Investigation of the Theory and Mechanism of the Origin of the Second Heart Sound

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SUMMARY To investigate further the origin of the second heart sound we studied human subjects, dogs, and a model in vitro of the cardiovascular system. Intra-arterial sound, pressure, and, where possible, flow and high speed cine (2,000 frames/sec) were utilized. The closure sound of the semilunar valves was of higher amplitude in the ventricles than in their respective arterial cavities. The direction of inscription of the main components of intra-arterial sound were opposite in direction to the components of intraventricular sound. Notches, representative of pressure increments, were noted on the ventricular pressure tracings and were coincident with the components of sound. The amplitude of the closure sound varied with diastolic pressure, but remained unchanged with augmentation of forward and retrograde aortic flow. Cines showed second sound to begin after complete valvular closure, and average leaflet closure rate was constant regardless of pressure. Hence, the semilunar valves, when closed, act as an elastic membrane and, when set into motion, produce transient pressure changes indicative of sound. The magnitude of the initial stretch is related to the differential pressure between the arterial and ventricular chambers. Sound transients which follow the major components of the second sound appear to be caused by the continuing stretch and recoil of the leaflets. Clinically unexplained findings such as the reduced or absent second sound in calcific aortic stenosis and its paradoxical presence in congenital aortic stenosis may be explained by those observations.

Although it is generally agreed that the second heart sound is related to closure of the semilunar valves, controversy remains as to the exact mechanism. In 1915 Wiggers suggested that there is silent approximation of the semilunar valves, and that aftervibrations of the closed valves and the column of blood produce the second sound. Studies utilizing an electrical conduction device showed that aortic coaptation occurs at least 5-13 msec before the sharp apex of the aortic incisura, and studies utilizing echocardiography showed that the aortic component of the second sound occurs at or slightly after coaptation. These observations support the work of Wiggers. Others have suggested that the main vibrations of the second sound are related to the magnitude of deceleration of blood during the latter part of ventricular systole. Some attribute the second sound to vibration of the leaflets caused by the snapping together of the corpora arantii. Rushmer related the origin of the second sound to vibrations of the valvular cusps initiated by abrupt deceleration of backward-moving blood following ejection. In view of these conflicting theories, our study was undertaken to further explore the mechanism of the production of the second heart sound. The scope of this investigation is related entirely to the origin of the second sound, and does not relate to the method of sound radiation in the cardiovascular system.

Methods

Simultaneous recordings of pressure and intra-arterial sound were obtained in 10 human subjects with no evidence of valvular disease, and in one with a Björk-Shiley tilting disk aortic valvular prosthesis. The study was approved by the appropriate human experimentation committees, and informed consent was obtained from each subject studied. Valvular disease in these 10 subjects was excluded on the basis of absence of murmurs at the chest wall and absence of a pressure gradient across the aortic valve. Pressure and sound-pressure fluctuations (which we refer to as intravascular sound) were measured with a single catheter-tip transducer in conjunction with a TCB-100 control unit which permits the recording of sound and pressure from the same transducer (Millar Instruments). The frequency response of the pressure sensor was essentially flat from 0 Hz to 10 kHz in the pressure mode. The audio mode low frequency cutoff, when used with 10-MΩ loading, was 6 dB at 40 Hz, 3 dB at 50 Hz, and flat above 90 Hz. The phase lag of the single-gauge transducer was 90° at 35 kHz, which is equivalent to a time delay of approximately 7 μsec. Tests showed no artifacts or apparent sound production by snapping or tapping the body of the catheter or motion of the tip of the transducer. Tests
with a multifunction pressure generator (Millar Instruments), and with this particular catheter-tip sound transducer and recording system, indicated that compression was registered as a downward deflection on the sound tracing, and expansion was registered as an upward deflection on the sound tracing.

Recordings of sound and pressure were obtained just above the aortic and pulmonic valves and within the ventricles. Data were recorded at various paper speeds ranging between 50 and 200 mm/sec, utilizing a photographic recorder (Electronics for Medicine). All recordings were made on DC amplifiers of the same recorder, thereby eliminating relative time lags of various signals due to the recorder.

Recording techniques identical to those used for human subjects were used for four dogs anesthetized with sodium pentobarbital. Elevation of systemic pressure was induced by an infusion of angiotensin II amide, and transient reductions in blood pressure were induced by a rapid intravenous injection of nitroglycerin. The behavior of the closure sound of both semilunar valves following these alterations was noted. Two additional dogs were studied with the chest open. In these dogs a cuff flow transducer connected to a BL-610 pulsed logic electromagnetic flowmeter (Biotronex) was placed around the root of the aorta. The time lag of the flow transducer is estimated to be 1–2 msec at 100 Hz. The transducer was placed approximately 1–2 cm distal to the aortic valve, and signals during diastole included coronary flow. A catheter-tip pressure-sound transducer was positioned within the lumen of the flow probe. The relation with respect to time of the second heart sound, pressure, and flow were documented.

Studies also were performed in an in vitro flow system (Fig. 1) which consisted of a rigid plexiglass model of the left ventricle and root of the aorta in which various types of valves can be attached and in which pressures and flows can be adjusted to correspond to physiological values. Pressure and sound were measured with Millar catheter-tip transducers introduced through pressure ports and positioned 3 cm from either side of the sewing ring of the valve. The tips of the transducers were fixed in the stream

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**Figure 1** Diagram of in vitro flow system. Bottom shows ventricle, aortic valve, and aorta with pressure ports which accommodate catheter-tip pressure-sound transducers. Cameras were positioned to record motion of valve and dual beam oscilloscopic display. Valve and oscilloscope as viewed by camera are shown at the top. Magnitude of systolic pressure was controlled by varying the fluid level in the compression chamber. The level of diastolic pressure was controlled by varying the adjustable clamp proximal to the compression chamber. The laminar resistor (synthetic sponges enclosed within a plexiglass chamber) and the corrugated rubber tubing permitted control of the resistance and compliance of the simulated arterial system. The size of the laminar resistor and the length of the corrugated tubing were adjusted with the stented porcine valve to achieve configurations of pressure and flow which were comparable to those seen in subjects.
of flow and were not in contact with the wall of the chamber or valve. Flow was measured with a cannulating transducer 19 mm in diameter attached to a model 501 square wave electromagnetic flowmeter (Carolina Medical Electronics). The time lag at 100 Hz was 6 msec. The magnitude of systolic pressure was controlled by varying the fluid level in the compression chamber. The level of diastolic pressure was controlled by varying the adjustable clamp proximal to the compression chamber. The laminar resistor (synthetic sponges enclosed within a plexiglass chamber) and the corrugated rubber tubing permitted control of the resistance and compliance of the simulated arterial system. The size of the laminar resistor and the length of the corrugated tubing were adjusted to achieve configurations of pressure and flow which were comparable to those seen in human subjects.

Simultaneous recordings of pressure, sound, and flow were obtained in the simulated root of the aorta and simulated ventricle in the presence of both a normal porcine aortic valve (Hancock Laboratories), and a Lillehei-Kaster pivoting disk valve (Medical, Inc.). Pressure was adjusted at 120/70 mm Hg during control studies and 150/100 mm Hg to 170/110 mm Hg during studies of the effects of augmented pressure.

High speed motion pictures (2,000 frames/sec) (Hycam high speed camera, Red Lake Corporation) were taken of aortic valvular motion and the simultaneous analog display of sound (Fig. 1). The duration of closure was measured independently by two viewers. Frames of the cine (0.5-msec intervals) were counted. The beginning of closure was judged as the time of onset of motion from the point of widest opening. Total closure was judged as the point of complete coaptation of all portions of the three leaflets. The filming speed of the camera is thought to be accurate within 1%. The extent of valvular opening previously has been shown to depend on the stroke volume and the ejection rate, and these were kept constant at various pressures. The extent of opening was confirmed to be constant in this study. Since the distance of leaflet travel to closure was constant, the rate of closure was inferred from the duration of closure. Sound and pressure were recorded on a miniature dual-beam oscilloscope (Hewlett-Packard). The time lag of this oscilloscope was less than 1 μsec. Sound and pressure were recorded on the same frames of the cine as the valvular motion by placing the oscilloscope within the same lens field as the aortic valve. The time of occurrence of the second sound in relation to closure of the valve was judged from these cines, as was the time required for closure of the valve at various pressures.

Results

In each of the subjects with no apparent valvular disease, the aortic closure sound was of greater amplitude in the left ventricle than just above the aortic valve (Fig. 2). Similarly, the pulmonic closure sound invariably was of greater amplitude in the right ventricle than just above the pulmonic valve (Fig. 2). The second heart sound in all dogs was also found to be of greater amplitude within the ventricles than above the semilunar valves (Fig. 3) and similarly this was found in vitro.

Figure 2 Top: left ventricular (LV) and aortic (Ao) pressure and sound. Bottom: pulmonary arterial (PA) and right ventricular (RV) pressure and sound. In both instances, intracardiac recordings of the second sound (S2) were of higher amplitude within the ventricle than above the respective semilunar valve. The first component of intraventricular sound, in both the LV and RV, was recorded as a downward deflection indicative of compression. The first component of intra-arterial sound, in both the Ao and PA, was recorded as an upward deflection. This indicates expansion that was coincident with the intraventricular compression. Inflections on the LV and RV pressure curves (arrows) indicate compression coincident with the major component of the second sound.

Following the initial slow onset of the first component of the second sound in human subjects, the direction of inscription of the various components of the aortic closure sound, when recorded in the aorta, was invariably opposite to the direction of inscription of the components of the

Figure 3 Simultaneous recording in dog of electrocardiogram (ECG), left ventricular (LV) intracardiac sound, intra-aortic (Ao) sound, and pressure within the Ao and LV. Pressures were equivalent, as were recordings of sound. LV sound was louder than Ao sound. Following the initial slow onset of the first component of sound, the inscription within the LV of the various components of the aortic closure sound were opposite in direction to those in the Ao. This indicated simultaneous compression in the LV and expansion in the Ao, and vice versa. Coincident with sound due to compression, inflections due to compression were recorded on the LV pressure (arrows). Transient sound vibrations of lower amplitude followed the major component of the second sound (S2).
aortic closure sound when recorded in the ventricle (Fig. 2). Simultaneous recordings of sound within the left ventricle and above the aortic valve in dogs confirmed that the direction of inscription of sound was opposite in the aortic and ventricular chambers (Fig. 3).

Notches on the left ventricular pressure curve of humans and dogs were observed (Figs. 2 and 3). The inflection on the pressure tracings coincided with the components of the aortic closure sound. As the second sound increased in intensity, the inflection on left ventricular pressure tracings increased in magnitude (Fig. 4).

The second sound in dogs, whether recorded within the left ventricle or within the aorta, increased in amplitude with augmentation of aortic diastolic pressure and decreased with a reduction of aortic diastolic pressure (Fig. 5). Similarly, the intensity of the pulmonic closure sound varied with pulmonary arterial diastolic pressure. The aortic closure sound in vitro was shown to vary with the changes of simulated aortic pressure when the pump was held at a nearly constant stroke volume (Fig. 6). In open-chest dogs (in which instantaneous flow could be measured) there was no change of the amplitude of the second heart sound during post-extrasystolic beats. During such contractions, both forward and retrograde flow increased, but aortic diastolic pressure did not change (Fig. 7). Augmentation of peak systolic flow from 250 ml/sec to 450 ml/sec and augmentation of peak retrograde flow from 10 ml/sec to 35 ml/sec at a nearly constant diastolic pressure caused no change of the intensity of the second sound in vitro (Fig. 8).

The incisura of aortic pressure in dogs did not coincide with the nadir of aortic flow, but occurred 12-15 msec earlier (Fig. 9). The second sound started during the deceleration of aortic flow and began before the incisura of aortic pressure. The major components of the second sound occurred before the nadir of aortic flow (Fig. 9). Studies in vitro confirmed the fact that the first deflection of the second sound began during the deceleration of aortic flow and it began before the incisura of aortic pressure. Studies in vitro also confirmed that the incisura of aortic pressure preceded the nadir of aortic flow.

Simultaneous high speed motion pictures of closure of a porcine valve and simultaneously recorded aortic pressure and sound indicated that the sound began one cine frame (within 0.5 msec) after complete closure of the leaflets. This was consistently observed on six separately filmed closures of the valve. The leaflets, when observed at 2,000 frames/sec were shown to close in a nonsimultaneous fash-
ion (Fig. 10). Comparisons of high speed motion pictures of closure of the porcine valve at control pressures of 120/70 mm Hg and 150/110 mm Hg showed no change of the duration of closure of the leaflets with the higher pressure (Fig. 10). At 120/70 mm Hg, the average duration of closure of 3 beats was 27 msec. At 150/110 mm Hg, the average duration of closure was also 27 msec. The extent of opening was constant at both pressures. Therefore, the average rate of closure was constant. In spite of this, the amplitude of the second sound doubled with the higher pressure (Fig. 10).

Transient sounds were observed following the main component of the second sound in humans and in dogs (Figs. 2–4). Studies in vitro also showed transient sounds following closure of the porcine leaflet valve, in spite of the rigidity of the simulated aorta and ventricle (Fig. 10). Coincident with these transient sounds, we were able to observe vibrations of the closed valve in vitro.

To highlight these observations and further demonstrate their importance in the production of the closure sounds, studies of closure of a pivoting disk valve were performed. These studies of disk closure demonstrate the absence of mechanisms attributed to a stretched membrane. The second sound generated by the disk valve upon closure in the simulated arterial system in vitro was of the same amplitude both above the aortic valve and within the simulated ventricular chamber (Fig. 11). The closure sound of the pivoting disk valve occurred coincidently with disk closure, precisely at the time of impact. In contradistinction, sound induced by closure of the semilunar valves just after closure of the leaflet valve, when compression was induced within the left ventricle. There was no increase in the amplitude of the second sound with disk closure even in the presence of a marked augmentation of aortic diastolic pressure in vitro (Fig. 12). As with the semilunar valve, there was no increased rate of closure with the disk as a result of the elevated aortic pressure, the duration of closure being 52 msec at both 120/70 mm Hg and 170/110 mm Hg (Fig. 12). Transient sounds following closure of the disk valve were virtually absent both in the subject we studied (Fig. 13) and in vitro (Figs. 11 and 12).

**Discussion**

This study indicates that the second heart sound originates from an initial driving force, namely, an arterial to ventricular pressure differential, which stretches the semilunar valves just after closure. As a result of this stretch and the consequent valvular recoil, expansion and compression of blood are initiated in the arterial and ventricular cavities, thereby producing transient pressure changes representative of sound. The semilunar valves, when in a closed position, can be considered analogous to a mechanical vibrating system known as a circular stretched membrane. Such a membrane must be flexible, thin, and stretched in all directions by a force which is not affected by the motion of the membrane. Any force that would set such a membrane in motion would result in the production of compression and expansion waves. Each of the observed phenomena in this study which are related to the second heart sound can be explained on the basis of such compression and expansion and their mode of generation. The results of this study are in accordance with the general concept of silent approximation of the semilunar valves, followed by aftervibration of the leaflets which are productive of the second heart sound.

The conflicting postulates of the mechanism of the origin of the second heart sound which brought about this investigation will be treated separately in this discussion. Our proposed view of the origin of the second sound as being the result of membrane deflections which originate...
Figure 10  Sequential frames from cine (2,000 frames/sec) showing closure of stented porcine valve at pressure of 120/70 mm Hg (top), and 150/100 mm Hg (bottom). Simultaneous sound and pressure were recorded on a photographic recorder; pressure and sound on the frames of the cine were not suited for reproduction. Intervals of 10 frames (5 msec) were selected for illustration. The rate of closure of the leaflets was constant, regardless of the pressure. Transient sounds followed the main component of the closure sound (S1). The leaflets closed in an asynchronous fashion, the leaflets shown at the left and bottom of each frame being fully closed 5 msec before the remaining leaflet.

at the semilunar valve and result in the compression and expansion of blood does not relate to the method of sound radiation in the cardiovascular system. The latter has been studied by others.15

Figure 11  Sound in simulated left ventricle (LV) and aorta (Ao) of in vitro model of cardiovascular system. With the porcine valve (left) sound in the LV was recorded with greater amplitude than in Ao. With the disk valve (right) sound was of the same amplitude in both the LV and Ao. The amplifier gain for the porcine valve and disk valve was not the same.

OCCURRENCE OF THE SECOND HEART SOUND IN RELATION TO OTHER HEMODYNAMIC PARAMETERS

Theories of the origin of the second heart sound must be compatible with observations of the time of occurrence of the sound in the cardiac cycle. We detected the beginning of the second sound 0.5 msec after closure of the aortic valve. This is the time when stretch of the valve is initiated, resulting in the initiation of compression within the ventricle. Studies by MacCanon et al.7 and our own studies indicate that the aortic valve is completely closed 5-13 msec before the sharp apex of the aortic incisura. Pienme et al.8 have shown, and we confirm, that the second sound begins coincident with the rapid deceleration of the aortic flow. At this time, however, the semilunar valves are fully closed (as judged from high speed cine in our study), and this is in contradistinction to their thought that the second begins significantly before the valve closes. In spite of the
fact that the semilunar valves are closed, forward flow continues but decelerates rapidly. This behavior is due to the forward momentum and inertia of the blood. Whether deceleration of flow still can be postulated to relate to the cause of the second sound in view of these observations is undetermined.

The major components of the aortic closure sound in dogs occurred before the onset of backward flow. These observations contradict the postulates of others\(^\text{11}\) that the hydrodynamic mechanism of the origin of the second sound is related to blood rushing back toward the ventricular chambers and being abruptly arrested by the semilunar valves. If this were the case, the loudest sound should occur simultaneously with the nadir of flow. Our study indicates that this is not the case.

**SOUND PRODUCTION BY FORCEFUL COAPTATION OF THE LEAFLETS**

The second sound has been attributed to the snapping together of the corpora arantii which, consequently, cause the tense semilunar leaflets to vibrate.\(^\text{10}\) It has been suggested that systemic or pulmonary hypertension brings about a more rapid closure of the aortic or pulmonic valve, resulting in a louder intensity of the second sound.\(^\text{18}\) The question we endeavor to answer is not whether the leaflets vibrate upon closure, but whether the snapping of the corpora arantii is the major cause of vibration. Through the use of high speed cinemographs of closure of a semilunar valve, it was apparent that the leaflets do not close simultaneously, but rather in an asynchronous fashion. This minimizing the energy that is generated by the striking of the leaflets against each other. This energy is largely dissipated before the occurrence of the second sound, since sound occurs only after complete closure of the leaflets. Furthermore, cine measurements of the closure rate of a semilunar valve indicate no increased rate of closure in the presence of increased arterial pressure. If the vibrations which produce the second sound were related to the energy of coaptation, then the increased intensity of the second heart sound, which results from increased arterial...
pressure, should be accompanied by an increased rate of closure of the leaflets in order to produce higher amplitude vibrations. This, however, was shown not to be the case. The rate of closure was constant at various levels of aortic pressure.

**FACTORS AFFECTING THE INTENSITY OF THE SECOND HEART SOUND**

It is generally agreed that the intensity of the second heart sound is affected by the magnitude of the diastolic pressure.16-18 The mechanism of this observation is unexplained. Rushmer11 presumes that retrograde velocity is higher in the presence of systemic or pulmonary hypertension. He suggests that the intensity of the second sound depends on the velocity attained by the blood gushing back toward the ventricle and the abruptness with which the motion is arrested.11 With presumed higher velocity, the sound would be higher. Our studies in vitro, however, showed that the intensity of the second sound did not vary in the presence of increased forward and retrograde aortic flow when pressure was held constant. In dogs, no increase of the intensity of the second sound occurred when forward and reversed aortic flow were augmented. Since the major components of the aortic closure sound occur before the beginning of the reversal of aortic flow, it would seem that backward flow does not initiate the stretching of the leaflets which is responsible for oscillations productive of sound.

The intensity of the second sound following disk closure was independent of pressure. The mechanism of production of the closure sound of a disk valve is related to impact of the disk on the seat of the valve, an entirely different mechanism of sound production than that of a normal semilunar valve. The intensity of the sound is related to the energy of impact, as indicated by the kinetic energy equation, $\frac{1}{2}mv^2$, where $m$ is the mass of the disk and $v$ is the velocity of disk closure. The rate of closure of disk valves, therefore, must change in order to change the intensity of sound.

**PROPOSED MECHANISM OF THE ORIGIN OF THE SECOND HEART SOUND**

The results of this study suggest that the semilunar valve, when closed, being tense, thin, and compliant, has the characteristics of a circular membrane.14 A pressure differential between the artery and ventricle causes the tensed valve to stretch. It thereby generates compression of the blood in the ventricular cavity and a relative expansion or rarefaction of blood within the arterial cavity. Recoil of the valve then produces expansion within the ventricle and compression within the arterial chamber. The presence of compression and expansion is associated with an instantaneous change of the ambient fluid pressure, which defines sound.14

The occurrence of compression and expansion of blood was shown by oppositely recorded directions of inscription of various components of the aortic or pulmonic closure sound relative to whether sound was recorded on the arterial or ventricular side of the valve. Transient alterations of the pressure indicative of compression were noted on the downslope of the ventricular pressure tracing coincident with each compression component of sound (Figs. 2, 3, and 5). This is expected since sound is a transient alteration of pressure superimposed on the ambient pressure. Similarly, the simultaneous expansion of blood in the aorta is marked by a rapid reduction of pressure which ends at the apex of the incisura. The fact that sound was recorded at higher amplitudes within the ventricle indicates that the higher driving force productive of compression was directed toward the ventricle. Disk valves appear to produce a closure sound by impact of the disk against the seat of the valve. Since the mechanism is independent of stretch and recoil, components of sound were of equal intensity in the arterial and ventricular chambers.

As do all flexible membranes, the tense leaflets recoil because of their elastic properties after the initial stretch. Recoil consequently creates an expansion in the ventricle simultaneous with compression in the arterial cavity. The driving force generated by recoil is presumably of lower magnitude than the initial stretching force, and therefore produces lower amplitude components of sound (Figs. 2, 3, and 5). Stretch and recoil cease when the pressure gradient between the ventricle and arterial cavity becomes nearly constant.

Continuing but attenuated stretch and recoil appear to be responsible for the transient sounds which occur after the main components of the closure sound. This differs from previously postulated theories which relate the tran-

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**Figure 13** Top: Electrocardiogram (ECG), aortic (Ao) pressure, and intra-aortic sound in a subject with a normal aortic valve (top), and a subject with a tilting disk aortic prosthetic valve (bottom). Vibrations of lower amplitude followed the main component of the second sound (S₂) in the subject with the normal valve. Such transient sounds were virtually absent in the subject with the disk valve. Vertical lines show that S₂ began before the incisura of aortic pressure with the normal valve (top) and at the incisura with the disk valve (bottom).
sient sound to arterial vibrations and to the opening of the mitral valve, resulting in low-pitch vibrations. Transient vibrations were observed in an in vitro system utilizing a semilunar valve, and occurred even in the absence of other structures comparable to the mitral valve. As further evidence of this mechanism, tests utilizing a disk valve showed a virtual absence of sound transients. This was confirmed in one subject with a Björk-Shiley aortic prosthetic valve. The noncompliant nature of the disk prevented secondary sounds that would be caused by continuing stretch and recoil of the valve.

Our proposed mechanism for the origin of the second sound suggests a relation between the amplitude of the second sound and the driving pressure. Driving pressure, in sound theory, refers to the difference in pressure between the force exerted upon a mechanical vibratory system and the ambient pressure. In the case of the heart, the driving pressure refers to the instantaneous difference between arterial and ventricular pressure shortly after semilunar closure. Kusukawa and associates found a good correlation of the amplitude of the second sound with the difference of pressure between the aorta and left ventricle coincident with the dicrotic notch. They found a better correlation, however, between the amplitude of the second sound and the peak rate of development of the aortic to left ventricular differential pressure gradient (first derivative maximum).

CLINICAL SIGNIFICANCE

Events which relate to the intensity of the second sound can be explained in view of the observations made in this investigation. Augmentation of the intensity of the second sound with increased diastolic pressure is expected since, with a given valve, augmentation of diastolic pressure in general increases the rate of development of the differential pressure between the arterial and ventricular chambers. The amplitude of the second sound relates to the rate of development of the driving pressure (Fig. 5).

The diminished or absent second sound in calcific aortic stenosis can be explained in terms of the observations in this study. The semilunar valve, in producing a closure sound, functions as a stretched membrane. If the leaflets are thickened and noncompliant as a result of disease such as calcific aortic stenosis, then they no longer can function as a membrane capable of vibration. Stretch and recoil are diminished and the sound is reduced.

The second sound in congenital aortic stenosis is not lost or diminished, and this apparent paradox is also consistent with the theory we propose. Even though such valves are stenotic, the leaflets are thin and flexible and retain their capability of becoming tense after closure. Therefore, congenitally stenotic semilunar valves retain the characteristics of a flexible membrane and produce the stretch and recoil required for the production of the second sound.

A regurgitant semilunar valve also would be expected to produce a closure sound of diminished intensity according to the mechanism of the second sound proposed in this study. In the case of semilunar insufficiency, the leaflets of the valve cannot become tense following closure. An important characteristic of a stretched membrane capable of vibration, therefore, is lost. We have observed and illustrated a markedly reduced second sound in pure aortic insufficiency, although this observation, in general, has not been made.

Acknowledgments

We thank George T. O’Shea, Chief, Medical Media Production Service, Veterans Administration Hospital, Oklahoma City, for his technical advice related to the photographic techniques utilized in this investigation.

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Circ Res. 1976;39:874-882
doi: 10.1161/01.RES.39.6.874

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7330. Online ISSN: 1524-4571

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