Accuracy of the Phonocardiogram in Assessing Severity of Aortic and Pulmonic Stenosis

By Raul Gamboa, M.D., Paul G. Hugenholtz, M.D., and Alexander S. Nadas, M.D.

A continuing need exists for the refinement of technics that could obviate the need for cardiac catheterization in assessing the severity of congenital heart disease. For many years the phonocardiogram has been considered useful in this regard, particularly in the estimation of the degree of pulmonic stenosis. Following the studies by Leatham and Vogelpoel attention has been centered mainly around the duration of the murmur and the behavior of the second sound, while the significance of the ejection click and the timing of the peak amplitude of the murmur have been incompletely studied. Furthermore, none of these items has been subjected to a statistical analysis evaluating their reliability in estimating right ventricular pressure. In aortic stenosis no detailed studies are available relating auscultatory phenomena to specific hemodynamic features in the assessment of the severity of the lesion.

It is the purpose of this study to describe the correlation of the phonocardiogram with the peak ventricular pressure, as well as the calculated valve area in patients with pure semilunar stenosis and to evaluate the position of this tool in the diagnostic armamentarium presently available to the clinician.

Materials and Methods

Phonocardiograms were recorded on selected patients admitted to the Children's Hospital Medical Center for cardiac catheterization, in the period from April 1961 to June 1963. Of a total of 243, 50 records of patients with congenital valvular pulmonic stenosis and 30 of patients with valvular aortic stenosis were selected for analysis on the basis of technical quality and availability of hemodynamic data. They represented 80 per cent of all phonocardiograms in patients with aortic and pulmonic stenosis. The patients' ages ranged from 2 to 28 years.

Cardiac catheterization and angiograms were available in all 80 cases and in 20 the diagnosis was further confirmed at operation. Right heart catheterizations were performed in routine fashion, left heart studies were obtained by the retrograde arterial approach. Details of these methods have been described previously.

All phonocardiograms were obtained in a sound-proofed room on a Sanborn Polybeam, model 550 M, photographic recorder with preamplifiers model 350-1700 B, with selected limited frequency bands between 25 to 400 c.p.s. and corresponding slopes at 12 or 24 db./octave.

In this manner multiple records were obtained from the second right and left intercostal space, the fourth intercostal space, and the apex, during held expiration at a paper speed of 100 mm. per second. An electrocardiographic lead and indirect carotid pressure pulse recording were utilized for timing reference. The early systolic sound considered to be the "ejection click" was differentiated in patients with pulmonic stenosis from tricuspid valve closure by its prominence at the second left intercostal space, its high frequency components, and its increase in intensity during expiration. The ejection click in patients with aortic stenosis was distinguished from the first heart sound by its high frequency components and the absence of respiratory variation in its timing at the apex. Simultaneous recording over the aortic or pulmonic area and over the apex, or tricuspid area, with different filter ranges was extremely helpful in this regard. With the onset of the electrical depolarization (Q wave) as a constant point of reference, the time interval to the first component of the early systolic sound (Q-x
interval) was measured to the nearest 0.005 second. The interval between onset of ventricular contraction and the timing of the maximum amplitude of the systolic murmur (Q-peak amplitude interval) was determined in a similar fashion. The configuration of the murmur and the timing of the peak magnitude were determined from recordings made with the 400 c.p.s. filter at optimal magnitude.

The degree of splitting of the second sound

| Figure 1 |

Schematic display of phonocardiographic findings in 30 patients with aortic stenosis. In the columns following the left ventricular peak pressure are indicated the Q-x interval, the Q-peak amplitude interval, and, after the contour of the murmur, the A$_2$-P$_2$ interval.

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Figure 2

Schematic display of phonocardiographic findings in 50 patients with pulmonic stenosis. In the columns following the right ventricular peak pressure are indicated the Q-x interval, the Q-peak amplitude interval, and, after the contour of the murmur, the $A_2-P_2$ interval.
was determined by identifying the aortic closure sound from the dicrotic notch in the carotid artery pressure pulse. The pulmonary component at times was difficult to record, requiring selection of a high-gain-low-frequency band for its identification.

All measurements were corrected for heart rate according to the formulas

\[
\frac{Q-x \text{ interval}}{\sqrt{R-R \text{ interval}}}, \quad \frac{Q\text{-peak of the murmur interval}}{\sqrt{R-R \text{ interval}}}
\]

and

\[
\frac{A_2-P_2 \text{ interval}}{\sqrt{R-R \text{ interval}}}
\]

Similar corrections were made for the right ventricular ejection time and the rate of ventricular pressure development (dp/dt) determined in 17 of the 30 patients with pulmonic stenosis and

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**Figure 3**

Representative examples of the phonocardiogram in mild and severe aortic stenosis. x, ejection click; SM, systolic murmur; A, aortic closure sound; P, pulmonary closure sound.

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**Figure 4**

Typical illustration of the phonocardiogram in (A) mild, (B) moderate, and (C) severe pulmonic stenosis. Legend as in figure 3. The Q-x and A_2-P_2 intervals are indicated in seconds below the horizontal bars.
PHONOCARDIOGRAM IN AORTIC AND PULMONIC STENOSIS

Figure 5

Scattergram reflecting the relation between right ventricular peak pressure and the Q-x interval corrected for the heart rate in 50 patients with pulmonic stenosis.

ejection clicks in whom catheters of sufficient size to warrant these calculations were used.

Amplification varied in all records depending on chest size, skin thickness, and contact. Consequently no correlations were attempted with the loudness of the murmurs or the heart sounds, and the configurations displayed in figures 1 to 4 therefore do not indicate true differences between the intensity of these murmurs.

Hemodynamic measurements were obtained on the day following the one the phonocardiogram was obtained. Only peak ventricular pressures recorded at heart rates close, or identical, to those seen in the phonocardiograms were selected for the correlations. The material in cases with pulmonic stenosis was divided according to severity of right ventricular hypertension into three groups: (1) peak right ventricular pressure less than 80 mm. Hg (13 cases, mild); (2) pressures between 80 to 120 mm. Hg (21 cases, moderate); (3) pressures exceeding 120 mm. Hg. (16 cases, severe). Patients with aortic stenosis were arbitrarily divided into two groups, one with peak pressures in the left ventricle of 150 mm. Hg or less, the other with pressures exceeding this level.

Routine statistical analysis was carried out. Student's t test was used to express the significance of differences between the groups.

Results

A schematic representation of the essential phonocardiographic features and the corresponding hemodynamic data in patients with aortic and pulmonic stenosis is given in figures 1 and 2. The correlations between these factors appear in figures 5 to 9.

An ejection click could be identified in 60 per cent of the patients with pulmonic stenosis and in all patients with aortic stenosis. Contrary to previous opinion it was recorded with equal frequency in patients with mild and with severe pulmonic stenosis. A significant inverse relationship between right ventricular peak pressure and the Q-ejection click interval was calculated (r = -0.77, p = 0.001, fig. 5), indicating that the earlier the click occurs, the higher is the right ventricular pressure. The regression equation is RVPP = -4.352 sec. + 588, SEE = 25 mm. Hg. When the timing of the click is arranged according to the three subgroups, significant differences are found between these categories (table 1A). In contrast, in patients with aortic stenosis, the ejection click did not show any significant correlation with ventricular peak pressure (r = -0.40, p < 0.5, fig. 6), and no significant differences were found in the timing of the click in these two groups of sever-
ity (table 1B). There was no instance of severe aortic stenosis, however, when the click occurred late.

The timing of the peak amplitude of the pulmonic stenotic murmur permitted clear separation of the three groups of severity (table 2A). There was no overlap in this regard between the group with mild and moderate right ventricular hypertension and only two cases with moderate pulmonic stenosis (nos. 31 and 33) showed a murmur indicative of severe pulmonic stenosis (fig. 2). In each of these two cases, however, right ventricular peak pressure exceeded 110 mm Hg. Moreover, there was a statistically significant linear relationship between the right ventricular peak pressure and the Q-peak amplitude interval \( (r = 0.72, p < 0.001, \text{fig. } 7) \). The regression equation is \( \text{RVPP} = 1,289 \text{ sec. - 338, SEE = 32 mm. Hg} \). In a simplified way this correlation can also be expressed by noting the end of the murmur in relation to the timing of \( A_2 \). In each of the 13 patients with mild pulmonic stenosis and in 19 of the 21 with moderate pulmonic stenosis, the murmur terminated before \( A_2 \) was recorded, whereas in 12 of the 16 with severe pulmonic stenosis, the murmur extended through the aortic second sound (figs. 2 and 4).

A significant inverse relationship could also be calculated between the logarithm of pulmonary valve area and the Q-peak amplitude interval \( (r = -0.72, p < 0.001, \text{fig. } 8) \). The regression equation was \( \log \text{PVA} = -37.69 \text{ sec. + 10.63, SEE = 0.23 cm.}^2 \).

**Figure 7**

Scattergram showing the significant relationship in patients with pulmonic stenosis between the right ventricular peak systolic pressure and the interval between the onset of ventricular contraction and the maximum amplitude of the systolic murmur.

**Figure 8**

Relationship between the valve area and the Q-peak amplitude interval in pulmonic stenosis.

**Figure 9**

Diagram showing the correlation between right ventricular pressure and the interval between aortic and pulmonic valve closure. The correlation coefficient is 0.84, and shows its most narrow spread in the patients with pressures higher than 130 mm. Hg.
In patients with aortic stenosis, the systolic murmur found its peak amplitude in the first half of the ejection period in nearly all cases irrespective of severity (figs. 1 and 3). Only a few cases with severe left ventricular hypertension showed the peak of the murmur appearing in mid-systole. No significant correlation with left ventricular peak systolic pressure could be found, nor was there a relationship between this phonocardiographic measurement and the calculated aortic valve area, \( r = 0.35 \), \( p > 0.05 \), table 2B.

The degree of splitting, a measurement available in all but two cases with pulmonic

### Table 1A

**Incidence and Timing of Ejection Click, Corrected for Heart Rate, in 50 Cases with Valvular Pulmonic Stenosis**

<table>
<thead>
<tr>
<th>Right ventricular pressure</th>
<th>Total</th>
<th>&lt; 80</th>
<th>80-120</th>
<th>&gt; 120</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of cases studied</td>
<td>50</td>
<td>13</td>
<td>21</td>
<td>16</td>
</tr>
<tr>
<td>Number of cases with click</td>
<td>30</td>
<td>6</td>
<td>15</td>
<td>9</td>
</tr>
<tr>
<td>Per cent of total</td>
<td>60%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean Q-x interval (sec.)</td>
<td></td>
<td>0.120</td>
<td>0.099</td>
<td>0.086</td>
</tr>
<tr>
<td>Range</td>
<td>0.150 &lt;-&gt; 0.114</td>
<td>0.120 &lt;-&gt; 0.084</td>
<td>0.107 &lt;-&gt; 0.063</td>
<td></td>
</tr>
<tr>
<td>t</td>
<td>3.80</td>
<td></td>
<td>1.90</td>
<td></td>
</tr>
<tr>
<td>Level of significance</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Table 1B

**Incidence and Timing of Ejection Click, Corrected for Heart Rate, in 30 Cases with Valvular Aortic Stenosis**

<table>
<thead>
<tr>
<th>Left ventricular pressure</th>
<th>Total</th>
<th>&lt; 150</th>
<th>&gt; 150</th>
</tr>
</thead>
<tbody>
<tr>
<td>mm. Hg</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of cases studied</td>
<td>30</td>
<td>17</td>
<td>13</td>
</tr>
<tr>
<td>Number of cases with click</td>
<td>30</td>
<td>17</td>
<td>13</td>
</tr>
<tr>
<td>Mean Q-x interval (sec.)</td>
<td>0.120</td>
<td></td>
<td>0.115</td>
</tr>
<tr>
<td>Range</td>
<td>0.092 &lt;-&gt; 0.156</td>
<td>0.099 &lt;-&gt; 0.144</td>
<td></td>
</tr>
<tr>
<td>t</td>
<td></td>
<td>0.78</td>
<td></td>
</tr>
<tr>
<td>Level of significance</td>
<td></td>
<td>N.S.</td>
<td></td>
</tr>
</tbody>
</table>

### Table 2A

**Timing of the Peak of the Systolic Murmur, Corrected for Heart Rate, in 50 Cases with Valvular Pulmonic Stenosis**

<table>
<thead>
<tr>
<th>Right ventricular pressure</th>
<th>Total</th>
<th>&lt; 80</th>
<th>80-120</th>
<th>&gt; 120</th>
</tr>
</thead>
<tbody>
<tr>
<td>mm. Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of cases (total 50)</td>
<td>13</td>
<td>21</td>
<td>16</td>
<td></td>
</tr>
<tr>
<td>Mean Q-peak interval (sec.)</td>
<td>0.305</td>
<td></td>
<td>0.342</td>
<td>0.385</td>
</tr>
<tr>
<td>Range</td>
<td>0.300 &lt;-&gt; 0.311</td>
<td>0.312 &lt;-&gt; 0.375</td>
<td>0.365 &lt;-&gt; 0.402</td>
<td></td>
</tr>
<tr>
<td>t</td>
<td>8.22</td>
<td></td>
<td>9.55</td>
<td></td>
</tr>
<tr>
<td>Level of significance</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\( p < 0.001 \)  \( p < 0.001 \)
steno'sis, varied according to the severity of the lesion. The highest degrees of splitting were generally observed in severe right ventricular hypertension (figs. 2 and 4, table 3). A significant correlation between right ventricular pressure and the A2-P2 interval was found over the entire range of pressures observed, as expressed by the coefficient r = 0.84, p < 0.001 (fig. 9). All three subgroups also showed significant differences in their mean values with an increase in splitting from 0.068 second in the mild to 0.115 second in the severe group. In contrast the A2-P2 interval in aortic stenosis was quite variable and no correlation with severity of the lesion could be found. No paradoxical splitting was observed in these 30 cases.

The relationship between the Q-x interval and dp/dt was found to be highly significant (r = -0.90, p < 0.001, fig. 10). Significant correlation was also found between the Q-peak amplitude interval and the duration of the right ventricular ejection time (r = 0.59, p < 0.01, fig. 11).

**Discussion**

In a detailed study Vogelpoel and Schrrie showed conclusively that in pulmonic stenosis the configuration of the stenotic murmur and the degree of splitting of the

<table>
<thead>
<tr>
<th>Left ventricular pressure</th>
<th>mm. Hg</th>
<th>&lt; 150</th>
<th>&gt; 150</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of cases (total 30)</td>
<td>17</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>Mean duration (sec.)</td>
<td>0.189</td>
<td>0.198</td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>0.160 - 0.220</td>
<td>0.177 - 0.218</td>
<td></td>
</tr>
<tr>
<td>S.E.</td>
<td>0.047</td>
<td>0.004</td>
<td></td>
</tr>
<tr>
<td>S.D.</td>
<td>0.188</td>
<td>0.014</td>
<td></td>
</tr>
<tr>
<td>t</td>
<td></td>
<td>0.190</td>
<td></td>
</tr>
<tr>
<td>Level of significance</td>
<td></td>
<td>N.S.</td>
<td></td>
</tr>
</tbody>
</table>

**Figure 10**

Composite diagram reflecting the relationship between the mean value of the Q-peak amplitude and Q-x interval, and of the A2-P2 interval to the right ventricular peak pressure. In patients with pressures lower than systemic the A2-P2 interval shows its greatest deviation from the ideal relationship, and in those with pressures in excess of systemic the Q-x interval shows the least reliability.

**Figure 11**

Relationship between Q-x interval and rate of pressure development (dp/dt) in 17 selected patients with pulmonic stenosis of varying severity.

*Table 2B*

Timing of the Peak of the Systolic Murmur, Corrected for Heart Rate, in 30 Cases with Valvular Aortic Stenosis

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second sound relate significantly to the degree of right ventricular hypertension. These observations, added to those reported earlier by Leatham and Weitzman, have established the definite value of the phonocardiogram in the estimation of the severity of pulmonic stenosis. Yet most clinicians have reserved only a supplementary role for the phonocardiogram in the diagnosis and evaluation of this lesion, because the predictability of the severity of right ventricular hypertension in a given case appeared uncertain. The data presented in this study demonstrate the reliability of this tool, particularly when the three major phonocardiographic features are used in combination for the estimation of right ventricular peak pressure or valve area (fig. 12). It shows that in every case a reasonably accurate prediction of the right ventricular pressure may be made. It also indicates that the Q-x interval is a more reliable measurement, when the right ventricular pressure is less than systemic, than with higher pressures, while for the A2-P2 interval the reverse applies. In contrast, the Q-peak amplitude interval proved to be more accurate than either of these over the full range of pressures observed in this series. Except in patients with

## Table 3

Duration of A2-P2 Interval, Corrected for Heart Rate, in 50 Cases with Valvular Pulmonic Stenosis

<table>
<thead>
<tr>
<th>Right ventricular pressure</th>
<th>mm. Hg</th>
<th>80-120</th>
<th>&gt; 120</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of cases</td>
<td>13</td>
<td>21</td>
<td>14*</td>
</tr>
<tr>
<td>Mean A2-P2 interval (sec.)</td>
<td>0.068</td>
<td>0.082</td>
<td>0.115</td>
</tr>
<tr>
<td>Range</td>
<td>0.054 &lt;--&gt; 0.097</td>
<td>0.050 &lt;--&gt; 0.125</td>
<td>0.084 &lt;--&gt; 0.177</td>
</tr>
<tr>
<td>S.E.</td>
<td>0.007</td>
<td>0.006</td>
<td>0.011</td>
</tr>
<tr>
<td>S.D.</td>
<td>0.20</td>
<td>0.020</td>
<td>0.030</td>
</tr>
<tr>
<td>t</td>
<td>2.17</td>
<td>2.64</td>
<td></td>
</tr>
<tr>
<td>Level of significance</td>
<td>p &lt; 0.05</td>
<td>p &lt; 0.02</td>
<td></td>
</tr>
</tbody>
</table>

* In two cases P2 could not be registered.

## Table 4

Duration of Ejection Time, Q-Peak of the Murmur Interval, dp/dt, and Q-x Interval in 17 Selected Patients with Valvular Pulmonic Stenosis

<table>
<thead>
<tr>
<th>No.*</th>
<th>RVPP (mm. Hg)</th>
<th>Ejection time (sec.)</th>
<th>Q-peak interval (sec.)</th>
<th>dp/dt</th>
<th>Q-x interval (sec.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>38</td>
<td>0.024</td>
<td>0.301</td>
<td>65</td>
<td>0.115</td>
</tr>
<tr>
<td>9</td>
<td>54</td>
<td>0.027</td>
<td>0.305</td>
<td>84</td>
<td>0.127</td>
</tr>
<tr>
<td>10</td>
<td>60</td>
<td>0.014</td>
<td>0.310</td>
<td>70</td>
<td>0.150</td>
</tr>
<tr>
<td>12</td>
<td>78</td>
<td>0.020</td>
<td>0.304</td>
<td>105</td>
<td>0.111</td>
</tr>
<tr>
<td>13</td>
<td>78</td>
<td>0.028</td>
<td>0.307</td>
<td>86</td>
<td>0.114</td>
</tr>
<tr>
<td>16</td>
<td>80</td>
<td>0.026</td>
<td>0.320</td>
<td>111</td>
<td>0.113</td>
</tr>
<tr>
<td>18</td>
<td>87</td>
<td>0.028</td>
<td>0.340</td>
<td>122</td>
<td>0.117</td>
</tr>
<tr>
<td>31</td>
<td>115</td>
<td>0.027</td>
<td>0.365</td>
<td>134</td>
<td>0.084</td>
</tr>
<tr>
<td>35</td>
<td>130</td>
<td>0.033</td>
<td>0.390</td>
<td>156</td>
<td>0.098</td>
</tr>
<tr>
<td>38</td>
<td>154</td>
<td>0.035</td>
<td>0.375</td>
<td>163</td>
<td>0.077</td>
</tr>
<tr>
<td>39</td>
<td>160</td>
<td>0.047</td>
<td>0.365</td>
<td>215</td>
<td>0.090</td>
</tr>
<tr>
<td>40</td>
<td>160</td>
<td>0.043</td>
<td>0.370</td>
<td>206</td>
<td>0.063</td>
</tr>
<tr>
<td>41</td>
<td>160</td>
<td>0.030</td>
<td>0.390</td>
<td>130</td>
<td>0.098</td>
</tr>
<tr>
<td>42</td>
<td>160</td>
<td>0.039</td>
<td>0.395</td>
<td>142</td>
<td>0.097</td>
</tr>
<tr>
<td>47</td>
<td>180</td>
<td>0.037</td>
<td>0.402</td>
<td>137</td>
<td>0.091</td>
</tr>
<tr>
<td>49</td>
<td>196</td>
<td>0.040</td>
<td>0.375</td>
<td>219</td>
<td>—</td>
</tr>
<tr>
<td>50</td>
<td>200</td>
<td>0.040</td>
<td>0.400</td>
<td>181</td>
<td>0.080</td>
</tr>
</tbody>
</table>

* Corresponds to numbers in figure 2.  
All values are corrected for heart rate.

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in our patients was uniformly low and therefore affected the timing of valve opening but little, it seems likely that the increased rate of change in the ventricular pressure \((dp/dt)\) is the chief remaining variable related to the reduction in the Q-x interval. In pulmonic stenosis, therefore, the timing of the click affords an indirect but highly sensitive measurement of \(dp/dt\). Data obtained in a selected group of patients in whom catheters of sufficient size were employed to guarantee accurate measurement bear out this conclusion \((r = -0.90, p < 0.001, \text{fig. 10})\). On the other hand, the lack of correlation between the Q-x interval and the left ventricular peak pressure in patients with aortic stenosis must reflect the influence of the often variable and uniformly higher resistance in the systemic circuit. However, a late click \((0.135 \text{ second or over})\) was not observed with pressures higher than 155 mm. Hg, and this measurement may serve to exclude severe hypertension \((\text{fig. 6})\). This absence of late clicks in severe left ventricular hypertension indicates further that with a large increment in the range of ventricular pressure development, the influence of peripheral resistance again becomes less important.

The chief factors responsible for the change in configuration of the ejection murmur may be increased resistance to the ejection of the stroke volume leading to prolongation of the ejection period, increased velocity of the blood flow across the valve, and an altered sequence of right ventricular depolarization. The last appears unlikely to play a large role, since significant prolongation of right ventricular depolarization occurs only in severe right ventricular hypertension and then but rarely. Furthermore, experimental studies of varying degrees of right bundle-branch block have shown no delay of right ventricular contraction until advanced degrees of block are reached. The latter was not present in any of our patients. More important to the explanation of the progressively later occurrence of the maximum amplitude in the murmur is the prolongation of the ejection time \((\text{fig. 11})\) where this measurement is demon-
strated in seven patients with mild and 10
with severe degrees of pulmonic stenosis.
Another factor, particularly important in the
timing of the murmur, is the increased velocity
of the blood flow across the narrowed valve
during the later stages of systole. Not only is
the pressure differential across the pulmonary
valve at least as high toward the end as during
the beginning of systole but, as Kjellberg
and co-workers have shown, the summit of
right ventricular peak pressure often occurs
in late systole. In addition, application of La-
place's law to hypertrophied hearts shows that
the force of contraction for a given amount of contracting chamber increases as
the length of its fibers decreases. Thus, partic-
ularly in severe pulmonic stenosis, the hyp-
ertrophied heart delivers its greatest force
under the greatest pressure differential toward
the end of the ejection phase, the very moment
when less volume remains to be ejected. This
combination of factors results in maximal
velocity of blood flow across the valve orifice
during that time. Thus it is understandable
that the degree of hypertrophy of the ventricle
is reflected in the configuration of the murmur
and in the timing of the maximal amplitude
of this murmur. In aortic stenosis, on the other
hand, the pressure gradient decreases toward
the end of systole on account of the rise in
pressure in the aorta, and blood velocity thus
reaches less extreme values. This course of
events, then, is different from that encoun-
tered in pulmonic stenosis, where, probably on
account of the characteristics of the pulmo-
mary vascular tree, there is no appreciable rise
in pulmonary artery pressure accompanying
the ejection of blood through the stenotic pul-
monary valve. Thus the configuration of aor-
tic murmurs and the timing of maximal am-
plitude cannot be expected to reflect peak
pressure to a significant degree.

Finally, in regard to the behavior of the
A₂-P₂ interval, prolongation of ventricular sys-
tole seems to play a very important role in
valvular pulmonic stenosis and in valvular aortic stenosis. Recent studies have shown that prolongation of the proto-
diastolic phase, due to the low pressure above
the pulmonary valves, is also an important
factor, contributing to the wide splitting of
the second sound in valvular pulmonic steno-
sis. On the other hand, in aortic stenosis the
vascular resistance distal to the valve plays an
additional role. In fact, it may constitute the
foremost reason why the timing of aortic closure is an dependable indicator of left ventricular pressure, a clinically well-known fac-
tor. This is borne out by the rare occurrence of "paradoxical splitting" in aortic stenosis, even in cases with severe valve narrowing.

Summary
A critical evaluation was made of the reli-
ability of three easily obtainable phonocardi-
ographic criteria in the estimation of ventricu-
lar peak pressure in congenital pulmonic and
aortic valvular stenosis.

In 50 patients with pulmonic stenosis the
Q-ejection click interval showed a significant
inverse relationship to peak pressure (r =
−0.77, p < 0.001), while no significant relation-
ship existed in aortic stenosis (r = 0.40,
p < 0.5). The timing of the peak magnitude of
the murmur again related significantly to the
right ventricular pressure (r = 0.72, p <
0.001) and with the log of pulmonary valve
area (r = −0.72, p < 0.001). In contrast no
significant relationships existed in aortic ste-
nosis. The degree of splitting also reflected the
severity of right ventricular hypertension
(r = 0.84, p < 0.001), while this measurement
in aortic stenosis proved quite variable and
did not reflect ventricular peak pressure.

The significance of a combination of these
criteria in predicting the severity of right
ventricular hypertension and in obviating the
need for cardiac catheterization is stressed.

An explanation for the mechanism under-
lying the observed changes is given. Q-ejec-
tion click interval in particular constitutes an
indirect but sensitive way (r = −0.90, p <
0.001) of estimating the rate of pressure de-
velopment (dp/dt) in the hypertrophied right
ventricle.

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References


The Biologic Effects of Unrestricted Food-Supply

It is well recognized that normally, and in a general way, the inclination for food is directly related to emptiness of the stomach rather than to the nutritional needs of the animal concerned. The usefulness of this arrangement is obvious: if the impulse to seek food were felt only when the body was in need of it to carry on its functions, there would be a risk that the animal would already have lost the energy necessary for the search. The normal animal eats, then, because the stomach is calling for food, not because the body is calling for nourishment, and the physiological situation is satisfactory as long as the food is of low nutritive value, as in the herbivora, or has to be worked hard for, as with the carnivora. Civilized man has broken through the natural safeguards of the process by obtaining a food-supply unlimited both in nutritive value and in amount, but he still regulates his eating by the demands of his stomach rather than by the needs of his body. The result is that by the time middle age is reached the absence of some degree of adiposity is statistically abnormal, and may well give rise to a suspicion of disease. Here again we find a gross functional discrepancy between man and animals which may well have a confusing effect in the application of experimental results.—The Collected Papers of Wilfred Trotter, F.R.S. London, Oxford University Press, 1946, p. 109.
Accuracy of the Phonocardiogram in Assessing Severity of Aortic and Pulmonic Stenosis

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