

## **Intracardiac Phonocardiography: A Valuable Diagnostic Technique in Congenital and Acquired Heart Disease**

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Intracardiac phonocardiography represents a further attempt, following esophageal,<sup>1</sup> tracheal,<sup>2</sup> and epicardial phonocardiography,<sup>3</sup> to overcome the many limitations of the transmission of heart sounds and murmurs from their site of origin to the chest surface.

In recent years, many techniques have been described for direct intracardiac sound recording. In 1954, Yamakawa and associates<sup>4</sup> reported the first intracardiac phonocardiograms recorded in the intact animal by means of a condenser microphone soldered to the leading tip of a catheter. In the same year, and more extensively in 1957, Soulié and associates<sup>5,6</sup> recorded intracardiac sounds with the aid of an intracardiac micromanometer. Lewis and associates,<sup>7</sup> in 1957, reported the first group of patients with congenital and acquired heart disease who had been studied with an intracardiac microphone of barium titanate which was derived from the acoustic technology developed for undersea warfare. Luisada and Liu,<sup>8</sup> in 1957, recorded intracardiac sounds by using glucose solution within a standard catheter as the carrier of sounds and a pressure transducer of high-frequency response situated outside the body. Finally, in 1958, Moscovitz and associates<sup>9</sup> used a piezoelectric microphone sealed in the tip of a double-lumen catheter for the purpose of recording and correlating simultaneous mechanical and acoustic events of the cardiac cycle in animals and in patients.

Among these various techniques, that described by Lewis and associates<sup>7</sup> has been proved<sup>7,10</sup> to have, with the advantage of easy performance, the property of producing recordings of high quality. Moreover, it has been suggested<sup>11,12</sup> that this technique, by providing precise localization of the source of heart sounds and murmurs, would not only increase knowledge of the site and mechanism of production of cardiovascular sound, but would also be an important aid in the diagnosis of congenital and acquired heart disease.

It has been the purpose of this investigation to emphasize the clinical value of this technique by studying the intracardiac phonocardiograms in 160 patients

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with congenital and acquired heart disease, and by correlating the intracardiac phonocardiographic features with the clinical and hemodynamic data, and with the angiogram, the results of surgery, or postmortem findings when available.

#### MATERIAL AND METHODS

The 160 patients included in this study ranged in age from 3½ to 59 years. One hundred of these patients were fully investigated in the Cardiovascular Unit of the Toronto General Hospital and the remaining 60 in the Cardiovascular Center of the University of Padua.\*

Intracardiac sounds in all patients were obtained by means of the phonocatheter described by Wallace, Brown, Lewis and Dietz.<sup>13</sup>

The microphone is incorporated in the leading tip of the catheter. It consists of a hollow cylinder of activated barium titanate, which has the properties of a piezoelectric crystal in that it is able to convert the sonic vibrations into electrical current. It is ½ inch in length, has an outer diameter of 0.038 inch, and is soldered to a coaxial cable of the same diameter and approximately 5 feet long. Both the barium titanate element and the coaxial cable are protected with a non-irritant plastic coating, which prevents direct contact of the sound transducer with the blood and cardiac walls (a possible source of artefact) and allows treatment of the catheter with antiseptic solutions for sterilization purposes. The barium titanate transducer cannot be autoclaved because it is inactivated by high temperatures.

The voltage output of the barium titanate microphone is very small and requires the use of a specially designed preamplifier before being fed into a recording apparatus for routine chest phonocardiography. The preamplifier used for this investigation is a three-stage transistorized amplifier, which has an essentially flat response from 30 to 10,000 c.p.s.†

A two-channel photographic recorder (Sanborn Twin-Beam) was used for most of this work. This apparatus provides logarithmic and stethoscopic curves of amplification. A three-channel (Moedek and Shörner, Multicard) or an eight-channel (Electronics for Medicine, DR\$ Research Recorder) photographic recorder was also used. Intracardiac phonocardiograms were recorded routinely with an electrocardiogram as a reference tracing and with a simultaneous chest phonocardiogram for comparison studies.

Recordings on tape were also obtained by using the Sanborn-Ampex heart sound tape recorder.

In all instances intracardiac sound recordings were combined with routine right heart catheterization, during which pressure measurements and blood samples for oxygen studies were obtained and the cardiac output estimated by the direct Fick principle. Also, in the majority of cases, dye dilution studies were carried out by injecting T-1824 or cardiolgreen at various sites and by sampling at the right ear by means of an ear oximeter.<sup>14</sup>

The sound catheter was introduced into the cavities of the heart after the routine heart catheterization had been completed. The phonocatheter could be clearly seen by means of fluoroscopy because of its metallic components (Fig. 1). Localization of the sound transducer within the heart chambers and great vessels was made possible by reference marks placed on the fluoroscopic screen during routine heart catheterization, for location of the valve orifices.

In addition to right heart catheterization, 24 patients with chronic rheumatic heart disease, involving the mitral or the aortic valve or both, had left heart catheterization via the transthoracic route. Another 32 had a biplane selective angiogram recorded at a speed of 2 to 12 frames per second. Finally, 68 had surgical correction of their cardiac lesions,‡ and in 8 there was postmortem examination.

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†Phonocatheters and preamplifiers used in this investigation were built and made available to the author by the U. S. Naval Air Development Center of Johnsville, Pa.

‡Dr. W. G. Bigelow, Dr. R. O. Helmbecker, Dr. J. A. Key (University of Toronto and Toronto General Hospital), and Prof. G. Ceccarelli (Clinica Chirurgica, University of Padua) carried out the surgery in these patients.

## RESULTS

The diagnoses in the 160 cases included in this study are given in Table I. Table II presents the cases in which selective angiography, surgery, or postmortem examination were carried out. Following is a summary of the intracardiac phonocardiographic findings in each diagnostic group.

*Normal Hemodynamic Findings.*—The 20 patients, ranging from 6 to 45 years of age, who had normal hemodynamic findings in the right chambers of the heart and pulmonary artery showed the following intracardiac phonocardiographic features (Fig. 2): Within the *pulmonary artery* the first heart sound was very rudimentary or absent. The second sound was loud and single, representing the pulmonary valve closure. A soft systolic ejection murmur was recorded in all patients. This murmur was present in simultaneous external phonocardiograms in 9 patients only (Fig. 3). Within the *right ventricle* the first heart sound was



Fig. 1.—The sound catheter as it appears on the fluoroscopic screen.

recorded as a single group of vibrations occurring 0.06 to 0.08 second after the onset of the QRS complex, which in normal subjects is the time of the tricuspid closure. The second sound was rudimentary or absent (Fig. 2). Within the *right atrium* both the first and the second heart sounds were present in 10 out of 20 patients. They were of reduced intensity compared with those recorded in the ventricle and pulmonary artery. In only 2 patients was a fourth heart sound of moderate intensity observed, occurring 0.05 second after the onset of the P wave. In 9 patients no sounds at all were recorded.

In 3 subjects (4, 9, and 22 years old, respectively) with normal hemodynamic findings in both sides of the heart the sound catheter crossed a patent foramen

ovale and entered the *left atrium* and *left ventricle*. Within the latter chamber (Fig. 4) the first sound was recorded as a single group of vibrations which were much louder than the first sound recorded in the right ventricle. These vibrations occurred 0.04 to 0.06 second after the onset of the QRS complex (which is the time of the mitral valve closure) and 0.02 second before the first sound recorded within the right ventricle. The second sound in the left ventricle was recorded as a rudimentary group of vibrations. No sounds were recorded within the left atrium in these 3 normal subjects.

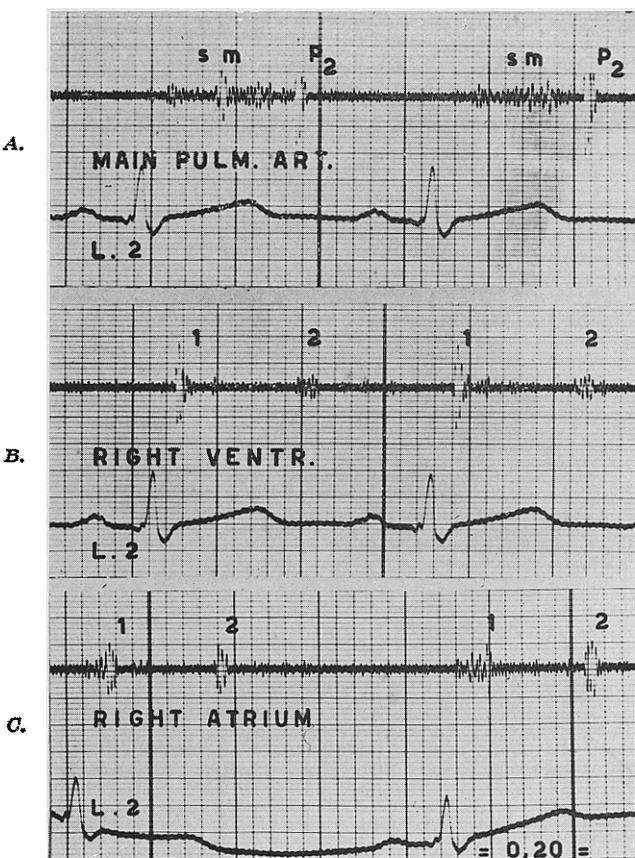


Fig. 2.—Intracardiac phonocardiogram in a subject with normal hemodynamic findings. A, Pulmonary artery. B, Right ventricle. C, Right atrium.

*Ventricular Septal Defect.*—Of the 21 patients with ventricular septal defect (V.S.D.), 16 had uncomplicated ventricular septal defect and 5 had associated pulmonary hypertension.

In all 16 patients of the former group a very loud pansystolic murmur was recorded within the right ventricle (Fig. 5). In addition, in 9 patients an early diastolic murmur of low intensity was observed. Both these murmurs disappeared abruptly when the sound transducer was moved out of the right ventricle.

In the pulmonary artery an ejection murmur of varying intensity, ending before or with a loud pulmonary closure, was observed (Fig. 5). No murmurs were recorded within the right atrium.

TABLE I. CLINICAL DIAGNOSIS IN 160 CASES STUDIED BY INTRACARDIAC PHONOCARDIOGRAPHY

DIAGNOSIS	NUMBER OF CASES
Congenital Heart Disease	97
Ventricular septal defect	21
Atrial septal defect	29
Lutembacher's syndrome	2
Persistent atrioventricular canal	3
Tetralogy of Fallot	15
Pentalogy of Fallot	1
Tricuspid atresia	2
Isolated pulmonary valvular stenosis	4
Idiopathic pulmonary artery dilatation	1
Patent ductus arteriosus	15
Common truncus	1
Isolated anomalous pulmonary veins	3
Chronic Rheumatic Heart Disease	35
Mitral stenosis	12
Mitral stenosis and tricuspid insufficiency	6
Mitral stenosis and insufficiency	8
Aortic stenosis and insufficiency	4
Mitral and aortic valvular disease	5
Primary Pulmonary Hypertension	1
Chronic Cor Pulmonale	2
Idiopathic Cardiac Enlargement	4
Intrathoracic A-V Fistula	1
Normal Catheterization Findings	20
Total	160

TABLE II. CASES IN WHICH SELECTIVE BIPLANE ANGIOCARDIOGRAPHY, SURGERY, OR POSTMORTEM EXAMINATION WERE CARRIED OUT

DIAGNOSIS	NUMBER OF CASES		
	BIPLANE ANGIO-CARDIOGRAPHY	SURGERY	POSTMORTEM
Ventricular septal defect	6	7	-
Atrial septal defect	2	19	2
Lutembacher's syndrome	-	2	-
Persistent A-V canal	1	-	-
Tetralogy of Fallot	10	5	2
Pentalogy	1	-	-
Tricuspid atresia	2	-	-
Isolated pulmonary stenosis	4	2	1
Patent ductus arteriosus	4	14	-
Isolated anomalous pulmonary veins	2	-	-
Mitral stenosis	-	10	-
Mitral stenosis and tricuspid insufficiency	-	4	-
Mitral stenosis and insufficiency	-	3	1
Aortic stenosis and insufficiency	-	2	1
Aortic and mitral valve disease	-	-	1
Total	32	68	8

In the 5 patients with V.S.D. and associated pulmonary hypertension (80 to 120 mm. Hg systolic) and bidirectional shunt, a systolic murmur (pansystolic in 2 patients and with mid- or late systolic accentuation in the remaining 3) was recorded within the right ventricle, together with an early diastolic murmur, loudest in the outflow tract. In the pulmonary artery a soft ejection murmur with a very loud pulmonary closure was observed.

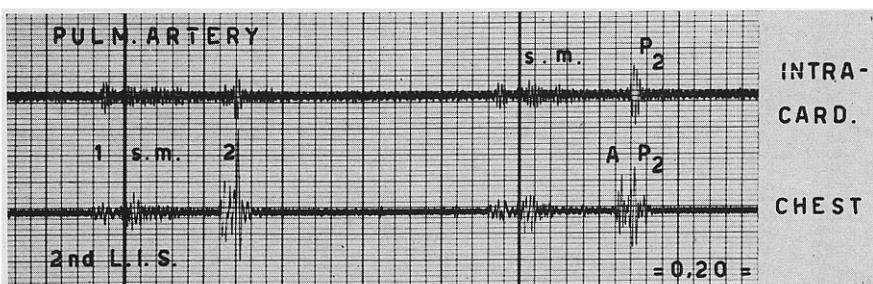


Fig. 3.—Simultaneous intracardiac and external phonocardiograms from within the pulmonary artery and the second left intercostal space in a normal subject. A soft mid-systolic murmur is present in both tracings. Taking the intracardiac phonocardiogram as a reference, the pulmonary component of a split second sound on the external phonocardiogram can be identified.

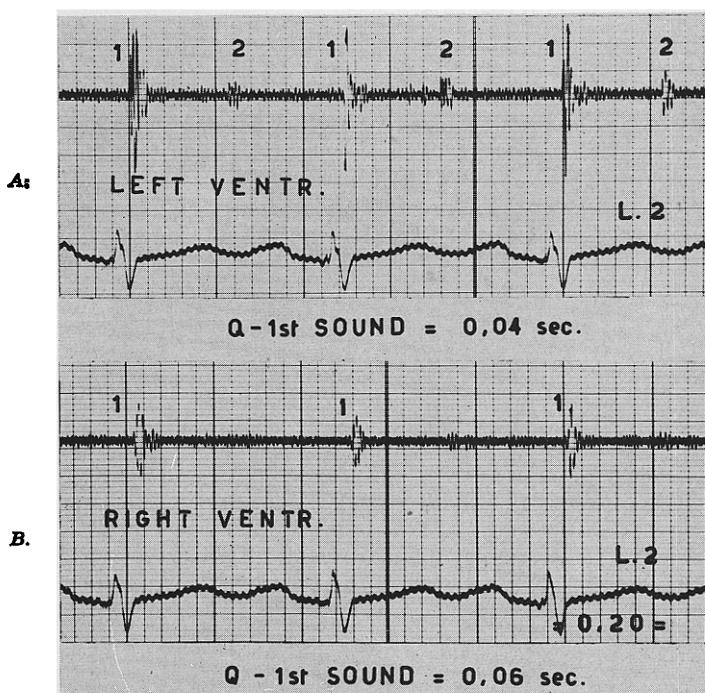


Fig. 4.—Intracardiac phonocardiogram in a normal subject. A, From within the left ventricle; B, from within the right ventricle. In the left ventricle the first heart sound is much louder and it occurs 0.02 sec. earlier than in the right ventricle. In the left ventricle, only the mitral closure is recorded, and in the right ventricle, only the tricuspid closure.

A few problem cases of V.S.D. included in this series are worthy of mention. In 2 patients, despite the finding of a significant rise in oxygen in the right atrium with no further rise in the right ventricle, suggesting an atrial septal defect rather than a ventricular septal defect, a loud pansystolic murmur recorded within the latter chamber led to the correct diagnosis of V.S.D., and this was proved later at surgery. In another patient with V.S.D. a significant rise in oxygen was detected only within the pulmonary artery, and despite the clinical picture, the catheterization findings suggested a patent ductus arteriosus. Again, the intracardiac phonocardiogram revealed a loud pansystolic murmur within the right ventricle and enabled the correct diagnosis to be made. One patient in whom the diagnosis at right heart catheterization was that of an isolated infundibular pulmonary stenosis (no shunt was detected by oxygen and dye dilution studies) had a rather large third infundibular chamber, which was demonstrated by selective angiography. While the sound catheter was lying within this low-

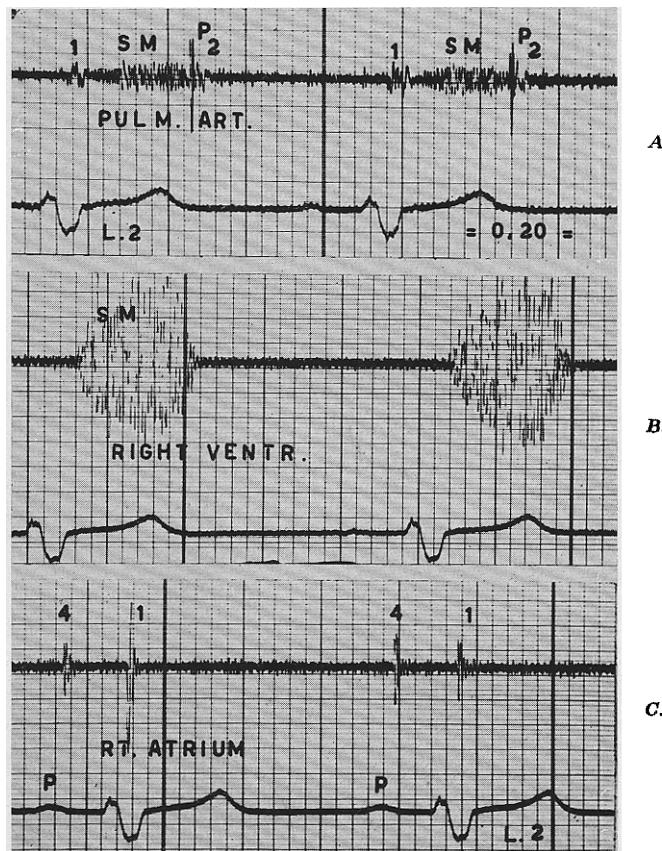
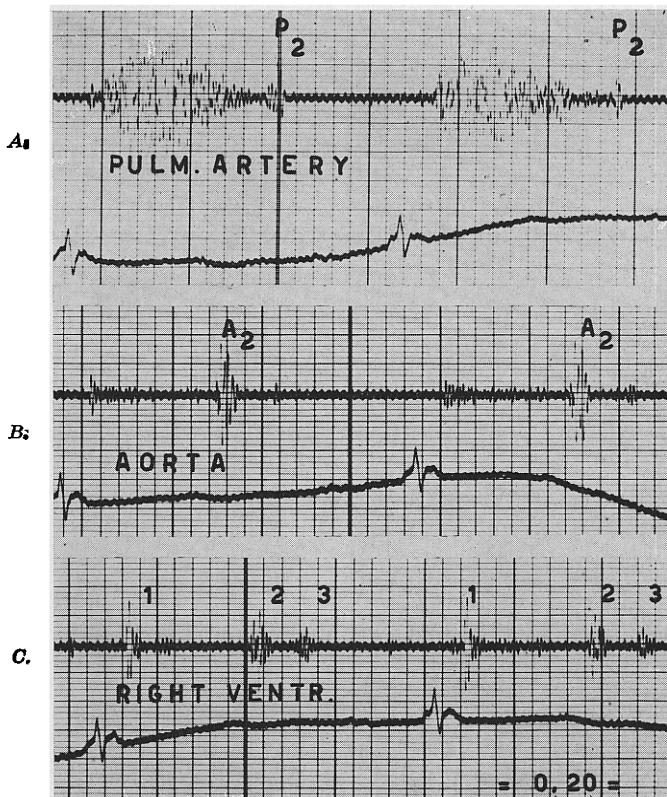


Fig. 5.—Uncomplicated ventricular septal defect. A loud pansystolic murmur (B) which is the equivalent of that heard over the mid-precordium is localized within the right ventricle. C, No murmurs are recorded within the right atrium, and within the pulmonary artery (A) a mid-systolic murmur and a loud pulmonary closure are present.

pressure cavity, a very loud "pansystolic" murmur was recorded and the diagnosis of a V.S.D. facing the infundibular chamber was suggested. Surgery was performed to relieve pulmonary stenosis, and a V.S.D. less than 1 cm. in diameter was found and successfully closed.



**Fig. 6.—**Tetralogy of Fallot. *A*, A loud systolic ejection murmur is recorded within the pulmonary artery, with a soft, delayed pulmonary closure. *B*, A very soft systolic murmur is present within the aorta. *C*, No murmurs are recorded within the right ventricle, suggesting that the ventricular septal defect in this case is silent.

**Tetralogy of Fallot.**—In the 15 patients who had tetralogy of Fallot with infundibular stenosis the intracardiac phonocardiogram revealed no murmurs within the right ventricle (Fig. 6), in which a loud first sound and a relatively loud second sound, the latter corresponding in time with the aortic closure, were recorded. Within the aorta, which was entered in 6 patients, a very soft ejection murmur was observed, followed by a loud aortic closure (Fig. 6). Within the pulmonary artery and its main branches a very loud, long, diamond-shaped murmur was recorded, which "overflowed" the aortic closure and ended with a soft, delayed pulmonary second sound. This murmur disappeared gradually as the catheter was being withdrawn across the infundibulum into the right ventricle (Fig. 9,4).

In these 15 patients the pulmonary closure was *always* recorded on the intracardiac phonocardiogram, although it was not audible or recordable from the chest in 11 patients. During the pullback of the phonocatheter from the pulmonary artery into the right ventricle the pulmonary sound disappeared long before the systolic murmur. Simultaneous recordings by phonocatheter from the pul-

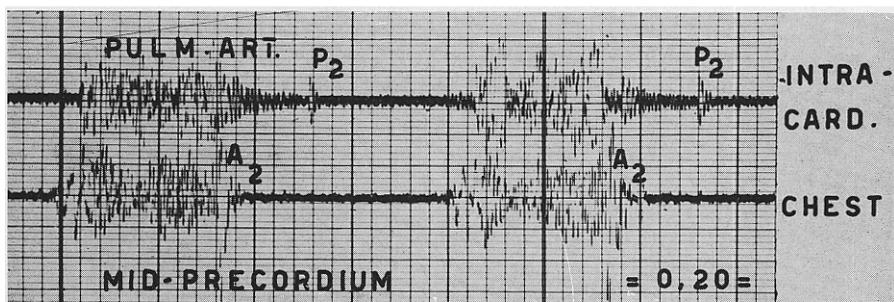


Fig. 7.—Tetralogy of Fallot. Simultaneous recordings from within the pulmonary artery by the sound catheter and externally from the mid-precordium where a loud aortic closure is recorded. The systolic murmur within the pulmonary artery overflows the aortic closure and there is a 0.16 sec. interval between the aortic and the pulmonary closure indicative of severe pulmonary stenosis.

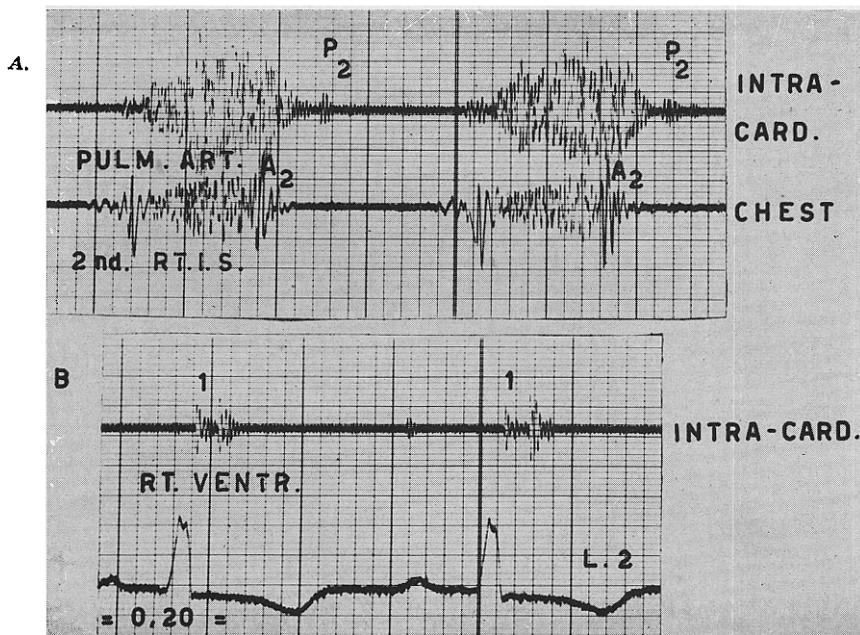


Fig. 8.—Isolated valvular pulmonary stenosis. A, A very loud ejection murmur with a soft, delayed pulmonary closure is recorded within the pulmonary artery, whereas no murmurs are present within the right ventricle. B, A, Simultaneous recordings from within the pulmonary artery and externally from the second right intercostal space show how the murmur overflows the aortic closure and how the pulmonary closure is delayed 0.12 sec., as compared with the aortic closure which indicates severe pulmonary stenosis.

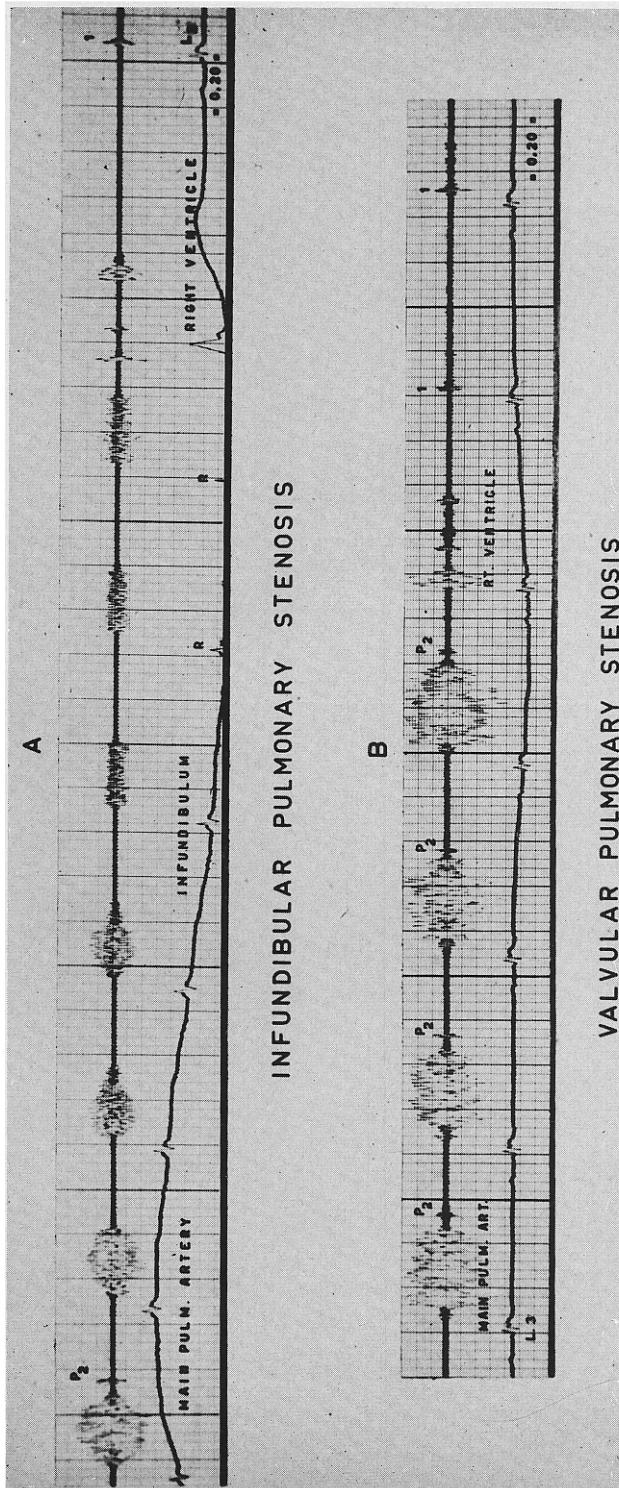


Fig. 9.—*A*, Infundibular pulmonary stenosis. The continuous tracing recorded during withdrawal of the sound catheter from within the main pulmonary artery, through the infundibulum into the right ventricle, shows that the systolic murmur disappears with a gradual reduction of intensity. The pulmonary second sound disappears long before the murmur. *B*, Valvular pulmonary stenosis. The loud systolic murmur recorded in the pulmonary artery disappears suddenly during withdrawal of the catheter across the pulmonary valves into the right ventricle, and the pulmonary closure disappears with it.

monary artery and by chest microphone from the mid-precordium (where the aortic closure is well recorded) revealed a delay in the pulmonary closure up to 0.17 second as compared with the aortic. In all cases there was a good correlation between the aorto-pulmonary closure interval and the pressure gradient across the infundibular stenosis. Intervals of 0.12 second or over were always accompanied by pressure gradients of 100 mm. Hg or more (Fig. 7).

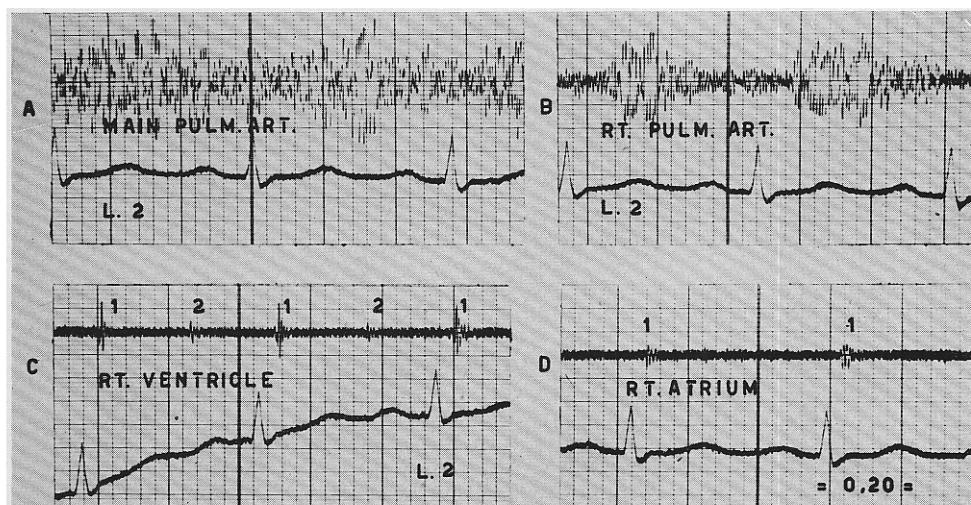


Fig. 10.—Patent ductus arteriosus. *A* and *B*, A continuous murmur is localized within the pulmonary artery. No murmurs are recorded within the right ventricle (*C*) and right atrium (*D*).

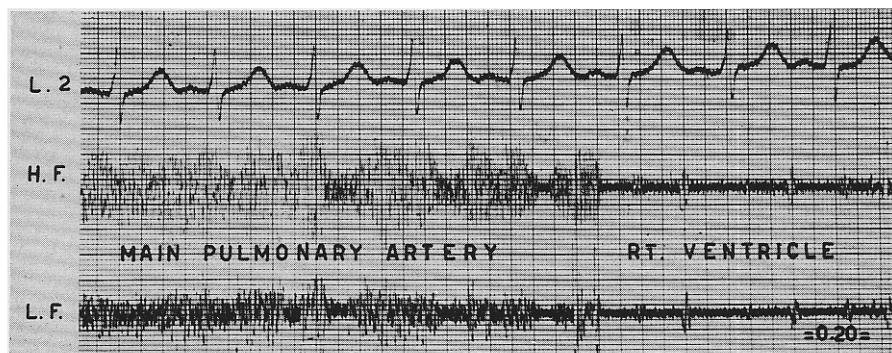
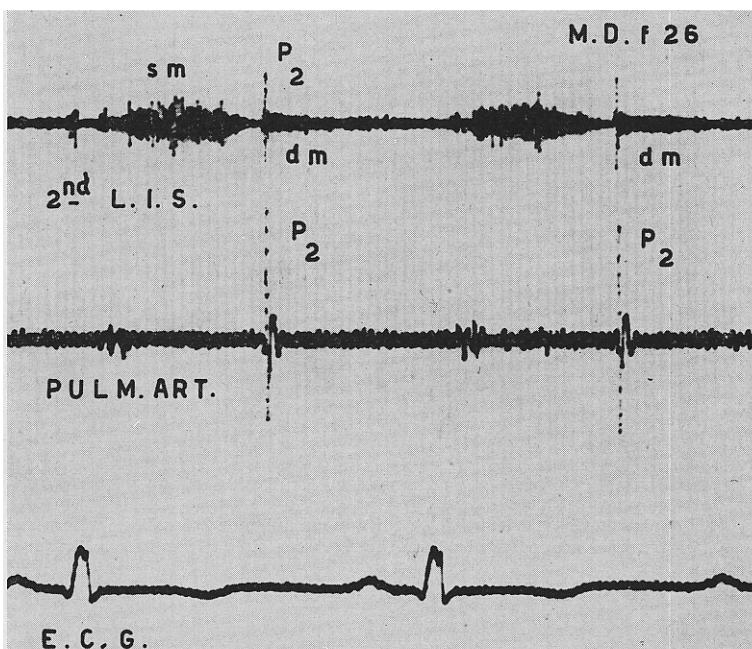


Fig. 11.—Patent ductus arteriosus. The continuous machinery murmur recorded within the pulmonary artery disappears suddenly once the sound catheter is withdrawn across the pulmonary valves into the right ventricle. (H.F., 300 to 600 c.p.s.; L.F., 30 to 300 c.p.s.)

*Isolated Pulmonary Valvular Stenosis.*—Isolated pulmonary valvular stenosis was encountered in 4 patients. The diagnosis was confirmed by selective angiography in all patients and at surgery in 2 patients. Within the pulmonary artery in all patients a very loud, diamond-shaped murmur and a soft, delayed

pulmonary closure were recorded (Fig. 8). The murmur disappeared abruptly when the sound catheter was withdrawn from the pulmonary artery into the right ventricle (Fig. 9,B), unlike the gradual disappearance observed in infundibular stenosis (Fig. 9,A). There was good correlation between the delay in pulmonary closure and the pressure gradient across the pulmonary valves as in cases of infundibular pulmonary stenosis (Fig. 8). No murmurs were recorded within the right ventricle and right atrium.



**Fig. 12.**—Simultaneous recordings from the second left intercostal space and from within the pulmonary artery in a subject clinically suspected of having patent ductus arteriosus. Whereas on the external recording there is a systolic and an early diastolic murmur, no murmurs are recorded within the pulmonary artery, which finding excludes the possibility of patent ductus arteriosus.

**Patent Ductus Arteriosus.**—Of the 15 patients with this condition, 12 had an uncomplicated patent ductus arteriosus (P.D.A.) with a typical continuous machinery type of murmur in the second and third left intercostal spaces. One patient had severe pulmonary hypertension, bidirectional shunt, a mid-systolic murmur at the base on auscultation, and a very loud second sound followed by an early, blowing diastolic murmur along the left sternal border in the third and fourth left intercostal spaces. The remaining 2 patients had normal pulmonary pressures, no significant rise in oxygen at any level, and a soft, systolic murmur in the first and second left intercostal spaces as the only auscultatory sign.

The intracardiac phonocardiogram in all 15 patients showed a loud, continuous, machinery type of murmur within the pulmonary artery and its main branches (Fig. 10). This murmur was typical even in those 2 patients in whom only a soft systolic murmur was audible over the chest.

The murmur of P.D.A. was present only within the pulmonary artery. At times it was loudest at the bifurcation or in the left pulmonary artery. It disappeared suddenly when the sound transducer was withdrawn from the pulmonary artery into the right ventricle (Fig. 11). In this chamber and in the right atrium no unusual findings were observed (Fig. 10.).

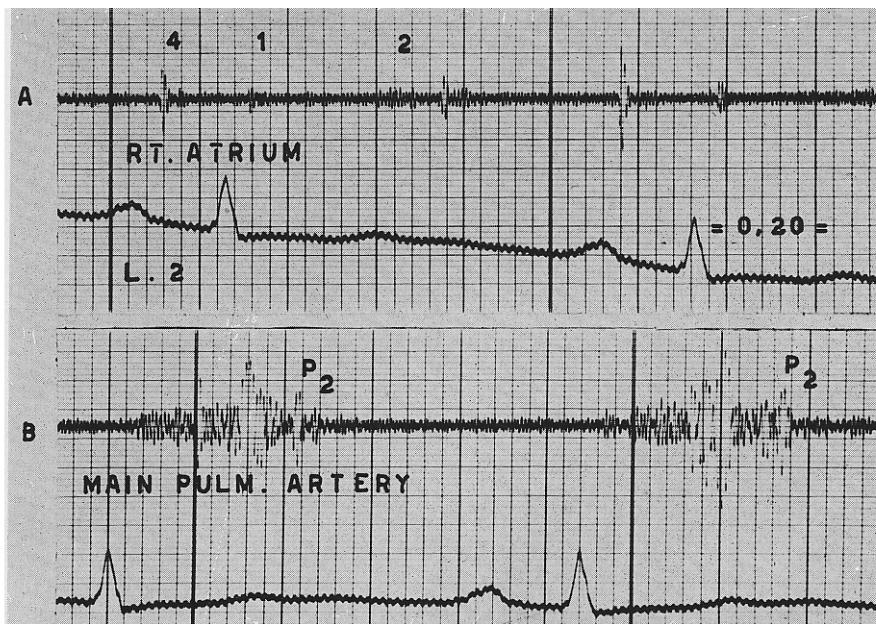


Fig. 13.—Uncomplicated atrial septal defect. A, No murmurs are recorded within the right atrium. B, A mid-systolic murmur, louder than in normal subjects, is present within the pulmonary artery.

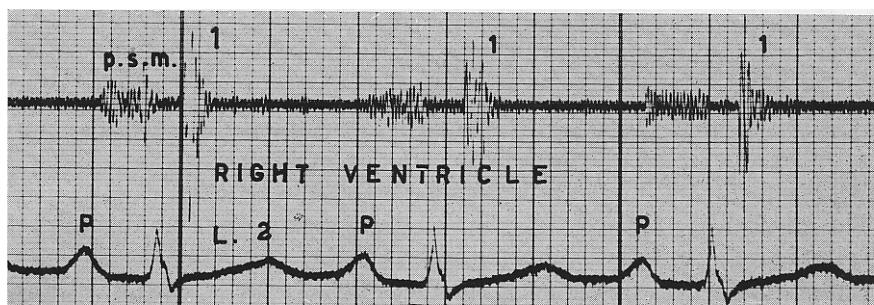


Fig. 14.—Whereas in cases of atrial septal defect with relatively small left-to-right shunts and pulmonary flow less than three times the systemic flow, no murmurs are usually encountered in the right ventricle, in cases with greater left-to-right shunts a mid-diastolic or presystolic murmur is commonly present within the inflow tract of the right ventricle. The figure demonstrates such a case. The diastolic murmurs disappear after surgical closure of the septal defect, suggesting that they are due to the increased flow across the tricuspid valve. In the majority of cases of uncomplicated atrial septal defect a very loud tricuspid closure is recorded within the right ventricle.

All patients with P.D.A., except the one with severe pulmonary hypertension (whose diagnosis was confirmed by means of a retrograde aortogram), had surgical closure of their ductus. The 2 patients with uncomplicated P.D.A., who had only a basal systolic murmur on auscultation, but a typical machinery type of murmur within the pulmonary artery, revealed at surgery a rather small patent ductus. It is noteworthy that in these two cases intracardiac phonocardiography was the only diagnostic technique which led to the correct diagnosis.

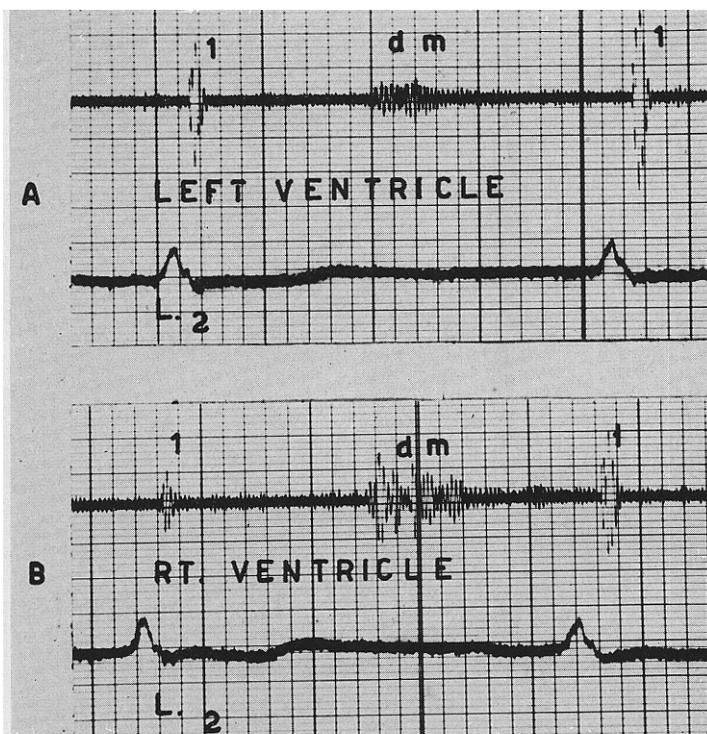


Fig. 15.—Lutembacher's syndrome with atrial fibrillation. A, A mid-diastolic murmur within the inflow tract of the left ventricle in this condition is diagnostic of mitral obstruction despite the absence of any significant pressure gradient across the mitral valve. B, A mid-diastolic murmur is recorded also within the right ventricle because of the increased flow across the tricuspid valve. On auscultation these two murmurs could not be separated.

The intracardiac phonocardiogram has been most helpful not only in establishing but in ruling out as well the diagnosis of P.D.A. in the presence of auscultatory signs or catheterization findings simulating such a condition. In fact, P.D.A. could be excluded in one case of ventricular septal defect with aortic insufficiency, in two cases of ventricular septal defect with pulmonary insufficiency, in one case of intrathoracic arteriovenous fistula, and in one case of aortic stenosis and insufficiency (Fig. 12). Intracardiac phonocardiograms in these cases showed that the ductus-like auscultatory features were not originating from the pulmonary artery.

*Atrial Septal Defect.*—Intracardiac phonocardiography in 29 patients with atrial septal defect (A.S.D.) has been most helpful in excluding associated congenital malformations (e.g., ventricular septal defect) and localizing the site of origin of the many auscultatory features in this condition.

In 12 patients with uncomplicated A.S.D. who had a pulmonary blood flow less than three times the systemic, no murmurs or unusual findings were recorded within the right atrium and right ventricle. In the pulmonary artery (Fig. 13) an ejection murmur of varying intensity was recorded along with a loud pulmonary closure. Both the murmur and the pulmonary sound increased in intensity during inspiration. The intensity of the murmur was considerably decreased after surgical closure of the defect in 5 patients who were catheterized postoperatively.

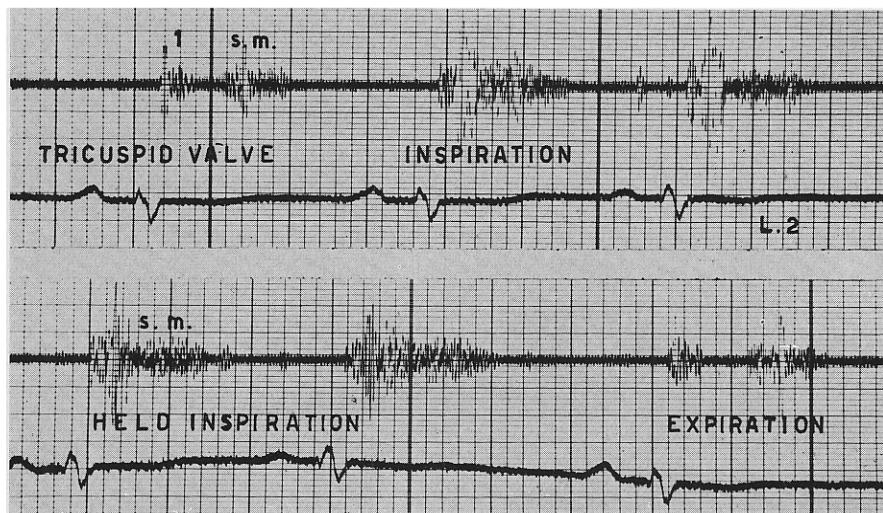


Fig. 16.—Pure mitral stenosis with apical systolic murmur. This murmur is localized by the sound catheter at the tricuspid valve. It becomes louder during inspiration.

In the 17 patients with A.S.D. who had a pulmonary blood flow over three times the systemic, a mid-diastolic or a presystolic murmur was recorded within the inflow tract of the right ventricle (Fig. 14), suggesting relative tricuspid stenosis secondary to the increased flow across this valve. This murmur disappeared in the 10 patients who underwent surgical closure of the septal defect and had postoperative catheterization.

In no cases of uncomplicated A.S.D. were murmurs recorded within the right atrium or left atrium or on pullback of the sound catheter across the septal defect.

*Other Diagnostic Groups.*—In one of the 2 patients with surgically proved Lutembacher's syndrome, in whom the sound catheter entered both sides of the heart, a mid-diastolic murmur was recorded in the inflow tract of both the right and left ventricles (Fig. 15). The latter finding suggested mitral stenosis, although no significant pressure gradient was recorded across the mitral valve. In 2 patients

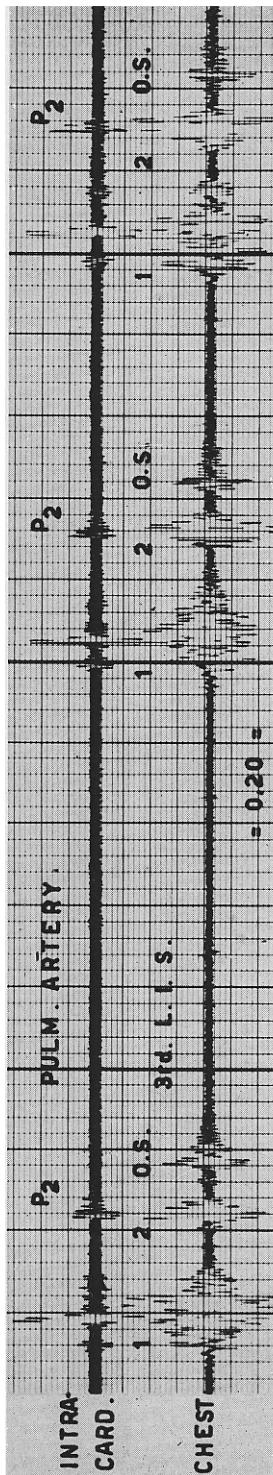
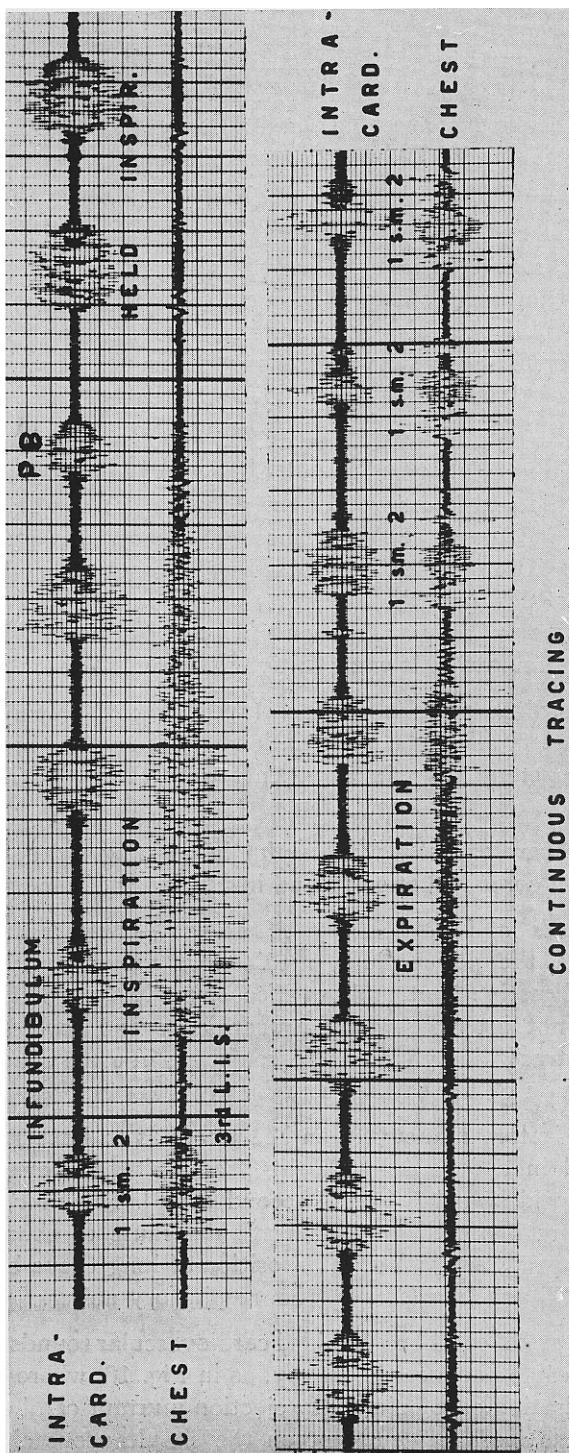


Fig. 17.—In this case of mitral stenosis and insufficiency there was clinical confusion between a delayed pulmonary second sound and a mitral opening snap. Simultaneous recordings from within the pulmonary artery by the sound catheter and externally from the mid-precordium, where the opening snap was best heard, allowed differentiation between the two auscultatory signs.



CONTINUOUS TRACING

Fig. 18.—Infundibular pulmonary stenosis. Simultaneous intracardiac and external recordings to demonstrate how respiratory sounds, while interfering with the external phonocardiogram, do not affect the intracardiac phonocardiogram. Furthermore, whereas the systolic murmur actually increases during inspiration (see the intracardiac tracing), it decreases paradoxically on the external recording, probably because of interposition of lung tissue between the outflow tract of the right ventricle, where the murmur originates, and the chest wall. (P.B. = Premature beat.)

with tricuspid atresia a mid-diastolic murmur was recorded in the inflow tract of the left ventricle. In 1 patient with persistent atrioventricular canal a pansystolic regurgitant murmur was recorded within the atrial chambers, suggesting either mitral or tricuspid incompetence, and a pansystolic murmur of much greater intensity was recorded in the outflow tract of the right ventricle, indicative of a defect of the interventricular septum, with left-to-right shunt at this level.

*Acquired Heart Disease.*—In acquired valvular heart disease, intracardiac phonocardiography has not been so helpful as in congenital heart disease. Nevertheless, it has been useful in clarifying some puzzling auscultatory findings, such as a systolic apical murmur in pure mitral stenosis, or an early opening snap in tight mitral stenosis, often mistaken for a loud delayed pulmonary closure.

Of the 18 patients with pure mitral stenosis, 6 had, on chest auscultation, a definite apical systolic murmur which was localized in the inflow tract of the right ventricle, and in the mid-lower part of the right atrium by phonocatheter, indicative of tricuspid insufficiency. All of these 6 patients had elevated pulmonary pressure (80 to 110 mm. Hg) and dilated right ventricle on fluoroscopy. The sign of Rivero Carvallo (increased intensity of the systolic murmur during inspiration) was positive in all patients on intracardiac phonocardiography (Fig. 16), whereas on auscultation it was positive in 2 patients only.

Simultaneous recordings from the chest (apex or mid-precordium) and within the pulmonary artery allowed differentiation between a delayed pulmonary closure and a mitral opening snap in 3 patients with severe mitral stenosis (Fig. 17).

Intracardiac phonocardiography was helpful in defining right-sided gallop bruits in 2 patients with combined stenosis and insufficiency of the mitral valve.

*Extracardiac Sounds and Respiratory Maneuvers.*—Extracardiac sounds which may arise from the pericardium, mediastinal vessels, pleurae, lungs, bronchi, chest wall, or digestive tract, and can affect so easily the ordinary external phonocardiogram, do not interfere with intracardiac sound recordings. As a consequence of this, intracardiac phonocardiograms are, in some conditions, the only faithful reproduction of the cardiac sounds.

The effects of respiratory maneuvers on heart sounds and murmurs are on many occasions useful in differentiating right- from left-sided events and in defining fixed or reversed splitting of the second sound. However, not infrequently these effects are obscured on auscultation because of changes in the position of the heart during respiration, interposition of lung tissue between the heart and the chest wall, and interference from respiratory noises.

The actual respiratory effects on right-sided cardiovascular sounds can always be detected by intracardiac phonocardiography, as in Fig. 18, where it is shown that the effects of inspiration on a pulmonary ejection murmur can be evaluated only on the intracardiac recording and not on the simultaneous chest phonocardiogram.

## DISCUSSION AND CONCLUSIONS

In agreement with the observations of Lewis and associates,<sup>7</sup> in all subjects with normal hemodynamic findings included in this series a rather soft systolic murmur was present within the pulmonary artery. This murmur had the characteristics of a flow murmur; it was high-pitched, had a mid-systolic accentuation, and ended before the pulmonary closure. In simultaneous recordings from the second intercostal space this murmur was observed in less than 50 per cent of the cases. No close relationship was noted between the loudness of the murmur within the pulmonary artery and its presence or intensity on simultaneous chest recordings. Therefore, factors other than the characteristics of the murmur itself must be involved in the transmission of this "physiologic" murmur to the surface of the thorax.

Analysis of the results in cases of congenital heart disease, which represent 61 per cent of the entire series, shows that intracardiac phonocardiography was of diagnostic value in many instances; in others it provided useful information as to the site and mechanism of production of the complex auscultatory signs.

In cases of isolated ventricular septal defect, a pansystolic murmur, sharply localized within the right ventricle, was a diagnostic sign even in cases in which oxygen studies were doubtful, or suggested the presence of an interatrial or aorto-pulmonary shunt, rather than an interventricular one. The intracardiac phonocardiogram was particularly useful in diagnosing small ventricular septal defects causing shunts not detectable by oxygen or dye dilution studies.

In cases of tetralogy of Fallot no murmurs were recorded within the right ventricle, and only a very soft ejection murmur was present in the aorta (Fig. 6). This confirms the fact that the defect of the interventricular septum in this condition is usually silent, as already suggested,<sup>15,16</sup> and that the loud ejection murmur recorded within the pulmonary artery is responsible alone for the harsh systolic murmur audible over the precordium. It is of interest to note that whereas the pulmonary closure was present on the ordinary chest phonocardiogram in only 20 per cent of our cases, and in only 15 per cent of the cases with infundibular stenosis in other series,<sup>16</sup> it was always present in the intracardiac phonocardiogram recorded from the pulmonary artery. This allowed measurement in every case of the delay in pulmonary closure compared with the aortic, and confirmed that there is a good correlation between this delay and the pressure gradient between the right ventricle and pulmonary artery, as suggested by various authors.<sup>17,18</sup>

The two consistent and entirely different intracardiac phonocardiographic patterns in valvular and infundibular pulmonary stenosis (Fig. 9), even when the pressure tracings were equivocal, established the differentiation between the two types of lesion. It is not fully understood why the murmur in infundibular pulmonary stenosis disappears gradually during pullback of the phonocatheter from the pulmonary artery into the right ventricle. Probably, within the infundibulum, where the blood velocity is higher, the murmur becomes so high-pitched that some of its vibrations are not recorded, or are recorded at lower intensity by the phonocardiograph.

In cases of patent ductus arteriosus, in agreement with Lewis' observations,<sup>7</sup> and our previous experience,<sup>10,11,19</sup> the intracardiac phonocardiogram has been of diagnostic value. A continuous machinery type of murmur, localized within the pulmonary artery, and loudest at times at the bifurcation or in the left pulmonary artery, was the typical diagnostic sign in this condition. It is of great interest to observe that this murmur was present within the pulmonary artery, even in cases of patent ductus arteriosus which showed atypical auscultatory findings on clinical examination and normal hemodynamic features at right heart catheterization. Lewis and associates<sup>7</sup> described one of such cases in their series. Patent ductus arteriosus with absence of a continuous murmur is not infrequent<sup>20</sup>; this condition represented 5 to 10 per cent of all cases in a recent series<sup>21</sup> and in our own experience.

Intracardiac phonocardiography has been of diagnostic value not only in establishing but in excluding as well the diagnosis of patent ductus arteriosus in the presence of auscultatory and catheterization findings simulating such a condition, namely, other types of intrathoracic arteriovenous fistulae, ventricular septal defect with aortic or pulmonary insufficiency, mild aortic stenosis and insufficiency.

In cases of uncomplicated atrial septal defect, intracardiac phonocardiography has been very helpful in excluding possible associated congenital lesions, such as ventricular septal defect, and in achieving confirmation of the site and mechanism of production of the many auscultatory signs in this condition.<sup>22-26</sup> The basal systolic murmur has been localized within the pulmonary artery in agreement with previous observations by Soulié and co-workers,<sup>5</sup> Feruglio and Dalla Volta,<sup>11</sup> and Luisada and Testelli,<sup>26</sup> and the mid-diastolic and presystolic murmurs have been localized within the inflow tract of the right ventricle, due to the increased blood flow across the tricuspid valve, as suggested by various authors.<sup>22-24</sup> Similar mid-diastolic and presystolic murmurs have been localized within the inflow tract of the left ventricle in cases of tricuspid atresia. No murmurs attributable to flow were recorded across the septal defect in cases of atrial septal defect with uni- or bidirectional shunt; this is a conclusion not shared previously by others.<sup>22,27</sup>

In cases of Lutembacher's syndrome the presence of a mid-diastolic or presystolic murmur within the inflow tract of the left ventricle is a diagnostic sign of mitral obstruction even in the absence of a significant pressure gradient across this valve.

In cases of persistent atrioventricular canal a loud pansystolic murmur within the right ventricle (indicating a left-to-right shunt at this level) associated with a systolic regurgitant murmur within the atria (due to mitral or tricuspid incompetence) were two signs favoring the diagnosis.

When the results obtained in acquired heart disease were analyzed, it was shown that intracardiac phonocardiography has only a few practical applications, such as in establishing the tricuspid origin of the apical systolic murmur in pure mitral stenosis (due to relative tricuspid insufficiency), in differentiating between a mitral opening snap and a delayed pulmonary closure (by means of simultaneous

recordings from within the pulmonary artery and externally from the mid-precordium or the apex), and in defining right-sided gallop rhythm.

In both congenital and acquired heart disease, intracardiac phonocardiography has been of value in differentiating intra- and extracardiac sounds, and it has provided faithful records of the actual effects of respiration on heart sounds and murmurs.

Special attention has been paid in this investigation to the fact that in intracardiac phonocardiography, sounds and murmurs, even loud ones, are sharply localized in one chamber or vessel and are not recordable in any other contiguous cavity. This problem is still under investigation. At present it can be said only that even by using full amplification in the recording system, murmurs could not be recorded in any other chamber except that which received the blood flow responsible for their production.

#### SUMMARY

With the aid of a barium titanate microphone incorporated in the tip of a specially designed catheter, intracardiac phonocardiograms were recorded in 160 subjects, of whom 20 had normal hemodynamic findings, 97 had congenital heart disease, and the remainder had acquired heart disease.

The normal intracardiac phonocardiographic patterns from both the right and left cavities of the heart are described. The diagnostic value of intracardiac phonocardiography in congenital and acquired heart disease is stressed.

On intracardiac sound recordings, murmurs are sharply localized in that chamber or vessel which receives the blood flow responsible for their production. Therefore, in the presence of uncomplicated ventricular septal defect, a pansystolic murmur is recorded only within the right ventricle; in cases of patent ductus arteriosus a continuous murmur is recorded only within the pulmonary artery. Both these conditions could be diagnosed or excluded with certainty, even when oxygen studies and clinical signs were equivocal. In cases of pulmonary stenosis an ejection murmur was recorded only within the pulmonary artery and its branches. Infundibular and valvular pulmonary stenosis showed different patterns during withdrawal of the sound catheter into the ventricle. This allowed differentiation between the two types of lesion. In atrial septal defect it was confirmed that murmurs and sounds are produced by the increased flow across the tricuspid and pulmonary valves, rather than by the flow across the defect. In cases of Lutembacher's syndrome a mid-diastolic or presystolic murmur in the inflow tract of the left ventricle was a diagnostic sign.

In cases of pure mitral stenosis with apical systolic murmur the tricuspid origin of the murmur could be established. Simultaneous chest and intracardiac phonocardiograms allowed differentiation between valve closure and early diastolic sounds and between intra- and extracardiac sounds.

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