SUMMARY

The aortic valve echogram was recorded in open chest dogs simultaneously with aortic flow, acceleration of flow, aortic and left ventricular pressures, and intracardiac phonocardiograms.

Comparison of echographic with hemodynamic data showed the following: The aortic valve started its opening with the onset of flow, at the same point that left ventricular pressure exceeded aortic pressure. Complete valve opening preceded peak aortic flow by an average of 43 msec and the cusps started to move toward closure while flow was still accelerating. Final closure of the valve was achieved at the time of zero flow and preceded the aortic second sound by 4-10 msec. The echographically determined "valve orifice area" correlated well with aortic stroke volume ($r = 0.94$).

The intensity of the aortic first sound was related to peak acceleration of aortic flow. The intensity of the second sound was not related to the amplitude of cusp motion but correlated well with the aortic pressure at the time of closure and with peak flow deceleration.

Hemodynamic Correlates of the Normal Aortic Valve Echogram

A Study of Sound, Flow, and Motion

Shlomo Laniado, M.D., Edward Yellin, Ph.D., Reuven Terdiman, M.Sc., Ilan Meytes, M.D., and Jona Stadler, M.D.

UNTIL RECENTLY most of the studies describing the dynamic behavior of the normal aortic valve were done either with models or at autopsy. Attempts have also been made to infer the hemodynamics of the normal valve from studies of prosthetic valves.

Following the demonstration by Edler that motion of the aortic cusps can be recorded by ultrasound technique, the systematic studies of Gramiak and Shah helped to delineate the functional anatomy of the normal aortic valve as seen in the echogram.

A controversial issue has been the genesis of the major component of the second heart sound and its relation to aortic flow and valve closure. While it is generally believed that valve closure and sudden impedance to back flow are responsible for the production of sound vibrations whether the sound precedes or follows valve closure has not been established.

In this study we correlate the dynamics of aortic cusp motion, as recorded by the echogram, with changes in instantaneous transvalvular flow and stroke volume. Particular emphasis is placed on opening and closing events and the relationship between sound vibrations and pressure, flow, and cusp motion.

Material and Methods

Fifteen dogs, 19-25 kg, were anesthetized with pentobarbital (30 mg/kg), the chest was opened at the sternum and the heart exposed. Left ventricular and aortic pressures were measured with high-fidelity catheter-tipped transducers (Micro-Tip model PC-350, Millar Instruments). The left ventricular catheter was inserted through an apical puncture and access to the aorta was gained by retrograde passage via the right common carotid artery. Both catheters were adjusted for common zero and equal sensitivity, which was checked repeatedly throughout the experiments by momentarily positioning the aortic catheter in the left ventricle or by advancing the left ventricular catheter into the aorta above the valve. A short stiff catheter connected to Statham P23Db pressure transducer was introduced into the left atrium via a pulmonary vein for measurements of left atrial pressure.

An electromagnetic flow probe was placed around the ascending aorta just above its root and instantaneous aortic flow was recorded by a square wave electromagnetic flow-meter (Model 501, Carolina Medical Electronics). The fre-
Table 1. Hemodynamic and Echographic Data from Six Dogs

<table>
<thead>
<tr>
<th>Dog</th>
<th>CO (L/min)</th>
<th>HR</th>
<th>AoP (mm Hg)</th>
<th>AoVol (ml/beat)</th>
<th>AoF (ml/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Stroke</td>
<td>Opening</td>
<td>% opening</td>
<td>Peak</td>
<td>Opening</td>
</tr>
<tr>
<td>1</td>
<td>1.50</td>
<td>70</td>
<td>106</td>
<td>94.7</td>
<td>22.8</td>
</tr>
<tr>
<td></td>
<td>= 0.07</td>
<td>±</td>
<td>± 1.01</td>
<td>± 0.62</td>
<td>± 1.00</td>
</tr>
<tr>
<td>2</td>
<td>2.52</td>
<td>75</td>
<td>101</td>
<td>86.5</td>
<td>33.6</td>
</tr>
<tr>
<td></td>
<td>± 0.11</td>
<td>±</td>
<td>± 2.20</td>
<td>± 1.84</td>
<td>± 1.44</td>
</tr>
<tr>
<td>3</td>
<td>1.58</td>
<td>63</td>
<td>68</td>
<td>49.3</td>
<td>25.1</td>
</tr>
<tr>
<td></td>
<td>± 0.05</td>
<td>±</td>
<td>± 2.08</td>
<td>± 0.96</td>
<td>± 0.88</td>
</tr>
<tr>
<td>4</td>
<td>2.67</td>
<td>108</td>
<td>134</td>
<td>116.4</td>
<td>24.7</td>
</tr>
<tr>
<td></td>
<td>± 0.30</td>
<td>±</td>
<td>± 2.86</td>
<td>± 4.09</td>
<td>± 2.80</td>
</tr>
<tr>
<td>5</td>
<td>2.09</td>
<td>95</td>
<td>94</td>
<td>74.0</td>
<td>23.7</td>
</tr>
<tr>
<td></td>
<td>± 0.10</td>
<td>±</td>
<td>± 2.38</td>
<td>± 3.09</td>
<td>± 1.09</td>
</tr>
<tr>
<td>6</td>
<td>2.16</td>
<td>125</td>
<td>84</td>
<td>73.2</td>
<td>17.3</td>
</tr>
<tr>
<td></td>
<td>± 0.11</td>
<td>±</td>
<td>± 3.15</td>
<td>± 2.48</td>
<td>± 0.90</td>
</tr>
<tr>
<td>Mean</td>
<td>2.10</td>
<td>90</td>
<td>97</td>
<td>82</td>
<td>24.1</td>
</tr>
<tr>
<td>± SE</td>
<td>0.19</td>
<td>9.80</td>
<td>9.10</td>
<td>9.30</td>
<td>2.20</td>
</tr>
</tbody>
</table>

*At the time of dicrotic notch.

Abbreviations: CO = cardiac output; HR = heart rate; Syst = systolic; Diast = diastolic; VOA = valve orifice area; accel = acceleration; decel = deceleration AoF = aortic flow; AoP = aortic pressure; max = maximal; amp = amplitude.

The frequency response of the flow meter was selected at 30 Hz, with a time lag of less than 3 msec. The velocity curve recorded by the probe was the true reflection of actual flow since the area circumscribed by the aortic flow probe was constant and the probe was tightly placed around the aorta. The time derivative of the aortic flow (dQ/dt), obtained by electronic differentiation, was used to measure its acceleration or deceleration.

Sound vibrations from the aorta and left ventricle were obtained from the high fidelity pressure transducers by filtering their output using a method described previously to allow the recording of vibrations between 40-500 Hz.

The aortic valve echogram was obtained with a 3.5 MHz focused transducer 10 mm in diameter and a Unirad Series 100 echocardiograph. After achieving the characteristic pattern of the anterior mitral cusp echogram, using a technique that has already been described, the transducer was tilted medially and anteriorly following the anatomical continuity between the anterior cusp of the mitral valve and the posterior aortic wall until optimal registration of the aortic cusps, presumably the right and noncoronary, was recorded at a paper speed of 75-150 mm/sec.

The effects of changes in peripheral resistance and cardiac output on the dynamics of the aortic valve were investigated following administration of pharmacologic agents (isoproterenol 0.1 μg/kg/min or phenylephrine 1-2 μg/kg/min), or changes in left ventricular filling volume.

Complete echographic and hemodynamic data were obtained in six dogs and only partial data in the remaining nine, due to the complexity of the experiments and the difficulty in obtaining a good aortic valve echogram in the open-chest dog.

Calculations

Figure 1 is a schematic representation of the normal relations between pressure, flow, sound, and motion. Stroke volume was calculated by planimetry of the area under the calibrated aortic flow curve. Peak flow was measured as the highest point of the aortic flow curve (arrow 1), opening flow was defined as the instantaneous flow at the time of full valve opening (arrow 2) and opening volume was the amount of

![Figure 1. Schematic representation of the relations between the hemodynamic data and the aortic valve echogram (AoVE). dQ/dt = rate of rise of aortic flow. Point 1 marks the peak aortic flow. Point 2 = aortic flow at the time the aortic cusps achieved their maximal amplitude of opening. Area 3 = opening volume of the valve. 4 = vibrations of the aortic valve following closure, observed in some experiments. A1 = aortic component of the first heart sound. A2 = aortic second sound. [t1, t2] = duration of the ejection period. [t1, t2] = duration of the opening movement of the cusps.]
hemodynamics of aortic valve / laniado et al.

<table>
<thead>
<tr>
<th>Amp cusp separation (mm)</th>
<th>Speed of cusp motion (mm/sec)</th>
<th>Time from systolic closure to systolic closure (sec)</th>
<th>Time from systolic closure to aortic valve closure (sec)</th>
<th>Time from valve closure to systolic closure (sec)</th>
<th>Peak accel of AoF (mm/sec²)</th>
<th>Peak decel of AoF (mm/sec²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>In opening</td>
<td>Before closure</td>
<td>Opening</td>
<td>During slow systolic closure</td>
<td>In final closure</td>
<td>Max Amp of cusp motion</td>
<td>Peak aortic flow</td>
</tr>
<tr>
<td>11.5</td>
<td>8.2</td>
<td>290</td>
<td>64</td>
<td>219</td>
<td>0.53</td>
<td>0.009</td>
</tr>
<tr>
<td>± 0.90</td>
<td>± 0.70</td>
<td>± 9.52</td>
<td>± 7.63</td>
<td>± 5.62</td>
<td>± 0.03</td>
<td>± 0.0010</td>
</tr>
<tr>
<td>15.1</td>
<td>8.8</td>
<td>342</td>
<td>85</td>
<td>316</td>
<td>0.65</td>
<td>0.008</td>
</tr>
<tr>
<td>± 0.50</td>
<td>± 0.40</td>
<td>± 18.30</td>
<td>± 6.18</td>
<td>± 21.2</td>
<td>± 0.02</td>
<td>± 0.0008</td>
</tr>
<tr>
<td>11.9</td>
<td>8.0</td>
<td>277</td>
<td>70</td>
<td>290</td>
<td>0.58</td>
<td>0.015</td>
</tr>
<tr>
<td>± 0.30</td>
<td>± 0.40</td>
<td>± 6.20</td>
<td>± 7.44</td>
<td>± 6.74</td>
<td>± 0.08</td>
<td>± 0.0030</td>
</tr>
<tr>
<td>12.0</td>
<td>9.3</td>
<td>275</td>
<td>70</td>
<td>247</td>
<td>0.55</td>
<td>0.013</td>
</tr>
<tr>
<td>± 0.90</td>
<td>± 0.70</td>
<td>± 2.18</td>
<td>± 11.0</td>
<td>± 4.13</td>
<td>± 0.02</td>
<td>± 0.0020</td>
</tr>
<tr>
<td>11.0</td>
<td>9.3</td>
<td>230</td>
<td>56</td>
<td>196</td>
<td>0.47</td>
<td>0.008</td>
</tr>
<tr>
<td>± 0.40</td>
<td>± 0.60</td>
<td>± 12.10</td>
<td>± 5.34</td>
<td>± 7.81</td>
<td>± 0.09</td>
<td>± 0.0017</td>
</tr>
<tr>
<td>12.3</td>
<td>9.0</td>
<td>241</td>
<td>60</td>
<td>212</td>
<td>0.43</td>
<td>0.0095</td>
</tr>
<tr>
<td>± 0.60</td>
<td>± 0.50</td>
<td>± 13.30</td>
<td>± 9.11</td>
<td>± 9.13</td>
<td>± 0.06</td>
<td>± 0.0009</td>
</tr>
<tr>
<td>12.4</td>
<td>8.6</td>
<td>270.8</td>
<td>67.5</td>
<td>237</td>
<td>0.53</td>
<td>0.0266</td>
</tr>
<tr>
<td>0.60</td>
<td>0.17</td>
<td>16.1</td>
<td>4.16</td>
<td>17.4</td>
<td>0.03</td>
<td>0.0008</td>
</tr>
</tbody>
</table>

Blood that had passed through the valve by the time the cusps achieved their full excursion of opening (fig. 1, shaded area). The amplitude of valve opening was measured at the point where the maximal distance between the two cusps was achieved at the beginning of systole (E-E' in fig. 1) and the amplitude of closing was taken as the distance between the cusps at the point where they started their rapid movement toward closure (B-B' in fig. 1). The speed of cusp motion during the events of opening or closing was measured from the slopes of the different segments of the echogram: The O-E slope gave the rate of valve opening, the E-B slope gave the speed of the slow systolic valve closure, and the B-C slope was the rate of final valve closure at the end of systole. "Valve Orifice Area" was used to describe the amount of valve opening during systole and was defined as the area circumscribed by the echograms of the two aortic cusps during systole. This area was measured by planimetry and was assigned arbitrary units for the purpose of comparing different beats.

In any given experiment there was no change in the sensitivity of the intraaortic sound recording system since the position of the aortic catheter was not changed relative to the aortic valve and thus it was possible to compare variations in sound intensity in the same experiment by measuring the maximal amplitude of the major sound components. During the short periods of recording the respirator was turned off to avoid unwanted movements and vibrations.

Statistical Methods

Linear regression analysis was performed using a Monroe 1860 statistical programmable calculator.

To estimate the effects of diastolic blood pressure (BP), deceleration of aortic flow (dQ/dt), and stroke volume (SV) on the intensity of the second sound (S₂), multiple regression analysis was performed. The second sound was considered as a function of the above independent variables (S₂ = a + b₁ BP + b₂ dQ/dt + b₃ SV). We looked for interrelationships between the variables; thus we considered the multiple correlation and the partial correlation coefficients.⁴⁴

Results

The Relationship of Aortic Flow to Cusp Motion

Figures 2 and 3 are original records taken from two different dog experiments demonstrating typical relations between flow, sound, pressure, and the aortic valve echogram. Table 1 summarizes the hemodynamic and echographic data in the six dogs on which all data were obtained. In each experiment, measurements were made under stable conditions on 20 consecutive beats; the average ± SEM are given.

The initial opening motion of the aortic valve occurred simultaneously with the onset of aortic flow and at the point that left ventricular pressure exceeded aortic pressure. The opening motion of the aortic valve was smooth, with an average speed of 270 mm/sec, and the cusps achieved their

**Figure 2.** An original record showing the relations between the aortic flow (AoF), acceleration or deceleration of flow (dQ/dt), pressure, intracavitary phonocardiograms, and the aortic valve echogram. Following the achievement of maximal opening the cusps start immediately to move slowly toward closure. Final closure occurred at zero flow and about 5 msec before the aortic dicrotic notch and the start of the major component of the second sound. Following closure, and at the time of the second sound, the cusps vibrated slightly. Timelines are 40 msec apart. Paper speed is 150 mm/sec. (Dog #4)
peak amplitude of excursion while flow was still accelerating at an average of 43 msec before peak flow. Full valve opening was achieved when the velocity of the flowing blood was 40% of its peak and when 5–9% of the total stroke volume had passed through the valve.

Following the completion of the opening movement the cusps started moving slowly toward closure. This systolic closing motion (E-B slope) was faster, and thus more apparent, in beats with small stroke volume, but nevertheless was a constant phenomenon persisting in every beat, though to different degrees. In some experiments the echogram showed a small but rapid movement toward closure occurring immediately following opening, resembling an "overshoot" (point E, fig. 1). Final closure of the valve was achieved at the time of zero flow and the average speed of cusp motion during final closure was 237 mm/sec.

When stroke volume varied from beat to beat, as seen in arrhythmias, it was demonstrated that with larger stroke volume the rate of aortic valve opening was faster, the maximal amplitude of excursion occurred sooner and the cusps achieved wider separation. Figure 4 demonstrates the relationship between stroke volume and the rate of cusp opening in 65 beats from a dog in which there was a marked variation in stroke volume due to arrhythmia (r = 0.80), and figure 5 gives the relationship between the speed of valve opening and acceleration of aortic flow (r = 0.87) in the same dog. The correlation between aortic stroke volume and the echographically determined valve orifice area is presented in figure 6 (r = 0.94) and demonstrated in an original record taken from another dog (fig. 7) where the stroke volume is spontaneously decreasing. In each case the above relationships are based on data from a single animal, not a group. However these results were reproducible in other dogs as can be seen from table 2 which demonstrates

**Figure 3.** This record clearly demonstrates vibrations of the closed valve which came simultaneously with the dicrotic notch in the aortic pressure and the second sound. Timelines are 100 msec apart. Paper speed is 100 mm/sec. (Dog #6)

**Figure 4.** The rate of cusp opening movement is plotted against the stroke volume in 65 beats in the same experiment in which arrhythmia resulted in marked variations in stroke volume. The best-fitting regression (straight) line is \( Y = 12.98x + 70.55 \).

**Figure 5.** A linear relation was found to exist between the speed of aortic valve opening movement and peak acceleration of aortic flow \( Y = 39.2X + 98.9 \) (Dog #4).
that there were no significant differences between the various coefficients of correlation in five different dogs. Dog 4 was selected as representative for the graphic illustrations.

Analysis of Aortic Sounds

Temporal Relation of Aortic Sound Vibrations to Opening and Closing Events

Examination of the aortic component of the first sound revealed that it started before the onset of aortic valve opening and acceleration of flow (table 1 and fig. 2).

The echogram clearly shows a close temporal relation between the aortic valve closure, the dicrotic notch of the aortic pressure, and the large amplitude vibrations of the second sound (figs. 2, 3). However there was a short time lag, in the range of 4–10 msec (table 1), between the point of the initial cuspid apposition and the beginning of the major sound component and the dicrotic notch. Some records demonstrated a rapid vibrating motion of the sealed cusps which started immediately following closure and occurred simultaneously with the major sound component (fig. 3).

Factors Responsible for the Intensity of the Aortic Sounds

The relative intensity of the major vibration of the aortic sounds was obtained by measurements of the amplitude of the phonocardiograms recorded from a location just above the valve, where the vibrations indicated sounds originating in the vicinity of the valve. The mitral component of the first heart sound could be recognized and differentiated from the aortic component since it came earlier and its amplitude in the aortic phonon was smaller than that in the left ventricular phonon.

Table 3 summarizes hemodynamic and echographic data from a dog in which changes in left ventricular filling volume and the effects of pharmacologic agents (isoproterenol and

<p>| TABLE 2. Coefficients of Correlation Between the Different Variables in Five Dogs |
|-----------------------|-------|-------|-------|-------|-------|-------|-------|-------|</p>
<table>
<thead>
<tr>
<th>Dog</th>
<th>SV/cusp rate</th>
<th>Peak accel AoF/ cusp rate</th>
<th>SV/VOA</th>
<th>Peak accel AoF/ Amp Ao S1</th>
<th>AoP/amp hf S1</th>
<th>SV/amp hf S1</th>
<th>Decel AoF/ amp hf S1</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.73</td>
<td>0.90</td>
<td>0.91</td>
<td>0.80</td>
<td>0.91</td>
<td>0.81</td>
<td>0.91</td>
</tr>
<tr>
<td>3</td>
<td>0.83</td>
<td>0.81</td>
<td>0.90</td>
<td>0.79</td>
<td>0.93</td>
<td>0.77</td>
<td>0.92</td>
</tr>
<tr>
<td>4</td>
<td>0.80</td>
<td>0.87</td>
<td>0.94</td>
<td>0.84</td>
<td>0.95</td>
<td>0.79</td>
<td>0.94</td>
</tr>
<tr>
<td>5</td>
<td>0.75</td>
<td>0.75</td>
<td>0.95</td>
<td>0.82</td>
<td>0.97</td>
<td>0.80</td>
<td>0.90</td>
</tr>
<tr>
<td>6</td>
<td>0.84</td>
<td>0.80</td>
<td>0.88</td>
<td>0.78</td>
<td>0.86</td>
<td>0.82</td>
<td>0.96</td>
</tr>
</tbody>
</table>

Dog #2 is excluded because limited variations in rhythm, rate and blood pressure during the experiment resulted in mean data only (table 1).

Abbreviations: r = rate of cusp opening movement; Peak accel AoF = peak acceleration of aortic flow; Amp AoS1 = amplitude of aortic component of first heart sound; AoP = aortic diastolic pressure; amp hf S1 = amplitude high frequency component of second heart sound; Decel AoF = peak of deceleration of aortic flow. For others see table 1.
phenylephrine) induced marked variations in stroke volume and/or aortic pressure. Twenty beats of each phase of the experiment were analyzed and four groups of beats with different stroke volume and aortic pressure are presented.

The intensity of the aortic component of the first sound appeared to have a smaller range of variation than the second sound. It was not affected by the aortic blood pressure and little affected by peak flow ($r = 0.52$) and stroke volume ($r = 0.76$). It was more closely associated with changes in acceleration of aortic flow as shown in figure 8 which correlates the amplitude of the aortic component of the first sound and peak acceleration of aortic flow ($r = 0.84$). Figure 7 is an example from a single dog of the relationship between flow acceleration ($dQ/dt$) and the amplitude of the aortic first sound. In the weaker beats flow accelerates slowly and the first sound is reduced in its intensity.

The intensity of the second aortic sound did not correlate with the maximal amplitude of cusp opening (table 3, fig. 10). Figures 9, 10, 11, and 12 demonstrate the effects of diastolic aortic pressure (at time of valve closure), stroke volume, and peak deceleration of aortic flow on the intensity of the second sound. These results, obtained from one dog (experiment 4), were reproducible in the other dogs (table 2).

In a single dog, the components, peak deceleration of aortic flow, stroke volume, and aortic blood pressure, were correlated with the intensity of $S_2$ over 35 beats (tables 4–6). Aortic blood pressure and peak deceleration of aortic flow exerted the major influence over intensity of the second heart sound.

**Discussion**

The normal aortic valve echogram is usually described as having the configuration of a “box” with two parallel lines reflecting the movement of the right and noncoronary cusps during the period of ejection. However, since Gramiak and Shah showed that the amplitude of the opening movement of the valve is greater than that of closing, the orifice must be decreasing in size during midstroke, with the margins of the cusps moving toward each other. This phenomenon of early closure of the valve during systole was observed in all our experiments. A review of our patient records and records presented in the literature further confirm this observation.

In their study using an aortic valve model, Bellhouse and Bellen found that the valve opened rapidly, before peak flow; stayed open until after peak flow; and moved toward closure after flow started to decelerate. They postulated a vortex mechanism which formed in the sinuses and contributed to efficient valve closure. Our results show a different temporal relation. When the ventricular pressure exceeds the aortic pressure, flow starts and the valve opens widely, achieving its maximum opening before peak flow, and starting its slow movement toward closure while flow is still accelerating. It appears that internal forces caused by the displacement of the cusps, in combination with vortices in the sinuses, cause the leaflets to begin to close before flow begins to decrease.

In low output states (fig. 7), or in premature ventricular contractions in patients, the forces applied to the leaflets by the fluid are reduced during both acceleration and deceleration and the cusps move toward apposition more rapidly under the action of external and internal forces.

Unlike the mitral valve which may stay open during diastole even with no flow across it, our results show that the aortic valve does not stay open unless forward flow is passing through it. This observation indicates that substantial internal forces exist in the opened aortic valve and its equilibrium position, in vivo, is closed.

**TABLE 3. Hemodynamic, Echographic and Phonocardiographic Data from a Single Dog Following Changes in Blood Pressure and Stroke Volume**

<table>
<thead>
<tr>
<th>CO (L/min)</th>
<th>HR</th>
<th>Synt</th>
<th>Diast*</th>
<th>Stroke</th>
<th>Opening</th>
<th>% opening</th>
<th>Peak</th>
<th>Opening</th>
<th>% opening</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.66</td>
<td>108</td>
<td>134</td>
<td>116.4</td>
<td>24.7</td>
<td>1.19</td>
<td>4.87</td>
<td>83.8</td>
<td>34.20</td>
<td>41.2</td>
</tr>
<tr>
<td>± 0.30</td>
<td></td>
<td>± 2.86</td>
<td>± 4.09</td>
<td>± 2.80</td>
<td>± 0.09</td>
<td>± 0.34</td>
<td>± 2.73</td>
<td>± 0.38</td>
<td>± 0.67</td>
</tr>
<tr>
<td>2.24</td>
<td>120</td>
<td>97</td>
<td>81.4</td>
<td>18.7</td>
<td>0.93</td>
<td>5.01</td>
<td>119.1</td>
<td>55.74</td>
<td>46.8</td>
</tr>
<tr>
<td>± 0.19</td>
<td></td>
<td>± 3.71</td>
<td>± 2.94</td>
<td>± 1.59</td>
<td>± 0.07</td>
<td>± 0.18</td>
<td>± 3.29</td>
<td>± 1.29</td>
<td>± 1.29</td>
</tr>
<tr>
<td>2.68</td>
<td>112</td>
<td>87</td>
<td>52.5</td>
<td>23.9</td>
<td>1.59</td>
<td>8.08</td>
<td>83.9</td>
<td>35.42</td>
<td>40.2</td>
</tr>
<tr>
<td>± 0.16</td>
<td></td>
<td>± 2.03</td>
<td>± 3.44</td>
<td>± 1.46</td>
<td>± 0.07</td>
<td>± 0.43</td>
<td>± 2.89</td>
<td>± 1.87</td>
<td>± 1.36</td>
</tr>
<tr>
<td>1.51</td>
<td>90</td>
<td>72</td>
<td>30.1</td>
<td>16.8</td>
<td>1.10</td>
<td>6.56</td>
<td>65.0</td>
<td>30.75</td>
<td>47.3</td>
</tr>
<tr>
<td>± 0.12</td>
<td></td>
<td>± 2.01</td>
<td>± 1.18</td>
<td>± 0.78</td>
<td>± 0.17</td>
<td>± 0.08</td>
<td>± 4.40</td>
<td>± 1.65</td>
<td>± 0.39</td>
</tr>
</tbody>
</table>

*at time of dicrotic notch.
For abbreviations see table 1.

**FIGURE 8. The relationship between the intensity of the aortic component of the first sound and peak acceleration of aortic flow.**

The best-fitting regression line is $Y = 0.46x + 0.36$. (Dog 4).
Our studies also show that echographically determined "aortic valve orifice area" is a reasonably accurate reflection of aortic flow and stroke volume; this is in contrast to the situation in the mitral valve. This finding, which confirms previous observations by Feigenbaum and by Yeh et al., may be useful in the clinical situation (provided an adequate aortic valve echogram is obtained) to estimate relative changes in stroke volume and cardiac output in an individual patient following arrhythmias, pacing at different rates, etc.

Origin of the Aortic Sounds

Our analysis of aortic valve sounds was based on the study of vibrations registered in the vicinity of the valve by an intracavitary high-fidelity transducer. We are aware of the possibility that the recorded vibrations may not be the true replica of external aortic sounds. However, since these vibrations were registered from a site as close as possible to the valve, they are probably the best representatives of true valvular sound activity.

The aortic component of the first heart sound was found to be clearly related in its intensity to acceleration of aortic flow. This supports previous suggestions that early acceleration of the ejected blood is responsible for vibrations originating in the aortic root. There was no fixed temporal relation between this sound and the time at which the aortic cusps achieved their maximal amplitude of opening (fig. 7). This confirms Waider and Craig's recent observations that in the normal aortic valve the cusps do not participate in the genesis of the first sound, and the findings of

![Figure 10](image_url)

**Figure 10.** This record presents two beats taken from the same experiment. The right panel was recorded following infusion of phenylephrine which caused an increase in peripheral resistance and blood pressure, and reduction in stroke volume. The traces represent (from top to bottom) aortic flow, aortic and left ventricular phonocardiograms, aortic pressure, left ventricular and atrial pressures, and the aortic valve echocardiogram. Despite the reduction in stroke volume and the amplitude of cusps' separation, the amplitude of the second sound was much greater (this can be seen in both the aortic phono and the LV phono) and correlated with the increase in the aortic pressure. (Dog #4).
Whittaker et al.\textsuperscript{22} that aortic ejection sounds heard in subjects with no aortic valve disease are the exaggerated ejection component of the first sound.

Since our aortic phonocardiogram did not record sounds originating in the vicinity of the tricuspid valve, we cannot appreciate the contribution of tricuspid valve closure to the second major component of the surface first sound.

The origin of the aortic component of the second heart sound is more controversial. The classic explanation attributes the first high frequency vibrations of the second sound to closure of the aortic valve and the sudden impedance to back flow.\textsuperscript{23} Several investigators have challenged this explanation. Piemme et al.\textsuperscript{24} postulated that the aortic second sound starts significantly before the aortic valve closure, while forward flow was still going on, and that it is the sudden deceleration of aortic forward flow that is responsible for the second sound. However their assumption that the onset of the second sound occurs 25 msec before valve closure was apparently incorrect. They mistakenly assumed that valve closure was accomplished at the time of the nadir point in the aortic flow curve. In fact, valve closure occurs earlier, at the time of zero flow, and the negative dip in the aortic flow is due to "cocking" of the closed valve. Even their own record (fig. 4, in reference 7) demonstrated that the dicrotic notch at the aortic pressure curve came significantly before the nadir of the aortic flow. This was also evident in all our tracings.

While many workers described a close temporal associa-

\begin{table}[h]
\centering
\caption{Correlation Coefficient of Second Heart Sound and Three Variables*}
\begin{tabular}{|c|c|}
\hline
Variables & \(r\) \\
\hline
\(S_2\) BP & 0.94 \\
\(dQ/\text{dt}\) & 0.96 \\
\(S_2\) SV & 0.80 \\
BP & 0.94 \\
\(dQ/\text{dt}\) & 0.79 \\
BP, SV & 0.79 \\
\(dQ/\text{dt}\) & 0.79 \\
\hline
\end{tabular}
\textsuperscript{*}Derived from 35 beats in a single animal.
\end{table}

\begin{table}[h]
\centering
\caption{Partial Correlation Coefficients of Second Heart Sound and Three Variables}
\begin{tabular}{|c|c|c|c|}
\hline
Variables & \(r\) & Variables & \(r\) & Variables & \(r\) \\
\hline
\(S_2\) BP & 0.66 & \(S_2\) BP & 0.39 & \(S_2\) BP & 0.84 \\
\(dQ/\text{dt}\) & \(S_2\) BP & 0.39 & \(dQ/\text{dt}\) & 0.89 \\
\(S_2\) SV & 0.27 & \(S_2\) SV & 0.24 & \(S_2\) SV & 0.24 \\
BP & \(dQ/\text{dt}\) & 0.23 & BP, SV & 0.23 & BP, \(dQ/\text{dt}\) & 0.84 \\
\hline
\end{tabular}
\end{table}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure11.png}
\caption{The relationship between the amplitude of the second heart sound and stroke volume. (\(Y = 0.43x + 1.58\)). (Dog \#4).}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure12.png}
\caption{The relation between the amplitude of the second sound and the maximal deceleration of aortic flow; \(r = 0.94 \pm 0.02\). (\(Y = 2.50x + 4.62\)). (Dog \#4).}
\end{figure}

\section*{TABLE 4. Correlation Coefficient of Second Heart Sound and Three Variables*}

<table>
<thead>
<tr>
<th>Variables</th>
<th>(r)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(S_2) BP</td>
<td>0.94</td>
</tr>
<tr>
<td>(dQ/\text{dt})</td>
<td>0.96</td>
</tr>
<tr>
<td>(S_2) SV</td>
<td>0.80</td>
</tr>
<tr>
<td>BP, (S_2) SV</td>
<td>0.79</td>
</tr>
<tr>
<td>BP, (dQ/\text{dt})</td>
<td>0.79</td>
</tr>
</tbody>
</table>

*Derived from 35 beats in a single animal.

Abbreviations: \(S_2\) = intensity of second aortic sound; \(dQ/\text{dt}\) = peak deceleration of aortic flow; BP = Aortic pressure at time of valve closure; SV = stroke volume.

tion between the first major group of vibrations of the second sound and the dicrotic notch in the aortic pressure,\textsuperscript{24, 26} MacCanon and coworkers, using a special electronic contacting device, showed that the aortic valve closed an average of 8 msec before the dicrotic notch in closed-chest dogs.\textsuperscript{11}

Criley et al. demonstrated by rapid cineangiographic films that the start of the second sound coincided with a sharp descent and distension of the aortic valve and root, few milliseconds following valve closure,\textsuperscript{28} and Chandraratna and co-workers, using simultaneous recording of phonocardiograms and aortic valve echograms in patients, showed that the aortic valve closure preceded the aortic component of the second sound by an average of 12 msec.\textsuperscript{27} These observations were recently supported by the study of Anastasiades et al.\textsuperscript{28}

Our records showed that the dicrotic notch of the aortic pressure followed the initial point of cusp apposition by about 4-10 msec and was accompanied by the major component of the aortic sound, which was seen in some experiments to occur simultaneously with a rapid vibrating motion of the sealed aortic cusps (figs. 2, 3).

The intensity of the second sound was not related to the cusps' amplitude of motion but was closely associated with the rate of flow deceleration and the aortic pressure at the time of valve closure.

These observations suggest that the second sound originates in the aortic valve but not at the time of initial closure and cusp apposition but shortly after, when the sealed cusps tense and vibrate under the action of a rapidly applied force.
The findings that the amplitude of the aortic component of the second sound is proportional to the level of the aortic pressure at the time of closure (fig. 9) is consistent with the common clinical observation of louder aortic second sound in states of systemic hypertension.

In summary, our observations emphasize that while movement of the normal aortic valve does not contribute to the first heart sound, it has a major role in the genesis of the second sound. However, unlike the situation in the mitral valve in which the intensity of the first heart sound was found to be related to valve position and amplitude of motion, the intensity of the aortic sound is not related to the magnitude of aortic cusp motion at closure. The functional anatomy of the mitral and aortic valves are entirely different. The mitral valve cusps are attached to the chordae tendineae and a smaller amplitude of closing movement is expected to generate less tension in the combined apparatus of cusps and cords at the final point of closure. The aortic cusps are unrestrained structures that tend to stay in the closed position, and the intensity of their vibrations is directly related to the forces acting on them following the completion of closure.

References
5. Edler I, Gustafson A, Karleforts T, Christensson B: Mitral and aortic

Table 6. Partial Correlation Coefficients of Second Heart Sound and Three Variables

<table>
<thead>
<tr>
<th>Variables</th>
<th>r</th>
</tr>
</thead>
<tbody>
<tr>
<td>$S_0$, SV (BP, dQ/dt held constant)</td>
<td>0.73</td>
</tr>
<tr>
<td>$S_0$, dQ/dt (BP, SV held constant)</td>
<td>0.96</td>
</tr>
<tr>
<td>$S_0$, BP (dQ/dt, SV held constant)</td>
<td>0.94</td>
</tr>
</tbody>
</table>

HEMODYNAMICS OF AORTIC VALVE/Laniado et al. 737

Hemodynamic correlates of the normal aortic valve echogram. A study of sound, flow, and motion.

S Laniado, E Yellin, R Terdiman, I Meytes and J Stadler

_Circulation_. 1976;54:729-737
doi: 10.1161/01.CIR.54.5.729

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1976 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/54/5/729