Investigation of genesis of gallop sounds in dogs by quantitative phonocardiography and digital frequency analysis

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ABSTRACT  Several investigators have noted external gallop sounds to be of higher amplitude than their corresponding internal sounds (S₃ and S₄). In this study we hoped to determine if S₃ and S₄ are transmitted in the same manner as S₁. In 11 closed-chest dogs, external (apical) and left ventricular pressures and sounds were recorded simultaneously with transducers with identical sensitivity and frequency responses. Volume and pressure overload and positive and negative inotropic drugs were used to generate gallop sounds. Recordings were made in the control state and after the various interventions. S₁ and S₄ were recorded in 17 experiments each. The amplitude of the external S₁ was uniformly higher than that of internal S₁ and internal gallop sounds were inconspicuous. With use of Fourier transforms, the gain function was determined by comparing internal to external S₁. By inverse transform, the amplitude of the internal gallop sounds was predicted from external sounds. The internal sounds of significant amplitude were predicted in many instances, but the actual recordings showed no conspicuous sounds. The absence of internal gallop sounds of expected amplitude as calculated from the external gallop sounds and the gain function derived from the comparison of internal and external S₁ make it very unlikely that external gallop sounds are derived from internal sounds.


RECENT INVESTIGATIONS in which high-fidelity, piezoresistive transducers and techniques of quantitative phonocardiography were used have shown that gallop sounds are inconspicuous in intraventricular pressure tracings irrespective of their external intensity. Since sounds are likely to be filtered and reduced in amplitude to a variable degree during their transmission from inside the ventricle to the chest wall, Reddy et al.¹ have postulated that the externally recordable gallop sounds of higher amplitude than internal sounds are not caused by simple passive transmission of intracavity sounds. Before accepting this hypothesis, one has to exclude technical factors that might contribute to the comparatively higher amplitude of externally recorded sounds. There are two major factors that might contribute to this phenomenon: (1) chest wall–transducer resonance, which might vary depending on type, method of application, and pressure of application of the transducers, and (2) the fact that a small external transducer applied with significant pressure and causing obvious indentation of the chest wall may perceive greater pressure because of stretching (shear forces) of the surrounding structures. It is not easily possible to define the degree to which these and other factors contribute to the relatively high amplitude of external gallop sounds. However, if we compare the sounds produced internally to the external recordings, the gain function of the total system could be derived.² We have therefore used first heart sound as a reference to calculate the gain function of the total system. Since the gain function could vary with varying frequencies, the sounds have been analyzed in the frequency domain and then compared to determine the gain function of the system. If the externally recordable gallop sounds exhibit higher amplitude than corresponding internal sounds in spite of correction for the gain function, then it can be assumed that these sounds are not generated within the ventricular cavity.

This experimental study was undertaken to answer

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the following question: Given the gain function of the chest wall–transducer system, which is derived from comparison of internal $S_I$ to external $S_F$, are amplitudes of internal gallop sounds of sufficient magnitude to give rise to external gallop sounds? Since internal gallop sounds are usually not discernible, their amplitude was predicted from external sounds and the gain function. If the amplitude of the internally recorded gallop sound is less than the value predicted from the external sound and the gain function, then it is unlikely that external gallop sounds are caused by simple passive transmission of internal sounds.

**Methods**

**Experimental protocol.** Experiments were performed on 11 mongrel dogs of both sexes weighing 17.1 to 25.4 kg. Anesthesia was induced with a subcutaneous injection of a solution of 0.05 mg/kg fentanyl and 2 mg/kg droperidol (Innovar Vet). Sodium pentobarbital, 20 mg/kg iv, was given to maintain a constant level of anesthesia during the experiment. Each animal was intubated with a cuffed endotracheal cannula and ventilated with room air by means of a Harvard ventilator pump (type 601). A femoral vein was cannulated for drug administration. Standard limb leads were attached for recording lead II of the electrocardiogram. Under fluoroscopy, a Millar micromanometer catheter was inserted into the left ventricle to record left ventricular pressure and the intracardiac phonocardiogram. This transducer has a flat response of from 0 to 20 kHz, 0.5% nonlinearity, and hysteresis.

During the experiments the dogs were in a semilateral lateral recumbent position so that a maximal apexcardiogram (ACG) could be obtained. The quantified ACG was recorded with a high-fidelity, infinite time constant system described previously that has a flat frequency response of 0 to 20 kHz. This system consists of a piezoresistive strain-gauge mounted on a thin stainless steel diaphragm 2 mm in diameter and is calibrated with air pressure by a sphygmomanometer. The apexcardiographic transducer was held to the chest wall at the place of maximal impulse by means of a three-legged holder fastened to the dog with elastic straps. The loading pressure was determined electronically. All recordings were made with a loading pressure of 200 to 400 mm Hg. The quantified ACG was recorded after passing through a low-pass filter with a cutoff frequency of 25 Hz and a roll off of 60 dB/decade. Millar catheters and apexcardiographic transducers were equisensitive, with identical frequency response when calibrated and tested in a liquid-filled chamber.

Intracardiac phonocardiograms obtained from the micromanometer and external phonocardiograms from the apexcardiographic transducer were both recorded by use of identical phonocardiographic transducers. As was the external phonocardiogram, the internal phonocardiogram was quantified in millimeters of mercury.

The electrocardiogram, left ventricular pressure, and intracardiac and external phonocardiograms were recorded simultaneously on an oscillographic strip-chart recorder (Electronics for Medicine) and on magnetic tape with use of an FM recorder (Honeywell 101) at a speed of 3.75 inches/sec. At this speed, the data bandwidth has a flat response (within 1 dB) from 0 to 1250 Hz.

Observations were made during a control period (figure 1), 30 min after onset of anesthesia, during acute volume overload (induced with Ringer's solution), during acute pressure overload (phenylephrine), and after administration of positive (isoproterenol) and negative (propranolol and verapamil) inotropic drugs. The purpose of the pharmacologic interventions was to generate diastolic heart sounds by means of hemodynamic changes.

In 11 experiments a total 66 recordings of effects of the interventions were made. Of these 66 recordings 12 showed $S_3$ sounds, another 12 showed $S_4$ sounds, and five recordings showed both $S_3$ and $S_4$ sounds. An experimental recording in which an external $S_3$ and an $S_4$ were present is shown in figure 2. Peak-to-peak amplitudes were measured for all sounds.

Student’s two tailed t test for paired data was used to calculate statistical probabilities. Group averages were expressed as mean ± SD.

In the frequency domain spectra were calculated for internal and external $S_I$ with Fourier transform methods (figure 3). These spectra were used to calculate a gain function. If present, spectra of external diastolic sounds were also noted (figure 4). Knowing the gain function and with use of an inverse transform,

**FIGURE 1.** Recording of phonocardiograms and pressures in the control situation. LVP = left ventricular pressure; QLAC = quantified left apexcardiogram; $S_I$ = intracardiac phonocardiogram; $S_F$ = external phonocardiogram.

**FIGURE 2.** Experimental recording showing an $S_3$ and $S_4$ on the external phonocardiogram and no discernible deflection during the corresponding periods on the intracardiac tracing. AP = aortic pressure.
amplitude of intracardiac diastolic sounds was calculated from external gallop sounds. Intracardiac gallop sounds were reconstructed in time domain from the calculated intensity frequency spectra (figure 4). Amplitudes of the reconstructed gallop sounds were measured and compared with the actual tracings from the left ventricle (figure 5).

Results

Hemodynamic data for all interventions are summarized in table 1. A typical control recording is shown in figure 1. The intensity of external $S_1$ was significantly ($p < .001$) greater than that of internal $S_1$ (table 2). Usually, heart sound spectra showed one major peak and several smaller peaks at higher frequencies. The peak frequency content of the intracardiac heart sound...
tended to be slightly higher than that of the external sound, but the difference was not statistically significant. External S₃ was discernible in 17 experiments. The intensity of external S₃ ranged between 0.1 and 3.7 mm Hg and was at least 0.5 mm Hg in 14 cases. There were no discernible S₃ sounds on the left ventricular tracings. Amplitude of the internal S₃ was calculated from the external gallop sounds and the gain function derived from comparing the external and internal S₃ sounds of the same beat. The predicted amplitudes of internal S₃ corresponding to the external S₃ ranged from 0.01 to 1.7 mm Hg (mean 0.27 ± 0.39 mm Hg), but there were no discernible S₃ sounds on intracardiac tracings.

**TABLE 1**

<table>
<thead>
<tr>
<th>Intervention</th>
<th>HR (beats/min)</th>
<th>EDP (mm Hg)</th>
<th>LVP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>118 ± 23</td>
<td>15.7 ± 5.7</td>
<td>128.1 ± 25.1</td>
</tr>
<tr>
<td>Volume overload</td>
<td>102 ± 24</td>
<td>34.5 ± 7.7</td>
<td>136.0 ± 18.0</td>
</tr>
<tr>
<td>Isoprenaline</td>
<td>123 ± 31</td>
<td>32.7 ± 16.0</td>
<td>139.0 ± 30.5</td>
</tr>
<tr>
<td>Phenylephrine Cl</td>
<td>113 ± 40</td>
<td>34.7 ± 16.9</td>
<td>169.7 ± 42.1</td>
</tr>
<tr>
<td>Propranolol</td>
<td>89 ± 29</td>
<td>23.8 ± 14.0</td>
<td>122.4 ± 43.8</td>
</tr>
<tr>
<td>Verapamil</td>
<td>86 ± 11</td>
<td>25.3 ± 8.3</td>
<td>87.3 ± 29.4</td>
</tr>
</tbody>
</table>

HR = heart rate; EDP = end-diastolic pressure; LVP = systolic (maximal) left ventricular pressure.

External S₄ sounds were also present in 17 experiments, with an intensity ranging from 0.37 to 8.2 mm Hg; the intensity was at least 1.3 mm Hg in 14 cases. Intensity of calculated internal S₄ ranged from 0.12 to 3.67 mm Hg. There were no distinctly discernible S₄ sounds on internal recordings (figure 2).

**Discussion**

It is generally believed that external cardiac sounds are transmitted intracardiac sounds that have lost some amplitude during their transmission; consequently, it would follow that their amplitude at the source should be higher than their external amplitude. However, recently it has been observed that gallop sounds are of extremely low amplitudes or absent on intracardiac phonocardiograms,¹ ² ³ ⁶ even when the amplitude of the external sounds is high. These observations have raised serious doubts about the intracardiac origin of gallop sounds. However, differences in transducers, filters, and gain used in recording internal and external phonocardiograms, coupled with the lack of a method of calibration of phonocardiographic signals, does not permit valid comparison of amplitude of internal and external phonocardiograms. Quantitative phonocardiographic techniques developed at the University of Pittsburgh¹ ³ ⁷ eliminated the differences between in-
ternal and external phonocardiograms with respect to transducers, filters, and gain. This technique has also permitted calibration of internal and external sounds in units of pressure (mm Hg). In the studies in Pittsburgh, in which similar transducers and identical filters and gains were used to record internal and external phonocardiograms, gallop sounds were found to be absent or of lower amplitude internally. The authors therefore suggested that external gallop sounds were not the result of simple passive transmission of internal sounds with a variable filtration. This conclusion was not totally validated, however, because the method used did not take into consideration the resonance of the chest wall–transducer system and amplification caused by shear forces generated on the surface by the external transducer as a result of stretching of the skin. It is unlikely that resonance of the chest wall lasts more than one cycle and summates over a number of cycles to produce amplification of gallop sounds, but shear forces can amplify the external signal significantly.

Since there is a general agreement that the external S1 is a product of internal S1, we have calculated the "gain function" of the whole system by comparing intracardiac to external S1. This gain function takes into consideration many factors, including frequency, resonance, and shear forces. If gallop sounds are generated within the left ventricular cavity as S1 sounds are thought to be, then the amplitude of intracardiac gallop sounds should be equal to the amplitude calculated from external gallop sounds and the gain function. The limitation of our method is that it does not take into consideration the differences in conduction in the various phases of the cardiac cycle: A stiffened contracted left ventricle during systole has been shown to be better conductor than a relaxed ventricle during diastole. By not taking into consideration these phase differences, if any, we have underestimated the predicted calculated value of internal gallop sounds. Since gallop sounds of calculated amplitude were absent in the internal recordings, it is unlikely that external gallop sounds are simply transmitted amplified internal sounds. Reddy et al. have postulated that the forces generated on the chest wall by cardiac motion in early diastole at the point of measurement of heart sounds are primarily responsible for the rapid filling wave on the quantified ACG, including its higher frequency contents displayed as S3. The cardiac motion may be translational (transverse or axial), rotational, torsional, or expansile. According to these authors, early diastolic thrust (the rapid filling wave) is caused by the expanding moving heart, just as the systolic thrust is caused by the motion of the contracting heart. The energy for diastolic motion may be primarily derived from the elastically stored potential energy generated during systole and the thrust on the chest wall caused by the elastic recoil of the heart may vary from site to site.

Recently Ozawa et al., using uniaxial accelerometers, have demonstrated the exaggerated acceleration and deceleration of the heart (anterior surface and apex) in early diastole in dogs made hypoxic to allow acquisition of S3. They elected to label the exaggerated second derivative of motion (acceleration) the "sound." We believe that the exaggerated motion of the heart in early diastole demonstrated by these authors is one component of the momentum that is responsible for the forces generated on the chest wall that in turn give rise to the rapid filling wave of the ACG (including its high-frequency components displayed as S3). The same force is also responsible for the displacement of the chest wall that, when coupled with the bell of the stethoscope, produces an audible S3. The information regarding motion obtained by these authors is somewhat limited because of their use of uniaxial accelerometers. Understanding of the genesis of gallop sounds can be further improved by the use of triaxial accelerometers and the recordings from multiple sites.

The relationship of the internal to the external S4 was similar to the relationship of the internal to external S1. The fourth heart sound was hardly discernible in internal recordings, irrespective of the amplitude of

### TABLE 2

<table>
<thead>
<tr>
<th>Sound</th>
<th>n</th>
<th>Amplitude</th>
<th>Frequency</th>
<th>Amplitude</th>
<th>Frequency</th>
<th>Amplitude</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>S1</td>
<td>66</td>
<td>10.7 ± 6.7</td>
<td>22 ± 7</td>
<td>2.7 ± 2.7</td>
<td>25 ± 10</td>
<td>0.3 ± 0.4</td>
<td>21 ± 8</td>
</tr>
<tr>
<td>S2</td>
<td>17</td>
<td>1.0 ± 0.8</td>
<td>20 ± 5</td>
<td></td>
<td></td>
<td>1.0 ± 1.1</td>
<td>27 ± 12</td>
</tr>
<tr>
<td>S3</td>
<td>17</td>
<td>2.4 ± 2.0</td>
<td>24 ± 8</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Peak-to-peak amplitudes (mm Hg) of external, intracardiac, and calculated intracardiac heart sounds (n = number of beats) and frequencies (Hz) of spectral maxima.
external S₄. In all cases the amplitude of the internal S₄ was less than expected based on external gallop sounds. The absence of an S₄ of calculated amplitude in internal recordings makes it unlikely that the external S₄ is a result of filtration, resonance, and amplification of the internal S₄. The A wave of the ACG, and its higher frequency components displayed as S₄, are caused by the thrust of the heart on the chest wall as a result of atrial contraction.

In summary, the absence of internal gallop sounds of expected amplitude, calculated from external gallop sounds and gain function derived from comparison of internal and external S₁ sounds, lends credence to the theory that external gallop sounds are not a result of simple passive transmission of internal sounds, and suggests that they may be generated by the thrust of a moving heart on the contiguous structures at the site of detection.

**Appendix 1: smoothing and time windowing phonocardiographic signals**

The digitized data were read onto disk files after smoothing. Smoothing is required to overcome the leakage effects of the finite record lengths and also to remove noise, which is likely to be of higher frequencies. Moreover, an adequate window has lower side lobes in the frequency domain than a rectangular window. Smoothing is performed by subroutines from the signal processing library first by integration and subsequently by differentiation of the array of digital data. It was shown by Aubert et al.⁴ that this procedure results in a low-pass filter with the same transfer function as a von Hann filter given by a cosine bell function

\[
H(f) = \frac{1}{2}(1 + \cos 2\pi f \Delta T)
\]

(1)

where \( f \) = frequency component of the signal; \( T \) = time increment between two adjacent samples points. Its cutoff frequency (-3 dB) is given by

\[
f_c = 0.183/\Delta T
\]

(2)

which corresponds to a frequency of 197.4 Hz for a sampling frequency of 1024 Hz.

The digital data were stored on floppy disk for subsequent processing. The records were visualized on a graphics terminal and different heart sounds were identified by the operator and isolated. The length of the data window must be chosen so that it covers the entire signal to be analyzed. Selection of a data window too close to the beginning or the end of the signal leads to artifacts in the frequency analysis and produces spurious peaks in the spectrum. Also, the baseline of either side of the signal being analyzed should be relatively free of background noise.⁵,⁶ Therefore, the data were gated with a raised cosine window (corresponding to a von Hann function) at both ends⁷ with an amplitude given by

\[
A(l) = \begin{cases} 
\frac{1}{2}(1 + \cos\pi(l - 1)/M) & 0 \leq l \leq M \\
1 & M + 1 \leq l \leq N - M \\
\frac{1}{2}(1 + \cos\pi(1 - (N - M - l))/M) & N - M + 1 \leq l \leq N 
\end{cases}
\]

(3)

where \( N \) = number of digital data points within the indicated heart sound; \( M \) = fraction of total signal to be tapered; \( M + 0.1N \); \( l \) = running number of data point.

This procedure produces a taper at the beginning and at the end of a sound to be isolated. The remaining part of the signal is padded out with zeros. The advantage of use of this window is that it allows analysis of isolated sounds that are completely within the flat portion and eliminates sharp corners in the data going into the FFT.

The effect of introducing the raised cosine window is a reduction in the amplitude of the frequency side lobes.

**Appendix 2**

The gated heart sounds were analyzed in the frequency domain with an FFT available from the Signal Processing Library TEK SPS BASIC (Tektronix). Since the heart sound to be analyzed is always completely contained within the window, the total power will be contained in the spectral estimates and no scaling is necessary after gating. Frequency spectra of intracardiac and external S₁ sounds are determined for all interventions. An example is shown in figure 3.

The chest wall was considered a “black box.” It was assumed to be a linear system with intracardiac and external phonocardiograms as input and output signals. The dynamic characteristics of such a constant-parameter linear system may be described by the weighting function \( h(t) \), which is defined as the output of the system at any time to a unit impulse input applied at a time \( \tau \). For any arbitrary input \( x(t) \) the system output \( y(t) \) is given by the convolution integral

\[
y(t) = \int_{-\infty}^{+\infty} h(\tau)x(t - \tau)d\tau
\]

(4)

That is, the value of the output \( y(t) \) is given as a weighted linear sum over the entire history of the input \( x(t) \). The dynamic characteristics of the system can be described by a frequency-response function \( H(f) \), which is defined as the Fourier transform of \( h(t) \), and is in fact a special case of the transfer function

\[
H(f) = \int_{-\infty}^{+\infty} h(\tau)e^{-2\pi i f \tau} d\tau
\]

(5)

where \( j = \sqrt{1} \). Equation 4 can be modified by taking the Fourier transform of both sides

\[
Y(f) = H(f)X(f)
\]

(6)

where \( X(f) \) is the Fourier transform of the input \( x(t) \) and \( Y(f) \) is the Fourier transform of the resulting output \( y(t) \). Hence, in terms of the frequency-response function of a system and the FFT of the input and output, the convolution integral in equation 4 reduces to the simple algebraic expression in equation 6. The frequency-response function is generally a complex valued quantity and can be written as:

\[
H(f) = | H(f) | e^{-j\theta(f)}
\]

(7)

The absolute value \( | H(f) | \) is the system gain factor and the associated phase angle \( \theta(f) \) is the system phase factor.

The input signal is the intracardiac \( S_1 \) and the output the external \( S_1 \) (chest wall phonocardiogram). Their spectra were determined by an FFT subroutine and the frequency-intensity spectrum \( S_1 \) was displayed on linear scales (figure 3). From equation 6 the complex quantity \( H(f) \) was calculated. Finally, if present, externally recorded diastolic sounds were used as output signals. An experimental recording, in which an external \( S_1 \) as well as an internal \( S_4 \) are present, is shown on figure 2. After determination of their frequency content the corresponding intracardiac sounds were calculated in the frequency domain (figure 4).

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after inverse transformation their time series representation was obtained (figure 5).

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