AORTIC STENOSIS: A CLINICAL STUDY

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Since early descriptions of aortic stenosis (Bonet, 1700; Burns, 1809; Hope, 1835) numerous studies on this subject have been reported—for example, Mönckeberg (1904), Christian (1931), McGinn and White (1934), Karsner and Koletsky (1947), Kumpe and Bean (1948), Lewes (1951), Mitchell *et al.* (1954). In addition, congenital aortic stenosis, the frequency of which was first noted by Gallavardin (1933), was recently reviewed by Campbell and Kauntze (1953).

The introduction of operative treatment (Larzelere and Bailey, 1953; Logan and Turner, 1954; Brock, 1954) led us to make a study of 50 consecutive patients with aortic stenosis attending the out-patient department of St. Thomas's Hospital, with special reference to the features which might determine their suitability for operation.

Material and Methods

The diagnosis of aortic stenosis was made from the characteristic harsh systolic murmur, maximal either in the aortic area or in the suprasternal notch. This murmur was usually loud, but, as is emphasized later, in cardiac failure its intensity was sometimes reduced. Those with evidence of mitral valvular disease were excluded. Only four of the patients have died; severe aortic stenosis was found in the three who came to post-mortem examination.

Of the 50 patients, 31 were males and 19 females. Thirty-one had an aortic diastolic murmur. In each case there was a full clinical examination, and the findings were agreed upon by at least two observers. All had fluoroscopy and electrocardiography (ten leads). In 39 an arterial pulse-pressure record was made from a needle in the brachial artery, using a Sanborn or Tybjaerg Hansen manometer and Sanborn direct writing recorder. Cardiac catheterization was performed in 20, and, for comparison, in five patients with syphilitic aortic incompetence. The detailed results of the findings at cardiac catheterization are reported elsewhere (Gorlin *et al.*, 1955).

Age, Sex, and Aetiology

The age and sex of the patients and the aetiology of the disease are shown in Table I. The patients have been divided into three aetiological groups—congenital, rheumatic, and undetermined. Those whose heart disease had been diagnosed at or before the age of 6 were arbitrarily considered to have a congenital aortic valvular lesion. In some of this group it is possible that the aortic stenosis may have resulted from fibro-elastosis; such a case, with histological proof, has recently been brought to our attention by Dr. D. H. Davies (Fig. 1), but is not included in this series. Of those classified as rheumatic 16 (7 male, 9 female) gave a history of rheumatic fever, 2 (1 male, 1 female) of chorea, and 2 (1 male, 1 female) of both. The incidence of a rheumatic history is given in more detail in Fig. 2. In common with other series—for example, Campbell and Shackle (1932)—a greater incidence of rheumatism was found in the females (11 of 19) than in the males (9 of 31), and it will be seen that, whereas in the females the incidence is about the same in all age groups after 20, in the males the greatest frequency is in middle age. Of the 20 patients with a rheumatic history five did not have an aortic diastolic murmur, which throws doubt on the conclusion of Kiloh (1950), who stated that "the evidence is in favour of accepting this type of aortic stenosis (pure aortic stenosis) as being of non-rheumatic origin."

TABLE I

Aetiology	Males	Females	Total	Average Age	Age Range
Congenital ..	1	5	6	28	9–41
Rheumatic ..	9	11	20	47	22–68
Undetermined	21	3	24	54	14–76
Total ..	31	19	50	50	9–76

Of the patients listed as "undetermined" no fewer than 21 of the 24 were male. It has been suggested in another

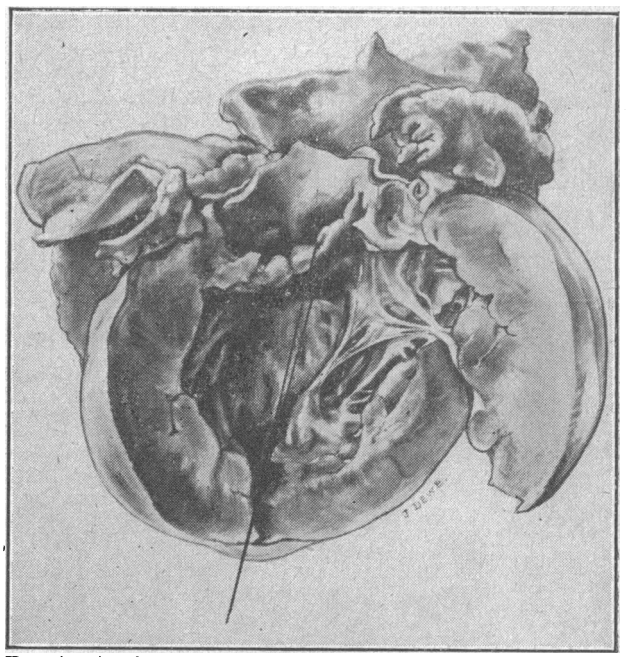

FIG. 1.—Aortic stenosis due to fibro-elastosis. Specimen from a boy of 6 whose murmur was discovered at the age of 2. Note the thickened edges of the valve cusps. (Dr. D. H. Davies's specimen. Photographed from an original drawing by Miss Joan Dewe.)

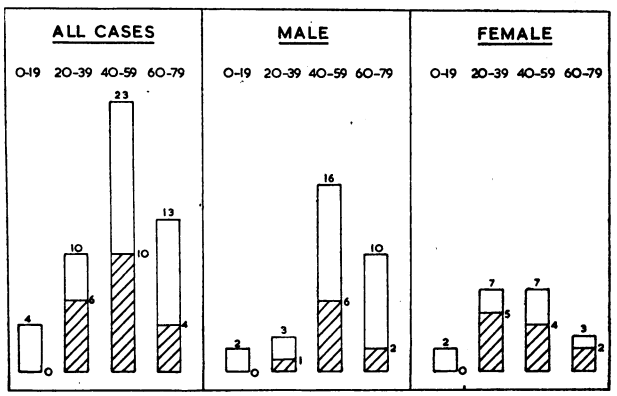

FIG. 2.—Incidence (cross-hatched) of a history of rheumatic fever or chorea.

communication (Smith and Matthews, 1955) that in some cases the aortic stenosis may be attributable to degeneration in a congenital bicuspid valve, a view first proposed by Peacock (1865) and which appears sporadically in the literature—for example, Koletsky (1941) and Kiloh (1950).

Symptoms

The distribution of symptoms is shown in Table II.

TABLE II

	No.	No Symptoms or Slight Dyspnoea	Moderate or Severe Dyspnoea	Paroxysmal Nocturnal Dyspnoea	Angina Pectoris	Effort Syncope
Males:						
Congenital ..	1	—	—	—	1	—
Rheumatic ..	9	5	3	2	1	2
Undetermined	21	9	7	2	7	4
Females:						
Congenital ..	5	3	—	—	2	—
Rheumatic ..	11	2	6	3	7	1
Undetermined	3	—	3	1	1	—
Total ..	50	19	19	8	19	7

Dyspnoea.—Nineteen patients had either no symptoms or only slight dyspnoea. Only eight had paroxysmal nocturnal dyspnoea. These severely disabled patients are considered in more detail below.

Angina Pectoris.—The distribution of angina pectoris is shown in Fig. 3. Our series supports Wood's (1950) statement that the type of pain in aortic stenosis is no different

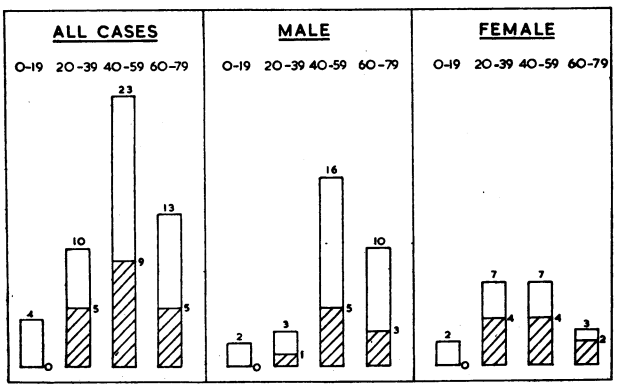

FIG. 3.—Incidence (cross-hatched) of angina pectoris.

from that of coronary arterial disease (contrary to the suggestion of Kumpe and Bean, 1948). The ability of aortic stenosis to cause angina of effort at an early age is confirmed. Four of the women aged less than 30 had this symptom. It was nearly as common (11 out of 31) in the patients without or with only slight dyspnoea as in those who were more breathless. We agree with Davies and Steiner (1949) that the "mechanism of angina of effort in this disease is not clear." Some of the relevant factors are discussed in another communication (Gorlin *et al.*, 1955).

Effort Syncope.—This occurred in seven patients; six of these were males. Their average age was 65 (range 41 to 76); the average age of those without this symptom was 50. We have no evidence to account for the preponderance of this symptom in elderly males.

Signs

The signs are set out in Table III.

Pulse.—"I have more than once observed old and eminent practitioners make such different judgments of hard, full and weak and small pulses, that I was sure they did not call the same sensations by the same name. It is to be wished, therefore, that physicians, in their doctrines of the pulses, and descriptions of cases, had attended to such circumstances of the pulse in which they could neither mistake nor be misunderstood" (Heberden, quoted by Ryle, 1948). Our experience supported Heberden's contention, and

TABLE III

	No.	Bisferiens Brachial Pulse	"L.V." Apex	Systolic Thrill	Aortic Diastolic Murmur	A_2 Less than Normal or Absent
Males:						
Congenital ..	1	—	—	1	1	1
Rheumatic ..	9	7	6	6	6	5
Undetermined	21	8	16	14	12	8
Females:						
Congenital ..	5	2*	4	5	2	2
Rheumatic ..	11	3	8	9	9	2
Undetermined	3	1	2	2	1	2
Total ..	50	21	36	37	31	20

* One patient had no aortic diastolic murmur (see text). "L.V." apex = Apical thrust of left ventricular overactivity. A_2 = Second heart sound in aortic area.

we often found it impossible to differentiate the anacrotic or plateau pulse from the normal, or from the small pulse which accompanies cardiac failure from other causes.

The double wave of the bisferiens pulse, though also a "makeweight" sign, is more useful, as it leaves less room for error. For its detection palpation of the brachial artery is more reliable than the radial; in no instance was it present in the radial but absent in the brachial, but the reverse was common. The double impulse can best be felt on the upper border of the thumb while it occludes the brachial artery against the humerus. It is important to realize that partial occlusion may lead to a false impression of the bisferiens pulse in normal individuals.

Of the 31 patients with an aortic diastolic murmur, 20 had this pulse. The single patient without an aortic diastolic murmur was of particular interest. When first seen at the age of 31 she was pregnant, and the bisferiens quality of the pulse was agreed by three observers. After delivery there was no bisferiens pulse. A subsequent pregnancy was accompanied by a return of this pulse at the second month; it disappeared after a therapeutic abortion. Evidence that with aortic stenosis an increased stroke volume is the cause of the bisferiens pulse is to be presented elsewhere (Matthews and Medd, in preparation). Usually, of course, the increase results from aortic incompetence.

Two of the 50 patients had auricular fibrillation, the remainder sinus rhythm. One was a man of 46 who died soon afterwards, and the other a woman of 73 with few symptoms. The rarity of "perpetual arrhythmia" was realized by Hope (1835) and Stokes (1854), though Lewes (1951) found that 5 of the 25 patients in his series had auricular fibrillation. His patients had all come to necropsy, so that a terminal arrhythmia may partly account for this greater incidence.

Apex.—A thrusting apex beat suggestive of left ventricular overactivity was found in 36 patients, and was commoner (26 out of 31) in patients with associated aortic incompetence than in those with pure aortic stenosis (10 out of 19), presumably due to the increased left ventricular work caused by the aortic regurgitation.

Aortic Second Sound.—It was notable that the aortic second sound was normal in 30 patients. Contrary to expectation there was no correlation between diminution or absence of the aortic second sound and calcification of the aortic valve on fluoroscopy. So far as we could judge, a knowledge of the intensity of the aortic second sound contributed nothing to the evaluation of aortic stenosis.

Systolic Thrill and Murmur.—The thrill was sought in the usual way with the patient leaning forward and with the breath held in full expiration. Though usually most pronounced at the base of the sternum, occasionally it was maximal in the suprasternal notch. No attempt was made to grade the intensity of the systolic murmur, because it was realized that it varies not only with the valve orifice size but also with blood flow and the factors which control the transmission of the sound. In one patient, a man aged 68 with a calcified aortic valve, the systolic murmur, previously loud, became almost inaudible when cardiac failure developed. This has been noted by others (Kumpe and Bean,

1948; Lewes, 1951); and Contratto and Levine (1937) suggest that "terminal cardiac weakness" may have been the explanation of their finding that only 21 of their 51 patients who came to necropsy had a systolic thrill in life. The difficulty of diagnosis in aortic stenosis that sometimes occurs when the patient is first seen in cardiac failure is referred to by Conner (1931), and Christian (1931) states that "if cardiac decompensation is marked, the thrill may be absent, and the murmur lose its harshness and loudness, and so this diagnostic evidence often disappears a few days before death." It is likely that the reduction in blood flow accounts for this finding, and the fact that, whereas an aortic systolic thrill was found in 10 out of 12 patients without disability, it was present in only four out of eight with paroxysmal nocturnal dyspnoea lends support to this interpretation.

Fluoroscopy

The fluoroscopy findings are shown in Table IV.

TABLE IV.—*Fluoroscopy and Electrocardiography*

	No.	L.V.+	Prominent First Part of Aorta	Calcification of Aortic Valve	E.C.G. of L.V.H. (incl. L.B.B.B.)
Males:					
Congenital	1	1	1	1	1
Rheumatic	9	7	3	8	9
Undetermined ..	21	19	12	17	15
Females:					
Congenital	5	5	3	2	3
Rheumatic	11	10	9	3	10
Undetermined ..	3	2	2	2	2
Total	50	44	30	33	40

L.V.+ = Apparent enlargement of left ventricle in 60 degrees left anterior oblique view. L.V.H. = Left ventricular hypertrophy. L.B.B.B. = Left bundle-branch block.

The appearance of enlargement of the left ventricle in 44 patients, and prominence of the first part of the aorta to the right and anteriorly in 30, conformed with the experience of others—for example, Campbell and Kauntze (1953). Of the six patients in whom the left ventricle appeared normal on screening, five had pure aortic stenosis and only one had associated incompetence, despite the fact that cases of the former constitute only 38% of the series. This reinforces the clinical finding noted above, that there is a greater tendency for aortic stenosis with incompetence to cause obvious left ventricular enlargement than aortic stenosis alone.

Particular care was taken to achieve adequate dark adaptation by wearing red glasses for at least a quarter of an hour before a search was made for calcification of the aortic valve. The high incidence of calcification (found in 33 of the 50 patients) is probably largely attributable to this. It was recorded as present only if clearly seen by all observers; doubtful flecks were disregarded. The site of the aortic valve when calcified seemed to us more constant than that of the mitral valve, presumably owing to the greater variation in the enlargement of the left atrium and right ventricle in mitral valvular disease. It was usual, in a 45 degrees right anterior oblique position, to find the shadow of the calcified valve moving in a small ellipse on a line dropped perpendicularly from the anterior margin of the aortic shadow. The position of the calcified valve is well illustrated by Davies and Steiner (1949).

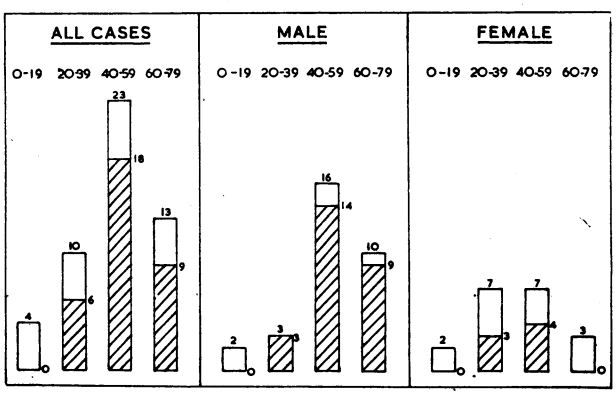

FIG. 4.—Incidence (cross-hatched) of calcification of the aortic valve on screening.

The incidence of valvular calcification is shown in more detail in Fig. 4. It is notable that it was found in 26 of 29 males aged more than 20. The equivalent figure for females was 7 of 17. In mitral stenosis there is a similar disparity between the two sexes in the incidence of valve calcification (Matthews, 1954). The finding of calcification did not relate to the severity of the obstruction or to the presence of aortic incompetence.

Electrocardiogram

In 38 patients the electrocardiogram (E.C.G.) showed the inversion of T waves, with or without depression of the ST segments in left ventricular surface leads that is sometimes described as "left ventricular strain" (Table IV). In two patients there was left bundle-branch block. In the remaining 10 patients the T waves were upright. In one of them the sum of the R in V_5 and the S in V_1 was more than 35 mm., thus fulfilling the criteria for left ventricular hypertrophy proposed by Sokolow and Lyon (1949); however, their method is open to the objection that false positives may arise from the fact that an R of 33 mm. may occur in V_5 in health (Kossman and Johnston, 1935) and false negatives from reduction of the voltage in emphysema.

In contrast to the clinical and fluoroscopic findings there was no significant difference in the E.C.G. attributable to coexisting aortic incompetence.

None of the patients in whom the E.C.G. was normal suffered from paroxysmal nocturnal dyspnoea; five were symptomless; two had mild, and two (aged 58 and 73) moderate exertional dyspnoea. One of those with mild dyspnoea also had angina pectoris (female aged 41). Conversely, although left ventricular hypertrophy can be associated with a normal E.C.G. (Goldberger, 1953), all the patients with paroxysmal nocturnal dyspnoea had the T wave changes of left ventricular strain. It would appear that the E.C.G. of left ventricular hypertrophy is one of the most reliable indications of the severity of the stenosis, though severe stenosis can occur with a normal E.C.G.

Brachial Arterial Pressure Pulse

Wiggers (1949) has described the characteristic arterial pressure pulse in normal animals (p. 671 *et seq.*) and in experimental aortic stenosis (p. 986 *et seq.*), and quotes previous workers. In aortic stenosis he stresses the slow rise of pressure, the transmission of the central pulse to the periphery with little change, and the rarity of the "anacrotic phenomenon."

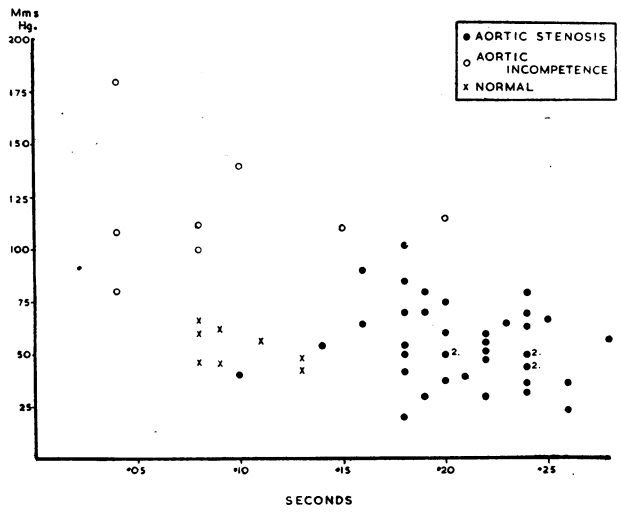

FIG. 5.—The brachial arterial pressure pulse. Systolic upstroke time and pulse pressure. Findings in 39 patients with aortic stenosis, with or without incompetence, in eight patients with aortic incompetence and a positive Wassermann reaction, and in eight normal controls.

The brachial arterial pressure pulse was recorded in 39 of our patients. Because of the many variables, both physiological—for example, stroke output, left ventricular performance, aortic incompetence, peripheral resistance, elasticity of the arterial system, transmission, and distortion factors (Alexander, 1949)—and technical—for example, site and direction of the needle—we do not consider it fruitful to analyse the pulse-pressure contour in detail. We have therefore restricted ourselves mainly to the measurement of the systolic upstroke time from the end of diastole to the final sustained peak of systole, although we appreciate that this measurement is dependent on the variables already described. This measurement was greater than 0.15 second in 37 patients. One of the remaining two appeared to have minimal stenosis; there was no explanation for the normal upstroke time in the other. The upstroke times are plotted against the pulse pressure in mm. Hg in Fig. 5, where they are compared with eight patients with severe aortic incompetence and a positive blood Wassermann reaction, and eight normal subjects using the same method. Of the eight patients with aortic incompetence, six had normal or shorter than normal systolic upstroke times; in one patient it was 0.15 second, and in one 0.20 second. The latter probably had some stenosis; in addition there was a well-marked anacrotic notch. The arterial tracing is thus of use in giving evidence that the aortic stenosis is severe while the E.C.G. is still normal. Examples of brachial arterial pressure pulses are shown in Fig. 6.

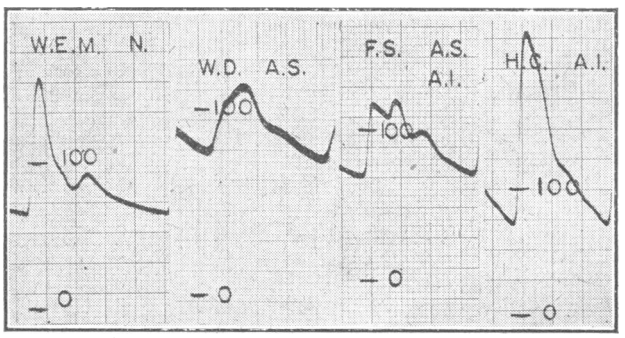

FIG. 6.—Brachial arterial pressure pulses in (from left to right) a normal control, aortic stenosis, aortic stenosis and incompetence, and syphilitic aortic incompetence. (Time marker 0.2 and 0.04 sec. Pressure standard 0–100 mm. Hg.)

Cardiac Catheterization

These findings have been made the subject of a separate communication (Gorlin *et al.*, 1955). There was evidence to show that aortic stenosis prevented the increase in stroke work which normally occurs on exercise before the symptoms were incapacitating, and that the "pulmonary capillary pressure," although normal at rest except for those patients in frank congestive failure, rose abnormally on exercise. This gave a good indication of the poor ventricular reserve.

In combination with post-mortem studies of the aortic valves in an artificial perfusion system (McMillan *et al.*, 1952) it appeared that the critical valve area in isolated aortic stenosis was of the order of 0.5 square cm.; with coexisting aortic incompetence it is variably larger. This reduction is generally due to commissural fusion, but may arise from increased valve-cusp rigidity alone (Smith and Matthews, 1955).

We believe, however, that cardiac catheterization has no important part to play in the routine assessment of a patient with aortic stenosis.

Natural History of the Disease

As our patients have in most cases been followed only for a maximum of one year since the study, the only guide to the natural history of the disease they can provide is from a consideration of the duration of their symptoms at the time we saw them. Nineteen patients (10 male, 9 female) had more than slight dyspnoea on exertion. In the males this symptom had been present for less than one year in five patients, for two years in three patients, and for four and seven years respectively in the remaining two (average duration two years). In the females the duration was longer, with an average of four years. Of eight patients with paroxysmal nocturnal dyspnoea, only one was aged less than 50 (46), and in this patient the symptom had been present for four years; in the remainder the duration was in each case less than one year. In 19 patients with angina the duration of the symptom was more variable; in 11 it was one year or less, in four, one to five years, in three, five to ten years, and in the remaining patient nineteen years.

Severe dyspnoea appears, therefore, to be incompatible with a long survival. This supports the results of Grant (1933), who followed for ten years 43 male war pensioners with aortic stenosis. At the end of this period 28 had died; of these, 10 died of cardiac failure, seven suddenly, two of

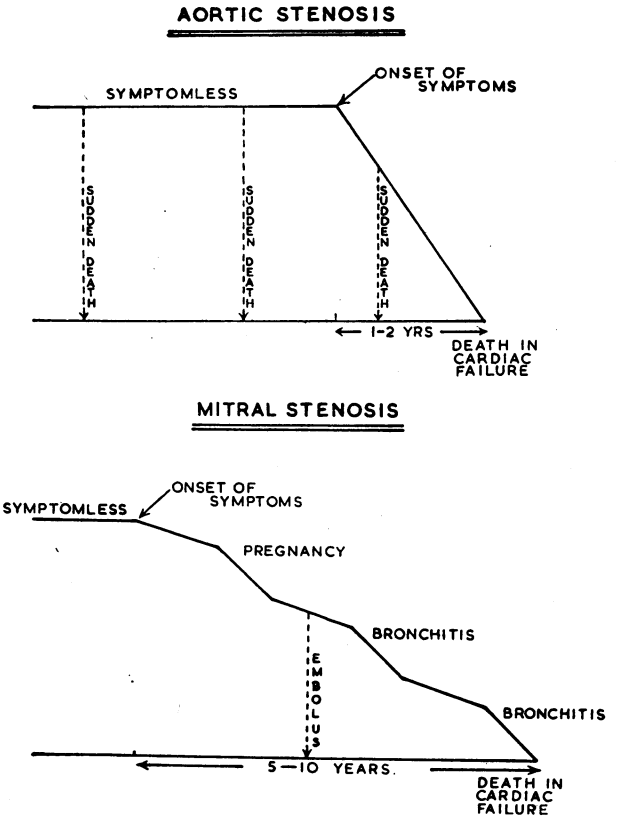

FIG. 7.—Diagrammatic representation of the natural history in aortic stenosis (above) and of mitral stenosis (below).

subacute bacterial endocarditis, and eight of unrelated causes. Thus between one-third and one-half died of the disease itself in ten years. The poor prognosis once severe symptoms develop is also emphasized by Contratto and Levine (1937), who found that the average length of survival after the first appearance of dyspnoea was twenty-three months and of pitting oedema four months. There is evidence that since the introduction of mercurial diuretics the prognosis has been considerably improved (Mitchell *et al.*, 1954).

The natural history of aortic stenosis is compared with that of mitral stenosis in Fig. 7. In the former there is a long symptomless period (specifically noted by Peacock, 1877) and, when left ventricular failure occurs, a short period of severe symptoms until death, but at any moment sudden death may occur. In the latter there is a comparatively long period of severe symptoms and the downhill progress is marked by exacerbations which are often associated with attacks of bronchitis, with pulmonary infarction, or with pregnancy.

With a view to deciding whether the onset of left ventricular failure was mainly due to the stenosis reaching a critical degree or to the progressive ischaemic changes that occur in the left ventricular muscle throughout years of established stenosis, a comparison was made between the 12 patients who had no disability (as judged by their exercising for more than five minutes on a standard exercise test) and the eight patients with paroxysmal nocturnal dyspnoea (Table V). It will be seen that there is little

TABLE V.—*Comparison of Least and Most Disabled Patients*

	12 Patients who Exercised more than 5 mins. on a Standard Exercise Test	8 Patients with Paroxysmal Nocturnal Dyspnoea
Sex	10 male; 2 female	4 male; 4 female
Average age	41	60
Age range	16–59	46–68
Left ventricular apex beat	8	5
Systolic thrill	10	4
Screening L.V.+	11	7
Calcified valve	9	5
L.V. hypertrophy on E.C.G.	9	8
Arterial tracing	9	5
Systolic upstroke more than 0·15 sec.	8	5
Average systolic upstroke (sec.)	0·22	0·19

difference between the two groups in the incidence of the factors that indicate the severity of the stenosis. The main difference is that the average age in the first group is 41 and in the second 60. These findings suggest that it is the duration of the aortic stenosis rather than its severity alone which precipitates left ventricular failure.

Two further patients in the series developed cardiac failure temporarily when they became severely anaemic (haemoglobin 30% and 48%).

Discussion

The diagnosis of severe aortic stenosis can usually be made from evidence of left ventricular hypertrophy in the presence of the characteristic murmur. For reasons already given, the intensity of the murmur is an unreliable guide to the degree of obstruction. When there is doubt an arterial pressure record may be helpful. Moreover, it should not be necessary to submit the patient to thoracotomy to demonstrate the severity of the aortic stenosis by measurement of the systolic pressure differential across the aortic valve, as suggested by Brock.* Such measurements are likely to be unreliable under the abnormal conditions of the operation.

Effective relief of the stenosis should make a more significant difference to the natural history of the disease than any medical treatment. However, the difficulty in the selection of patients for operation lies in the fact that cardiac failure, once it develops, runs a rapid course, and, conversely, at the time when the operation is likely to be most effective the patient is often symptomless and may not appreciate its desirability. This contrasts with mitral stenosis, in which there is often a comparatively long period when the patient has severe symptoms and is suitable for surgery. In aortic stenosis the selection of patients for operation divides itself into a technical and a moral issue. The theoretical advantages of early operation include: (1) the lower mortality and morbidity for all operations in younger patients; (2) the calcification of the valve is not so far advanced, so that the prospect of producing a functional valve is greater and the risks of calcific embolus are less; (3) subsequent fibrosis of the left ventricular myocardium may be retarded or delayed; (4) the repair of the left ventricular myocardium is easier (when the ventricular approach is used); and (5) the danger of ventricular fibrillation is reduced (Bailey *et al.*, 1954).

The disadvantages of early operation are that at the moment the operation is experimental, and though few data have been published it is likely that the overall mortality is high. However, the operation has so far been used mainly in terminal cardiac failure. On theoretical grounds the ideal patient for surgery is the young or middle-aged woman with severe symptoms and an uncalcified valve.

Operations for aortic stenosis are not likely to be so effective as for mitral stenosis, since valve calcification seems to be more common in the former. Unless there is some mobility in the valve, even splitting the commissures may produce a negligible increase in the functional valve orifice size, as the valvotome applies an opening force to the valve considerably greater than the ejected blood (McMillan, 1955).

*Unpublished observation delivered to Cardiac Society meeting on April 9, 1954.

Summary

Fifty patients with aortic stenosis were studied, and their clinical features are discussed. The findings from the brachial arterial pressure pulse in 39 patients are described. In the presence of the characteristic murmur the severity of the stenosis is established by evidence of left ventricular hypertrophy, and supported by a systolic upstroke time of more than 0.15 second in the brachial arterial pressure tracing. The critical aortic valve area appears to be of the order of 0.5 square cm. in isolated aortic stenosis, and is variably larger with coexisting aortic incompetence.

The natural history of the disease is discussed and contrasted with that of mitral stenosis.

The problem of selection of patients for valvotomy is more difficult than in mitral stenosis, since severe aortic stenosis is often compatible with an active life, and when left ventricular failure eventually occurs the valve may well be so abnormal as to prohibit an effective valvotomy. At the present time the operative mortality is high and the long-term results are unknown.

Our thanks are due to Drs. D. E. Smith, Evan Jones, Raymond Daley, and I. K. R. McMillan for much help in this investigation; to those physicians and surgeons of St. Thomas's Hospital who kindly allowed us to study their patients; to Miss Joan Dewe for the drawing and Mr. K. G. Moreman for the photograph; and to many colleagues for helpful criticism of the text.

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