Hemodynamic Correlates of the Various Components of the First Heart Sound

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The origin and significance of the different components of the first heart sound have been variously explained. The rapid vibrations of the first heart sound have been attributed by some authors to valvular events occurring during early systole, i.e., mitral and tricuspid closures at first, pulmonic and aortic openings later. Leatham, with the help of medium-high frequency phonocardiograms in healthy subjects, demonstrated two rather close components of the first sound and attributed the first to mitral closure, the second to tricuspid closure.

Earlier work in our laboratory revealed that the initial rapid vibrations of the first heart sound in each ventricle occurred after closure of the AV valves (average delay: LV = 26 msec from mitral closure; RV = 23 msec from tricuspid closure). It was therefore concluded that closure of AV valves is not directly responsible for the initial rapid vibrations of the first heart sound. Because this vibration coincided in time with the rapid rise of ventricular pressure, it was tentatively concluded that tension of the left ventricular structure would produce this first group of vibrations. From this study it seemed that a detailed investigation of the hemodynamic correlates of the various components of the first heart sound would provide additional information regarding the genesis of these vibrations.

Methods

Thirty experiments were done in adult mongrel dogs weighing from 14 to 23 kg. While in a postabsorptive state, they were lightly anesthetized with sodium pentobarbital intravenously (10 mg/kg). Heparin (10 mg/kg) was given after premedication with subcutaneous morphine (0.5 mg/kg) was given at two-hour intervals. Additional small doses of sodium pentobarbital were given when required.

Three different series of experiments were performed. In the first series of 16 heparinized (10 mg/kg) dogs, four 50 cm #10 Goodale-Lubin catheters were advanced into the pulmonary artery and right ventricle from the jugular veins, and into the aortic root and left ventricle from carotid or femoral arteries. Under fluoroscopic control, the catheters were placed so that their tips were free in the cavity of each chamber or vessel. The end openings of the catheters placed in arteries were plugged to minimize effects from end pressure. In addition, two AEL phonocatheters were advanced into the main pulmonary artery and the aortic root respectively, to be later moved to the right and left ventricles.

Four intracardiac pressure tracings, two intracardiac phonocardiograms, one external phonocardiogram, and an electrocardiogram were simultaneously displayed on an 8-channel oscilloscope and recorded on a film of bromide paper.

Pressures

These were recorded by Statham pressure transducers (P 23 D). The baselines of the two channels recording homolateral ventricular and arterial pressures were carefully superimposed and the pressure pulses were equally amplified. The amplifiers were adjusted to give on the film one mm deflection per mm Hg pressure or a multiple of this. The strain gauges were placed at the level of the respective chambers. The baselines were rechecked at the end of the experiment.

Phonocardiograms

External phonocardiograms were recorded by a dynamic microphone provided with a 2.5 cm open chest piece placed at the left of the sternum and about 2 cm above the apex in order to separate clearly the various groups of vibrations as checked by the oscilloscope. Intracardiac phonocardiograms were simultaneously recorded with barium titanate phonocatheters (American Electronics Laboratory), first from the two major arterial trunks, and later from the two ventricles. High-pass filters with a 24 db/octave slope were used; the tracings were commonly recorded at the 200 cycle/sec and 100 cycle/sec positions.
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Electrocardiogram

The lead showing a distinct Q and a large QRS deflection was recorded throughout the experiments.

Paper Speed

To distinguish different groups of vibrations, and for accuracy of measurements, paper speed was 200 mm/sec.

Special Procedures

In a second series of 10 experiments, in addition to the above data, the first derivative (rate of change) of the left and right ventricular pressures was also recorded. In a third series of four experiments, the heart sounds were recorded directly from the epicardial surface of the exposed heart and from the adventitia of the roots of the large vessels. In order to compare the amplitude in different tracings, the same amplification was used throughout. The device used was a modification of that of Bertrand et al., as previously employed in this laboratory.

Measurements made on 10 consecutive cycles were averaged as follows: 1) R-R interval; 2) P-R interval; 3) Q to onset of LV pressure rise; 4) Q to onset of RV pressure rise; 5) Q to onset of aortic pressure rise; 6) Q to onset of pulmonary artery pressure rise; 7) Q to 1st group of rapid vibrations; 8) Q to 2nd group of rapid vibrations; and 9) Q to 3rd group of rapid vibrations. Measurements of 7, 8, and 9 were made in the external phonoeardiogram and in the intracardiac phonocardiograms of LV, RV, PA, and Ao. The above measurements permitted correlation of the various vibrations with dynamic events.

From each experiment, three to four records of 10 consecutive cycles each were measured. The averages obtained were analyzed separately for each series of consecutive cycles to avoid possibility of comparing data obtained under somewhat dissimilar basal conditions. All measurements were made independently by two observers and compared for possible discrepancies. The rapid paper speed allowed a reading accuracy of two msec. The time relationship of different groups of rapid vibrations to some distinct points on the first derivative of the ventricular pressure was also studied. The delay in transmission of pressure by the catheter systems was five msec, and the presented data have been corrected for this delay.

Results

In 16 experiments on dogs, 400 cardiac cycles were studied in detail and 40 averages of 10 consecutive cycles were made. At the high speed of recording, it was often possible to identify three distinct groups of rapid vibrations within the first sound (fig. 1). The first group of rapid vibrations (first component) of the first heart sound followed the onset of left ventricular pressure rise by an interval of 14 to 30 msec (average 22 msec). The amplitude of this component was maximal in the left ventricular intracardiac phonocardiogram. This first component could also be detected, although greatly attenuated, in the intraluminal phonoeardiogram at the aortic root but was rarely seen in the tracings from the right ventricle or pulmonary artery. Analysis of the first derivative of the ventricular pressure disclosed a constant relationship between the first component and an abrupt change in the rate of pressure rise in the left ventricle (range —2 to +3 msec; figs. 2 and 3). Epicardial phonocardiograms revealed that this component was much larger over the left ventricle than over either the septum or the right ventricle.

The second group of rapid vibrations (second component) of the first heart sound fol-
allowed the first by 10 to 28 msec (average 19 msec) without any constant relationship to the dynamic events of the right heart. It followed initiation of right ventricular pressure rise by an interval which ranged widely from 8 to 48 msec. In some instances, it preceded pulmonary artery pressure rise while, in others, it followed it. This component was rarely recorded in the pulmonary artery or right ventricle. On the other hand, a fairly constant correlation was noted between this component and the first derivative of the left ventricular pressure pulse (fig. 3). Three common patterns presented by this derivative are diagrammatically presented in figure 4. The second component coincided with either the peak of pressure rise during isometric contraction (Type I) or the subsequent notch (Type II). In Type III, which presents a sharp rise, a single peak, and then a drop, the second component is often inseparable from the first. This type of tracing is often seen in the beat subsequent to a premature beat or following an infusion of a small dose of norepinephrine. The absence of a distinct second component seemed to be related to a shorter isometric tension period. This second component was maximal in the left ventricle, and epicardial tracings showed it to be larger over the left ventricle than either the septum or right ventricle. In two instances, where right bundle branch block was induced by probing with the catheter in the right ventricle, the interval between the first and the second component was not modified by this conduction disturbance causing delayed activation and contraction of the right ventricle.

The third group of rapid vibrations (third component) followed the first by 32 to 66 msec (average 46 msec) and always occurred after aortic valve opening. It followed the onset of aortic pressure rise by 10 to 31 msec (average 16 msec), was closely related to the early stage of systolic ejection, and coincided with, or immediately followed, a decline in the rate of change of pressure of the left ven-
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tricle (first derivative) which followed aortic valve opening. This third component was registered best in the intraluminal phonocardiogram from the aortic root and less well in that from the left ventricle. It was consistently most prominent over the left ventricle in the vicinity of the aortic root.

Discussion

The first sound has been described in the past as consisting of four components: an initial group of slow vibrations, followed by two groups of rapid vibrations, and terminated by another group of slow vibrations. The two rapid components are normally regarded as audible. Instead of two rapid components, it has been possible to register three separate groups in the present study with the use of high paper speed and adequate filtration.

Numerous theories have been advanced in order to explain these vibrations. Wiggers and Dean recorded sound vibrations produced by the contraction of an isolated perfused strip of myocardium while Kounatz et al. tried to eliminate valvular action by clamping the venae cavae or blocking the AV valves by balloons, and still recorded a "first sound." In contrast, Dock observed the disappearance of the first sound after ligation of the AV groove or of both cavae plus the azygos vein. Thus, the paramount importance of a valvular factor was emphasized by Dock, as well, as by Smith et al. after experiments on perfused hearts. Subsequent work by Dock indicated that myocardial tissue requires very high pressure changes in order to generate sounds.

A series of animal experiments by Luisada et al. confirmed the earlier findings of Dock and of Smith. However, these studies showed that it is impossible to alter valvular function without causing profound changes of ventricular pressure and volume. Also, it was impossible to alter myocardial function without altering the changes of pressure which cause valvular closure.

Orias and Braun Menendez followed by Rappaport and Syrague admitted the existence of separate vibratory phenomena within the heart sounds caused by atrial, valvular, muscular, and vascular vibrations. Both maintained that the second part of the first sound is related to the opening of the aortic and pulmonary valves and to the vibrations of the walls of the great vessels. This concept was supported by Luisada and coworkers, who demonstrated that clamping of the large arteries in animals abolished the second group of vibrations in the first few beats during which ventricular dynamics were still powerful. This interpretation was further supported by pulse tracings in man.

The progress of phonocardiographic technique in the last 20 years has been made largely through the use of filters, which permit study of the sound characteristics in adjacent frequency bands. This was initiated by Mannheimer, and continued through the
work of Mass and Weber,14 Holländer,13 and Luisada and coworkers.14 18 22 Leatham,1 24 using a medium-high frequency recording system in 30 healthy subjects, demonstrated two components separated by 0.02 to 0.03 sec. He further noted with indirect carotid tracings that the second component occurred after the rise of "aortic" pressure, and attributed it to tricuspid closure. Even though many workers in the field accepted this view, the underlying dynamic sequence is not clear because tricuspid closure precedes aortic valve opening by an average of 0.05 sec in normal human subjects (Braunwald et al. 23 28). The two high-pitched components of the first sound were confirmed and further studied by one of the present authors with Di Bartolo22 recording only the high frequency vibrations (500-1000 cycle/sec). On the other hand, Luisada et al.14 showed that, by using a medium-low frequency band (60-120 cycle/sec), one can often observe a pattern made of four large vibrations within the first heart sound. At that time the four components were ascribed to mitral closure, tricuspid closure, pulmonic opening, and aortic opening and appeared to be confirmed by the sequence of valvular events demonstrated by Braunwald et al.23 24 and by Mosovitz et al.22 While Leatham1 •23 24 explained the two high frequency components as due to mitral and tricuspid closure, Luisada et al.14 22 as well as Minhas and Gaul14 ascribed them to mitral closure followed by aortic opening. More recently Heintzen,29 working with children, appeared to confirm Leatham's interpretation. However, he stated that, in several cases, three vibrations could be observed, and explained them as due to mitral closure, tricuspid closure, and aortic opening. The above studies were made in man and statements about valvular timing were based on external phonocardiograms and either carotid tracings or (occasionally) pressure tracings of the right heart.

Previous work in this laboratory2 demonstrated that AV valve closure is not primarily responsible for the initial rapid vibration of the first heart sound. This has been supported recently by the findings of Saroff et al.30 and van Bogaert et al.31 The former have shown that atrial activity alone is sufficient to close the mitral valve. The latter confirm the existence of a delay of the "first sound" from AV valve closure, admit that the first sound is the acoustic expression of the increase of ventricular pressure, and believe that the contribution of the right ventricle to the first sound is negligible under normal conditions. We then considered it likely that the rapid rise in left ventricular pressure was responsible for the first component. The results of the present study confirm the earlier observation that there is a significant delay between the onset of left ventricular pressure rise and the first component of the first heart sound. The remarkable coincidence between this component and the rapid rise of the first derivative of left ventricular pressure appears to offer significant evidence in favor of the above hypothesis. This finding seems to confirm Wig- gers32 concept that vibrations recorded by the phonocardiograph originate in the entire heart and cannot be attributed to a single cardiac structure.

The second component followed the first by 10 to 28 msec. Since it invariably occurred before the onset of aortic pressure rise, it cannot be related to the events associated with the opening of the aortic valve. Its relation to the onset of right ventricular pressure rise varied widely and it either preceded or followed the onset of pulmonary artery pressure rise. In the absence of a significant temporal correlation, the dynamic events of the right heart do not appear to be responsible for this component. Furthermore, this component was maximal in the left, and seldom seen in the right ventricular intracardiac phonocardiogram. The most significant feature has been its constant association with the first derivative of the left ventricular pressure, where it seems to coincide with a sudden change of isometric tension. Any connection between this component and tricuspid closure is unlikely. The right ventricle is normally considered as a low pressure pump and it is not surprising that, under normal conditions, it fails to generate sudden and marked pressure
changes capable of producing a distinct group of high frequency vibrations within the first heart sound. On the other hand, it is conceivable that, in certain abnormal conditions, where the pressure relations of the two circuits are grossly altered, the situation may be different.

The third component of the first sound followed the first by 32 to 66 msec and occurred slightly after aortic valve opening. Therefore, it seems related to the sudden increase of tension occurring in the aorta during early ejection. Its relation to the decline of the derivative of left ventricular pressure appears to confirm this mechanism. Furthermore, its prominence in the intraluminal phonocardiogram from the aortic root strongly favors the aorta as its source. The authors believe that this component is the physiological counterpart of that sound which, when found in abnormal conditions (aortic ejection click or ejection sound), is larger and delayed.

Possible delay between the pressure curve and its first derivative was studied. It was noted that any sudden change in pressure caused a definite change in the curve of the first derivative without appreciable delay, though peak effect was reached later. Since the first two components of the first sound have been related in the present study to sudden changes in the pressure derivative, the factor of delay does not seem relevant.

This type of hemodynamic correlation between the heart sounds and the first derivative of the pressure pulse has not been emphasized thus far. Since the first derivative indicates rate of change of pressure, it seems ideally suited for such correlation which might relate heart sounds and those phenomena which are likely to throw the "cardiohemic system" into vibration. The close correlation of the various components of the first heart sound to the several events in the pressure derivative of the left ventricle seen in the present study confirms its usefulness. The present study tends to confirm Rushmer's hypothesis\(^\text{33}\) that the heart sounds are caused by "accelerations and decelerations." This hypothesis is applicable to the two first components as related to the left ventricular pressure rise, and to the third component as related to the aortic pressure rise.

Since most hemodynamic data from dogs are considered qualitatively applicable to man, the above correlation of the different components of the first heart sound is probably true for healthy human subjects, with slightly greater intervals. The first two components are separated in the dog by an interval of less than 0.03 sec, and in man by 0.03 to 0.04 sec.\(^\text{33}\) In the dog, the third (aortic) component is separated from the first by more than 0.03 sec. In man, this interval ranges from 0.05 to 0.06 sec.

**Summary**

Previous studies in this laboratory having demonstrated that AV valve closure is not directly responsible for the rapid vibrations of the first heart sound, the mechanism of production of this "sound" was studied further in 30 experiments in dogs. The experiments were performed with multiple pressure catheterization, and intracardiac phonocatheterization, and were compared with external phonocardiograms and electrocardiograms. In 10 experiments, the first derivatives of the left and right ventricular pressures were also recorded. In four experiments, direct epicardial and arterial sound tracings were obtained.

These experiments revealed that three groups of vibrations, or components, were often visible in the external phonocardiogram, and in intracardiac and epicardial tracings. The first component follows the onset of left ventricular pressure rise and closure of the mitral valve but coincides with the rapid rise of the first derivative of LV pressure. The second component has no relationship with right heart events, occurs before aortic valve opening, and coincides with the sudden change of course of the first derivative of LV pressure. The third component occurs slightly after aortic valve opening, and seems related to sudden increase of aortic wall tension. It is probably the physiological equivalent of the aortic "ejection sound." Thus, the first two components are both left ventricular in origin while the third is of aortic origin.
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References


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