Temporal Relation of the First Heart Sound to Closure of the Mitral Valve

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SUMMARY
The objective of this study is to describe the mechanism of production of the first heart sound (S1). The temporal relation between the major components of S1 and (1) cessation of flow across the mitral valve, (2) closure of the valve, and (3) time of peak rate of rise of left ventricular pressure (peak LV dP/dt), were investigated in open-chest dogs. Phasic flow across the mitral valve was recorded by a supraannular electromagnetic flow probe, and timing of valve closure was determined by synchronized cinefluorogram of the motion of the valve cusps after they were made radio opaque. Closure of the mitral valve did not occur at the crossing point of atrial and ventricular pressures (at the beginning of systole), but 20–40 msec later due to inertia of mitral flow. The first major component of the first sound coincided with two simultaneous events: cessation of mitral flow and closure of the valve. It did not show a fixed temporal relationship to time of peak dP/dt.

Additional Indexing Words:
Aortic flow Inertia of flow LV dP/dt Mitral flow Mitral cusp motion

THE ORIGIN of the first heart sound has been debated for more than a century, and the controversy over the precise mechanism by which sound energy originates in the heart has sharpened in the last decade. Lusita concluded recently that "a point of collision between old and new concepts concerning mechanism of heart sounds has been reached."

The traditional view which relates the first sound to closure of the mitral valve, has been challenged by workers who related the genesis of the first sound to rapid vibrations of the whole ventricular mass, induced by the ventricular contraction. The basis for opposition to the valvular theory was the widely held but incorrect concept that closure of the mitral valve is complete when the pressures in the left atrium and ventricle equalize at the onset of systole. The significant delay which was found to exist between the point of pressure equalization and the first major component of the first sound seemed thus to preclude the role of the valve in the genesis of sound.

It is unfortunate that students of heart sound phenomena directed their thinking only toward the correlation between recorded sound vibration and changes of A-V pressures and pressure gradients, and ignored the dynamic relationship between pressure and flow across the mitral valve. Since it has already been demonstrated that the time of zero flow across the mitral valve (at onset of ventricular systole) lags significantly after the time of zero A-V pressure gradient, the latter cannot be the time of complete valve closure.

In this study we present the results of an investigation which correlates the timing of the major components of the first heart sound with phasic measurements of left atrial and left ventricular pressures, mitral flow, and mitral cusp motion. The results are shown to be consistent with established physical principles.

Methods
Twenty large mongrel dogs weighing 30–50 kg were anesthetized with pentobarbital 30 mg/kg. The chest was opened through the left fourth intercostal space, and the dogs were put on total cardiopulmonary bypass. A toroidal electromagnetic flow probe was sutured to the mitral annulus in a supraannular position, and the wires were brought out through the left atrial appendage. A second flow probe was placed...
around the ascending aorta immediately above the aortic valve. Phasic flow was measured with a two-channel electromagnetic flowmeter (Carolina Medical Electronics, Inc.).

In the early experiments, short stiff catheters were placed in the atrium and ventricle, and Statham P23H differential pressure transducers were used to measure intracardiac pressures and the A-V gradient. Aortic pressures were similarly measured. In the later experiments, Konigsberg P-20 implantable transducers were used to measure these pressures simultaneously with the Statham gauges, thus providing high-fidelity records, equal sensitivity, and accurate zero level. An intracardiac phonocardiogram was recorded from the Konigsberg transducer as follows: The unfiltered output of the pressure amplifier was differentiated to remove the low-frequency pressure signal and then filtered through an AC amplifier set to pass frequencies between 40 and 500 Hz. The high-frequency response of the implantable transducer (flat to 1.6 kHz) made this possible. Simultaneous recordings of sound vibrations by both intracavitary and epicardial microphones showed matched appearance of the major sound components. The electrocardiogram (lead I or II) was also recorded; rates of rise of left ventricular pressure \((\frac{dP}{dt})\) and acceleration of flow through the aortic valve \((\frac{dV}{dt})\) were differentiated electronically from left ventricular pressure and aortic flow signals, respectively. Tracings were recorded by a multichannel oscillographic recorder (DR-8 Electronics for Medicine) at a paper speed of 100 or 200 mm/sec. In order to elucidate the motion of the mitral cusps, three methods of making their cross-section radio opaque were tried. In the first two dogs minute amounts of barium were injected subendothelialy at three locations along the cusp: the margin, midpoint, and annulus. In the next two dogs thin wires were sutured to these points, and finally in seven dogs Ray-tec (Johnson and Johnson, New Brunswick, New Jersey) strings were sutured to these same points (Ray-tec is a very extensible thread used in X-ray detectable surgical sponges). In three of these dogs the Ray-tec string was cut between sutures to minimize any potential distortion of the cusp. Cinemfluorograms of cusp movement were taken at 60 fps and synchronized with the oscillographic record by electronic signal.

Synchronization of the Dynamic Events and Possible Delay in the Systems

A most important aspect of this study is the temporal relationship of pressure, flow, cusp position, heart sounds, and peak left ventricular \(\frac{dP}{dt}\). At a paper speed of 100 mm/sec a linear measurement of 0.55 mm corresponds to 5 msec and any transmission delay of more than 5 msec between the origin of an event, its amplification, and the final record will be significant. We investigated three types of delays: mechanical propagation, electric propagation, and frequency-dependent phase lags. Propagation times were studied using two appropriate channels of the recorder and were determined by measuring the intervals on the photographic record. Frequency-dependent phase lags were studied using a two-channel oscilloscope with sweep speeds much faster than the 100 or 200 mm/sec paper speed of the recorder. An impulsive pressure or flow signal experienced no electric propagation delay. The aortic, ventricular, and atrial pressure catheters introduced mechanical transmission delays of 5, 2, and 2 msec, respectively. The Konigsberg pressure gauge had no mechanical delay; an impulsive pressure or flow signal applied directly to its transducer appeared immediately on the record. There was a subsequent distortion of the high frequency components, but there was no delay in appearance time. When the atroventricular pressure difference and the mitral flow are crossing zero, the wave forms are dominated by the frequency of the fundamental, and phase lags of the higher harmonics are of little or no significance. We are confident that the temporal relations between pressure and flow are accurate to better than 2 msec or less than 0.2 mm on the oscillographic record. In the phonocardiogram there was no phase lag greater than 1 msec between 100 and 500 Hz, and a 6 msec lag in the 40 Hz signal. However, an impulsive signal appeared immediately, indicating that the phase distortion did not influence the appearance time of the phonocardiogram.

The recording of \(\frac{dP}{dt}\) was accomplished by taking the derivative output of the pressure amplifier, (an RC circuit) and amplifying it with a DC amplifier. There was no measurable delay between the zero and maximum of the derivative and their corresponding points on the ventricular pressure record. Since cinemfluorograms were exposed at 60 fps, the synchronization of any one frame with its oscillographic record could be in error by as much as 16.7 msec or 1.7 mm. This error was eliminated by using as many as 10 synchronization marks on the cine and record during a run of 10 sec. The probability that all 10 marks would give the same one frame error is \(3^{10}\). We were able to accurately align the cine frames with their associated pressure flow records by using a 60 Hz signal as the marker on the records to correspond with the 60 fps film speed.

Results

The radio-opaque markers sutured to the mitral cusps were found to cause no detectable interference with normal cusp function. This was evident from records which demonstrated no change in the
dynamic relationship between pressure gradient and phasic mitral flow, before and after cusp opacification. Moreover, analysis of the range and mode of cusp movement and of dynamic changes in cusp configuration, did not show any difference when our various technics of cusp opacification were used.

Temporal Relation of the First Heart Sound to the Point of Equalization of Atrial and Ventricular Pressures and to Mitral Valve Closure

In normal sinus rhythm the first sound, as registered on the phonocardiogram, usually showed some low-amplitude vibrations with the onset of ventricular contraction, and its first major (i.e. large amplitude) component coincided with cessation of mitral flow. Flow, in turn, ceased 30-40 msec after ventricular systole had reversed the A-V pressure gradient. This is clearly shown in figures 1-5. In figure 2, the bottom tracing is an enlarged record of the A-V pressure gradient during diastole (subtracted electronically). Flow across the mitral valve starts as soon as left atrial pressure exceeds left ventricular pressure, but does not stop when pressures equalize again. Flow continues against

Figure 1

Original record taken at paper speed of 100 mm/sec demonstrating typical pressure, flow, and sound relationship. Phonocardiogram was recorded through an intracardiac transducer at 40–500 Hz. Time lines are 100 msec apart. Closure of the mitral valve could not occur before a period of 30 msec had passed from the time of the equalization of atrial and ventricular pressures, since, as evident from the record, blood was still crossing the mitral valve during that period. The flow continued against the adverse pressure gradient because of its inertia (for explanation see text). The small reverse flow seen after the valve is closed is not a true regurgitant flow but movement of the sealed mitral cusps and the blood behind them into the atrium during early systole, as is evident from the atrial “C” wave. The first major component of the first sound starts with cessation of mitral flow and reaches its maximal intensity with the peak of the “C” wave and the nadir of the mitral flow tracing. Both represent the point of maximal tensing and oscillation of the closed valve. In this and other records of mitral flow the zero is somewhat unstable because the probe is immersed in blood and perturbed by the electric activity of the heart. This is also evident by the artifact due to atrial and ventricular depolarizations.
the adverse A-V pressure gradient, and ceases 35 msec after the crossing point of the atrial and ventricular pressures. The first major component of the first sound starts at the end-diastolic cessation of mitral flow, and reaches its maximal intensity with the atrial C wave.

Evidence that the valve actually closes at the time of zero flow across the mitral valve, and not earlier at the point of pressure equalization, is included in figures 3 and 4. In this dog the mitral cusps were opacified with a thin wire.

Figure 4 shows selected frames taken from a synchronized cinefluorogram, which demonstrate the position of the cusps at different periods of time throughout diastole. True closure of the valve in this record occurred 40 msec after the crossing point of atrial and ventricular pressures. In the beginning of diastole the closed valve (frame A) opens rapidly when left atrial pressure exceeds left ventricular pressure. Frame B shows the early state of cusp movement toward opening. Frame C shows partial opening, and in frame D complete opening occurred between 25 and 40 msec after the beginning of diastole (maximal valve opening occurred before maximal flow). The valve stayed widely open during the period of rapid early filling and moved toward closure in mid-diastole (frame E). With atrial contraction, the cusps once again opened widely (frame F) and then moved toward closure (frame G). Following the onset of ventricular contraction (point X), the cusp margins moved rapidly to complete closure (frames H and I), 40 msec after the A-V pressure gradient reversal. Frame J shows the excursion of the closed valve toward the atrium induced by the rise in left ventricular pressure, which correlates with what appears on the record to be a reversal of mitral flow.

Figure 2

An original record taken from another dog at a paper speed of 100 mm/sec, displaying (from top to bottom) mitral flow, aortic flow, ECG, left ventricular and left atrial pressures, ventriculoatrial pressure gradient (magnified x5) and phonocardiogram. Time lines are 100 msec apart. This record demonstrates some degree of mitral stenosis. Cessation of flow coincided with the first major component of the first sound, and came 35 msec after the point of A-V pressure crossover.
Temporal Relation of the First Heart Sound to Peak $dP/dt$

Most tracings showed an almost simultaneous occurrence of the first heart sound, valve closure, and point of maximal rate of rise of left ventricular pressure. Figure 5 demonstrates that peak $dP/dt$ occurs 15 msec after closure of the mitral valve (as evident from the cessation of flow); 15 msec after the first major component of the first sound; and at the moment of aortic valve opening. Records taken at a fast paper speed demonstrate clearly that peak $dP/dt$ lags behind mitral valve closure and the first major component. This is even more evident in cases of slow rhythm, with long preejection period and long isovolumic contraction period (fig. 3).

The Aortic Component

Figure 6 is a typical record demonstrating the temporal relationship between the aortic pressure, flow, acceleration of the aortic flow, and different components of the first sound. Pressures were recorded by implantable transducers. A second major component of the first sound seems to coincide with peak acceleration of the aortic flow (peak $dV/dt$). The phonocardiogram was recorded from within the left ventricular cavity, which may partially account for the marked differences be-


Figure 4

Enlarged section taken from figure 3 (showing mitral flow) with the synchronized cine frames. Ten frames were selected to demonstrate various positions of the cusps throughout diastole and early systole. X marks crossing point of A-V pressures. It is clear that the valve stays open beyond the point of pressure equalization (frame H). (For explanation, see text.)

tween the intensity of the mitral and aortic components.

Discussion

Various components of the first heart sound have been attributed to different events occurring in the heart during early systole. Initial low-frequency vibrations were described as resulting from atrial contractions or from gliding movements of contracting fibers of the left ventricular myocardium. The appearance of these vibrations even in patients with atrial fibrillation seems to preclude any role for atrial contractions.

Two major components of high-frequency vibrations were described as following the initial component. The second major component starts with the rise of aortic pressure and onset of aortic flow and is attributed to opening of the aortic valve and peak acceleration of blood flow. Additional components of the first sound were described and were related to change of pressure patterns in the aorta. Luisada et al. excluded the right ventricle and showed that it did not contribute to the first heart sound.

The controversial issue concerns the first major component of $S_1$. Luisada and his associates demonstrated that the point of atrioventricular pressure crossover (zero gradient) precedes the first major component by a period of about 30 msec. They then concluded that this sound cannot be produced by the closure of the valve, since the latter was believed to occur earlier.

Rushmer emphasized that acceleration and deceleration of blood flow can induce sound vibrations and suggested that at the onset of ventricular contraction the first movement of blood closes and seals the A-V valves before ventricular pressure rises, and that onset of ventricular contraction induces high-frequency vibration of the whole "cardiohemic system": ventricular muscle, blood, and closed valves. Van Bogaert et al. suggested
that there is no participation of the valves in the genesis of heart sounds,13,14 and MacCanon and his colleagues postulated that movements of the mitral valve apparatus cannot account for the production of the first sound since the mass excursion of the mitral leaflets, judged from the measurement of maximal motion of cusps and weight of valve is far too small to produce the vibration energy of the first sound.15 The proponents of the nonvalvular theory were further encouraged in their belief that the rise of ventricular pressure per se was responsible for the sound production by the apparent simultaneous appearance of the first major component of the first sound with the point of peak dP/dt during the short period of isovolumic contraction.3

Our findings demonstrate conclusively that the first major component of the first sound is related to the completion of the rapid movement of the mitral cusps toward closure, and reaffirm the observations of Nolan et al.6 and Yellin et al.7,8 that closure of the mitral valve is not complete at the time of zero atrioventricular pressure gradient at the end of diastole, but only some time after the reversal of the pressure gradient, as previously suggested by Faber.16 The blood flowing from the left atrium across the mitral valve has inertia. Moreover it is in motion, and, by Newton’s law of motion, tends to remain in motion unless force is applied to stop it. In this case the required force is supplied by the pressure difference between the ventricle and atrium. Ventricular pressure must exceed atrial pressure for a finite interval of time before the flow can be brought to rest.

Figure 5
A tracing showing the temporal relationship between phasic mitral flow, first sound, phonocardiogram, and peak LV dP/dt. The major component of the first sound coincided with cessation of mitral flow while peak LV dP/dt came 15 msec later, coinciding with the opening of the aortic valve (arrow marks the time of cessation of mitral flow). Time lines are 100 msec apart.
S1 AND MITRAL VALVE CLOSURE

![Diagram of aortic flow, dV/dt, A P, dP/dt, L V P, and phonocardiogram]

**Figure 6**

*Demonstration of the time relationship between components of the first sound and peak acceleration of aortic flow (peak dV/dt). The phonocardiogram was recorded from a transducer located in the left ventricular cavity. This record has been redrawn for clarity.*

Our data also confirm previous studies which showed that in normal sinus rhythm partial closure of the mitral valve occurs at mid-diastole prior to atrial contraction, and again following atrial systole, even before the onset of ventricular contraction (fig. 4, frames E and G). We found no fixed temporal relationship between S1 and point of peak dP/dt, but only between S1 and valve closure. Closure of the mitral valve marks the beginning of the short period of isovolumic contraction of the left ventricle, and under normal conditions peak dP/dt will occur at the end of this period—when the aortic valve opens. During the isovolumic period the system is sealed and the potential energy of contraction cannot be converted to kinetic energy of flow through the aortic valve or used to overcome the inertia of the blood flowing across the mitral valve. Consequently, there is a rapid build-up of left ventricular pressure which normally reaches its peak at the end of this period. Thus the shorter the duration of the isovolumic period, the closer will be peak dP/dt to the first sound, and vice versa.

The vibrations of the second major component of the first sound (fig. 6) may be generated by maximal acceleration of blood flow through the open aortic valve as suggested by Piemme et al. or by the continued oscillation of the cardiohemic system. It is also conceivable that the initial low-frequency vibrations preceding the first major component (figs. 1, 2) are generated by the sudden deceleration of flow across the mitral valve (due to the negating ventriculoatrial pressure gradient at the beginning of systole) before the valve is completely closed.

We therefore conclude that to accept the nonvalvular theory of genesis of the first major component of the first sound is to ignore the existing relations between mitral flow, cusp motion, and sound vibrations. Moreover it complicates the understanding of phenomena which can be explained simply by relating sound to valve closure.

Echocardiographic studies in patients with acute severe aortic insufficiency demonstrated in some cases that the mitral valve closed prematurely in diastole, before ventricular contraction even started, and phonocardiographic tracings revealed that the first sound, though soft because of lack of vigorous closure, still occurred at the time of closure.

In patients with mitral valve prostheses, the mechanical sound of valve closure is clearly defined and does not come at the time of zero atrioventricular pressure gradient but later in the cycle. If the theory that the valve is closed at the point of zero gradient but that sound is evoked later (at the time of peak dP/dt) is correct, in patients with artificial valves one should expect to hear a split sound: a first component, the mechanical closure sound of the artificial valve, and a second component coming later at point of peak dP/dt. But that is not the case. Experiments that elicit loud sounds from isolated valve and chordae do not produce any sounds from the ventricular wall, and the massive ventricular walls mufffe sounds rather than produce them. The fact that children who have less bulky mass of muscle often possess louder sounds than adults makes it hard to accept the notion that the vibrating mass of blood and contractile muscle is responsible for the sound. The argument that the valve alone cannot contribute to the vibration energy of the sound because of
its limited mass excursion\textsuperscript{15} is misleading, since it is not solely the kinetic energy of the moving cusps and chordae that is transformed into sound vibration. The cusps are pushed and tensed by the moving mass of blood and contracting muscle. It is obvious that the main source of energy to produce sound vibrations is the contracting left ventricular myocardium, and that the source of vibrations is the mitral apparatus.

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