

## ASSESSMENT OF AORTIC STENOSIS FROM THE EXTERNAL CAROTID PULSE WAVE

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The introduction of aortic valve surgery has made the assessment of aortic valve disease increasingly important, and attention has been focused on the arterial pulse as an aid to diagnosis.

Experimental studies on aortic stenosis were carried out by De Heer in 1912 and later by Allan (1925) and by Katz, Ralli, and Cheer (1928), who showed that an increasing systolic pressure gradient across the aortic valve occurred proportional to the degree of obstruction, and that there was an associated alteration of the pulse wave on the distal side of the stenosis. Katz and Feil (1925) showed that alterations in the indirect subclavian pulse consisted chiefly of prolongation of the ejection time and an anacrotic appearance in patients with aortic stenosis.

Studies of the direct arterial pressure pulse, usually from the brachial artery, have been carried out in recent years and have proved disappointing in assessing the severity of the aortic stenosis, largely because of the overlap between the normal and the abnormal (Goldberg, Bakst, and Bailey, 1954; Gorlin and Case, 1956; Fleming and Gibson, 1957; Hancock and Abelman, 1957; Wood, 1958; Robinson, 1963).

Attempts have also been made to assess the degree of aortic stenosis from the indirect carotid pulse wave (Duchosal *et al.*, 1956; Donoso *et al.*, 1956; Eggink, Hartog, and Kuipers, 1958; Daoud, Reppert, and Butterworth, 1959; Benchimol, Dimond, and Shen, 1960; Robinson 1963), and it is probable that this wave form reflects central events more clearly than the direct brachial arterial pulse. In the majority of these papers an objective assessment of the degree of aortic stenosis was not available, although the diagnosis of aortic stenosis could frequently be made from an analysis of the carotid pulse.

The main purpose of this paper is an attempt to determine whether the presence and the degree of aortic stenosis can be assessed from an externally recorded carotid pulse wave. The carotid pulse has been recorded and analysed in a group of patients with aortic stenosis where an objective assessment of the severity of the obstruction was available.

### SUBJECTS AND METHOD

All recordings were made with a four-channel Cambridge photographic instrument. The carotid pulse wave was recorded with a piezo-electric microphone connected via a short piece of rigid plastic tubing to a funnel-shaped cup applicator. The frequency response of this crystal microphone is linear from 1 to about 800 cycles per second. Simultaneous heart sounds were recorded at medium and low frequency together with electrocardiogram lead II. Records were taken at a paper speed of 50 mm. per second with respiration halted in partial expiration.

As controls 35 normal subjects aged from 17 to 50 years have been compared with 47 patients where pure or dominant aortic valvar stenosis was present. Patients with more than trivial regurgitation were excluded from the study, and all 47 patients studied had some form of objective assessment of their aortic valvular disease. The peak systolic gradient across the aortic valve was measured in all 47 patients, by left heart catheterization in 37 and at operation in 10 patients: 36 had an operation and were assessed by the surgeon, and 13 came to autopsy.

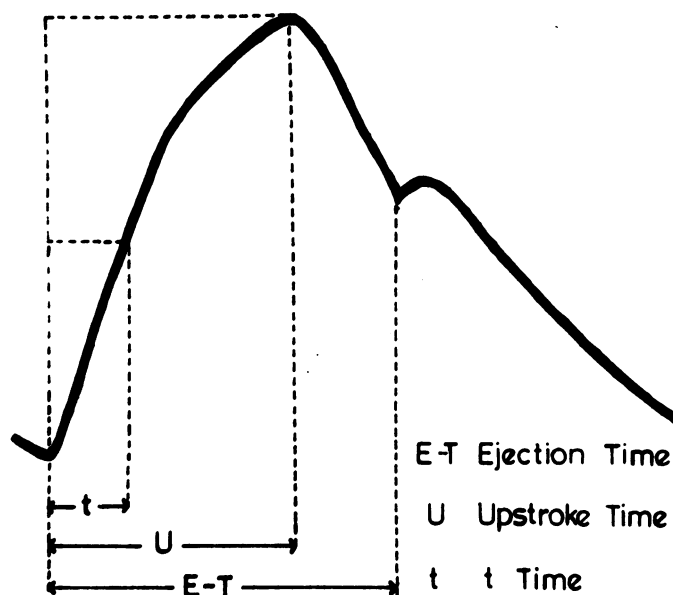


FIG. 1.—Diagram showing the measurements made on the carotid pulse. E-T ejection time; U upstroke time; t time.

The degree of aortic stenosis was graded as mild, moderate, or severe on the basis of one or more observations using the following arbitrary criteria.

**Mild Stenosis.** This was present in 8 patients, who had a systolic gradient of less than 45 mm. Hg, or a cross-sectional aortic valve area of more than 1 cm.<sup>2</sup>, calculated or measured.

**Moderate Stenosis.** This was present in 17 patients, who had a systolic gradient from 45 to 75 mm. Hg, or a valve area of 0.75 to 1.0 cm.<sup>2</sup>

**Severe Stenosis.** This was present in 22 patients, who had a systolic gradient above 75 mm. Hg, or a valve area of less than 0.75 cm.<sup>2</sup>

We have also reviewed 21 patients with combined aortic and mitral stenosis. All had left heart catheter studies and also an operation, thus confirming the diagnosis. Two patients came to autopsy. Lastly, we have compared the findings with 30 patients suffering from severe mitral stenosis, all of whom had an operation and hence a surgical assessment of valve size.

**Analysis of the External Carotid Pulse Wave.** The measurements made from the carotid pulse are shown diagrammatically in Fig. 1, and are as follows.

1. **The Systolic Ejection Time (E-T).** This was measured from the initial rise of the carotid upstroke to the catacrotic notch. Where the notch was not clearly defined the end of the ejection was taken as the position of the aortic component of the second sound with the addition of 0.03 second.

2. **The Systolic Upstroke Time (u).** This was measured from the beginning of the carotid upstroke to the peak of the pulse curve, that is to the highest point or maximal excursion of the curve.

3. **The 't' Time.** This is the time taken to achieve half the maximum height of the carotid pulse curve, measuring from the onset of the carotid upstroke (Duchosal *et al.*, 1956).

4. **The Ascending Index (u/E-T Ratio).** The ratio of the upstroke time (u) to the systolic ejection time (E-T) was simply calculated from the absolute values of u and E-T as described by Eggink *et al.* (1958), who designated this value as the 'ascending index'.

All these values were an average of measurements made from at least four cardiac cycles, and were corrected for heart rate using the Bazett formula, dividing the absolute values by the square root of the preceding cycle length (R-R interval).

## RESULTS

The four main features analysed in each of the four groups are shown in Table I. The mean figures and range are given for each measurement.

TABLE I

	Controls (35 subjects)	Isolated aortic stenosis (47 patients)	Combined mitral and aortic stenosis (21 patients)	Severe mitral stenosis (30 patients)
<i>Systolic ejection time</i>				
E-T { Range .. ..	0.26-0.34	0.30-0.48	0.29-0.43	0.26-0.346
Mean .. .. .	0.309	0.365	0.347	0.302
<i>Upstroke time</i>				
u { Range .. ..	0.05-0.12	0.08-0.33	0.10-0.28	0.05-0.14
Mean .. .. .	0.08	0.193	0.21	0.08
<i>'t' time</i>				
Range .. .. .	0.02-0.046	0.025-0.11	0.034-0.10	0.024-0.04
Mean .. .. .	0.032	0.059	0.056	0.035
<i>Ascending index</i>				
U/E-T ratio { Range ..	0.20-0.36	0.24-0.79	0.30-0.77	0.21-0.46
Mean .. .. .	0.26	0.53	0.60	0.27

All times are given in seconds.

TABLE II

Degree of aortic stenosis	Ejection time	Upstroke time	't' time	Ascending index (u/E-T ratio)
Mild (8 patients)	0.34	0.12	0.041	0.33
Moderate (17 patients)	0.34	0.18	0.054	0.55
Severe (22 patients)	0.38	0.22	0.066	0.60
All degrees (47 patients)	0.365	0.19	0.059	0.53

#### *Aortic Stenosis*

In general the carotid tracings showed a slow ascending limb, often with a prominent anacrotic notch or shoulder, a delayed peak with superimposed systolic vibrations, and sometimes a systolic plateau. As a rule the catacrotic notch was distinctly seen.

The systolic ejection time, the upstroke time, the 't' time, and the ascending index were each compared with the peak systolic gradient across the aortic valve.

*The Systolic Ejection Time.* This was never less than 0.3 sec., even when the aortic stenosis was graded as mild. The mean for the whole series was 0.365 sec. (range 0.30 to 0.48 sec.). This was greater than in the normal controls where the mean was 0.309 sec. (range 0.26 to 0.34 sec.).

When the patients were graded as mild, moderate, and severe, there was a greater average systolic ejection time in the severe group, viz. 0.38 sec. compared with 0.34 sec. in the moderate group (Table II). From the graph (Fig. 2), it can be seen that all the patients with an ejection time of 0.36 sec. or more had a peak systolic gradient of at least 45 mm. Hg, although there were several patients with a smaller ejection period and a large gradient. Therefore, a normal ejection time did

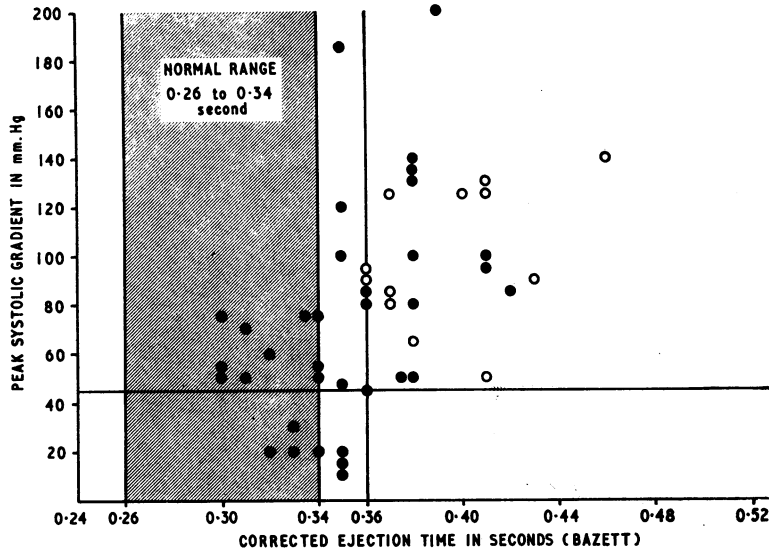


FIG. 2.—The ejection time compared with the peak systolic gradient across the aortic valve. The shaded area in this and Fig. 3, 4, 5, 7, and 8 represents the limits of normal. ● A.S. without left ventricular failure. ○ A.S. with left ventricular failure.

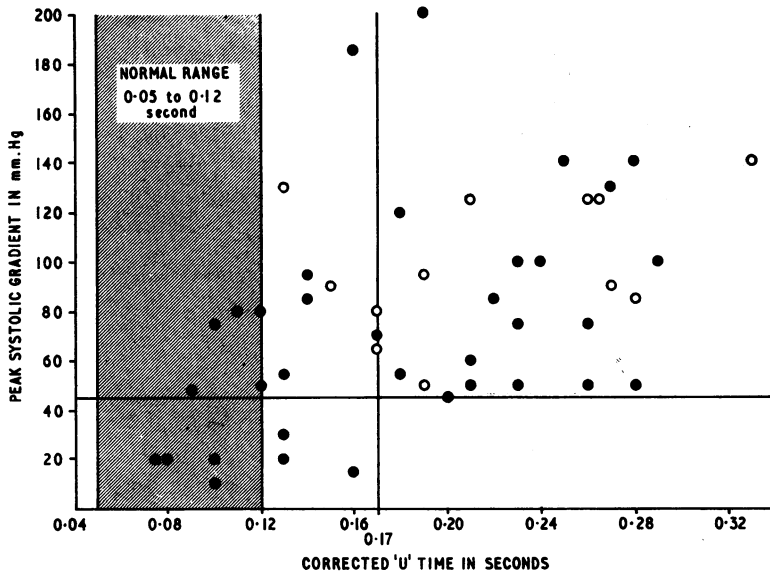


FIG. 3.—The 'u' time compared with the peak systolic gradient. ● A.S. without left ventricular failure. ○ A.S. with left ventricular failure.

not rule out severe aortic stenosis, but a corrected systolic ejection time of 0.36 sec. or more indicated a significant degree of aortic obstruction. Of 33 patients who had an ejection time greater than normal, 26 had an ejection time of 0.36 sec. or above.

*The Systolic Upstroke Time ('u').* Every patient with aortic stenosis had an upstroke time of 0.08 sec. or more with a mean of 0.19 sec. (range 0.08 to 0.33 sec.). The normal controls had a mean upstroke time of 0.08 sec. (range 0.05 to 0.12 sec.). Mild, moderate, and severe aortic stenosis showed average upstroke times of 0.12, 0.18, and 0.22 sec. respectively (Table II).

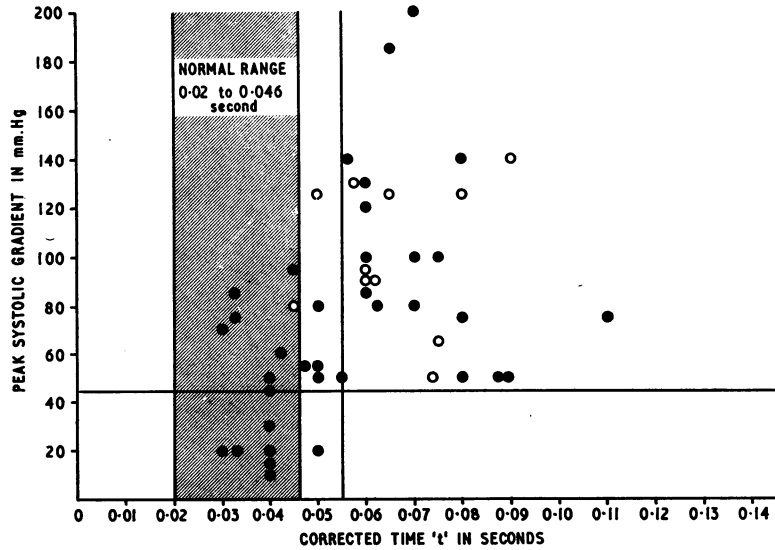


FIG. 4.—The 't' time compared with the peak systolic gradient. ● A.S. without left ventricular failure. ○ A.S. with left ventricular failure.

Fig. 3 shows upstroke time plotted against the peak systolic gradient. With an upstroke time of 0.17 sec. or more the systolic gradient was invariably greater than 45 mm. Hg, although there were several patients with large gradients and a shorter upstroke time.

An upstroke time greater than normal was found in 38 patients, and in 29 it was 0.17 sec. or above. A normal upstroke time did not exclude severe aortic stenosis, but an upstroke time of 0.17 sec. or more was always associated with significant aortic obstruction.

*The 't' Time.* This ranged from 0.025 to 0.11 sec. with a mean value of 0.059 sec. (Table I).

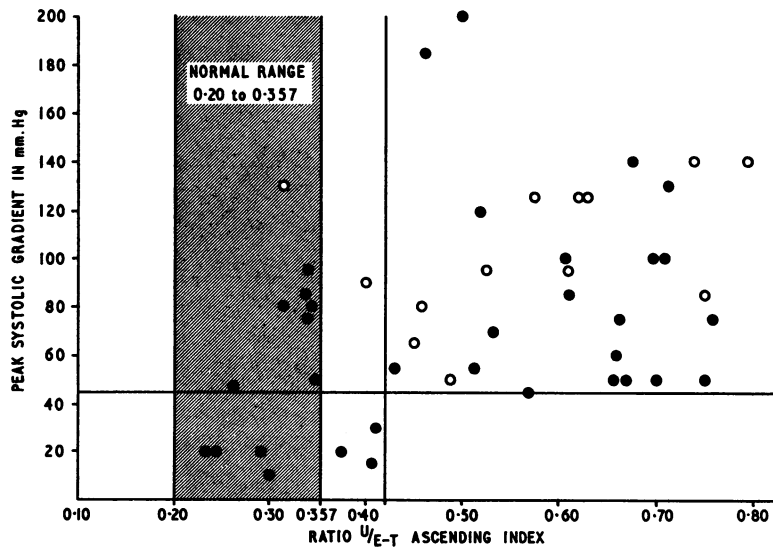


FIG. 5.—The ascending index,  $U/E-T$ , compared with the peak systolic gradient. ● A.S. without left ventricular failure. ○ A.S. with left ventricular failure.

The normal controls had a mean value of 0.032 sec. (range 0.02 to 0.046 sec.). The mean values for mild, moderate, and severe aortic stenosis were 0.041, 0.054, and 0.066 sec. respectively (Table II). All patients with a 't' time of 0.055 sec. or more had a gradient of at least 45 mm. Hg (Fig. 4). A 't' time above normal was recorded in 33 patients, and in 27 patients it was 0.055 sec. or greater. A normal 't' time did not exclude severe aortic stenosis, but significant obstruction was invariably present with a 't' time of 0.055 or above.

*The Ascending Index (u/E-T Ratio).* This ratio ranged from 0.24 to 0.79 with a mean value of 0.53. The mean for the normal controls was 0.26 (range 0.20 to 0.36). Mild, moderate, and severe aortic stenosis showed mean values of 0.33, 0.55, and 0.60 respectively (Tables I and II). A ratio of 0.42 or above was associated with a gradient of at least 45 mm. Hg (Fig. 5). Of 35 patients with an index greater than normal 31 had an index of 0.42 or more. A normal ascending index did not rule out serious aortic obstruction but a value of 0.42 or more was always associated with significant aortic stenosis.

Increasing severity of aortic stenosis was associated with a larger number of abnormal measurements on the indirect carotid pulse tracing (Table III). For each of the four parameters assessed

TABLE III

Parameter	Above normal upper limit		At or above critical level	
	Number	Percentage	Number	Percentage
Ejection time	33	70	26	55
Upstroke time	38	81	29	61.5
't' time	33	70	27	57
Ascending index	35	75	31	66

TABLE IV

Number of positive factors	Mild aortic stenosis (8 patients)		Moderate aortic stenosis (17 patients)		Severe aortic stenosis (22 patients)	
	No.	Percentage	No.	Percentage	No.	Percentage
None .. .. .	7	87	2	11.5	—	—
One .. .. .	1	13	3	18	2	9
Two .. .. .	—	—	5	29	3	13.5
Three .. .. .	—	—	4	23.5	4	18.5
Four .. .. .	—	—	3	18	13	59

there was a 'critical' level above which the peak systolic gradient was 45 mm. Hg or more. In Table III the percentage of patients with measurements above the normal upper limit is compared with that of patients where the measurements are at or above the 'critical' level for each parameter.

The upstroke time was above the upper normal limit in more patients (81%) than any of the other three measurements. When the 'critical' levels were considered the largest number of positive results (66%) was shown by the ascending index.

The four parameters have been combined to produce a numerical index. A factor was counted as positive if above the 'critical' level defined above. Each patient has been assessed for the number of positive factors with a maximum score of 4, and Table IV shows the number of positive measurements in relation to the severity of the aortic stenosis. The score increases as the aortic stenosis becomes more severe. This is shown in a different way in Fig. 6 where the peak systolic gradient is plotted against the score for each patient. With severe aortic stenosis, 59 per cent had a score of 4, and a further 18.5 per cent had a score of 3, that is 77.5 per cent of patients with severe

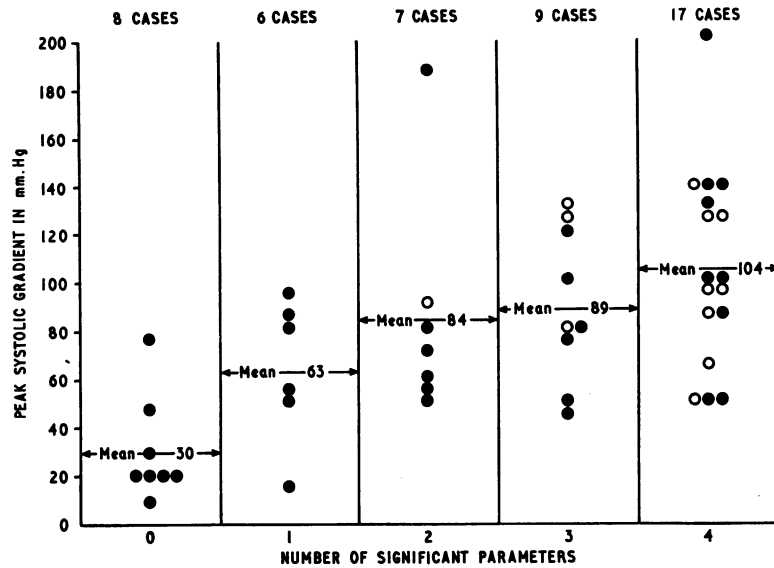


FIG. 6.—Comparison of number of positive findings with the peak systolic gradient.  
 ● A.S. without left ventricular failure. ○ A.S. with left ventricular failure.

aortic stenosis had at least three positive measurements. However, with four positive measurements the gradient may be no more than 50 mm. Hg in the individual case although the average gradient in this group was more than 100 mm. Hg, while a score of 2 was always associated with at least moderate stenosis and the gradient across the valve was never less than 45 mm. Hg. Occasionally one of the four measurements can be significantly increased in the presence of mild aortic stenosis, and thus one positive factor does not establish the diagnosis of significant stenosis with certainty. Two patients with moderate stenosis had relatively normal carotid tracings, and a significant lesion cannot be excluded by the absence of any positive measurements.

#### *Combined Aortic Stenosis and Mitral Stenosis*

External carotid pulse wave measurements on 21 patients with combined aortic and mitral stenosis and also on 30 patients with severe mitral stenosis are shown in Table I. The systolic ejection time was above normal in 9 patients and exceeded the critical level defined for isolated aortic stenosis in only 4 patients, all of whom had systolic gradients of more than 45 mm. Hg (Fig. 7). The upstroke time (u) was above normal in 19 and exceeded the critical level in 16 patients. However, in 4 of the latter patients the peak systolic gradient was less than 45 mm. Hg (Fig. 8). In addition, there were 2 false positive results out of 9 patients with a critically raised 't' time and 4 false positive findings in 17 patients with an ascending index above the critical level.

Although the average value for each of the four parameters was increased as compared with normal, the criteria used for assessing lone aortic valvar stenosis were not valid in the presence of combined mitral and aortic stenosis. The systolic ejection time was the only helpful measurement and when this was above the critical level as defined for lone aortic stenosis, then the aortic stenosis in the combined cases was at least of moderate severity.

Aortic stenosis was combined with mild mitral stenosis in 7 patients and the average ejection time was 0.37 sec.; with moderate mitral stenosis in 4 patients whose average ejection time was 0.36 sec.; and with severe mitral stenosis in 10 patients with an average ejection time of 0.32 sec. It appears, therefore, that the systolic ejection time is diminished in the presence of severe mitral

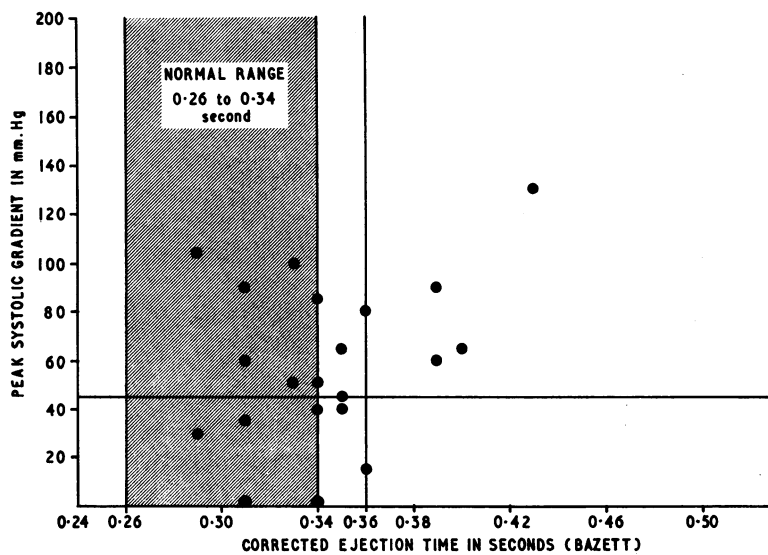


FIG. 7.—Comparison of ejection time and peak systolic gradient in cases of combined aortic and mitral stenosis.

stenosis. The longest ejection times were found when mild mitral stenosis was combined with severe aortic stenosis.

Measurements on the carotid pulse wave in severe isolated mitral stenosis showed values for the ejection time and the 't' time within the normal range. The upstroke time and the ascending index both had upper limits exceeding the normal range, although the average values were the same as in the controls. A slight prolongation of the upstroke time can, therefore, occur in severe mitral stenosis without any corresponding lengthening of the ejection period.

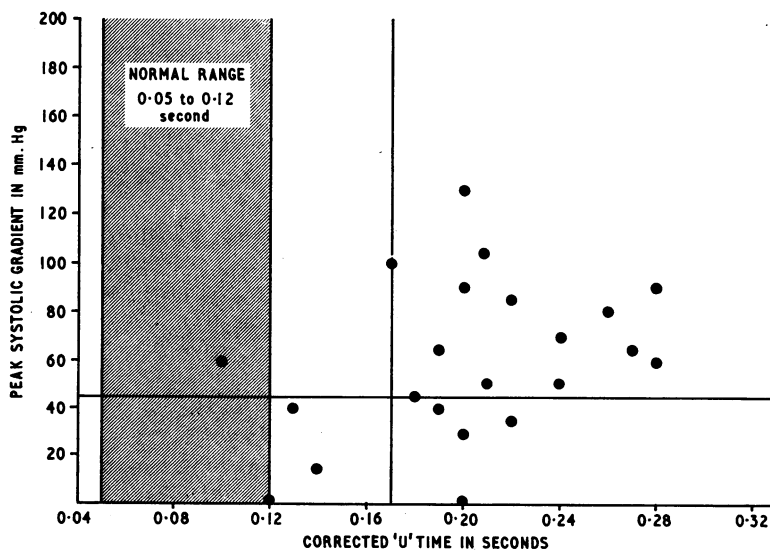


FIG. 8.—The same cases as Fig. 7, comparing upstroke time and peak systolic gradient.



## DISCUSSION

Marey (1863) first introduced graphic methods to record the peripheral pulse wave and noted the different pulse contours in aortic stenosis and aortic regurgitation. Bowen (1904) used the carotid pulse tracing to measure the duration of left ventricular ejection and Lombard and Cope (1926) observed an inverse relation between heart rate and ejection time in normal subjects. Katz and Feil (1925), using optical recordings, showed a prolonged systolic ejection time in patients with aortic stenosis, and Katz *et al.* (1928) later showed a similar prolongation of ejection time in experimental aortic stenosis recorded from the central aortic pressure pulse in dogs: ejection time increased with the severity of the stenosis. Similar experimental observations were made by Wiggers (1949).

During the past 10 years, several papers have been published describing the direct and indirect arterial pulse tracing as an aid to the diagnosis of aortic stenosis. The majority of authors have demonstrated the presence of a prolonged systolic ejection time and a delayed summit. Although these features aid in diagnosis there have been few attempts to correlate the severity of the aortic stenosis assessed objectively with the indirect carotid pulse tracing.

Donoso *et al.* (1956) compared indirect carotid pulse tracings in normal subjects with a group of 38 patients with aortic disease. They showed a prolongation of the upstroke time and of the systolic ejection time, but did not relate their findings to the severity of the stenosis. Duchosal *et al.* (1956) measured the 't' time and found that it increased with the severity of the stenosis. None of their patients was objectively assessed and a purely clinical grading was used.

Eggink *et al.* (1958) recorded the carotid pulse wave in 48 children with congenital aortic stenosis and stressed the value of the upstroke time and the ascending index (u/E-T ratio) in diagnosing the presence of aortic stenosis. They did not attempt to relate their findings to the degree of stenosis. Meyer-Heine *et al.* (1958) analysed the indirect carotid pulse in 40 patients with aortic stenosis, of whom one-third had associated mitral stenosis, and they found a prolonged systolic upstroke time in patients with severe stenosis: most of their patients were graded clinically although three came to autopsy. Daoud *et al.* (1959) found an increased systolic upstroke time, 't' time, and ejection angle in the carotid pulse curves of 40 patients with aortic stenosis, but without any correlation with the degree of obstruction. Benchimol *et al.* (1960) analysed the carotid sphygmogram in 25 patients with pure or dominant aortic stenosis. All their cases were confirmed at operation with the recording of pressure gradients in 22 patients and additional autopsy evidence in 8 patients. They found that the systolic ejection time was prolonged in all their patients, the upstroke time in 92 per cent and the 't' time in 72 per cent.

In our own series of 47 patients, the systolic ejection time, the upstroke time, the 't' time, and the ascending index were all prolonged in proportion to the severity of the aortic stenosis as assessed objectively, and the upstroke time was above the upper normal limit in more patients (81%) than any of the other three measurements. In contrast, when the 'critical' levels are considered with a minimal gradient of 45 mm. Hg the largest number of positive results (66%) was shown by the ascending index.

The ascending index combines two of the essential features of the pulse in aortic stenosis, namely the delayed summit and the prolongation of the period of ventricular ejection, findings that have been observed clinically and experimentally. It expresses the time occupied in attaining peak systolic pressure as a fraction of the total period of systolic ejection. A combination of the four parameters, assessed on the basis of a simple system of scoring, gave more useful information than any single factor alone. The severity of the aortic stenosis and the average peak systolic gradient across the aortic valve increased with the 'score' (Fig. 6). With two or more 'significant' positive factors the aortic stenosis was always of at least moderate severity.

Only 1 of the 8 patients with mild aortic stenosis had a single measurement above the 'critical' levels defined. However, in 6 of these 8 patients at least 1 of the 4 measurements was above the upper limit of normal, although less than the 'critical' level. In addition, 2 patients with moderate aortic stenosis had no significant positive measurements although in one of them the ejection time

was above normal. Mild or moderate aortic stenosis may, therefore, be present with a relatively normal carotid pulse contour.

The contours of the aortic and left ventricular pressure curves are normally parallel during the ejection period (Wiggers, 1949, 1952). This alters in aortic stenosis, the peaks no longer coincide in time, and there is a delay in the aortic peak associated with a diminished rate of ascent (Katz *et al.*, 1928; Wiggers, 1949). The systolic ejection period is prolonged as a compensatory mechanism for the reduced rate at which blood leaves the left ventricle.

Although the central aortic pressure pulse in aortic stenosis is principally determined by the degree of aortic obstruction, other factors can significantly alter the pulse form. The stroke volume, the peripheral vascular resistance, and the distensibility of the aorta are all capable of influencing the pulse contour. The systolic ejection period in normal subjects is prolonged in a linear fashion with increasing stroke volume. In aortic stenosis there is abnormal prolongation of ejection time relative to the stroke volume as shown by Gorlin *et al.* (1955) and by Weissler, Peeler, and Roehll (1961).

Goldberg *et al.* (1954) and Doyle and Neilson (1957) have shown alterations in the peripheral pulse contour during the Valsalva manœuvre in patients with aortic stenosis. The latter authors showed reduction in the systolic upstroke time during the straining period, which appeared to be due to diminished stroke volume and was associated with a fall in pulse pressure. In severe stenosis there was a greater increase in upstroke time for a given change of pulse pressure than in lesser degrees of aortic stenosis. The pulse contour thus appeared to vary with the stroke volume. This observation was also borne out when studying those of our own patients with atrial fibrillation or with ectopic beats, when the pulse contour was relatively normal after a short diastole although typical of severe aortic stenosis following a long diastolic interval.

Prolongation of the systolic ejection period has been postulated in hypertension and myocardial failure (Wiggers, 1952; Braunwald, Sarnoff, and Stainsby, 1958; and Smith *et al.*, 1959). Wiggers (1952) has shown experimentally in dogs that large increases in aortic pressure prolong the systolic ejection time. Braunwald *et al.* (1958) also showed that ventricular ejection was only prolonged in dogs when the mean aortic pressure was much raised. In addition, they demonstrated some increase in the period of ejection in the failing dog heart. However, Weissler *et al.* (1961) found clinically that the ejection time was not prolonged in a group of 11 patients with systemic hypertension.

Twenty patients with congestive cardiac failure due to ischæmic heart disease were studied by Benchimol *et al.* (1960) and shown to have a shortened total ejection time, and Weissler *et al.* (1961) found the ejection time to be diminished relative to heart rate in 12 patients with myocardial failure due to non-valvular heart disease. The ejection time was normal relative to stroke volume and thus reflected a low stroke volume in their patients. Thus clinical and experimental observations differ regarding the effects of hypertension and heart failure on the duration of systolic ejection. This may be related to the acute nature of the experiments compared to the clinical situation where compensatory mechanisms, such as ventricular hypertrophy, have had time to develop.

Significant aortic regurgitation can also prolong the ejection time (Katz and Feil, 1925; Wiggers, 1949; Weissler *et al.*, 1961). This is due to the time taken by the left ventricle to eject a large stroke volume consisting of the effective stroke volume together with the amount regurgitated. Although aortic regurgitation increases the systolic ejection time it is associated with a steep ascent of the pulse wave which quickly reaches its peak level. The upstroke time and the 't' time are less than normal and the ascending index will be correspondingly diminished. In practice a prolonged ejection time as an isolated finding is not likely to give rise to any difficulty if the clinical situation is known and particularly if the other measurements are below the normal upper limit.

It has long been recognized that the typical murmur and anacrotic pulse of aortic stenosis may both disappear during an episode of cardiac failure only to appear again as the patient's clinical state improves. This phenomenon is probably related to low cardiac output during the period of failure.

In combined aortic and mitral stenosis the peripheral pulse contour is modified by the presence

of the obstruction at the mitral valve. The cardiac output is diminished in severe mitral stenosis (Gorlin and Gorlin, 1951), and this also applies when the mitral stenosis is combined with aortic stenosis. Uricchio *et al.* (1959), Katznelson *et al.* (1960), and Honey (1961) found subnormal cardiac outputs in their series of patients with combined aortic and mitral stenosis.

For a given aortic valve area the gradient across the aortic valve and the systolic ejection time depend on the stroke volume. However, in aortic stenosis the ejection time increases more for a given rise in stroke volume than it does in normal subjects (Gorlin *et al.*, 1955; Weissler *et al.*, 1961). Benchimol *et al.* (1960) showed that the ejection time increased with stroke volume and cardiac output in pure mitral stenosis. The improved cardiac output following mitral valvotomy was associated with an increase in ejection time.

In our own series of 21 patients with combined lesions the peak systolic gradient was diminished in the presence of severe mitral stenosis. At operation in many of these patients the aortic valve gradient increased after mitral valvotomy before aortic valvotomy. Similar observations were noted by Brock (1957) and Honey (1961). The increase in aortic valve gradient reflects an increased flow across the valve. Mitral stenosis therefore appears to affect the carotid pulse curve in combined lesions by limiting the cardiac output. This probably accounts for the poor relation between peak systolic gradient and the various carotid pulse parameters in combined aortic and mitral stenosis. The less severe the mitral stenosis the more helpful the carotid pulse in assessing the aortic stenosis.

A linear correlation could not be established between the peak systolic gradient and any of the four parameters assessed in isolated aortic stenosis. This perhaps is not surprising in view of the many factors that influence the carotid pulse wave. Although the degree of aortic valve obstruction is the most important factor in determining pulse contour, the stroke volume makes a significant contribution, and this is well shown in those patients with combined aortic and mitral stenosis. The recordings of the carotid pulse were made under varying conditions and, although the patients were resting quietly on an examination couch, none of them were in a basal state and there must have been large variations in cardiac output.

By measuring the range of values in normal subjects, recorded under similar conditions, and defining a critical level based on physiological studies at some distance above the upper normal limit, it was possible to avoid false positive findings. There were a number of false negative observations although all but one of those patients with severe lone aortic stenosis had at least two measurements above the critical level. Normal findings in the presence of moderate or severe aortic stenosis appear to be due to a small stroke volume and hence normal values cannot exclude the presence of aortic stenosis. Values greater than normal but less than the critical levels allow a diagnosis of aortic stenosis to be made but do not provide information regarding its severity. Values above the critical levels are virtually diagnostic of aortic stenosis of at least moderate severity since there is no other clinical situation that produces similar findings.

#### SUMMARY

The external carotid pulse wave in lone aortic stenosis showed certain distinctive changes in contour when compared with the normal. These were principally related to obstruction to outflow at the aortic valve and they provided valuable information concerning its severity.

Four measurements were made from the carotid pulse curves: the systolic ejection time, the systolic upstroke time, the 't' time, and the ascending index (u/E-T ratio).

When compared with similar measurements in normal people, patients with aortic stenosis showed levels above the upper limits of normal—the ejection time and the 't' time in 70 per cent, the ascending index in 75 per cent, and the upstroke time in 81 per cent of patients.

Critical levels were defined for each of the four parameters at or above which the peak systolic gradient was at least 45 mm. Hg. The ascending index was the measurement most frequently above the critical level (66%).

The number of factors above the defined critical level increased with the systolic pressure gradient across the aortic valve. Similarly, when the patients were graded as having mild, moderate, or

severe aortic stenosis, the number of critical measurements increased with the severity of the stenosis.

Two or more critical measurements were always associated with at least moderate aortic stenosis and the gradient across the valve was then never less than 45 mm. Hg. Severe aortic stenosis could not be excluded by the absence of any positive measurements, and the reasons for this finding are discussed. Detailed analysis of the indirect carotid pulse tracing, therefore, appears to be a valuable way of assessing the severity of lone aortic stenosis.

In combined mitral and aortic stenosis the mitral stenosis reduced the cardiac output and modified the effects of the aortic stenosis on the carotid pulse contour. The reduced aortic valve flow appeared to be responsible for lower systolic pressure gradients across the aortic valve and also for less obvious alterations in the indirect carotid pulse curve. Apart from the systolic ejection time, the criteria used for assessing lone aortic stenosis were not valid in combined lesions.

Although the carotid pulse was generally of no value in assessing the severity of the aortic stenosis in combined lesions, the changes were often distinctive enough to allow aortic stenosis to be recognized even in the presence of severe mitral stenosis. The less severe the mitral stenosis the more accurately the carotid pulse reflected the degree of aortic stenosis.

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