Original Articles

AORTIC STENOSIS*

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THE fact that mitral stenosis, the unquestionable stigma of previous rheumatic infection, is a commonplace in many parts of India bespeaks the widespread presence amongst us of the seed, or hypothetical agent, that induces the rheumatic infection. In certain respects, India is the soil *par excellence* for the infectious agent : I refer to the sociologic factor, namely, the poverty of the masses in India and to the fact that India is the classical domain of deficiency diseases. The climatic factor too may be comprehensively assessed in the wide terrain of the Indian subcontinent with its unique range of climates. Finally, some parts of India have a seasonal abundance of streptococcal infections, such as tonsillitis, sore throats, acute sinusitis, and middle-ear disease which are often the precursors as well as important complications of our predominantly subacute rheumatism, which is not less serious than the rheumatic fever of England is dangerous. Accordingly, the rendition of satisfactory figures for our incidence of rheumatic heart disease would be much appreciated by those physicians abroad that are concerned with the rheumatic problem. If, however, we are to submit unimpeachable figures for our incidence of rheumatic heart disease and of other ætiological types of organic heart disease, we must adopt standard methods of classification and investigation and our clinical, electrocardiographic, radiological and pathological reports must be correspondingly accurate. I feel sure that if acceptable figures for the incidence of rheumatic heart disease were available to Homer J. Swift, he would hardly have stated at the First International Health Broadcast from New York in 1938 that 'Rheumatic heart disease rarely occurs among inhabitants of the tropics, unless they have contracted it elsewhere'. This paper on aortic stenosis and a previous one 'Some Notes on Clinical Heart Disease' (Kelly, 1939) are an attempt to indicate briefly some modern concepts of heart disease and methods of inquiry into it.

There are two main clinical groups of cases of aortic stenosis, the young and the elderly group. The fundamental pathology of both groups is essentially rheumatic. In young people aortic stenosis is the hall-mark of a severe rheumatic carditis in the past. In elderly men, that is to say in men over fifty years of age, aortic stenosis, according to Dry and Willius (1939) of the Mayo Clinic, is the outcome of progressive calcification of aortic valves that have been slightly damaged by the rheumatic infection in early life. 'The time factor', remarked Dry and Willius, 'explains why calcification need not be a universal concomitant of healed rheumatic lesions, because those harbouring the more serious forms of the disease, especially when accompanied by mitral stenosis, are weeded out by death before such an event as calcification can occur, thus leaving the solitary aortic lesion, one that the heart tolerates far better than mitral stenosis, to continue for a long time without embarrassing the cardiac reserve'. 'The evidence derived from the study of this material (288 cases of calcareous disease of the aortic valve) has led us', stated Dry and Willius, 'to accept unequivocally rheumatic infection as the ætiological factor in calcareous disease of the aortic valve. Rheumatic infection which eventually culminates in calcareous stenosis of the aortic valve was originally a mild form of rheumatic carditis which has allowed both the mitral valve and the myocardium to escape with minimal or no damage'. 'Our material', they observed, 'was derived largely from the Middle Western States where, on the whole (as perhaps in India), mild and atypical forms of the disease may be anticipated'. 'The frequent absence of a rheumatic history in calcareous disease of the aortic valve clearly substantiates, in the opinion of Willius, his hypothesis that 'the original acute inflammatory episode was mild and perhaps so atypical that its significance could not be realized at the time'. He concludes that calcareous disease of the aortic valve 'is not rare' in the Middle West of America. 'It is possible', he remarks, 'that surveys in the regions where the disease (rheumatic infection) is more prevalent may show a relatively lower incidence of calcareous disease of the aortic valve'. The prevalence of rheumatic heart disease in Bengal may be approximately gauged from our hospital statistics given later in this paper. Until the distinctive features of calcific aortic stenosis are more widely known to the general practitioner its incidence cannot be fairly estimated.

Rheumatic heart disease in hospital practice

In the ten-year period, 1930 to 1939, there were 32,907 admissions (28,107 Indians and 4,800 Anglo-Indians) from all causes, to the medical wards of the Medical College Hospitals, Calcutta. Of these, 351 (310 Indians and 41 Anglo-Indians) were cases of rheumatic heart disease, as detailed below. Otherwise stated, one medical admission in every hundred was a rheumatic heart case. The prevalence however of cardiac rheumatism in Bengal is, in my opinion, somewhat less than our hospital figures suggest. Furthermore, the rheumatic infection in Bengal is more subacute than florid. 'Climate appears to be an important factor in the incidence both of the rheumatic infection and of the rheumatic type of heart disease. For example, in Boston at the Peter Bent Brigham Hospital

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the incidence of rheumatic fever in the years 1914 to 1923 was 1.85 per cent of all medical admissions, the clinical incidence of mitral stenosis was 3.89 per cent, and the incidence of mitral stenosis in the autopsy room was 4.68 per cent, while in New Orleans at the charity hospitals these percentages from 1916 to 1923 were 0.3, 0.08, and 0.23, respectively, and at Baltimore at the Johns Hopkins Hospital from 1914 to 1922, 0.73, 2.01, and 1.30, respectively' (White, 1937).

Clinical diagnoses in the 351 *cases of rheumatic heart disease treated at the Medical College Hospitals, Calcutta*

Mitral stenosis	120
Mitral incompetence	15
Double mitral	70
Mitral and aortic valve disease ..	25
Double aortic	4
Rheumatic carditis	107
Rheumatic aortic incompetence ..	4
Rheumatic pericarditis	6
	351

I have deliberately omitted a statement of the sex incidence, as in view of the purdah system the figures would necessarily be misleading. The criteria given in the *Nomenclature and Criteria for Diagnosis of Diseases of the Heart,* 1939, should be more generally employed in India for the classification and diagnosis of heart disease. Diagnostic inexactitudes, such as 'double mitral' and 'double aortic', should be abandoned: surely one of the lesions is predominant.

Incidence of aortic stenosis—clinical group

Calcareous disease of the aortic valve was diagnosed clinically in two of 1,832 cases of cardiovascular disease admitted to the Medical College Hospitals, Calcutta, from 1930 to 1939. McGinn and White (1934) of the Massachusetts General Hospital reported 113 cases of aortic stenosis (2.3 per cent) in 4,800 cardiac patients. Campbell (1937) observed that out of every six rheumatic cases three had aortic incompetence and mitral stenosis, the fourth had aortic stenosis as well, and the fifth and sixth were clinically without mitral disease, one having aortic incompetence alone and the other aortic stenosis and incompetence. A case of aortic stenosis and incompetence with mitral stenosis is described later in this paper.

Incidence of aortic stenosis—post-mortem series

McGinn and White (1934) reported 123 cases of aortic stenosis (1.8 per cent) in 6,800 autopsies of all types of disease. Of these 123 cases, 86 had calcareous changes in the aortic valve and 37 had aortic stenosis without calcareous changes: the majority of the former group were over 50 years of age, whereas the vast majority of the latter were under 50. The subjects of calcareous disease of the aortic valve are notoriously prone to sudden death. Hence calcific aortic heart disease is not infrequently encountered as a medico-legal cardiac case. Our post-mortem material consists of a total of 1,995 cases of all types of disease autopsied in the period 1915 to 1937. I am grateful to Dr. M. N. De, lately Professor of Pathology, for the connected records, which suggest that aortic stenosis is practically non-existent in Bengal. Incidentally, if we are to develop a precise and scientific knowledge of heart disease in this country we must wherever possible supplement our clinical cardiology by pathological studies of our cardiovascular diseases and anomalies. Useful guidance may be bad from the section entitled 'An Outline for the Pathological Diagnosis of Cardiovascular Diseases and Anomalies', in the New York Heart Association's standard work mentioned above. Seven international authorities on cardiovascular pathology are responsible for this section.

Rheumatic aortic stenosis

'As a rule, aortic stenosis is merely one element in rheumatic heart disease. It may be accompanied by aortic regurgitation, mitral defect, and myocardial and pericardial disease. In the pathogenesis of heart failure in such cases, aortic stenosis most often plays only a subordinate rôle. Actually, as a previously leaky valve narrows, the added work thrown on the left ventricle by the stenosis may no more than substitute for that spared the chamber by the diminution in regurgitation due to the constriction of the aortic aperture' (Fishberg, 1937). Aortic valvulitis, according to Horder, is particularly common in the endocarditis occurring during scarlet fever, a specific fever which I have never encountered in the tropics. Possibly school medical officers in certain parts of India may have some experience of it. Gallavardin's own rheumatic type of aortic stenosis in young subjects is probably congenital in origin rather than a subacute endocarditis of unknown ætiology. Miller advises us that aortic stenosis due to rheumatism is practically unknown in children. Signs of aortic stenosis in a child should arouse a suspicion of a congenital heart lesion or of malignant endocarditis.

Calcific aortic stenosis

Priority of description of calcific aortic stenosis belongs to Bonetus, who in his *Sepulchretum,* 1679, the early forerunner of Cabot's *Differential Diagnosis,* 1911, presented the case of the robust middle-aged Parisian tailor who dropped dead in the street and showed calcified stenosed aortic valves on autopsy. In 1931, Christian, the doyen of American cardiologists and the *guru* of Levine and others, wrote a significant paper on aortic stenosis with calcification. In this landmark in the study of aortic stenosis Christian not only concluded that calcific aortic stenosis was of rheumatic origin, but he also adumbrated the finding of calcification of aortic valves radiologically. In 1933, Sosman and Wosika described a fluoroscopic procedure whereby calcific aortic leaflets can be

visualized during life. Our interest in calcific aortic stenosis was immediately and enthusiastically revived.

Evidence of the rheumatic origin of calcific aortic stenosis

From a critical analysis of 200 cases of calcific aortic stenosis studied at necropsy, Clawson *et al.* (1938) decided that this lesion was invariably due to rheumatic infection. The following observations furnished the basis of their conclusion. The incidence of a rheumatic history and of stigmata of previous rheumatic infection in calcific aortic stenosis approximated that of other healed rheumatic deformities of the valve. The stigmata of previous rheumatic infection included deformities of the mitral or other valves, adherent pericardium, the presence of Aschoff nodes in the myocardium, a definite inflammatory reaction in 68 per cent calcified aortic cusps and aortic-valve vascularization in 95 per cent of cases. We know that blood vessels do not exist in human heart valves and that valve vascularization is secondary to rheumatic valvulitis (Gross and Friedberg, 1936, and Gross, 1937). 'That deposition of calcium', remarks Willius, 'should occur in a region of low vascularity, from which inflammatory products cannot be absorbed adequately, is one expression of the phenomenon of calcification in general'. 'The leaflets of the heart valves', continues Willius, 'satisfy this set of circumstances perfectly'. Cholesterol crystals, so commonly seen in atherosclerosis, were not seen by Clawson in any of the calcified aortic valves. The gross structure of the aortic valves in calcified valve deformity was, in Clawson's opinion, in all respects similar to the structure of the calcified mitral valves sometimes observed, and not at all characteristic of an atherosclerotic process. Atherosclerotic thickening of the aortic cusps is further negatived, according to Clawson, by the remarkable smoothness of the aorta in the subjects of calcific stenosis. He suggests that the stenosed aortic valves, diseased from early life, buffer the systolic impact of the aorta and thus protect the ascending aorta. Willius' paper is on somewhat similar lines to that of Clawson's in the matter of evidence. They both stress the frequency of a rheumatic history and of stigmata of rheumatic infection, which they think is too great to be regarded merely as a casual and not a causative factor. Willius believes 'that rheumatic infection which culminates in calcareous disease of the aortic valve differs only in a quantitative manner and not in any qualitative manner, from other types of rheumatic carditis'. Other advocates of the rheumatic ætiology of calcific aortic stenosis are Christian (1931), McGinn and White (1934), Contratto and Levine (1937), and Boas (1935).

Other views

Friedberg and Sohval (1939) do not preclude a non-rheumatic form of the disease. Margolis, Zeillesson and Barnes (1931) and others hold that in some cases the lesion is the result of a non-inflammatory degenerative process. Cabot's idea (1926) that calcareous disease of the aortic valve represents the healed stage of subacute bacterial endocarditis is, according to Willius, another intriguing but unsupported supposition. Libman suggests that congenitally bicuspid aortic valve may furnish the basis of some cases. Finally, it is to be remembered that syphilis is never a cause of aortic stenosis. About 30 cases of combined syphilitic aortitis and rheumatic disease of the heart have been reported up to date.

Symptomatology

'The only symptoms of aortic valve disease are the tendency to faintness, dizziness, or even syncope in patients with marked aortic stenosis' (White, 1937). We shall here confine ourselves to calcific aortic stenosis : the symptoms encountered in a case of aortic stenosis in young subjects are recounted later in this paper.

Calcific aortic stenosis is remarkably well borne by the left ventricle for many years. It is amenable to faultless compensation by the substitution of hypertrophy for dilatation at each stage, not only because of the inherent gradualness of the narrowing process but also because aortic and coronary sclerosis are likely to occur in inverse proportion to the degree of stenosis of the aortic valve. Eventually, the left ventricle yields behind the obstructed aortic orifice. The resultant effort-dyspnœa is often the earliest and may be the sole symptom for years. Cough due to chronic pulmonary congestion is commonly complained of and 'bronchitis' is often misdiagnosed, because of the patient's age and the absence of systemic œdema. Hæmoptysis occasionally occurs. Later, paroxysmal dyspnœa may gravely distress the patient at night. The progress of heart failure in aortic stenosis is relentless. Finally, right-sided heart failure appears and death from cardiac failure, which is the commonest mode of termination of aortic stenosis, follows in about six months. The specific symptomatology of calcific stenosis, dizziness, syncope and angina pectoris, commonly occurs in the midst of these distressing symptoms of heart failure, and so may be displaced to the background of the clinical picture and pass unrecognized unless direct inquiry is made. The para-dyspnœic angor of Gallavardin, or the sense of intense oppression in the chest sometimes amounting to actual pain associated with severe paroxysmal dyspnœa, must be clearly distinguished from true angina pectoris. 'Angina of decubitus' usually does not occur in aortic stenosis of calcific type. The not infrequent confusion of cardiac asthma with angina pectoris is ably discussed by Bedford (1939) in his excellent account of left ventricular failure in the Strickland Goodall Memorial Lecture.

Angina pectoris

About half the cases of calcific aortic stenosis exhibit angina pectoris. The mechanism of its production is myocardial ischæmia due to coronary insufficiency. For purposes of simplicity and clarity, I shall endeavour to summarize briefly the various explanations for this coronary insufficiency, ably advanced by Harrison (1939), Green (1936), Fishberg (1937), Contratto and Levine (1937) and Friedberg and Sohval (1939).

When the aortic orifice is stenosed, thus obstructing the emptying of the left ventricle, the intraventricular pressure rises and the ejection velocity of the blood is greatly enhanced in order to maintain a given volume flow per unit of time. Now, the elevated intraventricular systolic pressure compresses the peripheral coronary vessels after the fashion of the blanching of a tightly-clenched fist and perhaps the accelerated blood flow past the coronary orifices produces suction of blood from the coronary vessels. Thus, coronary inadequacy may be present even at rest. Usually, however, this is more notable on exertion because the left ventricle, operating against the already extremely high intraventricular pressure, finds it difficult if not impossible further to augment the force of its contraction and the aortic pressure requisite for exertion. Increased heart size greatly intensifies such relative coronary insufficiency. The concentric hypertrophied heart of aortic stenosis is more appropriately described as 'heavy heart' than in terms of heart size. Verification of the theory of coronary insufficiency in these cases is frequently afforded by the coronary-thrombotic-like pattern of the T-wave and RST segments, which is in keeping with the clinical picture. The frequent presence of angina pectoris in 'pure' aortic stenosis, that is, without clinically evident aortic regurgitation, promptly dismisses aortic insufficiency as a causal factor of anginal pain in these cases. The occurrence of coronary vaso-constriction in calcific stenosis, with resultant coronary insufficiency, is purely hypothetical. In hypertensive arteriosclerotic heart disease subjects, the situation of coronary pain is not uncommonly atypical.

Dizziness, fainting and syncope in calcific aortic stenosis

These symptoms tend to occur in those aortic stenotics that suffer from angina pectoris as well. And like angina pectoris, attacks of faintness or downright syncope are complained of, mostly on exertion. Harrison attributes the syncopal attacks of calcific aortic stenosis to sudden diminution of the cardiac output, because of the aggravation by exercise of a pre-existing myocardial oxygen deficiency, with resultant cerebral anoxia. It may be extremely difficult to differentiate calcific aortic stenosis from coronary artery disease (arteriosclerotic heart disease). Both conditions are due to a common factor, namely, coronary ischæmia, with the result that certain clinical and electrocardiographic features are common to both. Thus, they may both cause angina pectoris, cardiac failure and sudden death, and both may show conduction disturbances and T-wave changes electrocardiographically. In coronary arteriosclerotic subjects, however, syncope on effort fails to occur because, according to Harrison (1939), their myocardial anoxia is focal, whereas such syncope is a classical symptom of calcific aortic stenosis wherein myocardial anoxæmia is diffuse. A history of dizziness and syncope in an elderly man should therefore suggest the possibility of aortic stenosis rather than arteriosclerotic heart disease. The finding of an aortic systolic murmur and thrill would render the differentiation from coronary artery disease clear. In all doubtful cases the aortic valves should be carefully scrutinized fluoroscopically for evidence of calcification. Levine (1936) and others attribute the tendency of calcific aortics to faintness and syncope to hypersensitivity of the carotid sinus reflexes.

DIAGNOSTIC CRITERIA

In 1934, McGinn and White advocated a considerable broadening of the previously rigid diagnostic criteria of aortic stenosis, which they concluded we were more often missing when it was present, than diagnosing when it was absent. In the opinion of Willius, the diagnostic criteria had not been recognized by the clinician until relatively recent years. Willius has closely studied aortic stenosis for many years and in such of his cases as failed to satisfy the old-time requirements necessary for a clinical diagnosis of aortic stenosis he sought radiological demonstration of calcification of the aortic leaflets. Truly 'the eye often misses what is not in the observer's mind but sees what it looks for'—Horder.

Before 1934

In his *Medical Notes,* Horder (1921) writes regarding a systolic murmur heard at the aortic base—'the first thing to say (to oneself) about a systolic bruit heard at the aortic base is that the case is probably not one of aortic stenosis. More likely causes of the bruit are . . . But if, in addition to the presence of a systolic aortic bruit, the following features are also made out during the examination—

(1) good conduction of the bruit towards the right side of the neck;

(2) considerable hypertrophy of the left ventricle;

(3) systolic thrill in the second right interspace; and

(4) a small pulse—

it may be said with confidence that the patient suffers from aortic stenosis. If to these findings were added—

(5) diminution or absence of the aortic second sound; and

(6) an aortic diastolic murmur—
the most fastidious physician of previous decades would probably have registered his complete approval of Horder's excellent criteria. Incidentally it may be noted that five signs, namely, aortic systolic murmur, aortic diastolic murmur, diminished or absent aortic second sound, aortic systolic thrill, and calcification of the aortic valve on fluoroscopic examination in calcific stenosis, emanate directly from the deformed aortic cusps.

Since 1934

A fair sample of British and American requirements for the clinical diagnosis of aortic stenosis is given respectively by Campbell (1937) and by White (1937).

Concerning the diagnosis of aortic stenosis, Campbell states 'It is not so simple as that of aortic incompetence, although in cases of high-grade aortic stenosis it may at once be suggested by the feel of the pulse, both the slow rise and the sustained plateau being readily appreciated by the finger. A systolic murmur is often present as well as diastolic and may indicate nothing more than some roughening of the valves. Whenever there is a systolic murmur, a special search should be made for a systolic thrill and the rougher the murmur the more confidently may it be expected'.

'The combination of aortic stenosis and incompetence', he continues, 'is much commoner than pure aortic stenosis. It is sometimes said that only the expert should diagnose aortic stenosis in the absence of aortic incompetence, but the intention of this warning can be expressed better by the following statements. In the absence of aortic incompetence, stenosis should never be diagnosed unless there is a systolic thrill as well as a murmur, or the characteristic pulse of aortic stenosis or both.

In the presence of aortic incompetence, stenosis may be suspected even without a thrill if there is a very rough systolic murmur, especially if the pulse is less characteristic of aortic incompetence than would be expected from the loud diastolic murmur. The blood pressure should always be taken as a measure of the degree of stenosis and the pulse in both arms should be taken as a routine to detect the slighter cases of coarctation of the aorta'.

White's statement of the American attitude is as follows—'The triad of murmur, thrill and small pulse', he says, 'is the essential finding : the other findings are corroborative. It is not necessary to wait for an aortic systolic thrill or a plateau pulse to make a diagnosis of aortic stenosis : the diagnosis can be made on the systolic murmur alone in a patient without aortic dilatation or hypertension provided the murmur is loud and harsh. Accuracy of diagnosis has increased greatly in our hands since we have made this change in the diagnostic criteria of aortic stenosis'.

The essential criteria defined by Willius as diagnostic of calcareous stenosis of the aortic valve are as follows :—

'(1) A loud rough systolic murmur over the base of the heart conducted into the vessels of the neck and in many instances over the præcordium.

(2) The second heart tone is absent or diminished in intensity.

(3) It is replaced by a soft blowing diastolic murmur where there is associated aortic regurgitation.

(4) A thrill is usually palpable over the upper part of the sternal region.

(5) Evidence of cardiac hypertrophy usually can be elicited.

(6) Radiological examination reveals the presence of deposits of calcium within the aortic cusps or annulus'.

BASAL SYSTOLIC MURMURS

Murmurs are obtrusive and impressive signs, and, if the physician is uncertain of their origins, he necessarily lacks self-assurance and diagnostic discrimination. While pulmonary systolic murmurs are commonly due to physiological factors and mildly pathological states, aortic systolic murmurs frequently own a definitely pathological basis of varying significance. The aorta is thicker, less elastic and much longer than the pulmonary artery, hence the aorta offers more resistance to distension and at the same time affords accommodation for a larger stroke volume than does the pulmonary artery. Therefore the increased cardiac output and heightened systolic force of the overactive heart (of excitement, anæmia, fever, neurocirculatory asthenia, thyrotoxicosis and early hypertension) are seldom sufficient to produce appreciable dilatation of the aorta. On the other hand, the resultant physiological dilatation of the soft, thin, easily dilatable short pulmonary artery in the same hyperkinetic circumstances is usually responsible for the pulmonary systolic murmur, the commonest of all heart murmurs, heard in Balfour's 'area of pulmonary romance'. (The right lung apex behind is another area of auscultatory romance.) Dilatation of the pulmonary artery and of its 'ring' would of course result in relative pulmonary incompetence : dilatation of the aorta and its 'ring' would likewise produce relative aortic incompetence.

Causes of aortic systolic murmur

I. Relative stenosis due to—

(1) Cardiac overaction, *e.g.,* in anæmia systolic murmurs are heard most frequently in the pulmonary area, next most commonly in the mitral area, the tricuspid area and the aortic area, in the order named.

(2) Simple dilatation of the aorta due to arteriosclerosis, chronic hypertension and syphilitic aortitis.

(3) Aneurysm usually of the ascending aorta.

II. Conditions favouring relative stenosis by producing kinking and so a tendency to proximal dilatation of the aorta, *e.g.*, high diaphragm, as in extreme obesity, pregnancy, ascites and other forms of abdominal distension.

III. Absolute stenosis due to—

(1) Monckeberg's sclerosis of the aortic valve without evident aortic dilatation, productive of slight aortic stenosis in some elderly people.

(2) Aortic stenosis due to rheumatic valvulitis or calcification of the valves (rheumatic and non-rheumatic calcific aortic valves).

(3) Congenital aortic stenosis productive of aortic dwarfism.

IV. Transmission of a systolic murmur from elsewhere, *e.g.*, pulmonary area, mid-sternum, lower sternum, apex.

The systolic murmur of aortic stenosis

Acoustic characters.—The systolic murmur of aortic stenosis is characteristically loud and harsh. It may be rough, coarse, grating, croaking, vibratory, whistling, musical or even blowing. Rough murmurs bespeak stenosis, while rasping or tearing sounds often characterize the louder varieties of murmur associated with calcareous deposits on the valve, or marked stenosis. The murmur is apt to be very loud in recumbency. The loudness of the murmur is maintained until the grave heart failure of aortic stenosis supervenes, whereupon the murmur becomes less intense and may even disappear. The widespread transmission of very loud aortic systolic (stenotic) murmurs over the chest, back, vertex of head, sacrum and humeral condyles and their very occasional audibility, even at a distance from the patient, seems to support Cabot's view that 'the distance a murmur is transmitted is purely a function of its loudness'. Hence the 'loud and harsh aortic systolic murmur' postulated by White generally implies an obstructive stenotic murmur well conducted into the neck. Loud mitral stenotic murmurs are almost invariably confined closely to the patient's cardiac impulse because in diastole the apex is not firmly applied to the chest with the result that the vibrations are rapidly lost (Cowan and Ritchie, 1935). Thus mitral stenotic murmurs fail to endorse Cabot's theory of the transmission of murmurs.

Timing

In aortic stenosis both the isometric and ejection phase of systole are prolonged. Sound records show that the isometric component of the first sound is clear of murmur provided that the disease process is limited to the semilunar valves. They also show that the murmur of aortic stenosis attains its maximum intensity in the phase of maximal ejection. Clinically, the loud and harsh murmur of aortic stenosis is early systolic in point of time, commonly masks the first sound and frequently extends throughout the whole of systole.

Punctum maximum

This corresponds with that of the accompanying thrill, namely, the clinical aortic area. The murmur may, however, be best heard over the manubrium sterni or even to the left of the sternum. Rarely, the cardiac apex is the site of maximum intensity of the harsh stenotic murmur, in which case mitral regurgitation is simulated as it may be to a less extent when a loud aortic systolic murmur is heard all over the præcordium. In both instances, transmission of the murmur to the lung bases behind, significant of mitral regurgitation, is notably absent. Moreover, mitral regurgitant murmurs are not transmitted to the base of the heart, nor are they audible in the neck.

Selective propagation

The transmission of the aortic systolic murmur of stenosis is characteristically widespread, because its vibrations are conveyed along the blood vessels in the direction of the blood current and also through the left ventricle, and because the louder it is the greater is its distance of transmission in accordance with Cabot's law, as stated above. Thus the murmur may be well conducted along the carotid and subclavian vessels into the neck and along the arms, it may be heard all over the front of the chest, and when it is heard in the back its point of maximum intensity precisely corresponds with the first point of contact of the aorta with the spine, namely, the fourth thoracic vertebra.

The transmission, however, of an aortic systolic murmur even into the neck is not pathognomonic of true stenosis: indeed, it is more frequently indicative of dilatation of the aorta with roughening of the intima. An interesting feature of aortic systolic murmurs is their tendency to 'tunnel', *i.e.*, 'to travel some distance underground and emerge with a change of quality'—Clifford Allbutt. 'The murmur of aortic stenosis', remarks Cabot, 'is often heard well at the apex and at the aortic area and faintly in the intervening space, probably owing to the interposition of the right ventricle'. Levine, who followed up many instances of such 'systolic murmurs—cause unknown', observed, 'as years went on a definite basal thrill would become palpable or calcification of the valve would be found on *x*-ray examination'. 'Dyspnœa or anginal pain', he adds, 'were frequent eventual developments in such patients'.

THE THRILL

In the matter of physical signs in clinical medicine, not only is it vital to know them thoroughly and the precise technique of their elicitation but also to know when precisely to elicit them. Obviously, time for one thing does not permit a practitioner to perform all the tests in his repertoire every time he sees a new patient. With reference to aortic stenosis, Lewis aptly remarks that 'a harsh murmur is the hint to examine for thrill'. A thrill is the analogue of

the murmur, the conditions necessary for its production being more exaggerated as a rule. In fact, low-pitched murmurs are generally palpable as thrills. The essential requisite for thrill production is a sudden change in the calibre of the blood stream from narrow to wide. The most intense systolic thrills are those of aortic stenosis and pulmonary stenosis. Intensifying factors are a thin chest, closeness of the occurrence of the thrill to the palpating hand, and speed of blood flow. A common associate of calcific aortic stenosis is pulmonary emphysema, which causes recession of the heart from the chest wall. The force of the thrill varies with the intensity and pitch of the murmur. Hence a loud and harsh aortic systolic murmur is attended by a thrill, and if the murmur is a gentle one no thrill at all is felt. Thus, if an aortic systolic murmur is not fairly loud, generally it may be ignored. It is important to remember that when heart failure supervenes in aortic stenosis the thrill, like the murmur, becomes faint. The fainter thrills of aortic stenosis are often missed owing to faulty technique. For their detection it is necessary to employ light palpation with the patient sitting up and leaning forward, particularly with his breath held in expiration, whereby the sternum and adjacent ribs come into closer contact with the aorta, and the intervening lung border is retracted. One should carefully search for thrill in this fashion in the second right interspace, and over the upper and middle portions of the sternum. Occasionally the thrill may be felt at the cardiac apex, where it may very rarely be better appreciated than at the base. If in doubt about a thrill, the answer is 'thrill is absent'. Definite sustained purring vibrations must be felt before the observer can declare that thrill is present. An overacting heart throws the ribs, and more especially rigid ribs, into vibrations with the production of a 'pseudo-thrill', in which case, if one separates the fingers and places them in the intercostal spaces, the osseous vibrations will not be felt. The transmission of the aortic systolic thrill is similar to, but less widespread than, that of the corresponding murmur, and is likewise non-pathognomonic of aortic stenosis. A systolic thrill maximal in the aortic area is an indisputable sign of aortic stenosis, provided the other causes of thrill to the right of the upper end of the sternum, more especially those due to relative stenosis, *i.e.*, aortic dilatation and congenital heart disease, are excluded. The rarer causes of thrill to the right of the sternum are :—aneurysm of the ascending aorta or of the commencement of the aortic arch, aneurysm of the innominate or of the right subclavian artery, arterio-venous aneurysm (ascending aorta and superior vena cava) and compression of the aorta or its great branches by a mediastinal tumour or other factor. The thrill of arterio-venous aneurysm is continuous rather than simply systolic, especially over the site of perforation, *i.e.*, in the area about the aortic cartilage. Kurtz has described the occurrence in the supersternal notch of 5 to 6 visible vibrations with each systole of the heart in cases of well-marked thrill due to rheumatic aortic stenosis.

The aortic diastolic murmur

An aortic diastolic murmur is the rule in rheumatic aortic stenosis, whereas it is an inconstant accompaniment of calcific aortic stenosis. The post-mortem evidence of calcific stenosis clearly suggests that leakage is inevitable, nevertheless clinical evidence of such leakage is not infrequently wanting, which reminds us that the murmur of mitral regurgitation is often conspicuously absent in fully developed mitral stenosis. In either case, the louder one murmur becomes the less loud is the other; that is to say, the greater the stenosis, the less the regurgitation. At all events a deliberate search should be made for a basal diastolic murmur in all cases of suspected aortic stenosis. A soft and distant aortic diastolic murmur is notoriously elusive. Such a murmur may be heard best after exercise with the patient standing or sitting and leaning a little forward, which manœuvre brings the aortic valves nearer the chest wall. We then listen attentively, employing a Bowle's chest-piece or direct auscultation with the naked ear, especially when the patient holds his breath in full expiration. The aortic diastolic murmur commences abruptly with, and may somewhat blur, the second sound; it quickly attains its maximal intensity during the first moment of diastole and is both less intense and less prolonged than its dominant partner, the loud, harsh and prolonged systolic murmur of aortic stenosis. In aortic stenosis, a maximal intensity and more clearly waterfall character of the diastolic murmur in the left third interspace close to the sternum, *i.e.*, approximately over the aortic valve itself, is highly significant of a rheumatic ætiology and of an undilated aorta. A loud aortic diastolic murmur should lead one to interpret a high intra-aortic pressure, the proof of a vigorous left ventricle, and a correspondingly less excessive leakage. 'The degree of aortic incompetence', Lewis reminds us, 'cannot be gauged from the loudness or length of the murmur'. A loud musical aortic diastolic murmur promptly excludes aortic stenosis in that such a murmur is usually due to retroversion or eversion of the right anterior aortic leaflet, produced by syphilitic involvement : a tear or rupture of an aortic valve is less commonly responsible for this loud musical murmur. The 'double aortic', beloved of students, is a finding of purely localizing value in that it implies nothing further than disease of the aortic valves. The confirmatory value of an aortic diastolic murmur in a case of suspected aortic stenosis is considerable in the absence of Corrigan's pulse, of alteration in the size and contour of the aorta and of evidence of syphilis.

The aortic second sound

The careful determination of aortic second sound intensity is regarded by some observers as being of more importance in the diagnosis of aortic stenosis than the study of the pulse. The classical quartette of signs of aortic stenosis, however, is the loud, harsh aortic systolic murmur, the corresponding thrill, the diminished or absent aortic second sound and the plateau pulse. The aortic second sound and the pulse changes being usually difficult of interpretation are relegated to the third and fourth positions.

The second sound, which definitely precedes the opening of the mitral and tricuspid valves, gives acoustic expression to the vibrations set up in the semilunar valves at the moment of their closure and also on the wall of the artery and in the blood column itself (Orías and Braun-Menéndez, 1939). In classical aortic stenosis, the aortic second sound is weak or absent because the stiff and rigid valve segments are incapable of snapping together. Total absence of the aortic second sound documents gross stenosis. Elsewhere a second heart sound of subnormal intensity is easily audible. Even in well-marked aortic stenosis, the second sound is commonly heard at the aortic base and is due to the closure of the pulmonic cusps. Definite aortic stenosis can be present with a normal aortic second sound. The second sound, as heard at the aortic area, is the summation of the second sounds, originating in the aorta and in the pulmonary artery. Hence, when studying the aortic stenosis, it is advisable to auscultate the carotid artery also, where the aortic component of the second sound is isolated. Factors germane to consideration of aortic second sound intensity in aortic stenosis are the degree of stiffness of the aortic cusps, the presence or absence of an aortic diastolic murmur, the level of the systemic blood pressure, and the condition of the aorta. Extrinsic factors, such as obesity and emphysema, must of course never be overlooked in the evaluation of heart-sound intensity. A co-existent aortic diastolic murmur beginning abruptly in early diastole will somewhat blur the aortic second sound.

Lewis discusses accentuation of the aortic second sound in hypertension, wherein an accentuated aortic second sound is an expected, but by no means an invariable, finding. Obesity or emphysema may be responsible for its absence on occasions. Stiffness and enlargement of the aorta impart a slightly musical quality to the aortic second sound, hence an intoned or ringing aortic second sound suggests changes in the aorta rather than in the aortic cusps. Thus, in hypertension when the aortic second sound is reduplicated and ringing in quality (*bruit de tabourka*), the ringing quality is to be ascribed to sclerosis and calcification of the aorta and not to the hypertension. In aortic sclerosis without hypertension, the aortic second sound may be greatly accentuated and ringing, owing to the close approximation of the sclerotic unfolded aorta to the chest wall. Otherwise stated, an aortic second sound with a characteristic snappy emphasis, comparable to the note of a small drum, is highly significant of aortic dilatation, especially if associated with normal blood pressure. As a general rule, therefore, state Norris and Landis (1933), if the aortic second sound is normal and more especially if it is loud and ringing in character, the systolic murmur heard at the aortic base originates in the aorta and is not due to rigid and diseased aortic valves. In calcific aortic stenosis the first part of the aorta is often remarkably free of atheromatous changes, owing to buffering by the calcific valves. Cabot's remark concerning the aortic second sound in certain cases of aortic stenosis is at least stimulating.

'Another strange fact is that even in a case with rigid and immovable valves the aortic second sound may not only be audible but may be actually accentuated and the systolic blood pressure high. How physiologists would account for this I do not know. So far as I see, it definitely attacks the theory, ordinarily held by physiologists, that the aortic second sound is due to the closure of the aortic valves and to this cause alone.'

We do not blame our physiologists for this apparent discrepancy.

THE PULSE

The study of the pulse by palpation and tracings has greatly declined since the introduction of the baumanometer and the electrocardiograph. These instruments are of course infinitely more accurate in their assessment of pulse quality and rhythm : the rate being best obtained by auscultation of the heart. Nevertheless, it appears from Dudgeon's writing (1882) that the nineteenth-century physician armed with his ' loud-ticking gold chronometer ' was at least more mysterious and consequently a greater oracle in his day.

'The physician of old made his diagnosis chiefly by observation of the pulse and tongue. But as the tongue could be rapidly inspected, and anyone could judge of its foulness or cleanness as well as himself, he concentrated his attention mainly on the pulse, in the feeling of which there was always scope for affecting the possession of peculiar skill and insight. To the uninitiated, who regarded the doctor as a depository of occult knowledge, and who received his dicta as though they were oracles, there was something very imposing in his method of pulse palpation. The fingers of the right hand daintily grasping the patient's wrist, while the doctor's eyes are riveted on the loud-ticking gold chronometer he held in his left hand, his head gravely nodding the while synchronously with the arterial pulsation—all this formed a picture calculated to inspire beholders with reverence and awe.'

' The outstanding diagnostic sign of aortic stenosis is a small pulse, rising slowly to a delayed summit . . . Aortic stenosis should never be diagnosed without this sign '—Lewis. The characteristically slow, small, retarded pulse (*pulsus rarus, parvus, tardus*) is the peripheral expression of ' pure ' aortic stenosis of high-grade severity. Its wave conveys to the finger the impression of a gradual rise to a summit which

is unduly prolonged in the form of a plateau. The sustained plateau is best appreciated by palpation with the approximate fingers simultaneously, when the pulse wave will be felt to take time to pass the palpating fingers, like a crowd going through a door. Left ventricular hypertrophy may compensate for the stenosis, and so the size of the pulse may be approximately normal. Whereas the plateau pulse feels full between the beats, Corrigan's pulse feels empty. The slow rise bespeaks obstruction to the ejection of blood from the left ventricle into the aorta due to the absolute narrowing of the valvular orifice. Anacrotism, or the presence of an additional wave on the upstroke of the pulse, marks the transition from a rapid to a slower ejection by the left ventricle.

The causal factors of this sudden change of ventricular tactics are not yet precisely known. The valvular obstruction and perhaps coincident arteriosclerosis are probably responsible. The anacrotic pulse is more diagnostic of aortic stenosis than the *pulsus bisferiens*, or the double pulse beat, but it is not pathognomonic of stenosis in that it is also observed in conditions of obstruction distal to the aortic valve, *e.g.*, aortic aneurysm, compression of the radial artery by a tumour proximal to the site of palpation, severe arteriosclerosis, etc. Both the anacrotic pulse and the *pulsus bisferiens* are usually more evident in pulse tracings than they are to the palpating finger. Another feature of the classical pulse of aortic stenosis is delay, *i.e.*, increase of the normal interval between the cardiac apex and the radial pulse to perhaps one-fifth of a second, which increase is most evident on simultaneous auscultation of the heart and palpation of the radial pulse. This retardation of the pulse is connoted by *tardus*. The heart rate in aortic stenosis is often remarkably slow, even 50 to 60 beats per minute, in the absence of heart block and digitalization.

Harrison (1939) suggests that the carotid sinus is reflexly concerned in the prolongation of systole common in these cases and that the duration of diastole is correspondingly lengthened, which conditions tend to delay the onset of the next heart beat. Levine remarks—'the slow heart rate may possibly be due to the same factor that makes the patient subject to syncope, or may in some way be related to the vagus apparatus'. However it may be, the bradycardia of aortic stenosis maintains a satisfactory diastolic pressure, thus obviating anginal seizures, which tend to occur with higher heart rates. Most patients with congestive failure exhibit tachycardia and may have arrhythmias. In aortic stenosis, however, the heart rate, even in advanced congestive failure, may be under 70 or 60 in the absence of heart block and digitalis therapy. The rhythm, moreover, is generally regular. Such a congestive failure, or for that matter any congestive failure of obscure ætiology, should always remind one of the possibility of calcific aortic stenosis. The three characteristic pulses of valvular disease of the heart are Corrigan's pulse, significant but by no means pathognomonic of aortic regurgitation, the plateau pulse of aortic stenosis, and the small pulse with firmness found in mitral stenosis. Corrigan's pulse is a popular finding not only in aortic regurgitation but also in conditions of peripheral vaso-dilatation and high pulse pressure, *e.g.*, Graves described it in hyperthyroidism, it may be a conspicuous feature of certain anæmias, it is a common feature of neurocirculatory asthenia and during fevers : it may be observed in some cases of hypertension.

For the occurrence of Corrigan's pulse in aortic regurgitation, the leak must be appreciable and the left ventricle must be fairly powerful. Hence in cases of slight leakage, widely-gaping aortic orifice, or of left ventricular failure, Corrigan's pulse is absent or indeterminate. Its form is the antithesis of the plateau pulse and both aortic stenosis and mitral stenosis definitely modify it. The classical plateau pulse is not a feature of the majority of the cases of aortic stenosis. The young rheumatic group, for example, is characteristically polyvalvular and so the pulse is the resultant of aortic stenosis, aortic incompetence, and perhaps of mitral stenosis. Aortic stenosis tends to lessen both the rise in systolic pressure and the fall in diastolic pressure due to aortic regurgitation. Mitral stenosis modifies the signs of aortic regurgitation in much the same way. In the calcific group, in addition to the usual presence of aortic leakage, one not infrequently finds a complicating hypertension, the classical pulse of which is described as '*magnus, durus, tardus*'—large, hard and slow.

Other associates of the elderly group are arteriosclerosis and renal disease. Both groups may exhibit a superimposed neurocirculatory asthenia. Obviously the pulse, like the aortic second sound, is often difficult of correct interpretation. 'Indeed I have in two cases observed a well-marked "Corrigan" pulse in life, and been confronted post mortem with a narrowed, rigid aortic valve!' Cabot's perplexity is eloquent.

The cardiac impulse

The characteristic cardiac impulse of gross aortic stenosis is large, rises gradually, is overlong sustained and leads to a slow, deliberate and well-defined displacement of the thoracic wall, downwards and slightly outwards. Briefly, it displays increased force and a slow and deliberate out-thrust. 'The out-thrust of the apex beat may be even slower and longer than in the case of hypertrophy—"slow heave", which is sustained'—Price (1937). It is reminiscent of the slow steady heave of a bullock starting to drag a heavy bullock cart. Libman suggests that the contrast between the slow heaving cardiac impulse and the absence of retromanubrial pulsation may afford a clue to the recognition of

aortic stenosis. Broadbent describes the cardiac impulse of aortic stenosis as 'a well-defined and deliberate out-thrust of no great violence'. The distinctive impulse should be compared with the delayed flat-topped pulse wave of small or average volume. When heart failure supervenes, the slow heaving impulse of aortic stenosis becomes more diffuse. The combination of a feeble first heart sound and a heaving impulse is as significant of cardiac failure as it is striking to the observer.

The first heart sound

Four factors—muscular, valvular, vascular and auricular—contribute vibrations to the first heart sound (Orías and Braun-Menéndez, 1939). For the normal production of the first heart sound mitral and tricuspid valve closure must be prompt and efficient, and strong ventricular muscle must produce a sudden well-marked presphygmic tension preceding the outflow of blood. Two groups of ventricular vibrations are essential for the formation of the first heart sound: the first group, produced by the sudden contraction of the ventricle, is known as the isometric component, and the second group, produced by the opening of the semilunar valves, is called the ejection component. Furthermore, the intensity of the first heart sound varies directly with the rate of rise of intraventricular pressure during systole and does not depend on cardiac output (Wright, 1936). The degree of tension to which the mitral and tricuspid valves are subjected at the beginning of systole, is the other principal factor determining the intensity of the first heart sound. Recently, Stead *et al.* (1939) concluded that in normal hearts the position of the auriculo-ventricular valves at the beginning of ventricular contraction is the primary factor in determining the character of the first heart sound. Variations in the P-R interval within normal limits, they observed, may produce striking alterations in the first heart-sound intensity. In aortic stenosis there is obstruction to the outflow of blood and consequently a super-normal intraventricular systolic pressure with prolongation of both the isometric and ejection phases of systole. Hence the first sound at the apex in aortic stenosis is typically booming, *i.e.*, prolonged, low-pitched and resonant, signifying concentric left ventricular hypertrophy. At the aortic base the first sound is commonly masked from its commencement by the loud harsh aortic systolic murmur. Marked prolongation of the first sound in aortic stenosis may be the whisper of an approaching gallop rhythm, indicative of left ventricular failure. Presystolic gallop rhythm is 'the cry of the heart for help'. Not rarely, however, gallop rhythm suggests the presence of bundle branch block, which is sometimes a helpful diagnostic feature of calcific aortic stenosis. The Austin-Flint murmur is interpreted by some as a presystolic gallop rhythm (Laubry and Pezzi, 1926).

Blood-pressure readings

Classical aortic stenosis exhibits a low systolic and relatively high diastolic pressure, *e.g.*, a frequent reading is 110/90. The resultant is a small pulse pressure. We have already referred to the influence exerted by the stenosis on the commonly associated aortic leakage, which influence is less notable in rheumatic stenosis. Reference was also made to the fact that the essential hypertension not infrequently complicates the calcific group. 'To some extent', remarks Willius, 'the height of the blood pressure bears an indirect relationship to the degree of stenosis, although even in extreme degrees of stenosis severe hypertension and all its peripheral associates may occur'.

Speaking of blood-pressure readings, I might mention in passing that John Parkinson constantly insisted that 'there was no such disease as low blood pressure'. 'Usually', said Stroud (1939), 'individuals with low blood pressure can be patted on the back and told that God has been very kind to them. They do not accomplish quite as much as the high pressure individuals, but they are wonderful from the standpoint of the physician. They never feel quite right, they are always coming back to them and they live for ever'. The fat type of Bengalee gentleman is rather more concerned with high blood pressure: indeed he not infrequently displays Musser's syndrome, namely, obesity, hypertension and glycosuria. 'In old age, when the aorta is sclerosed, the diastolic pressure may be relatively low, and such a reading as 200/90 obtained. These readings may not be indicative of true hypertension'—East and Bain (1936).

Electrocardiographic changes

The electrocardiogram in aortic stenosis expectedly reflects left ventricular strain. This is evidenced electrocardiographically by (1) left axis deviation and by (2) changes in the T-wave in lead I or in leads I and II. Disturbances of rhythm may be present also.

Axis deviation

Left axis deviation is the common finding. A large excursion of R in lead II is not uncommon. The differential effect in the two ventricles caused by combined aortic and mitral lesions may be shown by left axis deviation, right axis deviation, or no preponderance. Auricular fibrillation is usually associated either with right axis deviation or with no preponderance of either ventricles. This, in the opinion of Willius, suggests that as long as the left ventricle carries the major strain, the auricles are far less likely to fibrillate than when the reverse is true.

T-wave changes

Inversion of T-wave in leads I and II is due either to factors which act directly upon the muscle or to a change in sequence of invasion as in bundle branch block. In the latter case, the

T-wave changes are associated with QRS changes. In aortic stenosis, with the development of left axis deviation, the T-wave in lead I becomes flattened and finally inverted. Such inversion of the T-wave in lead I may be indicative of the beginning of a not uncommon conduction disturbance in calcific aortic stenosis. I refer to left bundle branch block, which may sometimes be explained in these cases by an extension of the calcific process from the aortic valve to the bundle branch. Inversion of T in leads I and II is common in great enlargements of the left ventricle, when it may signify a relative myocardial ischæmia, due more to the enormous muscle mass increasing the territory of the coronary circuit than to the decrease of the coronary blood supply by the lowered cardiac output of aortic stenosis. T-wave changes in leads II and III suggest the presence of a complicating factor, such as mitral stenosis or pulmonary arteriolar sclerosis, in both of which cases right axis deviation will be in evidence. Otherwise digitalization or coronary infarction may provide the explanation. T-wave changes in all three leads suggest a complicating mitral stenosis or some other less easily discernible factor. A diphasic T-wave in lead I or II of the ∓ type has the same significance as inversion of the T-wave. T-wave inversion tends to lengthen the relative duration of systole. It is much less ominous in aortic stenosis than when associated with grave myocardial involvement.

Diagnosis

The clinical diagnosis of aortic stenosis should be primarily founded upon the secure basis of a fairly loud aortic systolic murmur and its corresponding thrill, as described above. If a diagnosis of aortic stenosis is going to be made by physical signs at all, the murmur and thrill are fundamental signs without which diagnosis will generally proceed uncertainly, until radiological and electrocardiographic aid are invoked. In the *mofussil* such instrumental assistance is rarely available. Amongst the foremost contributors to our present knowledge of aortic stenosis are Christian and his former pupil Levine.

'Without the thrill in addition to the murmur, it is unsafe to make a diagnosis of aortic stenosis. If diagnosis is limited to the cases which present both the thrill and the murmur, the percentage of correct diagnosis will be high. On the other hand, a few cases will be missed for we do see aortic stenosis in which no thrill is produced or even without a murmur. Sometimes this failure to feel a thrill and hear a murmur results in examining the patient only in the late stages of the disease when decompensation is marked' (Christian, 1935).

'The clinical diagnosis of aortic stenosis will in most cases depend on finding a systolic thrill at the base of the heart' (Levine, 1936).

The next step is a diligent search for an aortic diastolic murmur. Its discovery will aid a focal diagnosis of aortic valve disease, and will further suggest that the loud aortic systolic murmur bespeaks absolute stenosis. The intensity of the aortic second sound should then be carefully estimated in the manner described. Its decrement confirms a diagnosis of absolute stenosis. In the absence of a typical plateau pulse, the observer should at least satisfy himself that the pulse is not an unmodified Corrigan pulse. The first heart sound, the cardiac impulse, and the neck and radial pulses should be studied and contrasted the one with the other. The variability of the blood pressure and the connected factors are briefly discussed above. In emphysematous and obese calcific aortic subjects especially, *x*-ray examination is the only precise method of determining heart size, especially left ventricular enlargement and local or general aortic dilatation : the technique of visualization of the calcified valves is described by Sosman and Wosika (1933); left auricular enlargement finally confirms any clinical signs of mitral stenosis. The electrocardiographic findings in the calcific group have been recorded. Those in the rheumatic group likewise add to the completeness of the clinical picture, *e.g.*, the type of preponderance or neutralization, the severity of the cardiac lesions, the progression of carditis, and so forth.

A pure rheumatic ætiology is indicated by the youth of the patient, and a previous history of rheumatism or the presence of mitral stenosis clinically or enlarged left auricle radiologically and the electrocardiographic picture. The calcific group, on the other hand, is over fifty, has a distinctive symptomatology, exhibits conduction disturbances electrocardiographically, and perhaps calcified valves on fluoroscopy. We have already referred, under 'syncope', to the differentiation of calcific aortic stenosis from arteriosclerotic heart disease or coronary artery disease.

Sosman advises us to suspect the possibility of combined rheumatic aortic stenosis and syphilitic aortitis in cases of obscure and bizarre cardiovascular disease in which there is a history of previous rheumatic fever and clinical evidence of syphilis. Clinically, it is usually impossible to establish the diagnosis of such a combination, which occurs more frequently in localities with a higher incidence of syphilitic aortitis. There is, however, no clear evidence as yet that one predisposes the heart to a subsequent infection by the other.

PROGNOSIS

'Rheumatic aortic stenosis', Campbell (1937) tells us, 'is of serious significance in the young subject, its gravity being related to the size of the heart : but if all signs and symptoms are favourable, aortic stenosis is compatible with a good prognosis'.

The subjects of calcific aortic stenosis, and more especially those with angina pectoris, dizziness, syncope, conduction disturbances and marked cardiac enlargement, exhibit a distinct liability to sudden death, sometimes in the midst of their usual health, that is, before heart failure

has finally supervened. In such cases the occurrence of sudden death may be very occasionally explained on a mechanical basis, namely, by locking of the valve by an unusually forceful diastolic recoil thrust, or by thrombotic occlusion of the stenotic aortic orifice, as may happen in very rare cases of mitral stenosis with a ball thrombus in the left auricle. In some cases of calcific stenosis the cardio-inhibitory action of a hypertensive carotid sinus may be productive of cardiac standstill and sudden death. More usual explanations of sudden death in calcareous disease of the aortic valve are ventricular fibrillation, the result of oxygen deficiency due to severe myocardial ischæmia, which may also produce high-grade heart block with consequent cerebral anæmia or cardiac standstill. Acute coronary occlusion caused one death in Willius' series : another case of his exhibited all the features of coronary thrombosis, including the classical electrocardiographic pattern, yet necropsy revealed no evidence of any such vascular accident. Incidentally, a single attack of acute cor pulmonale may produce a remarkable clinical and electrocardiographic likeness of acute coronary disaster without morphological evidence of coronary insufficiency. The physician in charge of a puerperal case, for example, may invite one to see an acute pulmonary embolism whose clinical features, namely, shock, sudden air hunger, sense of impending dissolution and substernal oppressive pain, have not unreasonably raised a suspicion of coronary thrombosis. As a matter of fact, when the pulmonary artery or its main branches are suddenly obstructed, the lesion is in effect a coronary one, in that the increased tension in the right ventricle diminishes the blood flow through the right coronary artery with consequent coronary insufficiency, myocardial ischæmia, and a preponderantly right coronary pattern of the electrocardiogram. Coronary thrombosis itself implies a progressive degenerative arteriosclerotic process with eventually initial hæmorrhage, thrombosis, and occlusion of a coronary vessel, in this order. Lest the gravity of calcific aortic stenosis has been rather over-emphasized, we shall cite the cases of the Latin professor and college dean, who had evident valvular heart disease for at least 25 years. In his sixty-third year the professor consulted White (1932), who found marked calcific aortic stenosis and considerable cardiac enlargement but no congestive failure. 'During the following one and a half years he did very well' remarked White, until one summer 'he was fatigued by college commencement exercises and the hot weather, and then motored off into the country nearly 200 miles in one day'. That night the professor not undeservedly had an attack of pulmonary œdema. He died a few months later. 'The first qualification of a physician is hopefulness'—James Little. And at the bedside of a heart case a great deal too much optimism is, to quote Lindsay, a venial error compared with a little too much pessimism. 'As a rule', remarks Levy, 'even those, who stoutly assert that they "want to know everything", prefer to hear a word of encouragement'. If you feel inclined to flourish the sword of Damocles, you might advisedly recall the observation of Wilks, that 'the sleeping accommodation of all the London hotels would be insufficient to put up the individuals walking about the city who had at some time or another been condemned to death by the medical profession'.

MYOCARDOSIS

Hyman and Parsonnet in introducing their *Failing Heart of Middle Life* (1932) informed us that

> 'The intense publicity given (heart disease) by every agency has swept into many a physician's consulting room individuals who had never before sought medical advice. Probably no phase of health propaganda has excited more interest among the laity than the problem of heart disease. Presented by the startling fact that heart disease leads all other causes of death, notwithstanding the tremendous publicity given to cancer and tuberculosis, many persons have besieged their doctors to examine them and to allay their fears of sudden death from heart failure. With the daily press constantly relating the sudden demise of some prominent citizen from causes said to be heart disease, the problem is more sharply brought to the attention of newspaper readers approaching middle life.'

The writers tactfully met this situation by 'an attempt to portray for the reader a more or less tangible and subtle picture of the failing heart before there are demonstrable objective signs of frank cardiovascular pathology'—the so-called myocardosis syndrome. From the outset we have discountenanced their equivocal term 'myocardosis', convinced as we are in the truth of Stroud's remark that a great many of these 'failing hearts of middle life' are merely introspective, apprehensive individuals who are afraid of heart disease but have nothing wrong with their cardiovascular systems any more than God expects them to have as they grow older.

Willius (1931) found abnormal electrocardiograms in 55 per cent of 700 people over the age of 74 years. Everybody naturally is afraid of heart disease and in America its danger and fatalities have been disastrously over-emphasized by every agency, for example by pamphlets such as 'How is your heart', by posters entitled, 'Your heart is a pump, take care of it', complete with a picture of a pump and a skeleton working at the handle, by broadcasts on heart disease, by overcautious physicians, and by divers other means. In his presidential address to the American Heart Association in 1939, Dr. Stroud admitted that all this heart disease propaganda, originally designed to prevent heart disease, has been a source of worry to him for many years in view of the very many imaginary-heart-disease sufferers it had produced. Dr. Stroud then appealed to the profession in America 'to dispel some of the fear in the mind of the average man concerning cardiovascular disease'. He condemned the publicity methods mentioned above and expressed his agreement

with the sound British attitude by his remark that—'the English certainly feel that we are taking grave chances in bringing too much of this subject before the public, which is not well enough trained to really understand what it is all about'. Certainly, learned addresses to public audiences on 'sudden death' and on what the Irishman calls 'coroner's thrombosis' are not in the public interest. 'Certainly', continued Dr. Stroud, 'we have enough to be afraid of nowadays—social security, wars in Europe, many, many things. If we give our patients a philosophy of life and hope and faith, I believe that we are helping them more than if we make them fearful'. Problems in regard to sudden death and the Insurance and Workmen's Compensation Acts increasingly worry the American lawyer to-day. But he is not complaining. All these considerations decided us to exclude 'myocardosis' from our heart diagnoses in India; 'myocardosis' and 'silent heart disease' are much too subtle for the general public.

REMARKS

Clinical features are stressed throughout this paper because the fundamental basis of a sound knowledge of heart disease is necessarily clinical. Furthermore, substitution procedures such as electrocardiography are not really available to the rural practitioner who is still compelled to rely entirely upon his previous clinical training, stethoscope, blood-pressure instrument and watch for his cardiac diagnosis. The relative value of the different procedures employed in the investigation of a heart case is clearly stated by White (1937) in his *Heart Disease*. 'Electrocardiography', he said, 'ranks third in value after history taking and physical examination : cardiovascular radiography ranks fourth'. 'The electrocardiograph', he insisted, 'does not take the place of such other methods of examination as history taking, percussion, auscultation, and radiography'.

Obviously no cardiologist would attempt to exalt electrocardiography by depreciating clinical investigation. On the contrary, *Diseases of the Heart* by Lewis (1937*a*), the foremost authority on electrocardiography, is the finest appreciation of clinical heart disease I know. We briefly discussed cardiac symptoms and signs in a previous paper (Kelly, 1939). The doubtful symptoms and signs displayed by obese and emphysematous subjects had been discussed already in some detail in *Recent Advances in Cardiology* (East and Bain, 1936) and *x*-ray examination and electrocardiograms advised. John Parkinson, however, remarks that 'when a patient complains of pain about the sternum when he walks, the diagnosis of a healthy heart is out of the question'. The most innocent undergraduate knows that an isolated symptom or sign rarely makes a diagnosis, any more than that 'one swallow makes a summer'. 'Experience teaches', says Lewis, 'that to place reliance upon a single sign is precarious. Compare this sign with that, and confident recognition of the patient's state grows as these signs group themselves together to form a harmonious picture'. We used neurocirculatory asthenia to illustrate this linkage of symptoms and signs to form a clinical picture, which incidentally had not heretofore been sufficiently emphasized to the general practitioner in India. 'The modern routine examination of heart cases', we said, 'includes clinical, electrocardiographic and radiological examination'. The case reported demonstrates how the latter procedures corroborate and supplement the clinical findings. The routine investigation of a heart case in the outpatients' department of the National Heart Hospital, London, is as follows : The technician takes the electrocardiographic tracings of the case before the physician's arrival. The tracings are then taken over by the sister in charge, who places them in their rack in the physician's room. The physician personally takes the history, performs the physical examination and makes a clinical diagnosis in each case. The appropriate electrocardiogram is now picked out of the rack and handed to the physician, who interprets it in conjunction with the clinical picture. When all the cases are thus disposed of, the patients are taken to the *x*-ray department. The physician personally screens each case and outlines on tracing paper the radiological configuration of any case of special interest, correlating all the while the clinical, electrocardiographic and radiological findings. The technical assistant in most electrocardiographic departments can 'rattle off' electrocardiographic findings, *e.g.*, arrhythmias, on sight. They fail, however, to attain the status of the physician in that they cannot correlate the electrocardiographic with the clinical findings. The physician in charge of a case should not regard a mere statement of electrocardiographic findings, such as the technical assistant may provide, as the electrocardiographic interpretation of his case.

Clearly, every physician should interpret the electrocardiographic findings of his own cases in the light of his clinical knowledge of them. To this end physicians should acquaint themselves with the essentials of clinical electrocardiography. These are to be found in *Clinical Electrocardiography* (1937) by Lewis, who tells us that this handbook of only 120 pages is 'intended to serve as an introduction to students of electrocardiography and as a guide to practitioners and hospital physicians in understanding curves that may be taken by others from patients in their charge'. Lewis deals in his excellent way with disturbances of the cardiac mechanism. There is a great and rapidly increasing demand by the American practitioner of medicine who does not intend to specialize in cardiology for handbooks designed to give him a grounding in electrocardiography. *Essentials of Electrocardiography* by Ashman and Hull (1937) is such a handbook. These writers state

that they 'have removed the emphasis from disturbances of the cardiac mechanism and have placed it where it belongs, namely, upon the abnormalities, which reveal or suggest the existence of myocardial disease'.

The rough summation of an electrocardiogram consists in the simple addition of the observed deviations from the normal. Leads I and II are particularly scrutinized for abnormalities, which are usually less significant if confined to lead III. One minor electrocardiographic abnormality may be disregarded. The addition of several inconclusive deviations equals heart disease just as does the sum of several inconclusive clinical findings. Chest leads are of much value and should be requested more frequently, especially lead IV, F.

In the not too distant future the lag-screen belt electrocardiogram will be a commonplace adjunct in the bedside diagnosis of heart cases in India, as it already is in America. This enables the physician, who can interpret electrocardiograms, to see precisely what is going on in the heart. In fact, some physicians have already complained that the machine is not infrequently a source of embarrassment, in that it compels a decision on the spot as to treatment, whereas formerly the physician could look the matter up quietly while the film was being developed. Large numbers of army recruits with alleged heart disease could be quickly and finally disposed of by the lag-screen method of Asher.

'Finally', concluded White, 'it must be realized that the electrocardiogram may be perfectly normal even in the presence of serious heart disease'. The truth of the latter remark is specially emphasized by Lewis (1937)—'It is to be recognized', he said, 'that a thrombosis may happen in the coronary arterial system without appreciable change being displayed then or subsequently in the electrocardiogram'. Thus there are silent areas in the myocardium as well as in the frontal lobe. Nevertheless, I do not propose to disturb the profession or the public by over-stressing silent heart disease. The phrase is too sinister, too suggestive of sudden death, especially to the middle-aged man, who not unreasonably derives much consolation from the thought that his doctor can 'spot' something wrong with his heart by some means or other, and thereby forestall what is dramatically described as a myocardial catastrophy. The third-year medical student is aware of the dangers of dual failure in diphtheria, and the general practitioners I have met never fail to suspect circulatory failure in a case of diphtheria whatever the signs. The remarks of Boyle *et al.* (1939) concerning this circulatory failure are not uninteresting. 'Surprisingly', they said, 'death from this cause is frequently not entirely explained by the pathologic changes in the heart. The frequent paucity, or even absence of cardiac lesions, contrasted with the dramatic collapse in diphtheria, has led some authors to look elsewhere for the cause of death, or to hypothesize a functional alteration of the myocardium'. Paul White's attitude towards electrocardiography is well balanced. 'This method of study', he said, 'should be viewed modestly as helpful and supplementary but not accorded too great importance'.

Dr. Stroud (1939) has recently expressed considerable anxiety 'concerning unnecessary fears developed through electrocardiographic interpretations. He recalled Sir Thomas Lewis' last words to him—

> 'When you return to the United States, you must be careful in using the electrocardiographic galvanometer. Remember that the United States is a young country, and Americans are impressionable. They believe that almost anything may be accomplished through mechanical or scientific effort. If you use this machine, then look at the tracings in front of the patient, then look at the patient and shake your head, that patient is probably a cardiac cripple for the rest of his life.'

Stroud then tells of the awe inspired by electrocardiographic apparatus in the not-so-intelligent patient. He tells of the patient about to be electrocardiographed who appealed, 'Doctor, do you mind if I say a little prayer before you turn the thing on': and he tells of the slightly more intelligent patient who remarked to him a week after the tracing was made—'Doctor, I feel ever so much better since I took that electrocardiographic treatment'. Finally Dr Stroud continues—

> 'I believe that with all these machines now being sold throughout the country, we must be sure to educate not only the public, but also the medical profession, as to the relative importance of the electrocardiogram. We must persuade doctor and patient, alike, that it is impossible to read from the electrocardiogram alone the last word as to the future of the cardiovascular system. I am positive that all too many physicians are attempting to read from the electrocardiogram more than is justifiable.'

Case report

On the 12th of April, 1939, a 25-year-old Hindi tutor, S. N. S., had a syncopal attack which so alarmed him that he sought admission to the Medical College Hospital, Calcutta, that evening.

His family history was negative. He had malaria and eczema in childhood. At the age of ten he was confined to bed for several weeks on account of a moderately severe grade of fever, attended by sore throat and flitting arthritis. For a whole year thereafter he had an evening temperature and experienced palpitation and dyspnœa whenever he tried to get about. In fact, ever since he had that long-drawn-out fever fourteen years ago he has constantly noticed that moderate exertion brings on some degree of palpitation together with a less degree of dyspnœa. From time to time in the past five years a feeling of mild general weakness comes over him. Lately, he has been having night starts and bad dreams and also feelings of faintness. To-day he had an attack of actual syncope.

Physical examination

The patient was a moderately well-developed and well-nourished young man of average height. He exhibited slight pallor, mild acne rosacea, and some nervousness. His temperature was 98.6°F. His pulse was regular at a rate of 82: the right radial pulse had a slightly jerky character and both radials were appreciably thickened. The respiratory rate was 20: sighing respiration was not evidenced. His blood pressure was

135/65 right arm, and 125/80 left arm: his subclavian and carotid pulses, however, were equal. He weighed 144 pounds dressed.

Examination of the head and neck, including observation for carotid pulsation, revealed nothing abnormal. The pupils were equal, central, regular, moderately dilated, and reacted well to light and in accommodation. The throat was apparently healthy: the related glands were impalpable. The mouth seemed healthy.

The lungs were normal to percussion and auscultation.

Heart

The præcordium was slightly prominent. The obviously heaving apex impulse was diffused in the fifth and sixth interspace to about half an inch outside the left mid-clavicular line. The maximum apex impulse was found in the left fifth interspace in the mid-clavicular line. A well-marked systolic thrill was palpated at the aortic base, maximal at the junction of the right second costal cartilage with the sternum.

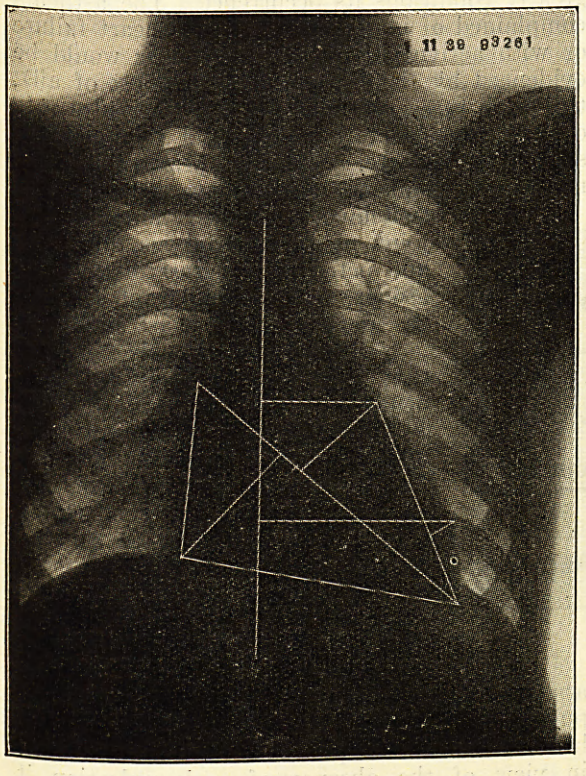
The abdomen was flat: there was no tenderness or rigidity and abdominal organs were not palpable.

The extremities were normal.

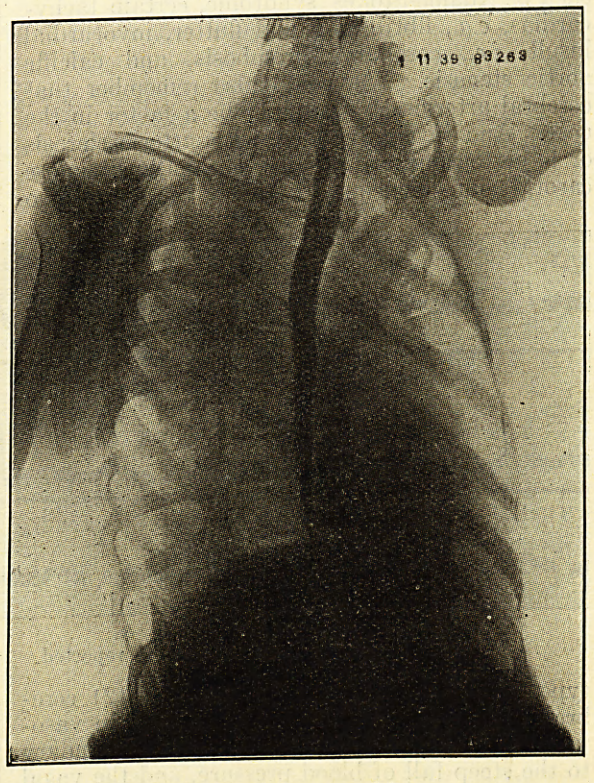
The reflexes were hyperactive and equal.

Electrocardiographic findings

Normal rhythm. A and V rate 72 (lead II). P-wave slightly prominent. P_2 width = 0.12 sec. (over 0.1 sec. in lead where it appears to be widest), P_2 height 3 mm. (over 2.4 mm. in lead II). P-R interval = 0.22 sec. (0.20 sec. is upper limit for large adult with a heart rate of 72). No deviation of electrical axis. QRS interval = 0.08 (not over 0.10 sec. in lead where widest in adult). Large excursion of R in lead II. R_1 slightly slurred upstroke, and downstroke especially near base. R_3 upstroke slurred (position of slurs fairly constant, *i.e.*, not caused by the alternating current oscillations). RST segments show apparently low and high take off of slight degree in leads I and III, respectively, due to slight downward and upward drift of the base line.



Antero-posterior position.



Right oblique position.

There was no supra-cardiac dullness. The low-pitched, prolonged and somewhat muffled apical first heart sound was modified by the presence of an emphatic component and was attended by a short systolic murmur. An aortic diastolic whiff commenced with the second sound at the apex, which was followed by a low-pitched mitral diastolic rumble with mid-diastolic accentuation. At the site of maximal intensity of the aortic systolic thrill, a loud, harsh, early systolic murmur was heard, masking the first heart sound at the aortic base and extending throughout the greater part of systole. The latter murmur was well conducted into the neck, down towards the apex and slightly into the back. The second sound in the aortic area was diminished in intensity and slightly blurred by a smooth soft diastolic aortic murmur, the distant waterfall character of which was more evident at its point of maximum intensity, namely, in the left fourth interspace close to the sternum. The second sound was absent over the carotid artery. The first heart sound was easily audible in the pulmonary area and the second was accentuated.

T-waves diphasic. Q-T = 0.36 sec. Normal (upper limit for this cycle length of 0.84 sec. = 0.384 sec.). I acknowledge my thanks to the electrocardiographic department for the tracing (*see* page 144). The findings are the writer's.

Radiological examination

The professor of radiology reported as follows:—'The heart shows considerable general enlargement. The aortic shadow appears normal. The œsophagus shows slight deviation as by an enlarged left auricle'. This report is confirmatory of the writer's fluoroscopic findings.

Laboratory findings

Blood.—Hæmoglobin—75 per cent; R. B. C.—4,500,000; and W. B. C.—6,120.

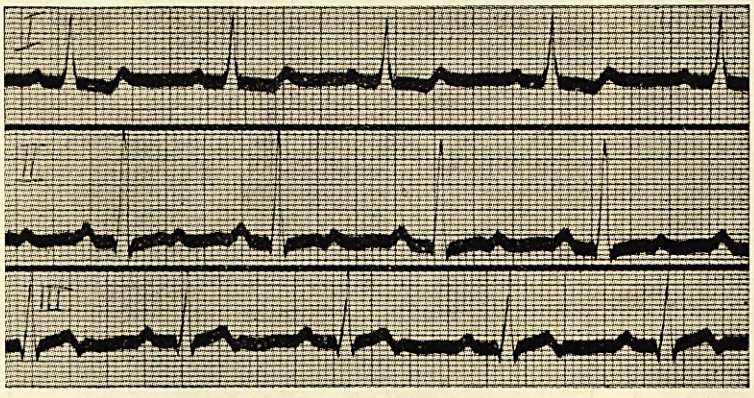
Sedimentation rate.—Within normal limits.

Blood Wassermann reaction.—Negative.

Throat.—*Streptococcus hæmolyticus,* pneumococcus, *M. catarrhalis,* and *Staph. albus.*

On culture—no Klebs-Löffler bacilli.

Comment.—This patient is evidently introspective and apprehensive. His reflexes and emotional responses are brisk. There is nothing, however, that shakes the general morale of any individual more than the occurrence of attacks of faintness or actual syncope. The underlying mechanism responsible for syncope in young people is usually neurogenic (vaso-vagal syncope) and in them syncope is generally benign. Paroxysmal tachycardia is an occasional cardiogenic cause of syncope in young persons. Aortic valvular disease is the only form of valvular disease causing syncope. In this case syncope owns a neurogenic mechanism. If, on the other hand, the patient were an elderly man, we would first exclude a cardiogenic origin such as Adams-Stokes' syndrome, certain tachycardias, *e.g.*, fibrillation and flutter, myocardial insult, *i.e.*, coronary thrombosis, and calcific aortic stenosis. We would also remember that cerebral arteriosclerosis might be a factor in the case. And in elderly men with arteriosclerosis or hypertension, we might occasionally diagnose carotid sinus syncope of which there are two types, (1) cardiac or circulatory, and (2) cerebral (Ferris, Capps and Weiss, 1935). In vaso-vagal syncope, loss of consciousness is secondary to the steep fall of blood pressure, and the vagal slowing of the heart is less important, whereas in the cardiac type of carotid sinus syncope unconsciousness is dependent upon slowing of the heart, which may amount to complete block for a few beats, and the usually accompanying fall of blood pressure is secondary to this slowing of the heart. Sweating aids recognition of the former type of syncope: in cardiac syncope sweating is rare.



Electrocardiogram of case reported.

The cerebral type of carotid sinus syncope is characterized by the occurrence of syncope and convulsion in spite of the fact that there is no cardiac slowing and no fall in the blood pressure, *i.e.*, in spite of a normal blood flow. Syncope of this type is due to altered reaction of the brain cells, dependent upon vaso-motor changes in strictly localized areas or upon a response to afferent nervous impulses originating in the (hypersensitive) carotid sinuses. Apparently these were the cases that Gowers relegated to the borderland between epilepsy and syncope: at least so Weiss suggests. Vaso-vagal syncope of Weiss and Ferris has a somewhat similar mechanism to that of the cardiac type of carotid sinus syncope. This very occasional form of syncope is due to reflex slowing of the heart caused by gastro-intestinal disorders acting through the vagus nerves. Night starts and bad dreams are more common in the subjects of aortic than of mitral disease. But a story of night starts and bad dreams is not infrequently told by an emotional and imaginative patient such as this one. Palpitation too is not uncommon in the subjects of aortic disease, but here again the sensitivity of the nervous system greatly conditions its occurrence. The asthenia complained of is unassociated with heart failure or with active valvulitis. The latter would be evidenced by the leucocyte count (best of all), the pulse rate especially the sleeping pulse rate over 90, temperature above 99°F., accelerated sedimentation rate, dyspnœa, præcordial pain, nausea and vomiting, by rheumatic polyarthritis, twitching, nodules, muscle and joint pains, hæmoptysis, diarrhœa and petechiæ, progress of valvular lesions, electrocardiographic changes and increasing heart size. We may therefore reasonably conclude that this case is complicated by neurocirculatory asthenia.

The slight pallor of this patient is unattended by appreciable anæmia. Active rheumatism has been excluded. Subacute bacterial endocarditis is negatived by the absence of symptoms and signs of infection including finger clubbing, splenic enlargement and superficial petechiæ. Another possibility is 'pale mitral stenosis', *i.e.*, mitral stenosis associated with aortic regurgitation. Neck pulsation, however, is absent. But the slow heaving cardiac impulse is a striking sign: in view of the absence of neck pulsation it prompts a diagnosis of aortic stenosis. This diagnosis is confirmed by the presence of the corresponding murmur and thrill, diminished aortic second sound, aortic diastolic murmur, moderately enlarged left ventricle, and by the absence of aortic dilatation radiologically. The presence of mitral stenosis further reinforces the diagnosis. The pulse is a compromise between the pulses of all three valvular lesions. A rheumatic ætiology is clearly indicated by the rheumatic history, prominent præcordium, clinically and radiologically evident mitral stenosis, and the electrocardiographic picture. We shall now briefly interpret the latter.

Auricular hypertrophy, the logical consequence of mitral stenosis, is evidenced by the excessive height and width of P. Ventricular preponderance is lacking in this case of rheumatic aortic stenosis and regurgitation with mitral stenosis,

because the effect of right ventricular hypertrophy associated with mitral stenosis is neutralized by the effect of hypertrophy of the left ventricle which has resulted from aortic stenosis and regurgitation. The cessation of rheumatic activity is marked by the return of the Q-T interval to normal. The P-R interval, however, remains prolonged. Such persistent prolongation of the P-R interval in a rheumatic subject indicates a distinct proclivity to the development of auricular fibrillation, the onset of which is precipitated by the simultaneous action of two factors, *i.e.*, vagal activity and the E factor of Nahum and Hoff. The QRS and P-wave changes tend to persist and will increase with further recurrence of rheumatic activity and the hypertrophies resulting from the diseased aortic and mitral valves. Permanent electrocardiographic changes in rheumatic heart disease include notching or slurring of the QRS group in two or more leads, T-wave inversion (T_1 T_2) and auricular fibrillation. T-wave changes and factors influencing the pattern of the T-wave in aortic stenosis are already described.

Supplementary note

Since the completion of this paper the patient has been readmitted on account of a rheumatic relapse. His aortic stenosis is clear-cut as before. His mitral stenosis is more in evidence than formerly, in that he now displays a diastolic thrill, snappy mitral first sound and a longer mitral diastolic murmur. His pulses have altered somewhat. His present blood pressure is 130/90 right arm and 120/75 left arm. The jugular chain of cervical lymphatic glands is just palpable bilaterally. Graded pressure over his right carotid sinus again failed to elicit evidence of carotid sinus sensitivity, such as may be induced to the point of syncope in a chronic dysenteric in our wards, a Hindu male, aged 60 years, who has never in his life complained of dizziness, giddiness or syncope. The vaso-vagal attacks described by John Ryle (1939) emphasize the great importance of nervous impulses in the production of syncope and serve to remind us that cerebral anoxia is not the only mechanism causing unconsciousness in man.

A few points in the treatment of calcific aortic stenosis in the stage of failure

Bedford (1939) has ably outlined the treatment of isolated left ventricular failure. Mackenzie and Lewis believed that depression of auriculo-ventricular conduction was the essential action of digitalis on the heart. In his treatment of failure with congestion, Lewis said : 'The most emphatic action of digitalis and its allies is in the case of auricular fibrillation'. Lewis (1937) dismissed digitalis therapy in failure with regular rhythm, with the remark that : 'Occasionally, however, cases of chronic congestion that present regular heart action, and that have been treated by all the usual methods without success, respond to full doses of digitalis'. Wenckenbach and Christian, on the other hand, attributed the efficiency of digitalis to its effect on myocardial tone and contractility, and on this basis advised its use in all congestive failures. Gavey and John Parkinson assessed the clinical value of digitalis in heart failure with normal sinus rhythm and compared the same with that in auricular fibrillation. Their main conclusions were given in a former paper (Kelly, 1939). The third effect of digitalis on the heart, namely its depression of the pace-making function of the sino-auricular and also of the auriculo-ventricular node with resulting tendency of the heart rate to be lowered, has long interested John Parkinson. In soldiers with cardiac symptoms and a frequent pulse John Parkinson (1917) found the reduction in rate from digitalis 'was almost negligible' and in his recent study of digitalis in failure with normal rhythm he remarks that 'Reduction in rate was not always accompanied by clinical improvement, though improvement was rather more common in the patients who showed it. Some good clinical results were seen without any reduction in rate'. Hence, in the regular rhythm failure of calcific aortic stenosis, which is generally documented by a remarkably slow heart rate, we must not strive after a further reduction of the heart rate : induction of the muscular effect of digitalis and not of its direct sinus action is the object of therapy.

Partial heart block does not contra-indicate the use of digitalis.

Convallan is recommended by Ralph Major and Leger (1939) in lieu of digitalis, should heart block or bundle branch block attend cardiac failure, as it not infrequently does in calcareous disease of the aortic valves. Convallan is a special extract of *Convallaria majalis* or lily-of-the-valley. Its activity is due to certain digitalis-like glucosides. Its pharmacological action in large doses is essentially the same as that of digitalis and strophanthin. In small doses it produces remarkable diuretic effect without causing heart block or increasing the degree of any existing block. Its cumulative action is negligible and it may be given before or after digitalization with complete safety. The minimum effective dose is 3,000 frog units, and up to 12,000 frog units may be administered daily with safety. We have had no personal experience of convallan yet.

'The partnership of a mercurial diuretic with digitalis should govern the treatment of heart failure'—John Parkinson. The former lessens œdema, even of the heart muscle itself, and the latter directly improves myocardial efficiency. Nearly ten years ago Bedford demonstrated the occasional superiority of salyrgan to digitalis in the treatment of heart failure. Salyrgan acts mainly on the kidneys, either directly or indirectly as part of a general vascular effect : Hermann and others suggest that it acts preponderantly through inhibiting

tubular reabsorption. Hence reasonably efficient renal function is essential for salyrgan diuresis, and impaired renal function contraindicates the use of salyrgan or other mercurial diuretic. Obviously, calcific aortic stenosis subjects with sclerotic kidneys are unlikely to respond to salyrgan. Other preparations similar to salyrgan are neptal and mersalyl. Esidrone (Ciba), the sodium salt of pyridine dicarboxy-β-mercuri-w-hydroxy-propyl-amide-theophylline, contains 32.2 per cent mercury in non-ionizable form and 28 per cent of theophylline which is bound chemically to the mercury molecule. Mercupurin is another mercury-theophylline compound. Fishberg (1937) enjoins upon us not to give mercurials to moribund patients: 'I have', he said, 'seen them add the *coupe de grace* by producing anuria'. Mercurials lower the venous pressure and so are inadvisable in the shock stage of coronary thrombosis.

The action of mercurial diuretics should be supplemented from time to time by xanthine derivatives. Theopyllin (theocin) according to Fishberg produces diuresis predominantly through increase in renal blood flow with consequent augmentation in glomerular filtration. Its chief compounds are theophylline ethylenediamine (aminophyllin or euphyllin) and theobromine sodium-salicylate (diuretin). Theobromine calcium-salicylate (theocalcin) may sometimes usefully replace diuretin. Animal experiments have probably over-emphasized the additive or synergistic effect of calcium and digitalis on the heart. Wall (1939) found that no untoward reactions followed the intravenous injection of 5 c.cm. of a 20 per cent calcium gluconate solution in congestive failure cases receiving digitalis. He suggests that the calcium so injected is diluted before it reaches the heart by the increased circulating blood volume and slowed circulation of heart failure subjects. In Wall's opinion the danger lies in a sudden increase in calcium-ion concentration in the heart and not in the synergism of calcium and digitalis. Finally, we might add that we find some advantage in 'ringing the changes' on diuretic drugs.

Summary

1. The modern ætiological concept of calcific aortic stenosis is that the vast majority of cases are clearly rheumatic in origin.

2. The incidence of rheumatic heart disease in our hospital practice was shown and an attempt was made to ascertain the incidence of aortic stenosis from our clinical and post-mortem records.

3. The distinctive symptomatology of calcific aortic stenosis was described.

4. The diagnostic criteria of aortic stenosis were discussed in moderate detail.

5. Calcification of the aortic valve demonstrable by fluoroscopy is indisputable evidence of calcific aortic stenosis.

6. The electrocardiographic and other findings were briefly outlined.

7. The essential requirements for a diagnosis of aortic stenosis were stated and the differentiation of calcific aortic stenosis from arteriosclerosis or coronary heart disease was mentioned.

8. A reference was made to the prognosis of aortic stenosis.

9. The importance of correlating clinical, electrocardiographic and radiological findings in a heart case was stressed.

10. A case of rheumatic aortic stenosis was presented.

11. The treatment of calcific aortic stenosis in the stage of failure was briefly reviewed.

I am grateful to Lieut.-Colonel J. C. De, I.M.S., Superintendent, Medical College Hospitals, Calcutta, for permission to report the case. I acknowledge gratefully the help I received from Dr. A. K. Ahmed, my senior house physician, and from Dr. G. B. Sinha, my medical registrar, in respect of our hospital statistics.

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(*Continued at foot of opposite page*)

OBSERVATIONS ON THE USE OF NICOTINIC ACID IN THE TREATMENT OF PELLAGRA AND ALLIED CONDITIONS

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IN September 1937 Elvehjem *et al.* discovered that a single dose of 30 mg. of Eastman Kodak Company nicotinic acid improved the appetite and stopped the diarrhœa in a dog suffering from black tongue; since this date many people have treated human pellagrins with nicotinic acid. Results from this form of treatment have been so successful that nicotinic acid is now established as a specific cure for pellagra.

A large amount of research has been carried out in the United States where pellagra is very common. Smith *et al.* (1937) reported a cure with 60 mg. of nicotinic acid daily for twelve days. The appetite improved after 24 hours and mental improvement was noted after 48 hours. The skin was improved after 3 days.

Spies *et al.* (1938) reported an immediate increase in appetite and cessation of nausea and diarrhœa. Twenty-four hours after administration of nicotinic acid, the tongue became less sore and salivation diminished. He recommended a full well-balanced diet in addition to nicotinic acid.

Spies and Aring (1938) drew attention to beriberi symptoms in pellagra cases. They found that many pellagrins in the U. S. A. suffered from alcoholic neuritis.

In India cases of pellagra are frequently met with. Rau and Raman (1936) reported 8 cases in Vizagapatam. They carried out blood analysis and found that the blood showed a fairly constant reduction in the albumin fraction.

In March 1939, Napier drew attention to the importance of this disease in India in a detailed description of it, and Sen Gupta, Napier and others (1939) recorded five cases treated in Calcutta. Bajaj (1939) treated one of six cases in the Punjab with nicotinic acid and found it to be 'very helpful in improving the local condition of the mouth and the skin'.

A Hindu agriculturist was treated successfully in Midnapore in July 1939 with six injections of nicotinic acid, and further interest in this form of treatment was aroused by two more cases in the Presidency General Hospital. At the same time it was observed that several other patients, particularly Anglo-Indians, showed some of the symptoms of pellagra though not clinically suffering from that disease. Many of this latter group showed improvement under nicotinic-acid treatment.

Altogether 20 cases were treated with nicotinic acid and for study purposes these were divided into three groups.

(1) Cases of true pellagra.
(2) Cases of nicotinic-acid deficiency.
(3) Miscellaneous cases.

Cases of true pellagra

Only three cases were allotted to this group. The distinction, however, between this group of cases and those classified under nicotinic-acid deficiency was mainly one of degree. Patients with marked skin lesions, gastro-intestinal and nervous symptoms were classified as true pellagra cases. Less definite cases were classed as cases of nicotinic-acid deficiency. All three were men between the ages of 30 and 50 years. One was a Hindu agriculturalist and the other two were unemployed Anglo-Indians.

Rice was the main article of diet in each case though the Hindu was in the habit of eating fish fairly frequently, and the Anglo-Indians ate meat when they could afford it.

The duration of symptoms varied from 7 months to 12 years. The most severe was case 1 : he complained of indigestion, itchiness, and 'insects flying out of his ears' and 'worms crawling in the skin'. His condition became worse each winter but he had managed to do his work till a year ago when fever and diarrhœa left him very thin, and he had to give up his employment as a boiler-maker. He had previously been treated in hospital for gastritis.

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