolic murmurs, and after closure of the defect they all had severe pulmonary hypertension. Two died within a few days of surgery and the third was in heart failure for over 1 year after operation. A similar trend was found in the 30 patients with grade-III/IV murmurs. A grade-III murmur was found in only 19 per cent with slight, in 43 per cent with moderate, and in 67 per cent with severe pulmonary hypertension after operation.

**Systolic Thrill**

In many patients the systolic murmur was accompanied by an obvious systolic thrill at the left sternal edge, but in 26 patients the thrill was soft or absent. Only one of these had a large uncomplicated defect, and in four the poor thrill was due to a rather small shunt through a small defect. In the remaining 21, there was an anatomic or physiologic complication in every patient, as shown in table 3.

In the majority of these patients severe pulmonary hypertension was the cause of the weak or absent thrill, acting probably by a reduction of flow through the defect. In five the defect was anatomically unusual, there being multiple defects in three and a muscular or supracristal defect in two others.

Therefore in a patient thought to have a large ventricular septal defect, the absence of an obvious thrill should lead one strongly to suspect either severe pulmonary hypertension or an anatomic complication of the types shown in table 3.
Table 2
Hemodynamic Data from Preoperative Cardiac Catheterization Related to the Type of Systolic Murmur

<table>
<thead>
<tr>
<th>Type of murmur</th>
<th>Pulmonary artery systolic pressure (mm. Hg)</th>
<th>Pulmonary-to-systemic flow ratio</th>
<th>Pulmonary arteriolar resistance (units)</th>
<th>Pulmonary stenosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pansystolic</td>
<td>15-120</td>
<td>1.2-5.0</td>
<td>0.5-20</td>
<td>3.1</td>
</tr>
<tr>
<td>Long ejection</td>
<td>65-88</td>
<td>1.3-4.5</td>
<td>1.5-20</td>
<td>8.0</td>
</tr>
<tr>
<td>Short ejection</td>
<td>70-100</td>
<td>1.3-2.0</td>
<td>6-17</td>
<td>10.1</td>
</tr>
</tbody>
</table>

Table 3
Anatomic or Physiologic Features in 26 Patients with Weak or Absent Systolic Thrill

| Multiple ventricular septal defects plus patent ductus | 2 | Severe pulmonary hypertension plus patent ductus | 11 |
| Multiple ventricular septal defects plus patent ductus | 1 | Severe pulmonary hypertension plus patent ductus | 2 |
| Muscular defect                                          | 1 | Severe pulmonary hypertension plus mitral incompetence | 1 |
| Supracristal defect                                      | 1 | Severe pulmonary hypertension plus muscular defect | 1 |
| Associated patent ductus                                 | 1 | Uncomplicated                                      | 1 |

Site of Systolic Murmur and Thrill
In the majority of patients the murmur and thrill caused by the ventricular defect were maximal at the left third to fourth parasternal space. In those with associated pulmonary valve stenosis, the murmur was often louder at the pulmonary area and was transmitted toward both clavicles. The chief interest in the site of the murmur concerns those patients with a defect situated in other than the usual infracristal position. In two patients with a defect shunting directly into the right atrium the thrill and bruit were definitely atypical in position, being maximal between the apex and the left sternal edge. There were three patients with a defect in the muscular septum and the systolic bruit was heard at the usual left sternal edge site in two; in the third patient associated mitral disease confused the picture. In four patients with a lone supracristal defect the systolic murmur was heard maximally toward the pulmonary area.

Associated Lesions Giving a Systolic Murmur
Three patients had associated mitral incompetence, and all had apical pansystolic murmurs. The murmur was not easy to separate from that caused by the ventricular defect, and in all three a loud apical diastolic murmur helped in the diagnosis. In two patients with associated aortic stenosis who are not in this present series, the appropriate systolic murmur was easily recognized. The auscultatory features of those with corrected transposition or peripheral pulmonary artery stenosis have already been reported by us.9 That paper also details the important syndrome associated with ventricular septal defect in which aorta and pulmonary artery both arise from the right ventricle; this syndrome often has a distinctive auscultatory feature, in that a loud ejection systolic murmur or thrill is noted at the right second space.

Mitral Diastolic Murmur
The apical diastolic murmur in ventricular septal defect is usually attributed to the increased flow across the mitral valve and the murmur certainly disappears after operation. In table 4 the length of the mitral diastolic murmur is related to the degree of pulmonary vascular disease and to the presence of pulmonary stenosis in 86 patients without mitral disease.

As can be seen, the length of the mitral diastolic murmur gives little or no indication as to what degree of pulmonary hypertension will be present after surgery. Long bruits occurred with severe, and short bruits with mild pulmonary vascular disease. Furthermore, in two patients with mitral valve disease the presence of a valve lesion was not suspected until the mitral bruit persisted after complete closure of the defect. To have used the diastolic murmur in these patients as an index of pulmonary vascular disease would, of course, have been very misleading.

Circulation, Volume XXVIII, July 1968
SOUNDS IN VENTRICULAR SEPTAL DEFECT

Table 3

The four varieties of pansystolic murmur are related here to the hemodynamic data.

Mitral Valve Disease

Seven patients had associated congenital mitral valve disease.

Four had mitral stenosis and the loud long diastolic bruit that was heard in all was accompanied by a thrill in two. Only one patient required mitral valvotomy, and in him there was in addition a presystolic thrill and murmur. Two had combined incompetence and stenosis with apical systolic and diastolic thrills. One patient had mild incompetence only, with an apical pansystolic bruit. One patient (with mild stenosis) had an opening snap.

Differentiation of the mitral systolic bruit from that caused by the ventricular defect was comparatively easy in those with severe pulmonary hypertension (fig. 4), for in them the ventricular septal defect bruit was ejection in character contrasting with the pansystolic murmur at the apex—and in fact the mitral signs dominated the clinical picture in two such patients. In others the presence of the loud diastolic bruit helped to focus attention on the mitral valve.

Early Diastolic Murmur

An early blowing diastolic bruit at the base of the heart was heard in 26 patients, and the causes are shown in table 5.

The great importance of the early diastolic murmur in ventricular septal defect lies in the fact that it may be the only clue to the presence of an associated patent ductus arteriosus. Extracorporeal circulation is impossible until the ductus has been ligated and so prior knowledge of its presence is essential. We could not reliably distinguish clinically between the various causes of an early diastolic bruit (fig. 5) and, hence, used aortography to confirm or deny the presence of a ductus. In the moderately severe instances of aortic incompetence, however, the murmur was well heard down the left sternal edge, whereas in patent ductus it was confined to the pulmonary area. The murmur of pulmonary incompetence was of interest because it occurred for two quite different reasons. In three patients the pulmonary valve was grossly deformed and all had a considerable systolic pressure gradient in the right ventricle due to infundibular hypertrophy. These patients, as shown

Table 4

Length of Apical Diastolic Murmur Related to the Degree of the Postoperative Pulmonary Artery Pressure and to Presence of Pulmonary Stenosis

Table 5

Lesions Giving Rise to Basal Early Diastolic Murmur in 26 Patients
Figure 6

Phonocardiograms from three patients with pulmonary incompetence due to a deformed pulmonary valve and associated with infundibular stenosis. Note the greatly delayed onset of the diastolic murmur.

Figure 7

Splitting of the second heart sound (a) close split, with a virtually single second sound, with severe pulmonary hypertension, (b) normal split with mild pulmonary hypertension, (c) wide split, with associated pulmonary stenosis.

in figure 6, had a very delayed onset to the diastolic murmur due presumably to much delayed pulmonary valve closure from the infundibular obstruction. In all three the bruit was loud and accompanied by a thrill. In two of them the valve lesion was probably congenital with the cusps being very small in one, and adherent to the pulmonary artery in the other. In the third patient, the cusps were small and deformed, probably from previous endocarditis.

Second Heart Sound

Our chief concern in relation to the second heart sound was its degree of splitting, i.e., the interval between aortic and pulmonary closure. This splitting was graded on the basis of the expiratory phonocardiogram, into close (less than 0.02 second), normal (0.02 to 0.04 second), and wide (over 0.04 second) (fig. 7).

The relation between the degree of splitting and the severity of pulmonary vascular disease is shown in table 6, as is the relation to pulmonary stenosis.

The degree of splitting is shown to be a quite useful guide to pulmonary vascular disease. When the second sound is closely split, the patients nearly always have moderate or severe pulmonary hypertension after operation, whereas a widely split second sound is confined to those with slight or absent pulmonary vascular disease. Over half of those with a wide split had pulmonary stenosis.

Table 6

<table>
<thead>
<tr>
<th>Width of splitting of second sound</th>
<th>Pulmonary artery mean pressure after operation as per cent of systemic mean pressure</th>
<th>Pulmonary stenosis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>10-29</td>
<td>30-49</td>
</tr>
<tr>
<td>Close</td>
<td>2</td>
<td>11</td>
</tr>
<tr>
<td>Normal</td>
<td>29</td>
<td>17</td>
</tr>
<tr>
<td>Wide</td>
<td>22</td>
<td>0</td>
</tr>
</tbody>
</table>

The intensity of the pulmonary component of the second sound at the pulmonary area was graded into soft, normal, and loud; this was done at auscultation and not on the phonocardiogram. Of 21 patients with a loud second sound, 18 had moderate or severe pulmonary hypertension postoperatively. Conversely, the 17 patients with soft pulmonary closure all had a near normal pulmonary artery pressure after operation, 14 of the 17 having pulmonary stenosis.

Pulmonary Stenosis

Twenty-two patients had pulmonary stenosis and this lesion had an important influence on the auscultatory findings, although in no patient was it severe enough to render the left-to-right shunt small or absent. In eight, the stenosis was primarily valvular and in 14 primarily infundibular—usually hypertrophied muscle, but occasionally a true diaphragm. With pulmonary stenosis the systolic murmur was always pansystolic and was often accompanied by an intense thrill at the pulmonary area, which sometimes had a “tearing” quality. Being loud and pansystolic, the murmur tended to obscure aortic valve closure. The second heart sound was abnormal in all patients. In the few with severe stenosis pulmonary closure was inaudible and the second sound was therefore single (fig. 8). In the majority the second sound was widely split and the loudness of pulmonary closure varied with the severity of the obstruction, being even accentuated in some with mild stenosis. The
site of the stenosis as distinct from its severity was not predictable from the auscultatory findings.

**Pulmonary Ejection Click**

An ejection click (fig. 4) over the pulmonary area was heard in six patients. In four there was moderate or severe pulmonary vascular disease, and the click was followed by an ejection systolic murmur. In one there was a very high pulmonary artery pressure due to a large shunt. The sixth patient had a moderate-sized defect with an infundibular gradient of 25 mm. Hg, and the reason for the click was obscure. No other patient with infundibular or valvular stenosis had an ejection click and neither did those patients with an associated atrial septal defect.

**Discussion**

The length and intensity of the systolic murmur provide a very clear guide to the severity of pulmonary vascular disease in ventricular septal defect. We have used the systolic thrill and murmur in the selection of patients for surgery for over 3 years and have found them to be most reliable. In this respect they contrast at times with the results of right heart catheterization. When catheterization demonstrates a considerable elevation of pulmonary vascular resistance, with a small left-to-right shunt, one would expect substantial pulmonary hypertension to be present after surgery. However, we have seen five patients with a pulmonary arteriolar resistance of over 9 units (720 dynes) who had a normal pulmonary artery pressure after operation. In all these five patients there was a pansystolic murmur which correctly indicated, in contrast to the catheterization findings, the absence of serious irreversible pulmonary vascular disease. The only exception found was in a patient with ventricular septal defect, pulmonary stenosis, and patent ductus in whom the pansystolic bruit was caused by the first two lesions, and the pulmonary vascular disease, it is assumed, by the ductus.

Conversely, there are occasionally patients in whom calculated pulmonary vascular resistance at catheterization is normal, but an ejection bruit indicates severe pulmonary vascular disease as confirmed by the persistence of pulmonary hypertension after operation.

Fyler et al.,

...
of pulmonary vascular disease, as measured by the postoperative pulmonary artery pressure.

The systolic murmur of the ventricular septal defect proved to be an excellent guide to the severity of pulmonary vascular disease. With a pansystolic murmur there was rarely severe pulmonary vascular disease, but with a short ejection murmur moderate or severe pulmonary vascular disease was always present.

The systolic thrill was weak or absent in 26 patients, and 80 per cent of these had an anatomic complication or severe pulmonary hypertension.

The mitral diastolic murmur was not a good guide to the severity of pulmonary vascular disease.

An associated patent ductus arteriosus gave rise to an early diastolic murmur, never to a continuous murmur, and on auscultatory grounds it could not reliably be distinguished from pulmonary or aortic incompetence.

The degree of splitting and the intensity of the second heart sound correlated well with the presence and severity of pulmonary vascular disease.

Pulmonary stenosis produced characteristic auscultatory features.

A pulmonary ejection click usually indicated the presence of moderate or severe pulmonary vascular disease.

References


Limits of Finite Knowledge

In practical terms . . . our knowledge is finite and never all-encompassing. There is always much that we miss, much that we cannot be aware of because the very act of learning, of ordering, of finding unity and meaning, the very power to talk about things means that we leave out a great deal.—J. Robert Oppenheimer. “On Science and Culture.” Encounter, October, 1962, p. 9.
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Circulation. 1963;28:94-100
doi: 10.1161/01.CIR.28.1.94

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