Intracardiac Phonocardiography in Ventricular Septal Defect

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It has been demonstrated that intracardiac phonocardiography provides precise localization of the source of cardiovascular sounds. Can this technique be of practical help in the diagnosis of small or complicated ventricular septal defects? Is a ventricular septal defect ever silent? What mechanisms are responsible for the diastolic murmurs in ventricular septal defect? In an attempt to answer these problems, intracardiac phonocardiograms have been studied in 47 cases.

The most useful clinical sign of ventricular septal defect is a loud, harsh, pansystolic murmur with maximal intensity over the midprecordium (Roger murmur). This murmur is often accompanied by a mitral diastolic rumble, and by an early diastolic murmur at the base or along the left sternal border of uncertain origin. This typical auscultatory picture may be greatly altered when ventricular septal defect is associated with high pulmonary vascular resistance, pulmonary stenosis, overriding aorta, or aortic insufficiency. The role of the septal defect in the production of sounds in these conditions, cannot be established by simple auscultation or ordinary chest phonocardiography.

Previous experience has demonstrated that intracardiac phonocardiography provides precise localization of the source of cardiovascular sound. The practical value of this information has been shown in particular in ventricular septal defect in which the intracardiac phonocardiogram is of diagnostic value even in the presence of equivocal catheterization and dye-dilution studies, by localizing a Roger murmur within the right ventricle.

It is the purpose of this paper to present the intracardiac phonocardiographic findings in 47 proved cases of ventricular septal defect and to emphasize the value of intracardiac phonocardiography not only in establishing the diagnosis of ventricular septal defect, but also in clarifying the site and mechanism of the basal and apical diastolic murmurs and the role of the interventricular communication in complicated ventricular septal defect.

Material and Methods

The 47 patients included in this study ranged in age from 8 to 43 years, the majority being in the second decade. There were 20 male and 27 female subjects. Right heart catheterization was done in all patients. The study included calculation of systemic and pulmonary blood flows and recording of indicator-dilution curves from multiple sites. In 12 cases the septal defect was crossed and in 8 the aorta was catheterized. In addition, 26 patients had selective biplane angiocardiography at a speed of 1 to 12 frames per second. Twelve had corrective surgery with use of cardiopulmonary bypass and 4 were examined post mortem.

The cases have been grouped as in table 1.

Table 1

Diagnoses in Forty-seven Cases of Ventricular Septal Defect

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Number of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uncomplicated ventricular septal defect</td>
<td>18</td>
</tr>
<tr>
<td>Ventricular septal defect with aortic insufficiency</td>
<td>2</td>
</tr>
<tr>
<td>Ventricular septal defect with pulmonary hypertension</td>
<td>10</td>
</tr>
<tr>
<td>Ventricular septal defect with pulmonary stenosis (normal aortic root and normal peripheral arterial oxygen saturation)</td>
<td>3</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>14</td>
</tr>
</tbody>
</table>

*Two cases in this group had complete atrioventricular block and 1 case was of traumatic origin.
Intracardiac sounds were recorded by means of the sound catheter originally described by Lewis et al. The intracardiac microphone is a hollow cylinder of activated barium titanate incorporated in the tip of a specially designed catheter which is approximately the size of a no. 5 Courand catheter. The output of the microphone after preliminary amplification by a 3-stage transistorized pre-amplifier was fed into a 2-channel photographic recorder used for routine clinical phonocardiography. All records were made at the paper speed of 75 mm. per second. The phonocatheter was introduced under fluoroscopic guidance following routine cardiac catheterization. During the latter procedure reference marks were placed on the fluoroscopic screen to locate the pulmonary and tricuspid orifices. Simultaneous intracardiac and external sound recordings were also obtained.

**Phonocatheters and pre-amplifiers were built and made available to the authors by the Naval Air Development Center of Johnsville, Pennsylvania.

Figure 1

Intracardiac phonocardiograms in a normal subject and in a patient with ventricular septal defect. In the normal subject (left) note, A, the short systolic flow murmur in the pulmonary artery; B, the absence of murmurs in the right ventricle; C, the absence of murmurs and sounds in the right atrium. In the patient with ventricular septal defect (right) note, D, the intense flow murmur in the pulmonary artery; E, the loud pansystolic murmur in the right ventricle; F, the absence of murmurs in the right atrium.

Results

Uncomplicated Ventricular Septal Defect

Of the 18 cases with uncomplicated ventricular septal defect, 7 had rather small defects and a pulmonary blood flow less than twice the systemic; 11 had large left-to-right shunts with a pulmonary blood flow 2 to 3 times the systemic flow. In the latter group of 11 patients were 2 cases of complete atrioventricular block. In the former group there was 1 case of traumatic origin.

The most striking intracardiac phonocardiographic feature in all, compared to normal subjects, was a loud, harsh, pansystolic murmur within the right ventricle (fig. 1). This murmur was usually loudest in the outflow tract of the ventricle and decreased toward the apex. In the case of the traumatic defect (fig. 2), however, in which the opening was localized by the course of the catheter
INTRACARDIAC PHONOCARDIOGRAPHY

Figure 2

Intracardiac phonocardiogram in a patient with traumatic ventricular septal defect. Note the loud pansystolic murmur recorded near the apex of the right ventricle and the flow murmur in the pulmonary artery.

Figure 3

Simultaneous intracardiac phonocardiogram from the right ventricle and chest phonocardiogram in a case of ventricular septal defect. Note the similarity in the character and duration of the murmur.

Figure 4

Intracardiac phonocardiogram in ventricular septal defect with large left-to-right shunt. Note, A, flow murmur in the pulmonary artery; B, absence of murmurs in the right atrium; C, pansystolic murmur in the right ventricle; D, middiastolic-murmur with presystolic accentuation in the left ventricle due to increased flow across the mitral valve.

Within the pulmonary artery, a flow murmur louder than that usually recorded in normal subjects (fig. 1) was observed. The pulmonary valve closure was of normal intensity or slightly accentuated.

An early diastolic murmur was recorded in the upper outflow tract of the right ventricle just beneath the pulmonary valve, in only 1 of the 11 cases with large left-to-right shunt. In 3 patients with large left-to-right shunt in whom an apical diastolic rumble was present on auscultation, the sound catheter was passed across the defect into the left ventricle. In this chamber no systolic murmur was recorded. There was a long diastolic murmur with the characteristics of a mitral murmur: low-pitched, “decrecendo” in middiastole, with presystolic accentuation (fig. 4D).

In the 2 cases with complete atrioventricular block and large left-to-right shunt, the intracardiac phonocardiographic features were
those encountered in the majority of uncomplicated ventricular septal defects. In the right atrium, however, an atrial sound followed each P wave of the electrocardiogram (fig. 5).

Ventricular Septal Defect with Aortic Insufficiency

The 2 patients with ventricular septal defect and aortic insufficiency proved by retrograde aortography, had on clinical auscultation and external phonocardiography a continuous murmur, which could not be differentiated from that of a patent ductus arteriosus, aortic septal defect, or ruptured sinus of Valsalva (fig. 6). In these cases, the correct diagnosis could not be established by routine heart catheterization.

The intracardiac phonocardiogram (fig. 6) showed a loud pansystolic murmur within the right ventricle as in isolated ventricular septal defect and no diastolic murmur. In the pulmonary artery a midsystolic murmur and a loud pulmonary second sound were recorded. There were no unusual findings in the right atrium.

On the basis of the absence of a continuous murmur within the pulmonary artery, the diagnoses of patent ductus arteriosus and aortic septal defect were excluded. The possibility of rupture of a sinus of Valsalva into the right ventricle or right atrium could also be excluded because of the absence of a continuous murmur in these chambers.

Ventricular Septal Defect with Pulmonary Hypertension

Ten patients with ventricular septal defect had a pulmonary systolic arterial pressure greater than twice the normal value. Of these, 7 had a small left-to-right shunt and 3 had a large left-to-right shunt with a pulmonary blood flow 2 to 3 times the systemic flow. In all patients a systolic murmur was recorded within the right ventricle. This murmur was
extremely loud occupying all systole, in the 3 cases with large left-to-right shunt; it was very soft and limited to the first part of systole in the 7 cases with small left-to-right shunts (fig. 7). A comparable difference in intensity was noted in the mid-systolic murmur recorded within the pulmonary artery; this murmur having less intensity with smaller pulmonary blood flow. Features common to both groups were an early diastolic murmur within the outflow tract of the right ventricle and a loud pulmonary second sound in the pulmonary artery. On auscultation an early blowing diastolic murmur had been heard in the second and third left intercostal spaces along the sternal border in 8 patients. No abnormal features were noted within the right atrium.

**Ventricular Septal Defect with Pulmonary Stenosis**

Three patients in addition to a ventricular septal defect had a pressure gradient of 50 mm. Hg or more across the pulmonary valve, a pulmonary blood flow up to 3 times the systemic flow, a normal aortic root, and normal peripheral arterial oxygen saturation.

The intracardiac phonocardiogram (fig. 8) from within the pulmonary artery, showed a midsystolic murmur of great intensity and a soft, delayed pulmonary second sound. These findings are similar to those observed within the pulmonary artery in cases of isolated pulmonary stenosis (fig. 8A).

In the right ventricle a loud pansystolic murmur was constantly present in cases of ventricular septal defect with pulmonary stenosis falling in this group (fig. 8E); it was absent in cases of isolated pulmonary stenosis (fig. 8B). No abnormal findings were observed within the right atrium in this group.

**Tetralogy of Fallot**

Fourteen patients had in addition to an interventricular communication, the other characteristic features of Fallot's tetralogy: severe pulmonary stenosis and overriding aorta. In all, the left-to-right shunt was insignificant and there was peripheral arterial oxygen unsaturation. The intracardiac phonocardiogram in these cases (fig. 9) revealed a loud diamond-shaped murmur within the pulmonary artery and a very soft and delayed pulmonary second sound occurring 0.08 to 0.12 second after the aortic second sound. No murmurs were recorded within the right ventricle in 8 patients; in 6 an early soft systolic murmur was observed. Within the aorta an early, usually soft systolic murmur and a loud aortic second sound were recorded.

**Discussion**

A loud pansystolic murmur sharply localized within the right ventricle and usually loudest in the outflow tract of this chamber, is a constant sign in uncomplicated ventricular septal defect. It corresponds in shape and duration to the murmur recorded externally from the third and fourth left intercostal...
spaces and described in detail by Roger 80 years ago. Although by auscultation and chest phonocardiography the murmur may be present over a large precordial area, when recorded by the phonocatheter it is localized in the ventricular chamber and disappears dramatically when the catheter is withdrawn into the right atrium or advanced into the pulmonary artery. It is because of this precise localization that intracardiac phonocardiography is of exceptional value in the diagnosis of ventricular septal defect.

The intensity of the intracardiac murmur is not related to the size of the defect in isolated ventricular septal defect. It is this fact which makes intracardiac phonocardiography the technic of choice in the diagnosis of small defects with shunts less than 0.5 L/min, in which blood oxygen determinations fail to establish the presence of an interventricular communication. A small ventricular septal defect is one possibility to be considered in the differential diagnosis of an "innocent left parasternal murmur."  

Errors in the diagnosis of ventricular septal defect from oxygen saturation studies are not infrequent and may be avoided with the help of intracardiac phonocardiography. It is known that depending on the position of the septal defect, the jet of arterial blood from the left ventricle may enter either the tricuspid or the pulmonary valve, yielding a misleading oxygen rise in the atrial or pulmonary blood samples. In this situation a ventricular septal defect may be overlooked on the basis of the oxygen studies alone.

In other conditions, low-lying atrial septal defect or large patent ductus arteriosus with pulmonary incompetence, a significant oxygen rise may be detected in the right ventricle suggesting interventricular communication which is in fact not present. In this case the
Intracardiac phonocardiograms in 2 subjects with pulmonary stenosis: one with intact ventricular septum (left), the other with ventricular septal defect and left-to-right shunt (right). Note in each subject, in the pulmonary artery (A and D) the diamond-shaped murmur of pulmonary stenosis, and a soft, delayed pulmonary closure. In B the silent right ventricle in the absence of ventricular septal defect is contrasted in E with the loud pansystolic murmur of ventricular septal defect.

intracardiac phonocardiogram from the right ventricle will reveal no systolic murmur and will exclude ventricular septal defect.

The combination of ventricular septal defect with aortic insufficiency, congenital or acquired, is not rare. It was reported to be present in 5 per cent of cases in a recent series of ventricular septal defects. This combination may be mistaken clinically and at catheterization for patent ductus arteriosus, aortic septal defect, or ruptured aneurysm of a sinus of Valsalva into the right ventricle. Thoracotomy has been performed in error because of the incorrect diagnosis of patent ductus arteriosus. The differential diagnosis is simplified by intracardiac phonocardiography. In cases of ventricular septal defect with aortic insufficiency there will be a typical Roger murmur in the right ventricle; in cases of patent ductus arteriosus or ruptured sinus of Valsalva into the right ventricle there will be a continuous machinery murmur within the pulmonary artery or the right ventricle respectively.

When ventricular septal defect is complicated by high pulmonary vascular resistance or pulmonary stenosis, the intensity of the systolic murmur in the right ventricle decreases proportionally to the left-to-right shunt. The murmur also becomes shorter and may be present only in one part of systole. It never disappears. In the presence of large left-to-right even with elevated pulmonary arterial pressure, a very loud pansystolic mur-
mur is to be expected in the right ventricle. This sign is of great importance in the differential diagnosis between ventricular septal defect with pulmonary hypertension and patent ductus arteriosus with pulmonary hypertension plus pulmonary valvular insufficiency. This diagnostic problem was stressed in a recent report\textsuperscript{15} in which 10 surgically proved cases of patent ductus arteriosus simulated so closely ventricular septal defect that surgical correction by extracorporeal circulation was initially considered for each of the patients and was attempted in 2.

Only in cases of severe tetralogy of Fallot is a systolic murmur absent in the right ventricle, despite the presence of ventricular septal defect. In this condition because of the equal pressure in the ventricles and the overriding aorta "blood does not flow from one ventricle to the other but from both ventricles into the aorta."\textsuperscript{16} The ventricular septal defect is silent as already suggested\textsuperscript{17,18} and the pulmonary stenosis is responsible alone for the harsh systolic murmur heard over the mid-precordium.\textsuperscript{19}

With respect to the early basal diastolic murmur in ventricular septal defect, 3 possible mechanisms may be suggested: pulmonary insufficiency, aortic insufficiency, and flow across the defect.\textsuperscript{2} It seems unlikely that flow across the defect is responsible for such a diastolic murmur. It was not observed in the right ventricular phonocardiograms of the 2 cases in this series, with complete atrioventricular block and large pulmonary blood flow in which the very prolonged diastole should have created the best hemodynamic conditions for an interventricular shunt. Both aortic and pulmonary insufficiency may be responsible for the early basal diastolic murmur in ventricular septal defect. Intracardiac phonocardiography permits differentiation of the aortic from the pulmonary diastolic murmur because only the latter is recorded in the outflow tract of the right ventricle. No diastolic murmur was observed in this chamber in the cases of ventricular septal defect with aortic insufficiency included in this series.

A diastolic murmur of functional pulmonary incompetence was recorded in all cases of ventricular septal defect with pulmonary hypertension, whether the left-to-right shunt was large or small. Such a murmur was recorded only once in uncomplicated ventricular septal defect. In this case the left-to-right shunt was large and pulmonary blood flow was over 3 times the systemic flow. One may conclude that with the exception of the cases in which aortic insufficiency is present, an early diastolic murmur in ventricular septal defect is indicative either of high pulmonary vascu-

\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure9.png}
\caption{Intracardiac phonocardiogram in a subject with severe tetralogy of Fallot. There is a loud ejection type of murmur in the pulmonary artery and a delayed soft pulmonary valve closure. The right ventricle is silent, despite the proved presence of the ventricular septal defect, due to the absence of left-to-right shunt across the defect. There is a soft systolic murmur within the aorta and a prominent aortic valve closure.}
\end{figure}

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lar resistance or high pulmonary blood flow.

The mitral origin of the apical diastolic murmur in ventricular septal defect, first described by Laubry and Pezzi in 1921 has been confirmed beyond doubt by the sound catheter, which demonstrated such a murmur within the inflow tract of the left ventricle just beneath the mitral valve. A similar murmur due to increased flow across the mitral valve has been recorded in other conditions such as tricuspid atresia.

Summary

Intracardiac phonocardiograms, with use of a barium titanate microphone, have been recorded in 47 proved cases of ventricular septal defect.

A loud pansystolic murmur localized within the right ventricle is a diagnostic sign of uncomplicated ventricular septal defect, even when clinical and catheterization findings are equivocal.

In ventricular septal defect complicated by pulmonary hypertension or pulmonary stenosis, the murmur is always present with left-to-right shunt and is proportional in duration and intensity to the volume of the shunt.

In severe tetralogy of Fallot with no left-to-right shunt, the ventricular septal defect is silent.

Intracardiac phonocardiography permits the differential diagnosis of ventricular septal defect with aortic insufficiency, from patent ductus arteriosus, aortic septal defect, and rupture of a sinus of Valsalva.

The mitral origin of the apical diastolic murmur in ventricular septal defect has been confirmed.

Evidence supports the view that the early diastolic murmur in ventricular septal defect does not rise from the defect itself but from pulmonary valvular insufficiency.

Summario in Interlingua

Per medio de microphonos a titaneo de barium, phonocardiogrammas intracardiaque esseva registrate in 47 provate casus de defecto ventriculo-septal.

Un forte murmure pansystolique, locate intra le ventriculo dextere, es un signo diagnostique de non-complicato defecto ventriculo-septal, mesmo quando le constatationes clinis e catheteria es equivoe.

In casus de defecto ventriculo-septal complicato per hypertension pulmonar o stenosis pulmonar, le murmure es semper presente con un shunting sinistro-dextere e es proportional in duration e intensitate al volumine del shunting.

In sever tetralogia de Fallot sin shunting sinistro-dextere, le defecto ventriculo-septal es silente.

Phonocardiographia intracardiaque permitte le diagnosto differential de defecto ventriculo-septal con insufficiencia aortic contra patente ducto arterioso, defecto aorto-septal, e ruptura del sino de Valsalva.

Le origine mitral del diastolic murmure apical in defecto ventriculo-septal eseva confirmate.

Es presentate datos que supporta le conception que le murmure codiastolic in defecto ventriculo-septal ha su origine non in le defecto mesme sed in insufficiencia pulmono-valvular.

References

10. —, Gunton, R. W., Sreenivasan, A., and Heim-

Four case reports of rupture of the infarcted ventricular septum are presented. The incidence of this complication was 0.5 per cent among all hospitalizations for myocardial infarctions during 1954-1958. The patients were elderly, ranging from 65 to 84 years. In each instance a characteristic harsh pansystolic apical or lower left sternal area murmur and thrill were detected within 1 week following the onset of a first myocardial infarction, and each patient then developed fatal hypotension and heart failure. The infarction was extensive in all cases and was located anteroseptally (with electrocardiographic QV1-V6) in 3 and posteroseptally in 1. The diameter of the perforation was 5 mm., 12 mm., and 12.5 mm. in the 3 autopsied cases. The first of these had become occluded by thrombus and had cardiac catheterizations been done under such circumstances, the septal perforation would not have been detected since there would have been no shunt. The high mortality rate on medical treatment, 87 per cent within 8 weeks, led the authors to view favorably the possibilities offered by surgical closure of the perforation.

Rogers
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