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Exploration of the Cause of the Low Intensity
Aortic Component of the Second Sound
in Nonhypotensive Patients with Poor Ventricular Performance

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FAREED KHAJA, M.D., AND DANIEL T. ANBE, M.D.

SUMMARY This investigation was undertaken to explore the cause of the diminished second sound (S₂) that may occur in normotensive patients with poorly performing ventricles. Intravo-
tolic sound and pressure were measured in 16 patients with angina; eight had normal ventricular performance (ejection fraction ≥ 60%) and eight had poor performance (ejection fraction < 50%). The amplitude of S₂ was lower in patients with poor ventricular performance as was negative dp/dt. Aortic pressure was com-
parable in both groups. The amplitude of S₂ was linearly related to the
rate of change of the pressure gradient that developed across the
aortic valve during diastole (r = 0.82). The latter also correlated with negative dp/dt (r = 0.82). These observations indicate that in patients with poor ventricular performance, isovolumic relax-
ation may be compromised. This would cause a reduction of the
rate of development of the diastolic pressure gradient, which would result in a diminished S₂.

THE AORTIC COMPONENT of the second sound may be
diminished in patients with myocardial infarction or con-
gestive heart failure, even in the absence of a reduced blood
pressure. 1-3 Traditional teaching that considers the amplitude of the second sound to be primarily determined by diastolic pressure does not explain this observation. 4-7 In order to explain these clinical observations, one must assume that factors other than diastolic pressure contribute to the intensity of the second sound. Other pressure related factors that have been suggested or shown to relate to the amplitude of the aortic component of the second sound include the diastolic pressure gradient that develops across the closed valve, 8-12 the maximal rate of change of the diastolic pressure gradient 9-12 and the pressure gradient at the in-
cisura. 11-12 The rate of change of the diastolic pressure gradient correlates best with the amplitude of the second sound.9 11 12 The velocity of retrograde aortic flow 8 and deceleration of flow 10 have also been suggested as factors which could affect the amplitude of the second sound. However, if one con-
ceives of the second sound as being caused by vibration of the closed cusps 11-12 then it can be demonstrated by mathematical analysis of factors that would effect vibration,

that the driving force productive of vibration is the diastolic pressure difference that develops across the valve. 8 The amplitude of sound that would result from such vibrations relates to the rate of change of that pressure difference. 8 Neither retrograde flow nor the deceleration of flow were shown to be the forces productive of valvular vibration. Thus, it seems from previous studies that the rate of change of the pressure gradient that develops across the valve in diastole is a primary determinant of the amplitude of the second sound. 11-12 The purpose of this study is to explore the extent to which the rate of change of the diastolic pressure gradient, and factors which affect it, may participate in caus-
ing a diminished aortic component of the second sound which is sometimes observed in normotensive patients following a myocardial infarction or in heart failure.

Methods

Intra-aortic sound was measured during diagnostic car-
diac catheterization in 16 patients with anginal-like pain. Three had no apparent cardiac disease, 12 had coronary heart disease, and one had cardiomyopathy. Eight patients had normal ventricular performance as judged by an ejection fraction of 60% or more; and eight patients had poor ventricular performances, indicated by an ejection fraction of less than 50%. One patient was excluded because he had an ejection fraction of 54% which may not be abnormal ac-

ording to the criteria of some investigators, 14 yet is below the range of normal found by others. 15 Patients were also ex-

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Pressure and sound in all patients were measured in the aorta and left ventricle using a Millar Instruments catheter-tip micromanometer. In all but two patients a single catheter-tip sensor was used. Aortic and ventricular pressure and sound were measured in rapid sequence during pull-back of the catheter across the aortic valve. The RR intervals of beats from which calculations were made were virtually the same in the aorta and left ventricle (0.82 ± 0.03 sec vs 0.81 ± 0.03 sec) (mean ± SEM). Aortic and ventricular pressure and sound were measured simultaneously in two patients using a dual catheter-tip micromanometer.

The amplitude of the aortic component of the second sound was measured as the peak-to-peak amplitude of the largest deflection. It was calibrated in terms of pressure (dynes/cm²) as previously described. The characteristics of the sound transducer and recording system also were previously described.

Three consecutive beats were selected for detailed analysis. In those beats, instantaneous values of the aortic and left ventricular diastolic pressure were digitized at 0.25 mm (1.25 msec) intervals using an electronic hand held digitizer (Numbics Corp.) on line with a Hewlett-Packard 21 MX computer. The data were referenced to the electrocardiogram by superimposing the QRS complexes. Instantaneous values of the difference between the aortic and left ventricular diastolic pressure were calculated from the point at which a pressure difference was measurable until the point at which the pressure difference was nearly constant (fig. 1). Instantaneous values of the rate of change of the diastolic pressure gradient were determined by curve fitting techniques.

**Results**

The amplitude of the aortic component of the second sound was lower in patients with poor ventricular perfor-

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**Table 1. Second Sound Amplitude and Related Hemodynamic and Clinical Data**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Diagnosis</th>
<th>Amplitude of sound (dynes/cm²)</th>
<th>Aortic pressure (mm Hg)</th>
<th>LVEDP (mm Hg)</th>
<th>Max d(Ap)/dt (mm Hg/sec)</th>
<th>Max -d(Ap)/dt (mm Hg/sec)</th>
<th>Max Δp (mm Hg)</th>
<th>Δp at incisura (mm Hg)</th>
<th>Ejection fraction (%)</th>
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</thead>
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<tr>
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</tr>
<tr>
<td>1</td>
<td>54</td>
<td>M</td>
<td>CAD</td>
<td>5800</td>
<td>137/90</td>
<td>9</td>
<td>2020</td>
<td>1620</td>
<td>117</td>
<td>33</td>
<td>71</td>
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<tr>
<td>2</td>
<td>44</td>
<td>M</td>
<td>CAD</td>
<td>5470</td>
<td>117/79</td>
<td>6</td>
<td>2170</td>
<td>2000</td>
<td>88</td>
<td>13</td>
<td>67</td>
</tr>
<tr>
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<td>44</td>
<td>F</td>
<td>NL</td>
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<td>111/63</td>
<td>8</td>
<td>1320</td>
<td>1430</td>
<td>88</td>
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<td>38</td>
<td>M</td>
<td>CAD</td>
<td>2520</td>
<td>104/77</td>
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<td>1290</td>
<td>1170</td>
<td>82</td>
<td>9</td>
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<td>51</td>
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<td>7</td>
<td>1570</td>
<td>2430</td>
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<tr>
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<td>31</td>
<td>M</td>
<td>CAD</td>
<td>1560</td>
<td>128/85</td>
<td>16</td>
<td>1190</td>
<td>1250</td>
<td>100</td>
<td>33</td>
<td>61</td>
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<td>53</td>
<td>M</td>
<td>CAD</td>
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<td>20</td>
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<td>46</td>
<td>M</td>
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<td>2020</td>
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<td>111</td>
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<tr>
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<td>3600</td>
<td>128/82</td>
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<td>1730</td>
<td>1750</td>
<td>99</td>
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<tr>
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<td>160</td>
<td>160</td>
<td>4</td>
<td>6</td>
<td>4</td>
</tr>
</tbody>
</table>

**Patients with normal ventricular performance**

| 1       | 59  | M   | CAD       | 3070                          | 170/80                | 19           | 1560                     | 1590                     | 105           | 46                   | 47                  |
| 2       | 61  | M   | CAD       | 2960                          | 145/78                | 22           | 1090                     | 1090                     | 99            | 25                   | 44                  |
| 3       | 41  | M   | CAD       | 2990                          | 120/84                | 7            | 1270                     | 1140                     | 93            | 17                   | 43                  |
| 4       | 46  | M   | CAD       | 2380                          | 106/69                | 14           | 1110                     | 1040                     | 76            | 18                   | 19                  |
| 5       | 46  | M   | CAD       | 1470                          | 133/95                | 22           | 750                      | 920                      | 94            | 4                    | 12                  |
| 6       | 45  | F   | CAD       | 1210                          | 120/96                | 22           | 1180                     | 800                      | 88            | 8                    | 16                  |
| 7       | 46  | F   | CAD       | 2080                          | 108/88                | 10           | 1160                     | 920                      | 95            | 6                    | 35                  |
| 8       | 54  | M   | CAD       | 1210                          | 103/65                | 14           | 1010                     | 860                      | 81            | 9                    | 33                  |
| Mean    | 50  |     |           | 2050                          | 126/82                | 16           | 1140                     | 1060                     | 91            | 17                   | 31                  |
| +SEM    | 3   |     |           | 250                           | 8/4                   | 2            | 80                       | 80                       | 3             | 5                    | 5                   |

**Patients with abnormal ventricular performance**

| 1       | 59  | M   | CAD       | 3070                          | 170/80                | 19           | 1560                     | 1590                     | 105           | 46                   | 47                  |
| 2       | 61  | M   | CAD       | 2960                          | 145/78                | 22           | 1090                     | 1090                     | 99            | 25                   | 44                  |
| 3       | 41  | M   | CAD       | 2990                          | 120/84                | 7            | 1270                     | 1140                     | 93            | 17                   | 43                  |
| 4       | 46  | M   | CAD       | 2380                          | 106/69                | 14           | 1110                     | 1040                     | 76            | 18                   | 19                  |
| 5       | 46  | M   | CAD       | 1470                          | 133/95                | 22           | 750                      | 920                      | 94            | 4                    | 12                  |
| 6       | 45  | F   | CAD       | 1210                          | 120/96                | 22           | 1180                     | 800                      | 88            | 8                    | 16                  |
| 7       | 46  | F   | CAD       | 2080                          | 108/88                | 10           | 1160                     | 920                      | 95            | 6                    | 35                  |
| 8       | 54  | M   | CAD       | 1210                          | 103/65                | 14           | 1010                     | 860                      | 81            | 9                    | 33                  |
| Mean    | 50  |     |           | 2050                          | 126/82                | 16           | 1140                     | 1060                     | 91            | 17                   | 31                  |

**P value**

- <0.02
- <0.01
- <0.01
- <0.01

**Key:** Δp = diastolic pressure gradient; d(Ap)/dt = rate of change of the diastolic pressure gradient.

P values refer to the difference of means between the two groups (unpaired t-test).
mance (2050 ± 250 dynes/cm²) than in patients with normal ventricular performance (3600 ± 520 dynes/cm², \( P < 0.02 \), unpaired t-test) (table 1). Negative dp/dt was lower in those with poor performance (1060 ± 80 vs 1730 ± 160 mm Hg/sec, \( P < 0.01 \)). The maximal rate of change of the diastolic pressure gradient was also lower in patients with poor ventricular performance (1140 ± 80 vs 1720 ± 160 mm Hg/sec, \( P < 0.01 \)).

The amplitude of the aortic component of the second sound showed a linear correlation with the maximal rate of change of the diastolic pressure gradient that developed across the valve (\( r = 0.82 \)) (fig. 2). Maximal values of the rate of change of the diastolic pressure gradient occurred coincident with the major component of the second sound. A linear correlation was shown between the rate of change of the diastolic pressure gradient and negative dp/dt (\( r = 0.82 \)). Negative dp/dt correlated with the second sound (\( r = 0.68 \)), but the correlation with sound was not as close as the rate of change of the pressure difference. The amplitude of the second sound also correlated with the ejection fraction (\( r = 0.67 \)). Again the correlation was not as close as the correlation between sound and the rate of change of the pressure gradient.

Aortic pressure was comparable in both groups (126/82 ± 8/4 vs 126/82 ± 5/4 mm Hg). The amplitude of the aortic component of the second sound showed a poor correlation with aortic diastolic pressure (\( r = 0.10 \)); the aortic pressure at the time of the incisura (\( r = 0.28 \)); the maximal value of the diastolic pressure gradient, (\( r = 0.47 \)); and with the pressure gradient at the time of the aortic incisura (\( r = 0.28 \)).

**Discussion**

Even though the aortic pressure of the patients with poor ventricular performance was virtually the same as the pressure of those with normal performance, the amplitude of the aortic component of the second sound was significantly diminished in those with poor performance. Both negative dp/dt and the rate of change of the diastolic pressure gradient were also lower in patients with poor performance.

A dependence of the amplitude of the aortic component of the second sound upon the rate of change of the diastolic pressure gradient is predicted by equations which describe sound produced by the aortic valve when the valve is modeled as a vibrating circular membrane.\(^9\) Since the rate of change of the diastolic pressure gradient is dependent upon negative dp/dt, the results imply a dependence of the amplitude of the aortic closure sound upon the rate of isovolumic relaxation. We have observed a diminished aortic component of the second sound on phonocardiograms taken at the chest wall in normotensive patients hospitalized because of recent ischemic episodes.\(^7\) In these patients, the duration of isovolumic relaxation was prolonged, which also implies a reduced rate of isovolumic relaxation.\(^13\) The diminished aortic component of the second sound in normotensive patients after a recent infarction or in heart failure, therefore, appears to reflect an impaired rate of isovolumic relaxation.

If the aortic valve during diastole can be considered to vibrate analogously to a vibrating membrane, then the driving force productive of valvular vibration is the diastolic pressure gradient.\(^13\) Acoustical theory indicates the amplitude of sound generated by a vibrating source in the form of a piston relates directly to the velocity of the vibrating source.\(^19\) Any hemodynamic or physiological factors that affect the velocity of the vibrating source would directly affect the amplitude of sound. In the case of a vibrating membrane, the velocity of deflection of the membrane relates to the rate of change of the diastolic pressure gradient.\(^12\) Since the rate of change of the diastolic pressure gradient is affected by negative dp/dt, changes of the rate of isovolumic relaxation will result in changes of the amplitude of the aortic component of the second sound.

The diminished aortic sound in aortic stenosis would seem to relate to increased stiffness of the valve.\(^16\) Because of the important effect that reduced flexibility of the leaflets may have upon the aortic component of the second sound, particular care was taken to assure that the aortic valve was normal in these patients. Regurgitant valves also are accompanied by a diminished amplitude of the second sound.\(^16\) Presumably, in an insufficient valve, the ability of the closed cusps to vibrate is diminished. Therefore, patients with aortic insufficiency were also excluded from this study. Other factors, not yet explored, perhaps may contribute to the second sound. These include the size and shape of the ventricle, the sound absorption of the aortic and left ventricular walls, and the viscosity of the blood.\(^13\)

In summary, in patients with poor ventricular performance, the rate of isovolumic relaxation may be compromised. This would cause a reduction of negative dp/dt which in turn causes a reduction of the rate of change of the pressure gradient that develops across the valve during diastole. A diminished second sound, therefore, would result due to the more slowly developing driving pressure which directly affects the characteristics of valvular vibration.

**References**

Spectral Energy of the First Heart Sound in Acute Myocardial Ischemia

A Correlation with Electrocardiographic, Hemodynamic, and Wall Motion Abnormalities

W. Bromley Clarke, Ph.D., Stephen M. Austin, M.D., Pravin M. Shah, M.D., Paul M. Griffen, James T. Dove, M.D., Judith McCullough, and Bernard F. Schreiner, M.D.

SUMMARY First heart sound (S1) energy spectra in isovolumic systole, hemodynamics, and angiographic left ventricular wall motion (LVWM) at rest and with atrial pacing were compared in 27 patients who underwent diagnostic cardiac catheterization and angiography because of chest pain. Eighteen patients were found to have coronary artery disease (CAD) and nine patients, normal coronary arteries. Eleven of the 18 CAD patients (61%) had a mean reduction in the spectral energy of S1 of 6.5 ± 1.4 (SEM) dB below control (−52%), during interruption of ischemic stress of rapid atrial pacing, compared to only one of nine patients without CAD (P < 0.05). Only five CAD patients (28%) had an abnormal rise (±5 mm) in left ventricular end-diastolic pressure (LVEDP) either during or upon interruption of pacing, and six (33%) had ischemic ST-segment depression ≥0.1 mv in the ECG. Similarly two patients free of CAD (22%) had an abnormal increase in LVEDP, and none had ECG evidence of ischemia. Seventeen CAD patients (94%) had segmental LVWM abnormalities at rest or with interruption of pacing, while three patients with normal coronary arteries (33%) had abnormal angiographic LVWM (P < 0.01).

Thus, reduction in S1 spectral energy is a common accompaniment of myocardial ischemia. In the present study, it was more frequently observed than abnormalities in either the ECG or LVEDP, but was not as consistently seen as segmental left ventricular wall motion abnormalities.

ACUTE MYOCARDIAL ISCHEMIA has been noted in the clinical setting to be associated with reduction in loudness of the first heart sound (S1). This study was designed to determine whether detailed analysis of the first heart sound during ischemic stress of rapid atrial pacing provides a quantitative measure of changes in energy spectra of S1.

Several theories have been proposed to explain the mechanism of generation of the first heart sound (S1).1-4 The more recent studies5-8 have supported the acceleration and deceleration theory of Rushmer7 as the most likely physiologic explanation. An echocardiographic study from our laboratory5 has shown that, although the timing of mitral valve closure is a critical factor in determining the intensity of S1, the energy of sound vibrations is probably derived from the force of left ventricular contraction during isovolumic systole.

Sakamoto and associates6 have reported a nearly linear relationship between the amplitude of S1 and peak rate of...
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P D Stein, H N Sabbah, F Khaja and D T Anbe

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