EARLY DIAGNOSIS OF RHEUMATIC VALVULAR DISEASE IN RECRUITS

BY

JOHN PARKINSON AND RONALD HARTLEY

From the Cardiac Department of the London Hospital

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During the world war of 1939-45 the call for men for the armed forces made the medical examination of potential recruits an important field of medical service, and many were rejected for heart disease. The major problem for cardiologists was to detect the presence of rheumatic heart disease when it was early and symptomless, a stage not seen except incidentally in peacetime practice. It might at first be thought that with the advent of peace the importance of very early diagnosis would quickly fall. But it is becoming evident that in economic and labour conditions after this war, comparable medical examinations will be multiplied and that in time few employees will be accepted without them.

From 1940 to 1945, 2500 men suspected of cardiac abnormalities were referred to one of us (J. P.) by medical boards. Among them were found 609 cases of early rheumatic heart disease, of which only a quarter had symptoms, and those slight. Only 264 had mitral valvular disease, a low proportion due to the fact that mitral stenosis is more easily identified by medical boards than aortic incompetence, and that those with mitral disease more often get symptoms at an earlier age and, therefore, come under medical observation. It is on the analysis and study of these 609 together with a limited survey of the literature, that this paper is mainly based. Table I shows the relative incidence of various cardiovascular defects. Cases of effort syndrome (neuro-circulatory asthenia) were not included.

TABLE I
CARDIOVASCULAR DISEASE AMONG 2500 MEN REFERRED BY MEDICAL BOARDS

Rheumatic heart disease	609	(76 per cent)
Congenital heart disease	71	(9 per cent)
*Hypertension	49	(6 per cent)
Enlargement, cause unknown	25	(3 per cent)
*Hyperthyroidism	12	
Angina of effort	12	
Coronary thrombosis	5	
Syphilitic aortic incompetence	4	
Parox. tachycardia	10	
Bundle branch block	2	
Complete heart block	1	
Auricular fibrillation	5	
Auricular flutter	1	
	806	

* These figures are smaller than might be expected because many such cases were referred to physicians other than cardiologists.

The figures in Table I may be compared with those given in a table shown among the individual reports of the chairmen of the special medical advisory boards in the United States (Fenn *et al.*, 1944). Of 698 rejected for cardiovascular disease and disorder, 396 had rheumatic heart disease, 43 had congenital heart disease, and 161 had hypertension; neuro-circulatory asthenia accounted for 61 rejections.

The proportion of cardiovascular disease among recruits in this country has been given as

9 per cent of 30,000 rejections for all causes ; of this 9 per cent, four-fifths were for rheumatic heart disease (Parkinson, 1945). In the United States, Rowntree (1945) estimated that 6 per cent of 2,976,000 total rejections were for cardiovascular reasons. About half were due to rheumatic lesions (Levy, Stroud, and White, 1943). Wilburne and Ceccolini (1944) examined 20,000 men and found that 6 per cent of all rejections were for cardiovascular lesions and that 63 per cent of these were rheumatic. Perrot (1944) found that 8 per cent of all rejections were for cardiovascular reasons.

METHOD OF EXAMINATION

Past History. No great reliance was placed upon a past history of rheumatic fever or chorea, whether negative or positive; too many men, in their eagerness to serve or to escape service, will falsify such a history; the same applies to a story of dyspnoea on exertion.

Inspection. This served to exclude obvious anæmia or goitre, and to note nutrition, physique, and any deformity of the chest or spine.

Palpation. The apex beat was then found by placing the whole hand lightly upon the chest wall under the left breast. It was noted whether the impulse was displaced or too forcible. Minor degrees of scoliosis frequently move the apex beat outwards. The anterior chest wall was then palpated for the presence of a thrill, the palm of the hand being laid lightly over the apex and then over the base of the heart. A thrill at the base of the heart may best be felt by the observer standing on the right of the subject who leans forward and stops breathing at the end of full expiration ; the hand should be merely applied to the chest wall, not pressed hard. No reliance whatever was placed upon a doubtful thrill at any site.

Auscultation. The heart sounds were then considered, whether normal or abnormal, their loudness, and any variations in character which frequently lead to doubt in diagnosis. A split first sound which is fairly common in health sometimes led to an unjustified suspicion that early mitral stenosis was present; an accompanying double quality in the feel of the apex beat added to this suspicion. The first sound may otherwise vary in loudness or clarity in different subjects without significance. It is often said that a pulmonary second sound, loud or duplicated or both, is a sign of early mitral disease. But this is not so—such variations are common in healthy young adults. The normal third heart sound, so common in the young (Thayer, 1909 ; Evans, 1942*b*), was also frequently mistaken for a mitral diastolic murmur. Bramwell (1943) found a reduplication of the second sound at the apex, which he considers to be identical with the third heart sound, to be frequently mistaken for mitral stenosis.

Murmurs were first considered with the subject standing, then when lying, and again when in the left lateral position after exercise. During this procedure a presystolic murmur is heard best at the higher range of rate, and an aortic diastolic during the lower range of rate. We have found it convenient thus to listen for an aortic diastolic murmur on the left of the sternum while the heart rate is falling after the subject has exercised and turned half over on his left side, and immediately after deciding on the presence or absence of a presystolic murmur at the apex. The aortic diastolic murmur is also well heard with the subject leaning forward with the breath held at the end of full expiration. We have often been exhorted to keep an *open* mind, and in general this is sound advice. We do naturally listen without preconceived ideas of what will be heard; and we cannot help noticing a first sound which is unusually loud or when there is an obvious systolic murmur. That is easy, and now comes the moment to *close* the mind to what is likely to preoccupy it and to concentrate upon answers to special questions :

(1) Is there a mid-diastolic murmur at the apex, a distant murmur beginning with the third heart sound but distinguishable from it ? If so there is mitral stenosis. (2) Is there a diastolic murmur immediately following the second sound ? If so there is aortic incompetence and the murmur can be tracked up the left border of the sternum to its aortic source. Murmurs were timed by the carotid pulse, felt best with the observer's left hand resting on the right side of the patient's neck, the thumb on the artery.

The Pulse. The rate and rhythm of the pulse were noted at the end of the examination, when the recruit was more composed. Its response in rate to effort was noted when auscultating after exercise, but no so-called exercise tolerance tests were made.

Radioscopy. Radioscopy was used in every case, employing the three standard positions, i.e. anterior, right oblique, and left oblique ; and all statements on heart size were based on radioscopy, occasionally supported by teleradiography.

Electrocardiography. Cardiograms were taken on the first 500 cases but their value was so limited that subsequently they were taken only for extra evidence when diagnosis was difficult or when an unusual rhythm was present. This agrees with the conclusions of Wood, Wolferth, and Miller (1941) based on a large experience. The exigencies of war practice had also to be considered. But a cardiogram was always taken in suspected myocardial disease, chiefly among the older recruits.

THE DIAGNOSIS OF EARLY MITRAL VALVULAR DISEASE

This entails a discussion of the significance of the apical systolic murmur which has been regarded as the commonest murmur in mitral stenosis (Graham Steell, 1906). We doubt whether this is true of young adults judged by our experience with recruits. Of 184 recruits diagnosed certainly as mitral stenosis, 104 had a presystolic murmur (in 72 alone, in 3 with a diastolic, and in 29 with a systolic murmur in addition) ; 13 had a mitral diastolic murmur only; 5 a loud apical first sound only; and 2 were diagnosed solely on the X-ray appearances, there being no abnormal auscultatory sounds. A further 60 had only a systolic murmur. One might expect to find in current textbooks, under mitral stenosis and its diagnosis, a special reference to the first signs permitting its early diagnosis ; but the description is usually that of the fully developed disease as seen in patients with symptoms seeking medical advice. In contrast, war requires, and peace should require, the recognition of the symptomless valve lesion.

The Apical Systolic Murmur. The commonest cause for a medical board to want a cardiologist's opinion was the presence of an apical systolic murmur. If it was not overlooked, the common soft pulmonary systolic murmur was apparently more easily assessed as insignificant.

We have reserved the term incidental—preferring it to the term functional—for those systolic murmurs that occur not only in health and, therefore, could be classed as innocent, but also in anæmia and pyrexia—murmurs that are not indicative of cardiovascular disease. Systolic murmurs also occur with “relative incompetence” of the mitral valve, as in association with aortic incompetence or stenosis, in hypertension, rheumatic carditis, subacute bacterial endocarditis, and heart failure. Rheumatic heart disease affecting the mitral valve is a third and the most important source of this sign. A harsh systolic murmur with thrill near the sternum in the fourth or fifth left interspace may be congenital—that of a ventricular septal defect.

The incidence of systolic murmurs in any group of apparently healthy people is high. Contratto (1943) heard such murmurs in 350 (12 per cent) of 2856 college students examined ; of these, 208 were classed as functional, 9 as rheumatic valvular disease, 6 as congenital lesions and 127 were regarded as more than functional, though the physical signs were insufficient to permit an exact diagnosis. Wilburne and Ceccolini (1944) found 297 functional murmurs among 20,000 recruits. In our own series 259 (10 per cent) incidental murmurs of impressive intensity were heard.

Studies of the natural history of the systolic murmur such as those by Blumenthal (1942) and by Baker, Sprague, and White (1943) underline the great difference in prognosis between the incidental (or functional) murmur and the organic mitral systolic murmur. The difficulties in the assessment of the systolic murmur are considerable; Master (1944) asserted that many men admitted to the U.S. Navy with so-called functional murmurs were later boarded out with valve lesions.

Incidental murmurs are generally soft and short, and they vary in intensity both with position and with respiration to a greater degree than organic murmurs. They are unaccompanied by symptoms or signs of heart disease. Radioscopy shows a heart normal in shape and size. There may be some chest abnormality such as a depressed sternum which is often associated with a systolic murmur, an association noted twelve times among our 2500 recruits. The term “cardio-respiratory” applies by definition to systolic murmurs varying with respiration, disappearing at some phase, often that of full expiration or inspiration. In practice

they are short and soft, and seldom present difficulty. Exocardial murmurs were heard in three men. Scadding and Wood (1939) have drawn attention to a systolic click that occurs in cases of left-sided early pneumothorax.

The important features in assessing a systolic murmur are loudness and length, the presence or absence of a diastolic murmur, and evidence of enlargement of the heart on X-ray. As regards the intensity, White (1927) showed that there is a distinct difference in prognosis between the three groups of loud, moderate, and slight murmurs. Levine (1933) and Freeman and Levine (1933) advised that systolic murmurs be graded into six groups according to their intensity: they found that only the first two groups of this classification were of no significance and that the majority of functional murmurs fell into the first group, which consisted of the very shortest and softest murmurs.

Evans (1942*a*) has lately again stressed the importance of the late systolic murmur, i.e. a murmur occurring late in systole, separated from the first heart sound by a short interval; although this murmur is often noticeable, Evans believes that it is always innocent. Twenty-four such murmurs were heard among the 2500 recruits seen, and there was never any reason to think they represented mitral disease. A medical board, however, might regard them as evidence of mitral stenosis or of aortic valvular disease (Table II).

TABLE II
ANALYSIS OF THE DESCRIPTION OF 24 "LATE SYSTOLIC MURMURS" AS STATED BY MEDICAL BOARDS
WHEN SEEKING A CARDIOLOGIST'S OPINION

(1) Systolic murmur at the apex	7
(2) No reference to the murmur	3
(3) Apical murmur with no reference to timing	4
(4) Diastolic murmur	4
(5) Presystolic murmur	2
(6) Systolic and diastolic murmurs	2
(7) Board stated difficulty in timing the murmur	2
	24

The intensity of murmurs may be graded simply into three groups, soft, moderate, and loud, and the length may be given as short, moderate, or long. The phrase "the murmur replaces the first sound" used often to be employed, but now it is assumed that if the systolic murmur is long and loud enough the first sound may be ousted. The conduction of a murmur is little more than a measure of its intensity (Levine and Likoff, 1944). We join with Levine (1933) and Freeman and Levine (1933) and Bramwell (1942) in hesitating to call a loud systolic murmur incidental or functional, even in the absence of collateral signs of heart disease. It is in such cases that the routine use of the phonocardiograph may be of great use in the near future. Whether a recruit should be accepted for the forces when he possesses a very noticeable but innocent systolic murmur is open to argument. We think that unless extremely keen, such a man runs the risk of a "doctor made" cardiac neurosis (Hill and Dewar, 1945). Some claim that this is placing the individual before the State, but such a soldier may become more of a liability than an asset in any active unit.

MITRAL STENOSIS WITH A PRESYSTOLIC MURMUR

One hundred and four (almost 40 per cent of all the cases of mitral valvular disease) cases of mitral stenosis with a presystolic murmur, and all with a loud first sound, were seen. Of these 37 per cent gave a history of rheumatic fever, 15 per cent of chorea, and 3 per cent of both (total 55 per cent). Among men without heart disease a history of rheumatic fever was given in 14 per cent, of chorea in 2 per cent, and of both in 0.1 per cent, a total of 16 per cent. This figure is higher than in the normal population because such histories often influenced the medical boards in deciding to seek a cardiologist's opinion. More reliable percentage frequency of rheumatic history in the normal is that of 1000 men studied during the first world war, namely 5 per cent (Parkinson, 1945).

Dyspnoea on exertion was the only symptom considered; in all cases of mitral stenosis, no matter what the physical signs, the proportion with dyspnoea proved to be about the same, 20 per cent. Short-windedness must always be considered in relation to the build, occupation,

and condition of the patient. Many men with mitral stenosis showed a loud and sudden first sound as the only obvious physical sign. In the majority, however, a presystolic murmur was evoked by exertion and then heard in the left lateral position. In the absence of a presystolic murmur, and in the absence of tachycardia, the characteristic apical first sound points to the probability, but not the certainty of mitral stenosis. Other causes of a loud first sound are hypertension, tachycardia, and thyrotoxicosis, and in the last especially it can resemble a presystolic murmur. In five cases with a loud and sudden first sound only, and tachycardia absent, exertion failed to elicit a presystolic murmur; yet the X-ray appearance was, in all five, diagnostic of mitral stenosis. Thirteen cases had a mitral diastolic murmur only, limited to the apex. The percentage having symptoms was essentially the same in this last group as in those with a presystolic murmur; the percentage with a positive rheumatic history was also identical with that of the presystolic group.

TABLE III
AUSCULTATORY SIGNS IN 184 CASES OF EARLY MITRAL STENOSIS

Presystolic murmur only	72
Presystolic murmur and systolic murmur	29
Presystolic and diastolic murmur	3
Loud mitral first sound only	5
Diastolic murmur only	13
Systolic murmur only	60
No abnormal auscultatory signs	2
	184

MITRAL STENOSIS WITH A MURMUR, SYSTOLIC NOT PRESYSTOLIC

In a total of 264 cases seen with mitral valvular lesions, 60 had a mitral systolic murmur of moderate or loud intensity and of moderate or long duration. This with a combination of an apical systolic thrill in 9; a rheumatic history in 48 per cent, chorea in 7 per cent, and both in 5 per cent (total 60 per cent); dyspnoea on exertion; and a loud first sound, or X-ray changes (90 per cent), allowed a diagnosis of mitral stenosis to be made with some certainty.

MITRAL INCOMPETENCE

Between the two world wars it was frequently taught that mitral incompetence was rare, and incidental (or functional) murmurs were diagnosed freely. A mitral lesion without stenosis is not commonly seen at necropsy. Cabot (1926) found only seven cases of mitral incompetence in 4000 autopsies, and three of these he thought were doubtful. Cabot followed many cases of the classical triad of apical systolic murmur, loud pulmonary second sound, and an enlarged heart, but post-mortem there was rarely evidence of mitral incompetence without mitral stenosis. Mackenzie (1925) held almost similar views and regarded mitral stenosis as the essential rheumatic valvular lesion. On the other hand, de la Chapelle, Graef, and Rottino (1934) record the necropsy findings in 7 cases of mitral incompetence. Of 100 necropsies reported by Bland, White, and Jones (1935) on patients dying from rheumatic heart disease below the age of 21, 25 had valve deformity without stenosis: of these 100 cases, recrudescence of the rheumatic infection was directly responsible for 85 deaths. Dana and Reidy (1936) graded 150 cases of mitral disease at the post-mortem examination; pure mitral incompetence was not uncommon, forming in fact one-fifth to one-half of all cases; and the writers expressed the belief that this condition can by itself cause death. Levine (1945) believes that mitral incompetence is occasionally found post-mortem; he reports cases with apical systolic murmurs and subsequent bacterial endocarditis where post-mortem a pure incompetence of the mitral valve is found. Bourne (1946) has recently recorded the necropsy findings in such a case. It seems that pure mitral incompetence though uncommon does occur especially in the young, and proceeds to stenosis later, so that it is rarely seen without it after the age of twenty.

Twelve cases were seen in which doubtless there was mitral disease, but in which the evidence was insufficient to warrant the diagnosis of mitral stenosis. These were labelled mitral incompetence (rheumatic). All had an apical systolic murmur, 6 had a positive rheumatic history, 5 showed slight general enlargement on radioscopy, and 2 others slight enlargement of the left ventricle. Other and almost identical cases as regards signs (described above) were classed as mitral stenosis, mainly because of X-ray evidence.

A greater problem was presented by as many as 68 subjects (25 per cent of the whole group of mitral valvular disease) classed as "probable mitral valvular disease (mitral incompetence)." The alternative diagnosis was a healthy heart with a noticeable but incidental systolic murmur. An apology is perhaps necessary for the size of this group; for in military practice it may be politic to exclude all who carry so suspicious a murmur lest later they should fall sick or claim unjustifiably a pension. In civilian life they might have been recorded as "For observation, ? organic heart disease" and would then have been followed up over a period of months or years. Here also phonocardiography will, in future, prove invaluable. Of this group, 45 gave a positive and circumstantial history of rheumatic fever or chorea in youth. Dyspnoea was claimed by 14, and 1 gave a history of hæmoptysis. A systolic murmur at the apex was heard in 45, varying from soft to moderate in intensity and short to moderate in length. In 24 of them the apical first sound was loud and sudden. In 9 doubt existed as to the presence of a mitral diastolic murmur. Of these cases 30 showed a doubtful or borderline enlargement of the left auricle in the right oblique position; in 8 the heart seemed to be generally though slightly enlarged; in 4 the cardiogram showed right axis deviation.

TABLE IV
CASES THOUGHT TO HAVE MITRAL VALVULAR DISEASE

Mitral stenosis	184
Mitral incompetence	12
Mitral valvular disease probable	68
	264

Mitral stenosis without murmur. In two cases the diagnosis rested on the radiological appearance alone. In one, auricular fibrillation was present and the heart was enlarged (X-ray), the size of the left auricle being diagnostic. The other case had tachycardia, and here the left auricle and the conus were both enlarged. Sosman (1940) has termed this variety the "sub-clinical type."

RADIOSCOPY IN MITRAL VALVULAR DISEASE

The difficulties of clinical diagnosis of mitral valvular disease have been reduced by the use of X-rays. In such inspections it is important to bear in mind the influence of the position of the diaphragm on the cardiac shadow. Just as a high diaphragm by raising the heart and rotating it may give an appearance similar to that of mitral stenosis, so a low diaphragm may conceal the minor changes to be expected in mitral disease.

Our radioscopic findings show no significant difference between the cardiac outline of mitral stenosis in those with a presystolic murmur and in those with a systolic murmur only. Among those with a presystolic murmur only, 104 in all, enlargement of the left auricle was the only certain X-ray change in 77 (74 per cent); in 13 the pulmonary artery and the conus were also enlarged; in a further 14 (13 per cent) there was no change in the size or shape of the heart. Of those with a diastolic murmur, 13 in all, 6 cases had a large left auricle, 4 enlarged pulmonary artery and conus; and 3 showed no change. Of those with an apical systolic murmur and mitral stenosis (60 in all), 40 (67 per cent) had left auricular enlargement, 14 had enlargement of the pulmonary artery and the conus, and a further 6 (10 per cent) showed no change. Thus about 10 per cent of cases of early mitral stenosis show no appreciable changes on radioscopy—a small percentage.

The first and commonest sign of mitral stenosis on the X-ray screen is enlargement of the left auricle; this occurred in about 70 per cent as the sole X-ray sign: it is seen best and earliest, in our opinion, in the conventional right oblique position with barium in the œsophagus. In the early case, enlargement is most noticeable with the subject turned through an angle of 60° rather than 45°; but the optimum angle should be chosen. In this position also the pulmonary artery, with perhaps the "conus" of the right ventricle, appears as a prominence on the anterior border of the heart, giving the appearance of great cardiac depth from front to back as compared with the slim-necked normal organ. Master (1942) considers the left oblique position to be of equal value; and so does Evans (personal communication).

In the right oblique position the course of barium in the œsophagus in the normal is straight

or at most a gentle and slight arc (Fig. 1) ; but in early mitral stenosis, on reaching the upper border of the left auricle, the barium hesitates, *changes its course*, turning backwards before continuing in a curve around the auricle. The bend forming an obtuse angle varies in position, but it is usually high on the posterior cardiac border (Fig. 2, 3, and 4). A wedge of barium may be left at the site of the bend, filling the obtuse angle. Occasionally the convexity may affect not the upper but the middle part only of the auricular curve.

When the left auricular enlargement is but part of a general enlargement, or when this chamber is pushed backwards by a large left ventricle, the barium veers backwards at a high point and runs in a longer curve with its convexity dorsally; there is seldom such a sharp change in direction as in typical mitral stenosis.

An isolated or disproportionate enlargement of the left auricle is almost pathognomonic of mitral stenosis. Apart from a high diaphragm, there are other causes for backward displacement of the œsophagus, for instance, complete heart block (Babey, 1937), pericardial effusion, or patent ductus arteriosus (Donovan *et al.*, 1943). The question as to whether or not the left auricle may enlarge in long-standing auricular fibrillation without mitral stenosis is a matter of opinion; to us it seems probable, but to a limited degree (Babey, 1937; Palmer, 1937). Enlargement of this chamber also occurs in the heart of thyrotoxicosis, but it is more a part of a general enlargement of all chambers.

In the early case in which the right oblique shows a doubtful or borderline enlargement

FIG. 1.—Normal heart. Right oblique position with barium in the œsophagus. Normal left auricular curve.

FIG. 2.—Early mitral stenosis. Right oblique view with barium in the œsophagus. Increased cardiac depth. Barium changes course at a high level with wedging of the barium at the upper border of left auricle as the œsophagus is pushed backwards.

FIG. 3.—Early mitral stenosis. Description as for Fig. 2.

FIG. 4.—Early mitral stenosis. Description as for Fig. 2.

of the left auricle, a straightening of the left border in the anterior view as a separate consideration may assist in coming to the right conclusion on the presence or absence of an early lesion, for the anterior X-ray of mitral stenosis is often also characteristic. The earliest sign is a straightening due to filling-in of the normal concavity of this border by prominence of one or both of the two middle arcs (Fig. 5, 6, 7, and 8).

As some others have done, we deprecate the use of the term "mitralization" for this straightening of the normally concave left border of the heart. By such mixing of the premise and the conclusion in describing an X-ray picture, its real value is diminished. What is seen should be accurately described, and the interpretation for diagnostic purposes should logically follow. Thus straightening of the left border is the observation, and the probable but not the invariable interpretation is mitral stenosis. The upper of the middle arcs is admittedly formed by the pulmonary artery, with or without its left branch. Undue prominence of the upper middle arc (the pulmonary) is almost invariable in established mitral stenosis, but we have only seen it in a small proportion, say 10 per cent, of early cases.

The position as regards the lower middle arc is not so clear. Most cardiologists have believed and taught for years that this arc represents the enlarged conus of the right ventricle which extends forwards and to the left; and that, as the heart rotates to the left, it is thrown

FIG. 6.—Early mitral stenosis. Anterior view. Straightening and lengthening of the left cardiac border by enlargement of the pulmonary artery and “conus.”

FIG. 5.—Normal heart. Anterior view.

FIG. 8.—Early mitral stenosis. Description as for Fig. 7.

FIG. 7.—Early mitral stenosis. Anterior view. Straightening and lengthening of the left cardiac border by enlargement of the pulmonary artery and "conus."

into more prominence. This teaching was founded on post-mortem study and found support from Laubry *et al.* (1939), and Roesler (1943). Robb and Steinberg (1939), and Grishman, Sussman, and Steinberg (1944), dispute this belief from their studies with angiocardiograms. They consider that this arc is formed by the left auricular appendage pushed into prominence by the left auricle enlarging to the left, not only to the right; they are convinced that the right ventricular conus never forms part of the cardiac outline as seen by X-ray. On the whole we accept this view.

Since reading this new conception of the identity of the lower middle arc we have taken every opportunity of noting whether while turning the patient to the left (right oblique position) the prominence disappears or persists, and we often find that it persists. If it were the left auricle or its appendage it might disappear with this rotation of the patient (see also Zdansky, 1939). This is not a valid objection to the newer view. The thickness of the ventricular wall is admittedly to be reckoned with. It may be that the undilated appendage sits upon the projecting conus augmenting the prominence. We do not know to what extent the auricular appendage is distended in life; at necropsy it is usually contracted.

Apart from invariable radioscopy which is part of our cardiac ritual, in order to study the anterior outline of the heart in the early stages of mitral valvular disease teleradiograms were taken of 32 men, 17 healthy students, 8 very early but certain mitral stenotics, and 7 men with mitral stenosis and aortic incompetence. This number is admittedly too small for statistical judgment, but they do give some indications of value. These 32 films were taken in 1943, but examined by each of us separately without reference to the clinical findings.

In 21 of the 32 films there was agreement as to the degree of the *hilar shadows*, in 10 of these 21 they were thought to be large or too dense, or both. Valvular lesions were present clinically in 7 of these 10, mitral stenosis with aortic incompetence in 4, mitral stenosis alone in 3. Among the 8 men with early mitral stenosis alone, there were increased hilar shadows in 3, in the other 5 we were not in agreement; that is in no case of pure mitral stenosis were the hilar shadows considered to be normal by both observers. Among the 7 men with combined valve lesions it was agreed that 4 showed increased markings and that in 1 they were normal; they were disagreement in 2 cases. The hilar shadows were thought to be normal by both observers in 9 of the 17 normal heart films, to be excessive in 3, and there was disagreement in 5 others. The conclusion may be drawn that the estimation of the degree of hilar markings carries a wide margin of error, yet increase in these shadows does occur rather more frequently in early valvular disease than in normal hearts.

The clinical diagnosis of early mitral stenosis had been made in 8 men; one of us (J. P.) recognized mitral stenosis in all these films but thought that in 3 it was associated with aortic incompetence; the other (R. H.) considered that 3 of these hearts were within normal limits and also that aortic incompetence was added to the mitral stenosis in 2 others.

The outline of these 8 hearts with early mitral stenosis was traced from the teleradiograms and they were approximated; for comparison, similar tracings of 8 hearts from normal students are shown (Fig. 9). The difference seems to lie first in the straight left border of the group with mitral stenosis, and secondly in the small aortic knuckle in this group, these two points in combination giving an appearance of added length and straightness to the left border of the heart with mitral stenosis. The buttressing, i.e. extension outwards of the right auricle, was possibly a little more prominent in those with mitral stenosis than in those without it, or their right auricular curve may extend a trifle higher. Fig. 6, 7, and 8 show the earliest changes that we consider to be recognizable in mitral stenosis in the anterior view. Fig. 2, 3, and 4 show the earliest changes in the right oblique position. Examples of normal hearts are shown in Fig. 1 and 5 for comparison.

Of the films, 7 in number, of hearts with the combined lesion of mitral stenosis and aortic incompetence, one of us (J. P.) was correct in 5, diagnosed mitral stenosis alone in 1, and considered 1 to be within normal limits. The other (R. H.) thought that 6 showed the combined lesion and that 1 was within normal limits. Both thought one particular film to be that of a normal heart when it was not. The conclusion to be drawn from the films is that, while enlargement of the left auricle in the oblique position is the most certain early sign of mitral stenosis, the anterior film is often abnormal, and nearly always so if a combined valve lesion is present.

FIG. 9.—Diagram to compare the superimposed outlines of eight normal hearts (A) with those of eight early mitral stenotics; (B) in the anterior view. The difference lies in the straightening and lengthening of the left cardiac border. Buttressing of the right auricle is a less noticeable feature.

To illustrate those changes that we consider to be the earliest in mitral stenosis we have produced the diagrams shown in Fig. 10 and 11. A normal cardiac outline, taken from a teleradiogram, was used as the normal outline shown by the dotted lines. On to this we have drawn the abnormal outline that we believe to be typical of the early condition. This belief is founded on our experience of radioscopy in the early and symptomless case as seen in recruits.

The question of the differential diagnosis of mitral stenosis from mitral incompetence on radioscopy does not appear easy to decide. Dietlen (1923) considered mitral incompetence to be by far the commonest pure valve lesion seen; he gave three points of difference from mitral stenosis in early cases: first, the left auricle is enlarged, but not to the same degree as in stenosis; secondly, the right ventricle is not enlarged except in failure; thirdly, the left ventricle is enlarged in incompetence, but not in stenosis. Assman (1934) says that there is hypertrophy and dilatation of all the heart chambers except the right auricle in mitral incompetence. Roesler (1943) considers mitral incompetence to be rare, and to cause enlargement of the left ventricle. He draws attention to the pulsations in the large left auricle; in mitral incompetence these are exaggerated and, as they alternate with those of the right lower border, cause a see-saw movement which may be quite distinctive. Zdansky (1939) also speaks of enlargement of the left ventricle, and of the left auricle, though not to the same degree as in mitral stenosis.

We have never felt that this differentiation between mitral stenosis and incompetence can be made radiologically with certainty until kymography is successfully applied to the problem. When, in the presence of a mitral systolic murmur, doubtful or certain enlargement of the left ventricle is seen, it is our practice to listen again for an aortic diastolic murmur. If in mitral incompetence the left ventricle enlarges, and if it is admitted that mitral incompetence develops at a later date into mitral stenosis, the question must arise why the left ventricle is not enlarged in mitral stenosis? Does the left ventricle regress as stenosis is installed? This is hard to believe.

FIG. 10.—Early mitral stenosis. Diagrammatic anterior view. Dotted line denotes normal cardiac outline. Straightening of the left cardiac border by enlargement of the pulmonary artery and “conus,” the earliest detectable change in this position.

FIG. 11.—Early mitral stenosis. Diagrammatic right oblique view, with barium in the œsophagus. Dotted line indicates the normal heart outline and the normal œsophageal course. Increased cardiac depth, with left auricular enlargement. Anterior and upper cardiac border is fuller and more rounded than the normal.

AORTIC VALVULAR DISEASE

Aortic incompetence

There is remarkable unanimity as to what is the essential early sign of aortic incompetence; it is the aortic diastolic murmur. In early cases this murmur may be inconstant, present only within a limited range of heart rate, or in one position, or indeed on one occasion and not on another. It is usually heard best on the left of the sternum and follows immediately after the second heart sound.

In view of the high incidence rate of aortic diastolic murmurs heard best on the left of the sternum, it is of interest in surface anatomy to note that Sosman and Wosika (1934) have shown that, in disease at least, the aortic valve lies not behind the second right intercostal space, but behind the centre of the sternum, or sometimes to the left of the mid-line. Zdansky (1939) pictures the aortic valve in health to be just below the junction of the superior vena cava and the right auricle, and lying at the left border of the vertebral column in about the centre of the heart shadow at this level. Laubry *et al.* (1939) also indicate that this is the normal site of the aortic valves. Grishman *et al.* (1944) picture the aortic valve as being rather to the left of the mid-line.

Auscultation should be practised with the subject standing, sitting leaning forwards, and lying. As with mitral stenosis, listening to the patient lying down after exertion is useful, for an aortic diastolic murmur may best be heard during the lower ranges of a falling heart rate. The patient should always be asked to stop breathing for a moment at the end of expiration, for this excludes its noise and the respiratory sound which may closely resemble a diastolic murmur. Sometimes an aortic diastolic murmur is audible at the apex and then its early position in diastole, i.e. immediately after the second sound and before the normal time of the third heart sound, aids in its identification (Evans, 1942a).

That this murmur is frequently overlooked in recruits is confirmed by the report of Delaney *et al.* (1943) who examined 45,000 air cadets and detected heart disease in 100, 43 of whom showed aortic valvular lesions only; these were men who had been examined several times at medical boards and had been passed fit.

In our series, 202 cases of pure aortic incompetence of rheumatic origin were found. This large figure is significant in that it is a measure of the difficulty experienced by doctors in making this diagnosis. The diastolic murmur may be very difficult to detect unless it is listened for with specific intent, and with ears attuned. Too often an apical systolic murmur, present in 56 per cent of these cases, distracted undue attention upon the mitral valve to the neglect of the aortic and, therefore, to the detriment of the true diagnosis.

That aortic incompetence is frequently missed in life is also shown by a comparison between the clinical and pathological incidence (Tables V and VI). The variation among different authors probably rests on their different criteria of diagnosis in double valvular lesions.

TABLE V
THE VALVES AFFECTED (CLINICAL)

Name	No. of cases	Mitral	Aortic	Both
		Per cent	Per cent	Per cent
Hirschfelder, A. D. (1918)	1781	51	22	20
Willius, F. A. (1927)	160	78	13	9
Wilson, Lingg, and Croxford (1928) ..	328	88	0	11
Findlay, L. (1932)	489	88	0	12
Schlesinger, B. (1938)	738	87	1	11
Ritchie, W. T. (1939)	179	83	1	16
Sosman, M. C. (1939)	738	29	22	49

TABLE VI
THE VALVES AFFECTED (POST-MORTEM)

Name	No. of cases	Mitral	Aortic
		Per cent	Per cent
Coombs, C. F. (1924)	97	100	59
Clawson, B. J., <i>et al.</i> (1926)	130	73	63
Glahn, von W. C. (1927)	109	91	58
Findlay, L. (1932)	37	95	59
Bland, White, and Jones (1935)	100	98	71

An aortic diastolic murmur was present in all 202 cases. Dyspnoea was present in 31 (15 per cent compared with 18 per cent in mitral disease). It was recognized during the last war that pure aortic incompetence seldom gave rise to symptoms (Cotton, 1919). Bourne (1940) also found aortic incompetence to be asymptomatic more often than mitral stenosis.

A history of rheumatic fever was obtained in 84 (41 per cent), of chorea alone in 6 (3 per cent), and of both in 8 (4 per cent). The low incidence of aortic incompetence resulting from chorea has often been emphasized (Abt and Levinson, 1916; Strong, 1923; Wallace, 1933; Schwarz and Leader, 1935; and Berghoff *et al.*, 1944).

Peripheral vascular signs are absent in early cases; capillary pulsation, collapsing pulse, and even a large pulse pressure on sphygmomanometry, are to be regarded as confirmatory signs in the developed disease, but render little service in earlier diagnosis.

The X-ray changes in aortic incompetence are confined to the left ventricle and to the aorta. The enlarged left ventricle moves the left border farther out, or the lower and apical portion becomes more rounded, or both these features are present (Fig. 12). In the second oblique position, with the patient turned through an angle of 45°–60°, the rounded left ventricle is in greater contrast with the straight right ventricular arc than in the normal (Fig. 13 and 14).

FIG. 12.—Early aortic incompetence. Anterior view. Left ventricle is enlarged to the left and also more rounded than is normal. Ascending aorta is slightly prominent for a young man.

The heart and aorta show excessive pulsation only if the reflux is great enough. A dynamic dilatation of the first part of the aorta is common in rheumatic aortic incompetence and should not easily suggest syphilis.

The diagrams in Fig. 15 and 16 show the changes that we consider to be the early radioscopic signs of aortic incompetence. Upon normal cardiac outlines in the anterior and left oblique positions, shown by the dotted lines, we have drawn that enlargement which our experience of the early and symptomless case, as seen in recruits, has lead us to consider to be the first typical change.

X-ray changes were present in 169 (84 per cent) of these 202 cases, and absent in 33 (16 per cent). The proportion with enlargement is noteworthy when it is remembered that these were early cases and the majority were asymptomatic. Aortic incompetence was strongly suspected in a further 40 cases which were returned as—rheumatic heart disease (probable aortic incompetence). In this group a combination of positive rheumatic history, symptoms, signs, and X-ray findings made the diagnosis likely, but no diastolic murmur was heard with any certainty. One such case was seen in 1942, and again one year later when a diastolic murmur was obvious.

Aortic incompetence of syphilitic origin was the lesion in a further 4 cases.

FIG. 13.—Normal heart. Left oblique view.

FIG. 14.—Early aortic incompetence. Left oblique view. Rounding and prominence of the left ventricle.

Aortic stenosis

Aortic stenosis alone was present in 14 cases. It is generally considered (Christian, 1931; Clawson *et al.*, 1938; Dry and Willius, 1939; and Reich, 1945) that aortic stenosis is mostly of rheumatic origin. Gibbs (1935) in his necropsy report found this to be so in 90 per cent under the age of 50, of 27 cases studied.

The diagnosis rests on the finding of an aortic systolic murmur and thrill, with left ventricular enlargement. Whether this diagnosis should be made in the absence of a thrill is debatable—we think it may be made. Reich (1945) recorded its presence in only 27 per cent of 22 cases confirmed post-mortem. A thrill was present in 13 of our 14 cases. The stenotic significance of a loud systolic aortic murmur as the main auscultatory sign in a person suspected of rheumatic heart disease cannot easily be discounted, and with left ventricular enlargement aortic stenosis would become the most likely diagnosis, thrill or no thrill. It is often stated that the aortic second sound is absent in aortic stenosis; but this is not true of early cases. In 13 of our 14 cases the second sound was heard; after the loud murmur, attention may then be directed to hearing the second sound. Analysing the 14 cases: 3 gave a rheumatic history, 6 an apical systolic murmur, and 12 exhibited left ventricular enlargement on radioscopy. Analysis of 13 cases of aortic stenosis with incompetence returned a rheumatic history in 1, dyspnoea in 2, an apical systolic murmur in 7, and X-ray enlargement of the left ventricle in all.

COMBINED VALVULAR LESIONS

Aortic incompetence and mitral stenosis

Aortic incompetence in combination with mitral stenosis was present in 72 cases (10 per

FIG. 15.—Early aortic incompetence. Diagrammatic anterior view. Dotted line is normal heart outline. Left ventricle enlarged to the left, and more rounded than the normal. Ascending aorta prominent.

FIG. 16.—Early aortic incompetence. Diagrammatic left oblique view. Dotted line is normal heart outline. Left ventricle enlarged dorsad, and more rounded than the normal. Ascending aorta prominent.

cent) of rheumatic heart disease seen. All these seemed more fully developed than were the single valve lesions and no difficulty was found in their diagnosis.

It must be allowed that 10 per cent of rheumatic cases with this combined lesion does not represent the true figure in all rheumatic heart disease because the grosser cases were graded by the Boards without reference. We believe that this combination is far more frequent than is generally thought, and that the aortic murmur is simply overlooked. Table V shows the clinical frequency as shown by various reported series, and Table VI the post-mortem incidence, the difference between them being obvious.

The findings in our 72 cases are shown in Table VII.

TABLE VII
ANALYSIS OF 72 CASES OF MITRAL STENOSIS WITH AORTIC INCOMPETENCE

Rheumatic history			Dyspnœa	X-ray findings		
Rheumatic fever	..	23 (32 per cent)	21 (29 per cent)	Left auricle enlarged	..	53 (74 per cent)
Chorea	..	6 (8 ")		Left ventricle enlarged	..	45 (63 ")
Both	..	3 (4 ")		General enlargement	..	12 (17 ")
Total	..	44		Normal	..	4 (6 ")

Mitral stenosis, aortic incompetence, and stenosis were present in combination in four cases. A single example of mitral stenosis and aortic stenosis in combination was seen.

Hypertension

The problem of appraising the uppermost limits of the normal blood pressure remains unsolved; for the purpose of admission to the British armed forces a diastolic pressure of 90 must not be exceeded. The question of the significance of a transient hypertension has been much discussed, more in the United States than in this country (Hines, 1940 ; Master, 1943 ; Rogers and Palmer, 1944; and Levy *et al.*, 1944). The conclusions drawn by these

workers is that men showing it, and those with a borderline reading, are more likely to proceed to true hypertension than those without it.

In our 2500 recruits, only 49 (2 per cent) were seen with hypertension, i.e. 6 per cent of all those with heart disease. As noted above, this figure is not a true representation of the frequency of hypertension as compared with rheumatic heart disease. In 45 the systolic and diastolic pressures were both raised, in 2 the systolic only, and in a further 2 the diastolic only. The figure of 160/90 was taken as borderline. The average reading for all these cases was 176/104.

We think that the upper limit of the systolic pressure (after the exclusion of renal disease and coarctation of the aorta) might with safety be placed higher than 160 mm., provided that the diastolic pressure is 90 or below, and that there is no evidence of left ventricular enlargement on radioscopy.

CHANGES OF PULSE RATE

Tachycardia. A simple nervous tachycardia seemed worthy of note in 72 recruits. The pulse ranged between 90 and 130, but usually it fell after rest and as the man became more accustomed to his examiner.

In the United States a persistent heart rate of 100 or over is a cause for rejection (White, 1943). Hoskin (1942) considers that nervous tachycardia should be graded low for service. We believe that a slightly more liberal attitude should be adopted, a rate of 120–130 which tends to fall on resting, in the absence of thyroid disease, may be accepted for full service. We would agree that a persistent rate of 100 should lead to a careful search for a cause other than simple nervousness, such as pyrexia, pulmonary disease, and thyrotoxicosis. Excessive smoking or consumption of alcohol may accelerate the pulse rate.

Bradycardia. This is seldom worthy of note. When the rate is under 50 an attempt to raise the pulse by exercise should be made; when under 40 an electrocardiogram is indicated. Parkinson (1936) has emphasized that simple bradycardia may produce a natural enlargement of the cardiac shadow on X-ray. Two such cases were seen; one because a radiological opinion had been sent to the medical board previously that the heart was enlarged; the other because of the co-existence of a soft systolic murmur.

In 1000 consecutive cases, 31 were referred for irregularity of the pulse.

Extrasystoles	16
Sinus arrhythmia	10
Auricular fibrillation	3
Auricular flutter	1
Tachycardia with ventricular escape	1

In all, 5 cases were seen with auricular fibrillation of undetermined ætiology.

Cardiac enlargement

A difficult problem arose in 25 recruits in whom the heart was enlarged on X-ray, but the cause could not be found. In view of the age group they must be regarded as either congenital or rheumatic. We believe that some of these will eventually develop an aortic diastolic murmur, and this we saw to occur in one case. A further 41 were referred on account of an apparent enlargement from a displaced apex-beat; but actually there was no enlargement, and the displacement was due to scoliosis, which was often more verified on radioscopy than on clinical inspection.

Congenital heart disease

Among the 2500 men seen, 71 had congenital heart disease compared with 609 with rheumatic heart disease.

Pulmonary stenosis	21
Patent ductus arteriosus	10
Atrial septal defect	9
Ventricular septal defect	8
Aortic stenosis	2
Congenital dextrocardia	1
Coarctation of the aorta	1
Right-sided aortic arch	1
Unclassified	18
	71

Finally we would like to support a recent statement by Moir and Shirley Smith (1946) that "the remarkable low incidence of cardiovascular disorders in the Army (less than 7 per 1000 of all sick admitted to hospital) reflects much credit on Recruiting Medical Boards who succeeded in eliminating at the early stage the vast majority of patients with organic cardiovascular disease and to a large extent weeding out those prone to effort syndrome and other neuro-circulatory upsets."

SUMMARY AND CONCLUSIONS

Medical Boards referred 2500 recruits to one of us for a cardiological opinion before acceptance for military service, and of these, 609 (24 per cent), about a quarter, were judged to have chronic rheumatic valvular disease. The commonest of reasons for reference was the presence of a systolic murmur; others were displacement of the apex-beat, an altered first heart sound, a noticeable third heart sound, arrhythmia, and tachycardia.

Incidental (or functional) murmurs were heard in 259 men (10 per cent). The basis for recognition was their softness and shortness, their variability with posture, respiration, and heart rate. An incidental murmur is seldom loud or long, unless it is of the "late-systolic" variety. A heart found to be of normal shape and size on radioscopy was valuable, and corroborative evidence in deciding that a murmur was incidental. Military as well as medical reasons will decide whether the occasional recruit with a noticeable yet innocent murmur should be accepted for war service.

A split *first-sound* at the apex is a normal variation often mistaken for a presystolic murmur; and slight modifications in the quality of the first sound are negligible. Only in the absence of tachycardia is a loud and abrupt first sound suggestive of mitral stenosis.

Mitral valvular disease was present in 264 men, 32 per cent of all rheumatic cases (609). Pathological evidence is quoted to support the view that mitral incompetence, without stenosis, must occur clinically though it is rare except perhaps in early life. If there is doubt the term mitral valvular disease is convenient.

(a) *Mitral stenosis with a presystolic murmur.* 104 cases; all had a loud first sound. In 77 cases enlargement of the left auricle was the characteristic X-ray change: 5 other cases had a loud first sound without a presystolic murmur, and yet X-rays left no doubt that mitral stenosis was present. A mitral diastolic murmur only was heard in 13 other cases.

(b) *Mitral stenosis with a systolic murmur.* 60 cases; strong rheumatic history in over half, moderately or very long and loud murmur in all, loud first sound in some and X-ray evidence in 54. In 9 of the 60 cases there was a systolic thrill. In 12 other cases, similar evidence supported in 7 by X-ray enlargement of the heart (though not of the left auricle) seemed to justify a diagnosis of rheumatic mitral incompetence.

(c) *Mitral valvular disease (probable).* 68 cases; the history, symptoms, and signs together strongly favoured the diagnosis, but they were not conclusive. In half of them, our X-ray note was "doubtful enlargement of the left auricle." Rejection was always advised.

(d) *Mitral stenosis without murmur.* In two cases, one with auricular fibrillation, no abnormal auscultatory signs were found, but the enlargement of the left auricle justified the diagnosis of mitral stenosis.

Aortic valvular disease, without mitral stenosis was present in 229 men.

(a) *Aortic incompetence alone.* 202 cases; all had an aortic diastolic murmur; more than half had also an apical systolic murmur of no great length or intensity. In none was the left auricle enlarged, but in 169 cases (84 per cent) there was enlargement of the left ventricle.

Aortic incompetence alone or in combination with mitral stenosis is far commoner than usually thought. Pathological evidence is quoted in support of our belief. During routine auscultation the possibility of aortic incompetence should always be born in mind, and especially perhaps where there is a systolic murmur at the apex otherwise unexplained.

(b) *Aortic stenosis alone.* 14 cases; 13 had an aortic systolic thrill and yet the aortic second sound was present; 12 showed enlargement of the left ventricle.

(c) *Aortic stenosis and incompetence.* 13 cases; all had enlargement of the left ventricle.

Combined valvular lesions. Aortic incompetence and mitral stenosis. 72 cases, i.e.

10 per cent of all rheumatic valvular disease seen. Aortic stenosis also was present in 4 other cases.

Radioscopy

This proved to be invaluable in early diagnosis, but electrocardiography was of little value. In no less than 80–90 per cent of these early cases convincing X-ray evidence in support was found.

In the right oblique position the abnormal auricular curve is perhaps seen better on radioscopy than in a film as the course of barium in the œsophagus may be followed; this method also allows the subject to be turned until the optimum position is reached. In the right oblique view the anterior cardiac border in mitral stenosis is prominent and the normal slim neck of the heart becomes filled in and convex.

Left auricular enlargement in mitral stenosis and left ventricular enlargement in aortic incompetence are easily the most important early changes. The enlarged left auricle is confirmed as a sign equal in importance to the presystolic murmur in the diagnosis of mitral stenosis.

Diagrams are shown to illustrate those X-ray changes that our experience in radioscopy of the early and symptomless case has led us to accept as the early signs of mitral stenosis and of aortic incompetence.

A high diaphragm, a trivial scoliosis, or a simple bradycardia simulate (in radiology) an early mitral stenosis or an early aortic incompetence. Either lesion may be concealed by a low diaphragm.

In early mitral stenosis the filling in of the left border combined with a small aortic knuckle, gives a characteristic appearance of added length and straightness to this border. Extension upwards of the right border (buttressing) is unconvincing as an early sign. The density and extent of hilar markings vary so much in health that the occasional increase seen in early mitral stenosis has little value in diagnosis.

In early aortic incompetence the left ventricle enlarges as is shown both by extension outwards of the left lower border and by increased convexity of it. These together are regarded as the earliest signs of left ventricular enlargement. This enlargement is best confirmed in the left oblique position.

Confirmatory teleradiograms were taken in a small series of early valvular disease and in controls, and a few are reproduced in illustration.

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