Special Articles

The First Heart Sound in Normal and Pathological Conditions

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Summary

Considerations of the physical basis of cardiac contraction and sound generation explain the mechanism of the first sound. Older theories examining this sound as the result of valve closure or stiffening are refuted. It has been demonstrated that the normal first sound originates in the left ventricle alone and that accelerations and decelerations, "timed" by mitral and aortic valves events, are its cause. Three components have been recognized in the first sound: *a* occurs when the left ventricular wall and septum have reached a certain tension; *b* when the aortic valve opens; *c* when the peak of the aortic pulse has been reached. The ventricular septum is an integral and essential part of the left ventricle. In *left bundle branch block,* abnormal activation of the septum transforms this into a passive structure resulting in a slower rise of left ventricular pressure and a longer isovolumic period. This causes a small and delayed first sound, whose components, however, are still separated by normal intervals. In *right bundle branch block*, the first sound has a normal amplitude and its components are separated by normal intervals. If there is a larger late component, it is a *c* component, similar to that of normal elderly subjects. A larger *c* component may also be found in *atrial septal defect.* The *cannon sound* of AV block is caused by more rapid deceleration due to higher atrial pressure at the onset of ventricular contraction resulting in intense vibrations. The first sound of *arrhythmias* varies in the different conditions and even in different subjects, due to the effect of several variable factors. Elevated left atrial pressure, stiffening of the mitral valve in *mitral stenosis*, causes a slow onset and a more rapid rise of LV pressure. This results in a delayed, but larger, first sound. The action of catecholamines on the myocardium dramatically increases the first sound. The latter

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can be considered as an *index of contractility* and may be of great interest during stress tests.

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Pressure Gradients and Cardiac Vibrations

Most of the energy involved in the heart beat results in the creation of *pressure gradients* in the chambers of the heart and great vessels. These gradients are responsible for motions of cardiac structures, progression of blood in the vascular trees and generation of mechanical vibrations. The *cardiac vibrations* are the result of energy applied to a system and are a single phenomenon regardless of their frequency and amplitude. Although there is general agreement that the lower frequencies express cardiac dynamics resulting in displacement, velocity, and acceleration, controversies have arisen in regard to the role of the cardiac valves in the production of high frequency vibrations (i.e. heart sounds). However, important findings published in the last 35 years allow specification of the valves-sound relation. Some early investigators believed that sounds were produced by forceful closure of the valves, while later researchers demonstrated that valve closure was silent. This implied that " valve tension ", which immediately follows closure, is the cause of sound (a better term is *valve stiffening*).

Findings that refuted a role of valve closure in sound generation were reported by Edler et al,[1] Pohost et al,[2] Tsakiris et al,[31] and Wexler et al[3] for the mitral valve. They showed that the leaflets of the mitral valve do *not* close uniformly and simultaneously. Often one leaflet closes slightly before the other, and even different parts of the same leaflet may close at different times. Another important finding, observed by us since 1961[4]-[6] and confirmed by Wexler et al,[3] is that sound occurs only when the ventricular walls and the septum have reached a certain degree of tension as a result of ventricular contraction. The threshold level appears to be 1/5 to 1/3 of the total pressure rise (Fig. 1).

First Heart Sound and Mitral Valve

The cardiac valves " time " the onset of the first sound with their closure and opening; they also " time " the onset and changes of acceleration and deceleration that occur during the phases of ventricular contraction and

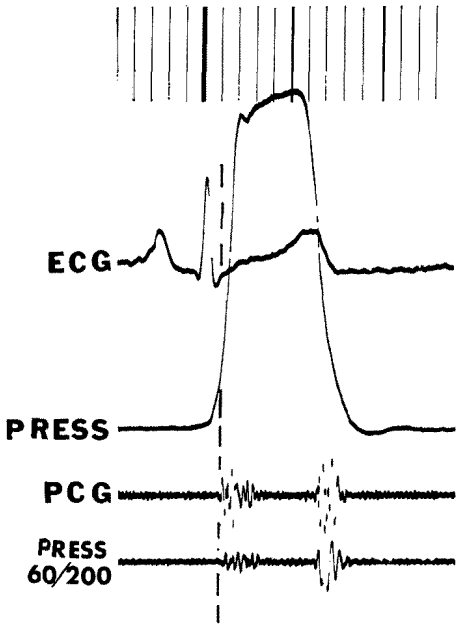


Fig. 1. Left ventricular pressure (PRESS) of an anesthetized dog, recorded with a catheter-tip manometer; filtered pressure (PRESS 60–200); and external phonocardiogram (PCG) at the apex (nominal frequency of 100 Hz; 24 db/octave slope). There is a close similarity between the filtered pressure and the external PCG. Both tracings show three components within the first sound. The first component occurs at a time that the LV pressure tracing is already fairly high (broken line). There is a slight electronic delay in the filtered pressure tracing.

relaxation. Time-coincidence is not sufficient to establish a cause-effect relation, and a more accurate analysis for the dynamic system is necessary. Such analysis was done by Mac Canon et al,[7] who studied the relation between the *energy* of the first heart sound, the *motion* of the mitral valve, and the *mass* of the vibrating valve structures. Their conclusion was that *not more than one tenth of the energy could be due to the mitral valve.* This suggests that the mitral complex has a minor role in the generation of sound and excludes either closure or stiffening of this complex as the major cause of the first sound.

The pressure gradients between the heart chambers and vessels cause the opening and closure of the valves. The lack of coincidence between pressure crossover and final closure of the mitral valve should not be interpreted as contradicting this statement because the delay is the result of the inertia of both the blood and the valve structures.

In 1948, we recorded two larger vibrations within the first heart sound, termed *a* and *b*.[8] In 1954, Leatham again recorded two larger vibrations within the first sound, termed " mitral " and " tricuspid ".[9] After our first

study on the first sound, Shah et al in our laboratory identified three components of the first sound, designated *a*, *b*, and *c*.[10] When valvular theory was generally accepted, the first component (a) was attributed to the mitral valve (either closure or stiffening). Component *b*, then, was attributed to either the opening of the aortic valve[8] or the closure of the tricuspid valve.[9] This application of the "valve closure" theory to component *b* was suggested,[9] on the basis of the occasional coincidence of this component with echo evidence of tricuspid closure. However, studies of the timing of aortic valve opening with an electric contact[11),20] did not support this concept (Fig. 2). Moreover, bypass of the right heart did not alter the first sound (Fig.

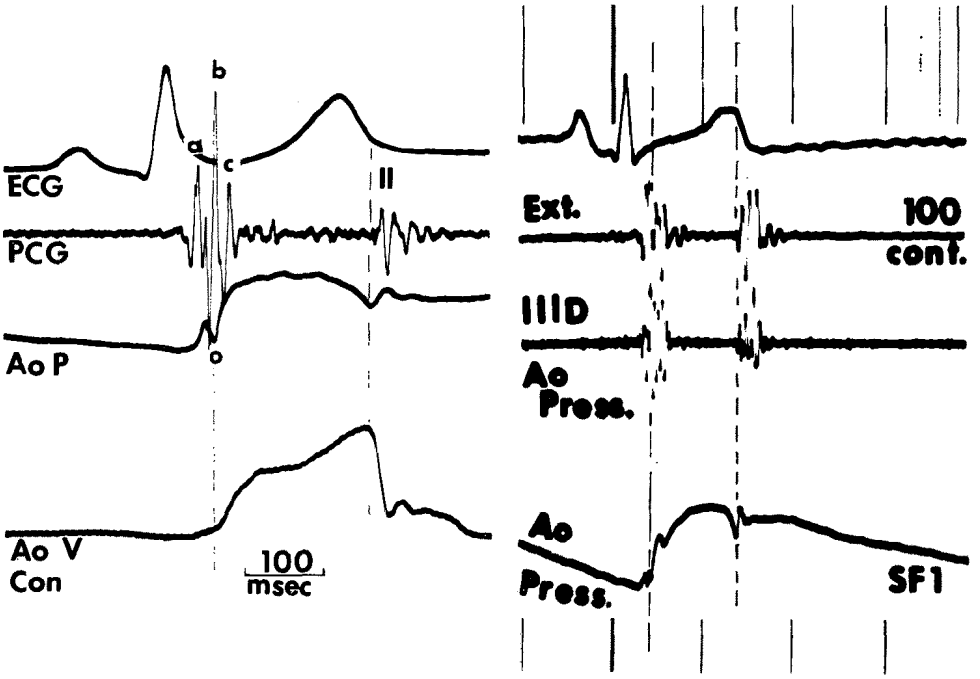


Fig. 2. Experiment in a dog. A (left): From above: electrocardiogram (ECG); external phonocardiogram (PCG); aortic pressure, recorded with a catheter-tip manometer (AoP); and opening and closure of the aortic valve, recorded by means of an electric contact device introduced into the valve (AoVCon). Tracing recorded at 200 mm/sec. The aortic pressure tracing seems to have an anacrotic notch. This is simulated by the fact that the isovolumic tension is slightly pushing the cusps (a fact already known). Opening of the valve (o) occurs later, as demonstrated by the contact, and coincides with component *b* of the first sound in the external phonocardiogram (PCG). B (right): Recording of aortic pressure (lowest tracing) and third derivative of the same (IIID–Ao Press.). The latter is practically identical with the external phonocardiogram (Ext.) recorded with a high pass filter with nominal frequency of 100 Hz.

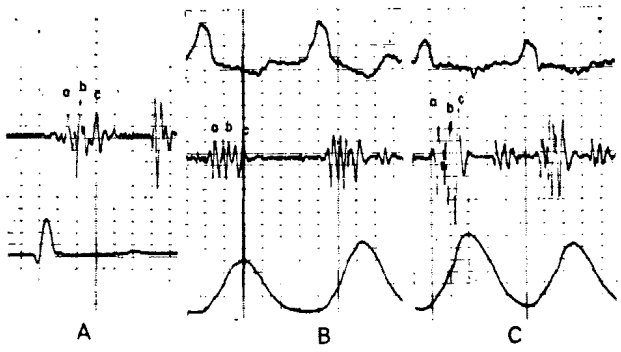


Fig. 3. Experiment in a dog with right heart bypass. (A) Heart sounds recorded at the apex with closed chest. (B, C) Heart sounds recorded within the left ventricle after opening the chest and performing the bypass. The lowest tracing is a left ventricular pressure tracing. The three components of the first sound (a, b, c) are present before and after the bypass (from Luisada et al, 1967, courtesy of Circulation).

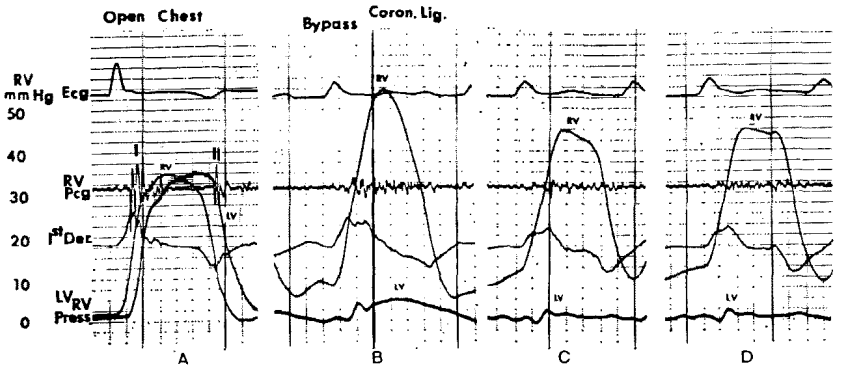


Fig. 4. Records of tracings obtained in an open chest dog. (A) Control. Right and left ventricular pressures recorded with regular catheters (LV pressure is recorded with lower amplification so that it is equal to RV pressure). Right ventricular sounds are obtained with a Dallons-Telco micromanometer. Medium tracing is a dp/dt of right ventricle; lowest tracing is a dp/dt of left ventricle. Between A and B, *a left heart bypass and a circumflex coronary ligation* have been performed. (C, D) While the right ventricle is strongly beating, the first heart sound gradually disappears in that chamber when it disappears in the left ventricle (from Luisada et al, 1968, courtesy of J Appl Physiol).

3), while bypass of the left heart, when the right ventricle was beating strongly, completely abolished the first heart sound[11] (Fig. 4). Prakash et al[12] documented a complete echo coincidence between the *b* component and the onset of opening of the aortic valve, while we demonstrated that the *b* component coincides with the onset of pressure rise in the aorta, recorded with a

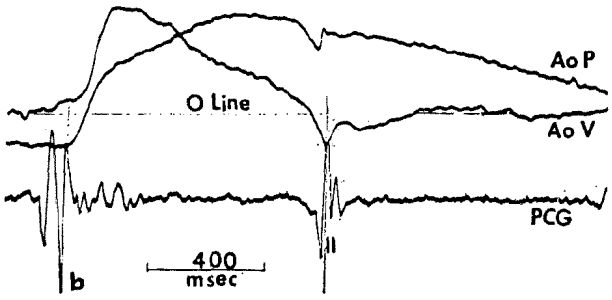


Fig. 5. Experiment in a dog. From above: aortic pressure recorded with a catheter-tip manometer (AoP); aortic flow velocity recorded with an intra-aortic probe (AoV); external phonocardiogram (PCG). Tracing recorded at 400 mm/sec.

catheter-tip manometer just above the aortic valve[13),20)] (Fig. 5). Thus, our original concept was confirmed.

First Heart Sound and Cardiac Dynamics

A cause-effect relationship between cardiac dynamics and the first heart sound was suggested first by Wiggers[14)] and advocated subsequently by Rushmer.[15)] In particular, the latter investigator proposed that accelerations and decelerations of the cardiohemic system would generate the heart sounds. Our subsequent studies have expanded this concept by demonstrating that the left ventricle is the sole cause of the normal first sound, and that the isovolumic and ejection phases of left ventricular contraction, "timed" by mitral closure and aortic opening, cause acceleration of the blood and deceleration of the cardio-aortic structures, followed by a reversal of the process. The contributions of these mechanisms to the first heart sound were initially studied by van Bogaert et al,[16)] Laurens,[17)] Piemme et al,[18)] and Guenther,[19)] and were finally confirmed from intracardiac recordings of accelerations and decelerations[20)] (Figs. 1, 6). It is noteworthy that: (1) component *a* is influenced markedly by the characteristics of left ventricular contraction and by the left ventriculo-atrial gradient at the beginning of contraction; (2) component *b* is influenced by the left ventricular-aortic gradient; and (3) component *c* is influenced by the functional and anatomical conditions of the aortic wall.

Intracardiac and Chest Sounds

A final point in establishing a cause-effect relation between heart dynamics and heart sounds is supplied by mathematical analysis. Different investigators have documented a linear relation between the phonocardiogram

recorded on the chest surface and the left intraventricular pressure tracing, the slope being a function of myocardial stiffness (Laurens,[17] Agress et al,[21] Cassot et al,[22] van Vollenhoven et al[23]). The phonocardiographic signal is

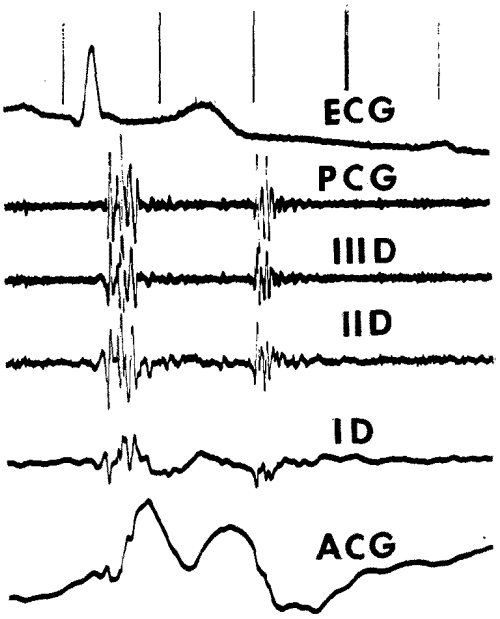


Fig. 6. Tracings recorded in a normal 24-year-old man. The external PCG is recorded at the apex (nominal frequency of 100 Hz; 24 db/octave slope); the apex cardiogram (ACG) is recorded together with the first, second, and third derivatives (1D, IID, IIID). Note the identical appearance of the first and second sounds in the PCG and the third derivative (rate of acceleration) of the ACG.

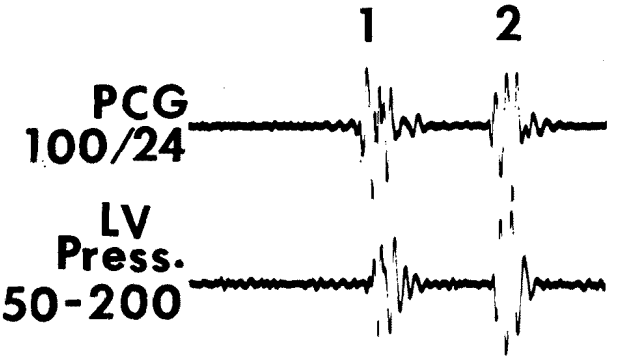


Fig. 7. Comparison between phonocardiogram on the chest (filter at normal frequency 100 Hz with slope of 24 db/octave) and left ventricular pressure, whose electric output is filtered through a band pass filter 50–200 Hz (slopes 24 db/octave).

proportional to the acceleration of the outer wall of the heart muscle, which is proportional to the changes of intraventricular pressure. As a result, it was possible to convert the LV pressure into a theoretical thoracic acceleration tracing that was almost identical to the phonocardiogram recorded on the chest surface, both in normal and clinical conditions.

Working along similar lines, we have recorded the third derivative (rate of acceleration) of both the left ventricular pressure and the apex cardiogram.[10),13)] In both cases, we obtained tracings that were similar to the external phonocardiograms: the first heart sound had three components, similar to those previously recorded intracardially by Shah et al[10)] (Fig. 1). Similar results were obtained by filtering of either LV pressure (Fig. 6) or apex cardiogram (Fig. 7).

Function of the Ventricular Septum

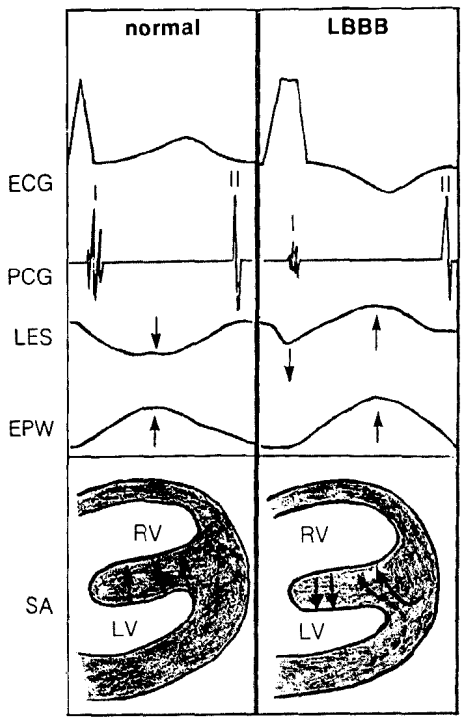
With rare exceptions, *the interventricular septum is an integral part of the left ventricle.* In normal hearts, 90% of this structure contracts at the same time as the free ventricular wall and is an essential element for the rapid rise of pressure that occurs in the left ventricle during early systole (Fig. 8). Weber et al[30)] has shown that the septum is an important element of left ventricular dynamics in animal experiments.

Left Bundle Branch Block

In *left bundle branch block*, the septum has an abnormal motion (demonstrated by echocardiographic studies), which is evidence that it behaves like a passive structure (Fig. 8). This behavior, due to abnormal activation of the septum, allows the pressure rise caused by contraction of the free wall to push the septum anteriorly. This process decreases the rapidity of the pressure rise and prolongs the isovolumic contraction period (Adolph et al[24)]). It also *delays* and *decreases the amplitude of the first sound*, but *intercomponent intervals* are unaffected[25)-29)] (Fig. 9).

Right Bundle Branch Block

In *right bundle branch block*, the three main components of the first sound are again separated by *normal intervals.*[25),28),29)] In several cases, chiefly older subjects, the *c* component, which occurs well *after* initiation of right ventricular contraction, is larger.[25),28),29)] This feature, due to senile changes of the aorta, is responsible for the old statement about " wide splitting of the first sound in right bundle branch block ", since the *c* component was thought to be a *b* component.



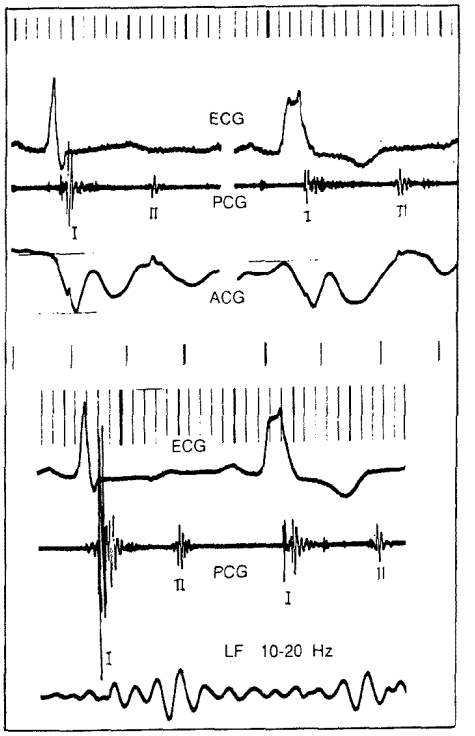


Fig. 8 (left). Scheme of septal motion and septal activation in normal heart and in left bundle branch block. From above: electrocardiogram (ECG), phonocardiogram (PCG), echocardiographic motion of the left endocardial surface of the ventricular septum (LES), and motion of the endocardial surface of the posterior wall of the left ventricle (EPW). They are correlated with a scheme that reproduces the depolarization of the septum (SA) in the normal heart (left) and in left bundle branch block (right). (Scheme drawn according to a description of Sodi-Pallares relative to depolarization of the septum.)

Fig. 9 (right). Tracings obtained in an 84-year-old patient with rate-related LBBB. Note the decrease of apical impulse (ACG), of the low frequency tracing (LF), and of the first sound (I) with the block.

Atrioventricular Block

The *timing and amplitude of the first high frequency component (a) of the first sound* are a function of the left ventriculo-atrial pressure gradient. The higher the left atrial pressure, the larger is this component.[32)-35)] A louder sound is caused by more rapid deceleration, resulting in more intense vibrations of the heart walls, the mitral valve complex, and the blood. This phenomenon has been attributed to the position and motion of the mitral leaflets when left atrial pressure is high. However, the loudest (cannon) sound in complete

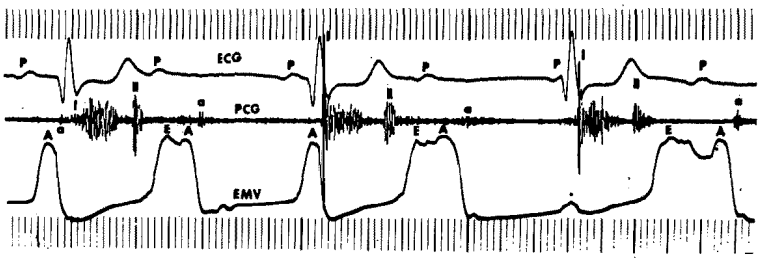


Fig. 10. Recording from a 91-year-old woman with complete atrioventricular block. The lowest tracing (EMV) is an echocardiogram of the anterior leaflet of the mitral valve (A mode). The first complex has a PR interval of 0.26 and a large *a* wave of the echo; despite large valve motion, the first sound is of minimal amplitude. The second complex has a PR interval of 0.16. A large *a* wave in the EMV is followed by closure of the valve; amplitude and rate of closure are similar to those of the previous cycle but the phonocardiogram shows a *cannon sound*. The third complex has a PR of only 0.08. Again, a *cannon sound* is seen but the previously closed valve shows only minimal reopening. Thus, 3 cycles show different dynamic events with no correlation with valve motion.

AV block may occur with a nearly or completely closed mitral valve[35] (Fig. 10). Therefore, the resistance of the valve, supported by high atrial pressure, is essential for the more rapid deceleration that causes a cannon sound in certain cycles.[35),36] However, a high atrial pressure at the onset of ventricular contraction may cause a *cannon sound*, even if the valve is initially open and then closes rapidly (the last occurrence is not always present).

Atrial Septal Defect

It has been claimed that a characteristic first sound exists in *atrial septal defect (ASD)* as a result of a larger " tricuspid component ". We have studied the electro- and phonocardiographic tracings, as well as the intracardiac pressure tracings of numerous patients with a *secundum* type of atrial septal defect.[41),42] These examinations indicated that the three high frequency components of the first sound are separated by normal intervals in ASD. Furthermore, the second high frequency component is *not* related to the tricuspid valve, as proven by comparison of the sound tracing with right ventricular pressure tracings and their first derivatives.[41]

Arrhythmias

The amplitude of the first component of the first sound in arrhythmias is a peculiar phenomenon that has been the object of numerous studies. Both in *ectopic beats*[37] and in *atrial fibrillation*,[38] several dynamic factors acting on

the causes of the first component are involved so that the behavior of the latter is not the same in all patients having a similar type of arrhythmia.

Mitral Stenosis

In *mitral stenosis*, our studies demonstrated that the left ventricular pressure rises slowly at first, then increases rapidly. The first high frequency component of the first sound is delayed when the left atrial pressure is higher than in normal subjects,[39] and there is a parallel increase of the left atrial pressure and of the first sound amplitude.[40] We have concluded that the increased left atrial pressure, by stiffening the mitral valve, causes a more rapid rise of LV pressure after closure of the valve. This results in a louder sound that is similar to the cannon sound of AV block. Finally, a recent study by Hada et al[43] has failed to observe that the tricuspid closure precedes mitral closure in mitral stenosis, as stated in the past.

First Sound and Sympathetic Stimulation

The amplitude of the first component of the first sound is also a function of *the rapidity of left ventricular contraction*, which can be influenced by sympathomimetic hormones, reflexes, and drugs. This feature is revealed by the left ventricular dp/dt (first derivative), and even better by the second and third derivatives in animals[44] (Figs. 11, 12). It has also been identified in normal subjects during *stress tests*,[45],[46] procedures that cause a marked increase of catecholamines in the blood. It also confirms the importance of the

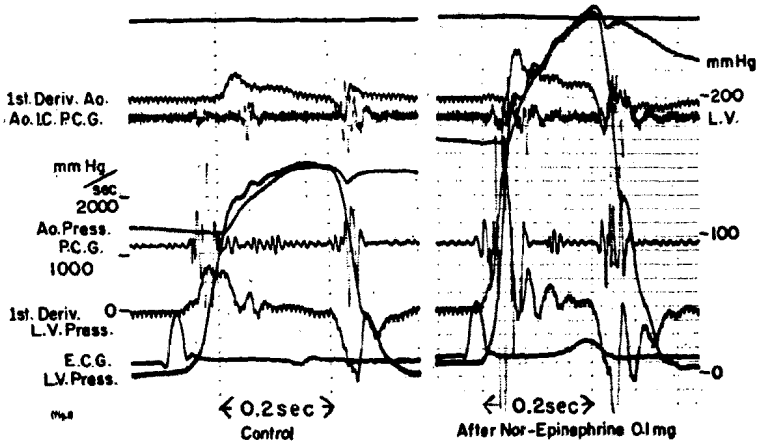


Fig. 11. Effect of l-norepinephrine in a dog showing a parallel increase of the early systolic wave of the first derivative (dp/dt) and the first heart sound amplitude together with an increase of left ventricular pressure (from Sakamoto et al, 1965, courtesy of the authors and Circ Res).

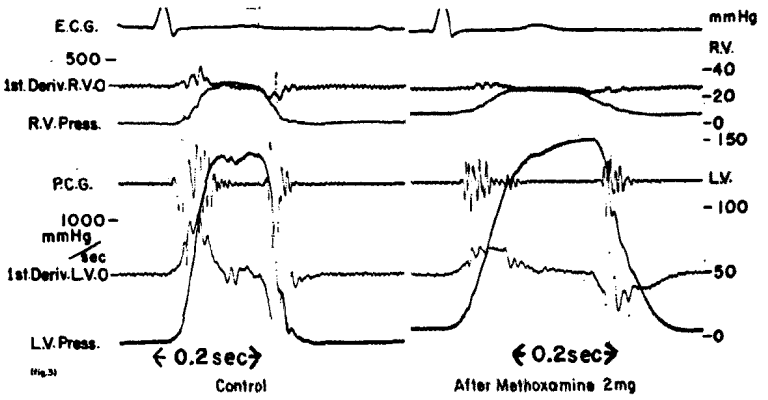


Fig. 12. Effect of methoxamine in a dog showing a parallel decrease of the early systolic wave of the first derivative (dp/dt) of left ventricular pressure and of the first heart sound amplitude in spite of some increase of left ventricular pressure (from Sakamoto et al, 1965, courtesy of the authors and Circ Res). Methoxamine contracts the peripheral arteries (increase of afterload) but does not increase the contractility of the left ventricle. In this phase of the experiment, a slight left ventricular failure is likely.

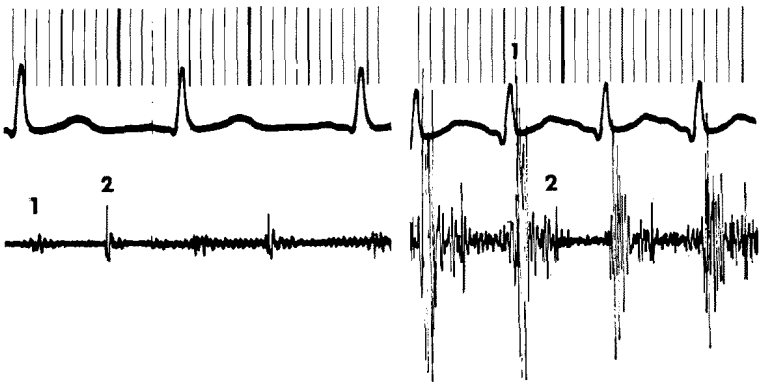


Fig. 13. Behavior of the first heart sound during a stress test on a treadmill. Left: control, right: after exercise.

isovolumic period for the amplitude of the first heart sound. In conclusion, this sound is a good *index of left ventricular contractility* and is valuable if studied during brief time spans, like in *anesthesia* or in *stress testing*[46] (Fig. 13).

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