

HEART MURMURS AND THEIR VALUE IN  
DIAGNOSIS AND PROGNOSIS IN AFFECTIONS OF  
THE MITRAL AND AORTIC VALVES.

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In this thesis the writer will show that the correlation of heart murmurs in aortic and mitral disease, with other evidence of cardiovascular disease is an integral part of the constructive art of diagnosis, and no less important a part in the more exacting art of prognosis. It will be shown that the significance of heart murmurs is at its maximum in patients presenting with few or no signs of heart disease, and that in the presence of signs indicating a failing cardiac reserve the value of the heart murmurs in prognosis diminishes, although they still retain their original value in diagnosis.

It will be shown that there are definite features and characteristics which differentiate heart murmurs with potentially grave implications, from those threatening no hazard to the individual, and mention will be made of the assistance to the clinician afforded by radiography and phonocardiography.

It will be shown that the attachment of any diagnostic and prognostic significance to heart murmurs must presuppose a thorough investigation for diseases which are not primarily of cardiac origin. This will include the search for such varied conditions as thyrotoxicosis, severe anaemia, hypertension, chronic pulmonary diseases and aneurysm of the aorta. Until a definite diagnosis has been made, it will be suggested that no prognosis be offered.

The widening scope of operative treatment for cardiac patients which justifies operation in some cases of mitral and aortic valvular disease, will be mentioned, to show a more recent responsibility for accurate diagnosis and prognosis in those with heart murmurs, which the physician is being required to accept in an ever increasing degree.

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Bramwell (1955) describes a series in which this sound was present in 43% of men under 20 years and in only 10% of men over this age. Splitting of the heart sounds may cause confusion with heart murmurs and will be considered in detail in a later section.

The production of heart murmurs arises from the combination of several factors. In the normal heart the relationships are such that no sound is produced when blood passes from one chamber to another through a communicating orifice. If the cavity into which the blood flows is enlarged, and the communicating orifice remains normal then a murmur is produced. This factor is responsible for the disappearing murmur in diastole which occurs in some cases of rheumatic carditis and described by Zilli and Gamna (1954).

This murmur is explained as being due to a relative mitral and tricuspid stenosis caused by ventricular dilatation, or in the absence of demonstrable dilatation, to oedema of the mitral leaflets. This rumbling diastolic murmur disappeared in about two-thirds of those in whom it was present initially. It is also the case that when the chamber into which the blood flows remains normal, but the communicating orifice is altered in size, then a murmur is produced.

In the production of obstructive murmurs several factors are involved, the size of the communicating orifice, the rate at which the blood is flowing through it, and the pressure gradient across the valve. If the production of an obstructive murmur depends on the size of the orifice relative to the rate at which the blood is flowing through it, then an increased blood flow might be expected to produce a murmur in the absence of any alteration to the orifice. In actual fact Bramwell (1943) has shown that in cases where the cardiac output is increased by exercise, or the heart is overactive as in hyperthyroidism, the first heart sound bears a close resemblance to that of mitral stenosis, and may simulate a presystolic murmur.

One cause of the crescendo quality of the presystolic murmur of mitral stenosis is that the final vibrations of the murmur are merged with the initial vibrations of the first heart sound. Another suggested cause is derived from the pioneer work of Hope (1832), on the back pressure theory in the causation of cardiac failure which had, as its basis, the belief that, as an obstacle to the circulation operated on the heart in a retrograde direction, the cavity situated immediately behind it was the first to suffer from its influence. It would follow from this, that a marked degree of mitral obstruction would result in compensatory hypertrophy of the auricular wall. This powerful auricle would then force its contents into the ventricle with great thrust, and the increased pressure gradient at the mitral orifice would manifest itself clinically by a loud crescendo diastolic murmur. Wood, how- (1954) has shown that the presystolic murmur does not indicate the degree of mitral stenosis and may occur with trivial obstruction.

Other general factors which contribute to the production of murmurs are (1) eddying of the blood stream setting up vibrations in the chordae tendineae and muscoli papillares (2) the vibration of diseased and distorted valve cusps (3) roughening of the wall of the chamber as in the atheromatous aorta and in (4) rigid fibrosis and calcification of the mitral ring there is interference with the systolic reduction in circumference and change of shape in the mitral orifice a factor favouring mitral incompetence.

When an additional sound is detected in the cardiac cycle, this may be due to a heart murmur and certain qualities characteristic of types of murmur strengthen this presumption. If the sound has a soft blowing or harsh, rumbling or crescendo quality it is probable that a heart murmur has been discovered. These qualities assist in the differentiation of heart murmurs from the other additional sounds which may be detected in the cardiac cycle. Even/

Even so a murmur may be confused with the other additional sounds heard on auscultation of the heart. A diastolic murmur at the apical region may be confused with the third heart sound or the opening snap of the mitral valve, and it is significant that in the Joint Report of the Rheumatic Fever Working Party of the Medical Research Council of Great Britain and the American Heart Association 1955 on the Treatment of Acute Rheumatic Fever in Children, the analysis of apical mid-diastolic murmurs from the American centres was not included, as a review of the records indicated that the varying terms, mid-diastolic murmur, third heart sound and gallop rhythm were recorded inconsistently by different observers and centres. It is easier to identify the systolic than the diastolic murmur because there are fewer alternatives to a systolic murmur.

The opening snap of the mitral valve is a sharp, high-pitched, clicking sound loudest between the apex and the left sternal border, and occurs earlier in diastole than the third heart sound which is a longer, low-pitched sound loudest at the apex. Mounsey (1953) states that the opening snap occurs 0.07 seconds after the second sound and the third heart sound occurs 0.20 seconds after the second sound.

The opening snap is due to severe mitral stenosis and is absent when the valve is incompetent or much calcified. The third heart sound is heard where the predominant lesion is mitral incompetence and is due to rapid distension of the ventricle. Cleland and others (1954) did not find the absence of the opening snap of value in differentiating between predominant stenosis and incompetence and stated that differentiation between a third heart sound and the opening snap may be difficult, since the third heart sound in mitral incompetence occurs early.

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The timing of murmurs is an integral part of correct diagnosis. This task is greatly simplified if two murmurs are heard at the same auscultatory area, as one murmur must be systolic and the other diastolic. This is exemplified by the to and fro murmurs of aortic incompetence. When only one murmur is audible the task is more difficult, as the murmur must be allotted to systole or diastole, and to which part of each it occupies, by careful timing. There are several methods of examination which help the ear in this undertaking. If the apex beat is palpated during auscultation, it may be possible to relate the murmur to the palpable impulse of systole. If the apex beat is not readily palpable, as is often the case in female or stoutly built male patients, the carotid artery in the neck is always palpable and gives a reliable guide, provided it is remembered that this impulse occurs 0.1 seconds after ventricular contraction. The difficulty of accurate timing is increased by the presence of a rapid heart rate and irregularity of rhythm. Many patients when examined have a heart rate as high as 120 beats per minute, in which case diastole has a length of  $\frac{1}{4}$  seconds and presystole  $\frac{1}{12}$  seconds. Wood (1954) suggested that when tachycardia was present in mitral stenosis the heart might be slowed by carotid sinus compression and the timing and length of the murmur could then be verified. When in addition to rapid heart action there is irregularity of rhythm, usually due to auricular fibrillation, it is often not possible to interpret with any certainty, murmurs which may be present. In these cases, treatment aimed at reducing the rate and irregularity of the heart action will eventually enable the murmurs to be assessed and timed.

The task of identifying murmurs calls for the correlation of associated physical signs. Whenever a heart murmur is heard, its site of maximum intensity, direction of any transmission and the area of propagation must be determined. The presence of an associated thrill may provide valuable complementary evidence.

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A thrill should be sought in every case and only a vibrant purring sensation accepted as a definite thrill and its relation to systole or diastole recorded. Any alteration of the heart sounds in other auscultatory regions should be noted, especial attention being given to the pulmonary area. The effects of change of position on the intensity of the murmur should be assessed, and any change of intensity during respiration should be accurately related to either the inspiratory or expiratory phase.

Phonocardiography is an aid to clinical diagnosis whose value has not yet been fully ascertained. There are two principal instruments in present use. One is the double string galvanometer of Einthoven type introduced by Lewis and used by Evans 1947 and Cowen and Parnum 1949 to record the phonocardiogram and electrocardiogram simultaneously. The other is the cathode ray oscillograph which is of high sensitivity, but is extremely susceptible to extraneous sounds and electrical interference and in addition the photographic records are not as clear as those obtained by the string galvanometer.

The main clinical use of phonocardiography to-day is in the analysis of triple rhythm and the identification and timing of murmurs (Evans 1948).

Evans (1948) showed that the vibration of murmurs as recorded with a string galvanometer are finer and of higher frequency than those of the heart sounds. He believed that the phonocardiograph could aid in the distinction of organic from innocent mitral systolic murmurs, and to distinguish the two groups he used the S line, a line drawn through the end of the S wave of Lead II of the electrocardiogram to touch the simultaneously recorded phonocardiogram. A systolic murmur occurring at the S line was found in the organic group, while a systolic murmur beginning after the S line was found in the innocent group.

Cowen (1949) collected and analysed a series of cases of congenital and valvular disease with systolic murmurs, and showed that these systolic murmurs had no constant relation to the S line.

Evans (1947) found that in cases of mitral stenosis with a systolic but no presystolic murmur on auscultation, that as shown by phonocardiography, the murmur was actually presystolic in time. He also found a mid-diastolic murmur in all of his cases. Cowen (1949) did not substantiate these findings, and believed that the only additional information which the phonocardiograph offered in his series was, that a mitral diastolic murmur occurred more frequently than it was heard.

In a series of 20 cases of aortic incompetence Evans considered that there was evidence of rheumatic aetiology in 16 cases, because phonocardiography revealed the presence of presystolic and mid-diastolic murmurs of mitral stenosis. Cowen in his series thought that no additional information to that afforded by clinical examination was given by phonocardiography in the aetiology of aortic incompetence.

The conclusions drawn by Cowen (1949) were that before cardiology could benefit from the assistance of phonocardiography, an instrument must be used whose basic characteristics were known; and that the electrocardiogram was an unsatisfactory reference tracing, because there was no strict correlation between the electrical and mechanical events of the cardiac cycle.

It would seem then that phonocardiography provides no substitute for careful clinical examination of patients with heart murmurs and it is significant that in a review of mitral stenosis and an analysis of methods used, Wood (1954) states that auscultation was often checked, but rarely influenced by phonocardiography.

But in cases where the presence of a systolic murmur at apex is discovered then phonocardiography may aid in diagnosis, by recording a mid-diastolic murmur of mitral stenosis and as in cases of aortic incompetence, the presence of mitral stenosis may be suspected clinically from the invariable associated systolic and early diastolic murmurs at the apex, the phonocardiograph may help to differentiate the early diastolic murmur of aortic incompetence from the mid-diastolic murmur of mitral stenosis at the apex.



The phonocardiograph may also assist in differentiating an apical systolic murmur due to mitral incompetence from one due to aortic stenosis. Leatham (1951) has shown that the relation of the murmur to the heart sounds on the phonocardiogram is a helpful feature. In aortic stenosis the murmur begins shortly after the first sound, rises to a maximum in mid-systole and ends before the second sound. In mitral incompetence the murmur fills the whole of systole and may obscure the second sound.

Radiology is a science, whose assistance in diagnosis, by providing confirmatory evidence changes in the cardiac outline, is of paramount importance.

Two of the principal methods in current use are cardioscopy and antero-posterior and oblique films. Cardioscopy reveals enlargement of the individual chambers, and the types of pulsation of these chambers may be observed. The films are more valuable as a record of the patients progress.

It is wise to stress that new techniques are primarily designed to advance knowledge. They lead to a greater precision in diagnosis and treatment, but as Bain (1949) stresses, an undue reliance on them at the expense of the clinical examination is to be avoided.

In cases presenting with heart murmurs radiology can throw much light on the possible cause of the murmurs. This is well demonstrated in cases where the features and characteristics of the murmurs point to a diagnosis of rheumatic heart disease.

Where mitral incompetence is suspected, radiology may show characteristic changes in the cardiac outline described by Parkinson (1949) and screening may show expansible pulsation of the left auricle synchronous with ventricular systole, which is seen in the postero-antero as well as the right oblique position.

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In cases where mitral stenosis is suspected, the radiological picture may show hypertrophy of the right ventricle hypoplasia of the aorta and backward displacement of the left auricle and where tight mitral stenosis has been present from and early age, the left ventricle may be very small. In cases of aortic stenosis and incompetence, radiology reveals enlargement of the left ventricle, and Davis and Steiner (1949) showed that tomography could be used to demonstrate calcification in the aortic valve.

This section will be devoted to a consideration of the commoner heart murmurs encountered clinically and with each of the murmurs will be mentioned the associated clinical findings on examination of the heart, which aid in separating the murmurs of aortic and mitral disease from those arising from other causes.

Murmurs tend to fall into three principal groups. Those associated with rheumatic heart disease, with congenital anomalies and with physiological causes.

A loud apical systolic murmur which fills the whole of systole and may obscure the second sound suggests mitral incompetence. This is strengthened by conduction towards the axilla, by the absence of a presystolic murmur Wood (1954), the rarity of an accompanying thrill, diminution of intensity during inspiration, splitting of the pulmonary second sound, a third heart sound and a pulse full of volume. Wood (1954) suggested that a small water hammer type of pulse was an important sign of mitral incompetence, a finding not confirmed by Cleland and others (1955).

A blowing murmur in early diastole, and filling the whole of diastole, at the right and left sternal borders, and usually associated with a systolic murmur in these areas, suggests aortic incompetence. This is strengthened by the rarity of an accompanying thrill, by associated apical systolic and early diastolic murmurs Evans (1948), by diminution of the aortic second sound and by increased intensity during full expiration and when the patient leans forward. This condition was first described as permanent patency of the mouth of the aorta, or inadequacy of the aortic valves by Corrigan in 1832.

A loud rumbling apical mid-diastolic murmur and a crescendo presystolic apical murmur both suggest mitral stenosis. There may be an associated systolic murmur at apex, very often a palpable thrill is present, respiration makes little difference to the intensity of the murmurs, but the mid-diastolic murmur is usually more noticeable in the recumbent position, and especially when the patient has turned to the left side. There may be accentuation or splitting of the pulmonary second sound.

In patients with auricular fibrillation a low pitched rumbling murmur, which completely fills diastole in short cycles, which occurs in early and mid-diastole in long cycles and leaves a gap between the end of the murmur and the first sound suggests mitral stenosis.

The presence of a diastolic murmur at the left sternal border associated with mitral stenosis, may be due to pulmonary incompetence due to an increase of the pressure in the pulmonary circuit, Steell (1888). This murmur is constantly associated with a greatly accentuated pulmonary second sound.

A controversial murmur may be discussed at this stage, namely that described by Flint (1862). This was an apical presystolic murmur occurring in patients with aortic incompetence, and resembling that of mitral stenosis, where no stenotic lesion was demonstrated at post-mortem. Flint (1862) suggested that the regurgitant stream through the aortic valve brought the leaflets of the mitral valve into apposition, and caused a functional stenosis of the mitral valve. Paul White (1926) suggested that the murmur was due to relative stenosis of the mitral valve, secondary to dilatation of the left ventricle. Warburg (1946) suggests that the murmur probably never occurs.

The presence of tricuspid valvular disease may be suspected when a harsh systolic murmur with a thrill, or a diastolic murmur resembling that of mitral stenosis is heard at the lower end of the sternum. These murmurs may also be heard to the right of the lower sternum.

It is now important to exclude tricuspid disease in cases of mitral stenosis, as Sellers (1953) regards tricuspid disease as a contra-indication to mitral valvotomy.

These murmurs are summarised in tabular form.

1. CHARACTER 2. POSITION IN THE CYCLE 3. POINT OF MAXIMUM INTENSITY.	THRILL	TRANSMISSION	CHANGE OF HEART SOUNDS	RELATION TO RESPIRATION & POSITION	ASSOCIATED MURMURS	DIAGNOSIS
1. Harsh and loud systolic, apical	Rarely present	Towards the axilla	Splitting of 2nd pulmon- ary. Third heart sound.	Diminishes during inspiration.	-	Mitral Incompet- ence.
2. Blowing early or complete diastolic right and left sternal borders	Rarely present	None	Diminution of 2nd Aortic	Increases with expir- ation and while lean- ing forward	Systolic & early diastolic at the apex.	Aortic Incompet- ence.
Harsh rumbling, mid-diastolic, apical	Often present	None	Accentuated 1st mitral Accentuated 2nd pulmonary	Louder re- cumbent & lying on left side	Systolic at the apex	Mitral Stenosis
Crescendo, pre- systolic, apical	Often present	None	Accentuated 1st mitral Accentuated 2nd pulmonary	Little or no change	Systolic at the apex	Mitral Stenosis
Harsh rumbling, whole diastole or early and mid-dia- stole, apical with auricular fibrillation	Often Present	None	Accentuated 1st mitral 2nd pulmon- ary	Louder re- cumbent or lying on the left side	Systolic at the apex.	Mitral Stenosis
Diastolic, left sternal border	Rarely Present	None	Greatly accentuated 2nd pulmonary	Little Change	Murmurs of mitral stenosis	Pulmonary Incompetence Steel
Rumbling, presystolic, apical	-	-	-	-	Murmurs of aortic incompetence	Functional mitral sten- osis (Flind
Harsh, systolic or rumbling diastolic at lower end of the sternum	Often present	May be heard to the right of the lower sternum	-	-	Other mur- murs of rheumatic heart disease	Tricuspid Disease 1 3 1

A loud harsh basal systolic murmur beginning after the first sound rising to a peak in mid-systole and ending before the second sound, is due to aortic stenosis. This murmur is conducted towards the neck and often associated with a thrill. The murmur may be associated with a loud apical systolic murmur. The aortic second sound may be diminished and the pulse of low volume in advanced cases. The aetiology is a matter of opinion. Some workers such as Krasner and Koletsky (1947) believe it to be rheumatic in origin, but others such as Kiloh (1950) disagree with this view. Bramwell (1955) suggests that the sex incidence which favours males, the benign course over a number of years, the usual lack of associated mitral stenosis and the rarity of a history of rheumatic infection support the view that the condition is not rheumatic.

1. CHARACTER 2. POSITION IN THE CYCLE. 3. POINT OF MAXIMUM INTENSITY	THRILL	TRANSMISSION	RELATION TO RESPIRATION AND POSTURE	CHANGE OF HEART SOUNDS	ASSOCIATED MURMURS.	DIAGNOSIS
7. Loud, Harsh, systolic, right sternal border.	Often present	Into the neck	Little change.	May be diminished 2nd aortic	Diastolic aortic murmur	Aortic Stenosis

The Second group comprises the commoner congenital heart murmurs. A continuous murmur throughout the systole and diastole, best heard at the second and third left interspaces, suggests a patent ductus arteriosus. There is usually an associated thrill and wide pulse pressure.

A rough systolic murmur at the fourth left interspace associated with a thrill, suggests the presence of a patent interventricular septum.

A systolic murmur in the pulmonary area associated in some cases with an apical systolic murmur and in others with a pulmonary diastolic murmur, with a split second pulmonary sound suggest an interauricular septal defect. This condition may be associated with mitral stenosis to give Lutembacher's syndrome.

A systolic murmur in the pulmonary area often associated with a thrill and a diminished pulmonary second sound, may suggest pulmonary valvular stenosis with closed interventricular septum.

A systolic murmur in the pulmonary area with a thrill, a diminished pulmonary second sound and marked cyanosis occurs in Fallot's tetralogy.



1. CHARACTER 2. POSITION IN THE CYCLE. 3. POINT OF MAXIMUM INTENSITY	THRILL	CHANGE OF HEART SOUNDS	CYANOSIS	BLOOD FLOW TO THE LUNGS	ASSOCIATED MURMURS	DIAGNOSIS
8. Harsh, throughout systole and dias- tole, left upper chest	Systolic or con- tinuous	Diminution of 2nd. aortic	NO	INCREASED	May simulate apical presystolic	Patent ductus arteri- osus
9. Rough, systolic, fourth left interspace.	Usually Present	NONE	NO	INCREASED	-	Inter- tricular septal defect.
10. Systolic pulmon- ary	-	Split 2nd pulmonary	NO	INCREASED	Apical sys- tolic or pulmonary diastolic	Inter- auric- ular septal defect.
11. Systolic, pulmonary	Often Present	Diminished pulmonary second	NO	DECREASED	-	Pulmonary stenosis with closed septum
12. Systolic pulmonary	Often Present	Diminished pulmonary second	YES	DECREASED	-	Fallot's Tetral- ogy.

The interesting condition of coarctation of the aorta occurs in this group, and usually presents with widespread systolic murmurs and a high blood pressure in the arms with lower pressure in the legs. There may be abnormal pulsation in the neck.

The other principal groups contains murmurs due to physiological causes.

Amongst these is a blowing or moderately harsh systolic murmur in the pulmonary area unassociated with any change in the heart sounds or with a thrill. This murmur is heard in patients presenting with anaemia, thyrotoxicosis and acute infections. It is a consequence of the rapidly acting heart.

It is of interest to record that in 1832 Hope described a systolic bellows-murmur, occurring in those under the influence of reaction from excessive loss of blood, whether by the lancet or by haemorrhage.

He also stated that the largest class of individuals in whom the bellow-murmur was apt to occur, consisted of young people of plethoric habit and delicate, irritable temperament, subject to hysterical and hypochondrial affection, and to nervous palpitation.

A short, inconstant superficial apical systolic murmur may be heard. This murmur may disappear during inspiration or expiration. This is a cardiorespiratory murmur and is also an accompaniment of the over acting heart. It is due to increased air entry into the lung during ventricular systole, when a vacuum is created in the region of the heart.

Several murmurs arising as a direct result of cardiovascular disease but not of rheumatic or congenital origin, remain to be considered.

A systolic murmur may appear at the base of the heart and less frequently at the apex, in cases suffering from hypertension. There may be accentuation of the aortic sound and a third heart sound at the apex.

A systolic apical murmur may be heard in the aged patient resulting from dilatation of the heart in disease.

To an fro murmurs may be heard at the base of the heart in cases of aneurysm of the thoracic aorta where there is associated aortic incompetence. Where incompetence is absent a basal systolic murmur often accompanied by a thrill and an accentuated aortic second sound may be heard.

The appearance of a systolic pulmonary murmur, associated with a friction rub over the area of the pulmonary artery is a reliable sign of massive pulmonary embolism. There may also be accentuation of the pulmonary second sound and a third heart sound.

1. CHARACTER 2. POSITION IN CYCLE. 3. POINT OF MAXIMAL INTENSITY.	THRILL	CHANGE OF HEART SOUNDS	ASSOCIATED MURMURS.	RELATION TO RES- PIRATION AND POSTURE.	DIAGNOSIS.
13. Blowing or harsh. Throughout systole. PULMONARY	-	-	-	May disappear with inspiration	Physio- logical
14. Blowing or harsh. Early or mid- systole. Apical.	-	-	-	May disappear during inspir- ation or expiration.	Cardio- respiratory murmur.
15. Systolic, Apex or base of the heart	-	Accentuation of aortic 2nd. May be a 3rd sound.	-	-	Hyper- tension
16. Systolic, Basal.	May be present	Accentuated 2nd aortic sound	May be the murmurs of aortic in- competence	-	Aneurysm of the thoracic aorta
17. Systolic. Pulmonary.	Friction sounds over pulmonary artery	Accentuation of Pulmonary second sound. Third heart sound.	-	-	Pulmonary Embolism
18. Systolic, apical	-	-	-	-	Failing heart in the aged.

This section of the thesis will discuss the features and characteristics which separate heart murmurs of potential gravity, from heart murmurs which threaten no ultimate hazard to the patient. The correlation of the heart murmurs with other signs of heart disease will be shown to play an integral part in building up the diagnosis. It will be stressed that heart murmurs of mitral and aortic valvular disease have greater value in prognosis when the patient shows little evidence of cardiac impairment and that when a failing cardiac reserve becomes increasingly obvious, their value in prognosis diminishes. The importance of excluding non-cardiac causes of heart murmurs will be mentioned. Recent advances in the operative treatment of mitral and aortic stenosis illustrate responsibility for accurate diagnosis of ever increasing importance and will be mentioned in this section.

Fundamental factors which assist in the classification of the significance of heart murmurs are, the position of the murmurs in the cardiac cycle, the point of maximal intensity, area of transmission, loudness, character, and multiplicity, which means the presence of associated systolic or diastolic murmurs in the same or other auscultatory areas.

All murmurs occurring in diastole are significant and much information can be adduced from the precise part of diastole in which they occur.

A tabulated scheme of the murmurs heard in diastole, with their inherent features and characteristics is outlined, and some space devoted to discussion of their more important implications.

Position in Diastole	Site of maximum intensity.	Transmission	Associated murmurs	Loudness	Diagnosis
Mid and late diastole with pre-systolic accentuation.	Apex	Localised	Usually organic apical systolic	Low pitched, rumbling	Mitral Stenosis
Early, or whole of diastole.	Base	Right and left sternal borders and apex	Usually accompanying systolic murmur	Blowing	Aortic Incompetence.
Whole of diastole, runs off from the second pulmonary.	Pulmonary area Inner end of 3rd left costal cartilage.	Down the left sternal border	Murmurs of mitral stenosis	Varies from day to day	Pulmonary incompetence (Steell)
Early diastole	Pulmonary area	-	Pulmonary systolic or apical systolic	Loud	Patent inter-auricular septum
Throughout systole and diastole	Second or third left interspaces near sternum	Over the left upper chest	Accentuation in systole	Harsh and louder in systole	Patent ductus arteriosus
Mid diastole	Lower sternum	To the right of lower sternum.	Tricuspid systolic	Rumbling	Tricuspid stenosis

Aortic incompetence is a lesion which is responsible for many cases of congestive cardiac failure, and the factors responsible for failure are the mechanical effect of the valvular lesion, cardiac enlargement, and myocardial incompetence. This valvular lesion may cause left heart failure or at a later stage right heart failure, due to high grade decompensation of the left ventricle.

It will be observed that the diastolic murmur of this lesion may be heard at three areas, namely at the base of the heart, down the left sternal border and at the apex. An organic systolic murmur is a frequent accompaniment.

The murmur of mitral stenosis commences in mid-diastole, and if a severe degree of stenosis is present, the murmur continues up to the first heart sound. This murmur is rarely heard without an accompanying organic apical systolic murmur when there is evidence of acute rheumatic carditis. There may be present a pulmonary systolic murmur, not transmitted from the apex, and attributable to dilatation of the pulmonary artery or to rheumatic arteritis.

The presystolic murmur of mitral stenosis indicates that a powerful left auricle is forcing blood through a stenosed mitral orifice. Wood (1954) points out that this murmur may occur with only a trivial degree of stenosis. A physiological presystolic apical murmur may be heard in some normal persons when the heart output is increased as in exercise, and in patients with a normal mitral orifice, when the blood flow through the orifice is increased in hyperthyroidism, Bramwell (1943).

If auricular fibrillation supervenes, the diastolic murmur has a crescendo termination when two cycles follow in quick succession, but when diastole is longer, there is a silent interval between the murmur and the succeeding first sound, and there is no crescendo termination. The diastolic murmur of mitral stenosis may appear early in the course of acute rheumatic carditis.

A diastolic murmur may also be heard in almost pure mitral incompetence but it is usually short and associated with a third heart sound.

The presence of a stenosed tricuspid valve may be suspected when a murmur similar to that of mitral stenosis is heard at, or to the right of the lower sternum. Lewis (1933) stated that the diagnosis should be confined to cases of mitral stenosis, and that even when made it did not affect the management of the case. Since the advent of mitral valvotomy this view has been superseded, because Sellors and others (1953) consider that triscupid valvular disease is, in the majority of cases, a contra-indication to mitral valvotomy. The lesion has therefore, an importance in the assessment of cases for cardiac surgery and must be diagnosed if present. However Brock and others 1955 report two excellent post-operative results in two patients with large hearts from tricuspid disease, maintained for the three year survey.

A diastolic murmur may be heard in the pulmonary area in a quarter of cases with patent interauricular septum, and is usually associated with a pulmonary or systolic apical murmur. In congenital heart disease, the presence of<sup>a</sup> diastolic murmur, generally at the pulmonary area, but also heard towards the apex, indicates an increased blood flow to the lungs, and means that the patient cannot be aided by surgical procedures designed to augment the pulmonary blood supply.

Unlike diastolic, some systolic murmurs have little clinical significance and certain criteria have been evolved, which assist the clinician to separate those of significance from the unimportant.

A tabulated scheme similar to that used in the classification of diastolic murmurs is outlined below, and some points of interest will be discussed in more detail.



POSITION IN CYCLE	AREA OF MAXIMUM INTENSITY	TRANSMISSION	ASSOCIATED MURMURS	CHARACTER	DIAGNOSIS
Throughout systole	Apical	To or towards axilla and back.	-	Harsh	Mitral Incompetence.
Throughout systole	Aortic	Towards the neck	Systolic at apex and dia- stolic aortic	Often Loud	Aortic Stenosis
Throughout systole	Pulmonary	Over the left upper chest	-	Loud	Pulmonary sten- osis, relative- ly uncommon.
Throughout systole	Pulmonary	Over the upper precordium	-	Blowing or moderately harsh	Physiological relatively common.
Throughout systole	Pulmonary	-	pulmonary diastolic or apical systolic	Loud	Patent inter- auricular septum.
Systolic	Lower sternum	To the right of the lower sternum	-	Harsh	Tricuspid Incompetence.
Systolic	Basal or apical	-	Systolic at aortic region	Blowing	Hypertension
Systolic	Apical	Localised	-	Blowing or Harsh	Cardiorespira- tory

Much has been written about the relationship between the presence of an apical systolic murmur and the diagnosis of mitral incompetence. Balfour, realised that mitral incompetence was not necessarily due to a damaged mitral valve, and used the term "curable mitral incompetence" for the apical systolic murmur heard in cases of chlorosis. Carey Coombs believed that an apical systolic murmur was heard in mitral incompetence, but unless it was long, loud and high-pitched, could not be attributed to endocarditis. Lewis (1933), described an apical systolic murmur as the only sign of mitral incompetence, and considered that its main value was as a guiding sign attracting attention, and leading to a closer examination of the heart. He maintained that the value of a diagnosis of mitral incompetence was reduced, because it was rarely possible to assess the degree of reflux.

Fishberg (1940) believed that the importance of the apical systolic murmur had been underestimated and in recent years the advent of mitral valvotomy makes a diagnosis of mitral incompetence of considerable importance, as it is almost always a contra-indication to valvotomy. Wood (1954) describes a series in which no case of significant mitral incompetence was encountered without a systolic murmur of at least moderate degree.

The report of the Medical Research Council (1955) on the treatment of acute rheumatic fever placed the apical systolic murmur into one of the five grades, according to loudness and conduction to or towards the axilla or back.

Aortic stenosis is an interesting valvular lesion which causes an apical systolic murmur, in addition to the more typical, harsh, systolic murmur at the base of the heart. The murmur may be confused with that of mitral incompetence, as both lesions cause left ventricular enlargement and in both the pulse may be of full volume. Leatham (1951) has shown that, as seen on the phonocardiogram, the murmur of aortic stenosis begins shortly after the first sound, rises to a maximum in mid-systole and ends well before the second sound, whereas in mitral incompetence the murmur fills the whole of

of systole and, if loud, may obscure the second sound at the apex.

As regards the aetiology, present opinion favours a non-rheumatic origin, possibly due to a non-specific infection producing endocarditis in early life. In a recent study, however, Zilli and Gamna found that in patients between the ages of two and fifteen years suffering from a first attack of rheumatic fever, and displaying an apical systolic murmur, one in five of the patients had, in addition a non-transmitted aortic systolic murmur.

This murmur persisted throughout the period of observation, which varied from several weeks to three years, and was interpreted as indicating organic aortic stenosis. In addition Levine and others (1954), believe that in 90 per cent or more of cases of aortic stenosis, the cause is a rheumatic infection.

Bramwell (1955) believes that rheumatic carditis generally leads to incompetence of the aortic valve, rather than to stenosis, because the cusps of the aortic valve are discrete, so that when cicatricial contraction follows inflammation they tend to be drawn apart.

Campbell and Kauntze (1953) believe that pure aortic stenosis under the age of thirty is more likely to be congenital than rheumatic.

Aortic stenosis is one cause of right ventricular stenosis, which is caused by a grossly thickened interventricular septum encroaching on the cavity of the right ventricle, and causing gross congestive failure without pulmonary embarrassment, in the presence of a predominantly left-sided lesion.

The majority of systolic murmurs heard in the pulmonary area, are, physiological, due to rapidly acting heart, as in anaemia, hyperthyroidism, acute infections and following extreme exertion. This type of murmur disappears when the underlying cause is removed. Other pulmonary murmurs occur where there is an underlying congenital lesion. These include pulmonary stenosis, patent interauricular septum and, the loud very harsh murmur of patent inter-ventricular/

inter-ventricular septum, heard to the left of the mid-sternum and transmitted widely over the left upper chest.

These should be differentiated from a systolic, non-transmitted pulmonary murmur sometimes heard in mitral stenosis and attributed to dilatation of the pulmonary artery or to rheumatic arteritis.

The systolic murmur of hypertension is due to the forcible heart action and in late stages, to dilatation of the left ventricle causing functional regurgitation.

The apical cardiorespiratory murmur is caused by a rapidly acting heart, and may be recognised by the fact that it disappears when respiration is voluntarily suspended, and perhaps also during either inspiration or expiration. It is due to the creation of a vacuum in the region of the heart during ventricular systole.

The systolic murmur of tricuspid incompetence has the same significance, with regard to suitability for mitral valvotomy, as has the diastolic murmur of tricuspid stenosis.

There are certain well-defined signs of heart involvement which must be integrated with the presence of heart murmurs in order to reach a reasoned diagnosis of mitral or aortic valvular disease.

These signs include enlargement of different cardiac chambers, accentuation, diminution, and splitting of the different heart sounds, the presence of a third heart sound or the opening snap of the mitral valve, and whether there are signs of subacute bacterial endocarditis.

Both clinical and radiological evidence of enlargement of the cardiac chambers can provide valuable confirmatory evidence in patients presenting with heart murmurs suggesting mitral or aortic lesions.

Left/

Left ventricular enlargement may be recognised clinically by movement of the apex beat to the left of the outer limit of normal, usually accepted as from three to four inches to the left of the mid-sternum. Lewis (1948) states that when the outer border of the cardiac impulse is situated  $4\frac{1}{2}$  or more inches to the left of the mid-sternal line, cardiac enlargement is present, especially if the impulse is in sixth interspace.

The apex beat in left ventricular enlargement has a sustained, localised thrust over the apex. It is a conspicuous feature of all but the earliest cases, of aortic incompetence. In the more severe grades of hypertension there is clinical enlargement of the left ventricle and in mitral incompetence there is also marked enlargement. Aortic stenosis does not cause noticeable left ventricular enlargement in the early stages, but when early heart failure due to this lesion occurs, then the displacement becomes prominent and since both lesions may present with a loud systolic murmur at the apex, may cause confusion with mitral incompetence.

Congenital lesions associated with left ventricular enlargement include patent ductus arteriosus, coarctation of the aorta, and tricuspid atresia with patent interauricular septum.

There is no left ventricular enlargement, unless there is associated hypertension, in anaemia, hyperthyroidism and chronic pulmonary disease.

In right ventricular enlargement the apex is likewise moved beyond the outer limit of normal. In this event the apical impulse is diffuse, and between the apex beat and the sternum a blow may be felt which resembles that from an open hand. The first sign of right ventricular enlargement is a palpable systolic impulse in the third left interspace due to enlargement of the infundibulum. Wood (1954) considers the force of this thrust to be an indication of the degree of pulmonary vascular resistance.

Mitral/

Mitral stenosis is a frequent cause of right ventricular enlargement and in severe degrees of stenosis the apex beat has a tapping quality. Congenital heart lesions causing this type of enlargement are, patent interauricular septum, pulmonary valve stenosis, and pulmonary valve stenosis with a right to left shunt through a patent foramen ovale.

Right auricular enlargement is a reliable sign of tricuspid disease, especially when associated with the typical murmurs at, and to the right of the lower sternum, and with gross right ventricular enlargement.

Enlargement of the left auricle occurs in mitral stenosis, and powerful hypertrophy of this chamber, in the absence of auricular fibrillation, is responsible for the crescendo pre-systolic murmur of mitral stenosis. Giant enlargement of the left auricle occurs in mitral incompetence and pericardial adhesion, and may reach aneurysmal proportions. Wynn (1953) found that significant mitral incompetence in the presence of mitral stenosis, could be detected by certain criteria, including a very large or aneurysmal left auricle. When the left auricle enlarges, it extends outwards to the right of the sternum and may reach to the lower right axilla in giant or aneurysmal dilatation. Radiological examination gives more precise information regarding selective enlargement of the different chambers when enlargement is suspected clinically.

The first heart sound is produced by auricular and ventricular contraction, and the second by closure of the aortic and pulmonary valves. The second sound in the pulmonary area is caused, partly by aortic closure, but mainly by pulmonary closure, and the second sound in the aortic and mitral regions partly by pulmonary, but mainly by aortic closure. There may be accentuation, diminution or splitting of the heart sounds.

The first sound at the mitral area may be accentuated in mitral stenosis, hypertension, or in health especially when associated with tachycardia. The presence of auricular fibrillation dampens the intensity of the first sound so that in patients with auricular fibrillation the first sound may not be markedly accentuated.

The accentuated first sound in mitral stenosis is due to the pressure gradient across the mitral valve billowing the cusps deeply into the left ventricle up to the moment when it contracts. The accentuated first mitral heart sound is an indication that mitral stenosis may exist, but does not indicate the degree of the obstruction. The second mitral and aortic sounds may be accentuated in early aortic incompetence and a soft blowing early diastolic murmur may be heard or elicited in the aortic area or down the left sternal border.

Even when an accentuated pulmonary second sound appears to be single, splitting can nearly always be demonstrated during deep inspiration. The second pulmonary sound refers to the second or pulmonary element of the second heart sound and is rarely heard outwith the pulmonary area. The sound may be accentuated in patients with mitral stenosis, congenital heart disease, hyperthyroidism and is also a common finding in healthy persons. It is also a constant feature in patients with a Graham Steell murmur of high pressure in the pulmonary artery.

Diminution of the first mitral sound most commonly occurs in mitral incompetence, probably because rapid ventricular filling, and the rapid equalisation of left atrial and ventricular pressure floats mobile cusps into apposition before the ventricle contracts. When the valve cusps are rigid and immobilised by calcification, as often occurs in mitral incompetence, the first sound may be diminished Wynn (1953). When auricular fibrillation occurs in the course of mitral stenosis the intensity of the first mitral sound tends to be damped down.

A high degree of pulmonary vascular resistance may diminish the intensity of the first sound by causing an enlarged right ventricle to form the apex beat.

The second aortic sound may be diminished in the presence of aortic stenosis or incompetence.

Accentuation and splitting of the second sound at the pulmonary area are usually caused by increased pressure in the pulmonary circuit, /

circuit, as occurs in mitral stenosis. In normal health the aortic valve closes before the pulmonary valve and the two components may be heard in healthy children and young adults. Splitting of the second sound must be carefully differentiated from the opening snap of the mitral valve. The split second sound is always more easily detected at the pulmonary area, whereas the opening snap is best heard at a point between the apex and the left sternal border. The opening snap occurs from 0.03 sec. to 0.07 sec. after the second sound, whereas the second component of the split second sound occurs 0.03 sec. after the first component. Inspiration increases the gap between the two components of the split second sound, but has no effect on the position in time of the opening snap. Where left ventricular systole is short, as in mitral incompetence, the two components of the split second sound are more widely separated.

Splitting of the first mitral sound must not be confused with the presystolic murmur of mitral stenosis and Evans (1948) suggested the following differentiating features. The split first sound may be represented phonetically as r - rup, unlike the pre-systolic murmur thur - rup, the split first sound is better heard toward the midline and there are no associated signs of heart disease.

Two additional groups of sounds heard in the cardiac cycle are triple rhythm and the opening snap of the mitral valve.

The additional heart sound giving rise to triple rhythm may be proto-diastolic, pre-systolic or summation gallop in time. Its presence is due to rapid distension of the left ventricle. The additional sound in protodiastole is known as the third heart sound and may be heard in health, in mitral incompetence and in right ventricular failure. The additional sound in pre-systole is known as the fourth heart sound and is commonly due to left ventricular failure caused by hypertension and only very rarely to aortic incompetence, Evans (1948).

The/



The third heart sound is a long low pitched sound best heard at the apex, and according to Mounsey (1953) occurs 0.20 sec. after the second sound. It is heard when mitral incompetence predominates over mitral stenosis. It may occur in healthy adults under the age of forty and to have significance below this age, it must be integrated with other evidence of heart disease.

The opening snap of the mitral valve is ascribed to the presence of severe mitral stenosis with the pliant diaphragmatic type of valve ideally suited for operation. It is absent when mitral incompetence predominates and when the valve is much calcified, Wynn (1953). It was formerly ascribed to a duplicated second sound and is a sharp high-pitched clicking sound heard, at its loudest, between the apex and the left sternal border.

Cleland and others (1955) did not find the absence of the opening snap to be of value in differentiating between predominant stenosis and incompetence, and suggest that differentiation between a third heart sound and the opening snap may be difficult, since the third heart sound in mitral incompetence occurs early.

The presence of subacute bacterial endocarditis yet constitutes a serious threat to patients suffering from well-compensated valvular lesions. Despite the use of antibiotics the recovery rate is not higher than 70%. This complication is rarely met with in patients with severe cardiac failure, or in those with auricular fibrillation. The prognosis is worst in those with aortic incompetence and in patients who develop cardiac failure in the course of the infection, Bunn and Cook (1954). The occurrence of subacute bacterial endocarditis is suspected when a patient with a valvular lesion develops fever, embolic phenomena, pallor or splenic enlargement.

We now turn to the main substance of the thesis, an attempt to evaluate the relative importance in prognosis of mechanical obstruction due to disease affecting the heart valves and causing organic heart murmurs and of myocardial incompetence caused by weakening of the heart muscle by disease processes. In/

In 1854 William Stokes originated the forward failure theory to explain the causes of heart failure and stressed the importance of myocardial incompetence, in patients with valvular lesions. He believed that it was necessary to decide whether heart murmurs originated from an organic valvular lesion and then to assess the condition of the muscular portions of the heart, by the presence or absence of signs indicating an impaired or failing cardiac reserve.

Two powerful advocates of this belief, men of stature who based their expositions on personal clinical observations were Sir Thomas Lewis and Sir James Mackenzie.

Lewis (1933) believed the part played by the valvular components in heart failure had been over-emphasied, and stated categorically that the only basis of prognosis was the correlation of signs and symptoms indicating the early onset of an impaired cardiac reserve, caused by a weakened myocardium. These included cardiac enlargement, a reduced exercise tolerance, abnormal heart rhythm and signs of commencing congestive changes. He maintained that to base prognosis on the burden imposed by a valvular defect was uncertain and undesirable. The valvular component, in his opinion, was of greatest significance in patients who displayed no evidence of an impaired cardiac reserve. In accordance with these views Lewis graded patients with organic heart murmurs into five prognostic groups. Those with an organic valvular lesion and no other signs, except perhaps a slight impairment of exercise tolerance after severe exertion, whose prognosis extended to many years of active life. Patients with slight cardiac enlargement, fair exercise tolerance and auricular fibrillation might expect to live at least ten years,

years, as could those with moderate cardiac enlargement, poor exercise tolerance and normal rhythm. Those with moderate cardiac enlargement and auricular fibrillation and those with great cardiac enlargement, poor exercise tolerance and normal rhythm might anticipate from three to six years. A similar prognosis might be extended to those with congestive cardiac failure relieved by treatment. When gross enlargement with acute pulmonary congestion or congestion unrelieved by medical treatment supervened, then life was rarely prolonged more than two years. The final group included patients in the terminal phases of persistent heart failure, in whom death was always an imminent possibility.

Lewis stressed that when a patient was placed in the more advanced grades where evidence of a failing cardiac reserve became more importunate, then the importance of the mechanical factor due to valvular defect became relatively unimportant<sup>t</sup>..

Recent clinical and physiological evidence has shown that the fundamental cause of heart failure is the inability of the diseased heart to increase its output in response to a rise in the venous pressure, and that this rise in venous pressure is a compensatory mechanism.

The mandate of this thesis does not arrogate to itself a discussion of the causes of heart failure, nevertheless it may be mentioned that the pioneer theory of backward-pressure failure originated by John Hope in 1832 has some place in present views. Hope believed that as an obstacle to the circulation operated in a retrograde direction, the cavity immediately behind the obstruction was the first to suffer. Modern knowledge has revealed that certain features described by Lewis as indicating a failing cardiac reserve are due to the mechanical effect of the valvular lesion. This knowledge clarifies understanding of the processes involved, but does not alter the principle that, in organic heart disease, signs of a failing cardiac reserve have more prognostic significance than the features and characteristics of the heart murmurs. In short, signs and symptoms of the failing reserve remain the foundation of accurate prognosis, although the mechanical factors causing the heart murmurs also produce many of these signs and symptoms.

The advent of heart surgery in the relief of mitral and aortic stenosis has compelled discrimination between the mechanical burden imposed by the valvular lesion and the burden imposed by a defective myocardium. When patients with mitral stenosis develop dyspnoea, paroxysmal dyspnoea with hemoptysis and acute pulmonary oedema, these are caused by back pressure, due to obstruction of the mitral valve, causing persistent pulmonary congestion. Increased right ventricular pressure is opposed by mitral obstruction and leads to an increase of pulmonary capillary pressure.

The onset of auricular fibrillation in mitral stenosis, usually at the stage of severe pulmonary congestion and provoked by the patient's age and distension of the left atrium, indicates myocardial damage which has weakened the auricular wall and caused it to lose efficiency under the burden imposed by the valvular lesion.

Right and left ventricular enlargement indicate myocardial damage mechanical or inflammatory.

Since the surgical treatment of certain patients with heart disease became possible these signs have a further importance in prognosis. Gross right ventricular enlargement is a relative and left ventricular enlargement: a complete contra-indication to heart surgery. Auricular fibrillation occurs in the more advanced cases of heart disease which carry a higher operative risk. Brock and others (1955) in a recent follow up of forty-five cases of mitral valvotomy have found that, it is the anatomical operative result rather than the pre-operative rhythm which is the decisive factor in prognosis.

The presence of a long loud mitral diastolic murmur is an important prognostic sign, since it means that a powerful hypertrophied auricle which has not begun to fibrillate is opposing a gross degree of mitral obstruction. The combination of signs indicating gross mechanical obstruction and the relative absence of signs suggesting myocardial weakness is ideal for surgery, Sellors (1953).

Much more is now known of the natural history of patients with rheumatic valvular lesions. In order to determine the prognosis in mitral stenosis when it was not treated surgically, Wilson and Greenwood (1954) reviewed a series of patients with mitral stenosis. They found that almost all with asymptomatic mitral stenosis when first seen had remained symptom free for the fifteen years covered by the survey. In the group with slight disability when first seen, five of the twenty had died and in three others the disease had progressed to later stages. After the onset of severe pulmonary congestion with orthopnoea, dyspnoea both paroxysmal and nocturnal 16 per cent of the patients died within six months and 50 per cent within five years.

After the commencement of right heart failure, 24 per cent of the patients died within six months and 50 per cent within three years. The average age at death was 45.8 years and only 38 per cent of the patients survived to over the age of fifty years. They believed that death in young persons was frequently associated with active rheumatic carditis, but with mechanical factors over the age of thirty years. They also considered that the best indications of prognosis were heart size and functional capacity and that although heart murmurs gave an indication of the nature of the valvular lesion, they did not indicate the degree of stenosis or incompetence present, and suggested that a poor prognosis should not be given simply because a patient had an organic heart murmur.

In aortic incompetence, Fishberg (1940) believed that damage to the myocardium by infection was more often the principal cause of heart failure, but that the mechanical effect of regurgitation or super-added stenosis was a contributory factor. Once the signs of a failing cardiac reserve became evident, then the stage was set for a slowly progressive deterioration, over perhaps as much as ten years, and that in such patients with a failing reserve the prognosis was worse than in mitral stenosis. He also believed that in rheumatic disease as a whole, the prognosis was poor when impairment of the cardiac reserve led to the necessity for bed rest, many succumbing within a year.

Wood (1950) believed that prognosis in aortic incompetence should be based on the size of the left ventricle, since exercise tolerance usually remained good and normal heart rhythm was common.

Aortic stenosis is a condition of, as yet, undecided origin of which Lewis (1933) said there were no symptoms, a view which has been superseded with the passage of time. Grant (1933) described a series in which one-third to one-half of the patients with aortic stenosis died of the disease in ten years. Campbell and Kauntze (1953) believed that the prognosis was good in children; few patients experiencing severe symptoms until adult or even middle age, when the prognosis worsened. Wood (1950) considered that prognosis depended on cardiac enlargement and pulse volume, and that/

that a low pulse volume meant critical narrowing of the aortic orifice, which in pure aortic stenosis has recently been shown to be in the region of 0.5 square cm. Matthews and others (1955). He found that 15 per cent of patients died abruptly and a further 10 per cent developed subacute bacterial endocarditis. Bramwell and others (1955) believe that when heart failure in aortic stenosis commences, often in the sixties, the course is downhill regardless of therapy, with angina, and cerebral anaemia causing irritability, drowsiness and delusions. Kumpe and Bean (1948) had already noted that those patients with angina in aortic stenosis might die suddenly and that gross left ventricular enlargement caused a lowered cardiac output, with cerebral anaemia and syncope.

Mitchell and others (1954) found, that the average age of death in pure aortic stenosis was 65.3 years, but when associated with aortic incompetence was only 52.5 years. They noted that angina occurred in 36.7 per cent of patients with aortic stenosis and after its onset, the expectation of life was about four years. They also confirmed the observation made by Wood, that subacute bacterial endocarditis developed in 10 per cent of patients.

Matthews and others (1955) state, that when left ventricular failure commences in aortic stenosis, there occurred a short period of severe symptoms until death, and that sudden death might occur at any time. They believed that it was the duration of the stenosis, rather than its severity, which precipitated left ventricular failure.

Diagnosis of mitral and aortic valvular lesions must be made by the features and characteristics of the heart murmurs, by associated clinical signs of heart damage, by enquiry into the origin, cause and course of the patient's ailment and by technical aids to diagnosis, such as phonocardiography, radiology and electrocardiography.

Whenever a specific heart lesion has been confirmed, many clinical series dealing with the course of heart lesions are available, which enable the clinician to assess the probable course of the disease in a particular patient. For/

For the purpose of prognosis reliance is yet placed on the presence of symptoms and signs indicating an impaired or failing cardiac reserve.

Whenever a heart murmur is encountered a careful history and thorough clinical examination may reveal a non-cardiac cause. In many conditions a rapidly and forcibly acting heart may produce functional murmurs. These include hyperthyroidism, fevers, diseases, which have an anaemia as part of their syndrome and hypertension.

The systolic pulmonary murmur of pulmonary incompetence may occasionally occur as part of the symptomatology in conditions such as emphysema, pulmonary fibrosis and kyphoscoliosis causing hypertension of the pulmonary circuit.

Heart murmurs may be heard in patients with no organic heart lesion and no systemic disease. Such are the cardio-respiratory murmurs often heard in children and systolic murmurs at the pulmonary and mitral areas heard when the heart is over-acting after exercise, one of these described by Bramwell (1943) may simulate the presystolic murmur of mitral stenosis.

The next matter to be considered is the importance of modern surgical techniques in the treatment of selected cases of heart disease, and the change in prognosis of mitral and aortic stenosis wrought by surgery.

It is believed that the total incidence of predominantly mitral disease in both sexes between the ages of 18 and 44 is approximately 160,000 and that about 80,000 of those require valvotomy, Wood (1954).

The present operative procedure is splitting of the stenosed mitral valve either with a finger or valvotome.

Sellers and others (1953) suggest criteria by which the possible place of surgical treatment may be adjudged. These are the presence of severe mitral stenosis, signs of an impaired cardiac reserve disabling enough to justify operative risk and a firm/



firm belief that the symptoms and signs are primarily due to valvular obstruction and not to myocardial damage. They believe that the pliant diaphragmatic type of valve associated with a tapping apex beat, a snapping first mitral sound, an opening snap and a presystolic murmur gives the most rewarding post-operative results. Pulmonary congestion, with attacks of pulmonary oedema means severe mitral obstruction and is an indication for operation, as are severe exertional dyspnoea and lack of ability to deal with everyday tasks. The absence of heart enlargement and the presence of normal heart rhythm are favourable signs, suggesting a relatively healthy myocardium.

The combination of dyspnoea, pulmonary congestion, minimal heart enlargement and normal rhythm indicates severe mechanical obstruction and relative myocardial integrity and is regarded as favourable for surgery.

Patients with certain clinical findings must be refused heart surgery, these include predominant mitral incompetence, aortic incompetence with marked left ventricular enlargement and a Corrigan pulse, tricuspid valvular disease, active rheumatic fever and subacute bacterial endocarditis.

Sellors (1953) and others consider the age limits to be from 20 to 50 years and suggest that where right heart failure cannot be relieved by medical treatments, valvotomy is unlikely to be of benefit. Wilson and Greenwood (1954), however, state that a few patients with chronic and intractable right heart failure have improved remarkably after operation. It seems reasonable, however, to assume that chronic congestive heart failure uncontrolled by medical treatments suggests irreversible myocardial changes.

Cleland and others (1955) stress the importance of accurate assessment of the degrees of dyspnoea and pulmonary hypertension. They found that dyspnoea was inversely proportional to the size of the valve and that the critical diameter for the onset of paroxysmal dyspnoea, bronchitis and haemoptysis was 1.5 cms. They suggested that patients otherwise suitable should be operated on

before the stage of dyspnoea on slight exertion (Grade 3) was reached, and that patients with dyspnoea on moderate exertion (Grade 2) should be kept under careful review, since dyspnoea was not necessarily progressive. Like Sellors, they considered an accentuated first mitral sound and an opening snap to be favourable indications for operation. They believed that absolute contra-indications to surgery were, severe mitral incompetence, intractable heart failure, active rheumatic fever and predominantly aortic valvular disease.

The surgical treatment of aortic stenosis is at the present time largely experimental and little information is available about post-operative results.

Effective surgical relief of the stenosis should make a more significant difference to the natural history of the disease than any medical treatment, Matthews (1955), but the difficulty in the selection of patients for operation is, that when symptoms of cardiac failure develop, the course is rapid and that when the patient is most likely to benefit from operation there may be no symptoms. A patient who feels perfectly fit is unlikely to view the possibility of a major heart operation with equanimity. In addition, severe aortic stenosis is compatible with longevity and when left ventricular failure does occur, valvular calcification and ventricular myocardial fibrosis may be so advanced, as to prevent a successful valvotomy.

In a review of twenty-five patients treated by surgery, Muller and others (1954), suggested, that all patients, except those with minimal disability, should be considered for aortic valvotomy and stressed the desirability of early operation, which reduces the operative risk of sudden ventricular failure. They found that the post-operative results in patients with pure aortic stenosis were superior to those in patients with associated mitral valvular disease, and concluded that their early results were encouraging.

It may be seen that mitral valvotomy has a definite role in the treatment of mitral stenosis and can effect a dramatic alteration in prognosis. The place of aortic valvotomy is less assured, but if early results continue to be encouraging more patients may be aided by this more recent surgical technique.

This section of the thesis will summarise the salient features of the cases which were selected, observed, recorded and followed up. Ten of these cases will be described and discussed in greater detail in a later section of the work.

### MATERIAL

Thirty-one patients suffering from affections of the mitral and aortic valves were included in the thesis, twenty-five of them were females and six of them were males.

Ten patients had predominant mitral stenosis, five had predominant aortic stenosis, three had predominant mitral incompetence, one had predominant aortic incompetence and twelve had mitral stenosis and aortic incompetence.

### IMPAIRMENT OF THE CARDIAC RESERVE

Most of these cases have been personally followed up for a little over a year, but in many of the cases, previous clinical records extended back for as long as five to ten years. In Fig.1. a comparison is drawn, between the number of patients in each age group presenting with heart murmurs only and the number presenting with heart murmurs and, in addition, the signs and symptoms of an impaired or failing cardiac reserve.

The signs of an impaired cardiac reserve included palpable right or left ventricular enlargement, auricular fibrillation, evidence of congestive cardiac failure and signs of pulmonary hypertension. The symptoms of an impaired cardiac reserve included breathlessness on exertion, pulmonary congestion, with paroxysmal and nocturnal dyspnoea, haemoptysis and 'bronchitis', angina of effort and paroxysmal tachycardia.

The age of onset of signs and symptoms of an impaired or failing reserve was found to be highest in patients with aortic stenosis, the average age of onset being 54.6 years. It/

It was lowest in patients with predominant mitral incompetence, the average age of onset being 27 years in that group.

The majority of the patients when first seen, had signs and symptoms of an impaired cardiac reserve already established. Nine patients presented with heart murmurs alone and no symptoms of an impaired reserve, all were in the younger age groups. They were composed of three patients aged 14, 15 and 30 years with mitral stenosis, one patient aged 23 years with mitral incompetence and four patients aged 13, 14, 16 and 28 years with combined mitral stenosis and aortic incompetence; although the four in the latter group all had radiological evidence of cardiac enlargement.

LESION	Total Number of Patients	Number with signs & symptoms of an Impaired Reserve	AGE DISTRIBUTION OF PATIENTS WITH AN IMPAIRED RESERVE				SEX DISTRIBUTION OF PATIENTS WITH AN IMPAIRED RESERVE	
			0-19	20-39	40-59	60-79	Males	Females
MITRAL STENOSIS	10	7	-	1	4	2	1	6
AORTIC INCOMPETENCE	1	1	1	-	-	-	-	1
AORTIC STENOSIS	5	3	-	-	2	1	2	1
MITRAL INCOMPETENCE	3	2	-	1	1	-	-	2
MITRAL STENOSIS & AORTIC INCOMPETENCE	12	7	-	5	2	-	1	6

Figure 1. Showing the number, age distribution and sex distribution of the patients with signs and symptoms of an impaired cardiac reserve.

AGE AND SEX.

Figure 2 illustrates the age distribution and sex incidence for the different lesions. The most frequent lesion discovered was combined mitral stenosis and aortic incompetence, closely succeeded by mitral stenosis.

Only one case of pure aortic incompetence was included in the series. This case is described in detail in a later section.

LESION	TOTAL PATIENTS	AGE DISTRIBUTION				SEX INCIDENCE	
		0-19	20-39	40-59	60-79	Male	Female
MITRAL STENOSIS	10	2	2	4	2	1	9
AORTIC INCOMPETENCE	1	1	-	-	-	-	1
AORTIC STENOSIS	5	-	-	3	2	3	2
MITRAL INCOMPETENCE	3	1	1	1	-	-	3
MITRAL STENOSIS & AORTIC INCOMPETENCE	12	3	6	3	-	2	10

Figure 2. Age distribution and sex incidence for all cases.

### INCIDENCE OF SIGNS AND SYMPTOMS

Figure 3 illustrates the incidence of the various signs and symptoms of an impaired or failing cardiac reserve.



LESION	GRADES OF DYSPNOEA				PULMONARY CONGESTION	EVIDENCE OF CONGESTIVE CARDIAC FAILURE	CARDIAC ENLARGEMENT	AURIC-ULAR FIBRILLATION	ANGINA OF EFFORT	SUBACUTE BACTERIAL ENDO-CARDITIS
	I	2	3	4						
MITRAL STENOSIS 10 Cases	4	2	-	4	6	4	8	5 (one post-operatively)	-	-
AORTIC INCOMPETENCE 1 Case	-	-	-	1	-	1	1	-	1	1
AORTIC STENOSIS 5 Cases	2	1	-	2	2	2	4	1	3	-
MITRAL INCOMPETENCE 3 Cases	1	1	1	-	1	-	2	1 (paroxysmal)	-	1
MITRAL STENOSIS & AORTIC INCOMPETENCE 12 Cases	3	3	-	6	8	5	12	5	2	1
TOTAL 31 Cases	10	7	1	13	13	12	27	12	6	3

FIGURE 3. Illustrating the number of patients in either group with the various signs and symptoms of an impaired cardiac reserve.

In figure 3 the degree of dyspnoea was assessed on the basis that Grade 1 meant dyspnoea on severe exertion only, Grade 2 meant dyspnoea on moderate exertion, Grade 3 meant dyspnoea on slight exertion and Grade 4 meant dyspnoea at rest. All the patients with evidence of congestive cardiac failure were placed in Grade 4.

Patients with orthopnoea, nocturnal and paroxysmal dyspnoea, haemoptysis, recurrent attacks of Bronchitis, accentuation and splitting of the pulmonary second sound, were considered to have pulmonary congestion.

Cardiac enlargement, which was always confirmed radiologically, was the commonest clinical finding; only four of the total thirty-one patients did not have this sign.

Subacute bacterial endocarditis was considered to exist on the evidence of positive blood cultures, or on unequivocal and classical clinical signs and symptoms, despite negative blood cultures.

In this series, cardiac enlargement was the most common sign of an impaired or failing cardiac reserve, being absent in only four cases.

Established auricular fibrillation was discovered in four patients with mitral stenosis, the average age of onset in this lesion being forty-eight years. Three of these four patients had apical systolic murmurs of at least moderate degree, conducted to or toward the axilla.

In addition, one patient in whom, at operation, a slight degree of mitral incompetence had been discovered, developed auricular fibrillation post-operatively.

Three/

Three of the five cases of aortic stenosis developed angina of effort, the average age of onset being fifty-four years; none of these patients was hypertensive, the highest reading of the three being 120/80 mm.Hg. In one of these cases a woman, aged sixty-two years, the anginal pain may have been due to an anaemia, since on admission her haemoglobin was only 6.8 Gms.

Three patients were considered to have sufficient clinical evidence to justify a diagnosis of subacute bacterial endocarditis and two of them had, in addition, positive blood cultures. One patient with aortic incompetence died of the infection despite treatment.

This section of the thesis is devoted to a description of ten cases selected from the total number of thirty-one. After each case history there follows a short discussion, to illustrate the way in which each case provides material support for the several main arguments of the thesis.

The cases comprise two patients with aortic stenosis, four patients with mitral stenosis, one patient with aortic incompetence, one patient with mitral incompetence and two patients with combined mitral stenosis and aortic incompetence.

CASE 1.

MR. J.S.

AGED 53 YEARS

ADMITTED ON 1/7/55.

This patient, a farmer, gave no history, of rheumatic fever. He had noticed mild angina of effort for three years and slight exertional dyspnoea for fifteen months. Four months before admission he developed gross oedema of legs and sacrum with ascites and since then had been largely confined to bed. He had also a productive cough and mentioned several recent haemoptyses. He had suffered from paroxysmal<sup>and</sup> nocturnal dyspnoea.

General examination showed cyanosis, gross oedema of legs and sacrum, ascites, cervical venous congestion and slight breathlessness. Cardiovascular examination revealed a regular pulse of low volume. The apex beat was forcible and greatly enlarged to the left. There was a blowing systolic murmur at the mitral area and a harsh systolic murmur, with a thrill, at the aortic area. There were crepitations at both lung bases. Blood pressure was 110/80 mm.

Blood urea was 58mg/%, a blood count was normal and his Wasserman reaction negative. X-ray of the chest showed marked generalised cardiac enlargement. A second X-ray of chest taken two weeks later showed no change.

On 4/8/44 a four pint abdominal paracentesis was performed and a further five pints were removed on 16/8/55. The patient remained cyanosed and grossly oedematous despite digitalis, marsalyl and the abdominal paracenteses, gradually becoming more breathless and ultimately orthopneic. He showed no signs of cerebral anaemia or syncope. He died suddenly on 28/8/55. Post-mortem showed marked calcification of the aortic valve, gross cardiac enlargement, aortic stenosis and terminal coronary artery thrombosis.

DISCUSSION/

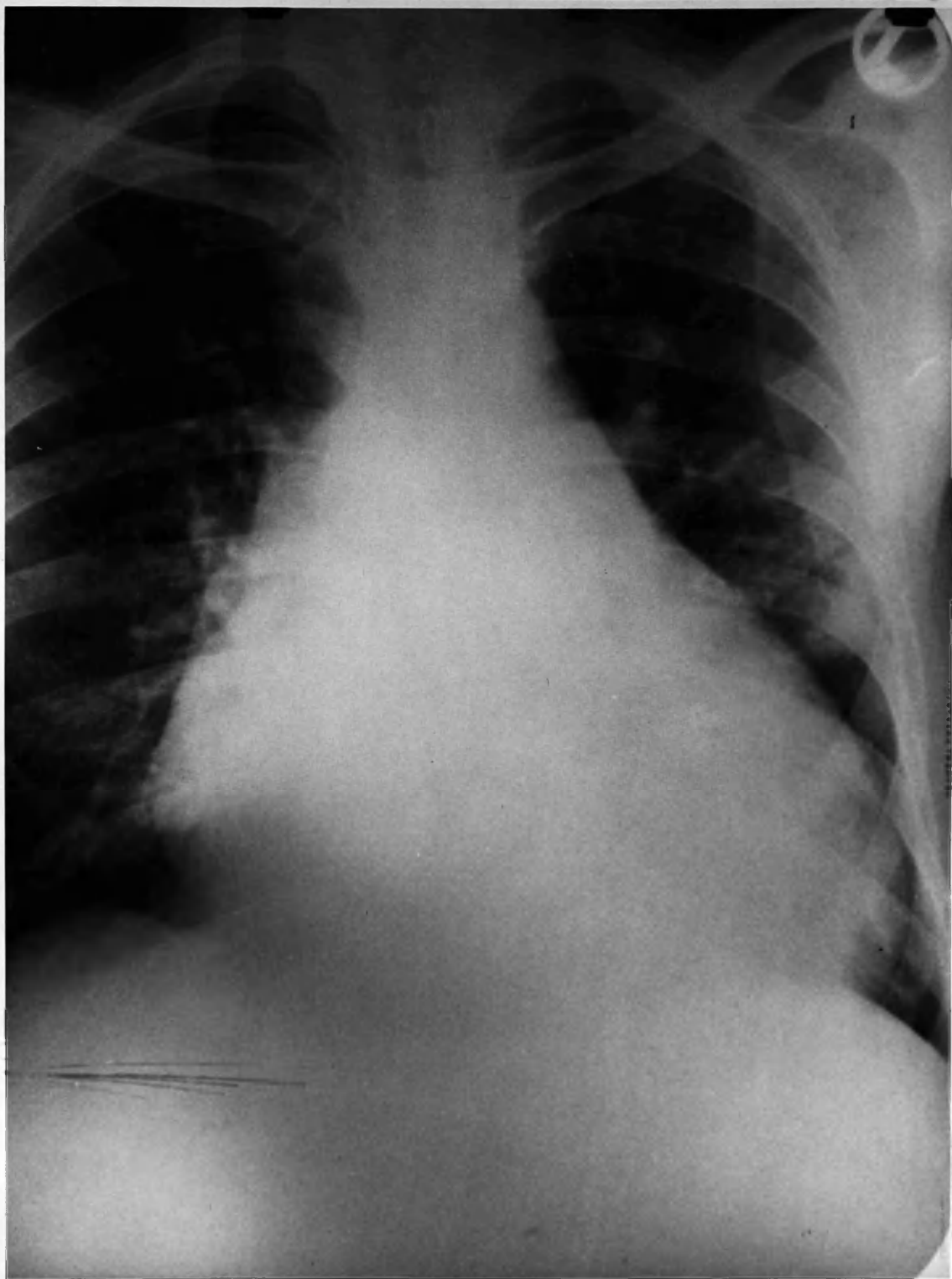


PLATE.I. 2.8.55. Mr.J.S. Gross enlargement of the cardiac shadow,especially the left ventricle and ascending aorta. Lung fields clear.

DISCUSSION:

The course of this patient's illness illustrates the belief, that in aortic stenosis, once heart failure commences, then the course is downhill regardless of therapy. His clinical course supports the view of Kumpe and Bean (1948), that those patients with angina in aortic stenosis might die suddenly. The diagnosis in this cases was made on the grounds of the loud harsh basal murmur with an accompanying thrill, the absence of hypertension and the gross cardiac enlargement with a low pulse volume. The prognosis was presented, not on the features of the murmur, but on the evidence of right heart failure due to high grade decompensation of the left ventricle due to aortic stenosis, on the great cardiac enlargement and the low pulse volume, which Woods (1950) considered to mean a critical narrowing of the aortic orifice. The onset of his anginal pain was a significant occurrence, as Mitchell and others (1954) noted that after its onset in aortic stenosis, the expectation of life was about four years. The absence of a history of rheumatic fever is of interest in view of the present day debates as to the aetiology of this condition.

CASE 2.            MRS. A.W.            AGED 62 YEARS            ADMITTED 15/7/55.

This patient, a housewife with a family of eleven, gave no history of rheumatic fever. For nine years she had experienced frequent anginal attacks and one year before admission had collapsed unconscious after an anginal attack, while walking in the street. She had suffered from slight, but increasing exertional dyspnoea for several years. Three months prior to admission she had an unusually severe anginal attack and had remained mainly in bed since, with ankle swelling if she remained out of bed for any length of time. She also mentioned that her ankles had swollen during several of her later confinements. She had no paroxysmal or nocturnal dyspnoea and no haemoptysis. General examination showed a stout, rather pale woman with slight ankle oedema, no cyanosis or cervical venous congestion, but breathless at rest. She was mentally alert but answered questions rather slowly.

Cardiovascular examination revealed a regular pulse of low volume. The apex beat was enlarged to the left but was not forcible. A harsh systolic murmur was heard at the aortic and mitral areas, loudest at the mitral area, the aortic murmur was not conducted into the neck. There were no thrills. The lung bases were clear. Blood pressure was 120/90 mm.

X-ray showed left ventricular enlargement and slight hilar congestion. E.C.G. showed left axial deviation. A blood count revealed a severe iron deficiency anaemia, haemoglobin 7.Gms. and she was given two pints of whole blood. She remained reasonably well on mersalyl therapy until 5/8/55 when she had several severe anginal attacks while in bed and became drowsy and mentally confused, with breathlessness, persistent anginal pain, ankle oedema and oliguria. Urine examination revealed a mild infection which was treated with sulphatriad. Her mersalyl was discontinued on 19/8/55 because of a mercurial rash. She became progressively drowsier with persistent oedema, mild confusion and increasing breathlessness at rest and died on 7/9/55.



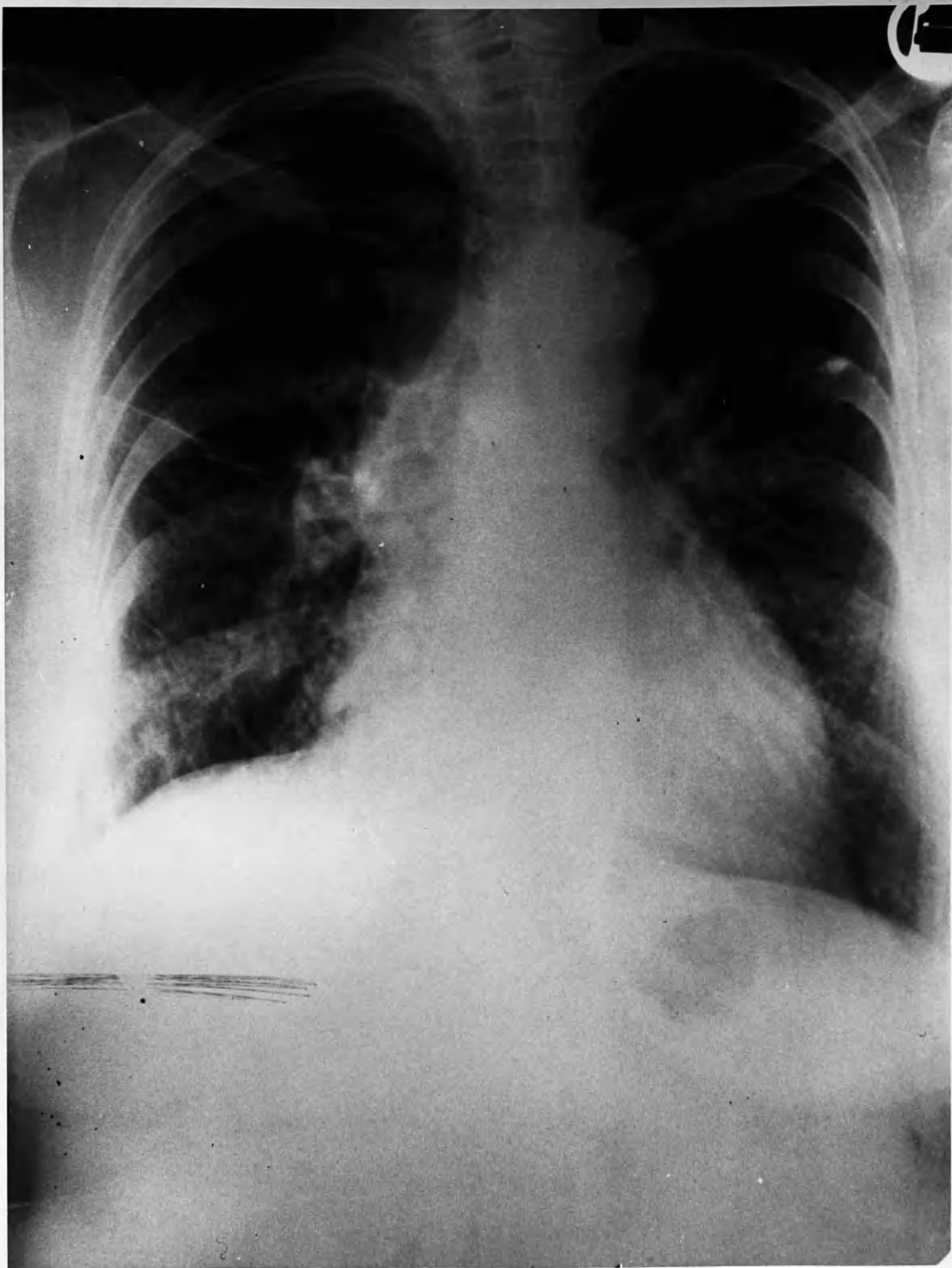


PLATE.2. 18.7.55. Mrs.A.W. Marked cardiac enlargement with unfolding of the aorta. There are mild congestive changes at the lung roots with some patchy mottling at the right lower zone.

Post-Mortem showed moderate cardiac enlargement, calcific aortic stenosis and myocardial fibrosis.

DISCUSSION:

As in Case 1 the diagnosis here was made on the basis of the heart murmur at the aortic area, the absence of any hypertension, the presence of left ventricular enlargement and a pulse of low pressure. She gave a longer history of anginal attacks than Case 1, but the length of survival from the time of onset of heart failure was almost the same, about five to six months. The rapidly downhill progress of this patient illustrates the poor prognosis once severe symptoms develop, a fact emphasised by Contratto and Levine (1937), who found that the average length of survival after the first appearance of dyspnoea was twenty-three months and of pitting oedema, four months. This patient developed signs of cerebral anaemia during the last few weeks of her life with slow, slurred speech and confusion of thought; features of aortic stenosis noted by Bramwell and others. Kumpe and Bean (1948) have noted that the lowered cardiac output in aortic stenosis causes cerebral anaemia with syncope. The attack of unconsciousness which this patient had one year prior to admission was probably of this nature. As in case 1 no history of rheumatic fever was obtained.

CASE 3.           MISS M.B.           AGED 15 YEARS           ADMITTED ON 6/8/55.

This patient, a schoolgirl, was admitted with acute rheumatic fever. Three weeks prior to admission she had developed a sore throat, following by migratory polyarthritides, erythema marginatum, malaise, sweating and three days before admission, epistaxis on two occasions. She gave no history of previous attacks of rheumatic fever.

General examination showed a flushed child with fever, a subcutaneous module on the extensor surface of the left elbow, a fading erythematous rash on the legs and a normal throat.

Cardiovascular examination revealed a regular pulse of good volume with tachycardia. There was no displacement of the apex beat and no thrills. The second pulmonary sound was split, but the heart sounds at the mitral and aortic areas were unaltered. At the mitral area there was a loud, blowing pan-systolic murmur conducted to the axilla. A harsh systolic murmur was heard at the pulmonary area and down the left sternal border. There was a faint blowing systolic murmur at the aortic area. Blood pressure was 140/75.

Her E.S.R. on admission was 7mm. for the first hour. She was treated with aspirin and Glycine powders and her general condition improved rapidly.

On the 8/8/55 however, a loud rumbling mid-diastolic murmur with pre-systolic accentuation made its appearance. Screening of the chest on 12/8/55 showed slight enlargement of the left auricle and clear lung fields. The patient's improvement continued, an E.S.R. on 25/8/55 was again 7 mm. for the first hour. A week later she was transferred, symptom free, to a convalescent home for six months. The heart murmurs remained unaltered.

DISCUSSION/

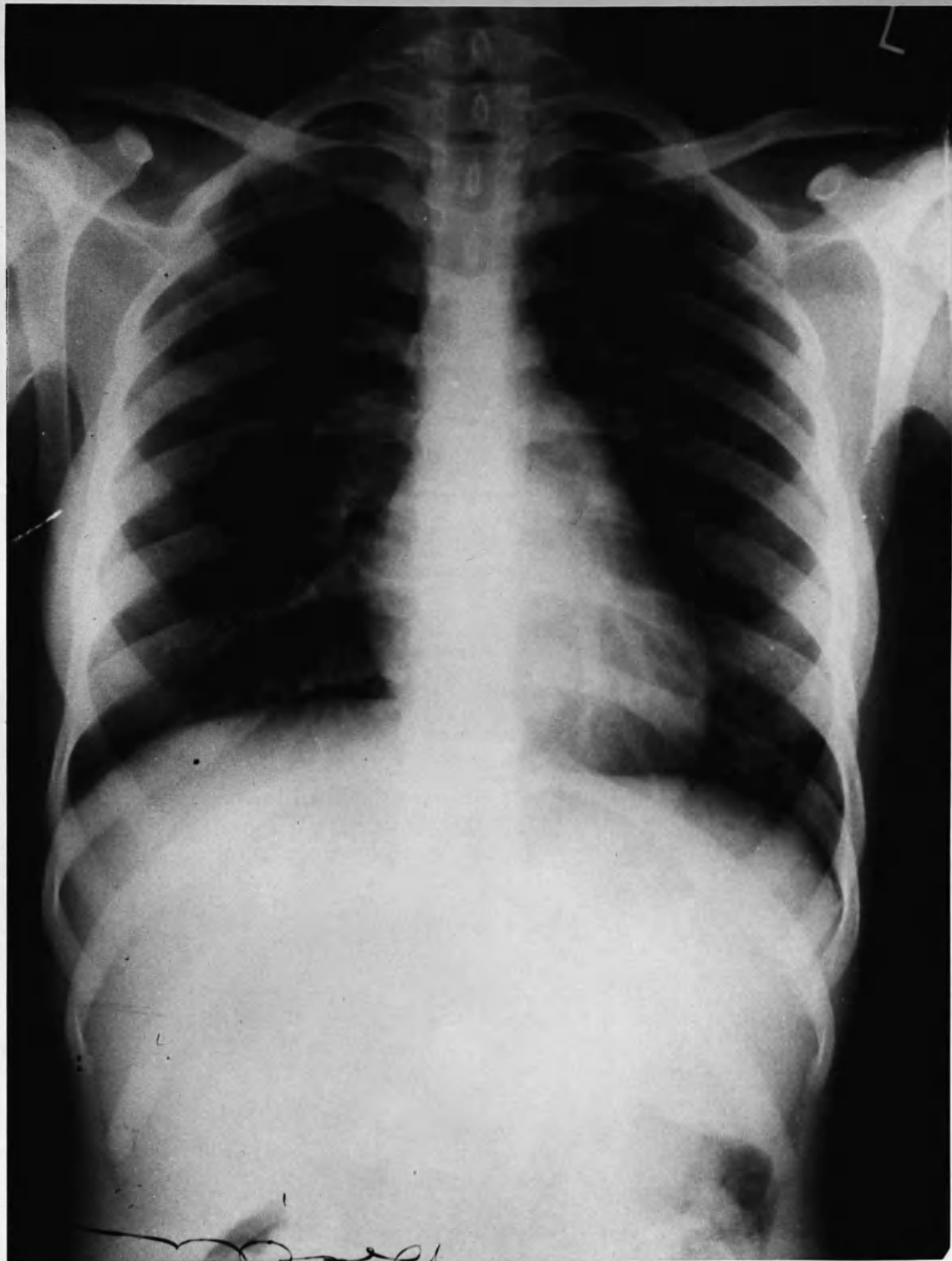


PLATE.3. 9.8.55. Miss.M.B. Very slight left auricular enlargement but no overall cardiac enlargement.Lung fields clear.

DISCUSSION:

This case is of interest in that it illustrates the rapidity with which a loud rumbling mitral mid-diastolic murmur with pre-systolic accentuation may appear. This child could not have developed a severe degree of mitral stenosis at such an early stage of her illness, and yet she had a well-marked mitral pre-systolic murmur. The view might be taken that this in fact was an organic murmur of mitral stenosis and if so, would tend to confirm the view expressed by Wood (1954). that a presystolic murmur might occur with a trivial degree of stenosis. It remained possible, however, that the appearance of the mid-diastolic and pre-systolic mitral murmurs in this case was due to relative mitral stenosis. Since the patient had no demonstrable left ventricular dilatation, this relative stenosis could have been caused by oedema of the mitral leaflets, as suggested by Zilli and Gamna (1954).

It remained possible then that this patient did not have organic mitral stenosis and that in the course of the succeeding months, the diastolic and pre-systolic murmurs might have disappeared.

However, when dismissed from the convalescent home at the end of her six months stay, the medical officer there reported that the mitral mid-diastolic and pre-systolic murmurs were unchanged; suggesting the presence of an organic mitral stenosis.

CASE 4.      MISS M. McC.      AGED 30 YEARS      SEEN AS OUTPATIENT

This patient, a secretary, was first admitted to hospital on 6/6/51. She had experienced rheumatic fever at five years of age and remained well until 1948 when she noticed slight ankle swelling. Two days prior to admission she had an attack of haemoptysis and breathlessness lasting for a week.

General examination revealed no abnormality. Cardiovascular examination showed a regular pulse of average volume. The apex beat was tapping and there was a diastolic thrill at the mitral area. There was narrow splitting and slight accentuation of the pulmonary second sound. There were no systolic murmurs, but a loud rumbling mid-diastolic murmur was heard at the mitral area. X-ray of the chest was negative. She was placed under observation as a possible candidate for cardiac surgery and has been seen at six-monthly intervals since 1951. At no time since has she shown signs of an impaired cardiac reserve.

When seen on 13/10/55 she was symptom free having spent her summer holiday abroad, participating in swimming and dancing with only very slight breathlessness. There had been no further haemoptysis.

General examination was negative. Cardiovascular examination showed a slow regular pulse of low volume. The apex beat was tapping in quality and not displaced. There was a mitral diastolic thrill. There was narrow splitting and accentuation of the pulmonary second sound.

At the mitral area there were accentuation of the first sound, an opening snap of the mitral valve, a long, loud, rumbling mid-diastolic murmur and a pre-systolic murmur. Blood Pressure 140/70 mm. X-ray of chest was negative.

DISCUSSION/

## DISCUSSION:

The presence of normal rhythm, lack of cardiac enlargement, accentuation of the first mitral sound and absence of an apical systolic murmur suggest that this patient has a well marked mitral stenosis, with mechanical obstruction predominating over myocardial damage. She has the opening snap of the mitral valve, associated with the pliant diaphragmatic type of valve ideal for operation and a tapping apex beat, a loud first mitral sound and a pre-systolic murmur, signs which Sellors and others (1953) believed would anticipate good post-operative results.

She has none of the absolute contra-indications to mitral valvotomy such as severe mitral incompetence, active rheumatic fever, predominant aortic valvular disease or advanced heart failure, Cleland and others (1955).

The reasons for deferring surgical treatment in this patient are that, despite evidence of severe mitral stenosis, as given by the features and characteristics of the heart murmurs and associated cardiac signs and the attack of pulmonary congestion with haemoptysis, four years ago, nevertheless she shows no signs of an impaired cardiac reserve disabling enough to justify the operative risks.

Her very slight breathlessness on severe exertion does not appear to be progressing to higher grades. She is able to deal adequately with everyday tasks, a fact stressed by Sellors and others (1955).

This patient will be referred for surgical treatment if progressive breathlessness appears, or if she develops paroxysmal dyspnoea, haemoptysis or bronchitis.

CASE 5.

MISS J.B.

AGED 50 YEARS

ADMITTED 16/7/54

This patient was first admitted to hospital on 25/4/52, She had suffered from chorea and rheumatic fever at the age of eight years and thereafter had suffered from occasional joint pains. Four years prior to admission she had noticed progressive exertional dyspnoea and about the same time suffered an attack of paroxysmal dyspnoea and palpitation. During the year before admission she had two attacks of haemoptysis lasting for several days. Cardiovascular examination at that time revealed a regular pulse of average volume. The apex beat was moderately enlarged to the left and she was found to have a mitral mid-diastolic murmur. Blood pressure 180/120 mm. X-ray of chest showed marked cardiac enlargement especially affecting the left auricle.

She was discharged very well on 21/5/52. She was re-admitted on 16/7/54, complaining of dyspnoea on slight exertion and frequent attacks of paroxysmal and nocturnal dyspnoea. For a year she had experienced attacks of paroxysmal tachycardia. For three months she had been confined to her house with extreme breathlessness and ankle swelling.

General examination showed orthopnoea, cyanosis, ankle oedema and slight cervical venous congestion. Cardiovascular examination showed a regular pulse of average volume. The apex beat was enlarged to the left and was tapping in character. There was a loud first mitral sound and splitting of the second pulmonary sound. At the mitral area there was a moderately loud blowing systolic murmur and a long loud rumbling mid-diastolic murmur and a pre-systolic murmur. X-ray of chest showed gross cardiac enlargement, especially left auricular and right ventricular. Blood cultures were negative.

She was accepted for mitral valvotomy which was carried out in Glasgow on 12/11/54, when mitral stenosis, slight incompetence and gross calcification of the mitral valve were found/



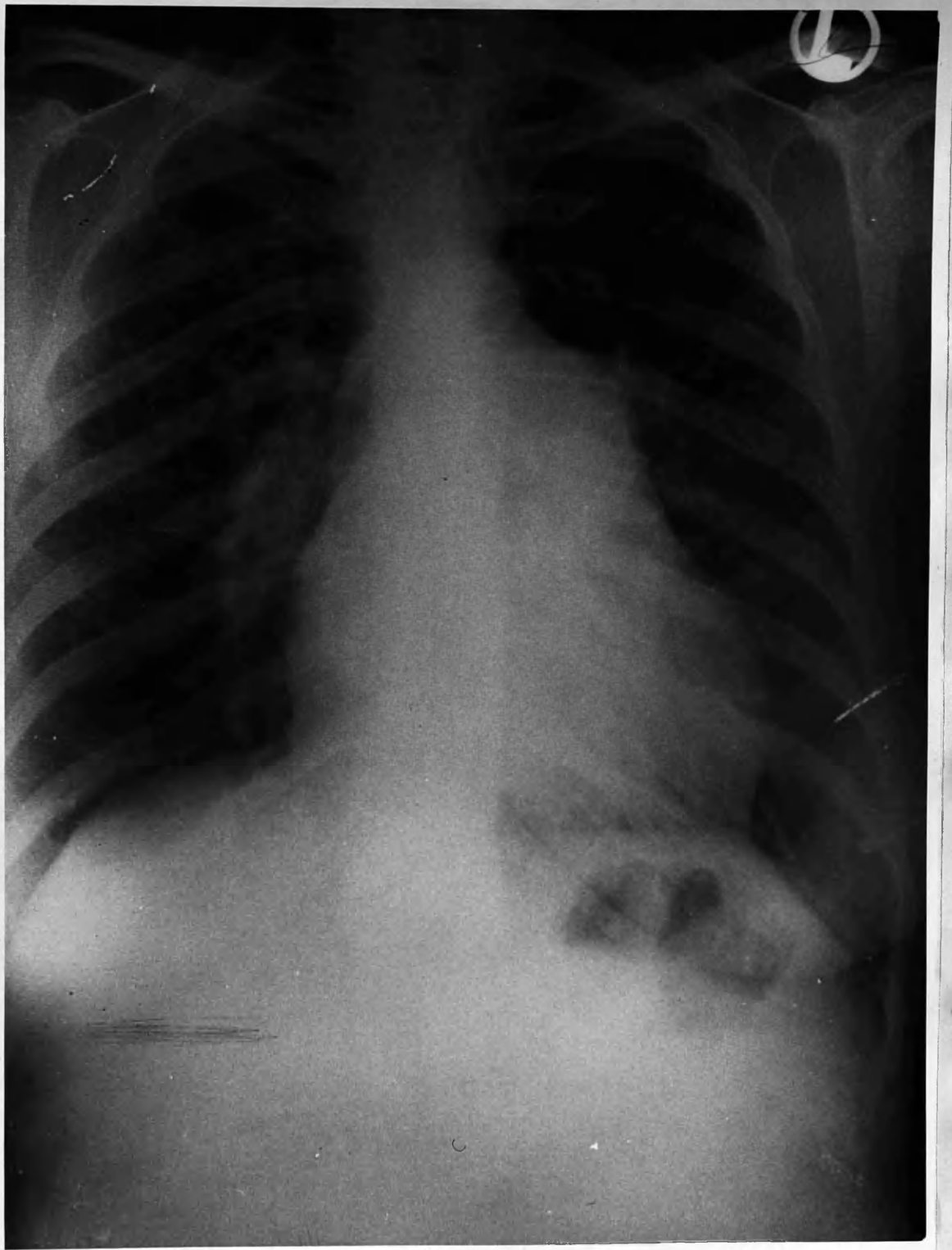


PLATE.5. 27.8.55.Miss.J.B. Cardiac enlargement with congestive cardiac failure.

found. She developed auricular fibrillation post-operatively and required digoxin 0.25 b.d. to control it. She was transferred back to this hospital on 19/1/54 and improving rapidly on 0.25 digoxin b.d. and mersalyl twice weekly was sent home much improved on 30/1/55.

#### DISCUSSION:

This patient had shown signs of an impaired cardiac reserve six years before operation, characterised by progressive exertional dyspnoea, paroxysmal dyspnoea and paroxysmal tachycardia. These symptoms became more severe, until three years before operation she developed haemoptysis, a sign of severe pulmonary congestion. Wilson and Greenwood (1954) found of those patients with mitral stenosis and severe pulmonary congestion, 16 per cent died within six months and 50 per cent within five years. According to the prognostic groups suggested by Lewis, this patient with marked cardiac enlargement, poor exercise tolerance and sound rhythm might have anticipated from three to six years survival at that time.

When admitted in the year of operation she had reached the stage of commencement of right heart failure with evidence of gross pulmonary congestion when, according to Wilson and Greenwood 24 per cent of these patients died within six months and 50 per cent within three years, and according to Lewis when the stage of gross cardiac enlargement was reached, then life was rarely prolonged for more than 2 years.

The patient was accordingly assessed for operative suitability. She had signs of an impaired cardiac reserve disabling enough to justify the operative risk, she had severe pulmonary congestion indicating severe mitral obstruction, exertional dyspnoea and was incapable of dealing with everyday tasks. The features of the diastolic murmur suggested a severe degree of stenosis. These factors favoured operation.

Against/

Against operation were the facts that she was suffering from congestive cardiac failure, that she had gross right ventricular failure, indicating myocardial damage either mechanical or inflammatory and that she had a moderately loud, blowing systolic murmur suggesting a moderate degree of mitral incompetence.

She was finally operated on, when gross stenosis, slight incompetence and gross calcification of the mitral valve were found. She developed auricular fibrillation post-operatively, improved slightly on medical treatment for a short while, but eight weeks later died at home of congestive cardiac failure. The poor post-operative result in this case supports the current view that calcification of the mitral valve and the presence of mitral incompetence carry a poor prognosis.

CASE 6.

MR. S.J.

AGED 48

RE-ADMITTED 27/12/55

This patient gave no history of rheumatic fever. He was admitted to hospital for the first time on 26/9/49 with a history of sudden onset of haemoptysis and breathlessness at work on that date. He had noticed slight breathlessness on exertion since 1945. He was found to have the murmurs of mitral stenosis and X-ray of his chest showed cardiac enlargement of mitral shape with no congestion. He made a rapid recovery and was discharged.

On 16/7/53 he was re-admitted with a history of occasional haemoptysis during the preceding seven months. He also had breathlessness on severe exertion and a cough. He had noticed no ankle swelling. He was reviewed for possible mitral valvotomy but was rejected, because of lack of severe breathlessness and because chest screening showed a severe degree of calcification of the mitral cups and ring.

On 27/12/55 he was re-admitted complaining of increasing breathlessness for six months, with occasional attacks of haemoptysis since 1953. One week before admission he had a severe haemoptysis with acute breathlessness and sudden tachycardia.

General Examination showed a pale, anxious, breathless man with finger clubbing and an emphysematous chest. He had no oedema or cervical venous congestion. The liver was palpable and tender. Examination of the cardiovascular system showed a rapid pulse with auricular fibrillation and low volume. There was enlargement of the apex beat outwards. There was a well-marked mitral thrill. The mitral first sound was not accentuated. There was narrow splitting and accentuation of the pulmonary second sound. At the mitral area there was a short soft systolic murmur and a medium length rumbling mid-diastolic murmur. No other murmurs were audible. E.S.R. was normal and blood area was 98 mgm%. X-ray of the chest showed cardiac enlargement of mitral shape, with an early

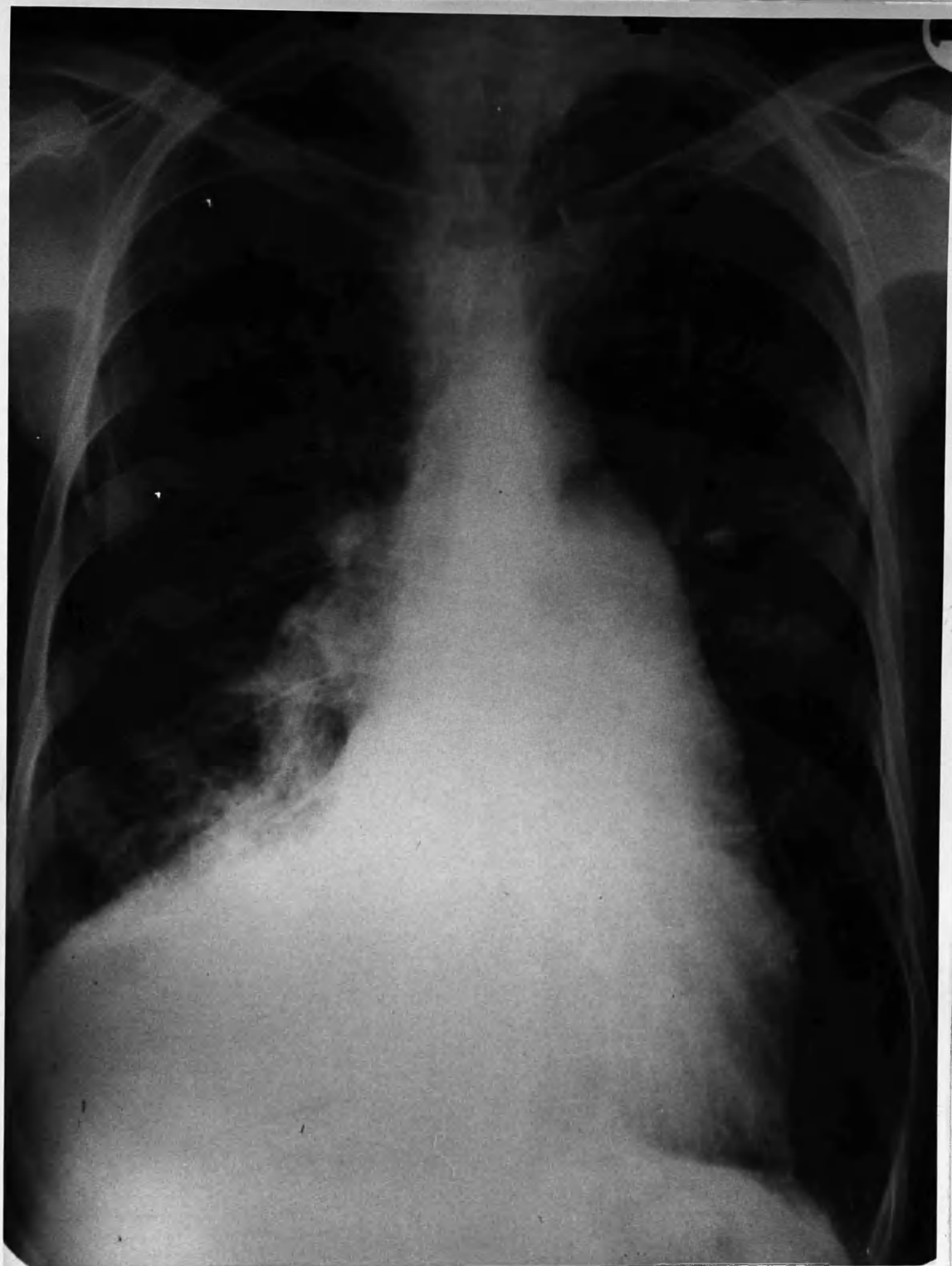


PLATE .6. 30.12.55. Mr.S.J. Enlarged cardiac shadow of mitral shape.

Consolidation in the right lower lobe, with raising of the right diaphragm.

early effusion at the right base. He made a good response to mersalyl and intravenous digoxin and his condition improved rapidly. He was discharged very well on 20/1/56.

#### DISCUSSION:

This patient appeared to have had a severe haemoptysis and an attack of severe pulmonary congestion with dyspnoea, occurring at the same time as the onset of auricular fibrillation. When the rapid fibrillation was controlled by digoxin therapy his dyspnoea rapidly subsided, which would indicate that some of the breathlessness was due to the commencement of the fibrillation.

He had, however, complained of progressive exertional dyspnoea for some two years prior to admission on this occasion, and must now have a severe degree of pulmonary congestion with paroxysmal dyspnoea. According to Wilson and Greenwood (1954) 50% of patients in this category are dead within five years.

In mitral stenosis, the onset of auricular fibrillation usually occurs at the stage of severe pulmonary congestion and is provoked by the patient's age - in this case 48 years - and distension of the left atrium. This patient would appear then to have a high degree of myocardial damage.

The onset of severe pulmonary congestion with paroxysmal dyspnoea and the slowly progressive exertional dyspnoea are factors in favour of mitral valvotomy at this stage, and even the onset of auricular fibrillation is not a contra-indication to surgery, since Brock and others (1955) have found that it is the operative anatomical result, rather than the pre-operative rhythm which is the decisive factor in prognosis.

The decisive factor in this patient is the presence of a severe degree of calcification of the mitral ring - a poor prognostic sign in mitral valvotomy.

It is unlikely that he would obtain striking benefit from surgery and it appears that the relief of some of his symptoms must be left to medical measures. He is still able to work and carry out everyday tasks without excessive breathlessness and at 48 years may probably anticipate at least six years of active life.

CASE 7. MISS M.S. AGED 15 YEARS ADMITTED 21.12.54.

This patient, a schoolgirl, was admitted with a three week history of fatigue, sweating, rapid weight loss and pain in the left ankle and knee. She had no sore throat.

She gave no history suggesting previous attacks of rheumatic fever. For several months prior to admission she had suffered from occasional splinter haemorrhages and progressive pallor. She had been breathless on severe exertion for a period of three months. There had been no cough, haemoptysis or ankle swelling.

General examination showed a pale, listless, fevered girl with poorly coloured mucous membranes. She had no skin rashes or subcutaneous nodules, no recent splinter haemorrhages. The throat was normal.

Cardiovascular examination showed a rapid regular pulse of average volume. There were no thrills. The first mitral sound was accentuated and a faint early diastolic murmur was heard in this area. At the base and down the left sternal border there was a soft blowing early diastolic murmur. There were no systolic murmurs. Blood pressure was 110/50 mm.

X-ray of chest showed cardiac enlargement, mainly left auricular, with no congestion of the lung fields.

The spleen was enlarged to two fingerbreadths below the left costal margin, the liver was not enlarged.

E.S.R. was 85 mm. for the first hour, Wasserman reaction was negative, the urine contained numerous red cells and the blood picture revealed a fairly severe microcytic hypochromic anaemia, haemoglobin 9.9 gms. Blood cultures were repeatedly negative.

She responded to large doses of intramuscular Penicillin, Aspirin and iron therapy and when she went home on 28.3.55 the spleen was no longer palpable, E.S.R. was 4 mm. for the first hour, haemoglobin was 12.2 gms. and she looked and felt very much better.



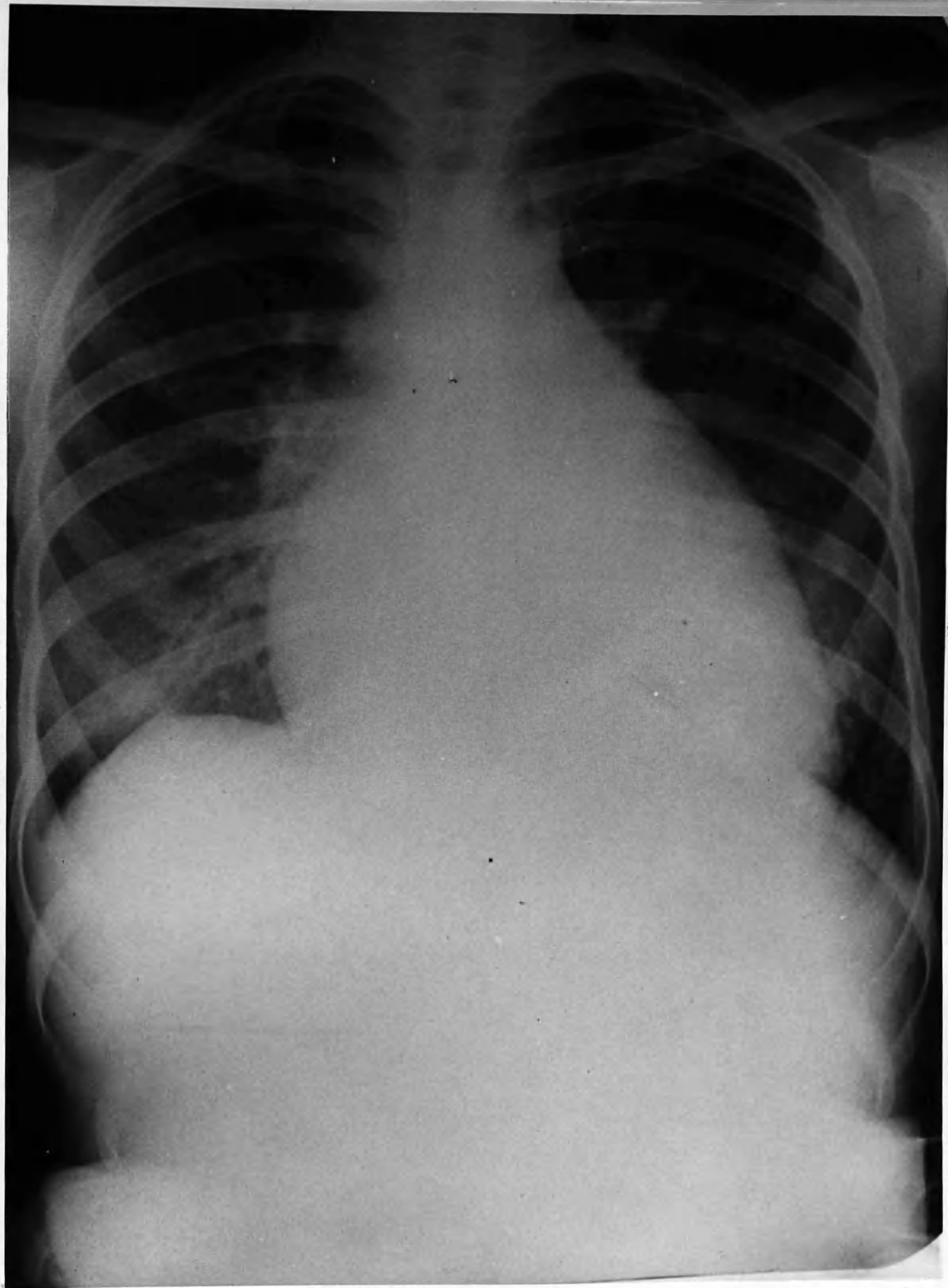


PLATE.7 Miss M.S. 27 . 5. 55. Great deterioration since films of 2I.2.55 which showed slight enlargement ,chiefly left auricular,and no enlargement of the hilar shadows. This plate shows very gross cardiac enlargement and passive congestion of the lung fields.

At home she unfortunately relapsed and for a month prior to readmission on 19.5.55 she was breathless on slight exertion. A week before readmission she developed breathlessness at rest, sacral oedema and chest pain on exertion.

General examination showed orthopnoea, cyanosis, sacral oedema and cervical venous congestion. The spleen was again palpable.

Cardiovascular examination showed little change except for the presence of medium crepitations at both bases.

X-ray of chest showed marked deterioration. There was an increase in the cardiac enlargement, with early congestion of the lung fields. Despite continuous oral iron her haemoglobin level had fallen to 9.7 gms. In hospital she was treated for cardiac failure and subacute bacterial endocarditis with accompanying anaemia. Her condition deteriorated rapidly and on 18.6.55 at her parents' request she was allowed home. She died there two weeks later.

#### DISCUSSION:

This youngster when first seen had a well-marked aortic incompetence with early cardiac enlargement, but no signs of an impaired or failing cardiac reserve, apart from slight breathlessness on severe exertion. The presence of normal heart rhythm and good exercise tolerance were not, however, of such favourable significance as might be anticipated from a study of mitral stenosis or aortic stenosis, since Wood (1950) holds the belief that, the prognosis in aortic incompetence depends on neither of these factors. He believed that the prognosis should be based on the size of the left ventricle, since exercise tolerance in aortic incompetence usually remained good and normal heart rhythm was common.

Nevertheless, if that view is accepted, the degree of left ventricular enlargement in this case was never sufficient to explain the rapid deterioration and early death.

The decisive factor in this girl's illness was the presence of subacute bacterial endocarditis. It is well known that the presence of this disease, suspected when a patient with a valvular lesion develops fever, embolic phenomena, pallor or splenic enlargement, still constitutes a serious threat to those suffering from well compensated valvular lesions. Despite the use of antibiotics, the recovery rate does not exceed 70 per cent.

The rapid course of this girl's illness supports the opinion of Bunn and Cook (1954) that, the prognosis was worst in those with aortic incompetence and in patients who developed cardiac failure during the course of infection.

The diagnosis of aortic incompetence was made on the basis of the heart murmurs, but the prognosis was based on the presence of subacute bacterial endocarditis, an infective process adding an additional burden to a myocardium already weakened, by the effects of an earlier rheumatic infection.

CASE 8.

MRS. S.McL.

AGED 42 YEARS.

ADMITTED 1/7/55

This patient, a housewife with two of a family, had been in hospital on two previous occasions. On 23.2.42, aged 29 years, she was admitted with a two week history of sweating, migratory polyarthrititis, palpitation and fever. Cardiovascular examination revealed no evidence of rheumatic carditis and after treatment with solicylates she was sent home completely well, after seven weeks in hospital.

A year later, on 3.2.43, she was re-admitted with a further attack of migratory polyarthrititis, sweating and fever. On this occasion she was found to have a soft systolic mitral murmur conducted towards the axilla; otherwise examination was negative. After two weeks in hospital she returned home very well.

When re-admitted on 1.7.55 she gave the following history since her previous admission. She had remained able to undertake her household duties and free from symptoms until 1952, when she noticed breathlessness on severe exertion and experienced attacks of palpitation, associated with a sense of oppression in the chest, not however suggesting angina of effort. She remained in this condition until two weeks prior to admission, when she developed pain in the left wrist and hand, malaise and fatigue, breathlessness on slight exertion associated with a right sided pleuritic chest pain.

General examination revealed pallor and poorly coloured mucous membranes, slight fever, slight cyanosis; slight breathlessness at rest and no oedema.

Cardiovascular/

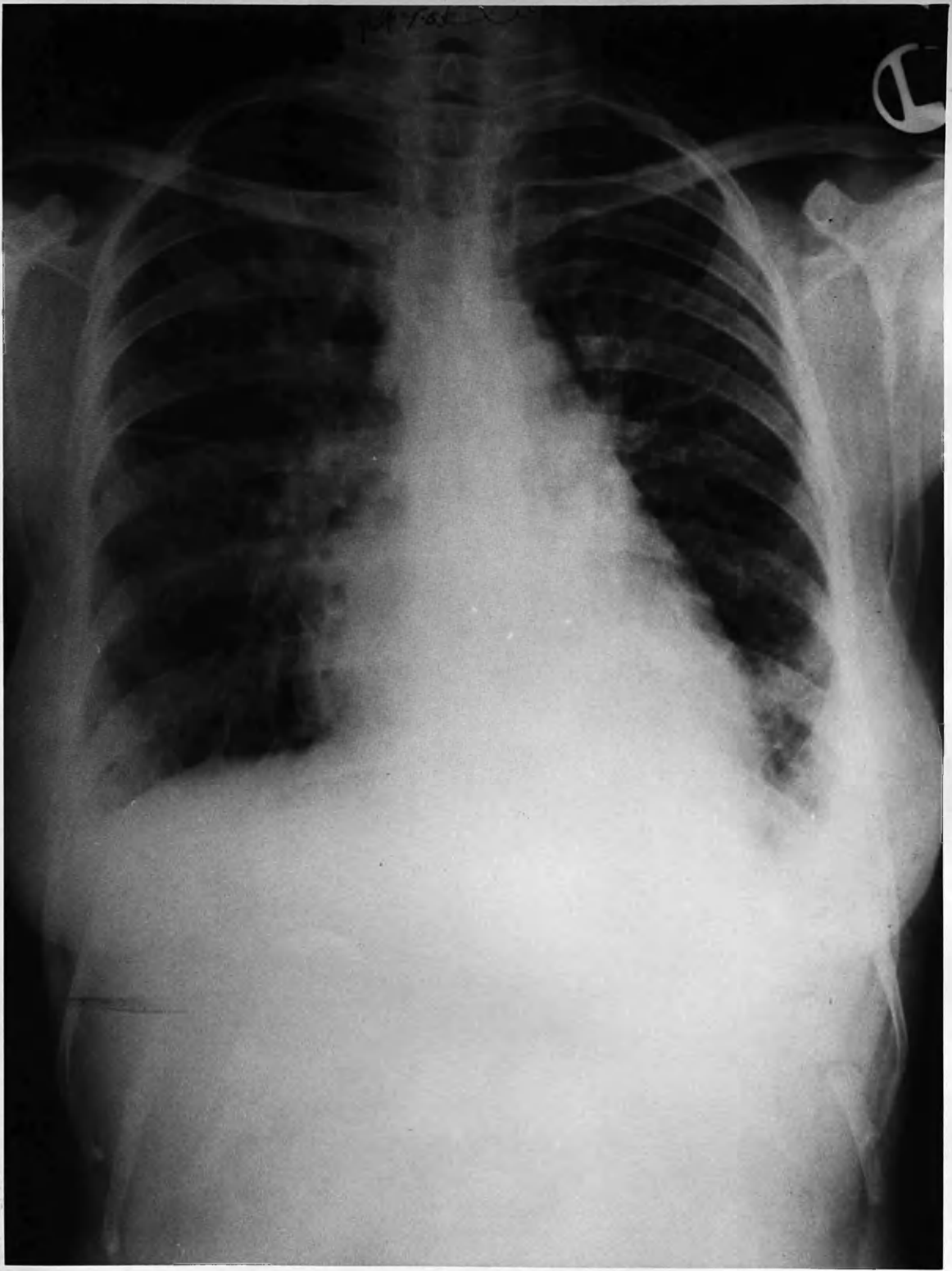


PLATE.8 Mrs S.McC. 18 . 7. 55..Cardiac enlargement with hypoplasia of the aortic arch and congestive changes present in both lung fields. No evidence of calcification of the mitral valve.

Cardiovascular examination revealed a regular pulse of low volume with tachycardia. There were no thrills. The first mitral sound was diminished and the second pulmonary sound slightly accentuated. There was a long, loud systolic murmur at the mitral area conducted to the axilla. There were no systolic murmurs elsewhere and no diastolic murmurs were heard or elicited. A third heart sound was well heard at the lower left sternal border. Blood pressure was 140/80 mm. There were crepitations at both bases. X-ray of chest showed hypoplasia of the aorta and a barium swallow revealed selective enlargement of the left auricle. There was no calcification of the mitral valve. There was congestion at both lung bases.

Blood examination showed a moderate microcytic hypochromic anaemia, Hb.9.1.gms. E.S.R. was 22 min. for first hour.

She responded well to medical therapy and was sent home quite recovered from her acute symptoms on 23.8.55.

#### DISCUSSION.

The presence of a diminished mitral first sound, a long loud systolic murmur conducted to the axilla, no apparent diastolic murmurs, a clearly heard third heart sound and radiological evidence of selective enlargement of the left auricle combine to reach a diagnosis of mitral incompetence. The loudness and area of propagation of the apical systolic murmur would suggest the presence of, at least, a moderated degree of incompetence and Wood (1954) described a series, in which no case of significant mitral incompetence was encountered, without an apical systolic murmur of, at least, moderate degree.

Where/

Where ventricular systole is short, as in mitral incompetence, the third heart sound occurs early in the cycle and may be confused with the opening snap of the mitral valve, unless it presents in its present form, best heard between the apex beat and the left sternal border and low-pitched in character.

Selective left auricular enlargement may occur in early cases of mitral stenosis, but great or aneurysmal dilatation of the left auricle is a feature of mitral incompetence; in this patient the enlargement was not great and could have been caused by either stenosis or incompetence.

A diminished mitral first sound may be caused by severe pulmonary hypertension in mitral stenosis, probably because the apex of the heart is, in these cases, formed by the enlarged right ventricle. Diminution of this sound, however, is usually suggestive of mitral incompetence or of calcification of the mitral valve.

The diagnosis in this case depended on a balanced integration of the various findings but principally on the presence of the long, loud, conducted apical systolic murmur and the diminished first sound in the mitral area.

The patient showed signs of early pulmonary congestion probably caused by the acute illness which brought her to hospital.

The pain in the left wrist and hand, the chest pain and pulmonary congestion were probably primarily due to a further attack of acute rheumatic fever, an opinion supported by the raised E.S.R., general malaise, fatigue, and pyrexia.

The rapid disappearance of the early pulmonary congestion and the slight breathlessness at rest, probably precipitated by the acute infection were encouraging; but the presence of an active rheumatic infection, evidence of at/

at least moderate mitral incompetence, radiological evidence of cardiac enlargement and the exertional dyspnoea, combined with her age and heavy domestic responsibilities, did not suggest an optimistic prognosis.



CASE 9.      MRS. M.B.      AGED 37 YEARS      RE-ADMITTED 26.7.55.

This patient, a housewife, had two attacks of rheumatic fever as a young girl. She remained well until 1948, when she developed progressive exertional dyspnoea. She was admitted to hospital in 1949 for investigation and was found to have murmurs of mitral stenosis, and X-ray appearances consistent with this diagnosis. In 1949 her fourth pregnancy was terminated because of her cardiac lesion.

During the succeeding six years she became progressively breathless and less able to carry out her household duties. She had no symptoms of pulmonary congestion, but several months before admission to hospital in January 1955 she developed oedema of the ankles.

General examination revealed breathlessness at rest, slight cyanosis, oedema of the ankles and cervical venous congestion. The liver was enlarged.

Cardiovascular examination revealed a pulse of average volume and rate, with auricular fibrillation. She had a soft blowing systolic and a rumbling, mid-diastolic murmur at the mitral area. Blood pressure was 120/70 mm. X-ray of chest showed enlargement of the left auricle, left ventricle and right ventricle, with a right-sided hydrothorax/<sup>which</sup> was large enough to require aspiration. She went home again, improved, on 16.6.55 still on digitalis and mersalyl. While at home she continued to attempt housework, cooking and shopping and as a result was re-admitted on 26.7.55.

She had slight breathlessness, gross ankle oedema, cervical venous congestion and the liver was hard, tender and enlarged. Cardiovascular examination showed auricular fibrillation, accentuation of the first mitral sound and narrow splitting of the pulmonary second sound. There was a diastolic thrill at the mitral area with a soft blowing systolic and long, rumbling, mid-diastolic murmurs.

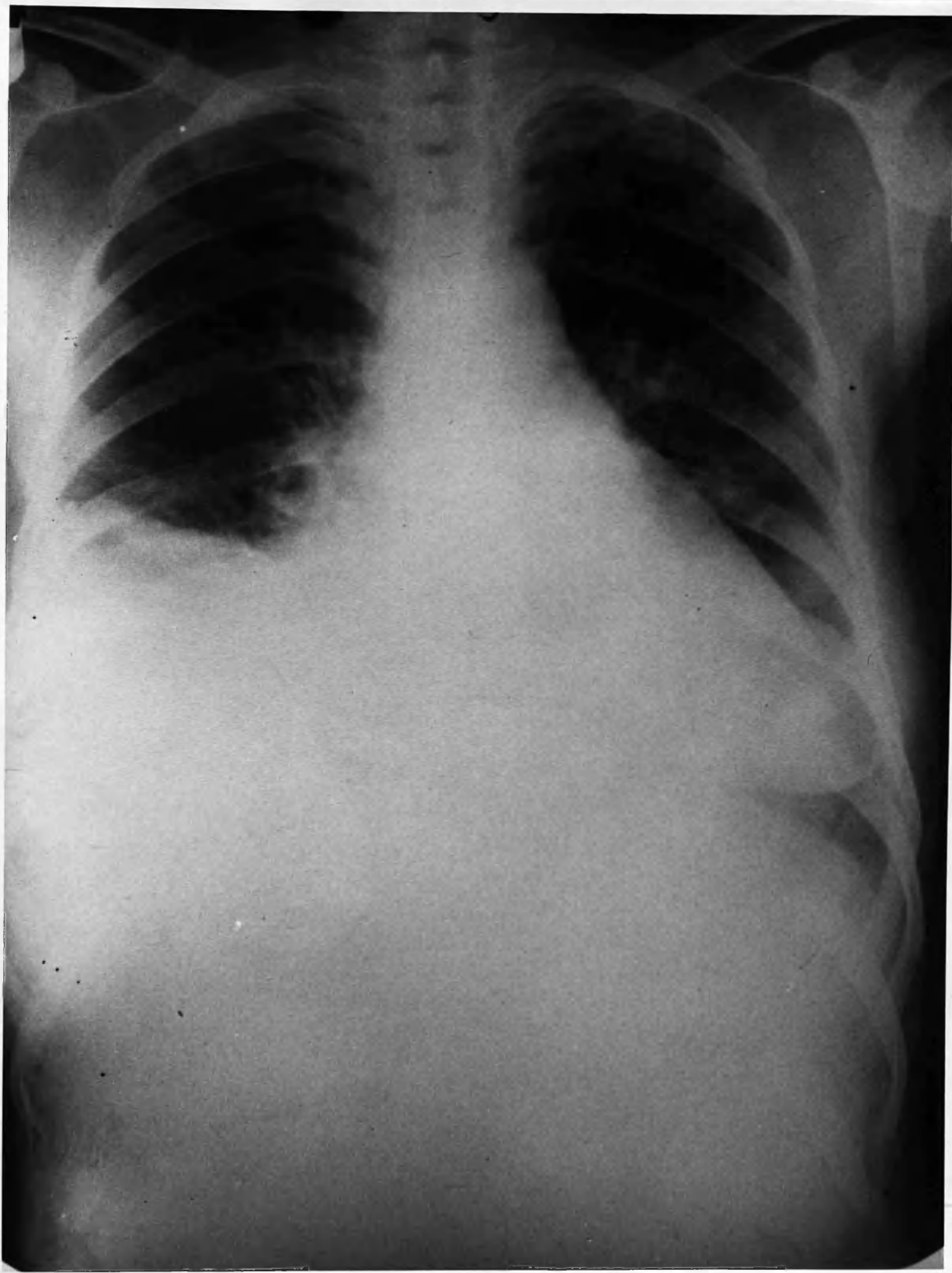


PLATE.9. 29.9.55. Mrs.M.B. Gross cardiac enlargement of mitral shape with congestive failure and an encysted effusion at the right base.

At the base and down the left sternal border there was a soft systolic murmur and an early diastolic murmur. There was a faint systolic murmur to the right of the lower sternum. Blood pressure was 120/70 mm. X-ray showed pulmonary congestion with a large right-sided hydrothorax. She again responded to rest and treatment and returned home six weeks after admission.

She was again re-admitted on 6.9.55 with advanced congestive failure. Clinical examination and X-ray of chest were similar to those of 26.7.55. Her oedema responded to treatment and she became less dyspnoeic. She went home again four weeks later. She died there in January 1956.

#### DISCUSSION.

The course of this patient's illness from the onset of signs of an impaired cardiac reserve, until her death at the age of thirty-seven was eight years. She had clear evidence of a combined mitral and aortic lesion. Evidence of mitral stenosis, in the form of heart murmurs and X-ray appearances supporting this diagnosis, was present in 1948, but it was not until 1955 that the murmurs of aortic incompetence were definitely apparent.

In the winter of 1954 she developed early congestive cardiac failure which was never completely controlled until her death fourteen months later. The onset of congestive failure in patients with rheumatic heart disease is an ominous portent and in a series of cases of predominant mitral stenosis described by Wilson and Greenwood (1954), 24 per cent of patients who developed right heart failure died within six months and 50 per cent within three years.

In/

In addition to the predominating mitral stenosis, this patient had aortic incompetence and the presence of a double valvular lesion carries a poor prognosis, indicating as it does a severe degree of myocardial and valvular damage. In predominant mitral stenosis the presence of aortic incompetence with left ventricular enlargement and a Corrigan pulse precludes operative treatment, which again worsens the prognosis.

This patient illustrates the comment made by Fishberg (1940) that, in rheumatic disease as a whole, the prognosis was poor when impairment of the cardiac reserve led to the necessity for bed rest, many succumbing within a year.

When first seen in 1948 she had slight cardiac enlargement, fair exercise tolerance, normal heart rhythm and no evidence of commencing congestive changes and according to Lewis, might have anticipated at least ten years. Probably due to her household responsibilities and inability to lead a quiet well-regulated life, - indeed only a few months before her death she was still doing a large part of the family washing in the tenement wash-house - it was only eight years until she passed into the final group, those in the terminal phases of persistent heart failure, in whom death is always an imminent possibility.

CASE 10

MISS E.G.

AGED 14 YEARS

ADMITTED 22/12/54.

This patient, a schoolgirl, was admitted with a history suggesting rheumatic fever.

Four months prior to admission she developed a sore throat followed by migratory polyarthrititis. The polyarthrititis continued and she had been resting at home for three months complaining of pain and stiffness of the knees, elbows, shoulders and wrists.

She had not noticed any nodules or skin rashes. She had felt fevered and generally unwell.

Six years previously she had had a tonsillectomy. She had never had any previous attacks of rheumatic fever.

General examination showed no abnormality except for, some pain and stiffness in the painful joints. There was no dyspnoea, oedema or other signs of an impaired cardiac reserve.

Cardiovascular system showed a regular pulse of average volume. The apex beat was tapping in character and enlarged to the left. There was a diastolic thrill at the apex. At the mitral area there was a moderately loud systolic murmur conducted to the axilla and a long rumbling mid-diastolic murmur.

Systolic and early diastolic murmurs were heard down the left sternal border. Blood pressure was 100/65mm.

E.S.R. was 76mm. for the first hour, and X-ray of chest showed gross cardiac enlargement of mitral shape with pulmonary congestion. Blood cultures were negative. She improved in hospital and when discharged to a convalescent home on 16/5/55 her E.S.R. was 20mm. for the first hour. X-ray showed a diminution in the pulmonary congestion, but the gross cardiac enlargement remained.

She/

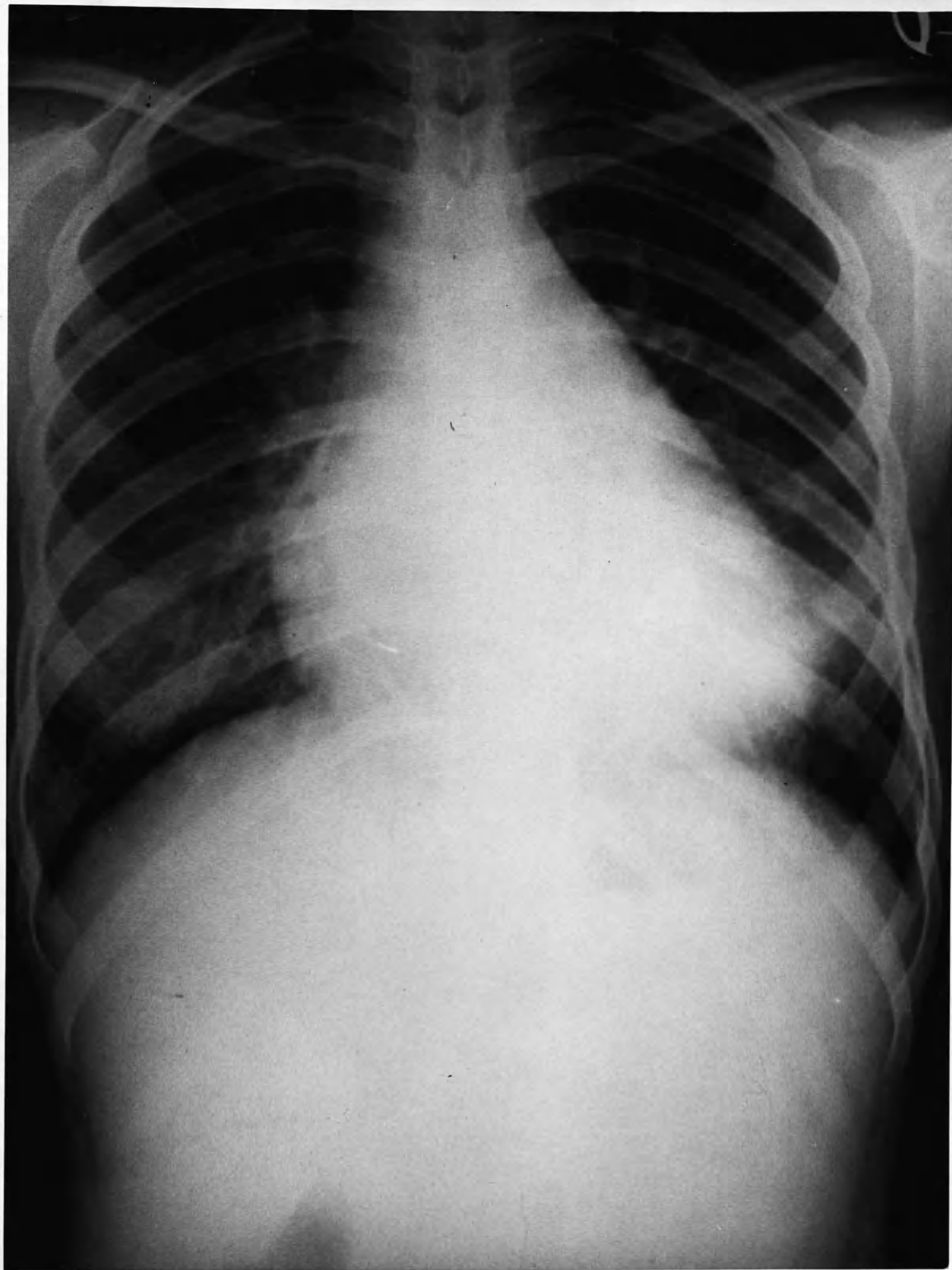


PLATE 10. Miss E.G. 3 . 2. 55 Gross cardiac enlargement with early congestive changes in both lung fields.

She was seen again on 18/1/56 when E.S.R. was 10mm. for the first hour, a blood count was normal, and X-ray of chest unchanged. The cardiovascular findings were similar to those of 22.12.54. She looked and felt well, her only symptom being breathlessness on severe exertion.

#### DISCUSSION.

This girl had her first acute attack of rheumatic fever four months prior to admission. Although no definite history of previous attacks was obtained, the fact that she had a tonsillectomy performed six years before admission does suggest that she may have experienced, at least, sore throats with possible subacute rheumatic fever.

She showed no signs or symptoms of an impaired or failing cardiac reserve, despite the radiological findings. According to Wood (1954) the prognosis in aortic incompetence depends more on the degree of cardiac enlargement, than on an evaluation of exercise tolerance or the type of heart rhythm since, in this lesion, effort tolerance usually remains good and normal heart rhythm is common.

Therefore, despite the absence of dyspnoea and auricular fibrillation, the clinical and radiological evidence of gross cardiac enlargement suggests that this girl will probably suffer a slow, steady deterioration of her cardiac reserve during the next ten years. Fishberg, (1940) believed that once evidence of an impaired or failing cardiac reserve became evident in aortic incompetence, the stage was set for a gradual deterioration and that the prognosis was worse than in those with mitral stenosis..

In addition to the murmurs of aortic incompetence, this girl had evidence of a well marked mitral stenosis; the length of the mitral mid-diastolic murmur suggesting at least a moderate degree of stenosis. The presence of the long moderately loud mitral systolic murmur conducted to the axilla would indicate at least a moderate degree of mitral incompetence.

The features and characteristics of the murmurs

point to a severe degree of valvular and myocardial damage and the degree of cardiac enlargement confirms severe myocardial damage, either mechanical or inflammatory.

The radiological evidence of early pulmonary congestion was an ominous sign but, in part, was probably aggravated by the presence of an acute rheumatic infection which slowly subsided.

When last seen four weeks ago after her sojourn at a convalescent home this girl was extremely well. Her E.S.R. and blood count were normal, she had no symptoms and no complaints. The heart murmurs were unchanged in character and the gross degree of cardiac enlargement remained. Her home conditions are fairly good and the mother is an intelligent woman, so that she has a fair chance of maintaining her present good health.

The eventual prognosis is poor, since she has a combined mitral stenosis and aortic incompetence, gross cardiac enlargement, evidence of early pulmonary congestion and for at least sixteen more years is liable to fall victim to further attacks of rheumatic fever.

Operation was not considered because of the gross cardiac enlargement, especially left ventricular.



Within the framework of this thesis, the general features and characteristics which differentiate heart murmurs of significance, from those threatening no ultimate hazard to the patient have been discussed and the methods, by which the murmurs of mitral and aortic affections may be recognised, timed and identified, have been described.

The correlation of additional data such as accentuation diminution or splitting of the heart sounds, the presence of additional sounds in the cardiac cycle and clinical and radiological evidence of cardiac enlargement, has been represented as an integral part of the constructive art of diagnosis.

The fundamental importance of an accurate assessment as to the relative role of the valvular and myocardial components in the production of mitral and aortic murmurs, their effect on the basic characteristics and usual timing of the murmurs, and their differing influence in the production of signs and symptoms of an impaired or failing cardiac reserve, has been analysed in some detail.

The value, in prognosis, of the murmurs of mitral and aortic valvular affections on the one hand and the signs and symptoms of an impaired or failing cardiac reserve on the other, have been examined.

The thesis has been argued that, as the signs and symptoms of an impaired or failing cardiac reserve become increasingly evident, then the value of the heart murmurs as a guide to prognosis decreases reciprocally.

Emphasis has been directed to the necessity for precise diagnosis in affections of the mitral and aortic valves, particularly since progress in operative technique has made surgical treatment of selected patients a practical reality. The criteria for surgical treatment have been enumerated in some detail, and the more recent views on this aspect of mitral and aortic valvular affections have been incorporated in the mandate of the thesis.

Thirty-one patients with the murmurs of mitral and aortic valvular lesions, personally recorded, observed and followed up, have been summarised in such a way, as to illustrate the main contentions of the thesis, and in a further section of the work, ten of these patients are discussed in full detail, with an individual summary of the support they lend to the several main contentions of this thesis.

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