Slopes of the Carotid Pulse Wave in Normal Subjects, Aortic Valvular Diseases, and Hypertrophic Subaortic Stenosis

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SUMMARY
Variations in certain slopes of the carotid pulse which exist among normal subjects, patients with aortic valvular disease, and those with hypertrophic subaortic stenosis were examined. Records from 256 subjects were quantified with computer technics. The externally recorded volume pulses were related to simultaneously recorded brachial artery pressures (cuff); thus the slopes are expressed in mm Hg/sec. Several slopes were studied: the maximum rate of pressure rise, the mean systolic upstroke (onset of carotid upstroke to peak systolic excursion), and the least square fits of the first two-thirds of the upstroke and the first two-thirds of diastole. The least square slopes of 98% of the patients with aortic stenosis were <457 mm Hg/sec, whereas none of those with hypertrophic subaortic stenosis was that low. The greatest overlap was between patients with aortic stenosis and those with aortic stenosis and aortic insufficiency. The slopes correlated poorly with valve areas and gradient in aortic stenosis and combined aortic stenosis and aortic insufficiency. The least square slope in normal subjects is not related to heart rate or age. Thus, the decreased slopes in patients with aortic stenosis are related to many factors, including myocardial function, but influenced by the size of the aortic valve opening and the properties of the vessels.

Additional Indexing Words:
Carotid pulse Computers

Clinically, the palpation of the carotid arterial pulse for determination of rapidity of pressure rise has often aided in the diagnosis of aortic stenosis and aortic insufficiency. Previous studies concerning the carotid pulse have dealt largely with time intervals such as upstroke time, preejection period, and systolic ejection time, and with ratios of the intervals. Computer technics have made it possible to analyze pulse wave variations that occur in diseased states, and a recent study demonstrated statistically significant variations in the contours of the carotid pulse in normal subjects and in patients with aortic valvular lesions. The present investigation was designed to quantify the pulse with respect to slope variations in mm Hg/sec among patients with various types of aortic valvular lesions, hypertrophic subaortic stenosis, and normal subjects.

Methods
Patient Selection
Recordings from 256 patients and normal subjects were used in this study. One hundred twenty were classified as normal, 48 had "pure" aortic stenosis, 35 had "pure" aortic insufficiency,
34 had combined aortic stenosis and aortic insufficiency, and 19 had hypertrophic subaortic stenosis. In the normal group, each of the 120 subjects had a negative history and physical examination and a negative maximum Bruce exercise test. The ages of the normal group ranged from 30 to 74 years, with a mean age of 51 years.

The patients with aortic valvular disease and hypertrophic subaortic stenosis were selected only on the basis of available carotid pulse recording and cardiac catheterization data, including aortic and left ventricular pressures. In addition, sufficient data were required from cineangiograms of the aortic root or from biplane left ventricular angiography to adequately evaluate the aortic insufficiency. Four patients meeting these requirements were excluded because of technically poor carotid pulse recordings.

The patients with aortic valvular lesions were classified according to the following criteria:

1. Aortic stenosis
   Patients with a peak gradient across the valve of 10 mm Hg or greater. No patients with an aortic diastolic murmur or demonstrable aortic insufficiency were included in this group. By these criteria, therefore, four patients with minimal stenosis (peak gradients less than 37 mm Hg) were included. These patients were deliberately included so that a wide spectrum of hemodynamics might be demonstrated.

2. Aortic insufficiency
   These patients had demonstrable aortic valvular regurgitation by angiocardiography. No patient was included whose peak gradient across the aortic valve was greater than 10 mm Hg. All had significant aortic diastolic murmur present. Obviously, some patients had minimal aortic regurgitation, but, again, these were included so that the various degrees of abnormality encountered might be presented.

3. Combined aortic stenosis and aortic insufficiency
   Each patient with combined aortic stenosis and aortic insufficiency had a peak gradient across the aortic valve of 10 mm Hg or greater, demonstrable aortic insufficiency, and an aortic diastolic murmur.

4. Hypertrophic subaortic stenosis
   Patients included in this category had demonstrable gradients across the obstruction and no evidence of aortic insufficiency. Characteristic angiographic findings of hypertrophic subaortic stenosis were present in each patient.

**Recording Technics**

A glycerin pellet* strapped to the subject's neck by a wide rubber band was used to record the carotid pulse wave. The pellet was attached to a PM5-0.2-350 Statham strain gauge transducer by a short strip of Tygon tubing. A great deal of effort was focused on obtaining a pure arterial pulse tracing, free of venous components. The carotid pulse was recorded simultaneously with the electrocardiogram (with the Y lead of the Frank vectorcardiographic system) and the kinetocardiogram. In the present study only the carotid pulse wave and the electrocardiogram, for timing purposes, were used.

**Computer Technics**

Two or three complexes of each recording were digitized on a Gerber digitizer at 0.0102-sec intervals. Simultaneously, the onset of the Q wave (determined from the electrocardiogram), the onset of the carotid upstroke, and the carotid incisural notch were marked for each complex. The curves were then calibrated according to the blood pressure. This was accomplished by setting the onset of ejection to the cuff diastolic blood pressure and the peak of the tracing to the systolic blood pressure. The remainder of the tracing was adjusted accordingly between these two points. This allowed the calculation of the various slopes in the pulse in mm Hg/sec. The data from two to three consecutive complexes were averaged so that more representative values could be obtained for each patient. The averaging of three complexes was an arbitrary decision. It is

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possible one representative complex would have been sufficient.

The various slopes of the carotid upstroke were determined in several ways. First, the upstroke was differentiated to obtain the maximum rate of pressure rise (max dp/dt). The mean carotid upstroke (CU-Peak slope) was determined by constructing a line from the onset of the upstroke to the peak of the curve and calculating the slope of this line (fig. 1). It was found that this line was a poor approximation of the upstroke of the tracing because of the rounded contour of the curve near the peak. The 2/3 least square slope, on the other hand, fitted the upstroke much more tightly. This slope was obtained by dividing the amplitude of the upstroke into thirds and fitting a line to the first two-thirds by a linear least squares method (fig. 1).

Two slopes were studied in the diastolic portion of the pulse. A point 0.0408 sec beyond the carotid incisural notch was designated as the starting position for each of the slopes. The 1/2 diastolic slope was determined by adjusting a line by least squares fit between the starting point and a position equal to 1/2 of the diastolic length beyond this (fig. 1). The 2/3 diastolic slope had as its terminal point a position 2/3 the length of the diastole beyond the same starting point. A line was placed between these points by the least squares fit method (fig. 1).

A previous study attempted to determine an index for the severity of aortic stenosis. This index was described as aortic dp/dt multiplied by stroke volume divided by aortic pulse pressure. However, Warner (personal communication) suggested a different correction factor for the central aortic pressure to indicate the severity of aortic stenosis; he calls it the aortic stenosis index. It is determined by dividing the 2/3 systolic slope by the pulse pressure to the 0.7 power. We have chosen to call it the aortic valve index (AVI), since we used the carotid pulse instead of the central aortic pressure.

Results

Table 1 presents the means and standard deviations for the different variables included for each group of subjects.

Figure 2 presents a cumulative per cent plot for the maximum rate of pressure rise. The abscissa contains the values for the variable in mm Hg/sec. The ordinate displays the per cent of patients. Each point on any given line represents the per cent of patients with values that fall below the corresponding value on the abscissa. It is evident that all the patients in the aortic stenosis group have slower maximum rates of pressure rise than do many of the patients in the other groups. It can be seen that 88% of the patients with aortic stenosis have maximum rates of pressure rise below 639 mm Hg/sec, whereas only 30% of normal subjects have values below this. It should be pointed out that only 9% of patients with aortic insufficiency and none of the patients with hypertrophic subaortic stenosis have values lower than this. The patients with aortic insufficiency tend to have much faster rates of pressure rise than do the subjects in the other groups. Eighty per cent of patients with aortic insufficiency have values above 1027 mm

Table 1

Means and Standard Deviations of the Variables for Each Group of Subjects

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Peak gradient</th>
<th>2/3 slope</th>
<th>Maximum rate of pressure rise</th>
<th>Mean CU-Peak slope</th>
<th>AVI</th>
<th>3/4 diastolic slope</th>
<th>2/3 diastolic slope</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aortic stenosis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>48</td>
<td>85 ± 36</td>
<td>257 ± 131</td>
<td>497 ± 204</td>
<td>165 ± 74</td>
<td>20.7 ± 8.1</td>
<td>–54 ± 51</td>
<td>–55 ± 50</td>
</tr>
<tr>
<td>Aortic insufficiency</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>45</td>
<td>2</td>
<td>1033 ± 458</td>
<td>1637 ± 767</td>
<td>558 ± 316</td>
<td>46 ± 14</td>
<td>–141 ± 67</td>
<td>–132 ± 52</td>
</tr>
<tr>
<td>Aortic stenosis and aortic insufficiency</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>44</td>
<td>57 ± 31</td>
<td>458 ± 203</td>
<td>755 ± 346</td>
<td>274 ± 139</td>
<td>29.0 ± 10.3</td>
<td>–76 ± 45</td>
<td>–80 ± 37</td>
</tr>
<tr>
<td>Hypertrophic subaortic stenosis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>44</td>
<td>53 ± 29</td>
<td>824 ± 389</td>
<td>1226 ± 449</td>
<td>559 ± 290</td>
<td>55.3 ± 12.0</td>
<td>–48 ± 61</td>
<td>–51 ± 52</td>
</tr>
</tbody>
</table>

Slopes are expressed in mm Hg/sec.
See Methods section and figure 1 for way in which these values were calculated.

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Cumulative per cent plot of values for the maximum rate of pressure rise for patients in each group which fall below a particular value. The slopes (numbers) on the abscissa read from top down. It is evident that the patients with aortic stenosis generally have smaller maximum rates of pressure rise, while the largest values for this variable are seen in subjects with aortic insufficiency. The steepness of the AS plot indicates that the values for this group of patients form a tight array. On the contrary, the AI group has much broader limits, as is evident from the smaller slope of this line.

Abbreviations: AS = aortic stenosis; AI = aortic insufficiency; AS + AI = combined aortic stenosis and aortic insufficiency; HSAS = hypertrophic subaortic stenosis; NO = normal.

Cumulative per cent plot showing the per cent of patients in each group in which the values for the 2/3 least square slope fall below a given value. It is evident that this variable separates the groups from one another well, with the AS group having the lowest values and the AI group having the highest. The steep slope of the AS linear plot indicates that the values for all the patients in this group fall within narrow limits. However, the AI plot is much less steep and, hence, this group of patients has values for the 2/3 least square slope that extend over a wide range. Abbreviations: same as in figure 2.

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Hg/sec, whereas none of the patients with aortic stenosis have values that high.

Figure 3 presents the cumulative per cent plot for the 2/3 slopes. Patients with aortic stenosis generally have smaller values for 2/3 slopes than do normal subjects, as evidenced by the fact that 75% of patients with aortic stenosis have 2/3 slopes below 275 mm Hg/sec, whereas only 9% of normal subjects do. Patients with aortic insufficiency or hypertrophic subaortic stenosis have considerably larger slopes than do the patients with aortic stenosis. It can be seen that all patients with hypertrophic subaortic stenosis and 91% of patients with aortic insufficiency have slopes above 397 mm Hg/sec, whereas only 4% of patients with aortic stenosis do.

Figure 4 presents the cumulative per cent
plot for the 2/3 diastolic slope. It can be seen that 66% of patients with aortic insufficiency have slopes more negative than −122 mm Hg/sec, whereas none of the normal subjects do. Also, it should be pointed out that only 6% of patients with combined aortic stenosis and aortic insufficiency, 5% of those with hypertrophic subaortic stenosis, and 4% of those with aortic stenosis have slopes below this same value.

Figure 5 presents the cumulative per cent plot for the aortic valve index. It can be seen that 96% of patients with aortic stenosis and 68% of patients with combined aortic stenosis and aortic insufficiency have indices below 32.68. On the other hand, only 31% of normal subjects, 17% of patients with aortic insufficiency, and none of the patients with hypertrophic subaortic stenosis have indices below this same value.

Table 2 presents the P values from the Student t-test for the significant differences between the groups for each variable studied. It should be noted that many of the values are less than 0.001 and are, therefore, highly significant.

Discussion

Obviously, the method of calculating slopes from the carotid pulse has certain limitations. The inaccuracy of cuff recorded blood pressures could significantly affect the slopes; however, the pressures in most instances were taken by carefully trained technologists at the time when the traces were recorded. In the few patients (six) where the pressure was inadvertently not taken at the time of recording, all blood pressures in the hospital record were averaged in order to obtain a representative sample. Even then, there are undoubtedly discrepancies between the true pressure in the carotid pulse and that recorded from the arm; however, this is a limitation in the technic which is unavoidable when noninvasive methods are used. Nevertheless, the slopes as calculated in this study (maximum rate of pressure rise; max dp/dt) are almost identical to those reported by Mason, Braunwald, Ross, and Morrow by direct measurement of the peak dp/dt or max dp/dt from the brachial artery. This was surprising since the externally recorded carotid pulse differs in contour from both the directly recorded carotid pressure pulse and the brachial arterial pressure pulse (see table 3).

A fair partition of patients with aortic stenosis from the other groups can be accomplished by use of any of the following slopes as mentioned in this study: the aortic valve index (fig. 5), the maximum rate of pressure rise (fig. 2), or the 2/3 slope (fig. 3). However, the 2/3 slope is the most reliable, particularly in distinguishing “pure” aortic stenosis from combined aortic stenosis and aortic insufficiency. It can be seen from figure

### Table 2

<table>
<thead>
<tr>
<th>Group</th>
<th>2/3 slope</th>
<th>Maximum rate of pressure rise</th>
<th>2/3 diastolic slope</th>
<th>AVI</th>
</tr>
</thead>
<tbody>
<tr>
<td>NO vs. AS</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>0.536</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>NO vs. AI</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>0.027</td>
</tr>
<tr>
<td>NO vs. AS and AI</td>
<td>0.002</td>
<td>0.029</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>NO vs. HSAS</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>0.243</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AS vs. AI</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AS vs. AS and AI</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>0.017</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AS vs. HSAS</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>0.710</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AI vs. AS and AI</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AI vs. HSAS</td>
<td>0.084</td>
<td>0.036</td>
<td>&lt;0.001</td>
<td>0.022</td>
</tr>
<tr>
<td>AS and AI vs. HSAS</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>0.019</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Abbreviations: NO = normal subjects; AS = aortic stenosis; AI = aortic insufficiency; AS and AI = combined aortic stenosis and insufficiency; HSAS = hypertrophic subaortic stenosis.

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Table 3

Comparison of Max $dp/dt$ from the Externally Recorded Carotid Pulse (Present Study) and the Directly Recorded Brachial Arterial Pressure Pulse (Reference 8)*

<table>
<thead>
<tr>
<th></th>
<th>Max $dp/dt$</th>
<th>N</th>
<th>AS</th>
<th>AS and AI</th>
<th>AI</th>
<th>HSAS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Direct brachial artery</td>
<td>811 ± 185</td>
<td>358 ± 85</td>
<td>724 ± 212</td>
<td>1736 ± 530</td>
<td>1092 ± 372</td>
<td></td>
</tr>
<tr>
<td>Present study</td>
<td>893 ± 317</td>
<td>497 ± 204</td>
<td>755 ± 346</td>
<td>1637 ± 707</td>
<td>1226 ± 449</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: Same as in table 2.
*All values are expressed in mm Hg/sec.

that 96% of the patients with aortic stenosis have values below 397 mm Hg/sec, whereas only 41% of the patients with the combined lesion have such low values. The 2/3 slope (fig. 3) or the maximum rate of pressure rise (fig. 2) may be used for separation of patients with aortic insufficiency from other subjects. For example (fig. 3), 89% of the patients with aortic insufficiency have 2/3 slopes above 518 mm Hg/sec, whereas no other group approaches such a high percentage. Figure 2 shows similar results. The 1/2 diastolic slope and the 2/3 diastolic slope are also significantly different in patients with aortic insufficiency and those in the other groups (2/3 diastolic slope as shown in fig. 4).

Hypertrophic subaortic stenosis is distinguished from valvular aortic stenosis primarily by the fact that the rapidity of the carotid upstroke is exceedingly fast in the former. For example, 96% of the patients with aortic stenosis had 2/3 slopes below 457 mm Hg/sec, whereas all the patients with hypertrophic subaortic stenosis had faster rates of pressure rise (figs. 3 and 5).

It should be noted that the slopes in aortic valve disease are poorly related to aortic valve area or to aortic-ventricular gradient ($r = -0.49$). The relationship is significant ($P < 0.01$); however, there is considerable scatter, such that prediction of valve area or gradient in a given patient would not be justified. Even correction of the 2/3 slope for pulse pressure (AVI) does not appreciably improve its relationship to gradient ($r = 0.50$, $P < 0.01$; fig. 6).

Some physiologic factors that could affect the rate of pressure rise in the carotid pulse, particularly in normal subjects, were also studied. Freis et al. demonstrated that the normal carotid pulse wave contains two systolic peaks. The first is related to maximum flow in the aorta and the second to maximum aortic pressure. He found that with increasing age the second systolic peak and the incisural notch increased in relation to the first systolic peak. These changes presumably reflect alterations in the arterial vessel distensibility. As a result of this, the slope of a line between the onset of the carotid upstroke and the highest systolic peak (mean CU-Peak slope) would tend to yield a bimodal array, which does occur. Thus, the decreased slopes generally appear in the older age group, while the steeper slopes appear in the younger group. However, with 2/3 slope a unimodal distribution occurs,
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and this slope correlated poorly with age (r = -0.16). Although increasing age could conceivably decrease contractility and reduce the rate of pressure rise, it apparently is not a major factor in determining the 2/3 slope of the carotid pulse. In addition, it is possible that heart rate could alter the slopes, since an increased heart rate shortens the duration of systole, and, therefore, possibly increases the slopes. However, again a poor correlation was found between the 2/3 slope and heart rate (r = 0.20). Thus, age and heart rate are not closely related to the slopes, and the aortic-ventricular gradient or valve area are not appreciably related to the slopes in aortic disease. Obviously other factors not apparent from this study, possibly including myocardial contractility, affect or determine the rate of pressure rise in the carotid pulse. Nevertheless, two positive correlations were found in normal subjects. The maximum rate of pressure rise as related to pulse pressure had an r of 0.78, and the 2/3 slope related to the systolic pressure had an r of 0.493; both were significant.

References

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