The Pulmonary Component of the Second Heart Sound
In Acquired Aortic Stenosis

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SUMMARY
The amplitude of the pulmonic component of the second sound in aortic stenosis was studied in 49 patients with this lesion. As controls, 50 normal subjects were also studied. Both groups were investigated by phonocardiography, apex cardiography and arterial tracings. Nineteen patients with aortic stenosis and four subjects without it were also studied by cardiac catheterization and angiography. The amplitudes of the two components of the second sound were compared, and the ratio of each with the amplitude of the first sound was determined. The ratios of both the aortic and the pulmonic component to that of the first sound were decreased in aortic stenosis, and the decrease of the pulmonic component was comparable to that of the aortic component. These findings could be related to prolongation of the isovolumic relaxation period of both ventricles caused by an influence of the left ventricle on the right, most likely due to functional changes of the interventricular septum.

Additional Indexing Words:
Heart auscultation   Heart valve disease   Phonocardiography
Second heart sound   Aortic stenosis

A common observation in our laboratory is that patients with aortic stenosis, not only have a small aortic component of the second sound, but also have a small pulmonic component. The combination of the two results in a small sound complex that is often difficult to study. Our studies in a series of patients offer a tentative explanation.

MATERIAL AND METHODS

This study was made by comparing the phonocardiographic, apex cardiographic, and carotid tracings of 50 normal subjects and 49 patients with valvular

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Received for publication November 10, 1980.
aortic stenosis*. The normal subjects were from 9 to 81 years old (mean 49 yrs). Their selection was made on the basis of history, physical examination, chest X-ray, electrocardiogram, phonocardiogram, and carotid tracing. The patients with proven aortic stenosis were from 7 to 86 years old (mean 52 yrs). The phonocardiograms were recorded with a Hewlett-Packard equipment including contact microphones having a linear frequency response from 0 to 1,000 Hz. Various high-pass filters were used but the calculations were made only in the tracings recorded with a filter having a nominal frequency of 100 Hz and a slope of 24 dB/octave. The microphone was applied to the third left i.c.s. at 2-3 cm from the sternal border and held in place by a rubber strap (the response of this microphone is independent of strap tension). Out of the 49 patients with aortic stenosis, 19 had been studied by left and right cardiac catheterization and angiography**. In all cases, the aortic stenosis was either pure or predominant according to clinical and laboratory studies.

The carotid tracing was recorded with a Hewlett-Packard pulse recorder which makes use of a small funnel applied to the neck and air transmission of the signal to a piezoelectric transducer.

The 19 patients of aortic stenosis studied by catheterization were classified as follows: a) mild to moderate stenosis (LV-aortic mean systolic gradient<50 mm Hg and aortic valve area greater than 0.9 cm²=9 subjects); b) severe stenosis (LV-aortic mean systolic gradient≥50 mm Hg and aortic valve area less than 0.9 cm²=10 subjects).

Six patients out of 19 had some degree of pulmonary hypertension (PA systolic pressure≥30 mm Hg; mean pressure≥20 mm Hg). Only four patients out of 19 had a reduced cardiac index (a value of 3.5 for younger patients and 3.0 for patients above 30 years was considered normal).

Due to the retrospective nature of our study, we were able to calculate the isovolumic relaxation period only in 5 out of 19 cases of aortic stenosis hemodynamically studied. One patient had simultaneous left and right ventricular (LV and RV) pressure tracings; in the other 4, the measurements made over the separate LV and RV pressures were compared in cycles of identical duration, both recorded at 50 mm/sec of paper speed. The isovolumic relaxation period was measured in msec from the moment of closure of the aortic or pulmonary valve (incisura of the correspondent arterial pressure tracing) to the 0 point of the ventricular pressure. The isovolumic relaxation period was calculated with the same method also in four normal subjects who underwent catheterization.

The amplitude of the first heart sound (I) as well as that of the two components of the second heart sound (IIₐ and IIₚ, respectively) was separately measured in millimeters in 5 to 10 subsequent cycles. The ratios IIₐ/I, IIₚ/I, and IIₐ/IIₚ were calculated in each of the measured cycles; then the smallest and the largest values were averaged for each ratio and the results were considered as the relative amplifi-

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* All control subjects and a case of aortic stenosis were studied at Oak Forest Hospital. Thirty-five cases of aortic stenosis were from our old laboratory at Mount Sinai Hospital. Five cases of aortic stenosis were studied at St. Mary of Nazareth Hospital.

** Fourteen of these cases were from our collection of Mount Sinai Hospital while 5 patients and 4 subjects without aortic stenosis were studied at St. Mary of Nazareth Hospital; their tracings were obtained through the courtesy of Dr. B. Cortis.
tude of each component of the second sound. As no reliable, non-invasive calibration of the heart sounds is possible as yet, the components of the second sound were then compared to the first sound. No cause of increase in amplitude of the first sound (mitral stenosis; thyrotoxicosis) existed in any of our patients. Left ventricular hypertrophy per se does not cause an increased amplitude of the first sound. On the contrary, by increasing the left ventricular mass, it tends to decrease the first sound or shift the vibrations of the latter to a lower frequency, as it was found by Adolph in normal athletes. Thus, this procedure gave evidence of the relative amplitude of the two components of the second sound irrespective of the degree of amplification.

The statistical analysis was made by calculating the mean values and the standard deviation of each group; then obtaining the significance of the differences using either the two samples-Student's t test (whenever the degrees of freedom were > 30) or the Wilcoxon-Mann-Whitney rank sum test (whenever the degrees of freedom were ≤ 30).

**RESULTS**

Six of our nineteen patients with aortic stenosis studied by cardiac catheterization had pulmonary hypertension while 13 had normal pulmonary artery pressure (Table I). This fact was not related to the degree of aortic stenosis as three of these patients had moderate aortic stenosis while the others had severe stenosis (Table II).

The study of the second sound revealed the following facts (Table III):

a) The ratio between amplitude of the aortic component of the second sound ($\Pi_A$) and that of the first sound ($I$) was 2.21 ± 0.78 in the control subjects but dropped to 0.88 ± 0.73 in the aortic stenosis studied by catheterization (and 0.92 ± 0.7 in the total group of aortic stenosis); thus it was con-

<table>
<thead>
<tr>
<th>No. of cases</th>
<th>Type of patients</th>
<th>Blood pressure (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Systolic</td>
</tr>
<tr>
<td>6</td>
<td>Aortic stenosis with pulmonary hypertension</td>
<td>Aortic pressure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pulmonary artery pressure</td>
</tr>
<tr>
<td>12</td>
<td>Aortic stenosis without pulmonary hypertension</td>
<td>Aortic pressure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pulmonary artery pressure</td>
</tr>
<tr>
<td>18</td>
<td>Total group of aortic stenosis</td>
<td>Aortic pressure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pulmonary artery pressure</td>
</tr>
</tbody>
</table>

Table I. Aortic and Pulmonary Pressures in our Group of Patients with Aortic Stenosis.
Table II. Degree of Aortic Stenosis and Relationship with Pulmonary Hypertension in 19 Catheterized Patients

<table>
<thead>
<tr>
<th>No. of cases</th>
<th>Type of patients</th>
<th>LV-Ao mean systolic gradient (mmHg)</th>
<th>Aortic valve area (cm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>9</td>
<td>Mild to moderate aortic stenosis</td>
<td>34.2 ± 6.6</td>
<td>1.2 ± 0.24</td>
</tr>
<tr>
<td>10</td>
<td>Severe aortic stenosis</td>
<td>83.5 ± 39.2</td>
<td>0.69 ± 0.26</td>
</tr>
<tr>
<td>6</td>
<td>Aortic stenosis with pulmonary hypertension</td>
<td>80 ± 56</td>
<td>0.74 ± 0.34</td>
</tr>
<tr>
<td>13</td>
<td>Aortic stenosis without pulmonary hypertension</td>
<td>56 ± 23</td>
<td>0.99 ± 0.29</td>
</tr>
<tr>
<td>19</td>
<td>Total group of aortic stenosis</td>
<td>64 ± 37</td>
<td>0.91 ± 0.32</td>
</tr>
</tbody>
</table>

The difference between the two groups with pulmonary hypertension is not statistically significant.

Table III. Relative Amplitude of Aortic and Pulmonic Components of the Second Sound

<table>
<thead>
<tr>
<th>No. of cases</th>
<th>Type of subjects</th>
<th>Ratio ( \Pi_A/I )</th>
<th>Ratio ( \Pi_P/I )</th>
<th>Ratio ( \Pi_A/\Pi_P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>50</td>
<td>Normal subjects</td>
<td>2.21 ± 0.78</td>
<td>1.12 ± 0.51</td>
<td>1.97 ± 0.93</td>
</tr>
<tr>
<td>49</td>
<td>Total group of aortic stenosis</td>
<td>0.92 ± 0.70   ***</td>
<td>0.62 ± 0.57   ***</td>
<td>1.73 ± 1.03 n.s.</td>
</tr>
<tr>
<td>19</td>
<td>Aortic stenosis with catheterization</td>
<td>0.88 ± 0.73   ***</td>
<td>0.63 ± 0.58   **</td>
<td>1.61 ± 1.04 n.s.</td>
</tr>
</tbody>
</table>

Asterisks give the statistical significance of a group versus the group of normal subjects.

** p<0.01  *** p<0.001

firmed that the \( \Pi_A \) is markedly decreased in aortic stenosis (Fig. 1).

b) The ratio between amplitude of the pulmonic component of the second sound \( \Pi_P \) and that of the first sound \( I \) was 1.12 ± 0.51 in the control subjects but dropped to 0.63 ± 0.58 in the group of cases of aortic stenosis studied by catheterization and 0.62 ± 0.57 in the total cases studied. Thus, it was proven that \( \Pi_P \) is also decreased in aortic stenosis (Fig. 1). The ratio between \( \Pi_A \) and \( \Pi_P \) was 1.97 ± 0.93 in normal subjects and 1.61 ± 1.04 in the group of aortic stenosis studied by catheterization (difference not significant). Thus, it was apparent that the decrease of \( \Pi_P \) was comparable to that of \( \Pi_A \) (Table III).

c) The relative amplitude of the aortic and pulmonic components of the second sound was then compared in patients with either moderate or severe aortic stenosis (Table IV). The ratios of \( \Pi_A/I \), \( \Pi_P/I \), and \( \Pi_A/\Pi_P \) were all more decreased in severe stenosis than in mild stenosis showing that the change
Fig. 1. Two cases of aortic stenosis.

A: 43-year-old male with severe stenosis (LV-Ao gradient = 176 mmHg, Aortic valve area = 0.15 cm²). Top: carotid tracing: slow half rise; marked shudder. Below: PCG recorded at 2 R with 100 Hz filter. Reversed splitting of the second sound with small P and A components. Long, diamond-shaped systolic murmur with late peak. Center: PCG recorded at apex with 100 Hz filter. The murmur is similar; the A component is very small. There is a third sound. Bottom: ECG.

B: 50-year-old female with mild stenosis (LV-Ao gradient = 25 mmHg, Aortic valve area = 1.46 cm²). Top: ECG. Center: PCG recorded at 3L with 100 Hz filter. Smaller and shorter, diamond-shaped systolic murmur with center peak. Normal splitting of the second sound with small A component and minimal P component. Bottom: Carotid tracing with rapid half rise and small shudder.
Table IV. Relative Amplitude of Aortic and Pulmonic Components of the Second Heart Sound in Moderate and Severe Aortic Stenosis

<table>
<thead>
<tr>
<th>No. of cases</th>
<th>Type of patients</th>
<th>Ratio $\Pi_A/I$</th>
<th>Ratio $\Pi_P/I$</th>
<th>Ratio $\Pi_A/\Pi_P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>9</td>
<td>Mild to moderate aortic stenosis$^*_x$</td>
<td>1.13±0.95</td>
<td>0.67±0.49</td>
<td>1.75±1.25</td>
</tr>
<tr>
<td></td>
<td>Significance versus 50 normal subjects</td>
<td>***</td>
<td>**</td>
<td>n.s.</td>
</tr>
<tr>
<td>10</td>
<td>Severe aortic stenosis$^{**x}$</td>
<td>0.63±0.3</td>
<td>0.59±0.29</td>
<td>1.06±0.24</td>
</tr>
<tr>
<td></td>
<td>Significance versus normals and versus the above group</td>
<td>***</td>
<td>***</td>
<td>***</td>
</tr>
</tbody>
</table>

** $p<0.01$  *** $p<0.001$  n.s. = not significant
$x$ LV-Ao gradient $< 50$ mmHg and aortic valve area $> 0.9$ cm$^2$
$^{**}$ LV-Ao gradient $\geq 50$ mmHg and aortic valve area $\leq 0.9$ cm$^2$

Table V. Relative Amplitude of Aortic and Pulmonic Components of the Second Sound in Relationship to Pulmonary Hypertension

<table>
<thead>
<tr>
<th>No. of cases</th>
<th>Type of patients</th>
<th>Ratio $\Pi_A/I$</th>
<th>Ratio $\Pi_P/I$</th>
<th>Ratio $\Pi_A/\Pi_P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>Aortic stenosis with pulmonary hypertension</td>
<td>1.23±1.08</td>
<td>1.01±0.87</td>
<td>1.24±0.52</td>
</tr>
<tr>
<td></td>
<td>Significance versus 50 normal subjects</td>
<td>***</td>
<td>n.s.</td>
<td>**</td>
</tr>
<tr>
<td>12</td>
<td>Aortic stenosis without pulmonary hypertension</td>
<td>0.71±0.44</td>
<td>0.44±0.25</td>
<td>1.79±1.2</td>
</tr>
<tr>
<td></td>
<td>Significance versus normals and versus the above group</td>
<td>***</td>
<td>**</td>
<td>n.s.</td>
</tr>
</tbody>
</table>

** $p<0.01$  *** $p<0.001$  n.s. = not significant

was related to the severity of the stenosis.

d) The relative amplitude of the aortic and pulmonic components was then compared between cases with aortic stenosis plus pulmonary hypertension and cases with aortic stenosis and normal pulmonary artery pressure (Table V). The ratio of $\Pi_A/I$ was lower in those without pulmonary hypertension but the difference was not significant; that of $\Pi_P/I$ was markedly lower and was significant. Finally, a difference between $\Pi_A/\Pi_P$ in cases with pulmonary hypertension and in cases without it was again present but was not statistically significant. In conclusion, while patients with aortic stenosis and pulmonary hypertension still have a significant decrease of the aortic component, while their pulmonic component is normal.

In five patients with aortic stenosis the isovolumic relaxation period was $134\pm9$ msec, while in four normal subjects it was $100\pm12$ msec. The difference between the two groups is highly significant ($p<0.001$). The iso-
volumic relaxation period was found identical in the two ventricles, both in the patients and in the controls.

**DISCUSSION**

A small aortic component of the second sound in the phonocardiographic tracing has been considered typical of valvular aortic stenosis for a long time\(^5\)\(^\text{-}^8\), a delayed aortic component has also been observed. The causes for these changes were revealed by the physiologic studies of Kusukawa et al.\(^6\) and Sabbah and Stein\(^7\) and were analyzed in catheterized patients by Kumar and Luisada.\(^8\)

Less known is the fact that a decrease of the pulmonary component of the second sound is also commonly observed. This fact has not been the object of special studies and has received, so far, no explanation.

The use of the ratios between the amplitude of the two sounds in order to document a decrease of the amplitude of the second sound is justified by the fact that left ventricular hypertrophy, left bundle branch block (present in five of our patients), myocardial ischemia and/or old myocardial infarct (present in 28 patients), left ventricular failure (present in 4 patients) were all factors able to decrease the first heart sound of our patients in comparison with normal controls. Thus, whenever the ratios \(\text{II}_A/\text{I}\) and \(\text{II}_P/\text{I}\) were found diminished in a statistically significant way, we could be all the more sure that a real decrease of the total second sound was present.

Our present observations have confirmed the small amplitude of the aortic component of the second sound, and also its correlation with the severity of the stenosis. A decrease of the pulmonic component was also documented. This decrease was comparable to that of the aortic component and was only slightly more marked in patients with more severe aortic stenosis than in those with mild stenosis. When pulmonary hypertension was present, the pulmonic component had a lesser degree of reduction; however, as it should have increased if the pulmonary hypertension had not been accompanied by aortic stenosis\(^9\)\(^,\)^{10}, this was a sign of relative decrease.

A possible, though non proven, explanation would be that the patients with aortic stenosis had a reduced cardiac output and that this fact was the cause of reduction of both the aortic and pulmonic components. However, out of 19 patients studied by catheterization, only four had a decrease of their cardiac index while the others had a normal output. Moreover, Stein et al.\(^10\) found that the large pulmonic component of patients with pulmonary hypertension fails to decrease with the onset of right ventricular failure if the left ventricle still strongly contracts. Thus, a different explanation should be
found.

The amplitude of the second heart sound has been demonstrated to be related to the rate of change of the diastolic pressure gradient across the closed aortic and pulmonary valves and to the rate of the ventricular isovolumic relaxation (as revealed by the depth of the trough of the dP/dt). In one of our cases where simultaneous pressure tracings of the two ventricles were recorded, the isovolumic relaxation of both ventricles was identical in all cycles (Fig. 2). In four other patients, in whom the tracings were not simultaneous but were comparable having the same heart rate and the same film speed, the isovolumic relaxation was again identical in both ventricles. In all five patients,

![Simultaneous tracings of pressure in the right and left ventricles of a patient with aortic stenosis.](image1)

**Fig. 2.** Simultaneous tracings of pressure in the right and left ventricles of a patient with aortic stenosis (LV-Ao gradient = 76 mm Hg). Same amplification of LV and RV. Recording speed = 50 mm/sec.

![Scheme of pathological sections of the heart.](image2)

**Fig. 3.** Scheme of pathological sections of the heart. At left = normal (A); at right = left ventricular hypertrophy (B). This scheme reveals the anatomical importance of the interventricular septum.
the isovolumic relaxation period was longer than in four normal subjects.

If we keep in mind that the strong and thick interventricular septum (demonstrated by echocardiographic and pathological studies—Fig. 3) is an integral part of both ventricles, it becomes obvious that a slower relaxation of this structure in aortic stenosis would prolong the rate of isovolumic relaxation of the right ventricle. Thus, the smaller pulmonic component would be caused by transmitted changes in function from the left to the right ventricle during early diastole. Such a dynamic influence of the left over the right ventricle is suggested by the following studies: 1) cauterization of the free right ventricular wall did not alter the blood flow into the pulmonary artery\(^{12}\); 2) aortic constriction increased intracavitary and intramuscular pressure of both sides of the interventricular septum while pulmonary artery constriction raised it only on the right side \(^{13},^{14}\); 3) clinical cases with pulmonary hypertension and right ventricular failure had a normal isovolumic relaxation of the right ventricle as a result of normal left ventricular relaxation\(^{10}\). These studies point out that the right ventricular pumping function is aided by an "in series left ventricle".\(^{14}\)

The characteristic left ventricular hypertrophy of aortic stenosis may be considered as the main cause of the prolonged phase of relaxation. Another factor that may be involved is septal calcification because extensive valvular calcification extending to the ventricular septum has been reported in 25% of 249 cases of aortic stenosis who underwent surgery\(^{15}\).

In conclusion, the amplitude of the pulmonic component of the second sound is partly related to the functional and anatomical situation of the right ventricle but is also affected by the changes in function of the left ventricle.

**References**

3. Luisada AA: Heart. Williams & Wilkins, Baltimore, 1948, p 231


