The Pulmonary Valve in Direct Phonocardiography

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The pulmonary second sound plays an important role in the clinical diagnosis of congenital and acquired heart diseases. Since phonocardiographic recordings from the chest wall present the pulmonary valve sound as part of a composite of sounds produced by 2 valves of the heart, it was our interest to demonstrate the pulmonary sound by direct phonocardiography.

Cardiac sounds and murmurs heard and recorded at the chest wall under normal and pathologic conditions are composed of audible vibrations associated with different mechanical events of the cardiac cycle occurring at the same time or in close sequence. Therefore, separation and identification of vibrations due to a single event, such as closure of I valve, is frequently difficult. The problems increase with anatomic variations, as they occur in congenital malformations of the heart and great vessels and with a combination of such defects. It is reasonable to assume, that recording directly from the surface of the heart, as close as possible to the presumed origin of the single sound will increase the understanding of those vibrations as well as their variation with pathologic conditions.

Direct phonocardiography has been used in the past under experimental conditions. Most investigators have recorded vibrations only from the ventricles. To our knowledge, Wiggers and Dean,1 were the first to have recorded sounds from the aorta and pulmonary artery as well. Bertrand and associates2 recently reported an experimental study in dogs employing a stethoscopic bell pick up held in place by suction. They recorded heart sounds and experimentally produced murmurs from all 4 chambers of the heart and from the aorta and pulmonary artery. Impressed by their excellent records we also used a similar stethoscopic bell. Apparatus was specially designed by one of us (WMR) to meet the peculiar requirements of direct phonocardiography. A direct recording technic was developed and applied to a series of dogs under experimental conditions. Since the tracings obtained were uniformly satisfactory, the method was transferred to the operating room and employed at surgical exploration of human patients having congenital or acquired heart disease.

Our present work is chiefly concerned with sounds and murmurs produced by the pulmonary valve. An experimental approach was therefore chosen in order to eliminate the sound due to the presence and function of this valve.

Heart sounds were recorded from the chest wall before opening the thorax. Direct recordings were made from the surface of the 4 chambers of the heart, the aorta and the pulmonary artery in a series of 20 adult dogs of 11 to 19 Kg. body weight. Fourteen of the 20 animals were operated on with the aid of extracorporeal circulation for various studies, most of them unrelated to the present communication.

Pulmonary stenosis was created surgically in 2 dogs by suture of 2 leaflets of the pulmonary valve through an incision in the pulmonary artery. Direct recordings were made before and after this operation. Then the incision was re-opened in 1 dog and the entire pulmonary valve excised and postoperative phonocardiograms obtained.

The other dogs were operated on without the aid of extracorporeal circulation.
PULMONARY VALVE IN PHONOCARDIOGRAPHY

Fig. 1. A. Normal dog. Chest wall phonocardiograms from the pulmonary area and lead II electrocardiogram. B. Direct recording over the pulmonary valve. Note systolic murmur (m) and the split second sound (2 and 2') lead II electrocardiogram. C. Simultaneous records from the pulmonary artery (top) and aorta.

Group received Nembutal anesthesia 30 mg. per Kg. body weight. In 4 dogs the pulmonary valve was excised during venous inflow occlusion. After the azygos vein was ligated and the superior and inferior vena cavae occluded by means of umbilical tape, the 3 leaflets of the pulmonary valve were excised, through an incision in the right ventricular outflow tract, with forceps and scissors. The inflow occlusion lasted 1 to 2 minutes. Pre- and postoperative recordings were obtained. Each operation was performed as an acute experiment and the specimen examined. In all animals the pulmonary leaflets were completely removed down to their origin and no free remnants were found. The heart in which a pulmonary stenosis was created and the valve left in place was also examined and the valve found to be competent.

RESULTS

Sounds recorded from the surface of the heart and great vessels showed variations in appearance according to the particular area from which they were recorded. The first heart sounds showed relatively the highest amplitude and greatest differentiation when they were obtained from a region overlying the mitral or tricuspid valve, and from over the atria where atrial sounds were also well recorded.

The second heart sound showed the highest amplitude and greatest differentiation when recorded from the aorta or pulmonary artery. Over the aorta, the second sound was single, high pitched, of high amplitude and short duration. The second sound over the pulmonary artery was either completely or incompletely split. When splitting was incomplete, that is, when there was a long group of continuous vibrations, the beginning and the end of the group was of higher amplitude, whereas the amplitude was diminished in the middle (fig. 1). Simultaneous tracings from aorta and pulmonary artery showed that the second part of the sound was usually absent in the aortic tracing. Occasionally, however, a small vibration could be recorded from the aorta coinciding with the second part of the sound that was present over the pulmonary artery. Amplitude of the second sound and
degree of splitting varied considerably within the same animal. Generally the splitting was confined to the right ventricular outflow tract, the pulmonary valve area, where it was most marked, and the base of the vessel itself. Occasionally it was transmitted to the right ventricle and both atria.

A systolic murmur was found over the right ventricular outflow tract and the pulmonary artery (figs. 1 and 2). When the murmur was loud it showed a peak in midystole. After creation of pulmonary stenosis, the murmur increased in amplitude and duration and extended across the aortic closure sound (figs. 2 and 3). After removal of the pulmonary valve, the first part of the second sound remained unchanged, whereas the second component disappeared. The systolic murmur also disappeared in every case and a low intensity crescendo-decrescendo diastolic murmur was found, starting at the place of the eliminated sound (fig. 3).

Since the method employed in experimental animals proved to be adequate and informative, it was subsequently applied to human patients subjected to closed and open heart surgery. The receiving bell used in human patients was the same size and shape as the one employed in the experiments, except that the suction groove was eliminated. The larger size of the hearts of human patients and the comparatively slow heart rates did not require special attachments. Careful holding of the receiving bell was sufficient to give clear tracings without artifacts. This is true also for infants, particularly with cardiac enlargement. Thus it was possible to avoid injurious effects of suction. As direct recording in human patients is in an early stage and the number of cases studied is small, we refrain from any further communication at the present time, however, an example is shown in order to demonstrate the potentialities of the method. Figure 4A shows a chest wall tracing from a young girl with coexisting atrial and ventricular septal defects. The tracing obtained from the fourth left intercostal space at the sternal border is compared with one recorded from the right ventricular surface, where the murmur was loudest. It can be seen that the murmur in the direct tracing is pansystolic and stops at the aortic sound (fig. 4B). The wide splitting of the second sound is typical of an atrial defect.

**DISCUSSION**

A split second sound in experimentally produced bundle-branch block was demonstrated by Braun-Menendez and Solari. Wofforth and Margolies found in human cases with
bundle-branch block, that one component of the split second sound was aortic, the other pulmonary in origin. Leatham, by means of simultaneous tracing from pulmonary, mitral, and aortic areas, arrived at the conclusion that the first part of a normally split second sound is aortic since it is synchronous with the dicrotic notch in carotid pulse tracings and the second is related to pulmonary valve closure. He stated that a split second sound is a persistent auscultatory finding in children and young adults. In our series of 30 young adults, only 5 failed to show it. The degree of splitting varied with respiration, being greatest in forced inspiration. In forced expiration it decreased to the point of fusion with the first component which always coincides with the aortic closure in other areas (fig. 5).

Bertrand, Milne, and Hornick found, by direct phonocardiography, a split second sound in 4 of 23 normal dogs. As the splitting was confined to the tracings from the pulmonary artery, they have asked whether it represents aortic and pulmonary valve closure, the first part being pulmonary valve closure, or whether both components are pulmonary in origin. In our tracings, splitting of the second sound, or at least recognizable sequence of 2 sounds, was a persistent finding. While the first component of the sound remained constant in amplitude within the same animal and was recorded from the aorta as well as the pulmonary artery, the second component was rarely found in the aortic tracing and in those cases only as small vibrations. After removal of the entire pulmonary valve, the second component disappeared whereas the first component remained unchanged. A diastolic murmur, obviously due to pulmonary insufficiency, appeared at the time the second component of the split sound had previously occurred. This seems sufficient evidence to justify the opinion that the second part of a normally split second sound is solely due to closure of the pulmonary valve, whereas it has no part in the first component. The aortic valve sound is transmitted to the pulmonary artery in its full amplitude, while only little of a pulmonary valve sound can be picked up from the aorta. Although the pulmonary valve is ordinarily located quite close to the chest wall, a single sound heard in the second or third left intercostal space at auscultation can not be interpreted.

**Fig. 5.** Phonocardiogram from chest wall of a normal young adult. In the pulmonary area wide splitting of the second sound occurs on forced inspiration and decreases to fusion on forced expiration. The first component of the split coincides with aortic closure over the lower left sternum and apex.
as necessarily pulmonary in origin, even though it may be louder than the second sound heard at the aortic area.

The systolic murmur heard over the pulmonary artery disappeared in every instance after removal of the pulmonary valve. Therefore, it can be no artifact, such as could be caused by compression of the thin walled vessel. It is known that a faint systolic murmur can frequently be heard in the pulmonary area of healthy children. It is thought that this is caused by the rapid flow rate through a relatively narrow pulmonary orifice. The similarity of such a murmur with the findings in the dogs is striking. The fact that the murmur in dogs disappeared with removal of the pulmonary valve suggests that flow through the pulmonary orifice is not the only causative factor of such a murmur. It appears that the valve leaflets play an important role in the creation of this functional murmur. Chisholm\textsuperscript{7} explains systolic murmurs associated with structurally normal pulmonary valves on the basis of physical changes in the valve cusps in relation to the lumen of the vessel, a process he termed “trigonoidation.”

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Circulation. 1958;18:992-996
doi: 10.1161/01.CIR.18.5.992

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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