A Clinical Study of the Brachial Arterial Pulse Form
With Special Reference to the Diagnosis of Aortic Valvular Disease

By ERNEST W. HANCOCK, M.D., AND WALTER H. ABELMANN, M.D.

A survey was made of the clinical usefulness of pulse forms from direct brachial arterial puncture of 250 patients. A prolonged duration of systolic upstroke and an anacrotic notch were found in most instances of severe aortic stenosis, but also frequently in patients with insignificant degrees of aortic stenosis, and occasionally in normal subjects. Characteristic, but nondiagnostic changes in the pulse form were noted in aortic insufficiency, mitral insufficiency, anemia, hyperthyroidism and exercise. The diagnostic significance of these findings is discussed.

The recording of the arterial pressure pulse by direct puncture of the brachial artery has become a routine procedure in clinical cardiac physiology. In the diagnosis of aortic stenosis, particularly, the arterial pulse tracing has been considered of diagnostic importance. The pulse contour has been used in recent years to confirm the clinical diagnosis of aortic stenosis, to assist in the selection of patients for surgical treatment of aortic stenosis, and to evaluate the results of aortic commissurotomy. The characteristic abnormalities of the pulse in aortic stenosis have been considered to be a prolonged duration of the systolic upstroke, an anacrotic notch, low systolic and diastolic pressures, and a narrow pulse pressure. These features have long been known to the clinician, and were described and illustrated in detail with externally recorded sphygmograms a generation ago. There have also been extensive studies in experimental animals, demonstrating in addition a prolonged duration of systole in aortic stenosis and a lesser degree of transformation of the arterial pulse contour in the course of transmission to the periphery. They have also suggested that the degree of alteration of the pulse form is related to the degree of the aortic stenosis. It is upon these experimental studies, in addition to the older clinical observations, that present-day interpretation of arterial pulse contours is largely based.

However, published material on direct human arterial pulse contours has been scant and fragmentary. There is a need for a more complete description of the contours observed clinically in normal and abnormal states. This paper is directed at that purpose, being a review of brachial artery pressure tracings from 250 patients taken during the past 4 years, with particular attention to the problem of exact diagnosis of aortic valve lesions.

Material and Methods

Direct brachial arterial pressure tracings were obtained in the following manner. A needle of 18 gage or larger was inserted percutaneously into the brachial artery in the antecubital space under local anesthesia, with the patient lying recumbent. The needle was connected by a rigid tubing up to 6 feet in length to an electromanometer (Sanborn) or strain-gage manometer (Statham P23A), and the tracing was recorded with appropriate amplifiers on a direct-writing oscillograph (Sanborn) at a paper speed of 25 mm. per second. Mean pressure was obtained by electric damping.

Artifacts and damping due to small clots in the needle, air bubbles or leaks in the connecting system, failure of the needle tip to lie free in the artery lumen, or improper angulation of the arm were eliminated by appropriate adjustments; an excessively damped normal tracing may be indistinguishable from the characteristic abnormal tracing of aortic stenosis. If there was not a free arterial flow from the needle, or if the pulse contour was not reproducible after repeated flushing of the recording system, then the validity of the tracing was questioned. Damped tracings were not included in the study.

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Dr. Abelmann is an Established Investigator of the American Heart Association.

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Exercise was performed on a bicycle ergometer in 81 cases.

Cardiac output in the normal subjects was determined by the dye dilution technic.

On each tracing, the systolic, diastolic, and mean pressures, the pulse pressure, the systolic upstroke duration, the duration of “systole,” the slope of the systolic upstroke, and the cycle length were measured. The duration of the systolic upstroke was taken as the time interval from the beginning of the systolic rise to the peak systolic pressure. “Systole” was defined as the time interval from the beginning of the systolic rise to the lowest point of the dicrotic notch, or to the point of abrupt change in rate of decline of the downstroke. The slope of the upstroke was measured as the rate of rise in pressure, in mm. Hg per 0.01 second over the steepest unbroken portion of the upstroke, usually the initial portion. The time measurements were usually accurate to the nearest 0.01 second, although some flattened or rounded pulse contours were difficult to measure accurately. The physiologic variation of cycle length, upstroke duration, and “systole” was usually at least 0.01 second in a continuous tracing over a short period of time, and greater than this when measured at different times.

From the total number of 250 patients, data from the pulse tracings of 106, constituted into 6 groups, are presented in detail. Patients not presented in detail included 56 with aortic valve lesions not verified by left heart catheterization, operation, or autopsy, 20 with mitral stenosis, 52 with alcoholism and cirrhosis of the liver, and 16 with miscellaneous conditions.

Group 1 consists of 40 “normal subjects,” of whom 6 were healthy house officers and 34 were convalescent patients from the wards of the Boston City Hospital. All were selected as control subjects for one or another cardiovascular or pulmonary study on the basis of showing no evidence of cardiovascular or pulmonary disease by clinical examination, electrocardiogram, chest roentgenogram or fluoroscopy. On the basis of age, this group is subdivided into group 1A, including 20 subjects ranging from 20 to 37 years of age, mean 27.1 years, and group 1B, including 20 subjects ranging from 38 to 84 years of age, mean 48.1 years.

Group 2 consists of 20 patients with aortic stenosis, proved severe, and represents a consecutive series of such patients from whom satisfactory brachial artery pressure tracings were available. All had typical clinical evidence of aortic stenosis with significant symptoms, and were under consideration for aortic valve surgery. Eight who had left heart catheterization had calculated valve areas in the range of 0.4 to 0.9 cm². Seventeen who were operated upon for aortic stenosis were estimated at the time of operation to have valve areas in the range of 0.4 to 0.8 cm² by direct transaortic palpation or, in 4 cases, by the passage of transventricular dilators. Fifteen of the operated patients showed systolic pressure gradients in excess of 50 mm. Hg across the aortic valve as determined by direct measurement at the time of operation. The 3 unoperated patients all came to autopsy and were found to have severe aortic stenosis, the valve orifice not admitting a fingertip, and judged to be in the range of 0.4 to 0.6 cm².

Four patients in this group showed evidence of some aortic insufficiency in the form of moderate or loud aortic diastolic murmurs and significant dilatation of the left ventricle, with diastolic blood pressures ranging from 45 to 83 mm. Hg (G. V., M. S., J. B., A. L.). It was thought in each case, after clinical study and palpation of the valve at operation, that predominant stenosis was present, with a degree of associated insufficiency impossible to measure with present methods, but evidently much less than “free” aortic insufficiency.

Group 3 consists of 13 patients with aortic stenosis, proved not severe. These patients were seen during the same period of time as those of group 2. All had typical clinical evidence of aortic stenosis and were under serious consideration for aortic valve operation, but were eventually judged to have no functionally significant narrowing of the valve. In 7, this conclusion was based on the finding at the time of operation of less than 10 mm. Hg pressure gradient across the aortic valve in systole. In 5 it was based on the finding at left heart catheterization of a left ventricular systolic pressure no higher than the brachial artery systolic pressure, and in 1 patient on the appearance of the aortic valve at autopsy, the valve being normal except for some sclerotic thickening of the leaflets, but associated with severe coronary artery disease.

Group 4 includes 20 patients with what was termed “myocardial failure.” These patients presented various degrees of congestive heart failure, attributed clinically to nonvalvular heart disease, usually hypertensive or coronary heart disease. None was thought to have high output failure.

Group 5 consists of 6 patients with the clinical picture of “free” aortic insufficiency, with seriously progressive signs of left ventricular failure, all subsequently shown at autopsy to have the expected pure aortic insufficiency without stenosis or other valvular disease.

Group 6 consists of 7 patients with severe rheumatic mitral insufficiency without stenosis. The absence of stenosis was proved at autopsy in 5, and was considered clinically evident in the other 2 (R. S., S. P.). All had marked cardiac enlargement, atrial fibrillation, and chronic congestive heart failure.

Results

Data from groups 1–6 are given in table 1, and estimates of the statistical significance of the differences are presented in table 2.
<table>
<thead>
<tr>
<th>Group</th>
<th>Age</th>
<th>Systolic</th>
<th>Diastolic</th>
<th>Mean</th>
<th>PP</th>
<th>Upstroke</th>
<th>Systole</th>
<th>R-R</th>
<th>Slope</th>
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<tr>
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<td></td>
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<td></td>
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<tr>
<td>Mean</td>
<td>37.6</td>
<td>125.0</td>
<td>72.3</td>
<td>94.1</td>
<td>52.7</td>
<td>0.112</td>
<td>0.283</td>
<td>0.818</td>
<td>8.94</td>
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<td>S.D.</td>
<td>13.6</td>
<td>34.1</td>
<td>7.55</td>
<td>2.8</td>
<td>9.4</td>
<td>0.039</td>
<td>0.033</td>
<td>0.128</td>
<td>4.39</td>
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<tr>
<td>C.V.</td>
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<td>27.2</td>
<td>10.4</td>
<td>2.98</td>
<td>17.8</td>
<td>35.3</td>
<td>11.6</td>
<td>15.6</td>
<td>49.1</td>
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<tr>
<td>Mean</td>
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<td>118.0</td>
<td>68.8</td>
<td>86.6</td>
<td>49.0</td>
<td>0.094</td>
<td>0.283</td>
<td>0.814</td>
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<td>5.3</td>
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<td>0.031</td>
<td>0.037</td>
<td>0.126</td>
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<tr>
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<td>7.7</td>
<td>7.8</td>
<td>15.5</td>
<td>32.0</td>
<td>13.1</td>
<td>15.4</td>
<td>48.5</td>
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</tr>
<tr>
<td>Mean</td>
<td>48.1</td>
<td>131.1</td>
<td>74.2</td>
<td>96.9</td>
<td>56.4</td>
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<td>0.033</td>
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<td>6.0</td>
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<td>12.7</td>
<td>18.1</td>
<td>31.2</td>
<td>11.7</td>
<td>16.9</td>
<td>64.5</td>
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<tr>
<td>Mean</td>
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<td>65.0</td>
<td>86.5</td>
<td>52.5</td>
<td>0.201</td>
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<td>19.7</td>
<td>0.03</td>
<td>0.08</td>
<td>0.03</td>
<td>1.70</td>
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<td>16.3</td>
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<td>10.1</td>
<td>37.5</td>
<td>14.9</td>
<td>24.8</td>
<td>3.9</td>
<td>33.9</td>
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<tr>
<td>Mean</td>
<td>46.8</td>
<td>140.0</td>
<td>74.2</td>
<td>97.2</td>
<td>65.8</td>
<td>0.171</td>
<td>0.307</td>
<td>0.801</td>
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<td>0.037</td>
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<td>26.8</td>
<td>14.9</td>
<td>17.6</td>
<td>52.7</td>
<td>10.9</td>
<td>12.2</td>
<td>17.3</td>
<td>45.2</td>
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<tr>
<td>Mean</td>
<td>59.7</td>
<td>156.2</td>
<td>82.6</td>
<td>106.2</td>
<td>73.7</td>
<td>0.111</td>
<td>0.331</td>
<td>0.846</td>
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<td>10.3</td>
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<td>0.140</td>
<td>0.197</td>
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<td>6.6</td>
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<td>5.5</td>
<td>10.2</td>
<td>11.5</td>
<td>48.2</td>
<td>23.3</td>
<td>69.1</td>
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<td>Mean</td>
<td>42.3</td>
<td>157.5</td>
<td>39.5</td>
<td>83.3</td>
<td>98.0</td>
<td>0.098</td>
<td>0.287</td>
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<tr>
<td>S.D.</td>
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<td>10.8</td>
<td>17.3</td>
<td>17.1</td>
<td>72.5</td>
<td>0.043</td>
<td>0.010</td>
<td>0.082</td>
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<td>40.9</td>
<td>6.9</td>
<td>43.4</td>
<td>20.5</td>
<td>45.4</td>
<td>3.47</td>
<td>11.0</td>
<td>62.6</td>
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<td>Group 6</td>
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<tr>
<td>Mean</td>
<td>35.3</td>
<td>120.9</td>
<td>67.1</td>
<td>85.7</td>
<td>53.7</td>
<td>0.079</td>
<td>0.230</td>
<td>0.774</td>
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<td>21.2</td>
<td>18.5</td>
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<td>100.1</td>
<td>16.9</td>
<td>17.6</td>
<td>81.8</td>
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</table>

* In this and other tables, BP = systolic and diastolic pressure in mm. Hg; mean pressure is in mm. Hg; PP = pulse pressure in mm. Hg; upstroke = duration of the systolic upstroke in seconds; systole = duration from the beginning of the systolic rise to the dicrotic notch, in seconds; R-R = cycle length, in seconds; slope = steepest slope of the upstroke, in mm. Hg per 0.01 second; S.D. = standard deviation; C.V. = coefficient of variation, in per cent.

**Table 2.**—Significance of the Differences of the Mean Values

<table>
<thead>
<tr>
<th>Groups</th>
<th>Age</th>
<th>Systolic</th>
<th>Diastolic</th>
<th>Mean</th>
<th>PP</th>
<th>Upstroke</th>
<th>Systole</th>
<th>R-R</th>
<th>Slope</th>
</tr>
</thead>
<tbody>
<tr>
<td>1A-1B</td>
<td>7.7</td>
<td>3.3</td>
<td>2.24</td>
<td>3.3</td>
<td>2.68</td>
<td>3.1</td>
<td>0.14</td>
<td>0.18</td>
<td>0.28</td>
</tr>
<tr>
<td>1 -1B</td>
<td>3.8</td>
<td>1.0</td>
<td>0.8</td>
<td>1.04</td>
<td>1.36</td>
<td>1.64</td>
<td>0.08</td>
<td>0.16</td>
<td>0.04</td>
</tr>
<tr>
<td>1-2</td>
<td>1.5</td>
<td>0.72</td>
<td>1.1</td>
<td>1.2</td>
<td>0.01</td>
<td>9.7</td>
<td>2.3</td>
<td>0.50</td>
<td>2.18</td>
</tr>
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<td>1B-2</td>
<td>0.56</td>
<td>2.03</td>
<td>3.34</td>
<td>3.18</td>
<td>0.75</td>
<td>6.3</td>
<td>1.1</td>
<td>5.16</td>
<td>1.30</td>
</tr>
<tr>
<td>1B-3</td>
<td>0.45</td>
<td>0.82</td>
<td>0.00</td>
<td>0.06</td>
<td>0.99</td>
<td>3.7</td>
<td>0.41</td>
<td>0.96</td>
<td>0.76</td>
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<td>1B-4</td>
<td>8.7</td>
<td>6.05</td>
<td>3.37</td>
<td>3.01</td>
<td>6.1</td>
<td>1.95</td>
<td>0.73</td>
<td>1.05</td>
<td>0.50</td>
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<td>2-3</td>
<td>0.93</td>
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<td>1.4</td>
<td>0.78</td>
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<td>1.03</td>
<td>0.64</td>
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<td>3-4</td>
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<td>1.6</td>
<td>1.86</td>
<td>0.81</td>
<td>5.0</td>
<td>0.75</td>
<td>0.67</td>
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<td>2-4</td>
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<td>2.5</td>
<td>4.7</td>
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<td>7.7</td>
<td>0.27</td>
<td>1.09</td>
<td>7.90</td>
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</table>

This table presents the ratios of the observed differences between the means of the 2 groups for each measurement to the standard error of the difference between the means. If the observed difference is more than twice the standard error of the means, then there is a less than 5 per cent probability that the 2 groups represent random samples from the same population, i.e., \( p = <0.05 \). If the ratio is 2.50, then \( p = <0.0125 \), and if the ratio is 3.00, then \( p = <0.003 \).
Normal Subjects (group 1, fig. 1)

The systolic upstroke duration in 40 normal subjects ranged from 0.06 to 0.20 second, with a mean value of 0.11 second. The upstroke duration was the most variable of the measurements. There was considerable variation in the details of the pulse forms (fig. 1); anacrotic (J. Sh., A. My.) and bisferiens (R. Mo.) forms were encountered. Comparison of the mean values of groups 1A and 1B showed that, as a group, the older normal subjects had significantly higher systolic, diastolic, and mean pressures, wider pulse pressures, and longer upstroke durations than the younger normal subjects. The slope of the upstroke was a highly variable measurement, and did not differ significantly between the younger and older groups of normal subjects. Thus, the

Fig. 1. A. Brachial arterial pulse forms of 20 normal subjects, ages 20 to 37 years (group 1A). B. Brachial arterial pulse forms of 20 normal subjects, age 38 to 84 years (group 1B). In this and other illustrations, the vertical scales in mm.Hg are not identical. All illustrations have an identical horizontal time scale, each heavy vertical line signifying 0.20 second.
highly significant difference in upstroke duration was predominantly due to the tendency of the pulse contours of older patients to show a gradually rounded peak instead of a sharp early peak as was characteristic of the pulse contour of almost all younger normal subjects.

The length of the cardiac cycle was not related to the upstroke duration in either group of normal subjects.

**Aortic Stenosis, Severe (group 2, fig. 2A)**

The upstroke duration in these patients ranged from 0.14 to 0.24 second, with a mean value of 0.20 second. The range overlapped that of the normal subjects to a mild degree (fig. 3), but the difference between the means was highly significant ($p = <0.01$). The patients with aortic stenosis also showed significantly lower systolic, diastolic, and mean pressures, and slower heart rates than the normal subjects of comparable age (group 1B), although the total group of normal subjects taken together differed significantly from the aortic stenosis group only in the duration of the systolic upstroke. The mean pulse pressure in the aortic stenosis group was smaller than that of group 1B, but the difference was not statistically significant. This finding documents a fact now widely recognized clinically; that a narrow pulse pressure is a characteristic feature in only a small minority of cases of severe aortic stenosis. The pulse pressure cannot be relied

![Fig. 2. A. Brachial arterial pulse forms from 20 patients with proved severe aortic stenosis (group 2). B. Brachial arterial pulse forms from 12 patients with aortic stenosis proved not severe (group 3).](http://circ.ahajournals.org/)


Fig. 3. Frequency distribution of the duration of the systolic upstroke (abscissa, seconds) in groups 1 to 4 (ordinate, number of patients).

upon as a guide to the severity of aortic stenosis.

The significant prolongation of "systole" and the more gradual slope of the upstroke in aortic stenosis as compared with the total group of normal subjects \( p < 0.05 \) are more likely due to factors related to age than to factors related to aortic stenosis, because there was no significant difference in these measurements between groups 2 and 1B, which are of comparable age. The prolonged upstroke, then, appeared to be the most characteristic abnormality of the arterial pulse form in aortic stenosis.

The abnormal pulse form that appeared to be most typical was that with an upstroke duration of 0.20 second or longer, and a distinct notch rather low on the anacrotic limb (cf. C. R., M. M., L. L.). This pattern was not seen in any patient without aortic stenosis. On the other hand, in some instances of severe isolated aortic stenosis there was no anacrotic notch.

The Valsalva maneuver often exaggerated the anacrotic notch during the initial cycles of the first phase, and occasionally during the overshoot, or sometimes brought out the appearance of an anacrotic notch where there had been little or none in the resting tracing (fig. 4E). One normal subject showed some degree of this change, however, and it has not always been present in severe aortic stenosis.

**Aortic Stenosis, Not Severe (group 3, fig. 2B)**

In group 3 the pulse forms resembled closely those of group 2. The systolic upstroke duration ranged from 0.12 to 0.20 second, mean 0.17 second. There was considerable overlap with normal subjects, and with the severe aortic stenosis group, sufficient to render diagnosis of an individual case unreliable on the basis of this measurement alone (fig. 3). However, the mean upstroke duration was significantly longer than that of the normal group and significantly shorter than that of the aortic stenosis group. None of the other measurements differed significantly from those of normal subjects of comparable age, nor from those of cases of severe aortic stenosis. These results support the idea that aortic stenosis not only leads to prolongation of the systolic upstroke, but that such prolongation actually is related in degree to the degree of valve narrowing, as suggested by experimental observations, but not yet proved in man.

In both groups 2 and 3, there was evidence of a direct relation between stroke volume and upstroke duration in individual patients. This was evident in plotting cycle length against duration of upstroke in patients with atrial fibrillation, and also in patients with frequent ventricular extrasystoles, in which case the large beat following a compensatory pause tended to show a longer upstroke and more prominent anacrotic notch than the resting pulse form.

**Myocardial Failure (group 4)**

This group showed a mean age of 59.7 years, significantly older than the other groups. Compared with group 1B, mean age 48.1 years, these patients with "myocardial failure" showed significantly higher systolic, diastolic, and mean pressures, and wider pulse pressures. The group included 5 patients with definite hypertension, greater than 200 mm. Hg systolic,
STUDY OF BRACHIAL ARTERIAL PULSE FORM

Fig. 4. A. Brachial arterial pulse forms from 3 patients (group 5) with severe pure aortic insufficiency. B. Brachial arterial pulse forms from 2 patients (group 6) with severe mitral insufficiency, showing the "small collapsing pulse." C. Rest and exercise arterial pulse forms in 2 normal subjects. J. W. at rest 114/68, mean 84 mm. Hg, cardiac output 7.7 L./min., stroke volume 103 ml., and peripheral resistance 870 dynes-sec-cm.\(^{-5}\); during exercise, arterial pressure 134/72, mean 92 mm. Hg, cardiac output 10.4 L./min., stroke volume 101 ml. and peripheral resistance 710 dynes-sec-cm.\(^{-5}\)

There is relatively little change in the pulse contour. C. S. at rest 116/66, mean 76 mm. Hg, cardiac output 6.5 L./min., stroke volume 87 ml., peripheral resistance 930 dynes-sec-cm.\(^{-5}\), and exercise arterial pressure 130/72, mean 100 mm. Hg, cardiac output 10.4 L./min., stroke volume 122 ml., peripheral resistance 645 dynes-sec-cm.\(^{-5}\). There is a characteristic change in the pulse contour. D. The arterial pulse form in other hemodynamic abnormalities. A. O. is a 47-year-old man with hyperthyroidism and atrial fibrillation, arterial pressure 205/75, rate 128, upstroke 0.05 second.

B. A. is a patient with fever, anemia, and severe liver disease, arterial pressure 92/50, rate 130, cardiac index 4.4 L./min./M.\(^2\), stroke volume 64 ml. The alteration of the pulse contour is apparently related to peripheral vasodilation without a striking increase in cardiac output.

105 diastolic, and 135 mean. The upstroke duration in group 4 ranged from 0.04 to 0.24 second, mean 0.11 second. This mean value is 0.02 second shorter than the mean of the older group of normal subjects, but because of the wide variability of the myocardial failure group, this difference does not quite reach the 5 per cent level of significance. Uncontrolled factors related to age, hypertension, functional mitral insufficiency, and arterial disease may be involved in this difference and may also be involved in the wide variability within the myocardial failure group. These data, therefore, do not show a significantly shorter upstroke duration in "myocardial failure" as herein defined, than in normal subjects. Because of the finding of a very brief upstroke duration in several patients with severe left ventricular failure, pulsus alternans, and gallop rhythm, however, it is suspected that further studies of larger and better controlled groups might reveal a significant difference.

Aortic Insufficiency (group 5, fig. 4A).

The pulse contours of the patients of group 5 showed some uniformity in the occurrence of abrupt upstrokes, bifid systolic peaks, and small or absent dicrotic waves, but none of these characteristics was constant, and all have been seen in tracings from patients without aortic insufficiency. The procedure was useful as an accurate measurement of the diastolic
pressure, which was not lower than 30 mm. Hg in any of these patients, although sounds were heard down to zero during sphygmomanometric determination of the blood pressure.

Mitral Stenosis and Insufficiency

Mitral stenosis has been associated with no particular features of the arterial pulse contour distinguishable from the normal, though a small pulse pressure with a flattened systolic peak is commonly seen.

Some patients with marked mitral insufficiency have shown the “small collapsing pulse” described by others. Group 6 (fig. 4B) includes selected patients showing some degree of this characteristic of the pulse. Their arterial pulse forms show rapid upstrokes, rapid downstrokes, short “systoles,” low dicrotic pressures, and prominent dicrotic waves.

Exercise

During exercise, there was a distinct tendency for the systolic and mean pressures to rise somewhat, and for the dicrotic wave to become lower and flatter (fig. 4C). The systolic upstroke duration changed very little unless it was prolonged initially, in which case it sometimes became much shorter. Flat-topped curves with apparently long upstrokes were sometimes revealed as quite normal during exercise. These changes were not constant and did not appear to be correlated with the degree of change in cardiac output or peripheral resistance. Many patients showed identical pulse forms during rest and exercise in spite of marked increases in cardiac output. Several patients with elevated cardiac output in association with hyperthyroidism, anemia, or liver disease (fig. 4D) showed pulse forms at rest similar to those of other patients during exercise and similar to those of patients with aortic insufficiency.

DISCUSSION

The brachial arterial pressure pulse form is best considered as representing the central aortic pressure pulse, altered in form in the course of transmission to the periphery. This alteration may take place by way of summation of one or more standing waves with the transmitted central aortic pulse. Thus, any changes observed in the brachial arterial pressure pulse may be the result of changes in the central pressure pulse, or of changes in the transmission of the central pulse to the periphery, or both.

The central aortic pressure pulse is a function of the rate and pattern of ventricular ejection, the stroke volume, the distensibility of the aortic chamber, the peripheral vascular resistance, and the viscosity of the blood. The pulse form may be modified by physiologic or pathologic changes in any of these parameters.

Certain pathologic lesions not only affect the central pressure pulse, but also the mode of transmission of the central pulse to the periphery. Dow’s experiments showed that the central aortic pulse in aortic stenosis is transmitted to the periphery more faithfully than the normal central pulse. Alexander has found evidence that the aortic standing wave may be reduced by aortic insufficiency, and Gupta and Wiggers have published similar findings in experimental coarctation of the aorta. Recent studies of central and peripheral pulses in man have suggested that the pulse in aortic stenosis is more faithfully transmitted to the periphery. This was also found in a patient with coarctation of the aorta. However, comparisons of the central and peripheral pulse contours in man have so far been few in number.

In view of the multiplicity of central and peripheral factors involved in the genesis of the peripheral pressure pulse, it is not surprising that the brachial arterial tracings encountered in normal subjects vary considerably and overlap to some extent the tracings associated with disorders of the circulation. Nor is the lack of correlation between the duration of upstroke or position of the anacrotic notch with the severity of aortic stenosis in individual patients difficult to accept. On the other hand, the findings presented here do not preclude the possibility that the central aortic pressure pulse may more closely reflect such hemodynamic lesions as aortic stenosis.

SUMMARY

Direct brachial arterial pressure tracings from 250 patients have been analyzed in relation to their potential clinical value.

In 40 “normal subjects” the duration of the
systolic upstroke ranged from 0.06 to 0.20 second, mean 0.11 ± 0.04 second, and the contour of the pulse form showed greater variation than heretofore reported for the human adult. Notching was present on the anacrotic limb in 2 cases. There was a significant increase in the upstroke duration with age.

In 19 of 20 patients with proved severe aortic stenosis characteristic abnormalities of the pressure pulse were evident. The duration of the systolic upstroke was prolonged to a mean of 0.20 ± 0.03 second in this group, and an anacrotic notch was present in 15 cases. While the mean duration of the systolic upstroke was significantly longer than in the normal group, there was some overlap.

The arterial pressure pulses of 13 patients with clinical aortic stenosis proved to be physiologically mild or insignificant resembled those of patients with proved severe stenosis, although the mean upstroke of the group was significantly shorter in duration.

Six patients with proved severe pure aortic insufficiency tended to show characteristic pressure pulses with rapid upstrokes, bifid systolic peaks, and low or flat dicrotic waves. None of these features may be considered diagnostic, since they are also seen in hyperthyroidism, anemia, and other high-output states.

Pulse pressure tracings from a group of 20 patients with decompensated nonvalvular heart disease showed wide variation and did not differ significantly from those obtained from normal subjects, but there was some evidence that both the systolic upstroke and "systole" may be shortened in myocardial failure.

Pulse contours in marked mitral insufficiency may be of the "small collapsing" type.

It is concluded that, because of the many factors that affect the peripheral pressure pulse, such tracings are difficult to interpret as a clinical diagnostic test. They are of value in determining blood pressure accurately. The brachial arterial pulse contour may serve to confirm a clinical diagnosis of aortic stenosis, but is not diagnostic of aortic stenosis and in any individual patient yields no information as to the severity of the lesion. A normal brachial arterial pulse form in a patient suspected of aortic stenosis speaks against physiologically significant stenosis, but does not rule it out. Further studies of the central aortic pulse form and its alteration in transmission to the periphery are indicated, in order to define further the clinical usefulness of arterial pressure tracings.

**Summario in Interlingua**

Registrationes directe del pression arterial brachial ab 250 patientes eseva analysate ab le puncto di vista de lor valor potential pro objectivos clinic.

In 40 subjectos "normal," le duration del ascendita systolic variava ab 0,06 a 0,20 secundas (valor medie = 0,11 ± 0,04 secundas), e le contorno del forma del pulso monstrava plus grande variationes que lo que ha previamente essite reportate pro le adulto human. Indentation eseva presente in le membro anacrotic in 2 casos. Esseva constatate un augmento significative del duration del ascendita con le augmento del etate del subjectos.

In 19 inter 20 patientes con demonstrate sever stenosis aortic, anormalitates characteristic del pulso de pression eseva evident. Le duration del ascendita systolic eseva pro-longate a un valor medie de 0,20 ± 0,03 secundas in iste grupo, e un indentation anacrotic eseva presente in 15 casos. Durante le valor medie del duration del ascendita systolic eseva significativamente plus longe que in le grupo de subjectos normal, le valores del duo series monstrava un corte region de coincidentia.

Le pulsos de pression arterial in le 13 patientes in qui physiologicamente leve o insignificantive grados clinic de stenosis aortic eseva demonstrate resimilava illos de patientes con demonstrate sever stenosis, ben que le ascendita medie del grupo eseva significativamente plus curte.

Sex patientes con demonstrate sever insufficiencia aortic pur tendeva a exhibir caracteristic pulso de pression con ascenditas rapide, bifide culmines systolic, e pauco elevate o plan undas dicrotic. Nulle de iste aspectos pote esser considerate como diagnostic, proque illos omnes occurre etiam in hyperthyroidism, anemia, e altere conditiones de rendimento elevate.
Registros del presión pulsátil ab un grupo de 20 pacientes con discompensate morbo cardiac non-valvular exhibita pronounced variaciones e non diferiva significativamente ab le registraziones obtenite ab subjectos normal, sed il pareva haber certe indicationes que tanto le ascendita sístolica como etiam le sístole pote esser accurtate in disfallimento myocardial.

Contornos del pulso in casos de marcata insuficientia mitral pote esser del "parve typo colaborante."

Il es concluside a causa del numero factors aufficienle le pulso de pression peripheric, tal registraziones es difficile a interpretar como test diagnostic clinic. Illos es de valor in le determination precisa del pression de sanguine. Le contorno del pulso arterial brachial pote servir a confirmar un diagnose clinic de stenosis aortic, sed illo non es diagnostic pro stenosis aortic, e in le paciente individual illo non forni informaciones in re le severture del lesion. Un forma normal del pulso arterial brachial in un paciente suspecte de haber stenosis aortic argue contra sed non exclude le possibilitade de un physiology significative stenosis. Studios additional del forma del pulso aortic central e de su alteration in le transmission al peripheria es indicate pro definir plus clarmente le utilitate clinic de registraziones de pression arterial.

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REFERENCES

A Clinical Study of the Brachial Arterial Pulse Form: With Special Reference to the Diagnosis of Aortic Valvular Disease

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