Intracardiac Phonocardiography in Man

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This paper describes a new method for the detection of sound from within the heart in man. It employs the technic and equipment of underwater listening developed and used by the Navy in antisubmarine warfare. Records are shown to illustrate the localization of heart sound production to an extent not possible by phonocardiograms from the chest wall. The technic has proved helpful in the evaluation of patients with valvular and congenital heart disease.

The purpose of this paper is to describe a new method for the detection of sounds from within the human heart. Records will be shown to illustrate that this technic enables localization of sound production to an extent not heretofore obtainable. By virtue of this property and by recording the sounds from their point of origin, new avenues of investigation of the production and transmission of sounds and murmurs are now available.

These investigations were made possible by the application to medicine of acoustic technology developed for undersea warfare. The technic was simply that of underwater listening, the passive phase of sonar. The transition from antisubmarine warfare to phonocardiography involved mainly miniaturization of the transducer and adaptation of the amplifiers to the recording instruments used in routine clinical phonocardiography.

Methods

The technical aspects of this work have been reported previously and will only be referred to briefly. The transducer is a hollow, circular cylinder of activated barium titanate. The activation process was carried out by a methodology developed by Wallace and imparts to the ceramic the qualities of a piezoelectric crystal. Catheters were made by drawing the transducer with its attached cable back into nonirritant plastic tubing. The transducer and cable were coated with silicone oil to provide lubrication for the insertion and also to provide acoustic coupling between the outer wall of the ceramic and the inner wall of the plastic tubing. The end of the catheter was then sealed chemically. In the present study 2 types of catheters have been used. The first was a single-lumen catheter for sound determinations alone and was approximately a no. 5F in size. The second was a double-lumen catheter using the sound element of the single-lumen catheter and having the other lumen for the usual pressures and sampling. Its size was approximately that of a no. 7½F catheter. In both types, the ceramic at the tip has been made ¼-inch long.

The signal from the catheter was fed into a cathode follower-preamplifier unit designed specially for this study. This unit allowed the sounds to be recorded on the phonocardiographic apparatus available in our laboratory, by matching impedances and by several stages of amplification. The records were taken on the Sanborn Twin Beam photographic instrument. Tape recordings were also made with the Cambridge Educational Electron Cardioscope at a tape speed of 1½ inches per second. These were made primarily for sound spectrographic analysis by the technic described by McKusick et al. and for comparison of intracardiac sounds with sounds heard on the chest wall as to duration, amplitude, and frequency. These studies will be reported in a subsequent communication.

The subjects of this study, the first 41 patients on whom intracardiac sounds were determined, were all patients from the wards and clinics of the Philadelphia General Hospital and the heart sound studies were done in conjunction with routine cardiac catheterization. The addition of intracardiac phonocardiography to the technic of cardiac catheterization alters the latter procedure very little. Two features of the technic of intracardiac phonocardiography are worthy of note: since the barium titanate is inactivated by autoclaving,† the catheters must

* Tygon tubing, obtained from the United States Stoneware Company, Akron, Ohio.
† Catheters are being developed using a transducer with a higher temperature tolerance that will permit autoclaving.
be sterilized in an antiseptic solution;* since the voltage output of the transducer is very small, special precautions must be taken to reduce electric interference. In our laboratory this has been obtained by careful grounding of all equipment and the patient, by making the sound catheters as short as possible, and by isolating the cathode follower-preamplifier unit in grounded aluminum foil. For the routine cardiac catheterization, pressures were determined with the Sanborn electromanometer. Blood samples were analyzed for oxygen content by the technic of Van Slyke and Neill6 and gas samples by the technic of Scholander.5 Cardiac outputs were done by the direct Fick principle with blood taken from the pulmonary artery for the mixed venous sample.

Since the sound catheter has a barium titanate element at the tip and has a metallic cable, it is visible by fluoroscopy. Figure 1, taken at the time of catheterization of the left pulmonary artery shows that the barium titanate transducer at the tip is radiographically more dense than the rest of the catheter.

Table 1 shows the diagnoses of the patients. The cardiac rhythm was normal in 37 and showed atrial fibrillation in 4. The intracardiac sounds in each patient are not described; since the number in each group is small, a detailed correlation is not justified. In this preliminary presentation we have chosen to discuss the various auditory events of the normal heart cycle, the abnormal sounds, and finally the heart sounds of congenital heart disease. The salient features of each record are given with each illustration.

* We have used Detergicide obtained from the United States Catheter and Instrument Company, Glens Falls, N. Y.

** First Heart Sound. In all of the patients the first heart sound was heard throughout the lesser circulation. It was heard in both veins near the heart, within the heart itself, and throughout the pulmonary tree, even with the catheter tip in the “wedge” position. With few exceptions, the maximal intensity was in the right ventricle (fig. 2). When differences in the intensity within the right ventricle could
be appreciated, it was found to be louder in the outflow tract than in the inflow tract (fig. 2B). Of very great interest to us was the observation that the first heart sound was never loudest in intensity in the region of the tricuspid valve. On occasion its intensity here (fig. 2B) was markedly reduced but usually to a lesser extent. These observations on the first heart sound suggested that we were hearing events from the left ventricle and that closure of the tricuspid valve was not a major contribution to the make-up of this sound.

In addition to observations on the over-all intensity of the first heart sound, differences in the intensity of components of the first heart sound have been noted depending on the position of the catheter tip. For example, in figure 3, the major component of the first heart sound begins 0.06 to 0.07 second after the Q wave and occurs during the early pressure rise in the right ventricle. * Most, if not all, of this component is over before the ventricular pressure has reached the level of the end-diastolic pressure in the pulmonary artery. At the time that this sound is produced, therefore, the pulmonic valve is closed. In the pulmonary artery, in contradistinction, the first major component of sound begins 0.14 second after the Q wave and is on the ascending limb of the pulmonary artery pressure curve. In the tracing taken in the pulmonary artery a few vibrations of low intensity are seen 0.06 to 0.07 second after the Q wave, and correspond, therefore, in time with the loud sound heard in the right ventricle. There is, then, a sound occurring with the onset of ventricular contraction that is loud in the right ventricle and is of much less intensity in the pulmonary artery. It would appear that this loud sound is transmitted poorly through the closed pulmonic valve. This same phenomenon, namely, that loud components of the first heart sound occur earlier in the right ventricle than in the pulmonary artery, is also present in figures 2A and 2B.

Second Heart Sound. In all of the patients, the second heart sound, like the first heart sound, was heard throughout the lesser circulation. Its maximal intensity was found to be in the pulmonary artery. There was little difference in intensity throughout the pulmonary tree, even with the catheter tip in the “wedge” position. During the withdrawal of the catheter from the pulmonary artery into the right ventricle, the intensity of the second heart sound decreased suddenly and the intensity of the first heart sound increased (fig. 2A). At the time of catheterization, the person listening to the intracardiac sounds was able to detect this change in the position of the catheter tip by the nature of the sounds without knowledge of the intravascular pressures or fluoroscopic image. The second heart sound could be heard in the inflow tract of the right ventricle and in the right atrium but was almost always much less intense. Of interest is the observation

* The photographic records are displayed as they were obtained. To correct for the time delay in pressure registration as compared with sounds, the pressure curve should be moved to the left 0.01 second.
Fig. 2. Sounds from the lesser circulation. In each strip the upper record is of intracardiac sounds and the lower record is lead II of the electrocardiogram; the time lines are 0.04 and 0.2 second respectively. (A) NC, 28-year-old man. Pulmonary tuberculosis, left upper lobe. Normal heart; no murmurs. The first heart sound is heard at all locations and is loudest (i.e., greatest amplitude) in the right ventricle. The second heart sound is heard at all locations and is loudest in the pulmonary artery and superior vena cava; there is a marked drop in intensity in going from the pulmonary artery to the outflow tract of the right ventricle. The third heart sound is heard in the right ventricle and is probably present in the tracing from the right atrium; it is loudest in the inflow tract of the right ventricle. The fourth heart sound is heard in the right atrium and right ventricle. There is a midsystolic murmur in the pulmonary artery. (B) CA, 47-year-old woman. Rheumatic heart disease with mitral stenosis and insufficiency and atrial fibrillation. The first heart sound is heard in all locations and is loudest in the right ventricle; in the region of the tricuspid valve (right ventricle low) the intensity is much less than at the apex of the right ventricle. The second heart sound is heard in all locations and is loudest in the pulmonary artery; its intensity drops abruptly from the pulmonary artery to the outflow tract of the right ventricle. No fourth heart sound is heard at any location. Systolic and diastolic murmurs are present at all locations with the exception that the diastolic murmur is not heard in the pulmonary artery.
that in some cases the second heart sound became loud again as the catheter was withdrawn into the superior vena cava (figs. 2A and 2B). We think that this difference is related to the proximity of the vena cava to the right pulmonary artery.

In addition to observations on the over-all intensity of the second heart sound, we have also been interested in the site of origin of the second heart sound heard in the pulmonary artery. This point is illustrated in figure 4B which shows phonocardiograms taken on a 41-year-old woman with rheumatic heart disease with mitral stenosis and insufficiency and possible aortic valvular disease. Roentgen examination showed slight left atrial enlargement, straightening of the left heart border, and prominence of the pulmonary artery. At cardiac catheterization, the pressure in the pulmonary artery was 20/9 mm. Hg, with a mean pressure of 13 mm. Hg. Systemic blood pressure was within normal limits. On auscultation, as seen in the upper tracings, which are precordial phonocardiograms, an accentuated P₂ was described. However, as seen in the lower tracing, the second heart sound in the pulmonary artery was not accentuated. Since the second heart sound in the pulmonary artery was not loud, the loud second heart sound heard at the pulmonic area on the chest was not due to pulmonic closure but to aortic closure. Others have suggested that in some cases, the loud second heart sound heard at the pulmonic area is due to closure of the aortic valve. We think that this record is strong evidence in favor of this viewpoint and believe that the location of the sound results from rotation of the heart and great vessels.

Third Heart Sound. This sound has been present in only a few of the cases studied to date (figs. 2A, 5B, and 6D). In 1 case a third heart sound was noted in the chest phonocardiograms taken at the apex. A third heart sound was also noted in the intracardiac recordings. In 2 cases a third heart sound was noted in the intracardiac phonocardiograms but was not recorded in those taken from the precordium. The third heart sound has been heard in the right ventricle and in the right atrium. It appears to be of greatest intensity in the infow tract of the right ventricle (fig. 2A), but has been heard with almost equal

Fig. 3. Intracardiac sounds and intravascular pressures. DS, 39-year-old woman. Rheumatic heart disease with mitral insufficiency and left bundle-branch block. Top row: intracardiac sounds and lead II of the electrocardiogram. Bottom row: intracardiac sounds and intravascular pressures from the pulmonary artery, right ventricle, and right atrium. The first heart sound is louder in the right ventricle than it is in the pulmonary artery; the initial loud component heard in the ventricle and in the atrium occurs at the onset of ventricular systole and is virtually complete before the ventricular pressure has reached the level of end-diastolic pulmonary artery pressure; it occurs, therefore, before the pulmonic valve has opened. The fourth heart sound occurs at the peak of the “a” wave in the right atrial tracing. The peak intensity of the systolic murmur in the pulmonary artery occurs at the peak of the pulmonary artery pressure.
Here, a premature ventricular contraction does not have the normally occurring systolic murmur, while the following normally conducted beat after a longer filling time has a systolic murmur of greater than normal intensity. B. OG, 41-year-old woman. Rheumatic heart disease with mitral stenosis and insufficiency and possible aortic valve disease. Pulmonary artery pressure 20/9 mm. Hg with a mean of 13 mm. Hg. The upper row of tracings shows chest phonocardiograms taken at the aortic and pulmonic areas and demonstrates that P₂ was accentuated and louder than A₂. The bottom tracing taken of intra-cardiac sounds in the pulmonary artery shows that here the second sound is not accentuated.

intensity in the right atrium (fig. 6D). No conclusions have been reached as to the etiology of this sound from the observations currently available.

Fourth Heart Sound. In all patients who had atrial contractions (i.e., a P wave in the electrocardiogram) a fourth heart sound was heard in some part of the heart. In a few cases it could be heard in all locations (figs. 3, 4B, and 6D), but in the remainder it was localized to the atrium and either diminished markedly or disappeared entirely as the catheter was moved into the ventricle or the venae cavae. On the other hand, it was unusual to observe a fourth heart sound in the chest phonocardiograms.

Frequently the sharp localization of the fourth heart sound within the heart was surprising. Figure 5A shows a loud fourth heart sound in the right atrium almost the intensity of the first heart sound and yet it is not heard in the superior vena cava just above the right atrium. In figure 5B when the tip of the catheter is in the right atrium near the tricuspid valve, there is a definite fourth heart sound, which is inaudible when the transducer lies at the mid-atrial level. Figures 5A and 5B suggest that the direction of blood flow may have something to do with carrying the sound away from certain parts of the heart. However, in figure 6C are records taken on either side of the tricuspid valve showing a clearly audible fourth heart sound on the atrial side, which is virtually inaudible on the ventricular side. This is occurring at a time when the tricuspid valve is open and blood is flowing from the right atrium into the right ventricle.

In much the same way that changes in the nature of the first and second heart sounds allow the listener to tell the transition from the pulmonary artery to the right ventricle, the appearance of a well-defined fourth heart sound allows the listener to note the transition from the right ventricle to the right atrium. The fact that the sound is loudest in the right atrium leads us to conclude that it is related to atrial events. It is related to atrial contraction as judged by the fact that the sound occurs at the peak of the “a” wave of the atrial pressure pulse (fig. 3, right atrium) and by the fact that the sound is not heard in patients with atrial fibrillation (fig. 2B). Whether it is due to muscle contraction or alterations in blood flow that occur during atrial systole, we are unable to state at the present time.

Effect of Respiration. Variations in the intensity of the first and second heart sounds due to respiration have been difficult to assess. In most cases, these sounds seemed unaffected by respiration. In contrast, respiration caused marked variations in the intensity of the fourth heart sound. Figures 5D and 6D (right atrium) show intracardiac phonocardiograms at the onset of respiration which, as expected, increased the intensity of the fourth heart sound. For comparison, the lower strip in figure 5D shows no fourth heart sound heard on the precordium.
Fig. 5. The fourth heart sound. A. TC, 24-year-old man. Idiopathic dilatation of the pulmonary artery. There is a loud fourth heart sound in the right atrium, which is not heard in the superior vena cava just above the right atrium. B. JC, 24-year-old woman. Rheumatic heart disease with mitral stenosis. A fourth heart sound is present in the right atrium near the tricuspid valve and is very faint on the ventricular side. D. JB, 56-year-old man. Chronic pulmonary fibrosis and emphysema, dilated pulmonary artery, pulmonary artery pressure 30/15 mm. Hg, with a mean pressure of 24 mm. Hg. In the top strip intracardiac sounds from the right atrium show variations in intensity of the fourth heart sound due to respiration. In the bottom strip no fourth heart sound is heard on the precordium.

Murmurs. Where murmurs were heard on the precordium, they were also heard within the heart (figs. 2B, 3, 4B, 5A, 5C and 7). The diastolic murmur of mitral stenosis, in patients who clinically and by cardiac catheterization did not have tricuspid valvular pathology, was heard in the right ventricle and in the right atrium but was not heard in the pulmonary artery. In those cases with the apical systolic murmur of mitral insufficiency, this murmur was also heard in the right ventricle and in the right atrium but was loudest in the pulmonary artery. The difference in the transmission of the 2 murmurs to the pulmonary artery was somewhat puzzling until it was observed that in those cases in which there were no murmurs present in the chest phonocardiograms and, indeed, elsewhere in the heart, there was a systolic murmur in the pulmonary artery (fig. 2A). In every tracing thus far obtained there has been a systolic murmur in the pulmonary artery. We believe that this localization is due to blood flow. This is based on the observations that (a) the peak of intensity of the
Fig. 6. Congenital heart disease. A, RC, 2-year-old boy. Patent ductus arteriosus. Top row: intracardiac sounds and lead II of electrocardiogram from the pulmonary artery, right ventricle, and right atrium. Middle and bottom row: continuous tracing of sounds and electrocardiogram. A machinery murmur is heard in the pulmonary artery that is not heard in the right ventricle or right atrium. Continuous tracing demonstrates that the change in the intracardiac sounds occurs abruptly at the pulmonic valve. B, NM, 18-year-old girl. Patent ductus arteriosus. Continuous tracing of intracardiac sound and intravascular pressure as the catheter is advanced rapidly from the outflow tract of the right ventricle into the descending branch of the left pulmonary artery. In the main pulmonary artery in the region of the left pulmonary artery is a loud machinery murmur that is not heard in the right ventricle or more distally in the left pulmonary artery. C, EW, 3-year-old boy. Ventricular septal defect, possible pulmonary stenosis. There is a systolic murmur high in the right ventricle that is not heard at the midventricular level.
murmur occurs at the peak of the pulmonary artery pressure curve (fig. 3, pulmonary artery), and (b) as seen in figure 4A a premature ventricular contraction is not associated with the normally occurring murmur while the next normally conducted beat after a longer filling time has a systolic murmur of greater intensity than that normally heard.

Gallop Sound. In 1 patient there was a loud gallop sound present at the time of cardiac catheterization. In figure 7, on the left, is the chest phonocardiogram taken with the patient in the left lateral recumbent position, which accentuated the gallop. The tracings on the right show that this sound was heard in both the right ventricle and the pulmonary artery. In the right ventricle, the sound is of a low pitch and on auscultation during catheterization it sounded very much like the gallop heard on the precordium. In the pulmonary artery the sound is shorter in duration and has a “snappier” quality. It would appear that the differences in the duration, intensity, and pitch are due to filtering of the sound by the various vascular structures.

Congenital Heart Disease. All the above results indicate that this new technic of heart sound registration localizes sound production to an extent not previously obtainable. This immediately suggests a possible usefulness in congenital heart disease, and, indeed, the most striking examples of localization have been seen in these cases.

Figure 6A shows tracings from a 2-year-old boy subsequently proved at operation to have a large patent ductus arteriosus. In the pulmonary artery there is a machinery murmur completely obscuring the first and second heart sounds. In the right ventricle and right atrium the sounds are well heard with little or no murmur. The 2 lower strips are a continuous tracing as the catheter was pulled back from the pulmonary artery into the right ventricle and show an abrupt transition at the pulmonic valve.

In the above case the typical murmur on the precordium left no doubt as to the diagnosis. In 1 of our patients, however, a murmur was heard at the pulmonic area and up under the left clavicle that was suggestive of patent ductus arteriosus but was not typical. Some observers doubted the presence of a ductus and suggested such possibilities as venous hum, coarctation of the pulmonary artery, pulmonary arteriovenous fistula, and a congenital anomaly of the pulmonic valve. Cardiac catheterization was completely normal. There was no evidence for a left-to-right shunt in the
pulmonary artery nor was there evidence for the other possibilities suggested. Figure 6B is a continuous tracing taken as the sound catheter was advanced rapidly from the outflow tract of the right ventricle to the distal left pulmonary artery. In the main pulmonary artery near the left pulmonary artery a typical machinery murmur of patent ductus arteriosus was heard that was not present elsewhere in the pulmonary tree nor could it be heard within the heart itself. On this basis a diagnosis was made of a small patent ductus arteriosus, which was subsequently shown to be present at operation.

Our studies in congenital heart disease are by no means complete and the exact diagnosis in many cases is at present unproved surgically. Two examples of systolic murmurs in association with congenital heart disease are shown in the last 2 figures.

Figure 6C shows recordings on a boy whose findings at catheterization indicated a high ventricular septal defect and probable pulmonary stenosis. High in the ventricle is a systolic murmur that is not heard in the lower portion of the ventricle. Figure 6D shows tracings from a boy in whom catheterization indicated isolated infundibular stenosis of a mild degree. There is a loud, long systolic murmur in the pulmonary artery and in the outflow tract of the right ventricle. Through a technical error the sounds from the inflow tract of the right ventricle were not recorded, but auscultation during the procedure indicated a marked drop in the intensity of the murmur in this region. This is also seen in the atrial recording. In these 2 cases, therefore, the characteristic murmur was heard best at the place where the lesion was located.

Discussion

To the best of the authors' knowledge this is the first medical report of this new method for the detection of sounds from within the human heart. However, this is not the first report on intracardiac phonocardiography. In 1954 Yamakawa et al.7 reported on studies of intracardiac sounds. Their studies were done in dogs and in "three human beings, in one of which the catheter microphone reached the pulmonary artery and then heart sounds at various parts in the heart were recorded on the tape recorder." However, no records of the sounds obtained in these patients are published in this report. They used a condenser microphone at 1 end of a catheter with the blood and body as the second electrode. This compromise imposed such severe limitations on the results that they say in their summary, "when the catheter tip is placed in the blood stream, chiefly coarse sounds due to a blood whirlpool are recorded, and when the tip is contacted with the inner wall of the heart, chiefly vibrations of a solid structure are recorded. These latter are thought to be similar to the heart sounds obtained from the chest wall." In our technic the barium titinate transducer, both sides of which are isolated from the blood, eliminated this problem. In our studies the best records were obtained when the transducer was free in the blood stream. Little or no "whirlpooling" sounds were noted though some of the high frequency noise may have been due to blood flow. When the catheter tip was in contact with the inner wall of the heart or the valves, loud knocking sounds were obtained that were similar to those obtained when the catheter tip was flicked with the finger nail. We have considered these to be artifacts. From a technical standpoint, too, research on underwater microphones indicates that the fidelity and frequency response of the barium titinate hydrophone, for this purpose, is far superior to the condenser microphone and especially so when the medium in which the sounds are produced is used as one side of the condenser.8

In the studies to date we have recorded intracardiac sounds at the time of cardiac catheterization.* In these preliminary investigations we have not considered ourselves justified in doing this procedure for intracardiac sounds alone when there was no indication for cardiac catheterization. In patients in whom cardiac catheterization was indicated we thought that it was justifiable to take the time

* Three studies were done to assess the feasibility of introducing the single-lumen sound catheter by percutaneous needle puncture. Two were done on patients in whom catheterization was indicated but who were opposed to a cut-down procedure and 1 was done on the senior author.
to record intracardiac sounds. The results have been rewarding in at least 1 case where intracardiac phonocardiography pointed out the diagnosis very clearly. In our hands this procedure has not increased the risk of cardiac catheterization. We believe that there is sufficient merit to this technic to make it part of the routine cardiac catheterization. At the present time steps are being taken to make the equipment available through commercial channels.*

It is clear that we have barely scratched the surface of the amount of information to be learned from this technic. In the realm of studies of immediate clinical importance, further work is being carried out on the value of this technic in the diagnosis of congenital heart disease and in the diagnosis of valvular lesions in patients considered to be candidates for valvular repair. In the realm of basic studies, work is being carried out on the absolute intensity of sounds from inside the heart and a comparison of intracardiac sounds with precordial sounds as to intensity, frequency, and duration. In this way it is hoped that further knowledge as to the method of production of heart sounds and murmurs within the heart and transmission to the chest wall will be obtained.

It should also be clear that a study of intracardiac sounds from the right side of the heart alone is incomplete. Studies in dogs indicate that the intensity of sounds is far greater from the left side than from the right side of the heart.1 There is no reason to suspect that this may not be true for man. We have rejected the idea of retrograde arterial passage with our present catheter because of the likelihood of valvular damage. Sounds from the left heart in man will be studied by the usual technic of left heart catheterization8 upon completion of a special catheter.

**Summary**

A new technic for the detection and study of heart sounds from within the heart in man has been described. This method uses the technic of underwater listening developed for underwater warfare and applies it directly to the study of heart sounds. These studies can be done at the time of right-sided cardiac catheterization with no additional hazard to the patient.

The characteristics of the heart sounds in the lesser circulation have been described. The first heart sound is loudest in the right ventricle. The second heart sound is loudest in the pulmonary artery. The third heart sound is loudest in the right ventricle. The fourth heart sound is loudest in the right atrium.

This technic is capable of localizing heart sounds and murmurs to an extent not heretofore obtainable. The addition of this instrument will materially increase knowledge of the origin of heart sounds and murmurs. The application of this technic to the other studies done at the time of cardiac catheterization should be of definite help in the diagnosis of congenital heart disease.

**Summary in Interlingua**

Es describite un nove technica pro le detection e le studio de sonos cardiac ab intra le corde human. Le metodo usa le technica del ascoltamento subaquatic disveloppate pro le objectivos del belligerentia submarin e applica lo directamente al studio del sonos cardiac. Iste studios pote esser effectuate al tempore de catheterisation dextero-cardiac sin hasardo additional pro le patiente.

Le characteristicas del sonos cardiac in le circulation minor es describite. Le prime sono cardiac es le plus intense in le ventriculo dextere. Le secunde sono cardiac es le plus intense in le arteria pulmonar. Le tertie sono cardiac es le plus intense in le ventriculo dextere. Le quarte sono cardiac es le plus intense in le atrio dextere.

Iste technica pote localizar sonos e murmures cardiac a grados de precision non previamente effectuabile. Le disponibilitate de iste instrumento va servir a augmentar considerablemente nostre cognoscensitas in re le origin de sonos e murmures cardiac. Le application de iste technica al altere studios interprendite al

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*Catheters and amplifiers are available commercially through the American Electronic Laboratories, Inc., Philadelphia, Pa.*
tempore del catheterisation cardiac debe provar se de adjuta definite in le diagnose de congenite morbo cardiac.

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REFERENCES


This report summarizes a 5-year experience in which 313 aortic aneurysms were resected. Surgical technics involved simple excision in the case of saccular aneurysms, and additional grafting in fusiform lesions. When the arch of the aorta is cross-clamped during the resection, a temporary shunt around the occluded segment or hypothermia is employed to protect particularly the central nervous system from ischemic effects. The frequent involvement of one or more major abdominal aortic branches by the aneurysm no longer prevents successful resection, and therefore diagnostic aortography is seldom needed. In dissecting aneurysms the false passage usually is led back into the aortic lumen proximally and distally it is obliterated; less commonly the dissecting area is totally excised. The over-all operative mortality rates have been 31 per cent for 83 thoracic aneurysms, 33 per cent for 27 ruptured abdominal aneurysms, and 8 per cent for 203 nonruptured abdominal ones. Factors increasing these mortality rates have been technical inexperience, patients over 60 years of age, and the presence of hypertension or heart disease. Follow-up studies of patients surviving abdominal operation for periods up to 3½ years have shown in general excellent relief of symptoms, good circulatory maintenance, and a significant increase in survival rates.
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