A Study of Heart Sounds and Murmurs by Direct Heart Recordings

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A method is described for recording heart sounds and murmurs on the surface of the heart chambers and great vessels. Studies were performed on normal dogs and in those with murmurs of known origin, chiefly pulmonic and aortic stenosis. In addition, the reappearance of the murmurs in such cases was studied, following the release of caval occlusion. It was found that the systolic murmur of pulmonic stenosis returned almost immediately after release of caval occlusion, whereas the aortic murmurs did not return until usually from six to 10 beats after release. This may provide a basis for the differentiation of right heart and left heart murmurs by a carefully controlled Valsalva maneuver.

Despite technical advances in phonocardiography, the source of many heart murmurs remains obscure. One example is the basal systolic murmur in patients with atrial septal defects. To investigate further the origin of heart murmurs, an experimental technic has been developed in which sounds are recorded directly from the heart and great vessels in dogs. This technic has been used to record heart sounds in normal control animals, and immediately after the production of a defect in the atrial septum. In addition, in order to test the hypothesis that recording directly from the heart's surface would identify the site of origin of murmurs, experiments were carried out in which aortic or pulmonic stenosis was produced. In the course of these experiments, it was also possible to evaluate the test suggested by Zinsser and Kay in which "right heart" and "left heart" murmurs may be differentiated by the length of time required for the murmur to return, following a Valsalva maneuver.

Methods

The recording of sounds from the surface of the heart chambers was described by Wiggers and Dean in 1917, using a Frank capsule. This method, and similar technics reported subsequently by others, involve suturing of part of the recording apparatus to the heart wall. Since the use of sutures makes it difficult to record rapidly and consecutively from a number of different sites and is not practical on the great vessels, suction was used in our experiments to secure the pick-up device.

A round brass cup measuring 1.9 cm. in diameter is used to transmit sounds from the heart surface. It consists of an inner funnel-shaped cup which is separated from an enclosing outer cup by a space of 1 mm. (fig. 1). The outer chamber so formed is connected via plastic tubing and a three-way stopcock to a constant suction apparatus. The stopcock allows suction to be interrupted, without varying the degree of suction which is maintained at minus 15 inches of mercury. The large inner chamber is connected via a 7 cm. length of plastic tubing of 0.6 cm. internal diameter to a crystal microphone. The signal from the microphone is transmitted to an amplifier which has two outputs, one to an oscilloscope for visual monitoring during the experiment, and the other to a mirror galvanometer in a Hathaway recording apparatus.

In order to determine the frequency response of the complete recording circuit, an alternating current generator was used to drive a condenser microphone. This microphone and the brass cup, which is ordinarily applied to the heart surface, were encased in a suitable housing to assure proper apposition of the surfaces and to decrease extraneous noise. The condenser microphone was then used as a calibrated sound generator. To ensure that the intensity of the sound signal remained constant over the frequency range tested, a carrier frequency circuit was used to measure directly movement of the condenser mem-

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brane. This movement was measured visually on an oscilloscope and necessary adjustments were then made by means of a potentiometer in the alternating current generator circuit. The frequency response of the amplifier alone and the complete recording apparatus is shown in figure 2.

Standard lead II of the electrocardiogram was recorded simultaneously with the heart sounds on a second galvanometer as a timing reference. A third galvanometer was often used to record pressures with a Lilly manometer from the right atrium, right ventricle, pulmonary artery or aorta; or for recording a direct epicardial lead. The paper speed was 8.2 cm. per second; since it was estimated that measurements could be made to within 0.5 mm., the error of measurement of sound duration corresponded to 0.006 second.

Studies were carried out in 32 dogs varying in weight from 8.2 to 19.1 Kg. The first 12 were anesthetized with intraperitoneal pentobarbital sodium; the remainder received morphine sulfate 3 mg. per Kg. subcutaneously, followed in 20 to 40 minutes by a mixture of equal parts of Dial-urethane* and pentobarbital sodium (50 mg. per milliliter) in the range of 0.25 ml. per kg., given intravenously. After the dogs were properly anesthetized, sounds were recorded from the chest wall in the second right intercostal space, the second to fourth or fifth left intercostal spaces, and over the apex. Following this, the trachea was intubated, an intermittent positive pressure respirator connected, and the chest opened. Sounds from the heart and great vessels were recorded in the following locations: (1) Left ventricle—mid-anterior portion, adjacent to the septum. (2) Right ventricle—mid-anterior portion. (3) Left atrium—at the junction of the auricular appendage and atrial wall. (4) Right atrium—at the junction of the auricular appendage and atrial wall. (5) Aorta—2 to 4 cm. distal to the aortic valve (suction often not used here). (6) Pulmonary artery—2 to 3 cm. distal to the pulmonic valve.

In five experiments, the records obtained while using suction were compared with those obtained from the same site while holding the cup manually against the epicardial surface. Records obtained by manual apposition of the cup showed no reproducibility of sounds. The records show less extraneous noise and were more reproducible when suction was used. In these experiments, an epicardial electrocardiographic lead was recorded using the brass cup

* Kindly supplied by Ciba Pharmaceutical Products, Inc., Summit, N. J.
as a conductor. At all times, slight S-T elevation was present when the brass cup was placed on the epicardium but, during suction, a marked “current of injury” appeared which regressed when suction was discontinued (fig. 3).

RESULTS

Normal Sounds: Nine preliminary experiments were necessary in order to obtain a reasonably proficient technic, to determine the optimum type of pickup and resolve other technical problems. These experiments are not included in the following observations.

Normal control sounds were obtained on the chest, heart, and great vessels in 23 dogs. Figure 4 illustrates the sounds recorded from the chest and figure 5 from the heart and great vessels in one such animal. The duration of the heart sounds at each site was measured and the mean and range of values are shown in table 1.

The relative intensity of first and second sounds on the left side of the heart was quite consistent. In all dogs, the first sound was much louder than the second over the left ventricle and the converse obtained for the aorta. The left atrial first sound always exceeded or equalled its corresponding second sound. On the right side, however, the findings were variable. On both the right ventricle and pulmonary artery the sounds were often equal but, occasionally, either the first or second sound was the louder. On the right atrium the first sound was usually louder than the second. The atrial sounds were of greater intensity than those recorded on the ventricular surface.

The second pulmonic sound varied in duration from 0.039 to 0.065 second in 19 dogs and averaged 0.055 second. In four dogs the second sound from the pulmonary artery was clearly split. In these cases this sound averaged 0.088 second in duration ranging from 0.079 to 0.095 second. Figure 6 illustrates such a split pulmonic second sound with a simultaneous aortic recording showing a systolic murmur. The first component of the split sound corresponds in time to the second aortic sound.

![NORMAL CHEST SOUNDS](image)

FIG. 4. Normal heart sounds, recorded from the chest in a normal dog.
Simultaneous sounds recorded from the surface of the right ventricle and right ventricular pressure tracings are illustrated in figure 7.

Atrial Septal Defect: In eight animals an attempt was made to produce an atrial septal defect by incising the septal wall. In five experiments an attempt to produce such a defect resulted in failure, as determined by later necropsy study. In none of these five experiments was a murmur produced over the four cardiac chambers or great vessels. In three animals atrial defects measuring respectively 0.2 cm., 0.5 cm. and 1.0 cm. in diameter were produced, the last being approximately one-half the area of the septum. Phonocardiograms were made up to 60 minutes after the production of the defect and in none of these animals was a murmur produced in any of the six recorded locations. Figure 8 shows the sounds recorded after the production of a 1 cm. diameter defect.

Production of Murmurs: In order to study the characteristics of a heart murmur of known origin, a ligature was placed about the

| Table 1.—Duration of Sounds Recorded on the Heart Chambers and Great Vessels |
|-----------------------------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
|                                   | Aorta | Pulmonary Artery | L. Ventricle | R. Ventricle | L. Atrium | R. Atrium |
| Duration of Heart Sounds in Seconds | 1st   | 2nd   | 1st   | 2nd   | 1st   | 2nd   | 1st   | 2nd   | 1st   | 2nd   | 1st   | 2nd   |
| Average                          | .069  | .043  | .077  | .055  | .098  | .087  | .058  | .084  | .043  | .083  | .040  | .090  | .044  |
| Range                            | .055  | .035  | .063  | .039  | .079  | .061  | .031  | .073  | .032  | .067  | .031  | .077  | .034  |
|                                  | .085  | .055  | .095  | .065  | .095  | .103  | .049  | .098  | .061  | .096  | .049  | .104  | .056  |
main pulmonary artery or aorta. By occluding these vessels to approximately one-half their normal diameter, a loud systolic thrill and murmur was consistently produced. The murmur was well propagated distally, along the vessel, but not proximally. The murmur was not transmitted to the other cardiac chambers or to the opposite great vessel.

Ligatures were placed about the aorta and sounds recorded from the four cardiac chambers and both great vessels in eight dogs. In each case a loud aortic systolic murmur was recorded and the murmur was not present in any of the other areas in seven of the eight animals. One dog had lower frequency vibrations over the main pulmonary artery as well; these occupied the first one-third to one-half of the systole and were of low intensity. Whether this represents transmission of the aortic murmur or is due to the operator’s holding the pickup too tightly against the thin-walled pulmonary artery (and thus producing some degree of stenosis) remains conjectural.

In six experiments, a ligature was placed about the main pulmonary artery; sounds were recorded from it, the aorta and the four cardiac chambers. In five of the six dogs there was no transmission of the systolic murmur from the pulmonary artery. In one of the six, there were some low intensity early systolic vibrations over the aorta.

The Effect of Decreased Venous Return on Aortic and Pulmonic Systolic Murmur: In these experiments ligatures were placed around the superior and inferior vena cavae so that venous inflow could be interrupted temporarily

**Fig. 6.** Illustrates simultaneous recordings of sounds obtained from the aorta and pulmonary artery. An aortic systolic murmur and a split pulmonic second sound are noted.

**Fig. 7.** Simultaneous sounds and pressure recordings from the right ventricle.
when desired, in order to imitate a strong Valsalva maneuver. The azygos vein was ligated in order that the venous return to the right atrium would be limited to coronary sinus flow when both cavae were occluded. Aortic or pulmonic stenosis was produced as above and the resulting murmurs recorded before, during and after five to 20 seconds of venae caval occlusion. One hundred thirteen caval occlusions in 11 dogs were done; 55 in dogs with induced aortic stenosis and 58 in animals with pulmonic stenosis.

In general, the pulmonic systolic murmurs returned to maximal intensity by the second or third postrelease beat, whereas the aortic murmurs returned more gradually, reaching maximal intensity from the sixth to the tenth postrelease beat, as in the experiment shown in figure 10. Figure 11 shows the time elapsed before return of the aortic and pulmonic systolic murmurs, following release of caval occlusion. Within the limits tested, the duration of caval occlusion appeared not to influ-

Fig. 8. Atrial septal defect. Sounds on the heart and great vessels, following production of a 1 cm. diameter atrial septal defect. No murmur is present at any recorded location.

Fig. 9. Caval occlusions. An aortic systolic murmur is shown that is not transmitted to the cardiac chambers or to the pulmonary artery.
The contrasting behavior of aortic and pulmonic systolic murmurs in the same dog is illustrated, recorded before, during and after release of caval occlusion. The pulmonic systolic murmur reappears abruptly with the first postrelease beat, whereas the aortic systolic murmur appears more gradually and reaches maximal intensity with the seventh postrelease beat.

The duration of caval occlusion plotted against the number of beats required for the systolic murmur to reach maximal intensity, following release of occlusion. Note that the pulmonary systolic murmurs usually reach maximal intensity with the second or third postrelease beat, while the aortic systolic murmurs are delayed in reaching maximal intensity until the fifth to tenth postrelease beat.
ence the interval required for the murmur to return to maximal intensity.

**Discussion**

The sounds recorded from the various cardiac chambers were, in general, not unexpected. The finding that the first and second heart sounds are of greater intensity on the surface of the atria than elsewhere may be related to the thinness of the atrial wall and the proximity of the pick-up device to the heart valves.

The recording of a split pulmonic second sound localized to the pulmonary artery in 4 of 23 control animals was quite unexpected. The source of the first component of the split sound may arise either in the pulmonary artery from closure of the semilunar valves, or may possibly be transmitted from the aorta. Since the second component is present only on the pulmonary artery, it is probable that this component arises within the pulmonary artery but its source remains unexplained. Leatham,5 by virtue of simultaneous chest recordings over the pulmonic and aortic areas in man, believes that the first component of a split pulmonic second sound represents aortic valve closure with the second component being due to pulmonic valve closure.

The lack of production of a heart murmur, following creation of an atrial septal defect, sheds no light on the source of such murmurs in man. However, these were acute experiments in open chest animals with recordings being made no longer than one hour following creation of the defect. Clinically, we have observed that, despite apparent adequate surgical closure of an atrial defect, the basal systolic murmur may persist for a variable length of time following operation. These findings suggest that this murmur is related more to the presence of increased pulmonary flow with dilatation of the main pulmonary artery and possible relative pulmonary stenosis, rather than the flow through the atrial defect. Long term animal experiments may clarify this point.

The murmurs produced by placing ligatures about the main pulmonary artery or aorta were in the optimum frequency range of our recording apparatus. Such murmurs (and thrills) were found to be well localized to the site of production and transmitted only distally along the course of the involved vessel. No significant transmission to other cardiac chambers was recorded. Should such localization apply to murmurs of other anomalies, this procedure may provide a basis for the determination of anatomic defects. Intracardiac phonocardiography, utilizing a suitable transducer at the tip of the catheter, may provide such information. These studies are in progress.*

The caval occlusion experiments, with induced pulmonic or aortic stenosis, show a distinct difference in the pattern of response in "left heart" and "right heart" murmurs. Following release of caval occlusion, the pulmonic murmurs returned to original intensity in two to three beats, whereas the aortic murmurs required six to 10 beats to do so. It, therefore, seems possible that a carefully controlled clinical procedure, employing the Valsalva maneuver, can aid in differentiating murmurs arising from the right and left heart.

**Summary**

1. Heart sounds have been recorded directly from the surface of the heart and great vessels in 23 normal dogs. Subsequent recordings were made in 14 of these animals with artificially produced aortic or pulmonic stenosis.

2. Four of the 23 control animals had a split second sound which was localized to the pulmonary artery.

3. The production of an atrial septal defect in three dogs was not associated with the development of a heart murmur.

4. Murmurs due to induced aortic or pulmonic stenosis were recorded at their site of origin and were found to be transmitted distally along the involved vessel but not transmitted to other cardiac chambers.

5. One hundred thirteen caval occlusion experiments were performed in 11 dogs with induced aortic or pulmonic stenosis. Following

* With the aid of Gulton Mfg. Corp., Metuchen, N. J.
release of caval occlusion, the pulmonic murmurs returned to original intensity in two to three beats; in contrast, the aortic murmurs increased gradually, reaching original intensity in six to 10 beats.

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**Summario in Interlingua**

(1) Sonos cardiac eseva registrate directemente ab le superficie del corde e de vasos major in 23 canes normal. Subsequentemente registrationes eseva obtenite ab 14 del mesme animals in que stenoses aortic o pulmonic habeva essite producute artificialmente.

(2) Quatro del 23 animales de controlo habeva un bipartite sono secunde que eseva localisate al arteria pulmonar.

(3) Le production de un defecto atrio-septal in tres canes non eseva associate con le disveloppamento de un murmure cardiac.

(4) Murmures que resultava de inducite stenoses aortic o pulmonic eseva registrate al sito de lor origine; il se mostrava que illos eseva transmititite distalmente al longo del vaso involvite sed que illos non eseva transmititite a altere cameras del corde.

(5) In 11 canes con inducite stenoses aortic o pulmonic un total de 113 experimentos occlusional eseva executate. Post relaxation del occlusion caval le murmures pulmonic retornava al intensitate original post 2 a 3 pulsos. Per contrasto con isto, le murmures aortic se augmentava gradualmente e re-attingeva lor intensitate original post 6 a 10 pulsos.

**References**


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