Assessing the Severity of Aortic Stenosis by Phonocardiography and External Carotid Pulse Recordings

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SUMMARY
Phonocardiograms and carotid pulse tracings were done on a group of 47 patients with all degrees of aortic stenosis and were compared with two groups of normals. Indices evaluated were pre-ejection period, left ventricular ejection time, maximum rate of arterial pulse rise, arterial half rise time (T time) and upstroke time, and timing of the peak intensity of the systolic murmur in relation to the electrocardiographic QRS and first heart sound. The indices most indicative of the presence of aortic stenosis and best correlated with its severity were the ejection time index, the maximal rate of rise of the carotid pulse and the timing of the peak of the systolic murmur. If, in a given case, all three of these indices fall outside of certain limits (ejection time index >0.42 sec, maximum rate of arterial pulse rise <500 mm Hg/sec, and Q wave to peak of murmur >0.19 sec), then severe aortic stenosis is almost invariably present.

Additional Indexing Words:
Phonocardiography
Aortic stenosis
Carotid pulse, in aortic stenosis
Arterial pulse, in aortic stenosis

ASSESSING the severity of aortic stenosis by non-invasive methods has intrigued many investigators. The three most popular avenues of research have been (1) analysis of the carotid pulse,1-7 (2) the timing and character of the aortic systolic murmur8-10 and, (3) correlation of abnormal heart sounds with the severity of stenosis.11-13

In this study, we examined carotid pulse tracings and phonocardiograms of a large series of patients with all degrees of aortic stenosis. Correlations of several of the most widely used indices with the degree of aortic stenosis were made. Each of the proposed indices for determining the severity of aortic stenosis was evaluated for its predictive value.

Materials and Methods
Phonocardiograms with carotid pulse tracings were done on a group of 47 patients with varying degrees of valvular aortic stenosis just prior to diagnostic cardiac catheterization. Criteria for selection were the presence of aortic stenosis without significant aortic insufficiency and good quality carotid pulse and sound tracings.

This group of patients was subsequently divided in two ways according to severity of aortic stenosis. One distinction was made by valve area above or below .75 cm², in the manner of Wood,14 who does not diagnose severe aortic stenosis in adult patients when the area of the opening in the valve is greater than .75 cm². Within this division, the severe group had 37 patients (13 females, 24 males), aged between 23 and 69 years, with valve areas between .32 and .75 cm². The mild group had 10 patients (3 females, 7 males), aged between 18 and 65 years, with valve areas between .75 and 3.70 cm². The second division of the patients was made by determining whether the peak aortic gradient was greater or less than 50 mm Hg. The severe group had 36 patients (11 females, 25 males), aged between 23 and 69 years, with a range of gradient from 50 to 145 mm Hg. The mild group had 11 patients (5 females, 6 males), aged between 18 and 69 years, with a range of gradient from 12 to 44 mm Hg.

Phonocardiograms and carotid pulse tracings were likewise done on two groups of 20 normal subjects. The first group consisted of an unselected series of normals ranging in age from 19 to 69 years (8 females, 12 males). The second group consisted of a series of 5 females and 15 males between the ages of 13 and 62 years who were considered on clinical grounds to have innocent systolic ejection murmurs.

During held mid expiration, phonocardiograms and carotid pulse tracings were simultaneously recorded, the latter by the method described by Tavel.15
filled cuff was placed around the neck and inflated to 5 to 10 mm Hg. The cuff was then connected by a short rubber tube to a piezo electric microphone (Lumiscope MP-1A). Phonocardiograms were obtained with a Cambridge microphone and were routinely recorded in the second right and second left intercostal space as well as in the lower left sternal border and apex.

Electrical signals were recorded graphically with an Electronics for Medicine Recorder (Model DR-8). Incoming signals were filtered with a band-pass filter set at 0.1 to 200 Hz. The time constant for this circuit was determined to be 1.8 sec. Although this was shorter than ideal, it was comparable with that obtained in most commercially available machines and, being approximately seven times the duration of ventricular systole, allowed a reasonably accurate pulse reproduction. Moreover, we have compared these pulse curves with those obtained with a strain gauge having an infinite time constant, and have found the arterial curves to be virtually identical. We have used this method to measure left ventricular ejection times in a series of 27 normal males and found a regression equation identical to that of Weissler et al., who used a strain gauge transducer. The upper limit for pulse recording was 200 Hz. Sound signals were filtered into 120 to 500 Hz and 400 to 2000 Hz bands. Recording speed was 100 mm/sec and time lines were set at 0.1 sec intervals.

Measurements

Several measurements were made from the simultaneous recordings. Preejection period (PEP) and left ventricular ejection time (LVET) were measured as suggested by Weissler and Garrard. Ejection time index (ETI) was calculated by the regression equation of the same authors, (LVET for males = 1.7 HR + 413; LVET for females = 1.6 HR + 418). Maximal rate of rise of the carotid pulse was obtained by fitting a line to the steepest portion of the carotid upstroke. This line (see fig. 1) was extrapolated to the peak of the carotid tracing. The time to reach maximum pulse pressure along this line was then taken and divided into the pulse pressure.

Pulse pressure was arrived at by careful sphygmomanometer measurement of brachial blood pressure just prior to the recordings. Upstroke time (U time) and time required for the pulse to reach half its height (T time) were measured directly from the carotid tracing as suggested by several authors.

Sound tracings were examined and the point of maximum intensity of the systolic ejection murmur was determined. This was expressed as a Q to peak and S₁ to peak time. All of the above measurements represented the average of values taken on five heart beats.

Right and retrograde or transseptal left heart catheterization was done on all patients in the aortic stenosis group. Simultaneous left ventricle-aortic pressures were obtained. The Fick method was used to determine cardiac output and aortic valve area was calculated by the Gorlin and Gorlin formula.

Figure 1

Example of typical carotid and sound tracing from a patient with severe aortic stenosis. Q and S₁ to peak of murmur were measured as indicated. T time and U time are illustrated on the carotid pulse. Maximal rate of rise of the carotid pulse was measured by fitting a line to the steepest portion of the carotid pulse as shown.
Table 1

Correlations of Hemodynamic and Phonocardiographic Variables in 47 Patients with Aortic Stenosis

<table>
<thead>
<tr>
<th></th>
<th>Pulse pressure</th>
<th>PEP</th>
<th>LVET corr.</th>
<th>PEP/LVET</th>
<th>Maximum rate rise</th>
<th>U time</th>
<th>T time</th>
<th>Q-Peak</th>
<th>S1-Peak</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valve area</td>
<td>.069</td>
<td>.038</td>
<td>-.380†</td>
<td>.061</td>
<td>-.289*</td>
<td>-.200</td>
<td>-.267</td>
<td>-.398†</td>
<td>-.576†</td>
</tr>
<tr>
<td>Gradient</td>
<td>-.273*</td>
<td>-.130</td>
<td>.468†</td>
<td>.013</td>
<td>-.375†</td>
<td>.329*</td>
<td>.293*</td>
<td>.389†</td>
<td>.365†</td>
</tr>
<tr>
<td>Cardiac index</td>
<td>.128</td>
<td>.042</td>
<td>-.130</td>
<td>.046</td>
<td>-.075</td>
<td>.128</td>
<td>.051</td>
<td>.082</td>
<td>-.071</td>
</tr>
<tr>
<td>LVET at rest</td>
<td>-.200</td>
<td>-.224</td>
<td>.217</td>
<td>-.306*</td>
<td>-.304*</td>
<td>.225</td>
<td>.039</td>
<td>.347*</td>
<td>.306</td>
</tr>
</tbody>
</table>

*Significant at 0.05 level.
†Significant at 0.01 level.

Abbreviations: PEP = pre-ejection period; LVET corr. = left ventricular ejection time, corrected for heart rate; U time = upstroke time; T time = time required for pulse to reach half its height; Q-Peak = beginning QRS to peak of murmur; S1-Peak = beginning of S1 to peak of murmur.

Results

The mean values for aortic valve area for all our patients with aortic stenosis was .79 cm², standard deviation .62, range .32 to 3.70 cm². The mean for peak aortic valve pressure gradient was 77.1 mm Hg, so 33.3, range 12 to 145 mm Hg, and the mean for cardiac index was 3.0 (so .07), with a range of 1.4 to 4.6 L/min/M². A system of linear regression analysis was used. Correlations of values obtained from indirect recordings with catheterization data is shown in table 1. It is noted that the most highly significant correlations are obtained with the timing of the peak of murmur, the ejection time index and the maximum rate of rise of the carotid pulse.

The patients with aortic stenosis are divided by gradient and valve area and compared with normal subjects in tables 2 and 3. Mean, standard deviation, and significance of the difference between the means of the three highly correlated variables are given. These data are represented graphically in figures 2, 3 and 4.

An attempt was made to utilize each of the best correlated variables in a cumulative manner (ejection time index >.42 sec, rate of rise <500 mm Hg/sec, and Q to peak of murmur >.19 sec). Twenty-seven of 28 patients (96%) with values outside the above limits for all three measures were found to have severe disease as determined by valve area and gradient. Records of the group of normal patients with murmurs were also examined and none had values outside the above limits for all three measures.

Table 2

Comparison of Patients with Mild and Severe Aortic Stenosis with Normal Subjects

<table>
<thead>
<tr>
<th></th>
<th>Gradient &lt;50 mm Mean</th>
<th>Gradient &gt;50 mm Mean</th>
<th>Normal Mean</th>
<th>P Value</th>
<th>Comparison</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maximum rate of rise (mm Hg/sec)</td>
<td>626 ± 432</td>
<td>328 ± 193</td>
<td>867 ± 365</td>
<td>&lt;.01</td>
<td>2-3</td>
</tr>
<tr>
<td>LVET corrected (sec)</td>
<td>.415 ± .028</td>
<td>.438 ± .022</td>
<td>.392 ± .022</td>
<td>&lt;.01</td>
<td>1-2, 2-3</td>
</tr>
<tr>
<td>S1-Peak of murmur (sec)</td>
<td>.195 ± .036</td>
<td>.197 ± .036</td>
<td>.134 ± .010</td>
<td>&lt;.01</td>
<td>1-3, 2-3</td>
</tr>
<tr>
<td>Q-Peak of murmur (sec)</td>
<td>.247 ± .029</td>
<td>.250 ± .035</td>
<td>.187 ± .015</td>
<td>&lt;.01</td>
<td>1-3, 2-3</td>
</tr>
</tbody>
</table>

Table 3

Comparison of Patients with Mild and Severe Aortic Stenosis with Normal Subjects

<table>
<thead>
<tr>
<th></th>
<th>Valve area &lt;.75 cm² Mean</th>
<th>Valve area &gt;.75 cm² Mean</th>
<th>Normal Mean</th>
<th>P Value</th>
<th>Comparison</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maximum rate of rise (mm Hg/sec)</td>
<td>365 ± 219</td>
<td>535 ± 491</td>
<td>867 ± 345</td>
<td>.01</td>
<td>1-3</td>
</tr>
<tr>
<td>LVET corrected (sec)</td>
<td>.438 ± .024</td>
<td>.410 ± .016</td>
<td>.392 ± .022</td>
<td>.01</td>
<td>1-3, 1-2</td>
</tr>
<tr>
<td>S1-Peak of murmur (sec)</td>
<td>.202 ± .038</td>
<td>.168 ± .038</td>
<td>.134 ± .010</td>
<td>.01</td>
<td>1-3</td>
</tr>
<tr>
<td>Q-Peak of murmur (sec)</td>
<td>.256 ± .029</td>
<td>.228 ± .034</td>
<td>.187 ± .015</td>
<td>.01</td>
<td>1-3, 2-3</td>
</tr>
</tbody>
</table>

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Figure 2

Scattergram comparing the maximal rate of rise of the carotid pulse in normals, mild aortic stenosis and severe aortic stenosis. Values shown are mean ± 1 SD.

Discussion

Much has been written regarding the diagnosis of aortic stenosis by pulse recordings and phonocardiography. However, some of the results of these techniques are limited in their usefulness. It has been well shown, for example, that the presence of an S₄ gallop correlates with a transvalvular gradient of 75 mm Hg. But this cannot be applied in the pediatric age group because of the frequency of a physiologic S₄ or in patients over 40 years of age because of the incidence of degenerative heart disease. The presence of paradoxical splitting of the second heart sound in association with a normal QRS and a basal systolic murmur suggests severe aortic outflow obstruction. This is often difficult to hear or record, however, not only because of the diminished intensity of the aortic closure sound in stiff and calcified valves, but also because of the tendency of the murmur to obscure the sound of pulmonic closure.

The three measurements we found to be most helpful were the ejection time index, the maximal rate of rise of the carotid pulse, and the timing of the peak of the murmur. Each of these was examined separately by several authors. Prolongation of the ejection time has been shown by many authors to be associated with the presence of aortic stenosis, though not necessarily an index of its severity. Our data not only confirm its usefulness in determining the presence of stenosis but also show a very statistically significant correlation with the transvalvular gradient at rest. LVET remains long when congestive heart failure complicates aortic stenosis. Our highest normal LVET (corr.) was 0.43 sec. Nineteen patients in our series had an LVET (corr.) of greater than 0.43 sec, and of these 18 had valve areas of 0.75 cm² or
ASSESSING SEVERITY OF AORTIC STENOSIS

Figure 4

Scattergrams showing the timing of peak intensity of murmurs in normals and patients with mild and severe aortic stenosis. Values shown are mean ± 1 sd.

less. The only exception had a valve area of 0.84 cm² with a gradient of 76 mm Hg.

Mild aortic stenosis usually yields normal LVET (corr.) values. Nobody with aortic stenosis in our series (any severity) had an LVET (corr.) of less than .39 sec although seven normals did have such values. We conclude, therefore, that only an LVET (corr.) of greater than 0.43 sec is very helpful clinically, virtually insuring the presence of severe aortic stenosis. If LVET (corr.) is less than 0.39 sec significant aortic stenosis may be ruled out.

PEP/LVET was calculated for each patient, and in the aortic stenosis group the mean was 0.25 ± 0.05 sec. In the normal group the mean was .355 ± .013 sec. Several authors, 4, 6, 20 have shown that this ratio is significantly smaller in the aortic stenosis group and that it persists in the presence of congestive heart failure. 22 23 PEP/LVET ratio does not correlate with the severity of stenosis in this group, however.

U time and T time have been used by several authors in the past as indices of the rate of rise of the carotid pulse. 6, 19 We found these less helpful than the maximum rate of rise of the carotid pulse. Recently, with the use of computer technology, more exact measurements of the rate of rise of the indirect carotid pulse have been obtained and utilized in the diagnosis of aortic outflow disease. Our results, obtained by a simpler method, compare very favorably with those of Lyle et al., 5 using the computerized technique. Nineteen of 20 normals had a rate of rise greater than 500 mm Hg/sec. A total of 32 patients in our aortic stenosis series had a rate of rise of 400 mm Hg/sec or less. Of these, 29 had valve areas of less than 0.75 cm². However, five patients with severe stenosis, aged 58 to 69 (as compared to a mean age of 53 for the entire group of diseased patients), showed normal rates of rise (greater than 500 mm Hg/sec) indicating that a normal finding does not rule out severe aortic stenosis. Such a rapid rise might be attributable to reduced distensibility of the peripheral vessels in this fairly aged arteriosclerotic population. Mild aortic stenosis resulted in variable values, overlapping considerably with both normals and those individuals with severe aortic stenosis. As a result, therefore, the only clinically useful finding with this measurement is a markedly reduced value of 400 mm Hg/sec or less, suggesting severe aortic stenosis.

The measurement which correlated most highly with the degree of aortic stenosis was the timing of the peak of the systolic murmur. This has been suggested by several authors as an index of severity, 8, 9, 10 but has been disputed by others. 24 We attempted to improve upon this correlation by
rate correcting each of the values obtained (value obtained \( \sqrt{R-R\text{ interval}} \)). This did not, however, improve our accuracy. No normal individual with an innocent ejection murmur had a Q to peak time of longer than 0.24 sec. In the aortic stenosis group, 24 cases had Q-peak times of greater than 0.24 sec, and of these, 22 had severe stenosis. Sixteen patients with severe stenosis did, however, fall into the normal range of 0.20 to 0.24 sec. Mild aortic stenosis also usually falls in the normal range. No patients with severe stenosis had values less than 0.20 sec, whereas 11 of the 20 normals had values in this range. We conclude, therefore, that a finding of a markedly prolonged time of greater than 0.24 sec strongly suggests severe stenosis, whereas a short time of less than 0.20 sec strongly denies such a diagnosis.

Combining the three most highly correlated indices yields more clinically useful information, particularly in assessing the severity of aortic stenosis. Values outside all three of the delineated limits, ejection time index > .42 sec, maximum rate of rise < 500 mm Hg/sec, and Q to peak of murmur > .19 sec are highly suggestive of severe aortic stenosis. All such patients except one (27 of 28, 96%) had severe stenosis as determined by pressure gradient and valve area. Also, the overwhelming majority of patients with severe aortic stenosis (27 of 36 or 75%) had measures outside all three limits.

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