Phonocardiographic Features of Atrial Septal Defect

By ROBERT EISENBERG, M.D., AND HERBERT N. HULTGREN, M.D.

The heart sounds of 20 patients with uncomplicated atrial septal defect have been analyzed, and the deviations from a normal control group were noted. Following complete closure of the defect 11 patients have been restudied, and the changes produced in the heart sounds by closure of the defect are discussed. By comparison of pre-operative and postoperative heart sounds and electrocardiograms it has been possible to ascribe some abnormalities of the heart sounds to the presence of the left-to-right shunt, and some to the presence of delayed electrical depolarization of the right ventricle.

THE development of safe and effective surgical technics for the closure of atrial septal defects has increased the importance of accurate preoperative diagnosis. This lesion is common and, especially in younger patients, the physical signs may occasionally simulate those of a normal patient with a functional systolic murmur. Ancillary diagnostic methods that might obviate the need for cardiac catheterization would therefore be valuable. Atrial septal defect is characterized by a large left-to-right shunt with increased blood flow across the tricuspid and pulmonary valves and by delayed electrical depolarization of the right ventricle resulting usually in a pattern of incomplete right bundle-branch block in the electrocardiogram. Since both these features may produce distinctive changes in the heart sounds, phonocardiography may be an important technic in the correct diagnosis of this lesion. It is the purpose of this paper to describe the phonocardiographic features of 20 patients with uncomplicated atrial septal defect. Eleven of these patients were studied again after complete surgical closure of the defect. Since there was essentially no change in the electrocardiograms of these patients following surgery, this study has provided the unique opportunity of separate examination of the effects of the left-to-right shunt and of the delay of right ventricular depolarization upon the heart sounds in this lesion.

Material and Methods

The diagnosis of atrial septal defect of the secondum variety was established in each patient by cardiac catheterization and further confirmed by surgical closure of the defect in 14 patients. Eleven of these patients had postoperative studies of their heart sounds. Complete closure of the defect in 10 of the patients was established by postoperative cardiac catheterization or by obvious complete surgical closure under direct vision with use of cardiopulmonary bypass.

The patients ranged in age from 4 to 50 years (mean 25 years). Left-to-right shunts were present in all patients and ranged from 2.1 to 22.4 L. per minute per M.² (mean 6.2 L. per minute per M.²). The systolic pressure in the pulmonary artery ranged from 16 to 65 mm. Hg (mean 34 mm. Hg). The duration of the QRS complex of the electrocardiograms ranged from 0.07 to 0.12 second (mean 0.09 second). Fifteen patients had incomplete right bundle-branch block and 1 had complete right bundle-branch block. Eighteen of 20 had secondary r waves or qR complexes in the right precordial leads, usually V₃.

Heart sounds were recorded with the patient in the relaxed recumbent position during quiet breathing or suspended respiration with a Sanborn Twin-Beam Cardiette at a paper speed of 75 mm. per second. A dynamic microphone supplied with the above apparatus was employed. Simultaneous electrocardiograms or carotid pulse tracings were recorded for timing purposes. In many patients, phonocardiograms were also recorded during cardiac catheterization, an electrocardiogram and intracardiac pressure being simultaneously recorded for timing. Reported time intervals consist of the mean value of measurements made from 10 successive cardiac cycles. The intensity of murmurs was graded from 0 to IV.

In all patients in this study measurements were made from the onset of the QRS complex of the electrocardiogram to the onset of pressure rise in the right ventricle and the pulmonary artery, as well as to the dicrotic notch of the pulmonary
artery pressure tracing. These intervals usually correlated well with the simultaneously recorded heart sounds but in all instances the sound provided a more precise point of reference than the event recorded on the pressure tracing. For example, it was usually difficult to determine the exact instant of the ascent of pressure in the right ventricle due to the initial gradual ascent of the pressure curve—whereas it was not difficult to identify precisely the onset of the sound of closure of the mitral and tricuspid valves. A study of heart sounds may therefore provide a more accurate method of examining the events of the cardiac cycle than examination of the pressure pulse.

Heart sounds were identified in the following manner: Pulmonic valve closure follows aortic valve closure and coincides with the dicrotic notch of the pulmonary artery pressure tracing or with the crossover point of this tracing and the right ventricular pressure curve when there is no good dicrotic notch. Aortic valve closure precedes the dicrotic notch of the carotid artery tracing by approximately 0.01 to 0.03 second. This delay is due to pulse transmission from the left ventricle to the carotid artery, plus a very slight delay in the pulse recorder. Mitral valve closure precedes the onset of the carotid artery upstroke by approximately 0.04 to 0.06 second. This time interval is composed of the isometric contraction time of the left ventricle (about 0.04 second) and the pulse and instrumental delay described above. Tricuspid valve closure follows mitral valve closure and occurs simultaneously with the onset of pressure rise in the right ventricle. Systolic ejection sound of the pulmonary artery follows tricuspid closure and occurs simultaneously with the onset of pressure rise in the pulmonary artery.

Pressure events obtained by the cardiac catheter were corrected for the 0.01 second delay in transmission time through the catheter and recording apparatus.

Fifteen normal subjects with the same age distribution had phonocardiographic studies performed in a similar manner. All of these subjects had normal electrocardiograms and chest roentgenograms. None had any evidence of cardiac disease.

**RESULTS**

Splitting of the first heart sound was identified in all patients, the interval from mitral to tricuspid closure ranging from 0.02 to 0.05 second (mean 0.038 second). Splitting of the first sound was identified in all normal control subjects with a mitral-to-tricuspid closure interval ranging from 0.02 to 0.04 second (mean 0.028 second). This range of normal values compares well with the previous work of Leatham. An analysis of the individual measurements in both groups reveals a greater degree of splitting in the patients with atrial septal defect as suggested by the difference in mean values between the 2 groups (fig. 1). This difference in mean values is statistically significant at the 1 per cent confidence level according to Fisher’s modification of Student’s t test. Following surgical closure of the defect there was essentially no change in the interval from mitral to tricuspid closure in the patients.

An early systolic ejection sound was recorded at the pulmonic area in 18 patients. A similar sound, but less intense, could be identified in the records of 7 of the control subjects. In occasional patients the ejection sound was loud and was responsible for an apparent wide splitting of the first sound on auscultation at the lower sternal margin. Following surgery the ejection sound diminished in intensity but did not disappear.

A systolic murmur was recorded at the pulmonic area in all patients. This murmur exhibited the following characteristics: 1. Its onset followed the first heart sound by a short interval of 0.04 to 0.08 second and the initial vibrations of the murmur were usually preceded by a systolic ejection sound. 2. It was an ejection murmur with its peak intensity in early or midsystole. 3. It ended before the sound of pulmonic valve closure and usually before the sound of aortic valve closure (fig. 2).

The murmur was of variable intensity and in some instances was faint or exhibited a scratchy character that suggested an innocent systolic murmur or a cardiorespiratory murmur upon auscultation. There was a rough direct correlation between the intensity of the murmur and the relative magnitude of the left-to-right shunt, the louder murmurs being associated with shunt volumes of 6 L per minute per M. or more. Two patients with grade-III murmurs had smaller shunts but these patients had prominent enlargement
of the main pulmonary artery demonstrated by chest roentgenograms. All patients with a loud murmur and a large shunt had a similar degree of pulmonary artery enlargement.

Following closure of the defect, the murmur disappeared in 3 patients and was greatly reduced in intensity in 8. It has been suggested by Cossio et al.\(^3\) that the systolic murmur in atrial septal defect is due to the increased blood flow across the pulmonic valve. If this concept is true, increasing pulmonary blood flow following closure of the defect should reproduce the murmur that was present prior to surgery. Pulmonary flow was increased by vigorous exercise in 8 patients and phonocardiograms were recorded immediately after exercise. In 6 of these patients, the original systolic murmur reappeared or the intensity of the residual systolic murmur increased (fig. 3). The characteristics of the murmur induced by exercise were the same as those of the murmur present prior to surgery.

The second heart sound at the base is normally split, with pulmonic valve closure occurring 0.01 to 0.03 second after aortic valve closure in expiration. Inspiration increases this interval to values of from 0.03 to 0.05 second due largely to a prolongation of right ventricular systole\(^1\) and probably also to a shortening of left ventricular systole as demonstrated by Boyer and Chisholm.\(^4\) The interval from aortic closure to pulmonic closure during expiration in the 15 control subjects in this study ranged from 0.01 to 0.04 second (mean 0.028 second). The expected respiratory variation in this interval was noted in all of these subjects. In the patients this interval ranged from 0.04 to 0.09 second (mean 0.059 second) during expiration (fig. 4). On inspiration 12 of the 20 patients showed an increase of about 0.01 second in this interval. Following closure of the defect the interval from aortic to pulmonic valve closure returned to normal in 9 of the 11 patients studied. In 7 of these 9 patients the normal pattern of respiratory variation was also noted. In 1 patient, in whom complete closure of the defect had been demonstrated by postoperative cardiac catheterization, the interval remained unchanged at 0.06 second and showed no respiratory variation. One patient maintained a wide interval, but showed normal respiratory variation. The duration of the QRS complex of the electrocardiogram in these latter 2 patients was less than 0.10 second. At the time of the postoperative studies the heart rates were not exactly the same as in the preoperative studies but the difference in rates was small (mean 9 beats per minute) and this difference did not affect significantly the aortic to pulmonic valve closure interval.

An early diastolic murmur was present along the lower left sternal margin in 6 patients. This murmur began from 0.06 to 0.10 second after pulmonic valve closure and it was loudest at the time of early rapid diastolic filling of the right ventricle. At this time right ventricular diastolic pressure is lower than
later in diastole and a small pressure gradient can usually be demonstrated across the tricuspid valve. No relationship was noted between the size of the shunt and the presence of the murmur. The murmur disappeared following surgery in all cases.

Presystolic sounds were recorded at the cardiac apex in 8 patients. These sounds could not be detected by auscultation. These sounds were comparable to similar sounds recorded in normal subjects. No presystolic murmurs were observed. A faint apical third heart sound was recorded in 1 patient, which could not be identified by auscultation.

In 1 patient a sharp early diastolic sound was heard and recorded along the left sternal margin. It could not be recorded from the apex. It occurred 0.12 second after pulmonic valve closure and 0.06 second after the crossover point of the right ventricular and right atrial pressures. It coincided with the trough of a slight early diastolic dip in the right ventricular pressure tracing (fig. 5).

**Discussion**

Splitting of the first sound in atrial septal defect is not marked and cannot be differentiated by auscultation from physiologic split-
Fig. 4 Top. Each dot, mean of 10 measurements of the interval from aortic to pulmonic valve closure, measured in hundredths of a second, during the expiratory phase of respiration. The mean value for each group is recorded.

Fig. 5 Middle. Relationship of the heart sounds (LSB) to the pressure changes in the right ventricle (RV) and right atrium (RA). Pressure tracings were recorded during withdrawal of the catheter from the ventricle to the atrium, and then were superimposed. The heart sounds were recorded separately and then superimposed diagrammatically by selecting cycles of identical length. The pressures at the right of the illustration are referred to midchest. Time lines, 0.10 second.

Fig. 6 Bottom. Diagrammatic representation of the phonocardiographic features of atrial septal defect and the changes following closure of the defect. CAR, carotid artery tracing; A1, atrial sound; M, mitral valve closure; E, systolic ejection click; TFM, tricuspid flow murmur.
in patients with a variety of heart diseases, and, in another study, in 2 of 13 patients with no cardiovascular disease.

The systolic ejection sound produced by abrupt tension of the annulus of the pulmonary valve is a frequent finding in atrial septal defect because the transmission of the sound to the chest wall is facilitated by dilatation of the pulmonary artery. Since it may be present in normal subjects, its occurrence is of little diagnostic value unless it is loud or widely separated from the first heart sound.

The basal systolic murmur in atrial septal defect is undoubtedly produced at the pulmonic valve for the following reasons: 1. It is usually maximal at the pulmonic area. 2. It is an ejection murmur frequently initiated by an ejection sound. 3. Increased flow across the valve and dilatation of the pulmonary artery occur in atrial septal defects and could produce the murmur and facilitate its transmission to the chest wall. 4. At surgery a palpable thrill is present over the pulmonary artery, which disappears when the defect is closed. 5. Phonocatheter studies have demonstrated that the murmur is loudest at the pulmonic valve and faint or absent in the right ventricle or right atrium. The data in the present study suggest that 2 factors are involved in the production of the murmur: increased flow across the valve, and anatomic changes (dilatation) in the pulmonary artery. That the increased flow was not the only factor in the production of the murmur was suggested by the presence of residual systolic murmurs in 8 patients whose defects had been closed. Pulmonary blood flow was increased by exercise in 8 patients postoperatively and in 6 the systolic murmurs appeared or, if present, became louder. The phonocardiographic characteristics and the location of these murmurs induced by exercise were similar to those observed prior to surgery.

As other investigators have noted, respiratory variation in the degree of splitting of the second sound was present in many of the patients, although the magnitude of the variation was less than in the normal control subjects. That this is not apparent on auscultation is due to the wide separation of the 2 components of the second sound in atrial septal defects, which makes small changes in the degree of splitting difficult to discern. The return of the degree of splitting to the normal range after closure of the defect indicates that the principal factor in the prolongation of right ventricular systole is the increased stroke volume and not the delay in depolarization of the right ventricle. Similar findings have been noted by others.

Early diastolic murmurs at the lower sternal margin probably are due to increased flow across the tricuspid valve at the time of rapid right ventricular filling in early diastole. No increase in the loudness of the murmur was noted during inspiration as is frequently found in tricuspid stenosis or insufficiency. This is probably due to the fact that the large flow across the valve in atrial septal defect cannot be increased proportionately as much by inspiration as can the smaller flow across the valve in tricuspid disease.

A faint third heart sound recorded at the apex of 1 patient probably arose in the left ventricle and is similar to third heart sounds present in a great proportion of children and young adults.

An early diastolic sound audible along the left sternal border was recorded in 1 patient (figs. 3 and 5). It probably represents a third heart sound arising in the right ventricle for the following reasons: 1. The sound occurred too late to be produced by pulmonic valve closure or by an opening snap of the tricuspid valve. It occurred at the time of rapid right ventricular filling in early diastole. 2. The sound was loudest at the left sternal border over the right ventricle, while third heart sounds arising in the left ventricle are loudest at the apex and are only rarely loud along the left sternal border. 3. The sound occurred slightly later than a third heart sound arising in the left ventricle would be expected. Leatham and Gray recorded early diastolic sounds in 10 patients. In 8 of these the sound occurred 0.03 to 0.08 second after pulmonic valve closure, and it was suggested that this sound was an opening snap of the tricuspid
valve. In the 2 other cases, the sounds occurred 0.11 and 0.12 second after pulmonic valve closure and appeared to be similar to the sound discussed above. (In addition, we have recorded a sound with similar timing in 1 other patient with evidence of a left-to-right shunt at the atrial level demonstrated by a subsequent cardiac catheterization.)

The rare occurrence of third sounds in the right ventricle is pertinent to the general origin of the third heart sound. Surely the hemodynamic events accompanying left ventricular third sounds must occur as frequently in the right ventricle as in the left. The rapid filling of the right ventricle in atrial septal defect and tricuspid insufficiency, however, is rarely accompanied by third sounds. It is possible that the tricuspid valve and its chordae are thinner and more delicate and therefore less capable of producing a third sound when tensed than the thicker leaflets and chordae of the mitral valve.

While most of the above features of atrial septal defect can be elicited by careful auscultation, phonocardiography is of value in determining the degree of splitting of the first and second heart sounds, especially when one component is faint, as well as identifying the presence of systolic ejection sounds and the more rarely encountered early diastolic sounds. Following surgery auscultation will usually reveal the change in intensity or disappearance of the systolic murmur and the systolic ejection sound, the disappearance of the tricuspid flow murmur and the return to a normal degree of splitting of the second sound. Phonocardiography should make the latter determination more precise. Figure 6 represents the characteristic sounds and murmur and the postoperative changes diagrammatically.

**Summary**

Phonocardiographic studies have been made of 20 patients with uncomplicated atrial septal defect and these studies have been repeated in 11 patients after complete surgical closure of the defect.

The following characteristic features were observed: Splitting of the first heart sound due to the delayed onset of right ventricular contraction. Systolic ejection murmur at the pulmonic area initiated by a systolic ejection sound. Both are due to increased flow and dilatation of the pulmonary artery. Wide splitting of the second sound due to prolongation of right ventricular systole. Early diastolic murmur along the lower left sternal border probably due to increased flow across the tricuspid valve. Rare presence of a third heart sound arising in the right ventricle.

Following complete closure of the defect, the following observations were made: Persistence of the split first sound. Disappearance or decrease in the intensity of the systolic murmur and the systolic ejection sound. Decrease in the degree of splitting of the second sound. Disappearance of the tricuspid flow murmur and the right ventricular third heart sound.

**Acknowledgment**

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**Summario in Interlingua**

Studios phonocardiographic esseva effectuata in 20 patientes con non-complicate defectos atrio-septal. In 11 patientes iste studios esseva repetite post le complete clausion chirurgic del defecto.

Le sequente observationes caracteristic esseva facite: Fission del primo sono cardiac in consequentia del retardate declaration del contraction dextero-ventricular. Murmure de ejection systolic in le area pulmonic initiate per un sono de ejection systolic. Ambes es causate per augmentos del fluxo e del dilataction del arteria pulmonar. Fissura large del secunde sono in consequentia del prolongation del systole dextero-ventricular. Precoce murmure diastolic al longo del margini sternal infero-sinistre, probablemente in consequentia del augmento del fluxo a transverso le valvula tricuspid. In rar casos, le presentia de un tertiae sono cardiac que prende su origine in le ventriculo dextere.

Post le complete clausion del defecto, le sequente observationes esseva facite: Persis-
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tentia del findite prime sono. Disparition o reduction del intensitate del murmur systolic e del sono de ejection systolic. Reduction del grado de fission in le secunde sono. Disparition del murmur del fluxo tricuspid e del tertia sono dextero-ventricular.

REFERENCES


In the meantime this I know and declare to all men, that sometimes the blood passes in less, sometimes in more abundant quantitie, and the circuit of the blood is perform'd sometimes sooner, sometimes slower, according to the age, temperature, external and internal cause, accidents natural or innatural, sleep, rest, food, exercise, passions of the mind, and the like.—William Harvey. De Motu Cordis, 1628.
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