Relationship of Heart Sounds to Acceleration of Blood Flow

By Thomas E. Piemme, M.D., G. Octo Barnett, M.D., and Lewis Dexter, M.D.

After more than 100 years of observation, no universal agreement exists regarding the precise nature of the cause of the familiar transient heart sounds. Most arguments for specific cause are based on the demonstration of an association in time between the sound and some other pressure or flow-related event, i.e., valve closure, chordae tensing, etc. There would appear to be two chief reasons for the existing disagreement.

In the first place there has been too often a failure to appreciate the physics of the system under investigation. This has led to errors in interpretation. No portion of the interdependent cardiohemic system may vibrate independently. Thus, a purely valvular cause of transient heart sounds is difficult to conceive. A realistic approach would seem to result from a consideration of those forces which set into vibratory motion the system of blood, heart walls, and valves. Such forces are associated with acceleration and deceleration of blood flow. Acceleration of blood flow is provided by the force of ventricular (or atrial) contraction per unit mass of blood. Deceleration of blood flow occurs in the presence of impedance exceeding the forces promoting forward flow. Heart walls, and tension within elastic vascular structures, as well as heart valves, contribute to total impedance. Some, but not all, of these factors have been taken into account in consideration of the origin of cardiac sound.

A second cause of disagreement has been the fact that instrumentation frequently used for the recording of events of the cardiac cycle (for the purpose of time comparison) has been inadequate to the task. All recording systems contain some time delay in the registration of the observed physical event. Such time delays in any study of the origin of heart sounds assume major importance inasmuch as events of the cardiac cycle to which audible pressure phenomena have been attributed follow so rapidly upon one another, especially at the onset of ventricular contraction, as to fall within the time delay of many transducer-recorder systems. Instrumentation for the registration of cardiac sound is reasonably precise, but instrumentation used for the recording of functions against which sound has been compared (e.g., pressure) is notoriously deficient for this purpose. The recording of jugular and venous pulse tracings involves, quite apart from instrument delay, a delay in pulse wave transmission from the heart to the transducer site. Whenever events, occurring within 20 to 40 milliseconds of one another, are being compared, the instrumentation for their registration must introduce a time delay of not greater than a few milliseconds.

The purpose of this communication is to relate the application of suitable high frequency response instrumentation to the investigation of the time relationships of cardiac
transient sounds to pressure and flow events of the cardiac cycle.

**Methods**

Simultaneous recordings have been made of intravascular and intracardiac pressures, aortic blood flow and acceleration, the electrocardiogram, and intracardiac sound in healthy anesthetized dogs.

Intracardiac and aortic pressure were measured with the Dallons-Telco* variable inductance microtransducer, a catheter-tip manometer with a frequency response that has been demonstrated in this laboratory to have a relatively constant ratio of output to input amplitude to well over 500 cycles/sec. The maximum time lag which could be introduced by this transducer would be on the order of less than 2 milliseconds. Access to the left ventricle and left atrium was gained by retrograde passage from the left carotid artery. Aortic pressures were obtained with the catheter-manometer located fluoroscopically within 2 cm of the aortic valve.

Intracardiac "sound" was obtained from the Dallons-Telco amplifier by filtering frequencies below 40 cycles/sec from the pressure output and amplifying the result. This is an integral part of the apparatus as obtained from the company. These higher frequency pressure transients, altered in their passage through the tissues by damping, and by the natural frequencies of intervening structures, appear on the chest wall, and are translated by the auditory mechanism into recognizable sound. That such recording of intracardiac "sound" is comparable in time with phonocardiographically recorded sound transients has been demonstrated.6

Aortic blood flow was obtained from the root of the aorta with the Medicon K-2000† electro-magnetic flowmeter. The amplifier employs a 400 cycles/sec carrier wave frequency. Circuit computations reveal a maximum phase lag of 6 to 8 milliseconds when the amplifier is used in the unfiltered position. Following placement of the flowmeter probe, the chest was closed and the lung reinflated before any observations were made.

Electronic differentiation of aortic flow velocity was used to measure acceleration. Differentiation of left ventricular pressure was occasionally done to detect subtle changes in slope not discernible by inspection of the tracing. Time derivatives were obtained with the use of the Philbrick P-2 different operational amplifier* with a resistance-capacitance network chosen so that the accuracy of computation was in error by less than 5% of all frequencies out to 150 cycles/sec. The time lag introduced by this computation was less than 1 millisecond.

The electrocardiogram was monitored from a modified lead 1 with electrodes placed in the axillae. Care was taken to reduce the resistance between electrodes to less than 500 ohms. Where necessary, subcutaneous contact was made.

Some recordings were made on an Electronics-for-Medicine® electron beam photographic recorder, others on a Sanborn model 350‡ directwriter. In the latter case a 60 cycle filter was occasionally used in the recording of sound and flow. Since identical circuits were employed when these functions were being compared with one another, there was no time delay introduced into the recording of one that was not identically introduced into the recording of the other. It was therefore possible to make a valid time comparison of the events occurring in the two different parameters.

The above instrumentation is reasonably precise. In every situation, careful attention was paid to any possible time delay that could be introduced by the transducer, amplifiers, computing elements, filtering devices, or the recording equipment. Direct comparison of recorded events within the constraints of the known time delays is valid.

Once control observations were made, many of the animals were given infusions of isoproterenol (0.4 µg/ml at 2 to 3 ml/min) or methoxamine (40 µg/ml at 2 to 3 ml/min) to determine, respectively, the effects of high flow and high resistance on the comparative timing of events of the cardiac cycle.

**Results**

**FIRST HEART SOUND**

The first heart sound was found to contain two major components, often separated by a discernible silent period. The separation of the two components was a function of the duration of isometric ventricular contraction. The first major component was seen to occur simultaneously with the onset of isometric ventricular contraction and the occurrence of

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*Philbrick Researches, Inc., 127 Clarendon Street, Boston, Massachusetts.
†Electronics-for-Medicine, Inc., 30 Virginia Road, White Plains, New York.
‡Sanborn Instrument Company, Waltham, Massachusetts.
the left atrial "c-wave" (fig. 1). The form of the atrial "c-wave" was not always as classically described, but was frequently biphasic and, on occasion, even more complex. There occurred at this time a distinct positive, or biphasic, wave in the central aorta. This wave was invariably present, even in one animal in the face of atrial fibrillation (fig. 2) and, therefore, could not possibly have been due to atrial contraction as has often been thought.

The presence of simultaneous low frequency pressure waves in the aorta and left atrium together with the first component of the first heart sound led to an inquiry as to whether a similar low frequency event might not be present in the left ventricle, but obscured by the rapidly rising intraventricular pressure. Accordingly, the left ventricular pressure was differentiated (fig. 3), revealing the presence of a subtle change in slope in early isometric ventricular contraction. This was present in all cases. It is not discernible by mere inspection of the undifferentiated pressure trace, since its amplitude is sufficiently low, and its "frequency" sufficiently high, to be hidden within the width of the rapidly rising electron beam inscription.

The second major component of the first heart sound began concomitantly with the rise of aortic pressure, and the onset of aortic blood flow. It peaked with peak acceleration of aortic blood flow, and ended just prior to maximum instantaneous flow. A comparison of sound with central aortic flow and pres-
FIGURE 2
Central aortic and left atrial pressures in the presence of atrial fibrillation in the dog. Atrial “c-wave” and small aortic presystolic wave (arrow) persist. Recording conditions as in figure 1.

SECOND HEART SOUND
The second heart sound began at that time during systole when instantaneous flow, which had been gradually declining, suddenly began a rapid descent to below baseline. It thus began with the onset of the rapid deceleration phase, while forward flow was still going on. Comparison of sound with aortic flow and its derivative are shown in figure 6 under control conditions, under conditions of high flow induced by isoproterenol, and under conditions of low flow and increased peripheral resistance induced by methoxamine. In each case the sound began with the onset of rapid deceleration, and peaked synchronously with peak deceleration. Later components, usually lesser in magnitude, were timed with the short period of reversal of aortic blood flow as the aortic valves closed, and with deceleration of pulmonary arterial flow. The onset of the second heart sound occurred approximately 25 milliseconds before the nadir of aortic flow, the latter being the point at which the aortic valves presumably close (figs. 4, 5, and 6). Such a time interval is clearly outside the maximum time delay of sound, flow, or acceleration recording.

The fundamental components of the second heart sound here recorded are on the order of 30 to 60 cycles/sec. It should be remembered that filtering circuits discriminated against higher frequencies of sound which were, nevertheless, concomitantly present. It is obvious, however, that there is significant energy at this effective band-pass, and that much of
HEART SOUNDS AND ACCELERATION OF BLOOD FLOW

FIGURE 3

Demonstration of the subtle change in slope of the left ventricular pressure in early isometric contraction. Upper trace is a recording of left ventricular pressure. Lower trace is the first time derivative of the signal. Distinct notching near the peak of the derivative is evidence of the presence of higher frequencies of pressure not seen upon inspection of the rapidly changing left ventricular pressure. The notch represents roughly a maximum change in the steepness of the slope of 20%, with instability persisting for less than 20 milliseconds. It is not surprising, therefore, that such information is not readily seen in the undifferentiated trace.

this energy occurs before the end of forward aortic flow. Furthermore, the magnitude of this early sound transient is roughly proportional to the magnitude of flow.

DIASTOLIC HEART SOUNDS

Third and fourth heart sounds are rarely heard in adult man in the absence of pathology. Similarly, they could not be recorded from the central aorta of the dog under normal conditions of pressure and flow. The administration of methoxamine, however, resulted in the production of a fourth heart sound that appeared simultaneously with the left atrial "a-wave" and a low frequency pressure wave recordable from the central aorta (fig. 7). The onset of heart failure and elevation of left ventricular end diastolic pressure with methoxamine, alone or in combination with dextran infusion, resulted in the appearance of a protodiastolic third heart sound and a new diastolic wave appearing in the aorta at the time of rapid ventricular filling (fig. 8).

Discussion

In a previous communication from this laboratory, the concept was presented of the heart as a second order mechanical system to account for the presence of distinct diastolic low frequency pressure transients appearing in the central aorta. The heart represents a distributed mass of fluid-filled muscle rather loosely suspended from the great vessels within the sleeve of the pericardium. These ele-
ments of mass, elastance, and resistance are constantly changing as blood flows in and out of the system. Should parts of the system be underdamped, oscillations would occur in the presence of an applied force. Oscillatory displacement of the system would further result in transient counterforces of induced pressure within the fluid chambers. Such transients would be complex wave-forms with frequency and amplitude spectra that are a function of the mass at the instant of displacement, the "spring" of suspension, and the resistance offered by the myocardium, pericardium, and surrounding mediastinal structures. The lower frequencies might be seen superimposed upon otherwise smooth intracardiac pressure functions, recorded via sufficiently sensitive transducers. The higher frequencies, recordable from within the chambers, would be translated through surrounding tissues to the chest wall, identifiable as external sound.

A number of observations appear to fit the above concept. That the system is underdamped, and oscillates at moderate frequencies (less than 60 cycles/sec), is seen in tracings of slit- and electro-kymography.8-10 The response of such instruments discriminates against higher frequencies. That similar frequency oscillations are reflected in induced counterpressures within the chambers has been shown in this laboratory.7 Further, these oscillations are seen at expected times of force inputs: isometric ventricular contraction, atrial contraction, and rapid ventricular filling. That vibration of the heart at higher (audible) frequencies does indeed occur is witnessed by the fact that we hear sound at all from the heart. Whatever the source within the heart, vibrations of the chest wall imply

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**FIGURE 4**

Pressure, sound, and flow recorded from the central aorta. Sound and flow recorded with 60-cycle filter permitting only dominant frequencies of sound to appear. Second major component of the first heart sound is seen to occur during early ventricular ejection. The onset of the second sound occurs 30 milliseconds before the nadir of flow reversal is inscribed. Sanborn 350 direct-writer recording.
vibration of intervening structures, including blood and heart wall.

Rushmer\textsuperscript{1} emphasized the concept of acceleration and deceleration of blood flow as forces sufficient to induce sound. McKusick\textsuperscript{11} states: It is . . . “probably most accurate to think of sounds such as the first and second sound, and the mitral ‘opening snap,’ as hydrostatic pressure transients produced by the abrupt interruption of the momentum of local flow. . . . Whether abrupt acceleration, as with valve opening, can produce sound is less convincingly demonstrated, but clinical experience would suggest it can.” Thus, McKusick felt that most, if not all, sounds were the product of abrupt changes in local flow. The observations related here extend the above concept, and lend weight to it.

\textbf{FIRST HEART SOUND}

For many years two schools of thought existed, attributing the first heart sound, re-

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Sound compared with flow (bottom) and the derivative of flow (top), under control conditions, under conditions of high flow (isoproterenol) and under conditions of increased peripheral resistance (methoxamine). Bracketing in each panel identifies onset of rapid deceleration, and peak deceleration, of aortic blood flow. In each instance, these events time respectively with the onset and peak of the second heart sound. The second sound begins 25, 20 and 30 milliseconds before the nadir of flow reversal under the three respective conditions. Such an event cannot be accounted for by phase lags in the systems (see text). Note that later components of the first sound increase as absolute magnitude of acceleration increases with increased flow (isoproterenol), and decrease with decrease of acceleration with high peripheral resistance (methoxamine). The latter effect is striking. Recording conditions as in figure 4.

spectively, to muscular contraction of the ventricle, and to closure of the A-V valves. Any significant muscular contribution was, however, firmly excluded by the experiments of Dock. Orias and Braun-Menendez, supported by others, then proposed that the first heart sound was comprised of four components: (1) initial vibrations due to atrial contraction, (2) a major component due to closure of the A-V valves, (3) a major component due to opening of the semilunar valves, and (4) terminal vibrations, perhaps due to accelerating aortic flow.

In spite of much support for initial vibrations of atrial origin many authors disagreed, calling attention to the fact that such initial vibrations persist in the presence of atrial fibrillation. The dispute was resolved with the discovery that under unusual pathological circumstances (e.g., arterial hypertension and decompensated aortic stenosis) the fourth heart sound did indeed move into the first heart sound, but that under normal circumstances atrial contraction played no part in the origin of the first heart sound.

There is general agreement, then, that the first major component of the first heart sound, including initial vibrations, is a result of forces set in motion during isometric ventric-
Effect of the pressor agent, methoxamine, upon the aortic pressure trace, and upon sound. A fourth heart sound appears, coincident with a distinct wave in the aorta in late diastole (arrow) in the presence of augmented atrial contraction. Electronics-for-Medicine recording.

Effect of left ventricular failure upon the aortic pressure trace. Three distinct waves (a, b and c) are seen during diastole in the central aorta, timing respectively with rapid ventricular filling, atrial contraction, and isometric ventricular contraction. Corresponding heart sounds are numbered. Failure was induced by sustained infusion of norepinephrine following administration of chloralose in a large volume of fluid.
ular contraction, and a seemingly obvious result of valve closure. Great doubt, however, has been shed on a valve closure mechanism by the accumulating evidence\textsuperscript{26, 27} that the A-V valves have at this point in time been already closed by inertial forces resulting from atrial contraction. This mechanism, first proposed in 1912,\textsuperscript{28} is hemodynamically persuasive.

One is left then with the concept, alluded to by others,\textsuperscript{1, 11} that isometric ventricular contraction induces a ballooning of the opposed valve curtains toward the atria. As these curtains are stretched to their elastic limit, flow in this direction is suddenly decelerated. If the present hypothesis is correct, this would result in the first component of the first heart sound, as well as low frequency pressure transients in all chambers of the heart. Figure 1 shows such a wave in the left atrium (the “c-wave”) and in the aorta (the presystolic wave). Indeed, simultaneously occurring waves may be demonstrated in all chambers of the heart.\textsuperscript{7}

Even less agreement exists regarding the second major component of the first heart sound. Previous recording methods have not clearly revealed whether the second major component occurred before, during, or after the instant of aortic valve opening. Thus, it has been thought by some to be due to valve opening,\textsuperscript{14–16, 29} by others to be due to systolic ejection,\textsuperscript{30} and by still others to be due to asynchronous closure of the A-V valves.\textsuperscript{31–38}

Results presented here show clearly that the second major component of the first heart sound occurs at the time of ejection of blood from the ventricle, a time too late to be accounted for by any asynchronous closure mechanism. Although there may be asynchronism of mitral and tricuspid valve closure, the time difference is probably so slight that both contribute to the first major component.

The widening of the gap between first and second components in bundle branch block. In fact, any process lengthening isometric ventricular contraction would be expected to produce a widening and splitting of the two components of the first sound.

That aortic flow acceleration bears a direct time correspondence to the second component of the first sound is clear from figures 4 and 5. Careful inspection of figure 6 shows that, as the magnitude of acceleration decreases in the face of high peripheral resistance (secondary to methoxamine infusion), the magnitude of the second major component of the first heart sound decreases accordingly. The reverse obtains with isoproterenol. Acceleration is a function of change in force, and greater acceleration implies increased force. This force would therefore be expected to be associated with a proportional increase in pressure transients in all frequency ranges. Acceleration of pulmonary arterial flow may contribute to the sound as well, but, since the duration of right ventricular ejection is longer, acceleration of flow in the pulmonary artery need be much less than that in the aorta. Such low pressure chambers are low energy producing and, consequently, must have much less influence on sound than would the high energy systemic chambers.

SECOND HEART SOUND

There has been no disagreement with the concept of the origin of the second heart sound. It has been thought to be due to asynchronous closure of the semilunar valves, aortic closure preceding pulmonic closure under normal conditions.\textsuperscript{37, 38} This has been reinforced by the clinical observation that high flow in either circuit delays closure of the respective valve and, correspondingly, widens or narrows the gap between the two components. Some observers have felt that terminal vibrations may be due to tricuspid and mitral valve opening.\textsuperscript{39}

The precise physical mechanism for the occurrence of the second sound was thought by Rushmer\textsuperscript{1} to be due to impedance to back flow as the semilunar valves abruptly closed. McKusick\textsuperscript{11} has agreed with this concept.

If the reversal of aortic and pulmonic flow
may be invoked to account for the origin of the second heart sound, then by similar argument forward deceleration should result in the production of sound. Indeed, the magnitude of forward deceleration (and associated force) is considerably greater than that of reverse deceleration, the mass of blood acting over a longer distance and at higher velocity. The evidence presented here shows clearly that the second sound begins at the onset of the phase of rapid deceleration of forward aortic flow. Furthermore, the first major component peaks with peak deceleration and ends with the nadir of flow reversal. This would appear to be strong evidence for a causal relationship, and is consistent with physical considerations. The remaining component is probably due to similar behavior of pulmonic flow.

That the onset of the sound earlier than has been thought is not due to artifact produced by the presence of the flowmeter probe is indicated by the consistent relationship of the sound to aortic pressure with or without the probe in place. In two dogs, simultaneous measurement of aortic and pulmonic pressures with the probe in place has confirmed that aortic valve closure preceded pulmonic valve closure in these preparations.

**DIASTOLIC HEART SOUNDS**

Under normal conditions of pressure, flow, and volume within the cardiac chambers, impedance to flow from atrium to ventricle may not result in sufficiently rapid deceleration to produce significant high frequency oscillations. This would certainly be true should ventricular relaxation be an active process, literally "sucking" blood into the ventricles. However, increased end diastolic volume and/or pressure, or disease restricting left ventricular filling (e.g., constrictive pericarditis), could result in augmentation (or change in character) of impedance to flow, giving rise to forces quite capable of displacing the system sufficiently to result in resonant motion. Further, the primary alteration of the volume-elastic characteristic in disease is a loss of compliance. Such loss of damping must predispose to ringing as a result of whatever increased forces are present.

As demonstrated here, left ventricular failure in the dog gave rise to both a third heart sound and an associated low frequency pressure transient within the ascending aorta. Indeed, the entire concept (and the evidence shown here) would appear to be supported by demonstrations of Lewis, Lewis and Dock, and Brady and Taubman that most normal adults have smooth outward motion of the ventricular border during early diastole, while children and patients with protodiastolic gallops demonstrate a sharp inflection with notching, as recorded by kymograms. This is certainly suggestive evidence for oscillation of the system, resulting in the observed pressure transients, including sound.

The etiology of the fourth heart sound may be similarly argued. The atrium and ventricle during atrial contraction act as a single chamber. Under the influence of a pressure gradient, blood flows from the atrium into an already distended ventricle. As the elastic limit of the musculature is reached, the rate of flow will be suddenly changed, the deceleration again resulting in an oscillation of all chambers and a coincident pressure transient. Both Kincaid-Smith and Barlow, and Weitzman have agreed that, although the appellation "atrial sound" is frequently attached to this phenomenon, it probably originates in the ventricle as a filling sound. More to the point, it originates from all participating resonant chambers.

Although the present communication contributes no evidence regarding the "opening snap" or the systolic ejection click, there is no reason to believe their origin is not on the same basis. The concept of valvular "snapping" in mitral stenosis is seriously damaged by the realization that "opening snaps" are commonly heard in left atrial myxoma. The hemodynamics of the two diseases are identical. Blood enters the left ventricle in early diastole at high velocity under a very large pressure gradient, and is impeded in the one instance by the elastic limit of the deformed mitral valve, in the other instance by mechanical
obstruction by the atrial tumor. Indeed, the authors believe that all transient cardiovascular sound has the same principle of origin.

The hypothesis presented here is consistent with the physics of sound production, and the observed behavior of the cardiovascular system. To say that valve closure is responsible for the origin of sound is to stop short of the essential point. Valve closure does indeed result in the production of sound, but only by providing a barrier to local flow. Furthermore, the concentration on valvular mechanisms as manufacturers of sound forces one to seek explanations for other sounds (third and fourth heart sounds) that are almost certainly not valvular in origin. Abrupt changes in momentum of flow, i.e., acceleration and deceleration of flow velocity, must set the heart in motion. This oscillatory motion is associated with a spectrum of induced pressure frequencies that will continue until the system is brought to rest through damping. Should one doubt that acceleration and deceleration of flowing fluids can be responsible for sound production, he need only enter an old house, and abruptly turn on and off the water faucets. The resultant noise can be impressive.

Summary
A concept has been presented that abrupt acceleration or deceleration of blood flow is associated with an energy source sufficient to displace the mass of the heart, and that, consequently, this mass will oscillate at a sum of frequencies that are a function of chamber mass and restoring forces. Induced pressure transients may be observed in all chambers, the lower frequencies appearing on conventional pressure tracings, the higher frequencies as intracardiac sound.

Using high frequency response instrumentation in dogs, intravascular sound has been compared with pressure and flow events throughout the cardiac cycle.

(1) The first component of the first heart sound occurs during early isometric ventricular contraction and is associated with lower frequency pressure transients that may be recorded from all chambers of the heart. The left atrial "c-wave" is one of these transients.

(2) The second component of the first heart sound occurs during ventricular ejection, and appears to be a function of acceleration of aortic blood flow.

(3) The second heart sound begins significantly before aortic valve closure, while forward flow is still going on, and is proportional to the magnitude of deceleration of blood flow.

(4) The third heart sound appears only in the presence of left ventricular failure at the time of rapid ventricular filling, and is associated with a low frequency wave recordable from the central aorta. The production of these transients is presumed to be deceleration of inflow as the limits of ventricular relaxation are reached.

(5) A fourth sound arises coincident with pressure transients in other chambers as atrial systole further distends the left ventricle.

It is proposed that acceleration and deceleration of blood flow is a sufficient and necessary condition for the origin of cardiovascular sound transients.

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