The Forces Needed to Evoke Sounds from Cardiac Tissues, and the Attenuation of Heart Sounds

By William Dock, M.D.

There has been no study of the efficiency of any sound-producing system. Here there is a description of a device that records the force of each pull applied to a valve segment or strip of myocardium while the sound evoked is recorded. The relation of force to sound intensity, and the attenuation of sounds in air, blood, and ventricular and chest walls provide some quantitative data on a neglected phase of cardiac physiology.

A BRIEF SERIES of experiments showed that relatively little force is required to evoke loud sounds when leaflets and chordae of the cardiac valves are drawn taut under water, although large forces are required to evoke sounds from strips of heart muscle. In order to obtain some quantitative data on the relation of teusing force to noise, it was necessary to devise a noise-free method for applying and recording forces of varying size. Once it was possible to get consistent records of force and noise with application of a given force, it was easy to compare the sonic potential of various cardiac structures, and the attenuation of sound in blood, air, myocardium, and the tissues of the chest wall.

METHOD

Records were made on a Sanborn Twin-Beam galvanometer. The acoustic elements were calibrated with a Maico audiometer.

An SRA-Type C strain gage, bonded to a steel spring, provided the data on forces applied to the tissues. A 12-volt alternating current was applied to a variable Wheatstone bridge, in which the strain gage was one element. This arrangement gave an output of 3.1 mv. (± 0.1 mv.) per 100 Gm. when forces of 25 to 400 Gm. were applied to the spring, in the device shown in figure 1. A Lucite tank, 14 by 18 cm. and 20 cm. deep, was used for immersion of the tissues. A stethoscope head with watertight diaphragm was fixed to the wall of the tank, with a tube leading through the wall to the microphone. Tank and microphone rested on foam rubber 5 cm. deep to insulate them from vibrations in the building. The apparatus was set up in a quiet room, not used for any other purpose.

As seen in figure 1, a heart valve cusp, V, is fastened at its annular margin to a balsa block firmly set in an 800-Gm. lead weight, W. This is done outside the water bath, with the beam, E, and attached fixtures are suspended so that the papillary muscles, P, can be fastened to the small balsa paddle, D. This paddle is firmly fastened to the spring, S, on one half of which the strain gage is bonded. The spring has its ends set in the prongs of the paddle, C, and the handle passes through a hole in the beam, F, to which it is fixed by an axle, A, on which the paddle swings freely. A long enterostomy clamp is used to lift the weight with one hand, while the beam is lifted from its support with the other. The device is thus transferred to the water-bath, and the beam slipped into place on the axle, F, which can be adjusted to any desired height above the bath. The depth of water is sufficient to cover the stethoscope diaphragm and the lower half of paddle, D. A lead weight, I, is placed on the beam, drawing the valve taut, and the axle is adjusted so that the beam is horizontal. Then the balsa block, B, on another adjustable mount, is placed so that it stops the descent of the left arm of the beam when the right end is lifted by hand. The usual setting permits 1 cm. descent of the beam, A, thus allowing 1 cm. of slack in the valve. The axles are steel, tight-fitting, and lubricated with graphite, so that the device generates no sound. The block, B, is faced with 3 mm. of foam rubber, so that contact between it and the beam is almost noiseless. This contact occurs only in the interval between tests.

The distance from A to F is 6 cm. When a lead weight, as a tight-fitting rider, is placed on the right arm 3 cm. from the axle, the force rais-

From the Department of Medicine, State University of New York-Downstate Medical Center, Brooklyn, N.Y.

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ing the paddle is half that of the weight, which falls half the distance the paddle rises when the beam is released after being pressed against \( B \). Since the riders vary from 25 to 200 Gm., and can be placed at 3, 6, 9, or 12 cm., force can be varied over a wide range. The beam is balanced by a lead weight built into the right end, but fine adjustment, to compensate for the buoyancy of the partly immersed lower paddle, is made with a small weight close to \( F \), on either side as needed, just before starting a series of tests. A metal point, at the right end of the beam, is touched with a finger to raise the weighted beam. It is released when the other end rests on \( B \). Lifting and release are repeated 6 to 8 times for each record, made at a paper speed of 2.5 or 7.5 cm./sec.

When attenuation of sounds in ventricular or thoracic wall is tested, disks of tissue more than twice the size of the stethoscopic diaphragm were held over the diaphragm by a wire ring that pressed the edges of the tissue against the wall of the tank. Only light pressure and support were needed and the pressure on the diaphragm was no greater than water pressure in ordinary recording.

**Calibration**

The strain gage was tested by lifting the weights attached by a short cord to the edge of the lower paddle. The voltage change, 3.15 mV. (± 0.1 mV.) per 100 Gm., remained constant over the range 25 to 400 Gm. and over 5 months of testing. Thus the voltage output was 1 mV./30,000 dynes. The records were made with a galvanometer sensitivity of 2 mm./mV., and in the figures 1-mm. deflection of the trace equals 15,000 dynes.

The Sanborn microphone was first tested with the standard stethoscope diaphragm attached directly as for heart sound recording. The normal adult gives a first apical heart sound record with a deflection of 8 to 12 mm., with the logarithmic amplifying circuit and the sensitivity control at "6." Deflections produced by heart sounds, by sounds elicited from valves under water, and by sounds from an audimeter were compared at various settings of the amplifier, from 4 to 8. With any given sound, if the deflection at setting 4 be taken as 1, the deflection at 5 is 3, at 6 it is 8, at 7 it is 20, and at 8 it is 35. All of the tests of valves were recorded with the logarithmic circuit at settings in the range of 4 to 7, and the deflections were reduced to equivalent deflections at 6 by multiplying the deflection at 4 by 8, that at 5 by 2.7, and the deflection at 7 by 0.4.

When the earphone of the Maico audiometer was applied to the Sanborn diaphragm, with the amplifier at 6 the deflection was 11.6 mm. for 70 db. at 128 c.p.s., 14.6 mm. at 256 c.p.s., and 4 mm. at 500 c.p.s. Thus the first sound is equivalent to 60 to 70 db. at the 125-cycle level; this also is the sound intensity of the unit, the sone, used by students of hearing and sound engineering.

When the bone conduction unit of the Maico audiometer was applied to the Sanborn diaphragm, the "10 db." setting gave a 6.4-mm. deflection at 125 c.p.s., 7.6 at 250 c.p.s., and 1.4 at 500 c.p.s. When the diaphragm and tube connection used in the tank were tested with the bone conduction unit, "20 db." at 125 c.p.s. gave a 7.2-mm. deflection. This result means that a pure sustained 125 c.p.s. tone, recorded through a tube from the tank, is attenuated to less than half of what it would be if recorded with the Sanborn diaphragm directly attached to the microphone. However, when the microphone, tube, and diaphragm from the tank were used to record apical heart sounds of a normal subject, the peak amplitude was only 15 per cent smaller than when the Sanborn diaphragm was applied at the apex.

With both types of Maico stimulator and over the range of 10 to 80 db., the Sanborn microphone with the logarithmic amplifier shows a 3-fold increase in deflection amplitude for a 10 db., and a 10-fold increase with a 20 db., increase in sound. The audiometric decibel is based on sound intensity levels as related to the logarithm of the
FIG. 2. In this and other figures, upper pattern is the strain-gage record of tension applied to the valve (1 mm. = 15,000 dynes); lower is sound. From a complete anterior mitral leaflet with 2 papillary tips and many chordae. At this setting (5) of amplifier, a "normal" first sound gives a deflection of 4 mm. This demonstrates increase in sound with increase in force tensing entire cusp-chordae system.

FIG. 3. High speed recording, from 2-chordae preparation; posterior tricuspid leaflet. From a normal man, age 20, killed violently. Recorded at 4.5 setting.

square of the pressure acting on the eardrum.  

\[ \text{Sound intensity in decibels} = 20 \log_{10} \frac{\text{sound pressure}}{0.0002 \text{ dynes/cm}^2} \]

Thus a 3.1 increase in sound pressure equals 10 db., a 10-fold increase 20 db., a 31-fold increase 30 db., 100-fold increase 40 db., etc. Since the "logarithmic" amplifier units of the Sanborn phonocardiogram give a galvanometer deflection, as described above, 3 times as large for 10 db., and 10 times as large for each 20 db. increase in sound at the 125 to 250 c.p.s. range, the response is linear with sound pressure, and not with the logarithm of the ratio of sound pressure to the standard threshold of 0.0002 dynes/cm². The unit records sound pressure, not decibels. The latter can be derived from the data when needed to show the relation of increased sound to increased force applied to a valve.

Loudness, on the sone scale, is very different from decibels or sound intensity. A rise from 60 to 75 db. at 125 c.p.s. gives a 10-fold increase in loudness. Thus, at levels close to the normal heart sound, an increase of 15 db. in sound or of 5-fold increase in the Sanborn signal would correspond with a 10-fold increase in loudness. Therefore, in our graphic records, changes in amplitude of sound signal are less than apparent loudness would be to an auditor.

RESULTS

Relation of Tensing Force to Sound Intensity

When an entire cusp with all its chordae and tips of papillary muscles is tensed with increasing force, the sound evoked becomes louder. Changes in quality and duration also are evident. A series of such force-sound curves from different valves shows very wide variation. This is due to the great variation in number and points of attachment of the chordae, especially with posterior mitral
Forces Needed to Evoke Sounds

cusps or any of the tricuspid cusps. If one uses only 1 papillary tip, with 2 chordae to the edge of the leaflet, tensing only the central segment of the cusp, the sonic properties of different valves become less variable. But, as increasing force is applied, changes in pattern as well as intensity do occur, and breaks in the curve are seen when force is plotted against sound. These are shown in figures 2 and 3, selected from the experiments on which table 1 and figure 4 are based.

When the sound intensity in decibels is plotted against maximum force recorded during tensing these cusp and chordae preparations, a straight line can be drawn through the center of the group lines (fig. 4). This line intercepts the 60-db. level at 100,000 dynes, and the 80-db. level at 300,000 dynes. On the average, sound intensity increased 10-fold with a 3-fold increase in force above that required to evoke noise equal to the normal first sound.

As is evident in figure 4 the lines of forcesound relations intercept the zero force level at relatively high sound levels. This means that the straight line relationship is valid only in the range of 100,000 to 500,000 dynes. In the group of mitral cusps with 2 chordae, the range of zero force interceptions varied from 37,000 to 58,000 dynes, and $\Delta F/\Delta S$ varied from 6,000 to 13,500 dynes/db. The lowest values for both were in a cusp from a young normal heart; the highest, in the cusp from a middle-aged hypertensive subject. The ratio of sound to force rises relatively rapidly from 10,000 to 50,000 dynes and is almost linear from 80,000 dynes up to 800,000 dynes.

Relative Sonic Potentialities of Cardiac Tissues

When entire cusps, with the complex of chordae and 2 to 4 papillary tips, are tested, it is found that sounds of a given intensity are more easily evoked from anterior mitral leaflets than from other preparations, and that the posterior tricuspid cusp also is relatively easily set into audible vibration. The shorter cusps and many chordae of the elements that make up the rest of the atrioventricular valve ring appear to be much less effective vibra-

Fig. 4. Figure based on data in table 1, with sound intensity $(S)$ in decibels as ordinate, force $(F)$ in dynes as abscissa. $\bigcirc$, from intact anterior mitral leaflet; $\bullet$, from 2-chordae preparations of anterior mitral leaflets; $X$, from similar tricuspid posterior leaflet preparations.

tors. But, as shown in table 1 and figure 4, the simple elements taken from all leaflets are comparable in their sonic responses to tensing. When a posterior mitral cusp giving a small response is cut down to provide such a simple preparation, a louder noise may be given by the small portion of the valve when the same tension is applied. And when the chordae only or the leaflet only is mounted in the testing device, the same tension may evoke almost as much noise from the small portion as from the entire segment from papillary tip to annulus fibrosus. Splitting up of tension between many chordae and parts of cusps with little probability that all will be equally tensed at the same instant appears to make the sound evoked from some entire valves less intense than from simple elements. Apparently the force per fiber, rather than the size of the fibrous structure being tensed, is a very important factor in determining the sonic behavior of the atrioventricular valves when tensed in our device. The force applied to a pair of chordae is also applied to the segment attached to it. Undoubtedly the difficulty in
TABLE 1.—Sonic Responses to Tensing Forces on Valve Leaflets

<table>
<thead>
<tr>
<th>Valve leaflet</th>
<th>Sound pressure*</th>
<th>Forces (dynes)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Anterior mitral</td>
<td>2.1</td>
<td>195,000</td>
</tr>
<tr>
<td>Age 28</td>
<td>4.0</td>
<td>270,000</td>
</tr>
<tr>
<td></td>
<td>7.8</td>
<td>310,000</td>
</tr>
<tr>
<td>2. Anterior mitral</td>
<td>2.1</td>
<td>210,000</td>
</tr>
<tr>
<td>Age 35</td>
<td>7.5</td>
<td>285,000</td>
</tr>
<tr>
<td>Hypertensive</td>
<td>24.0</td>
<td>530,000</td>
</tr>
<tr>
<td>3. Anterior mitral</td>
<td>2.8</td>
<td>150,000</td>
</tr>
<tr>
<td>Age 62</td>
<td>5.5</td>
<td>255,000</td>
</tr>
<tr>
<td></td>
<td>11.2</td>
<td>280,000</td>
</tr>
<tr>
<td></td>
<td>22.4</td>
<td>400,000</td>
</tr>
<tr>
<td></td>
<td>27.5</td>
<td>420,000</td>
</tr>
<tr>
<td>4. Anterior mitral</td>
<td>0.5</td>
<td>120,000</td>
</tr>
<tr>
<td>Age 24</td>
<td>2.6</td>
<td>300,000</td>
</tr>
<tr>
<td>(Fig. 2)</td>
<td>3.9</td>
<td>400,000</td>
</tr>
</tbody>
</table>

*Sound pressure 1 is that of average apical first sound and gives a sound intensity of 60 db. Sound pressure 10 gives an intensity of 80 db.

adjusting a series of papillary tips on the paddle so that tension will be applied equally to all is an important factor in impairing the sonic response of the complex leaflets. Slight shifts in position of the paddle in relation to the lead-weighted balsa block may markedly change loudness, duration, and character of the sound evoked, although in any given position each preparation gave a constant pattern.

The strain-gage records show that the entire system of paddles, beam, and weight goes into a low-frequency oscillation, or “bounces” when a valve or other fibrous structure is suddenly drawn taut. The pattern of this oscillation depends on the natural frequency of the weighted system and on the viscosity and resiliency of the tissue being tested. Thus, in figure 5, the relatively stiff and simple anterior mitral valve swiftly decelerates the rise of the paddle, pulls the weighted arm up and releases all force on the spring for 0.08 sec. The delicate, many-stranded posterior leaflet produces slower deceleration of the paddle, with small rapid bounces and a long series of audible vibrations rather than a sharp snap. These vibrations may be due to taut chordae rubbing against each other during the “bounce.” The strip of interventricular septum is so viscous that deceleration of the paddle is gradual, no real bounce occurs, and no audible vibration is evoked by a force 8 times that which produces from the anterior mitral leaflet a sharp sound as loud as the normal apical first sound.

The testing device, intended merely to apply variable forces, or the same force repeatedly, was thus useful in showing how fast the force was absorbed. The type of “bounce” gave some idea of the resilience and viscosity of the tissues. Often the rebound would evoke a sound, as in figure 5.

As had been previously noted,1, fig. 19, no. 4 strips of fat-free parietal pericardium the size of a mitral leaflet can be set into audible vibration, and so can small strips of atrial wall, or the entire membranous interatrial septum of an occasional heart. Faint sounds can be elicited by great force from ventricular walls (figs. 5 and 6), in strips 2 cm. wide, but not from a whole lateral wall of a normal heart.

Attenuation of Sounds in Blood and in Ventricular and Chest Walls

With the use of anterior mitral cusp and chordae tendineae, the relative sound production in air, water, and whole blood could be recorded. The valve system was not touched between tests, and force records in these comparative tests were constant. The most striking difference was the extremely feeble sound produced when valves were tensed in air as compared with water (fig. 7). The sound intensity is 20 times greater, with the micro-
forces needed to evoke sounds

Figure 5. Left upper, anterior mitral leaflet; lower left, posterior mitral leaflet, both at setting 5, beam weighted with 100 Gm. at 3 cm. Right upper, same mitral leaflet, beam weighted with 50 Gm. at 3 cm.; right lower, strip of ventricular septum, weight 200 Gm. at 6 cm.; both at setting 6. Normal heart, 300 Gm.

phone 2.5 cm. from the leaflet, in water than in air, and the sound is much sharper. The attenuation of sound in blood is slight. In the most marked example (valve to diaphragm, 5 cm.) the decrease in intensity in blood was 30 per cent. In most records it is evident that high-frequency vibrations are much more reduced than those under 100 c.p.s. The corpuscles may damp the vibrations of the fibers, or merely attenuate transmission, like snow flakes in air.

The most marked attenuation in passing through a normal right ventricular wall held in front of the microphone was 20 per cent, but with hypertrophied left ventricular wall, in rigor and 3 to 4 cm. thick, sound intensity fell 65 per cent in one experiment and 45 to 52 per cent in 3 others, passing through the ventricular walls of hypertensive subjects. In figure 8 are shown the results of interposing intact chest wall, with 2 cm. of fat and skin and 1.5 cm. of rib cage and pleura, between the valve and the diaphragm of the phonocardiograph. The loss of intensity in the entire wall taken from the left precordial region over the heart was 65 per cent. When the skin and fat were tested, the loss was 20 per cent; when the ribs, muscle, and pleura were interposed, the loss was over 40 per cent. Five other tests with chest wall gave a minimal attenuation of 42 and a maximal of 81 per cent. All these tests, as those with attenuation in blood, show changes in sound pattern, as well as in its intensity, so that attenuation varies with frequency.

It must be re-emphasized that these percentage changes in the Sanborn signal correspond with percentage change in sound pressure, not in decibels or loudness. The rise of 5-fold pressure on removing the chest wall means a 15 db. rise in sound, and about a 10-fold increase in loudness. The sound produced by
Fig. 6. From left to right, interatrial septum, interventricular septum, both at 6; mitral anterior leaflet from annulus to line of closure; same, chordae from line of closure to papillary muscle, recorded at 5. Weight, 200 Gm. at 3 cm., except for chordae, 100 Gm. at 3 cm. A 650 Gm. heart.

a given force acting on the valve in water would be reduced in a large heart, 30 per cent by blood, 50 per cent by the ventricular wall, and 50 per cent by the chest wall, or a total attenuation of 80 per cent on the average. With the maximal observed losses this might be 95 per cent, or 25 db. A loud sound would become only 2.5 per cent as loud with this attenuation, since 1 sone is 25 db. down from 40 sones. Whenever air-filled lung is interposed, even larger losses of sound transmission occur, since small air cells provide optimal sound-proofing, and valve sounds are markedly attenuated when produced and transmitted in air.

DISCUSSION

In our experiments the valve elements were tensed, as they are in the body, in a fluid medium. The force necessary to evoke sounds of varying intensity was measured in water, as well as the attenuation experienced when sounds are produced in and transmitted through air or blood, and when they pass through the ventricular walls and the thoracic wall. It was found that the sound evoked when force was applied to a segment of a cusp, or even to 2 chordae, might be faint, as loud as, or, rarely, even louder than when the same force was applied to the whole leaflet with all its chordae. A force of about 100,000 dynes is needed to evoke from a pair of chordae and attached segment of a valve leaflet a sound equal to the apical first sound. To allow for the attenuation in blood, ventricular and thoracic walls, sound pressures 4 to 10 times greater must be evoked, and the force needed may be 180,000 to 300,000 dynes.

At the time the first heart sound occurs the difference in pressure on the 2 sides of the valve must be small, for the cusps have been floating freely in blood and there is a common chamber until the valves are drawn taut. All the force available for causing the sound must come from the arrest of the mass of blood moving from the ventricle toward the atrium, and from the contraction of papillary muscles, drawing the ends of the chordae tendineae in the opposite direction.

In our experiments, the valve elements were stretched in a straight line, and the annular edge also was stretched straight. In life the annular edge is curved, the cusps fill out in ares, like a ship’s sail in the wind, and only the chordae are straight, like the rigging holding a flying sail. The chordae to the centers of leaflets are not properly tensed in the device we use, and the entire system would have to be redesigned to fasten the cusp to a circular base and adjust all papillary tips to exert equal tension on the chordae to all parts of the cusp, as they do in the living heart. It is quite probable that a careful re-examination with such a device would reveal a higher production of sound per dyne of force applied to the valve. Yet the fact that a given force ap-
Fig. 7. Upper row, anterior mitral leaflet in air on left, in water on right, both recorded at 6. Below, left to right, in air, in water, both at 7; in air, at 7; in water, at 4. A valve tensed under water 2.5 cm. from stethoscope diaphragm produces far more noise than when tensed in air by same force.

plied to very small segments of valves, or to chordae alone, evokes sounds almost as loud as when it is applied to an entire leaflet, makes us believe that the general relationship of sound to force, and of the force needed to evoke the heart sounds from atrioventricular valves, is given by experiments such as these.

Proper positioning will lead to sharper sounds, for it will prevent friction of chordae against other chordae, and insure instantaneous tensing of all units. Thus maximal sound production from an entire valve will be similar to that when simple units of 2 chordae and a small segment of leaflet are tensed, as in the experiments on which table 1 and figure 3 are based.

When short segments of fibers are tensed, as in the semilunar cusps or the portion of mitral valve from annulus to line of adhesion tensed in a mitral snap, the pitch is higher, the sound is sharp, but the intensity may be very great. In the semilunar cusps, all the force is supplied by the inertia of the mass of arterial blood that is accelerated back toward the ventricle, since there is no motion of the points of attachment of valve margins. In the stenotic mitral valve, the mass of blood moving toward the ventricle is large, and a large pressure difference may develop between atrium and ventricle as the membrane moves from its end-systolic to its diastolic position.

Since the ventricular walls require very large tensing forces to produce audible vibrations, it is doubtful if they contribute to the heart sounds. Certainly the tension developed during systole causes little or no sound, as has been shown by the delay in the first sound in mitral stenosis, even though fiber tension in the wall rises to more than double that attained when a normal heart produces a first sound. The arrest of blood entering in diastole may tense chordae,\(^5\)-\(^6\) in mitral insufficiency with shortened cusps and chordae, but there is not enough inertial force to tense the ventricular wall forcibly enough to cause a sound.
The effect of ventricular wall in attenuating sound may be of some importance in explaining the location of areas of optimal audibility of sounds. All the valves are located close together, with interlocking fibrous rings, but first sounds and gallop sounds are loudest close to the apex of the left ventricle, while opening mitral snaps are loudest somewhat medial to this and even in the third left interspace next to the sternal border. The aortic second sound is loudest close to the sternum on the right at the same level, and tricuspid sounds close to the sternum in the fourth or fifth left interspace.

The left ventricle is thinnest at the apex, and approaches the chest wall most closely at this point, so that attenuation of the mitral first sound is least over the apex. In gallop and first sounds, the chordae are tensed, and as they are closer to the apex probably provide more sound than do the cusps. In mitral snap the noise of the cusps is less well transmitted into the apical region, but by shaking of the roots of the aorta and pulmonary artery, as well as by transmission into the dilated left atrium, it may reach the chest wall close to the sternum with less attenuation in traversing these thin structures. The pulmonic and aortic sounds are heard best where the bends of the arteries in which they arise approach the chest wall most closely, for there is probably minimal attenuation of sound in fluid-filled semi-rigid tubes.

It is remarkable how feeble are the sounds evoked when valves are tensed in air as compared with those heard when the same force is applied to valves immersed in water. This shows how far astray speculation could lead even so fine an investigator as Magendie. He rejected Rouanet’s thesis that the first sound was valvular, in spite of the ingenious experimental support Rouanet had given it. Rouanet, in one series of experiments, had obtained sounds from membranes tensed in air, and Magendie objected that what happened in air was not likely to occur in fluid-filled ventricles. Actually, the sound would have been far louder if Magendie had repeated Rouanet’s experiment with the membranes and stethoscope tip under water.

A surprising outcome of these experiments was the finding of a linear relation between sound intensity in decibels and the increase in peak force developed in tensing a cusp or chorda. The general equation is

$$ S = x \frac{(F-n)}{(n)} $$

where $S$ is sound intensity, $F$ is force in dynes, $n$, the force in dynes needed to evoke a sound of intensity 1, and $x$ the constant for each valve. We tried to find other experiments relating sound to force, but failed to find any. Intensity of sound in relation to force has not interested experimental or theoretical physicists. There are good studies of sounds in tanks, which would aid in performing this type of experiment in echo-free chambers. But in the classic texts on physics and on sound we found nothing on the relation of sound intensity to the force applied to strings or to air columns. There appear to
be no studies of efficiency of sound production, giving relation between force and sound, when various types of cords or membranes are suddenly drawn taut or struck when tensed.

Summary
A quantitative method of comparing a sound due to tensing cusps of valves, or other cardiac structures, and the peak force applied, shows that efficiency of sound production increases at higher levels of force. A straight line describes the relation of the logarithm of sound intensity to increase in force, above that needed to evoke a sound equal in intensity to a standard such as the first sound heart at the precordium on auscultation. The relatively stiff valves of older people or those with elevated intraventricular pressures give off louder sounds, for a given force, than the delicate structures of young normal subjects. Very large forces are needed to evoke audible sounds from strips of ventricular muscle and no such force levels occur in living animals.

Attenuation of heart sounds in air (and hence in aerated lung) is great. Attenuation in blood, ventricular wall, and chest wall is such that 70 to 95 per cent of sound intensity must be lost between the mitral cusps and the precordium even when the apex is in contact with chest wall.

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Summario in Interlingua
Un metodo quantitativo pro comparar un sono producet per tensificacion de cuspides valvular o de altere structuras cardiac con le fortia maximal applicate monstra que le efficacia del production de sonos es plus alte al plus alte nivellos de fortia. Un linea recte describe le relation inter le logarithmos del intensitates de sono e le augmentos de fortia supra le nivello requirite pro evocar un sono equal in intensitate a un standard de base, per exampl le prime sono cardiac que es audite al precordio in le auscultation. Le relativemente inelastic valvulas de subjectos de etate aviantiate e le valvulas de subjectos con elevate pressiones intraventricular produce plus marcate sonos pro un date nivello de fortia que le delicate structuras de juvenile subjectos normal. Multo grande fortias es requirite pro evocar audibile sonos ab pecias de musculo ventricular, e nulle tal nivellos de fortia occurre in animales in vivo.

Le attenuation de sonos cardiac in acre-e ergo in pulmones acrete-es grande. Le attenuation in sanguine, pariete ventricular, e pariete thoracice es si pronunciate que inter 70 e 95 pro cento del intensitate del sono debe esser perdite inter le cuspides mitral e le precordio, mesmo quando le apice es in contacto con le pariete thoracice.

References
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WILLIAM DOCK

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