CARDIAC FAILURE

with special reference to

Valvular Defects and Auricular Fibrillation,

based on

An Experimental Investigation into the Extra Load put on the Heart by Damaged Valves,

and

An Analytical Study of 320 Consecutive Cases of Failing Heart.

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C O N T E N T S.

INTRODUCTION.

In recent years many writers, in their attempt to direct attention to the major part the heart muscle plays in maintaining the circulation of the blood, have relegated valvular lesions to an obscure position in the semiology of diseases of the heart. Sir James MacKenzie[1] has taken a foremost part in this propaganda and, perhaps mostly from his manner of writing, has left the impression that the recognition of valvular lesions is almost useless in considering the question of cardiac failure - certainly that much useful time is wasted in the attention devoted to them in the students' curriculum.

It is easy to admit that the reserve power of the cardiac muscle will determine whether or not the heart can keep up a satisfactory flow of blood. It is just as obvious that a mechanical defect in the heart, or at the periphery of the circulation, will use up some of that power.

This is well brought out in cases where Auricular Fibrillation, by adding to the load of the heart, produces heart failure, as will be discussed later. Removal of the effects of the fibrillation, or removal of the fibrillation itself, is sufficient to restore to the patient a certain amount of cardiac reserve. It is also brought out in those cases where cardiolysis[2] has been performed for adherent pericardium, the signs of failure in many cases passing off and remaining absent for many years when the pericardial adhesions have been removed.

Cardiac reserve has been the subject of detailed

study by physiologists, as well as by clinicians, but as far as I am aware no attempt has been made to measure directly the mechanical effect of a damaged valve on the circulation. It was with the object of determining the importance of this factor in lessening cardiac reserve as far as it could be done with a mechanical schema, that the present investigation was initiated.

Rather than accept the apparently contradictory statistics of different workers with regard to the etiology of valve lesions, the cases admitted, because of failing heart, into one set of Wards of a general Hospital over a period of ten years, were analysed and the results have been incorporated in the discussion.

It was also considered advisable to note symptoms which were complained of by patients in this series, as giving an indication of the chief effects produced by diminished reserve of heart muscle.

The clinical material was placed at my disposal through the kindness of Professor T. K. Monro, while the experimental investigation was undertaken in the Physiological Laboratory by the courtesy of Professor Noel Paton. To each of these gentlemen I wish to record my thanks.

CARDIAC FAILURE.

On commencing this inquiry one was struck by the absence of any succinct definition of heart failure, though the expression is common enough both in lay and medical writings. It cannot be defined in terms of special symptoms, and one is forced to define first of all the function of the heart, and then consider heart failure in terms of its function.

It is accepted that the function of the heart is to provide an efficient circulation of blood, and we may consider the circulation to be efficient when, the blood being of normal quality, the needs of the body are supplied without the production of any uncomfortable symptoms. But the needs of the body vary, depending on the amount of work to which it is subjected, and the heart can be trained for work of different degrees of severity.

A heart that is trained to supply the wants of a man doing sedentary work would be incapable of meeting the demands of heavy manual labour, but it would not therefore be inferred that such a heart was suffering from failure. The definition would thus require to be adjusted to meet the cases of different individuals, and some such definition as the following might be suggested:- Cardiac failure is a failure on the part of the heart to keep up a circulation sufficient for the ordinary needs of the individual (and for which it had been trained) without the production of uncomfortable symptoms. This would obviate any qualification regarding the quality of the blood, or defect

in the lungs or general malnutrition, as the heart is not trained normally for these conditions, though it may under certain circumstances be quite well able to overcome some of these defects. It would meet the case equally of the man who was engaged in a laborious occupation and one who was engaged in sedentary work. But it should be noted that it would make the definition so wide that it would include the case of an individual who, though unable to follow his own occupation, might be able to attend to a less arduous calling.

It is not possible to fix a degree of exertion, inability to reach which would determine failure. No absolute standard is possible for all people. Each individual has his own standard. Further, there must be grades of failure. Failure may be absolute or relative. Thus one may be unable to perform any movement without the production of symptoms; he may even have symptoms when lying at rest in bed. Such would be a very severe grade of failure. Absolute failure would result in death. Milder degrees of relative failure would determine symptoms with varying degrees of exercise.

CARDIAC RESERVE.

The heart performs work which is in proportion to its requirements at the moment. An expenditure of energy adapted to meet extra requirements is produced either by a direct effect on the heart muscle, or by a variation in the nerve regulating mechanism. The work done is usually estimated by reference to the arterial pressure in the aorta which the heart has to overcome, and to the amount of blood sent out at each beat, or per minute.

Krogh[3] states that the left ventricle pumps 3 litres of blood per minute with the individual at rest, and 12 litres during moderate muscular exercise, while in an athlete the amount may be as much as 21 litres.

Taking into account the fact that during severe exercise the aortic pressure, with a healthy heart, is increased and if we accept the figures which Krogh assumes, viz: 110 millimetres when at rest and 150 or 160 during exercise, then the expenditure of energy during exercise will be -

$$\frac{12}{3} \times \frac{150}{110} = \frac{60}{11} = 5.5 \text{ times the resting work.}$$

The reserve in this case is therefore at least four times as much as the total amount of work expended during rest.

Further, Starling[4] in his experiments with the heart-lung preparation has shown that a heart can deliver the same quantity of blood against an arterial pressure of 208 mm.Hg as it does against one of 44 mm.Hg., provided the venous supply is kept up. Thus, in a heart removed from the body, he found that the natural reserve of the heart was between three and

four times the total energy expended at the lower pressure. These figures are in correspondence with what one finds clinically. A person may have a blood pressure reading of 250-280 mm. and be very vigorous; that is, presumably his body is getting a sufficient blood supply though he may have used up the bulk of his reserve for this purpose, and may have, in addition, trained his heart for this extra load.

Two factors may obviously effect reserve power of the heart - either something which weakens the force of the muscular contraction, or something which adds to the load on the heart, or both of these in varying degree.

If it is accepted that cardiac failure is due to diminished cardiac reserve, then the commoner causes which operate in these two directions to limit cardiac reserve and therefore to produce cardiac failure, may be presented in tabular form somewhat as follows:-

CAUSES OF DIMINISHED CARDIAC RESERVE.

(1) Causes of damage to heart muscle:-

(a) INFECTIONS : e.g.

acute rheumatism
syphilis
pyogenic infection
diphtheria
gonorrhoea
typhoid fever
influenza.

(b) METABOLIC TOXINS : e.g.

nephritis
gout
diabetes
Graves' disease.

(c) DEFECTIVE CORONARY BLOOD SUPPLY :

atheroma
syphilis
embolism.

(d) DEFECTIVE BLOOD :

pernicious anaemia, etc.

(2) Causes of increased load :-

(a) VALVULAR DISEASE.

(b) INCREASED ARTERIAL RESISTANCE.

(c) PULMONARY RESISTANCE.

(d) PROLONGED OVERWORK.

(e) DISORDERED RHYTHM.

(f) ALTERED NERVE CONTROL.

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We may state that cardiac failure is due to a disproportion between the state of the heart muscle and the load which the heart is called upon to carry; that is, it is due to the loss of cardiac reserve. These effects are graphically represented on the diagram (Fig.1) which was prepared for the use of students. (1)

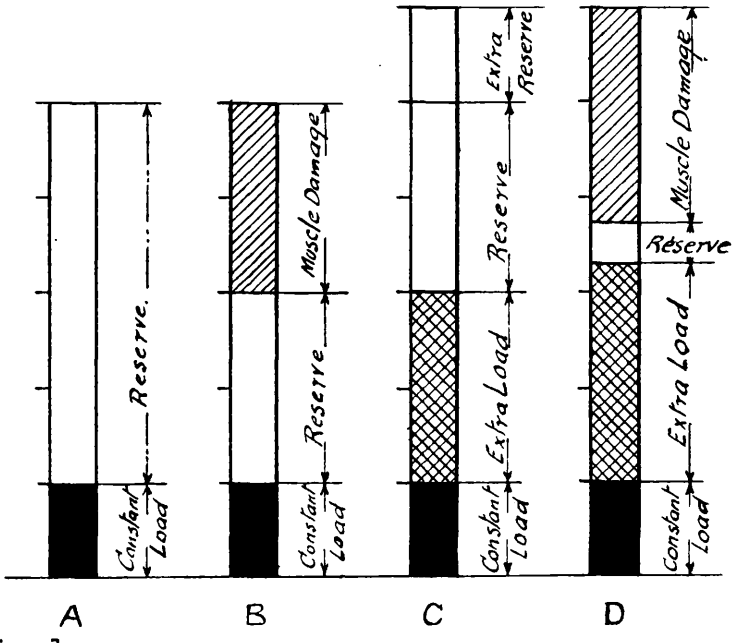


Fig.1. Diagram to illustrate the relationship of heart reserve to heart failure. *A*—healthy heart; *B*—heart with unhealthy muscle; *C*—heart with extra load due to valvular disease or high blood-pressure, and compensatory hypertrophy; *D*—failing heart due to load and muscle damage encroaching on reserve.

Assuming the figures derived from Krogh's and Starling's work A represents the proportion of reserve to constant load in the healthy heart.

In the case of B it is assumed that damage to heart muscle has lessened the reserve by two units, leaving only two above constant load for exertion.

In the case of C it is assumed that there is a constant extra load of two units which the heart has tried

to overcome by hypertrophy, adding a little to the reserve but not meeting fully the extra load.

In D is represented a heart which had hypertrophied to meet an extra load but in which the muscle is also damaged, and the gradual increase in the load and in the degree of impairment of muscle efficiency has encroached on the last unit of cardiac reserve and the heart is failing.

While some sign, such as dropsy, when proved to be dependent on the heart, is always indicative of diminished reserve or approaching failure, failure is not always associated with that sign. In many cases one must rely on the patient's symptoms; symptoms now produced by an amount of exertion which previously would have been carried out without discomfort, and in practice one usually, when certain symptoms are complained of, looks for some sign of disease in the heart, either in the direction of a valvular murmur, or enlargement, or some disorder of the rhythm which might indicate muscle damage, and then lays the blame for the symptoms on the heart, provided no more obvious cause is present.

It will be necessary therefore to examine (1) the symptoms which have been complained of by the 320 patients included in the clinical series, (2) the evidence which has been considered sufficient to classify them as cases of heart disease, and (3) the relative importance of various factors in these cases in diminishing the reserve power of the heart.

SYMPTOMS ASSOCIATED WITH LESSENED RESERVE IN 320 CASES OF FAILING HEART.

The symptoms which have been complained of in the series of 320 cases of failing heart, the subject of this investigation, taken in order of frequency are :-

Breathlessness	279	times	or in	87.2%	of the cases.
Pain	130	"	"	40.6%	"
Palpitation	107	"	"	33.4%	"
Exhaustion	78	"	"	24.36%	"
Cough	68	"	"	21.25%	"
Haemoptysis	37	"	"	11.55%	"
Giddiness	23	"	"	7.18%	"
Faintness	14	"	"	4.375%	"
Embolism	14	"	"	4.375%	"

while Dropsy was present in 125 of the cases, or 39.1%. (Details will be found in Tables in appendix A.)

Breathlessness.

This was complained of in 279, or 87.2% of the cases. No division has been attempted of the different types of dyspnoea, namely that which occurs as the result of exertion, and that which is paroxysmal and is frequently independent of exertion, though both types were noted. The former, i.e. that brought on by exertion, is usually the result of defective aeration of the blood and may be considered as a direct consequence of the heart lesion through stimulation by carbon dioxide of the respiratory

centre. When severe it is usually accompanied by some degree of cyanosis. When severe breathlessness is not associated with cyanosis there has frequently been found to be an altered hydrogen-ion concentration of the blood. In neither group has any attempt been made to separate those cases in which orthopnoea was present.

Breathlessness was found to be the most common symptom in each of the valve diseases, and also in the myocardial cases, and it was rather more frequent in the former than in the latter, viz: 89.8% and 79.6%. It was, if anything, a more frequent complaint in female than in male patients (88.8% and 85.75%) and this difference was entirely due to the less frequency of the complaint in male myocardial cases, the figures being - females, 87.2% and males, 74.5%. It was rather more frequent in females with mitral stenosis than in males with mitral stenosis, while the reverse was true of aortic incompetence.

Pain.

Pain has been present in the series in varying degrees of intensity and in very different situations. In 9 cases it was of the nature of Angina Pectoris, while in others of a neurotic temperament it might be said really only to exist in the brain. In some cases it had apparently no bearing on the cardiac disease, in others it was definitely produced on exertion. In some cases, while referred by the patient to the heart, it was found to be

present in the epigastrium and was really associated with an enlarged and tender liver. It was frequently associated with some hyperaesthesia of the skin of the praecordium, but this point was not systematically inquired into.

MacKenzie[6] has recently written at some length on this subject, and suggests that the pain in cardiac disease is due to the contraction of heart muscle against a force it is incapable of overcoming, due to defective coronary blood supply. Allbutt[7] believes that the severer pain of Angina is connected with the lesion in the first part of the ascending aorta, and that an attack of Angina Pectoris is due to the vagal spasm.

It occurs to the writer that the views of MacKenzie and of Allbutt might be correlated. The depressor nerve is connected with the first part of the arch of the aorta. Its function is believed to be the lowering of blood pressure through the agency of the peripheral blood vessels when the pressure in the aorta becomes unnaturally high as a result of effort or peripheral spasm. May it not be that if there is a lesion in the first part of the arch of the aorta, that the depressor nerve is unable to convey the necessary impulse and to produce the reflex lowering of pressure, and that so the heart has to bear an unnaturally heavy load and the result is the attack of pain explained in the way that MacKenzie suggests? It is to be noted, however, that pain in

the region of the heart, and hyperaesthesia, are not necessarily indicative of heart disease, as they occur in other states such as anaemia and functional disorders.

The complaint of pain was made in 130 cases in the series (40.6%) and it was found to be more frequent in male than in female patients (48.2% and 32.25%) and this was apparent in all types of disease. In valve cases the figures were, males - 53.7%; females - 33.88%; and in myocardial disease, males - 34.05% and females - 25.8%. The highest percentage in any one group, was reached in syphilitic aortic disease in males (60.8%), though the female aortic cases (a much smaller group) gave a reading of 55.6%. In other valve diseases, such as mitral stenosis, the male percentage was much higher than the females, the figures being 53.2% and 35%.

The main points to be noted in connection with this symptom were that it was not present in more than half the cases in either sex, and that it was more frequently a male than a female complaint.

Palpitation.

The term palpitation includes several conditions, any cardiac action which protrudes itself on the consciousness being frequently so described.

It was most commonly associated with exertion, but at other times with rest. Rapid heart action was noticed by some as palpitation, by others it was not observed; while

forcible rapid action of the heart produced complaints in varying degrees, which apparently depended as much on the sensitiveness of the patient as on the actual action of the heart. The consciousness of the strong beat which usually follows the pause associated with an extra systole was sometimes complained of as palpitation. In cases of rapid heart action complaint was found to cover such diverse conditions as simple acceleration, simple paroxysmal tachycardia, auricular flutter, auricular fibrillation, and paroxysmal auricular fibrillation. Palpitation may thus be of little significance so far as heart disease is concerned, or it may be of very grave significance, and it is to be noted that it is very common in other morbid states such as anaemia, general debility, and the neuroses.

Palpitation of some kind was present in 107 of the cases, or 33.4%. It was a much more common complaint in females than in males, the figures being 44.1% and 33.8%. It was a more common symptom in valve cases than in myocardial cases (37.2% and 21.18%), being found in 45.4% of female valve cases and 28.9% in male valve cases. It seems probable that the increased frequency in female cases is to be ascribed chiefly to the more sensitive nervous system in them.

Exhaustion.

Under this heading in the series have been included those who complained of undue fatigue. It may be present without exertion in severe cases of heart disease,

and it may be related to defective blood supply to muscles and nerves with consequent defect in nutrition and in the scavaging of waste products. MacKenzie[8] states that it is not truly a cardiac symptom but is to be related to vasomotor defect.

It was present in 24.36% of cases in this series and was more common in females than in males (29.6% and 19.65%) If anything it was more common in valve cases than in myocardial cases, the respective figures being 26.05% and 19.25%.

Cough.

Cough is probably more frequently significant of a complication of heart disease than of heart disease itself.

It was present in only 21.25% of all the cases and was more common in males than in females (23.8% and 18.4%) and likewise more common in valvular cases than in myocardial cases (22.75% and 16.7%). It reached its highest percentage in combined mitral and aortic disease in males (58.3%), but the number of cases in any one group was too small for any deduction to be made.

Haemoptysis.

Haemoptysis was present in 37 cases, or 11.55%, i.e. in a little more than half the number of cases in which cough was present. It was twice as common in valve cases as in myocardial cases (13.23% and 6.42%) and was slightly more common in males than in females (13.1% and 9.86% respectively)

the highest figures being in mitral disease, and combined mitral and aortic disease in males (21.9% and 25%).

Giddiness.

Giddiness was not a common complaint. It only occurred in 23 cases (7.18%). Its relative frequency in aortic incompetence used to be pointed out, and while in this series it reached its highest percentage in rheumatic aortic incompetence in males (15.8%), it is to be noted that it was not a complaint in any of the female patients suffering from aortic incompetence, and it is only noted in 4.2% of the syphilitic aortic incompetence cases.

Faintness.

Faintness was a rare complaint. It occurred in 14 cases, or 4.375%, and like the preceding complaint reached its highest percentage in rheumatic aortic incompetence (10.5%), but the figures were much too small to warrant any other deduction than that in general the complaint is comparatively rare.

Embolism.

Embolism was noted in the same percentage of cases as in Faintness.

Dropsy.

Dropsy was present in 125 of the cases or 39.1%, and was almost equally divided between the two sexes - (females, 40.7%; males, 37.5%). It was more common in

myocardial cases than in valve cases (46.2% and 36.8%) and this difference is rather more apparent in the male cases than in the female cases, the figures being - males: myocardial, 46.8%; valve, 33.88%, and in the females: myocardial, 45%; valve, 39.65%.

It is perhaps rather striking that the highest percentage found in any one group is in female aortic incompetence cases with 55.6%, and also that the percentage in syphilitic aortic incompetence and mitral stenosis in males are almost alike, viz: 30.5% and 31.3%, though both are below average for the whole series.

The relationship of dropsy to auricular fibrillation will be dealt with when dealing with the latter condition, but it may be noted here that while it was present in 39.1% of cases of failing heart, it was present in 48.8% of those cases of failing heart (86 in number) which were associated with auricular fibrillation.

The symptoms we find associated with cardiac failure (apart from the presence of embolism and dropsy) are thus found in persons with healthy hearts when these are subjected to over-exertion. Likewise no single physical sign is evidence of a failing heart if we except alternation of the heart, and it only occurs in some cases, not in all. Dropsy is not in itself evidence of cardiac failure, as it occurs in conditions apart from damaged heart. A heart may fail suddenly or gradually. No doubt in certain cases

of sudden failure some symptoms could have been elicited if carefully sought for, but the patient makes no complaint, or the symptom is so slight as not to attract attention; while in other cases no sign or symptom has been found even when it has been looked for. The type of failure which develops depends on the function of the heart which first gives way, as in the sudden death from failure of contractility, which may follow blockage of a coronary artery.

INDICATIONS OF HEART DISEASE.

GROSS ANATOMICAL CHANGE.

Enlargement:

Evidences of heart disease as obtained by physical examination are comparatively few in number, and one is really limited to determining whether there is gross anatomical change either in the direction of enlargement of the heart, or of disease of the valves.

It may be stated with moderate confidence that if the heart is enlarged the heart muscle is damaged, for while some state that a certain degree of enlargement of the heart may be physiological as a result of training, it is usually found that in later years those hearts are really more susceptible to secondary change than are hearts which are not so much hypertrophied. It is much safer to conclude that any heart which has hypertrophied, either as the result of over-training, or of undue load placed upon it by defect in the valves or by increased blood pressure due to peripheral causes, is a heart with damaged muscle.

Some would, in connection with the enlargement of the heart, differentiate between hypertrophy and dilatation, but while it may, in a number of cases, be possible to state that hypertrophy or dilatation is the predominant factor in the enlargement, in many cases it is impossible to determine this point from ordinary physical examination. (9)

Valve defects:

Valvular lesions are easily recognised by auscultation when the appropriate murmur of mitral stenosis or aortic incompetence has been detected, or, in other words, when one has determined the presence of a definite murmur during ventricular diastole it is safe to conclude that there is damage to either the mitral or the aortic valve. It is much more difficult to conclude from the presence of a murmur during ventricular systole, even when heard at the aortic or at the mitral area, that these valves are actually damaged. Many murmurs in the aortic area are due to dilatation of the aorta beyond the valve, while the latter may be perfectly normal. When due to actual stenosis they are usually accompanied by a thrill. Further, murmurs, frequently designated functional, may also be heard in this area during systole, as well as murmurs produced in neighbouring blood vessels. A systolic murmur at the mitral area presents even more difficulty, as many murmurs are heard in this situation with a perfectly normal heart muscle and mitral valve.

There still remains to be considered whether the murmur is indicative of damage to the valve, or whether it is due to relative incompetence from other causes. In either case it is not safe to conclude that there is damage to the heart, unless some enlargement of the heart itself accompanies the murmur. The propagation of the murmur from the apex towards the axilla, while it is more common

in organic disease than in functional disorder, is not a sign which can be used to differentiate between those two conditions, as loud functional murmurs may be conducted well to the left.

The question of primary disease of the pulmonic or tricuspid valve did not arise in the series.

The broad lines adopted in the present analysis have been, that murmurs heard during diastole were due to organic disease, and that murmurs heard during systole could not be taken as evidence of organic disease unless they were accompanied by enlargement.

The heart may be damaged without there being any enlargement or any evidence of damage to the valve, but to determine this, methods other than the ordinary methods of physical examination have to be employed, and even sometimes then one fails to determine serious damage though it may be present.

FINER INTRINSIC DEFECTS.

Arrhythmias.

Irregularity of the heart's action points to some defect in the finer intrinsic cardiac mechanism. But all irregularities of the heart's action do not indicate disease of the heart muscle, and it will be necessary to examine the various arrhythmias and to indicate their relation to disease of the muscle.

Extra systoles.

Extra systoles occur when there is no other evidence of damage to the heart, as in excessive smoking. On the other hand, they occur most frequently after middle life, and particularly in cases of high blood pressure, i.e. in cases where the muscle may be presumed to be past its best, but they may occur over a long period of years without the person showing any evidence of a weakening heart. Thus they cannot in themselves be taken as evidence of myocardial disease.

Paroxysmal Tachycardia.

Paroxysmal tachycardia, like its relation, extra systole, is not necessarily an indication of heart disease. It may occur as an apparently functional condition, or it may occur in a damaged heart. If the attack lasts for an hour or more, conclusions as to the state of the heart muscle may be drawn from the response it makes to the attack. If the heart dilates, and the liver enlarges, a weakened muscle is indicated. If the heart retains its normal size and distress is not great, it may be assumed that it is fairly healthy.

This condition was noted 5 times in the series.

Auricular flutter.

It may be accepted that this condition is an indication of definite disease of the auricular muscle. It is occasionally paroxysmal, but usually continues till

a further degree of disorder sets in.

Auricular fibrillation.

It may be accepted with confidence that this disorder is evidence of a damaged auricular muscle. Its relationship to cardiac failure will be discussed in detail later on.

Heart-block.

The lesions which produce heart-block are usually the result of rheumatism, syphilis, or other infection, while simple fibrosis also plays a part. It is seldom that the damage to the bundle is the only lesion of the heart, although it may be the only one that speaks in definite signs. It may be accepted then that heart-block is evidence of organic disease of heart muscle.

Sinus-block.

There is another type of heart-block which is very rare, called sinus-block, in which the auricle itself is prevented from contracting. The cause is probably in most cases to be found in a disorder of the vagus

Pulsus alternans.

This may be accepted as a sign of muscle exhaustion, and if present when the heart is quiet, may be taken as an indication of heart disease, and it is usually of serious omen. When it is only found during an attack of tachycardia its significance is not so certain.

Sinus irregularity is not indicative of disease of the heart muscle.

Summary:

Auricular fibrillation, auricular flutter, heart-block, and alternation of the heart, are all serious disorders and indicate diseased heart muscle, and bear an intimate relationship to failure of the circulation.

Paroxysmal tachycardia, if of long duration and frequent in its occurrence, may exhaust the heart, and if occurring in an already damaged heart may cause serious failure and death. If of short duration and infrequent, and occurring in a sound heart, it may only cause temporary discomfort.

Extra systoles throw little load on the heart and are thus of minor importance, and their relation to heart muscle damage is too indefinite for one to express any definite opinion. If not associated with other signs of heart weakness they are better neglected; if associated with other signs of heart weakness it is better to draw conclusions from the latter than from the extra systoles.

The nerve irregularities vary in importance with their cause. The respiratory type may be considered physiological in the young. Occurring in later life they suggest an unstable nerve mechanism, but not heart disease.

Other fine defects.

In addition to the arrhythmias, it is possible in a number of cases to determine the presence of disease by means of the electro-cardiograph even when the rhythm is normal. Such defects as mild degrees of heart-block, or bundle-branch block, or intra-ventricular block, or other abnormalities in the electrical deflections, may be of considerable value in pointing to serious disease.

It may briefly be stated, therefore, that one may obtain evidence of gross anatomical change by finding enlargement of the heart, or by finding a valvular murmur which is indicative of valve disease, and one may seek for finer intrinsic change by analysing any abnormality of rhythm that may be present, or by making an electro-cardiographic investigation into the deflections that the heart produces.

The criteria therefore, which determined the inclusion of any case in this series of failing hearts were (1) the presence of certain symptoms suggesting diminished cardiac reserve; (2) the presence of signs of disease of the heart muscle or valves; and (3) the absence of other conditions which could better explain the symptoms.

Any patient who was in Hospital on more than one occasion is only counted as one case in the series.

While it is necessary, therefore, to be quite clear as to what signs are definitely indicative of heart disease, it has to be remembered that it is still possible that signs of disease may be present without the heart being embarrassed and that therefore symptoms complained of by the patient must not be lightly ascribed to that organ. Valvular lesions must be viewed as evidence of heart damage but they must not be considered as in themselves producing cardiac failure, nor must all cardiac murmurs be considered as significant of valvular lesions. In the same way certain irregularities of the heart are always indicative of disease of muscle, while others have no such significance.

In addition to these facts, one has to bear in mind that in certain cases there may be no cardiac murmur, no enlargement, and no arrhythmia, and the patient may be dying from cardiac failure.

EXPERIMENTAL INVESTIGATION INTO THE EXTRA LOAD PUT ON THE HEART BY DAMAGED VALVES.

It is convenient at this point to describe the experiments conducted with a view to finding out the load added to the work of the heart by damaged valves.

The actual method adopted in the first place was to determine the effect of each of the left sided valvular lesions in diminishing output, from which it should be possible to estimate the relative extra load which each added to the work of the heart if an efficient circulation is to be carried on.

The apparatus about to be described was manufactured to my requirements in 1914, though during the period of the War, and for some time afterwards, sufficient time was not available to conduct the research.

The method of obtaining the force for the circulation was suggested on seeing an article on the Lodholz schema.(10)

APPARATUS USED.

A general view of the apparatus used will be seen in figs. 2 and 3, and the details of the valve in figs. 4 and 5. (pages 32 and 34).

The essential difference between this schema of part of the circulation and those that have been previously described in physiological or other text books is in the use of a variable distributing valve. By this

means it is possible to vary the relative lengths of systole and diastole in the cardiac cycle.

The fluid used in the experiments has been water and the force employed to represent the contraction of the left ventricle has been a head of water, the pressure of which can be varied within certain limits.

The highest pressure used in the experiments was obtained by syphon action from a supply in the room above that in which the apparatus was used and was constant throughout the experiments, representing the equivalent of 280 millimetres of mercury. Lower pressures were obtained by syphon from a supply which could be placed at any height from the level of the schema up to the ceiling of the work-shop. These lower pressures vary up to a maximum equivalent to 154 mm.Hg. The water under pressure is only allowed to pass into vessels which represent the aorta and arteries when the valve is open and the time of opening and closing can be altered. The valve is operated by a variable cam, the position of which can be adjusted to allow of the valve being open from 0.15 to 0.6 of a cycle, and closed from 0.85 to 0.4 (See figure 4c and d. and fig.5 c and d). Cardiac cycles can thus be imitated which have a systolic portion of 0.15 and a diastolic portion of 0.85, or at the other extreme a systolic portion of 0.6 and a diastolic portion of 0.4.

The cam was operated by a belt from the driving

shaft in the workshop of the Department of Physiology in the University.

By means of pulleys of different diameters the speed of the valve could be varied from a basal rate of about 70 per minute, to four times that speed, though only the three lower speeds were used. The speed varied slightly on different days, according to the electric power available.

The tube corresponding to the aorta, and through which the water is forced, has a diameter of 1/2". The aortic tube is made to divide into two tubes by a Y shaped connection, each of the tubes having a diameter of 3/8" and having thus together an area greater than that of the aortic tube. Each of these tubes is similarly divided into two, having a diameter of 5/16". These were the smallest arteries used in the schema. These four tubes pass through a perforated cork into a motor tube loosely packed with glass wool to imitate arteriole and capillary resistance. This tube had a diameter of $2\frac{1}{2}$" and was 10" long. The exit from this resistance area was by a tube of 15/16" diameter, to represent the great veins, with an area much larger than that of the aorta. Further, the outlet of this venous tube was raised $5\frac{1}{2}$" above the level of the schema to represent venous pressure. Incidentally this prevented air locks in the schema.

The means adopted for imitating valvular lesions is as follows:-

For Aortic Stenosis a series of diaphragms of measured size can be inserted into the commencement of the aorta. The aortic tubing has a diameter of 1/2 inch and the diameters of the openings in the diaphragms are 7/16th inch, 3/8th inch and 1/4th inch. These give areas roughly of three-quarters, one-half, and one-quarter that of the aortic tube.

For Aortic Incompetence a groove on the side of the piston allows a leak to take place from the aortic tube during the time that the valve is closed, i.e. during the period corresponding to ventricular diastole but not when the valve is open (Figs. 4a and 5a). The outlet tube, which can be opened or closed at will, has a diameter of 3/8ths inch, and by means of diaphragms this can be reduced to 5/16th inch, 1/4th inch and 3/16th inch, giving areas roughly of three-quarters, one-half, and one-quarter that of the full leak.

For Mitral Incompetence the internal arrangements of the valve are adapted to allow a leak to take place from the supply side during the time that the valve is open (figs 4b and 5b) i.e. during the period corresponding to ventricular systole, but not when the valve is closed. The size of the outlet for this leak, which can be closed or opened at will, is 3/8th inch in diameter. The size of the leak can be varied by inserting diaphragms having a

diameter of 5/16th inch, 1/4th inch and 3/16th inch. These give areas roughly of three-quarters, one-half, and one-quarter that of the full leak.

Mitral Stenosis presented special difficulties and the pressure had to be specially adapted for investigating this condition. A series of diaphragms of 7/16th inch, 3/8th inch and 1/4th inch diameter, allowed one to imitate stenosis in a mitral valve having a full opening of one-half inch, i.e. the area could be reduced to three-quarters, one-half, and one-quarter the full mitral opening. The details will be explained when discussing the results obtained in this condition.

It may be contended that the effect of leakage in the schema is not comparable to what obtains in the heart, in that leakage at the mitral valve passes back into the auricle and that at the aortic valve back into the ventricle. While this does not operate in the schema, it is not a disadvantage because the investigation is to find out the load added to the heart because of the leak.
Though in the heart the blood is not lost to the body, it still by its presence adds load, and in the experiment one is aiming only at determining the mechanical effect of the leak and not, in this connection, the method which the heart adopts to overcome this disability.

Schema of circulation.

DV. Distributing valve.

C. Cam, the position of which can be moved laterally.

H. Handle to alter position of cam.

Sc. Scale to indicate open and closed time of valve depending on position of cam.

PR. Piston rod.

P. Pulley wheels to take the drive and to vary speed.

B. Belt to drive the cam shaft.

V. Supply tube.

A. Aortic tube.

AL. Aortic leak tube.

ML. Mitral leak tube.

AB. Arterial branches.

CAR. Capillary and arteriole resistance.

VO. Tube taking venous outflow.

D. Diaphragms for altering size of outlet tubes.

K. Mercury manometer for kymograph.

CW. Clockwork.

SP. Smoked tracing paper.

RT. Tambour for radial tracings.

SG. MacKenzie's sphygmograph.

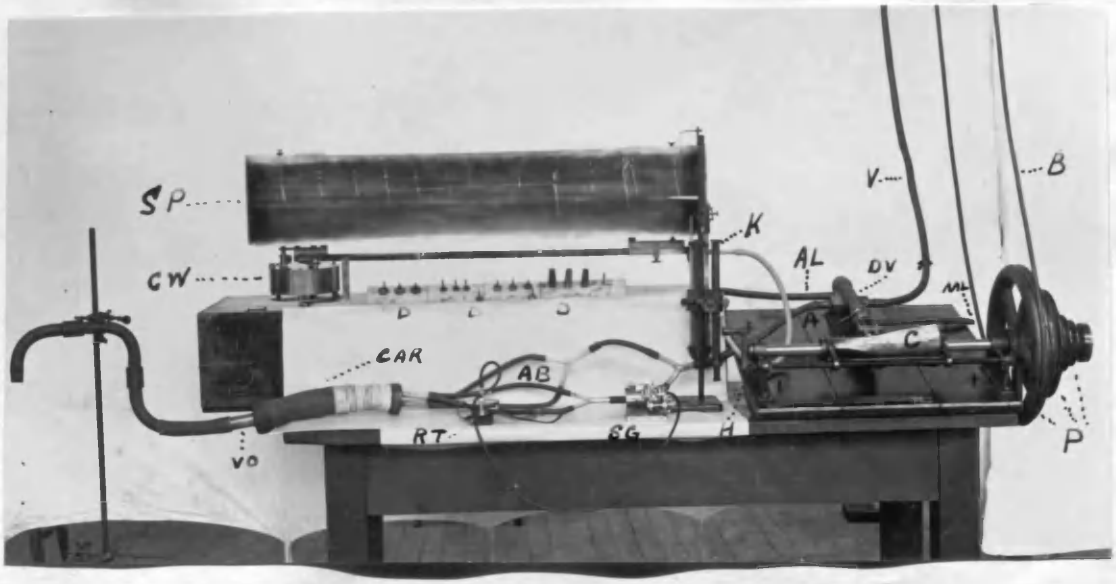


Fig. 2. General View of Schema.

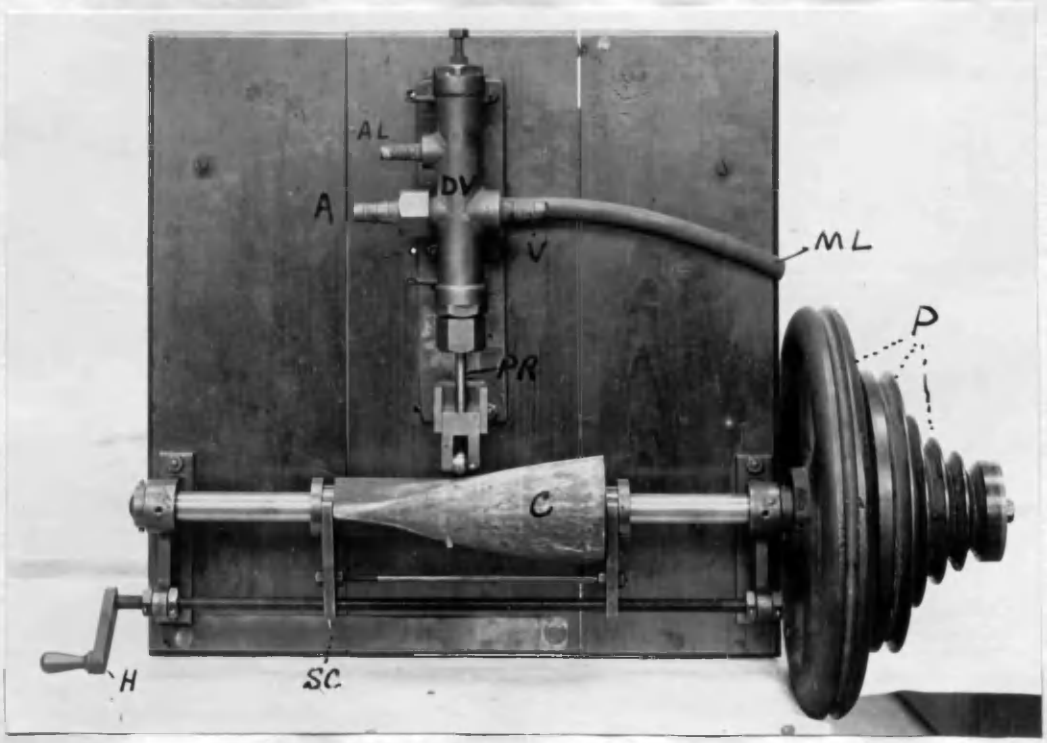


Fig. 3. View of the Valve and Cam.

Distributing Valve - internal arrangements.

V.	Supply tube.	VP.	Valve piston.
A.	Aortic tube.	PR.	Piston rod.
AL.	Aortic leak tube.	C.	Cam.
ML.	Mitral leak tube.	S.	Spring to return piston.

Scale -

FIGURES 4 - VENTRICULAR SYSTOLE.

Fig. 4a Valve open. Piston in most advanced position; ventricular systole(V.S.). No aortic leak.

Fig. 4b Section through V.A. showing the mechanism for permitting a leak during V.S. from supply V to ML - mitral incompetence.

Fig. 4c Part of piston rod and cam showing shape to produce Systole 0.15, Diastole 0.85, and position to secure opening of Valve.

Fig. 4d Part of piston rod and cam(valve open) showing shape to produce Systole 0.6 and Diastole 0.4.

FIGURES 5 - VENTRICULAR DIASTOLE.

Fig. 5a. Valve closed showing the mechanism for permitting a leak from A to AL, i.e. from the aorta during ventricular diastole (V.D.)

Fig. 5b. Section through V.A. during V.D. showing valve closed; no leak through M.L.

Fig. 5c. Part of piston rod and cam in position to secure closure of the valve.(Systole 0.15;Diastole 0.85).

Fig. 5d. Part of piston rod and cam in position to secure closure of valve (Systole 0.6;Diastole 0.4).

Fig. 4a.

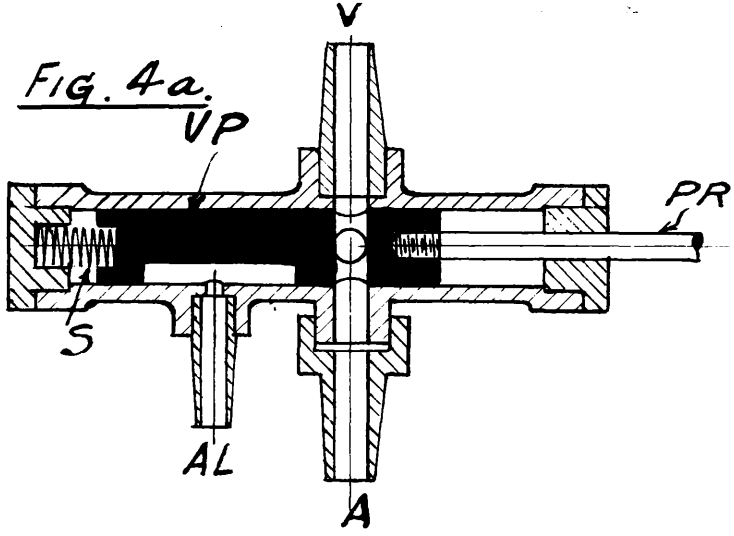


Fig. 4b.

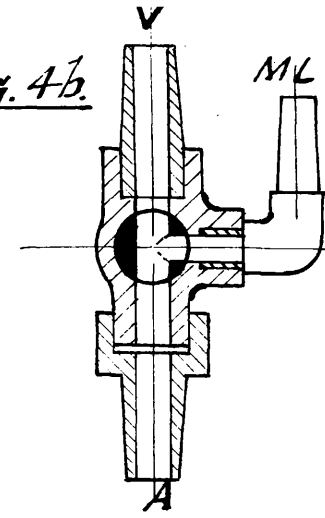


Fig. 4c.

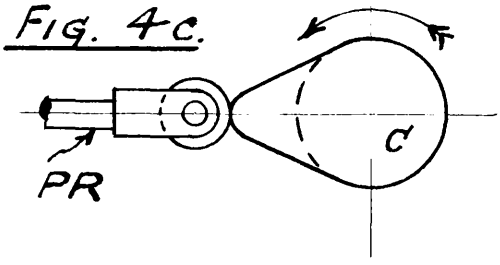


Fig. 4d.

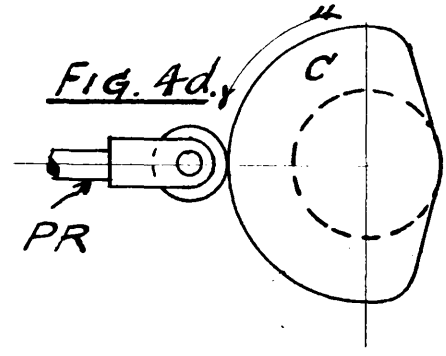


Fig. 5a.

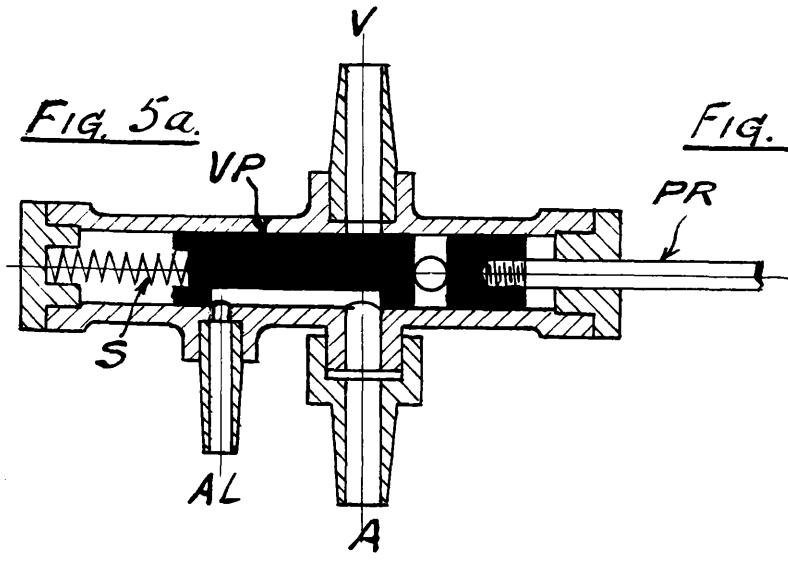


Fig. 5b.

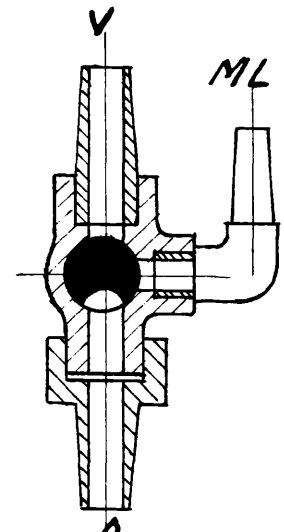


Fig. 5c.

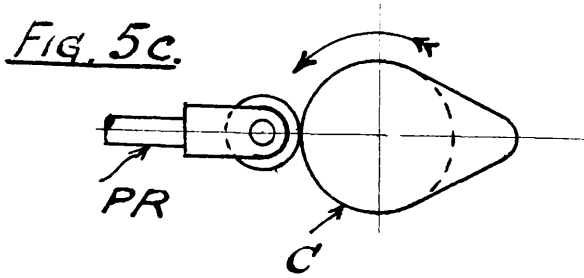
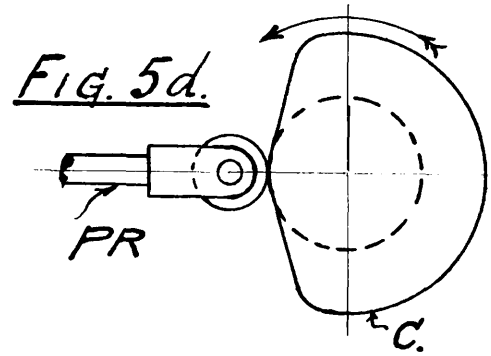


Fig. 5d.



METHOD OF RECORDING RESULTS.

The method for estimating the efficiency of the circulation was by measuring the output. The water that flowed out from the venous tube in measured time (usually 15 seconds), was collected in a glass cylinder graduated in ounces. The outflow from the leaks when being operated was measured during the same period of time into another cylinder.

The maximum and minimum pressures were taken from the aortic tube by means of a U shaped manometer, the connection being made by a length of pressure tubing. The pressures were recorded on a moving drum by means of a glass stilette fastened to a float on the mercury. The inertia of the mercury when oscillating presented considerable difficulty at first, but this was reduced to a minimum by narrowing the calibre of the pressure tubing. Even with this modification there is no doubt that the records of the systolic pressure may be a little too high, and that of the diastolic a little too low, particularly with big differences between systolic and diastolic pressures as in the case of aortic incompetence. The manometer used being of the U type, the graphic records shown in appendix C indicate a pressure which must be doubled to be transferred into millimetres of mercury.

It was found impossible to obtain satisfactory radial tracings from the rubber tubes already described, but this was overcome by taking from the smallest arterial

tubing a branch made of collapsible rubber tubing and re-inserting it a few inches distal to its origin. The tracings were taken from this tube by the ordinary radial adjustment of a MacKenzie polygraph. It was found necessary to compress the artery proximal to the tambour to get the best records.

The kymograph records and pulse tracings from all the experiments used in the argument are shown in the Appendix.

It has to be noted that as the output was measured for 15 seconds in a glass cylinder graduated in ounces, an error of half an ounce would make one of two ounces in the quantity per minute. Further, in the case of aortic and mitral leak, the flow being intermittent there is the further possibility of error in that the output for the last cycle might be just missed, or just obtained, according to the speed of the apparatus.

All readings of outputs must be considered as approximate and no deductions should be drawn from differences of 2 ounces or less.

Early experiments were undertaken to find out the resistance required to permit of a reasonable output when the apparatus was running under normal conditions. For this purpose systole was fixed at 0.3 of the cycle and diastole therefore 0.7 of the cycle, with a speed of about 70. The resistance was then adjusted to give an output of about 70 ounces per minute.

Considering that the aorta of the schema had an area between one-third and one-quarter that of the human aorta (schema 1/2" diameter: aorta 9/10" diameter), the output was comparable to what is usually accepted as the output of the left ventricle. Owing to the filtering action of the glass wool in the resistance bed, the output varied gradually and the wool had to be cleaned at times, but in any case the normal was determined each day prior to doing comparative experiments.

No deductions regarding the load put on the heart have been drawn from the radial tracings inserted in Appendix C, which have been obtained from the various experiments. They were first used to find out whether the curve of pressure in the peripheral vessels of the schema was at all comparable to what obtained in the radial artery. As they were found in most cases to be practically indistinguishable from these they were continued throughout the various series, and it may be noted that the tracings obtained from the conditions simulating aortic incompetence and aortic stenosis have a similar form to those which it is the custom to ascribe to these conditions in the Wards.

In the tables of results the following contractions are used :-

N.	Normal.
A.I.	Aortic Incompetence.
A.S.	Aortic Stenosis.
M.I.	Mitral Incompetence.
M.S.	Mitral Stenosis.
R.	Rate per minute.
S.	Fraction of cardiac cycle as systole.
D.	Fraction of cardiac cycle as diastole.
O.	Output in ounces per minute.
L.O.	Output in ounces per minute through leak.
B.P.	Pressure on kymograph tracings in mm.Hg.
A.	Diameter of aortic outlet.
L.	Diameter of leak outlet.
H.P.	Head of pressure from room above in millimetres of mercury.
L.P.	Head of pressure from workshop in millimetres of mercury.
Numbers	refer to number of experiment in the series and to kymograph and radial tracings in Appendix.

PRELIMINARY EXPERIMENT.

A preliminary tracing was taken to determine the effect of gradual increase in the length of systole and decrease in the length of diastole within the limits of the schema. The rate and head of pressure were constant at 74 per minute and 280 mm.Hg. respectively.

With systole 0.15 of the cycle and diastole 0.85, the output was 31 ounces, with a maximum pressure in the aorta of 60 mm.Hg and minimum pressure of 30 mm.Hg.

The position of the cam was gradually altered with the valve operating. At the other extreme, with systole 0.6 and diastole 0.4 of a cycle, output was 198 ounces per minute with a maximum pressure in aorta of 124 mm.Hg., and a minimum pressure of 50 mm.Hg.

Preliminary record to show the effect on output and on maximum and minimum pressures of increase in length of systole:

H.P. 280 mm.Hg. Aortic Opening - 1/2"diameter.

Rate - 74

S.	D.	O.	B. P.
0.15	0.85	31	60/30
0. 6	0. 4	198	124/50
0.3	0.7	80	90/42

RESULTS OF EXPERIMENTS, WITH TABLES, SHOWING THE MECHANICAL EFFECT OF VALVULAR LESIONS.

AORTIC STENOSIS.

Several sets of experiments were conducted with a view to showing the effect of this lesion in diminishing output, but only a few of these are given in the succeeding tables, as results were consistent throughout.

SERIES A shows the effect of Aortic Stenosis in diminishing output with varying lengths of systole and diastole. The normal readings are with the aorta of 1/2" diameter. The diaphragm used to produce Aortic Stenosis was the smallest available in the schema and gave a diameter of 1/4", i.e. the area was one quarter that of the full aortic tube. Other experiments were conducted with the intermediate sizes of diaphragms, but the variations in output were so slight as to make the results inconclusive within the limits of error of the apparatus.

It will be observed with Aortic Stenosis giving an area of only one quarter the aorta, the output is diminished very slightly with a systole of 0.3, the figures being from 62 to 59 ounces, and on another occasion from 56 to 46 ounces, which was the largest reduction obtained with this length of systole and amounted only to 18 per cent.

With prolongation of systole, the differences are likewise inconsiderable compared with the diminution in the area of output, and even with a systole of 0.5 the normal

output was 133 ounces, while that with Stenosis was only reduced to 108. At the higher speed slightly smaller differences were obtained.

Further, it may be noted from this table that increase in the duration of systole to 0.4 of a cycle fully compensates Aortic Stenosis at both of the speeds.

SERIES B likewise shows the effect of increase in the length of systole and also the effect of increasing the head of pressure in Aortic Stenosis. The standard pressure used was the low pressure - 154 mm.Hg. and the normal output with S - 0.3 was 34 ounces. With Aortic Stenosis of the same size as in the former experiment, the output was reduced to 31 ounces, but increasing systole to 0.4 the output rose to 43 ounces.

When the pressure was increased to 280 mm.Hg. even with a systole of 0.3, the output rose to 57 ounces, a very considerable increase over the normal output of 34 ounces. At the increased rate of 104 the result was still more striking(the systole being, of course, relatively longer, viz: 0.4). The output was increased to 72 ounces.

SERIES C gives confirmation of these results taken on another day. With a normal output of 36 ounces at low pressure, the Aortic Stenosis reduced the output only to 34 ounces, while an increase of pressure

raised the output to 54, and increase of pressure with increase in the length of systole raised the output to 82 ounces.

Several conclusions may be drawn from these experiments:-

(1) Narrowing of the outlet of the aorta does not reduce the output in proportion to the degree of the stenosis. Diminishing the aortic orifice to one-quarter its area produces a comparative reduction in output.

Arguing from those cases which have shown a larger reduction in output than some of the others, we find as in series A that a reduction in the size of the aortic opening by 75% only diminishes the output by about 18%.

(2) With the stenosis and reduction in output, the pressure is also reduced and this is slightly more marked in the systolic than in the diastolic pressure.

(3) The velocity of the flow through the narrowed orifice must be very much increased.

(4) Slight relative increase in the duration of systole, which would be accomplished in the human heart by a moderate acceleration of rate, is sufficient to overcome the defect.

(5) Increase in the ventricular pressure from 154 millimetres to 280 millimetres increased the output from 31 ounces to 57 ounces, the normal low pressure output being 34 ounces. In other words the increase in output was practically in

direct proportion to the increase in the force supplied, viz: a little over 80%.

If, therefore, the output with the aortic stenosis orifice reduced to one-quarter the size of the aorta only diminishes the output by 18%, it would appear that an extra percentage of force (a little more than 18%) would be sufficient to overcome the effects of the lesion.

Thus, if the figures adduced from Krogh's and Starling's work are correct, and there is four times the amount of force in reserve that the heart uses when acting quietly, then Aortic Stenosis of a considerable degree of severity would only use up about one-sixth of one of these units of reserve. The aortic orifice would require to be very small indeed to add any considerable load to the heart's work in this condition.

SERIES A.

AORTIC STENOSIS.

Series to show the effect of Aortic Stenosis in diminishing output at varying lengths of systole and diastole, and at different heart rates but with constant head of pressure :-

H.P. 280 mm.Hg.

Normal. A - 1/2" diameter.				Rate 67.	Aortic Stenosis. A.S. - 1/4" diameter.		
No.	S.	O.	B.P.	No.	S.	O.	B.P.
70	0.3	62	108/56	142	0.3	59	96/51
72	0.4	96	125/64	144	0.4	83	108/55
74	0.45	115	132/70	146	0.45	97	115/58
76	0.5	133	139/75	148	0.5	108	119/61.
				Rate 106.			
78	0.3	64	96/50	150	0.3	55	85/48
80	0.4	96	114/60	152	0.4	86	98/51
82	0.45	108	121/64	154	0.45	99	104/61
84	0.5	120	127/68	156	0.5	110	108/64
On another date:				Rate 68.			
281	0.3	56	108/66	282	0.3	46	100/64
				Rate 104.			
288	0.45	80		285	0.45	74	

SERIES B.

AORTIC STENOSIS.

Series to show the effect of increased lengths of systole and of increased pressure on Aortic Stenosis:

H.P. 280 mm.Hg.
L.P. 154 mm.Hg.

A.S. - 1/4" diameter.

Normal.			Rate - 68.	Aortic Stenosis.			
No.	S.	O.	B.P.	No.	S.	O.	B.P.
449.	LP. 0.3	34	71/55.	446	LP. 0.3	31.	66/52
448.	LP. 0.4	47	84/67	447	LP. 0.4	43	76/64
				440	HP. 0.3	57	111/87
				441	HP. 0.4	82	93/70
			Rate - 104.				
450.	LP. 0.3	32	68/54	445	LP. 0.3	30	62/50
451.	LP. 0.4	48	83/68	444	LP. 0.4	46	77/64
				442	HP. 0.3	48	83/65
				443	HP. 0.4	72	101/80

SERIES C.

AORTIC STENOSIS.

Series to show the effect of increased pressure and increased heart rate with shortened diastole on Aortic Stenosis:

A.S. - 1/4" diameter.

Low Pressure.				High Pressure.			
			Rate - 66				
No.	S.	O.	B.P.	No.	S.	O.	B.P.
N. 420.	0.3	36	69/46.	423.	0.3	63.	92/54
AS. 422.	0.3	34	64/43	424	0.3	54.	85/52
			Rate - 104.				
				A.S.428.	0.45	82	97/67.

AORTIC INCOMPETENCE.

In the experiments designed to estimate the mechanical effects of Aortic Incompetence, the leak had to take place from the aortic side of the system during the time that the valve was closed. This was obtained by the mechanism already described (see Figures 4a and 5a).

In the human heart the blood leaks back into the ventricle; in the schema this cannot be imitated exactly. It is necessary, however, to note the fact that in Aortic Incompetence the blood passes from the aorta with a definite pressure into the ventricle against comparatively little, if any, resistance. At the beginning of ventricular diastole in the human heart the pressure in the left ventricle is zero, and some believe that there is no positive pressure in the left ventricle till it contracts. Starling (11) states: "Pressure in the ventricles remains at or near the zero line during the greater part of diastole. With a big inflow there may be a slight rise towards the end of diastole which may be accentuated by the auricular contraction."

In the experiments on Aortic Incompetence the leak was allowed to operate against zero pressure, as it was considered that this most nearly approached the conditions present in the human heart.

SERIES D: shows the effect of Aortic Incompetence in diminishing output. The diameter of the aorta is 1/2" and the diameter of the tube taking the leak is 3/8", the area of the leak being thus 9/16", or a little more than a half of the full aorta led out at zero pressure. With a systole occupying 0.3 of the cycle, the output diminishes from a normal of 62 ounces to 22 ounces, i.e. a leak of 56% of the aorta reduces the output by 65%. This is apparent in lessening degree as diastole is shortened. Thus, with systole and diastole equal, the normal output is 133 ounces and with the same leak as before, the output diminishes to 97 ounces, a reduction of only 27%. Similar differences are noted at the higher rate speeds.

It is further to be noted - and this is important - that when with a constant rate (67) the diastole is shortened to be only 0.55 of the cycle, the output is then 78 ounces, i.e. by shortening diastole the lesion has been more than compensated, the normal output with diastole at 0.7 being only 62 ounces. At the higher speed of 106, diastole shortened only to 0.6 instead of 0.7, compensates the leak, the output being 68 ounces.

SERIES E. If the area of the aortic leak is equal to the full area of the aorta, as in series E, where both are 3/8" diameter, similar results obtain. The normal output with this size of aorta is 59 ounces per minute,

systole being 0.3 and diastole 0.7. If diastole is shortened to 0.55 the leak is compensated at any of the three speeds.

The conclusion that may be drawn from these two sets of experiments is that reduction in the length of diastole is sufficient to compensate for a lesion of aortic incompetence, even if the leak takes place through an opening equal in size to the whole aorta, and this would take place chiefly by increase in the heart rate.

SERIES F shows the effects of variation in the size of the aortic leak, other factors remaining constant, and with the aorta at full opening of 1/2" diameter. With a maximum leak through the tube of 3/8" diameter, the heart output is 23 ounces. When the leak is reduced to 3/16" diameter, or one-fourth of the area, the output only increases to 29 ounces. This is in keeping with the results obtained in Aortic Stenosis, in so far that in that condition the actual diameter of the aortic opening makes comparatively little difference in the output, the velocity of outflow through the narrow opening being apparently much increased. That experiment was conducted with the schema working at a rate of 66 per minute and with systole 0.3 and diastole 0.7.

When the rate is increased to 104 per minute,

as in SERIES F I, and if the increased rate were obtained entirely by shortening diastole, as is supposed to occur in the case of the human heart, then the actual duration of each systole would remain the same and would be obtained in the schema by a systole 0.45 of the shortened cycle. At the 66 rate the actual duration of systole is 0.27 seconds; at the 104 rate it would be 0.26 seconds. With this alteration the output at once increases and, with any of the sizes of aortic leak, fully overcomes the effect of the lesion. This corresponds as nearly as possible with what obtains in the human heart, as one finds that almost invariably in a patient who is suffering from Aortic Incompetence the heart rate is increased and diastole shortened.

The conclusion which may be drawn is therefore that increase in the heart rate in Aortic Incompetence is part of the compensatory mechanism.

With regard to the effect of Aortic Incompetence on blood pressure, it is to be noted that the reduction is more marked in the minimum than in the maximum pressure. Thus, with a leak of 3/16" diameter the pressure is reduced from 109/54 to 88/26, a reduction of 19% in the maximum and 52% in the minimum pressure; and with a leak of 3/8" diameter(or half the area of the aortic orifice) the pressure is reduced to 80/14, a reduction in the maximum of 27% and in the minimum pressure of 74%. The reduction in output is noted to be roughly in proportion to

the reduction in the minimum pressure.

An attempt may be made to find out the load added by such a lesion, by comparing the amount of work done under these two conditions. The same force operates in each case and one may estimate the energy expended by calculating the time during which the force acts in each case. As the actual duration of systole is the same at the 66 rate and at the 104 rate, the energy expended in the two cases would be proportionate to the rates; therefore the increase of force would be 58%. Or, one may calculate the work done in the two cases, this being in proportion to the output multiplied by the resistance. The normal output was 64 ounces and the resistance 54 mm.Hg. while with the leak, 106 ounces (78 plus 28) had to be moved against a resistance of 50 mm.Hg. This gives an increase in work done of 50%.

SERIES G shows the effects of increase in the force of the ventricle in overcoming Aortic Incompetence. In this series two pressures were used - a low pressure equivalent to 120 mm.Hg. and a high pressure equivalent to 280 mm.Hg. The normal output, without Aortic Incompetence, at the low pressure was 21 ounces, and with a moderate degree of aortic leak in the schema, viz: 1/4" diameter, the output was only 2 ounces, while the pressure fell from 64/46 to 36/11. When the high pressure was used under the same circumstances, the output was only increased to 14 ounces, while the blood pressure was raised to 73/25, the systolic pressure being

above and the diastolic pressure below the normal low pressure readings.

Stated briefly it is to be noted that more than double the force applied was not sufficient to compensate for a comparatively moderate leak. The greater the force the greater is the systolic blood pressure and consequently the greater the leak.

SERIES H. On a subsequent occasion a similar experiment was conducted, but the low pressure had been adjusted to give a pressure of 154 mm.Hg., while the leak was reduced still further to 3/16" diameter, or the smallest leak possible with the schema. The normal output at low pressure was 31 ounces, while with Aortic Incompetence of this degree, it fell to 8 ounces. With the high pressure (280 mm.Hg.) the output only rose to 26 ounces. Again, almost doubling the pressure did not compensate for a leak which was only 9/64ths (say one-seventh) of the size of the aorta. When, however, diastole was shortened from 0.7 to 0.55, even with low pressure, the output increased to 29 ounces, while with high pressure it shot up to 52 ounces.

The conclusion is similar to those already obtained, viz: that considerable increase in force is unable to compensate quite a small leak, while an alteration in the length of diastole is easily able to

overcome the difficulty.

The conclusions that may be drawn from these experiments are :

(1) Aortic Incompetence is a more serious mechanical disability than Aortic Stenosis.

(2) A leak of from 1/2 to 1/7th the area of the aorta reduces the output(other factors remaining constant) by more than one half, actually 65% to 55%.

(3) A leak within the limits of the schema reduces the maximum blood pressure by 20-25% and the minimum pressure from 50 to 75%.

(4) The size of the leak is relatively unimportant within the limits of the schema.

(5) Reduction in the duration of diastole is the most economical single method of overcoming the effects of the lesion.

(6) Doubling the ventricular force is barely sufficient to compensate the leak.

(7) Probably a combination of shortened diastole and increased force is the best method of compensation.

(8) A leak of moderate severity is capable of using up about one unit of reserve force, equivalent to the amount of force expended with the heart acting quietly.

SERIES D.

AORTIC INCOMPETENCE.

Series to show the effect of Aortic Incompetence on output with different lengths of systole and diastole, and at different rates, with a constant head of pressure and constant size of aortic orifice and aortic leak. The effect on blood pressure is also shown :

H. - 280 mm.Hg. A. - 1/2" diameter. A.I.L. - 3/8" diam.

Normal.				Rate - 67.	Aortic Incompetence.			
No.	S.	O.	B.P.	No.	S.	O.	L.O.	B.P.
70	0.3	62 oz.	108/56.	71.	0.3	22 oz.	46	81/15
72.	0.4	96 "	125/64	73	0.4	52 "	44	105/29
74.	0.45	115 "	132/70	75	0.45	78 "	41	119/39
76.	0.5	133 "	139/75	77	0.5	97 "	36	128/46
				Rate - 106.				
78.	0.3	64 oz.	96/50	79		36 oz.	25	83/30
80	0.4	96 "	114/60	81		68 "	24	108/44
82	0.45	108 "	121/64	83		80 "	20	115/52
84	0.5	120 "	127/68	85		92 "	16	122/57
				Rate - 156.				
86	0.3	40 oz.	70/44	87		28 oz.	8	59/31
88	0.4	56 "	88/50	89		44 "	9	73/43
90	0.45	63 "	82/51	91		56 "	6	78/48
92	0.5	73 "	86/54	93		66 "	4	86/55

SERIES E.

Shows effects of Aortic Incompetence on output. Effects of different lengths of systole and diastole and at different rates, with constant head of pressure, and with aortic leak equal to aortic orifice, each being 3/8" diameter :

H. - 280 mm.Hg. A.- 3/8" diameter. A.I.L. 3/8" diam.

Normal.			Rate - 68.		Aortic Incompetence.		
No.	S.	O.	B.P.	No.	O.	L.O.	B.P.
118	0.3	59	106/56	119.	19.	44.	78/18.
120	0.4	88	124/68	121.	46	43	102/32
122	0.45	104	131/72	123	62	40	114/39
124	0.5	118	138/78	125	78	39	125/47
			Rate - 104.				
126	0.3	59	94/51	127	27	25	80/31
128	0.4	92	116/24	129	58	26	106/47
130	0.45	104	122/68	131	68	22	114/54
132	0.5	124	(R.96) 123/70	133	88	22	(R.96) 116/56
						L.	
134	0.3	50	79/44	135	35	8.5	70/37
136	0.4	66	89/52	137	39	6.	85/50
138	0.45	72	90/53	139	62	5.	89/54
140	0.5	81	93/55	141	74	2.	94/59

SERIES F.

Shows effect of variation in size of aortic leak, other factors being constant:-

Constant aortic opening - 1/8"
Constant head of pressure 280 mm.Hg.
Constant rate - 66 per minute.
Constant cycle - Systole, 0.3 : Diastole, 0.7.
Varying aortic leak - 3/8" to 3/16".

No.	S.	O.	L. O.	B. P.	A.I.L.
166	0.3	64		109/54	
167	0.3	23	44	80/14	3/8"
168	0.3	22	44	80/15	5/16"
169	0.3	22	44	80/15	1/4"
170	0.3	29	38	88/26	3/16"

F.(a)

Similar to series F except - S. - 0.45
D. - 0.55
Rate - 104.

171	0.3	63		94/52	
172	0.45	118		120/66	
173	0.45	88	22	114/54	3/16"
174	0.45	80	26	112/50	1/4"
175	0.45	80	26	119/47	5/16"
176	0.45	78	28	114/50	3/8"

SERIES G.

To show the effect of increased force on Aortic Incompetence:-

H.P. 280 mm.Hg. A. 1/2" diameter.

L.P. 120 mm.Hg. A.I.L. 1/4" diameter.

Rate - 64.

No.		S.	O.	L. O.	B. P.
N. 295	L.P.	0.3	21. oz.	-	64/46
A.I. 294	L.P.	0.3	2. "	24	36/11
N. 289	H.P.	0.3	54 "	-	116/74
A.I. 299	H.P.	0.3	14 "	47	73/25

SERIES H. To confirm the effect of increased force:-

H.P. 280 mm.Hg. A. 1/2" diameter.

L.P. raised - 154 mm.Hg. A.I.L. 3/16" diameter.

Rate - 70.

No.		S.	O.	L. O.	B. P.
N. 324	L.P.	0.3	31 oz.	-	78/56
A.I. 325	L.P.	0.3	8 "	28	53/33
N. 328	H.P.	0.3	56 "	-	116/70
A.I. 329	H.P.	0.3	26 "	38	81/57

Rate - 104.

N. 320	L.P.	0.45	52 oz.	-	96/70
A.I. 321	L.P.	0.45	29 "	24	80/49
A.I. 331	H.P.	0.45	52 "	25	121/76

MITRAL INCOMPETENCE.

In arranging the apparatus to record the effects of Mitral Incompetence, note has to be taken of the level at which the leak is led out. During the whole of ventricular systole, the period during which mitral incompetence operates, the pressure in the left auricle does not, according to the physiologists, rise much above zero, the auricle gradually distending as blood enters it from the veins. It is only during auricular systole that the blood in that cavity is under any appreciable pressure, and even then it is probably not high. It is probable,however, that in cases of mitral incompetence, the pressure in the auricle may be increased during the time of operation of this leak.

In the experiments it will be observed that records have been made of the effect of mitral incompetence when the leak is led out against zero pressure, and also when it is led out against a pressure of 7" of water, which is equivalent to a pressure in the left auricle of about 13 mm.Hg. This was done lest the records of the leak at zero pressure were in any way exaggerated,but it is doubtful if this is necessary.

While the size of diaphragms used to determine the amount of the mitral leak were of exactly the same dimensions as those used for the aortic leak, they would thus be smaller in comparison with the size of a mitral valve, which has a diameter of one and one-

ninth greater than the aorta. Thus, while a disc of 3/16" diameter is about one-seventh the area of the aorta in the schema, it would only be about one-tenth the area of a corresponding mitral orifice.

SERIES J was designed to show the effect of varying degrees of Mitral Incompetence whether led out against zero pressure, or against a head of pressure of 7" of water. It is to be noted that while the size of the mitral leak affects the output, it does not do so in direct proportion to its size. With a leak through an opening of 3/8" diameter, or one-half the area of the aorta, the output is reduced from 64 to 32 ounces, or by 50%, and causes a diminution in pressure from 114/61 to 81/45, a reduction of 29% in the systolic pressure and 26% in the diastolic pressure. When the leak is 3/16" diameter, or one-seventh the area of the aorta, the output is reduced from 64 ounces to 46 ounces, or by 28%, and the Blood Pressure falls from 114/61 to 99/55, a reduction of 13% in the systolic and 10% in the diastolic pressure.

Thus, with the smallest leak, which is one-fourth the area of the largest, the output is increased by the lesion one half. And it has to be noted that though the smallest leak is only equivalent to about one-seventh the area of the aorta, when led out against zero pressure it delivers almost as much fluid as passes into the aorta. This is obviously due to the resistance offered by the pressure

in the aorta, This point is probably not so much appreciated in clinical conditions.

Given a leak at the mitral valve equal in size to the aorta, much more blood should pass into the left auricle than into the aorta, as the pressure in the auricle is very much less than in the aorta.

SERIES K shows the effect of relative increase in systole as may be produced by increase in rate. It is to be noted that increase in the length of systole produces an increase in the output in mitral incompetence, but this in no case in the series does increase in systole, even up to 0.5, compensate for the lesion, the highest figure obtained being 49 ounces at the low speed when systole is increased from 0.3 to 0.4 against a normal output of 56 ounces.

It is further to be noted that to obtain an output of 49 ounces, the heart had to deal with 185 ounces of water. This is in striking contrast to the results obtained in Aortic Incompetence.

SERIES L is similar to the last one with the exception that the diameter of the leak is reduced to 3/16". It is to be noted that when the leak is sufficiently small, in this case being equal to about one-seventh the area of the aorta, prolongation of systole from 0.3 to 0.4 is sufficient to fully compensate for the leak, the output being 73 ounces as against the normal at

0.3 of 66 ounces. But again it is to be noted that in order to obtain this result the heart had to deal with 135 ounces of water.

SERIES M shows the effect of increased pressure in Mitral Incompetence. It is to be noted that an increase of pressure from 154 to 280 mm.Hg. or of 80%, is fully able to compensate for a mitral leak equal to one-quarter the size of the aorta, the output being 40 ounces against the normal at the low pressure of 34 ounces. When systole is increased to 0.45, even at the low pressure the leak was just compensated, while at the high pressure it almost doubled, being 64 ounces against the normal of 34 ounces.

SERIES N is a further attempt to determine the effect of increased pressure in this case with the minimum mitral leak possible with the schema. It is to be noted that with a reduction in the size of the leak, increase in pressure from 154 to 280 mm.Hg., or by 80%, is more than ample to compensate for the loss due to Mitral Incompetence, the output with systole at 0.3 being doubled and almost 50% above the normal output without a leak.

The following conclusions may be drawn from this group of experiments :-

(1) Mitral Incompetence is more serious mechanically than Aortic Stenosis but does not reduce the output so much as

Aortic Incompetence.

(2) With leaks from one-half to one-seventh the area of the aorta. mitral incompetence reduces output from 50 to 28%, while aortic incompetence reduces it from 65 to 55%.

(3) A leak within the limits available with the schema, reduces the maximum blood pressure from 29 to 13% and the minimum blood pressure from 26 to 10%.

(4) In no case is the minimum blood pressure reduced so much as in aortic incompetence, the reduction with the latter being - maximum 25 to 20%, and minimum 75 to 50%.

(5) The size of the leak is more important in determining the output than in the case of aortic incompetence.

(6) Increase of pressure by 80% easily compensates any leak available with the schema.

(7) Relative increase in systole within reasonable limits cannot compensate a leak equal to one-quarter the size of the aorta but can compensate a smaller leak of one-seventh.

(8) A leak of moderate severity probably uses up about one-half of one unit of reserve, or half the force required by the heart when acting quietly.

MITRAL INCOMPETENCE.

SERIES J.

To show the effect of varying degrees of Mitral Incompetence when led out against zero pressure, and against a head of 7" of water, the size of the aorta, the rate of beat, the duration of systole and head of supply pressure remaining constant :

Aortic opening - 1/2".
Head of Pressure - 280 mm.Hg.
Rate - 67 per minute.
Cycle - Systole, 0.3: Diastole, 0.7.
Varying leak - 3/8" to 3/16".

Against zero pressure.					Against 7" water pressure or 13 mm.Hg.			
No.	L.O.	O.	L.	B.P.	No.	O.	L.	B.P.
272		64		114/61.				
274	3/8"	32	90	81/45	273	41	58	93/53
276	5/16"	36	84	83/50	275	40	56	94/54
278	1/4"	39	66	89/53	277	43	48	96/54
280	3/16"	46	42	99/55	279	54	37	102/57

SERIES K.

To show the effect of varying proportions of systole and diastole in Mitral Incompetence led out against zero pressure and at different speeds, the pressure, size of aorta and size of leak being constant (the leak being maximum, 3/8" diameter).

H.P. 280 mm.Hg. A.1/2" diameter. M.I.L. 3/8" diameter.

NORMAL.				MITRAL INCOMPETENCE.				
			Rate - 64.					
No.	S.	O.	B.P.	No.	S.	O.	L.	B.P.
196	0.3	56	98/54	197	0.3	20	78	60/40
199	0.4	105	136/70	198	0.4	49	136	80/52
200	0.25	52	120/52	201	0.25	25	73	68/43
			Rate - 98.					
202	0.3	56	92/54	203	0.3	24	82	60/42
205	0.4	91	115/67	204	0.4	40	128	75/51
			Rate - 146.					
208	0.4	74	102/60	209	0.4	28	116	65/49
211	0.5	89	109/68	210	0.5	29	144	65/50
212	0.3	56	90/57	213	0.3	23	76	61/47

SERIES L:

To show the same points as series K, the mitral leak being reduced to the minimum used in the schema, viz: 3/16" diameter:-

H.P. 280 mm.Hg. A. 1/2" diameter. M.I.L. 3/16" diam.

Normal.				Mitral Incompetence.				
No.	S.	O.	B.P.	No.	S.	O.	L.	B.P.
			Rate - 66.					
214	0.3	66	113/59.	215	0.3	50	44	97/53
217	0.4	100	133/75	216	0.4	72	63	116/65
218	0.25	52	97/51	219	0.25	36	36	83/45
			Rate - 100.					
220	0.3	56	90/52	221	0.3	40	44	76/46
223	0.4	90	115/66	222	0.4	65	62	96/57
			Rate - 150.					
227	0.5	80	107/66	226	0.5	52	76	86/55
228	0.4	67	100/59	229	0.4	44	58	78/50
231	0.3	52	95/61	230	0.3	32	40	71/46

SERIES M.

Series to show the effect of increased pressure in overcoming Mitral Incompetence - leak 1/4"diameter led out against a pressure of 7" of water:

L.P. 154 mm.Hg.			Rate: 66-70.		H.P. 280 mm.Hg.			
No.	S.	O.	L.	B.P.	No.	O.	L.	B.P.
N.333	0.3	34	-	66/44				
MI.335	0.3	24	16	55/43	354	40	34	80/55
MI.336	0.45	35	24	70/53	353	64	56	104/70

SERIES N.

To show the effect of increased pressure at different speeds and with the mitral leak reduced to the minimum of 3/16" :

L.P. 154 mm.Hg.			Rate : 66 - 69.		H.P. 280 mm.Hg.			
No.	S.	O.	L.	B.P.	No.	O.	L.	B.P.
N.357	0.3	33	-	70/51				
MI.358	0.3	24	18	57/41	383	48	28	81/48
MI.361	0.45	36	26	68/52	381	82	48	102/62
			Rate : 100-104.					
MI.365	0.45	43	33	75/56	379	72	44	96/64
			Rate : 150-152.					
MI.371	0.5	30	42	59/40	377	56	37	84/54
MI.373	0.6	35	52	64/46	375	62	50	89/59

MITRAL STENOSIS.

In adapting the schema to imitate Mitral Stenosis, certain physiological and anatomical facts have to be recognized :-

(1) The mitral orifice is larger than the aortic orifice, the respective diameters being 1.1 and 0.9 inches - the area of the mitral is therefore about one and a half times the area of the aorta.

(2) The pressure in the auricle, even during its systole, is never so high as that in the ventricle during its systole.

(3) The valve is open for a relatively long time, roughly 5/8ths of the cardiac cycle.

(4) Blood passes from the auricle with a relatively low pressure into the ventricle with zero pressure. There is no resistance comparable to that which the ventricle has to overcome. In diastole, pressure in the ventricle falls at first rapidly, and then more slowly until it reaches the line of zero pressure, and remains at or near this line during the greater part of diastole.

It was necessary to alter considerably the conditions in the schema, utilising the valve to represent the mitral orifice and at the same time obtain a flow through it roughly equal to what was obtained through the aorta with the normal high pressure. The valve had to be kept open for the longest possible time to imitate ventricular diastole, and resistance had to be removed to correspond with the lessened resistance in the ventricle.

By removing the resistance of glass wool, and the venous head of pressure, and by lengthening the outflow time to 0.6 of a cycle, an output of 56 ounces was obtained with a head of water pressure of 68 c.m. or equal to 50 mm.Hg., roughly one-fifth that used to produce High Pressure output with the schema as previously described.

It was not found possible to take satisfactory tracings but the results of output have been tabulated.

In SERIES O the effect of Mitral Stenosis on inflow to the ventricle is seen. Reduction of the mitral area to one-quarter of its size only diminished the flow from 56 to 50 ounces, i.e. a reduction of 75% in the inlet only reduced the flow by 11%. Shortening of ventricular diastole, which would be produced by any icrease of rate of the heart, made a much greater difference, Thus, with diastole reduced from 0.6 to 0.5 of a cycle, the inflow fell to 39 ounces, a reduction from normal of 17 ounces, or 30%. This point is worthy of note, in that the rapid rate which is associated with the onset of Auricular fibrillation, which is not uncommon in this condition, will make considerable difference to the efficiency of the organ from the point of view of this fact alone.

From SERIES P it can be concluded that increase of pressure from 68 c.m. to 80 c.m. was sufficient to overcome the obstruction of Stenosis of one-quarter the normal area, giving an inflow of 57 ounces compared with

the normal non-stenosis inflow of 56 ounces. This increase in the force by 17% was sufficient to overcome the resistance offered by a stenosis which reduced the area of the valve by 75%. It could not, however, do this if the diastole were shortened, such as would be produced by increase of heart rate. If diastole were shortened to be one-half the cycle, then the pressure had to be increased to 105 cm.H_2O., an increase in force of 54%. This reduction in the length of diastole corresponds to an increase in heart rate of only from 66 to 82.5 per minute, or 24%.

SERIES Q was performed without even the resistance offered by the arterial tube: the flow was taken direct from the main pipe, and it can be seen that a comparatively small increase in force was sufficient to overcome the effect of the lesion, but when diastole was shortened the force had to be increased.

The conclusions that may be drawn from the experiments in Mitral Stenosis are :-

(1) Narrowing of the mitral orifice to one-quarter of its size only reduces the inflow to the ventricle from 56 to 50 ounces, or from 68 to 60 ounces, depending on the degree of resistance. That is, reduction in the size of the orifice by 75% only reduces the inflow to the ventricle by 11 or 12%.

(2) An increase of pressure of 17% was sufficient to overcome the effect of reduction in size of the mitral

orifice of 75%.

(3) Increased heart rate with reduction in the length of ventricular diastole increased the effect of mitral stenosis on the flow. An increase in rate of 24% called for an increase in force of 54%.

(4) Considerable increase in heart rate, such as occurs in paroxysmal tachycardia, auricular flutter, or auricular fibrillation, would emphasise very materially the effects of a stenotic lesion simply by the great reduction in the length of ventricular diastole.

MITRAL STENOSIS.

SERIES O.

To show the effect of Mitral Stenosis on inflow to the ventricle, the only resistance used being that of the arterial tubing at the level of the schema.
The maximum flow period (ventricular diastole (V.D.)) available with the valve, was 0.6 of a cycle:

Pressure : 68 c.m.H_2O. or 50 mm.Hg.

Normal. Rate : 66. Inlet - 1/2" diameter.			Mitral Stenosis. Inlet - 1/4" diameter.		
No.	V.D.	O.	No.	V.D.	O.
458	0.6	56	459	0.6	50
			461	0.5	39

SERIES P.

To determine the increase of pressure necessary to overcome Mitral Stenosis of the same degree and with the same resistance as in SERIES O:

Mitral Stenosis - 1/4" diameter.
Rate : 66.

	No.	V.D.	Pressure	O.
Full Opening of 1/2".	458	0.6	68cm.H_2O.	56
Mitral Stenosis 1/4".	462	0.6	78cm.H_2O.	54
" " "	463	0.6	85cmH_2O.	59
" " "	464	0.6	80cm.H_2O.	57
" " "	465	0.5	80 cm.H_2O.	46
" " "	466	0.5	98 cm.H_2O.	53
"				

SERIES Q.

A series to demonstrate the effect of Mitral Stenosis without even the resistance offered by the arterial tubes, outflow being measured from the main outlet pipe:

Rate - 66.

	No.	V.D.	Pressure.	O.
Normal.	471	0.6	76 cm.H_2O.	68
Mitral Stenosis 1/4"	472	0.6	76 cm.H_2O.	60
" " "	468	0.6	105 cm.H_2O.	80
Mitral Stenosis 1/4"	469	0.5	105 cm.H_2O.	64
" " "	473	0.5	76 cm.H_2O.	54.

COMBINED LESIONS.

SERIES R shows the effect of aortic lesions singly and combined, at low and high pressures. Aortic stenosis reduces output 3 to 12%, Aortic Incompetence 52 to 64%, and a combined lesion 60 to 67%. Aortic stenosis therefore adds comparatively little load to the heart compared with Aortic incompetence.

Further, increase of pressure by 80% in aortic stenosis increases output 47% above normal, but with aortic incompetence it is 19% short of compensation, and with the combined lesion 33% short of compensation.

SERIES S contrasts the effect of Aortic Incompetence and Mitral Incompetence on output, the leak being led outthrough a 1/4" diameter tube. Aortic Incompetence reduces output by 64 to 56%, while with the same size of leak Mitral Incompetence reduces output by 49 to 42%, or if led out against small resistance, by 31 to 27%. The effect of increased pressure is also contrasted. Increase of 80% compensates Mitral Incompetence, while Aortic Incompetence is still 18% short of being compensated.

It has to be noted that 1/4" diameter is a smaller proportion of the mitral valve than of the aortic valve.

SERIES T shows the reduction in output which occurs when Mitral Incompetence is present along with Aortic Stenosis and

The double aortic lesion reduces output by 69 to 66%, but when combined with Mitral Incompetence the output is reduced by 85 to 80%.

Increase of pressure by 80% leaves the double aortic lesion still 39% short of compensation, but when combined with Mitral Incompetence, 64% below normal output at low pressure.

It is not possible with the schema to combine Mitral Stenosis with any of the other valve lesions.

The general conclusions which may be drawn from these experiments are confirmatory of those obtained from the pressure series:

(1) Aortic Stenosis is relatively benign and uses up little reserve, probably not more than one-tenth of a unit.

(2) Aortic Incompetence is the more serious of the three and probably uses about one unit.

(3) Mitral Incompetence is intermediate and probably uses up three-quarters of one unit.

(4) An increased head of pressure readily compensates Aortic Stenosis, with more difficulty Mitral Incompetence, and it does not compensate Aortic Incompetence even when increased by 80%.

(5) Aortic Stenosis adds a slight extra load when present with Aortic Incompetence.

(6) Mitral Incompetence adds a serious load when combined with aortic lesions - the combined lesions probably using up about one and one half units of reserve.

SERIES R.

To contrast the effect on output of Aortic Stenosis, Aortic Incompetence, and combined Aortic Stenosis and Incompetence, and also the effect of increased pressure:

L.P. 154 mm.Hg. H.P. 280 mm.Hg. S. 0.3

Rate - 68

	No.	O.	L.O.	B.P.	No.	O.	L.O.	B.P.
N.	436 (A - 1/2")	36		69/43	430	60		94/55
AS.	434 (AS.1/4")	35	-	63/29	432	53		84/51
AI.	437 (AIL.3/16")	13	26	50/22	431	29	34	74/32
A.S. A.I.	435 (AS.1/4") (AIL 3/16")	12	23	46/20	433	24	29	64/30

SERIES S.

To contrast the effects on output of Aortic Incompetence and Mitral Incompetence, and also the effect of pressure :

L.P. 154 mm.Hg. H.P. 280 mm.Hg. S. 0.3

Rate 68

	No.	O.	L.O.	B.P.	No.	O.	L.O.	B.P.
N.	392 (A. 1/2")	39	-	67/42.	389	73	-	98/55
A.I.	393 (AL.1/4")	14	28	48/18.	391	32	29	78/38
M.I.	395[x] (ML.1/4")	20	42	44/30.	396[x]	42	64	71/42
M.I.	394[o] (ML.1/4")	27	26	54/35.	390[o]	53	45	84/50

x In 395 and 396 mitral leak led out against zero pressure.

o In 394 and 390 mitral leak led out against 13 mm.Hg. press.

SERIES T.

To contrast the effect on output of Aortic Stenosis and Incompetence, and Aortic Stenosis and Incompetence combined with Mitral Incompetence, and also the effect of increased pressure :

L.P. 154 mm.Hg. H.P. 280 mm.Hg. Systole 0.3.

Rate - 69.

	No.		O.	B.P.	No.	O.	B.P.
N.	410.	(A. 1/2")	39	68/44	411.	70.	104/56
AS.	409	(AS.1/4")	36	62/40	405	52	80/50
AS. AI.	435	(AS.1/4") AL.3/16"	12	ALO. 23.46/20	433	24	ALO. 29.64/30
AS.	408.	(AS.1/4") AL.3/16" ML.3/16"	6	21.40/14.	MLO. 32.407.	14	26 MLO. 40. 52/22

ANALYSIS OF CLINICAL CASES.

CLASSIFICATION.

The cases included in this analysis have been a consecutive series admitted to Professor Monro's Wards in the Western Infirmary during ten years from 1st October 1913/30th September, 1923. They have been cases admitted on account of symptoms of failing circulation as defined in the earlier part of this communication.

Cases of renal disease have only been included when symptoms were present which appeared definitely to point to heart failure.

Cases of Aneurysm without involvement of the aortic valve have been excluded, while it has also been thought advisable to leave out of account 13 cases of Pericarditis and 4 cases of Paroxysmal Tachycardia for the main purposes of the analysis.

The classification which is used has been adopted with a view to excluding, as far as possible, debatable points in diagnosis. It is further to be noted that many cases of valvular disease are not included in the series, because the patients were suffering from some other condition and made no complaint of symptoms which might be ascribed to the valvular lesion. The criteria which have been used in the classification may be briefly noted :

Mitral Stenosis.

Mitral Stenosis was diagnosed in the presence

of a presystolic murmur at the apex, with or without an early diastolic murmur. It was not considered advantageous to attempt to distinguish those cases which also showed the presence of a systolic murmur from others in which this sign was absent, so that under the heading of Mitral Stenosis there are included cases which would, by some, be designated double mitral disease.

In the presence of Auricular Fibrillation, the presence of an early diastolic murmur at the apex in the absence of Aortic Incompetence was considered to be diagnostic. Only in an occasional case was the diagnosis made from the presence of an abrupt first sound at the apex in the absence of a presystolic or diastolic murmur, and then only if it occurred in a person before middle life and with a clinical history of rheumatic fever.

AORTIC INCOMPETENCE.

Aortic incompetence was diagnosed in the presence of a diastolic murmur at the aortic cartilage or down the left side of the sternum, and in most cases with confirmation from a large pulse pressure, particularly with the diastolic pressure less than normal and the presence of pulsatile arteries. No attempt was made to distinguish those cases in which the lesion was combined with that of Aortic Stenosis, as it was recognised that a systolic murmur in the aortic area was not in itself sufficient evidence to warrant such a diagnosis.

AORTIC INCOMPETENCE AND MITRAL STENOSIS.

The combination of Aortic Incompetence with disease of the mitral valve was only made in cases where it could be reasonably inferred that there was Mitral Stenosis. In no case was the presence of a simple systolic murmur at the apex taken as evidence of mitral disease. It will thus be evident that some cases may be included as pure Aortic Incompetence in which there was mitral disease, the only lesion being Mitral Incompetence. These will be referred to later.

MITRAL INCOMPETENCE.

Mitral Incompetence presented considerable difficulty and it was sometimes impossible to determine whether the patient was suffering from primary disease of the mitral valve, or from secondary and relative incompetence due to myocardial change. In patients with a systolic murmur at the apex, those with a definite rheumatic history, or those under 40 years in which no other cause for myocardial damage could be discovered, were included as cases of primary Mitral Incompetence. Cases of patients over 40 years, unless there was a very definite rheumatic history, were supposed to be cases of secondary Mitral Incompetence following myocardial change. Blood diseases were not included.

AORTIC STENOSIS.

The criteria taken as evidence of this condition

were a rough systolic murmur at the aortic cartilage, and a thrill in the same area and some cardiac enlargement. In the series there was no such case which was not present in combination with Aortic Incompetence, therefore Aortic Stenosis does not appear as a separate class in the clinical analysis.

MYOCARDIAL DEGENERATION.

Under this heading were included all those patients suffering from symptoms of failing heart which could not be placed in any of the groups of valvular disease.

ETIOLOGY.

In defining the etiological factors which have been present in the series, the ultimate object of the enquiry has been kept in view, viz: to determine from the gross lesion present and the knowledge of its cause, the probability of damage to cardiac muscle.

Cases have been included as rheumatic when there was a definite history of rheumatic fever, chorea, or definite growing pains in childhood, but muscular rheumatism and chronic rheumatism have been excluded or are specially mentioned. In the vast majority of cases noted as syphilitic, the Wassermann reaction was positive and other cases have only been included when there was a definite history of the infection, or other facts which clearly pointed

to its presence.

Cases of nephritis are those which were associated with high blood pressure and they are grouped along with the cases of arterio-sclerosis. Some of those cases also had a history of chronic bronchitis. Scarlet fever, influenza, diphtheria, frequent sore throats, and bronchitis, have been included under the general term of 'other infections'.

Alcohol is mentioned separately because it was in certain cases the only ascertainable cause and the amount consumed in each case was definitely excessive.

Under the heading of 'no history' are included 43, or 13.4%, of the cases. This may be taken to mean that there was no factor in the history which could reasonably be supposed to explain the heart disease. Of these, 25 or 10.62%, occurred in valve cases, and 17 or 21% in myocardial cases. The majority of the valve cases were probably rheumatic, as they occurred mostly in those groups with a high rheumatic history, but it was considered advisable to keep them in this group as there was no definite evidence. Absence of history was more common in female than in male cases.

The etiological factors will be fully considered when dealing separately with the different valve lesions and myocardial degeneration, but some general observations may be made at this point: Details will be found in tables in appendix A. 4 to 12;

Of the total of 320 cases of failing heart, 242 were valve cases and 78 myocardial cases. Of the 320 cases, 148 or 46.20% were definitely rheumatic, this being by far the largest group in the series.

In the 242 valve cases (table A 4) it was found to be the cause in 145 or 60%, accounting for 68.5% of the female cases and 51.2% of the male cases. A rheumatic history was more common in male patients who suffered from Mitral Stenosis than in female patients(75% and 68.6% respectively in pure stenosis, and 100% and 72.7% respectively in combined lesions).

Definite syphilitic infection was noted in 63 cases, or 19.7% of all cases, and in 53 or 22% of the valve cases. It reached its highest percentage in pure Aortic Incompetence when evidence of syphilis was obtained in 70.75% of the male cases and 66.66% of the female cases. The Wassermann reaction was positive in 51 of the 63 cases; in the other 12 cases there was definite clinical evidence; in no case was the Wassermann reaction negative.
It is to be noted that evidence of syphilis was not obtained in any case of pure mitral stenosis or of combined mitral stenosis and aortic incompetence in either sex.

Renal disease, or arterio-sclerosis,occupies the third place in etiological factors. It was present in 28 cases or 8.75% of the total, but when the 78 myocardial cases are considered alone it was found to account for 27 or 34.6% of these, and in the group it is the commonest

etiological factor. Other etiological factors occurred too seldom for the figures to be of any special value and it is to be noted, as already mentioned, that in 43 cases or 13.4% no history of any value was ascertainable.

Average ages. (Table A.10.)

The average age of all patients in the series was 39.76, the male patients being slightly older in the average than the females (42.09 and 37.19 years respectively). It has to be recognised that in general patients under 12 years of age are not admitted to Hospital.

As might be expected, patients suffering from valvular disease come under observation at an earlier average age than those suffering from myocardial disease, and likewise those valve cases which might be reckoned as rheumatic came under observation at an earlier age than those which are syphilitic, in both sex groups, the difference in the female group being about 20 years, and in the male group about 14 years.

Deaths. (Table A.11)

The death rate noted when in Hospital of the patients in this series was 19%. It is much greater among the male patients than among the female patients - 24% in the former and 14% in the latter, but in this connection it is to be noted (see table A.10) that the age at which male patients came under observation was usually five years

more than that noted for females.

The highest death rate occurred in syphilitic aortic incompetence in males, 33% of the patients dying, and the lowest death rate in any group of moderate size occurred in female mitral stenosis with a death rate of 11%.

It is to be noted that fibrillation was present in 27% of the cases in the series, and in 28% of those who died. This might be erroneously interpreted as indicating that auricular fibrillation does not specially embarrass the heart. It must, however, be taken along with the fact that in fibrillation the extra load can be in great part lifted by treatment with digitalis, while in other cases the load is not so amenable to drug treatment.

It may further be observed that while syphilis was present in 20% of all cases, it was noted in 29% of those who died, while rheumatism was noted in 46% of all cases but only in 43% of those who died.

Those suffering from other infections, or giving no history, constituted the balance, viz: 28%.

RELATION OF VALVE LESIONS TO:

1. Increased load.
2. Muscle damage.
3. Cardiac failure.

It is my intention to discuss only the four well recognised valve lesions of the left side of the heart, viz: Aortic stenosis, aortic incompetence, mitral incompetence and mitral stenosis.

Following the view that cardiac failure is due to a disproportion between heart load and muscle reserve, I shall, taking each in turn, treat -

1. Of the increased load that the lesion adds to the work of the heart, as shown by the results of the experiments with the schema:

2. Of the relation which the lesion bears to muscle damage in lessening reserve, as indicated by the commoner etiological factors found in the clinical analysis:

3. Of the relation to cardiac failure in view of these two considerations.

AORTIC STENOSIS.

Relation to load.

As has been shown in the experiments a degree of stenosis diminishing the aortic orifice to one quarter of its area produces a comparatively slight reduction in the output. Even if we argue from those cases which have shown a larger reduction than some of the others, we find that the diminution in output is about 16%, whereas the reduction in the aortic opening is 75%.

With the reduction in the output the blood pressure is also reduced, this being slightly more marked in the systolic than in the diastolic pressure.

The load added by aortic stenosis even of a severe degree must be very little and the deduction from the experiments is that it probably amounts to about one-tenth of one unit of reserve.

Relation to muscle damage.

Aortic Stenosis is not a common solitary lesion of the heart. It did not occur once in this series of cases. If the aortic valve is damaged by rheumatism, or by syphilis, aortic incompetence is practically always present with or without additional stenosis.

Primary pure Aortic Stenosis is due in most cases to atheroma of the senile type. As this seldom occurs before the sixth decade of life its significance is masked by the degeneration of heart muscle, which may

occur as a result of age, or as a result of co-existing atheroma of the coronary arteries producing fibrous or other degeneration of the muscle.

Relation to Cardiac Failure.

If the view is accepted that cardiac failure is due to a disproportion between the state of the heart muscle and the load which it has to carry (and this will be accepted throughout the discussion) then cardiac failure in aortic stenosis must be related very much more to the damage done to the muscle, than to the load of the aortic stenosis which is comparatively slight and could easily be met from cardiac reserve.

AORTIC INCOMPETENCE.

Relation to load.

Results of experiments show that Aortic Incompetence is the most serious of the valvular lesions considered mechanically. Whether judged from the diminution in output which it causes, or the difficulty in overcoming its effect, a leak of moderate severity probably doubles the resting work of the heart - that is uses up about one unit of reserve.

It therefore follows that if the heart muscle is healthy it should be able to overcome the lesion. The lesion thus would not by itself produce cardiac failure in a healthy heart, though normal reserve would be limited and symptoms would be produced more readily than before the onset of the valve defect.

Relation to muscle damage.

In the clinical series there were 74 cases of pure aortic incompetence and 23 cases in which it was combined with mitral stenosis. Of the pure cases 70% were syphilitic and 24% rheumatic. Of the combined cases no one was syphilitic and 84% were rheumatic. Of the total of 97 cases, 54% were syphilitic and 39% rheumatic. Included in the cases called pure are those in which a systolic murmur was present at the apex, but without other evidence of mitral disease. It is probable that some of these were really examples of combined lesions, in which case the [illegible] to syphilis

would be higher than that stated. My figures lead me to support the view of Cowan[12] that "the co-existence of mitral and aortic disease strongly suggests a rheumatic origin, while pure aortic disease suggests syphilis", and to confirm the percentage of syphilitic cases as found by Ritchie.[13] I feel induced to stress this point, as Russell Wells[14] has stated that "syphilis is not of much importance as a cause of aortic regurgitation in the case of men between the ages of 18 and 41". The statement was based on 307 cases of aortic incompetence in 10,000 cases of doubtful heart disease examined with a view to determining their fitness for army service. Wassermann test was not employed. The age grouping has a bearing on the statement and to meet the discrepancy I have divided the cases in the series into two groups - those within this age period and those above it (Table A.9). Of the pure aortic cases 50% within the lower age group were syphilitic, while taking all cases, pure and combined, 25% were syphilitic. I cannot therefore subscribe to the view enunicated by Wells, which appears to me to be misleading.

Syphilis.

It is rare to find syphilitic disease of the heart confined to the aortic valve. In most cases the valve lesion is associated with syphilitic aortitis, and as has been shown by Van der Stricht and Wingate Todd[15] there is often direct damage to the muscle fibres. The

coronary arteries are also frequently involved either with endarteritis in some part, or by pressure occlusion at the origin in the aorta. It may therefore be accepted that in Aortic Incompetence due to syphilis, damage to the heart muscle is a frequent, if not a constant accompaniment of the valve lesion.

Rheumatism.

In rheumatism the infection is seldom confined to the valve, but the heart muscle is also damaged in the primary infection though it may apparently recover in some cases more than the comparatively evascular structure of the valves.

It is to be noted, as has been pointed out by Grosse,(16) that blood vessels are present in healthy valves in early life, but that they gradually disappear during early adult life, and are seldom, if ever, found after the age of 30. Further, he points out that the aortic valve loses its blood vessels before the mitral and that the last cusp of the mitral to become evascular is the aortic cusp. If, as is believed, the rheumatic infection is blood borne then there will be a greater liability for involvement of the mitral than of the aortic valve, so that one may reason that if the aortic valve is involved in the rheumatic process the infection has probably occurred at an early age, and that it has likely been severe: Also that it is likely to be associated with disease of the mitral valve and also with

damage to the heart muscle. Rheumatism affecting the aortic valve therefore, in the majority of cases implies that there is other damage to the heart in muscle or in valve.

Atheroma.

Apart from the type which is sometimes called syphilitic atheroma, and which has already been considered, atheroma is a disease of later life. If the aortic valves suffer it is because the aorta itself has suffered and so there is special liability to involvement of the coronary arteries. There is thus reason to expect along with this type damaged cardiac muscle, both from defective blood supply and from the effects of age, while the lesion itself is likely from the nature of the case to be progressive. Long continued strain has been accepted as a cause of aortic incompetence, but unless it acts through the production of arterio-sclerotic processes, its action in producing aortic incompetence is difficult to understand if the valve has previously been healthy.

It will thus be seen that no matter which of the known etiological factors has been at work in producing Aortic Incompetence, there is a special liability to involvement of cardiac muscle.

It would further appear from the death rate (Table A.11) that syphilitic aortic disease is more serious than that due to other causes.

Relation to Cardiac Failure.

Both of the factors which limit cardiac reserve, viz: increased load and diseased heart muscle, operate very definitely in this condition. The relation to cardiac failure is therefore very close.

If one compares the mechanical effect of mitral incompetence with that of aortic incompetence, it is seen that while aortic incompetence is the more serious, the difference is not so great as that between the two conditions clinically. While it may double the work of the heart there would still be sufficient reserve for the heart to carry on, if it is accepted that the heart has a reserve of four times the force expended when acting quietly.

One must therefore infer that the special liability of the heart muscle to serious damage in this condition is the predominant factor in using up cardiac reserve and causing heart failure, though it is recognised that the lesion is itself a serious handicap. The relative incompetence of the mitral valve which may arise from dilatation of the left ventricle or mitral orifice adds a further load in this condition, and increases its gravity.

MITRAL INCOMPETENCE.

Relation to load.

Mitral Incompetence is found to add a considerable load to the heart, being comparable to Aortic Incompetence in this connection. Adopting the same method of estimation, it may be said that it reduces the reserve by three-fourths of one unit. The degree of the mechanical defect arises mainly from the fact that the ventricle has to force blood into the aorta against a head of pressure which is practically non-existent in the left auricle, so that regurgitation is relatively easy.

Relation to muscle damage.

Of the 33 cases of primary mitral incompetence in the series, 85% gave a history of rheumatic fever. It has been suggested that this lesion results from an old acute rheumatic endocarditis, while mitral stenosis is due to a sclerosing endocarditis.(17) This is not brought out in the series, as repeated attacks (up to 6) of acute rheumatic fever, have occurred in both groups. The only point which might suggest milder attacks in mitral stenosis is that in female cases a rheumatic history was obtained in 90% of the incompetence cases, and only in 69% of the stenosis cases.

It is generally recognised that after an attack of acute rheumatism there may be evidence of mitral incompetence, and without any further attack mitral stenosis is found to be present some years later, particularly if the

attack has been severe or has occurred in early life.

If mitral incompetence of rheumatic origin is present as a pure condition in middle life, it may be inferred that the attack has been mild and consequently there is mitral stenosis. Such a conclusion would not, however, be justified in early life or even after an attack of rheumatic fever.

Relation to Cardiac Failure.

Primary mitral incompetence bears a less close relationship to cardiac failure than does mitral stenosis or aortic incompetence, and it is much more difficult to assess the probability of muscle damage. Enlargement of the heart is possibly the only sure guide.

If the muscle is damaged then the load added by the lesion is sufficiently severe to be an important factor in limiting reserve and so encouraging cardiac failure. On the other hand it would appear that mitral incompetence may be present for many years with little evidence of muscle damage, in which case the cardiac reserve is able to deal with the defect.

There still remains the large group of cases in which mitral incompetence is present, but in which there has been no primary disease of the valve - cases of secondary or relative mitral incompetence. As far as possible these cases, if they presented signs of failing heart (and only such have been included in the figures),

have been placed under the heading of myocardial conditions. In them the question of load does not specifically arise, as the regurgitation has arisen as a result of failure on the part of the muscle to perform its function efficiently, and the gravity of the case depends on the disease which has damaged the heart muscle, particularly as the majority of these cases arise in the later years of life.
One must,however, assume that the mitral leak adds to the load of the already damaged heart, and that consequently it must further limit the already diminished cardiac reserve.

MITRAL STENOSIS.

Relation to load.

Judged from the result of the experiments, mitral stenosis is comparable with aortic stenosis rather than with lesions producing regurgitation. With reduction in the area of the orifice by 75% the flow was only reduced 11 or 12%, while increase in pressure by 17% was sufficient to overcome the defect. The fact that the load falls on a comparatively weak chamber like the left auricle has hardly a bearing on the result, as the defect is measured in relation to the normal weak force supplied.

The effect of increased rate must, however, have a bearing on the result, as it will operate with any exercise on the part of the individual. Increase of rate of 24% called for 54% increase of force to overcome the defect. An increase in rate of 100% or over, such as occurs in auricular fibrillation or paroxysmal tachycardia, must therefore add a very serious load to the heart in this condition. It might conceivably use up, according to previous methods of calculation, two units of reserve.

Relation to muscle damage.

There is a general concensus of opinion that the prime cause producing mitral stenosis is acute rheumatism, though it is not possible to get a definite clinical history of this in all cases. This is in great

part due no doubt to the comparative mildness of the attack of rheumatism in the child, although it is possible that other conditions allied to rheumatism produce similar effects.

The association of mitral stenosis with nephritis has also long been recognised, but that it accounts for any appreciable proportion of the cases is difficult to realise. In the statistics appended to this paper a definite history of rheumatism, or of conditions recognised as of the same nature, was obtained in 71% of the cases, and there is good grounds for supposing that at least the majority of the 18% in which no history was obtained were due to the same cause. The exact nature of the pathological process has been referred to by various writers, notably Poynton,[18] MacKenzie[19] and Lewis. There is good ground for believing that a certain degree of severity or of prolongation of the infection is necessary before the lesion that leads to mitral stenosis is produced, and as rheumatism does not affect the valve tissue solely, but also affects the heart muscle, though there it may not lead to the same degree of scarring, one can infer than when mitral stenosis has developed the infection has been sufficiently severe, or sufficiently prolonged to make almost certain the presence of heart muscle damage.

Relation to Cardiac Failure.

In making a comparison between mitral stenosis and the other valve lesions, account must be taken of the

very serious addition that is made to the load in this condition by acceleration of rate, though the effect of the stenosis with a normal proportion of systole and diastole is comparatively slight and compares with aortic stenosis. It then takes its place with, if not in advance of, aortic incompetence.

The relation to muscle damage, as has been noted, is very close and appears to be the predominant factor in determining heart failure. This is so far confirmed by clinical observation, in that cases of mitral stenosis go to autopsy in which signs of failure have been of comparatively short duration, and in which there is no reason to believe that the state of the valve has undergone any appreciable alteration during the same period. It is also confirmed by the long duration of symptoms in many cases before signs of actual failure are manifest.

A third factor often determines the onset of cardiac failure, viz: auricular fibrillation. It was present in 35% of the cases of pure mitral stenosis. The load added by the acceleration of rate alone, apart from other effects of this condition which are dealt with later, is often sufficient to use up the remaining reserve. This is best seen in cases which, in spite of some muscle damage and extra load, present no special symptoms till the onset of fibrillation with dramatic suddenness determines cardiac failure, usually accompanied by dropsy.

In this, as in the other valve lesions, therefore

the mechanical defect in the valve is not the predominant factor in producing heart failure. Failure must depend chiefly on the damage to the muscle which accompanies the valve lesion. Even in those cases where extra load is added by auricular fibrillation, it is the state of the heart muscle which is the determining factor in the production of this disordered rhythm.

Valvular lesions - General conclusions.

In reviewing the various points which have been raised in connection with these four left sided valve lesions, one finds a dual relationship of the lesion to cardiac failure.

There is the extra load forced on the heart by mechanical defect, the view which for so long has held a prominent place in medical writings. As a result of the experiments it has been shown that it is very easy to overcome the effects of narrowing of a valve orifice, even when cross section is reduced to as much as 1/4th of the normal and that the lesion in itself only produces comparatively slight diminution in output.

In the case of lesions producing regurgitation, one finds that though they produce a considerably greater load than those produced by stenosis, still both can be overcome by mechanical readjustment which is within the limits of the reserve power of the heart, using up about one-fourth of the estimated reserve at most.

This grouping according to mechanical effects does not indicate the clinical severity of the lesion, and one is forced to conclude that there must be some other factor which determines the gravity of aortic incompetence and mitral stenosis, as compared with mitral incompetence and aortic stenosis. This factor is found in the damage to heart muscle which is so closely associated with these two conditions.

A further point should be noted, though as it did not form part of this investigation it has not been previously mentioned, viz: late infection of the already damaged valves. This was noted in 9 out of 21 postmortems in this series, or 43%, but as the records were not scrutinised for this specific purpose the percentage may have been even more. So far it confirms the figures recorded by Cowan and Rennie,[20] who found acute reinfection in 37 out of 81cases which went to postmortem, or 45%. In these cases there can be no doubt that the toxic effect on the heart from the infection is the factor which determines the fatal issue in this group.

RELATION OF HIGH BLOOD PRESSURE TO:

1. Increased load.
2. Muscle damage.
3. Cardiac failure.

In the non-valvular cases of failing heart, high blood pressure is the most common associated condition, being present in 27 out of 78 cases, or 35%, the next in order of frequency being syphilis, which was present in 10 or 13% of the cases.

Relation to load.

There are no definite data in the experiments on which to estimate in figures the load added by a measured resistance. A rough idea may be obtained from certain results which were noted when removing resistance to imitate mitral stenosis.

With the low pressure used, 68 cm.H_2O.(or 50 mm.H the outflow with Systole 0.6, using all the resistance, was 14 ounces; on removing the glass wool it rose to 41 ounces; on removing the venous resistance of 5¼ inches(13 mm.Hg.) it rose to 56 ounces; and on removing the arterial tubing and taking outflow from the aortic tube, it rose to 64 ounces. Thus the total peripheral resistance reduced output from 64 ounces to 14 ounces, or by 78%.

It required a head of pressure of 154 mm.Hg. to obtain an output of 64 ounces with the full resistance on another date; that is, the resistance as used in the schema required three times the force to produce an output which could be obtained

if there were no resistance.

All one is prepared to say is that increasing the peripheral resistance adds considerably to the load on the heart.

High blood pressure, if due to a cause operating only at the periphery of the circulation, would in part compensate for its own load, the increase in the aortic pressure thus produced helping to provide an increased blood supply to the heart muscle. It is a common clinical experience for one to meet with patients having a blood pressure of 200 mm.Hg. or over, who make no definite complaint, the ventricle having hypertrophied to meet the strain within the limits of the patient's ordinary daily requirements, and Starling[4] has shown with his heart-lung preparation, that the heart can put out as much blood against 208 mm.Hg. as against 44 mm.Hg., the coronary arteries taking more blood in proportion to the blood pressure.

Relation to muscle damage.

Ultimately in these cases the heart muscle weakens, the hypertrophy of the muscle gives way to fibrous change, the cardiac reserve is reduced, and the heart fails to keep up an efficient circulation. But, in most cases the arterio-sclerosis which accompanies the high blood pressure is present in the coronary arteries, and tends to counteract the effect of the high blood pressure in providing a sufficient blood supply to the heart muscle.

Apparently, however, in some cases the heart muscle is damaged directly by the toxin (microbic or metabolic) which originally produced the high blood pressure.

Relation to Cardiac Failure.

It is true that in cases of high blood pressure death may occur from causes other than cardiac failure. Cerebral haemorrhage, or uraemia in cardiac-renal cases may kill the patient, but apart from these or other incidental causes, the end of the case is by way of cardiac failure.

As in the case of the valvular lesions, a disproportion between load and muscle efficiency determines the failure. The load added may be considerable, but the condition of the muscle determines the onset of serious symptoms, and in view of the fact that very high pressures are compatible with a fair degree of health, one must again come to the conclusion that where heart failure is the cause of death it is because of the damage of the muscle, and not simply from the high blood pressure.

RELATION OF AURICULAR FIBRILLATION TO:

1. Increased load.
2. Muscle damage.
3. Cardiac failure.

Of the various cardiac irregularities that have been described, one is outstanding in its relationship to cardiac failure, viz: auricular fibrillation. The actual lesion which determines in any individual case the onset of this disorder has not yet been definitely ascertained. That circus movement[21] in the auricle takes the place of the normal sino-auricular stimulation is generally accepted. MacKenzie,[22] as a result of recent investigations at St. Andrews, believes that auricular fibrillation is the natural effect of abolition of function of the sino-auricular node, whereas most believe that the onset of the circus movement leads to abolition of the s-a node function. In the present state of knowledge it would appear that the latter view has stronger grounds for its position. It is known that it is possible to cause a cessation of circus movement by the action of quinidine sulphate with a return to sinus rhythm. If the primary factor had been the abolition of sinus function, it is difficult to see how destruction of the effect of that abolition would lead to restoration of a function which was lost from another cause. In neither case, however, does the theory explain exactly how auricular fibrillation is ultimately brought about. In many cases the patient can point to a date when presumably this rhythm started.

In the series of 320 cases it was noted 86

times or in 27% of the cases - 50 times in the 242 valve cases and 36 times in the 78 myocardial cases. It was thus relatively more common in myocardial cases of failing heart (46%) than in valvular cases (21%). It was more common in female cases (33%) than in male cases(21%). The highest incidence in any one group occurred in female myocardial cases, the figure being 58%, while next in order of frequency came male myocardial cases 38%, male mitral stenosis 38%, female mitral stenosis 34%, male mitral incompetence 33%. It was present in 28% of those cases which died, the figure being highest in female myocardial cases, in which it was present in 80%.

Of the 50 cases associated with valvular disease, 80% were rheumatic. Of the 36 myocardial cases, 31% were associated with high blood pressure, while only 3% were rheumatic and 8% syphilitic.

Relation to load.

Auricular fibrillation adds load to the heart by its action on the ventricle, very much more than by the absence of the contraction of the auricle itself. The a-v node is bombarded with impulses numbering from 400 to 500 per minute; the majority of these, usually about two-thirds, are blocked in their passage to the ventricle, which responds to the remainder. As the auricular stimuli, coming from the irregular circus movement in the auricle, are irregular in themselves as shown by electro-cardiograph, so the ventricle responds to irregular stimuli in irregular

fashion. There is no constant relationship between the length of a pause and the strength of the succeeding contraction. The ventricle is stimulated probably in all cases before it is completely filled, and in many cases when it must be almost empty. Many of the pulsations communicated to the aorta do not reach the wrist. The acceleration on the heart, many of the beats being quite useless for increasing the blood pressure, must add considerable load to that organ, while at the same time by shortening diastole they prevent efficient filling of the coronary arteries.

The virtual paralysis of the auricle, which is present owing to the absence of any co-ordinate contraction of the auricular muscle fibres, withdraws from the circulation a muscular effort which at first sight might be supposed to have a definite influence in causing heart failure, but on considering the action of the auricle one knows : (1) that it only acts during one-eighth of the cardiac cycle; (2) that it raises blood pressure in the auricle to a maximum of only 30 mm.Hg.; and (3) that its chief action is probably in enlarging the cavity of the left ventricle.

The loss of the co-ordinate contraction of the auricle is distinctly less than the loss of its regular stimulation of the ventricle when controlled by sinus rhythm. When one recollects that the auricle may be paralysed owing to auricular fibrillation, over a period

of many years, without the onset of any symptoms, presumably because the ventricle is healthy, one must recognise that its contraction is not essential for carrying on the circulation, though it may be a very great convenience. In health, the auricle acts as a reservoir, and in its contraction the blood in the appendages is placed in a position of being readily swept into the ventricle. When fibrillation is present there is no such emptying of the appendages, and the blood tends to flow through the cavity of the auricle by virtue of any pressure that may be in the venous blood, whether of the general or pulmonary circulation.

Its effect in the case of mitral stenosis is even more striking. Simple acceleration is sufficient to add considerably to the load of that lesion, but when to this is added the other disabilities dependent on fibrillation it can readily be seen how it frequently uses up the remaining reserve.

Another indication of the load which auricular fibrillation adds to the work of the heart is obtained from the results of treatment. The limitation of the load on the ventricle by the action of digitalis often restores patients to a certain degree of comfort, while the effect of quinidine sulphate in abolishing the rhythm is even more striking.

In these cases it must be admitted that the load of the fibrillation has used up an amount of reserve,

which when restored to the patient has permitted of a moderate amount of exertion.

Similar deductions may be drawn from cases of paroxysmal auricular fibrillation. In one case (not included in the series) the patient, a medical man, was unable for sustained exertion during the attack, but within a few hours of its cessation was able to continue all his work, or engage in a round of golf, though the attacks had occurred on an average of once a week for over a year.

Relation to muscle damage.

As one looks at the etiology of auricular fibrillation it is evident that the vast majority of cases occur in two main conditions, viz: in mitral stenosis and in myocardial disease, and as has already been shown there is usually in mitral stenosis definite rheumatic damage to muscle, and in myocardial disease it is most commonly associated with high blood pressure.

It may be accepted generally that auricular fibrillation is evidence in itself of damage to the auricular muscle, and usually therefore to the ventricular muscle as well.

Paroxysmal cases, and cases where the rhythm is present without symptoms, are both rare and this rarity goes to support the general contention that auricular fibrillation is in itself evidence of damage to heart muscle.

Relation to cardiac failure.

The dual relationship of auricular

fibrillation to cardiac failure through increased load and muscle damage is thus particularly close.

In the great majority of cases the heart muscle is already damaged and reserve is still further lessened by the load of a valve lesion or of high blood pressure. The onset of the new rhythm, with additional load on the heart, is usually sufficient to determine not only a failing heart, but definite cardiac failure with dropsy.

In valvular lesions generally the progress towards failure may be comparatively slow; when this condition is superimposed the advent of cardiac failure is almost dramatic in its appearance.

In so far as cases have been observed in which fibrillation has been present over a period of some years, and, though untreated, the patient has been able to continue at light work, it must be inferred that fibrillation itself is not sufficient to use up all the reserve of a healthy heart, but that damaged ventricular muscle is necessary for the production of cardiac failure.

GENERAL CONCLUSIONS.

Valvular lesions add load to the work of the heart.

The load added by the lesion per se does not alone determine its position in the scale of clinical severity.

Lesions producing regurgitation add a greater mechanical load to the heart than those producing obstruction.

Within the limits available with the schema, namely a stenosis producing 75% reduction in the valve orifice, or incompetence through an opening equal to the valve orifice, it is mechanically possible for a healthy heart with a reserve equal to three or four times the resting power, to overcome the lesion and to leave some reserve for limited exertion.

In all the cases of cardiac failure that have come under review, there is reason to believe that the valvular lesion was accompanied by damage to cardiac muscle.

High blood pressure produces failure in the same way as valvular lesions, viz: because of the association of added load and damaged muscle.

Auricular fibrillation adds considerable load to the heart, but when the ventricle is healthy it can overcome the load. When auricular fibrillation produces cardiac failure it is always associated with damaged muscle.

Damage to muscle can alone produce cardiac failure, as in certain of the myocardial lesions in which there was no evidence of added load from valvular lesions, high blood pressure or disordered rhythm.

Healthy muscle, through its reserve, can deal with the load added to the work of the organ by valvular lesions, high blood pressure and auricular fibrillation, and failure is manifested only if the heart muscle is diseased and the amount of reserve is diminished from this cause.

Valvular lesions, high blood pressure , and auricular fibrillation add sufficient load to be considered as important factors in limiting reserve in the cases of cardiac failure in which they occur.

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APPENDIX A.

Tables incorporating the results of the analysis of 320 cases of Failing Heart with reference to symptomatology and etiology.

TABLE A.1.

SYMPTOMS IN 320 CASES OF FAILING HEART.

Females.

	Cases.	B.	Pn.	P.	Ex.	Co.	H.	G.	F.	Emb.	D.	A.F.
Mitral Stenosis.	80	73 91%	28 35%	38 47%	27 34%	15 19%	11 14%	6 7%	1 1%	8 10%	31 39%	27 34%
Mitral Stenosis and Aortic Incompetence.	11	9 82%	4 36%	2 18%	3 27%	3 27%	1 9%	0	1 9%	0	3 27%	1
Aortic Incompetence. (Rh.3. Syph.6.)	9	8 89%	5 56%	3 33%	2 22%	3 33%	1 11%	0	1 11%	0	5 56%	0
Mitral Incompetence.	21	18 86%	4 19%	12 57%	5 24%	4 19%	1 5%	0	0	0	9 43%	4 19%
	121	108 89%	41 34%	55 45%	37 30%	25 21%	14 12%	6 5%	3 2%	8 7%	48 40%	32 26%
Myocardial Cases.	31	27 87%	8 26%	12 39%	8 26%	3 10%	1 3%	4 13%	1 3%	2 6%	14 45%	18 58%
	152	135 89%	49 32%	67 44%	45 30%	28 18%	15 10%	10 7%	4 3%	10 7%	62 40%	50 33%

Males.

		Cases.	B.	Pn.	P.	Ex.	Co.	H.	G.	F.	Emb.	D.	A.F.
Mitral Stenosis.		32	28 87%	17 53%	9 28%	4 12%	9 28%	7 22%	2 6%	2 6%	1 3%	10 31%	12 37%
Mitral Stenosis and Aortic Incompetence.		12	10 83%	7 58%	4 33%	1 8%	7 58%	3 25%	1 8%	0	1 8%	6 50%	2 17%
Aortic Incompetence.	Rh.	19	17 89%	10 52%	4 21%	7 39%	3 16%	1 5%	3 16%	2 10%	1 5%	7 37%	0
	Syph.	46	42 91%	28 61%	11 24%	11 24%	10 22%	4 9%	2 4%	3 6%	0	14 30%	0
Mitral Incompetence.		12	12 100%	3 25%	7 58%	3 25%	1 8%	3 25%	1 8%	0	1 8%	4 33%	4 33%
		121	109 90%	65 54%	35 29%	26 21%	30 25%	18 15%	9 7%	7 5%	4 3%	41 34%	18 15%
Myocardial Cases.		47	35 74%	16 34%	5 11%	7 14%	10 21%	4 9%	4 9%	3 6%	0	22 47%	18 38%
		168	144 86%	81 48%	40 24%	33 20%	40 24%	22 13%	13 8%	10 6%	4 2%	63 37%	36 21%

B. Breathlessness.
Pn. Pain.
P. Palpitation.
Ex. Exhaustion.
Co. Cough.
H. Haemoptysis.
G. Giddiness.
F. Faintness.
Emb. Embolism.
D. Dropsy.
A.F. Auricular Fibrillation.

TABLE A. 2. SYMPTOMS IN 320 CASES OF FAILING HEART. (CONTINUED)

TOTALS:		Cases.	B.	Pn.	P.	Ex.	Co.	H.	G.	F.	Emb.	D.	A.F.
Valves.	Females.	121	108	41	55	37	25	14	6	3	8	48	32
	Males.	121	109	65	35	26	30	18	9	7	4	41	18
		242	217 90%	106 44%	90 37%	63 26%	55 23%	32 13%	15 6%	10 4%	12 5%	89 37%	50 21%
Myocardial Cases.	F.	31	27	8	12	8	3	1	4	1	2	14	18
	M.	47	35	16	5	7	10	4	4	3	0	22	18
		78	62 80%	24 31%	17 22%	15 19%	13 17%	5 6%	8 10.	4 5%	2 3%	36 46%	36 46%
GRAND TOTAL:		320	279 87%	130 41%	107 33%	78 24%	68 21%	37 12%	23 7%	14 4%	14 4%	125 39%	86 27%

B. Breathlessness. Ex. Exhaustion. G. Giddiness. D. Dropsy.
Pn. Pain. Co. Cough.. F. Faintness. A.F. Auricular Fibrillation.
P. Palpitation. H. Haemoptysis. Emb. Embolism.

MYOCARDIAL SYMPTOMS V. VALVULAR SYMPTOMS.

TABLE A 3.

Symptoms more common in Myocardial Cases.			Symptoms more common in Valve Cases.		
Auricular Fib.	by	25%	Palpitation	by	15%
Dropsy	by	9%	Pain	by	13%
Giddiness	by	4%	Breathlessness	by	10%
Faintness	by	1%	Exhaustion	by	7%
			Haemoptysis	by	7%
			Cough	by	6%
			Embolism	by	2%

E T I O L O G Y.

VALVE CASES.

TABLE A 4.

		Rh.F.	M.R.	S.	Other Inf.	N.H.
Females:						
Mitral Stenosis.	80.	55 69%	5 6%	0	6 8%	14 17%
Mitral Stenosis with Aortic Inc.	11.	8 73%	0	0	1 9%	2 18%
Aortic Incomp.	9	1 11%	0	6 67%	0	2 22%
Mitral Incomp.	21	19 90%	1 5%	0	1 5%	0
	121	83 68%	6 5%	6 5%	8 7%	18 15%
Males:						
Mitral Stenosis.	32	24 75%	0	0	2 6%	6 19%
Mitral Sten.with Aortic Incomp.	12	12 100%	0	0	0	0
Aortic Incomp.	65	17 26%	0	46 71%	0	2 3%
Mitral Incomp.	12	9 75%	0	1 8%	2 17%	0
	121.	62 51%	0	47 39%	4 3%	8 7%
VALVES:	F. 121	83	6	6	8	18
	M. 121	62	0	47	4	8
TOTAL:	242	145 60%	6 2%	53 22%	12 5%	26 11%

Rh.F. Rheumatic Fever. S. Syphilis. N.H. No history
M.R. Muscular [illegible]

E T I O L O G Y.

MYOCARDIAL CASES.

TABLE A 5.

	FEMALES.	MALES.	TOTAL
CASES:	31	47	
Rheumatic Fever.	2 (7%)	1(2%)	3 (4%)
Muscular Rheumatism.	0	0	
Syphilis.	1 (3%)	9(19%)	10 (13%)
Nephritis or Arterio-Sclerosis.	10 (32%)	17(36%)	27 (35%)
Bronchitis.	4 (13%)	4(9%)	8 (10%)
Goitre.	2 (7%)	0	2 (2%)
Other Infections.	6 (19%)	2(4%)	8 (10%)
Alcohol.	0	3(6%)	3 (4%)
No history.	6 (19%)	11(24%)	17 (22%)
	31	47	78

E T I O L O G Y.

GRAND TOTAL IN THE 320 CASES.

TABLE A 6.

	Valve.	Myocardial.	Total.	Per cent.
Rheumatic Fever.	145	3	148	46
Muscular Rheumatism.	6	0	6	2
Syphilis.	53	10	63	20
Nephritis or Arterio-Sclerosis.	0	27	27	9
Bronchitis.	0	8	8	3
Goitre.	0	2	2	1
Other Infections.	12	8	20	6
Alcohol.	0	3	3	1
No history.	26	17	43	13
TOTAL:	242	78	320.	

E T I O L O G Y

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VALVE AND MYOCARDIAL CASES CONTRASTED.

TABLE A 7.

	Valve Cases.	Myocardial Cases.
Rheumatism.	145 (60%)	3 (4%)
Syphilis.	53 (22%)	10 (13%)
High Blood Pressure. (?1)	0	27 (35%)
Others.	19 (7%)	21 (26%)
No history.	25 (11%)	17 (22%)
TOTAL :	242.	78.

E T I O L O G Y.

ALL CASES with MITRAL STENOSIS - pure and combined.

TABLE A 8.

	Cases.	Av. Age.	Rh.F.	M.R.	S.	Other Inf.	N.H.
Females:	91	32.1	63 69%	5 5%	0	7 8%	16 18%
Males :	44	30.66	36 82%	0	0	2 5%	6 14%
TOTAL :	135	31.71.	99 73%	5 4%	0	9 7%	22 16%

ALL CASES with AORTIC INCOMPETENCE - pure and combined.

	Cases.	Av. Age.	Rh.F.	M.R.	S.	Other Inf.	N.H.
Females:	20	40.55	9 45%	0	6 30%	1 5%	4 20%
Males :	77	42.77	29 38%	0	46 60%	0	2 3%
TOTAL :	97	42.31	38 39%	0	52 54%	1 1%	6 6%

E T I O L O G Y

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AORTIC INCOMPETENCE.

TABLE A 9.

		Cases.	Rh.	Syph.	Others.
In sex groups.					
Pure.	Males:	65	17	46	2
	Females:	9	1	6	2
		74	18 24%	52 70%	4 6%
Pure and Combined.	Males:	77	29	46	2
	Females:	20	9	6	5
		97	38 39%	52 54%	7 7%
In age groups.					
Pure.					
42 years and over :		56 76%	9 16%	43 77%	4 7%
41 years and under :		18 24%	9 50%	9 50%	0
		74	18	52	4
Pure and Combined.					
42 years and over :		61 63%	14 23%	43 70%	4 7%
41 years and under :		36 37%	24 67%	9 25%	3 8%
		97	38	52	7

AVERAGE AGES.

TABLE A 10.

Females:	Cases.	Years.	Average Age.
Mitral Stenosis.	80	2583	32.29
Mitral Stenosis with Aortic Incompetence.	11	351	31.91
Mitral Incompetence.	21	700	33.3.
Aortic Inc. Non-Syph.	3	143	47.66
	115	3777	32.84
Aortic Inc. Syph.	6	317	52.83 6
TOTAL VALVES :	121	4094	33.83
Myocardial Deg.	31	1559	50.29
TOTAL :	152	5653	37.19
Males:			
Mitral Stenosis.	32	978	30.56
Mitral Stenosis with Aortic Incompetence.	12	371	30.92
Mitral Incompetence.	12	369	30.75
Aortic Incompetence. Rh.	19	761	40.05
	75	2479	33.05
Aortic Incomp. Syph.	46	2161	46.98
TOTAL VALVES :	121	4640	38.35
Myocardial Deg.	47	2432	51.74
TOTAL:	168	7072	42.09
VALVE CASES: TOTAL	242	9734	40.22
MYOCARDIAL " "	78	3991	51.16
GRAND TOTAL :	320	12725	39.76.

DEATHS IN 320 CASES

in case groups.

TABLE A 11.

		Cases.	Deaths.	Average.	A.F.	Rh.	S.	Others
Mitral Stenosis.	F.	80	9 11%	35.11	5 56%	7 78%	0	2 22%
	M.	32	7 22%	36.57	2 29%	7 100%	0	0
		112	16 11%		7 44%	14 87%	0	2 13%
M.S.& A.I.	F.	11	2 18%	23.	0	1 50%	0	1 50%
" "	M.	12	3 25%	32.33	0	3 100%	0	0
		23	5 22%		0	4 80%	0	1 20%
Aortic Incomp.	F.	9	1 11%	49	0	0	1 100%	0
	M.Rh.	19	3 16%	46	0	3 100%	0	0
	M.S.	46	15 33%	45	0	0	15 100%	0
		74	19 26%		0	3 16%	16 84%	0
Mitral Incomp.	F.	21	4 19%	42.25.	1 25%	4 100%	0	0
	M.	12	2 17%	26.	1 50%	1 50%	0	1 50%
		33	6 18%		2 33%	5 83%	0	1 17%
Myocard. Cases.	F.	31	5 16%	57.2	4 80%	0	0	5 100%
	M.	47	10 21%	51.5	4 8%	0	2 20%	8 80%
		78	15 19%		8 53%	0	2 13%	13 87%
GRAND TOTAL :		320	61 19%		17 28%	26 43%	18 29%	17 28%

DEATHS IN 320 CASES

in sex groups.

TABLE A 12.

	Cases.	Deaths.	Av.Age.	A.F.	Rh.	S.	Others.
Females:							
Mitral Stenosis.	80	9 11%	35.11	5 56%	7 78%	0	2 22%
M.S. with A.I.	11	2 18%	23.	0	1 50%	0	1 50%
Aortic Incomp. 6 S. 3 Rh.	9	1 100%	49.	0	0	1 100%	0
Mitral Incomp.	21	4 19%	42.25	1 25%	4 100%	0	0
Myocardial Deg.	31	5 16%	57.	4 80%	0	0	5 100%
	152	21 14%	41.24.	10 48%	12 57%	1 5%	8 38%
Males:							
Mitral Stenosis.	32	7 22%	36.57.	2. 29%	7 100%	0	0
M.S. with A.I.	12	3 25%	32.33	0	3 100%	0	0
A. I. Rh.	19	3 16%	46.	0	3 100%	0	0
" Syph.	46	15 33%	45.	0	0	15 100%	0
Mitral Incomp.	12	2 17%	26	1 50%	1 50%	0	1 50%
Myocard. Deg.	47	10 21%	51.5	4 40%	0	2 20%	8 80%
TOTAL: MALES	168	40 24%	43.32	7 18%	14 35%	17 42%	9 23%
TOTAL :FEMALES:	152	21		10	12	1	8
GRAND TOTAL :	320	61 19%		17	26	18	17 28%

APPENDIX B.

Tables incorporating the results of the analysis of 80 cases of Auricular Fibrillation with reference to symptomatology and etiology.

TABLE B 1.

AURICULAR FIBRILLATION.

S Y M P T O M S.

	Cases.	B.	Pn.	P.	Ex.	Co.	H.	G.	F.	Emb.	D.
FEMALES.											
Mitral Stenosis.	27 34%	27 100%	13 48%	15 56%	9 33%	6 32%	4 15%	2 7%	1 4%	4 15%	16 59%
Mitral Stenosis and Aortic Incompetence.	1 7%	1 100%	0	0	0	0	0	0	0	0	1 100%
Aortic Incompetence.	0										
Mitral Incompetence.	4 19%	4 100%	1 25%	2 50%	1 25%	2 50%	0	0	0	0	3 75%
	32 26%	32 100%	14 44%	17 53%	10 31%	8 25%	4 12%	2 6%	1 3%	4 12%	20 62%
Myocardial Cases.	18 58%	18 100%	5 28%	6 33%	6 33%	2 11%	0	1 5%	0	1 5%	7 39%
	50 33%	50 100%	19 38%	23 46%	16 32%	10 20%	4 8%	3 6%	1 2%	5 10%	27 54%
MALES.											
Mitral Stenosis.	12 37%	12 100%	6 50%	3 25%	3 25%	3 25%	1 8%	1 8%	0	1 8%	4 33%
Mitral Stenosis and Aortic Incompetence.	2 17%	2 100%	1 50%	0	1 50%	2 100%	0	0	0	0	2 100%
Aortic Incompetence.	0										
Mitral Incompetence.	4 33%	4 100%	1 25%	2 50%	1 25%	0	1 25%	1 25%	0	1 25%	1 25%
	18 15%	18 100%	8 44%	5 28%	5 28%	5 28%	2 11%	2 11%	0	2 11%	7 39%
Myocardial Cases.	18 38%	15 83%	5 28%	4 22%	4 22%	6 33%	1 6%	1 6%	0	0	8 44%
	36 21%	33 92%	13 36%	9 25%	9 25%	11 31%	3 8%	3 8%	0	2 6%	15 42%

B. Breathlessness. Ex. Exhaustion. G. Giddiness. D. Dropsy.

Pn. Pain. Co. Cough. F. Faintness.

P. Palpitation. H. Haemoptysis. Emb. Embolism.

TABLE B. 2.

AURICULAR FIBRILLATION.

SYMPTOMS (CONTINUED)

TOTALS:

		Cases.	B.	Pn.	P.	Ex.	Co.	H.	G.	F.	Emb.	D.
VALVES:	Females.	32	32	14	17	10	8	4	2	1	4	20
	Males	18	18	8	5	5	5	2	2	0	2	7
		50	50 100%	22 44%	22 44%	15 30%	13 26%	6 12%	4 8%	1 2%	6 12%	27 54%
MYOCARDIAL CASES.	F.	18	18	5	6	6	2	0	1	0	1	7
	M.	18	15	5	4	4	6	1	1	0	0	8
		36 21%	33 92%	10 28%	10 28%	10 28%	8 22%	1 3%	2 6%	0	1 3%	15 42%
GRAND TOTAL:		86 26%	83 96%	32 37%	32 37%	25 29%	21 24%	7 8%	6 7%	1 1%	7 8%	42 49%

B. Breathlessness.
Pn. Pain.
P. Palpitation.
Ex. Exhaustion.
Co. Cough.
H. Haemoptysis.
G. Giddiness.
F. Faintness.
Emb. Embolism.
D. Dropsy.

E T I O L O G Y

O F

AURICULAR FIBRILLATION.

TABLE B. 3.

VALVE CASES.

Females.	Cases.	Rh.	S.	Other Infections.	No history.
Mitral Stenosis.	27	20	0	4	3
Mitral Stenosis with Aortic Incomp.	1	1	0	0	0
Aortic Incomp.	0				
Mitral Incompetence.	4	4	0	0	0
	32	25 78%	0	4 13%	3 9%
Males.					
Mitral Stenosis.	12	10	0	0	2
Mitral Stenosis with Aortic Incomp.	2	2	0	0	0
Aortic Incomp.	0				
Mitral Incomp.	4	3	0	1	0
	18	15 83%	0	1 6%	2 11%
TOTAL:	50	40 80%	0	5 10%	5 10%

E T I O L O G Y

O F

AURICULAR FIBRILLATION.

TABLE B.4

MYOCARDIAL CASES.

	Females.	Males	Total.
Cases.	18	18	36
Average Age.	54	53	
Rheumatic Fever.	1 6%	0	1 3%
Syphilis.	0	3 17%	3 8%
Nephritis or Arterio-Sclerosis.	5 28%	6 38%	11 31%
Goitre	2 11%	0	2 6%
Other Infections	5 28%	3 17%	8 22%
Alcohol.	0	2 11%	2 6%
No history.	5 28%	4 22%	9 25%
	18	18	36.

E T I O L O G Y

O F

AURICULAR FIBRILLATION.

GRAND TOTAL:

TABLE B. 5.

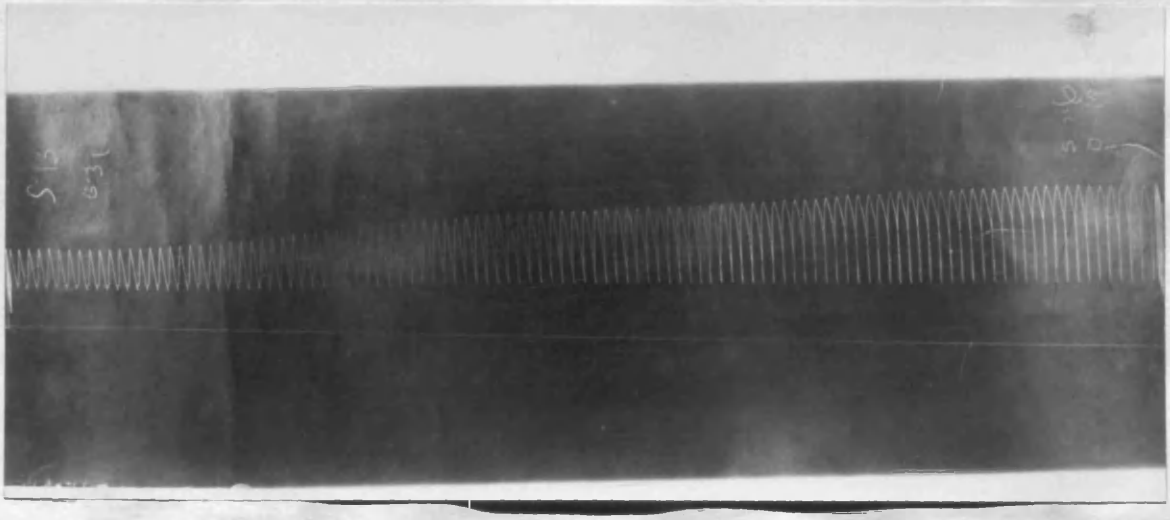
	Valves.	Myocardial.	Total.
Rheumatism	40	1	41 48%
Syphilis	0	3	3 3%
Nephritis or Arterio-Sclerosis.	0	11	11 13%
Other Infections	5	12	17 22%
No History.	5	9	14 16%
	50	36	86

APPENDIX C.

Kymograph records and radial tracings

to illustrate experiments used in the thesis.

PRELIMINARY TRACINGS.



AORTIC STENOSIS.

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Series A. AORTIC STENOSIS.

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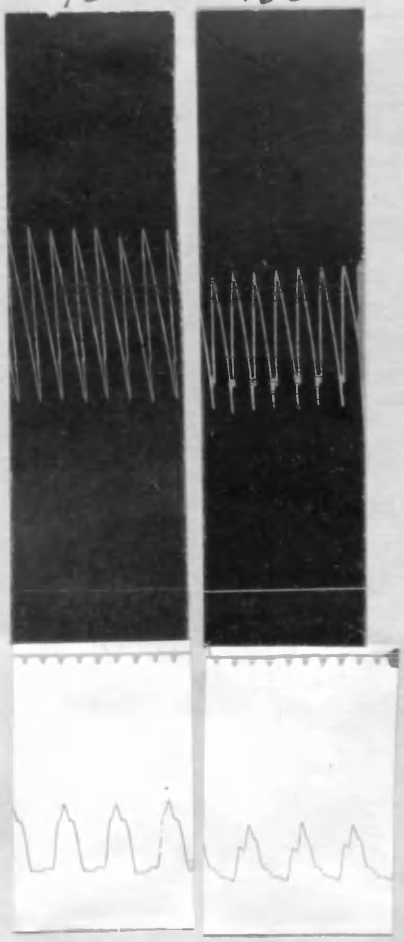

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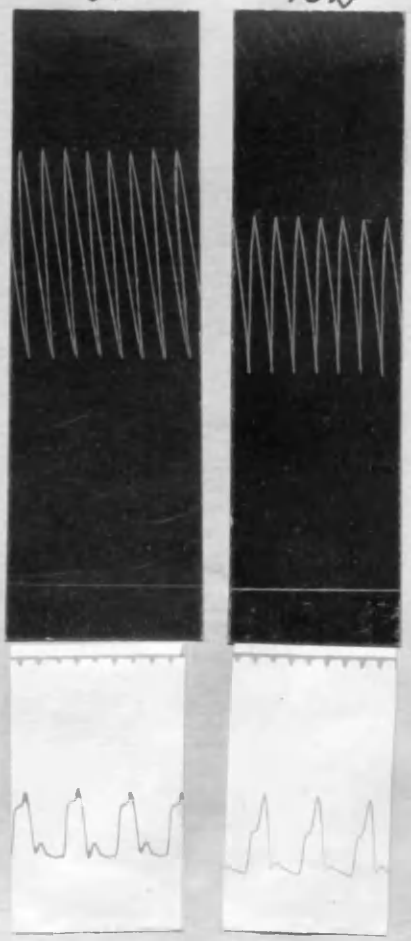

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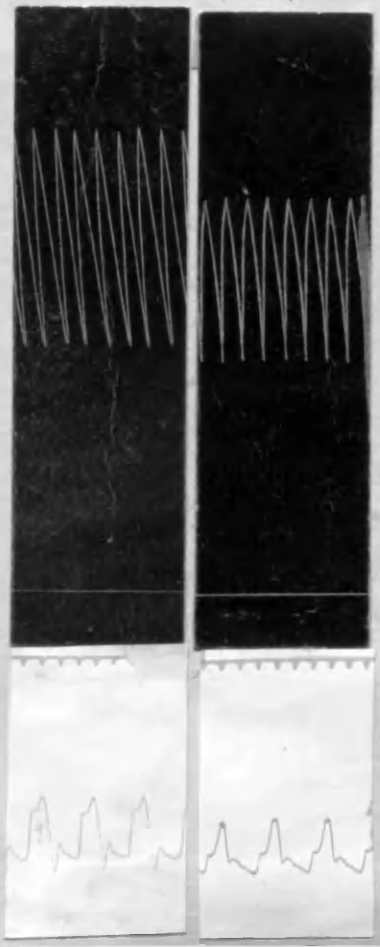

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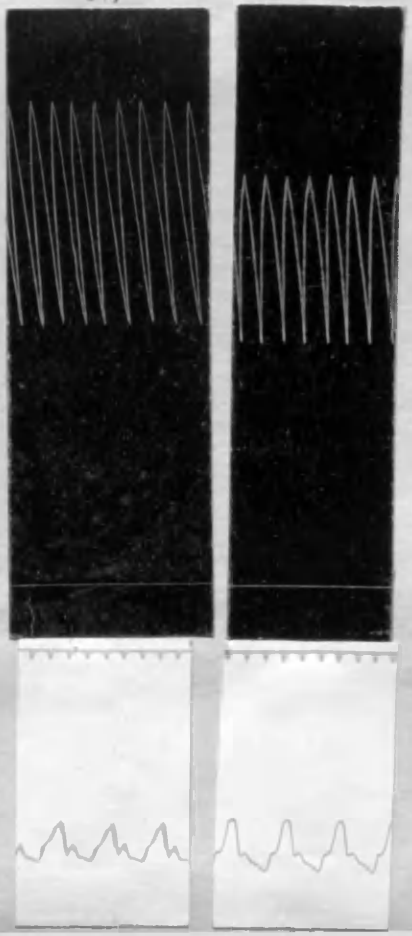

Series A.

AORTIC STENOSIS

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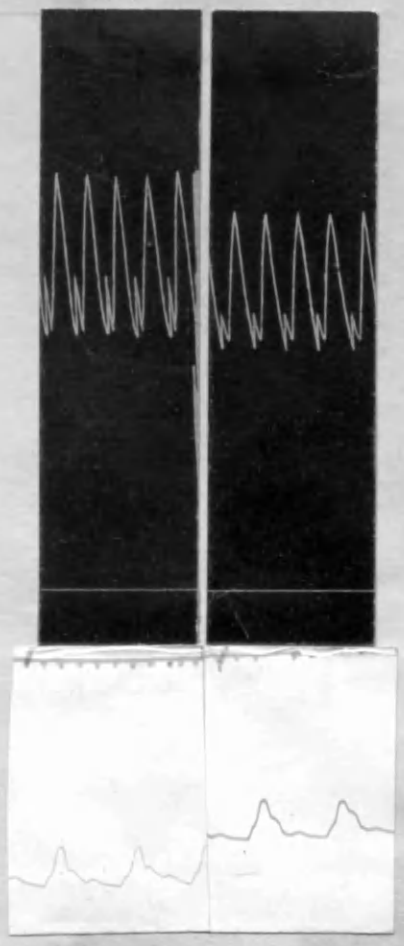

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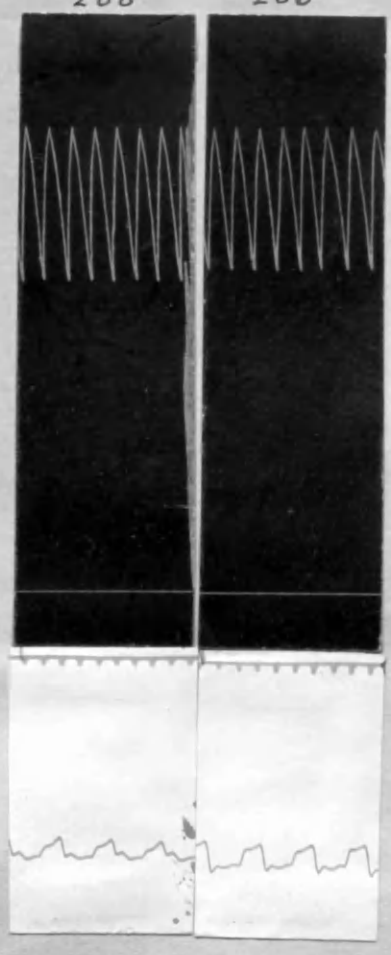

Series B. AORTIC STENOSIS.

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Series B. AORTIC STENOSIS

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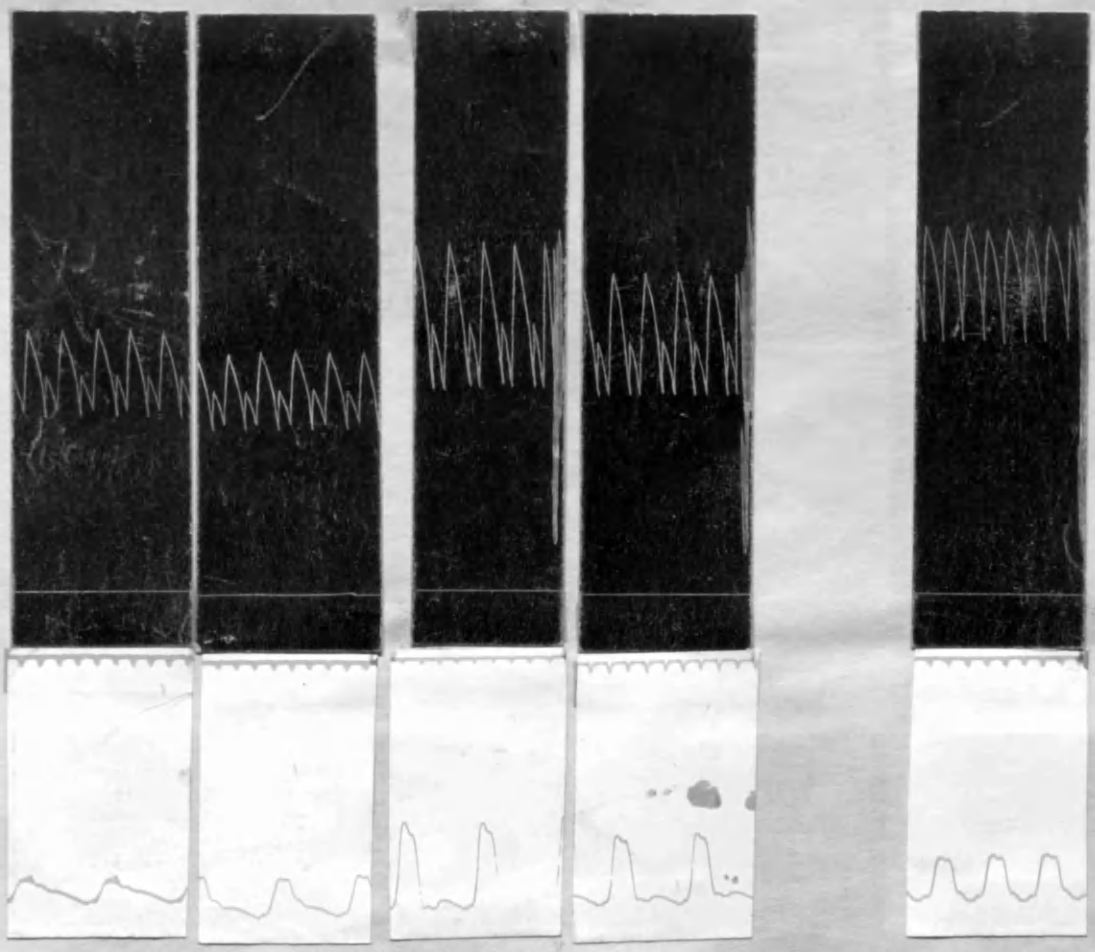
Series C. AORTIC STENOSIS.



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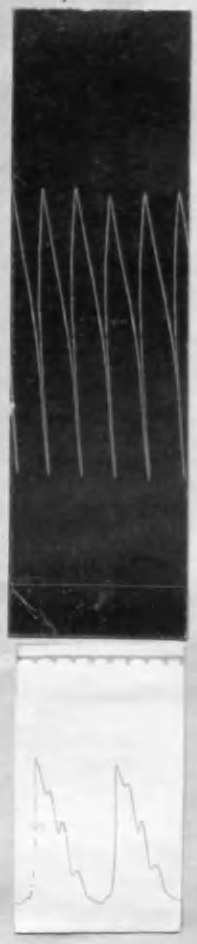

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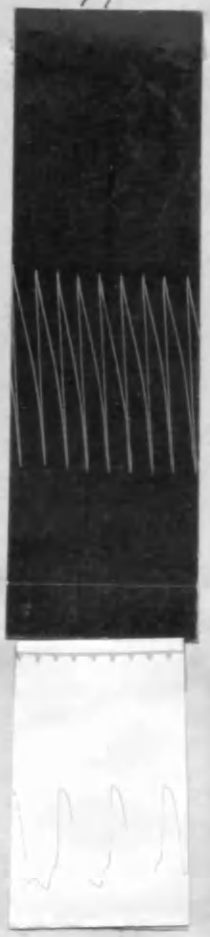

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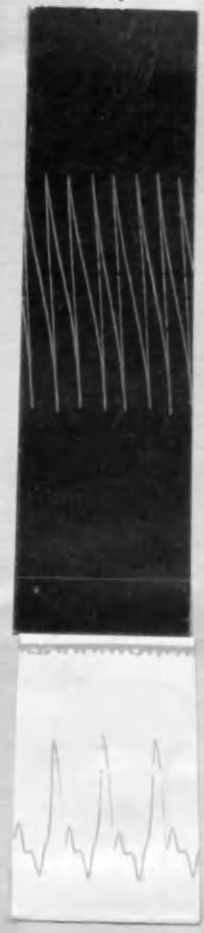

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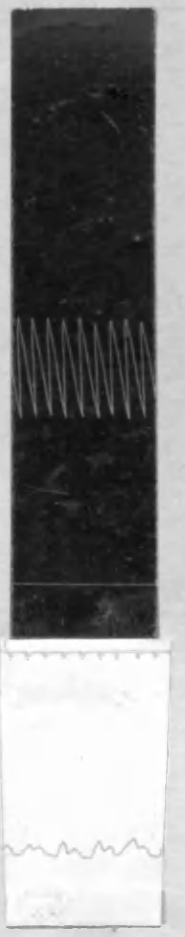

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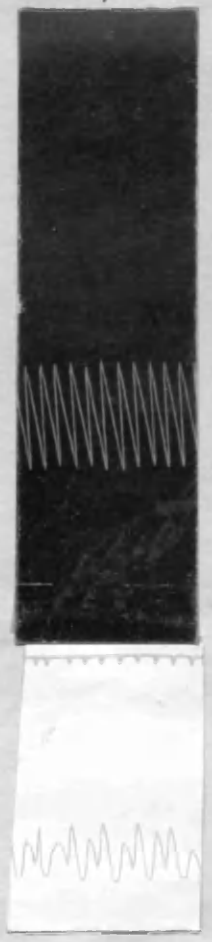

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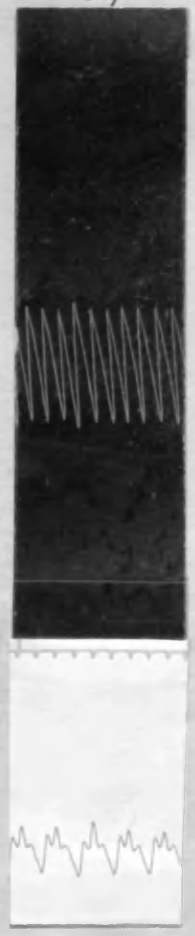

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Series E. AORTIC INCOMPETENCE.

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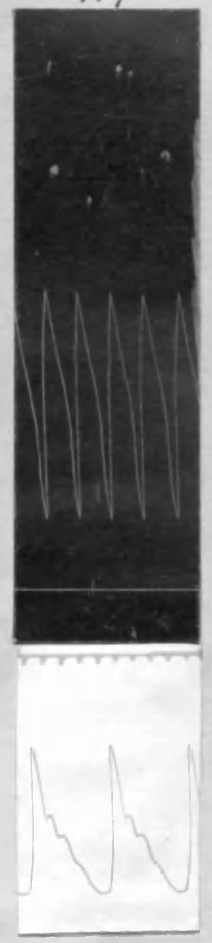

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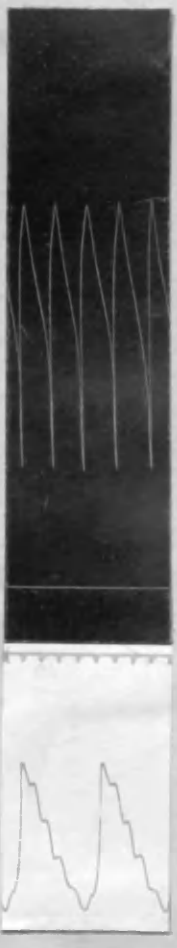

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Series E. AORTIC INCOMPETENCE.

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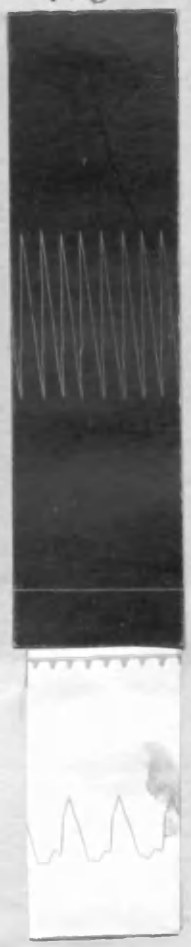

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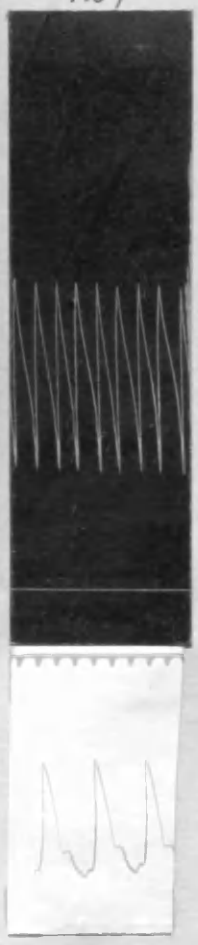

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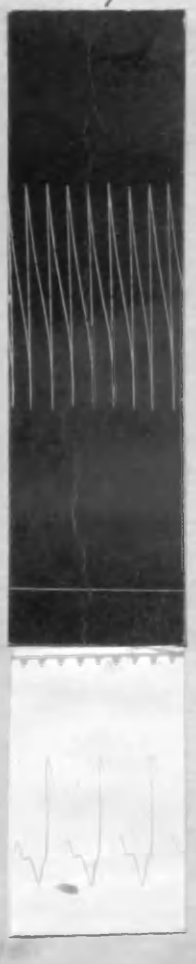

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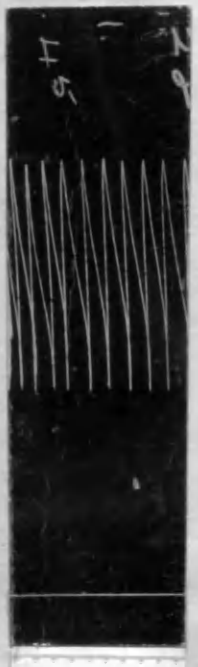

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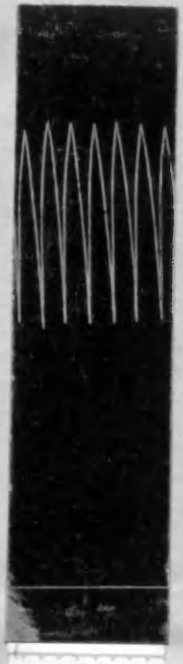

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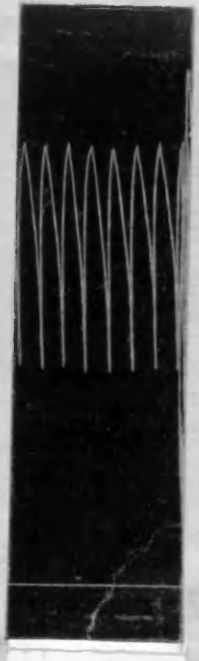

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Series E. AORTIC INCOMPETENCE.

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Series F. AORTIC INCOMPETENCE.

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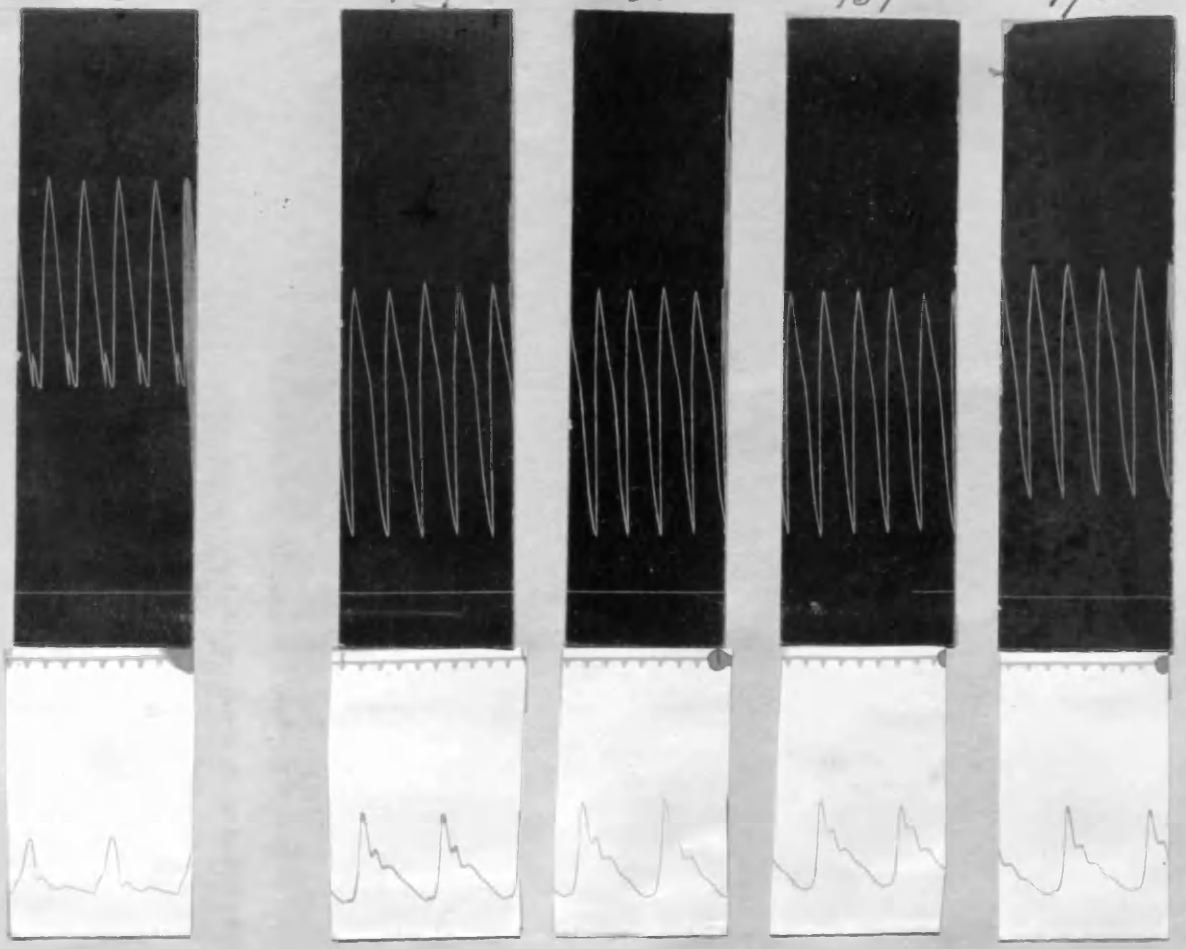

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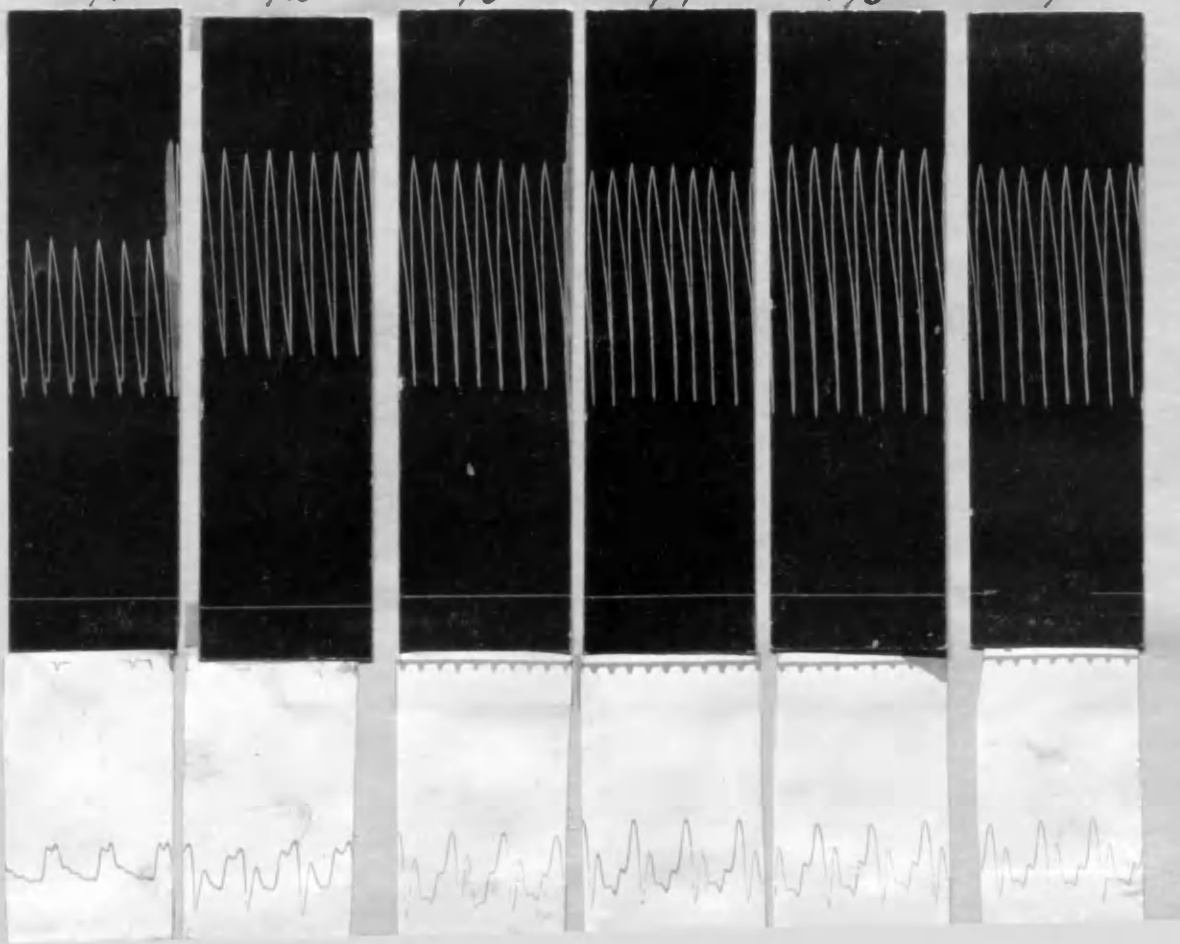

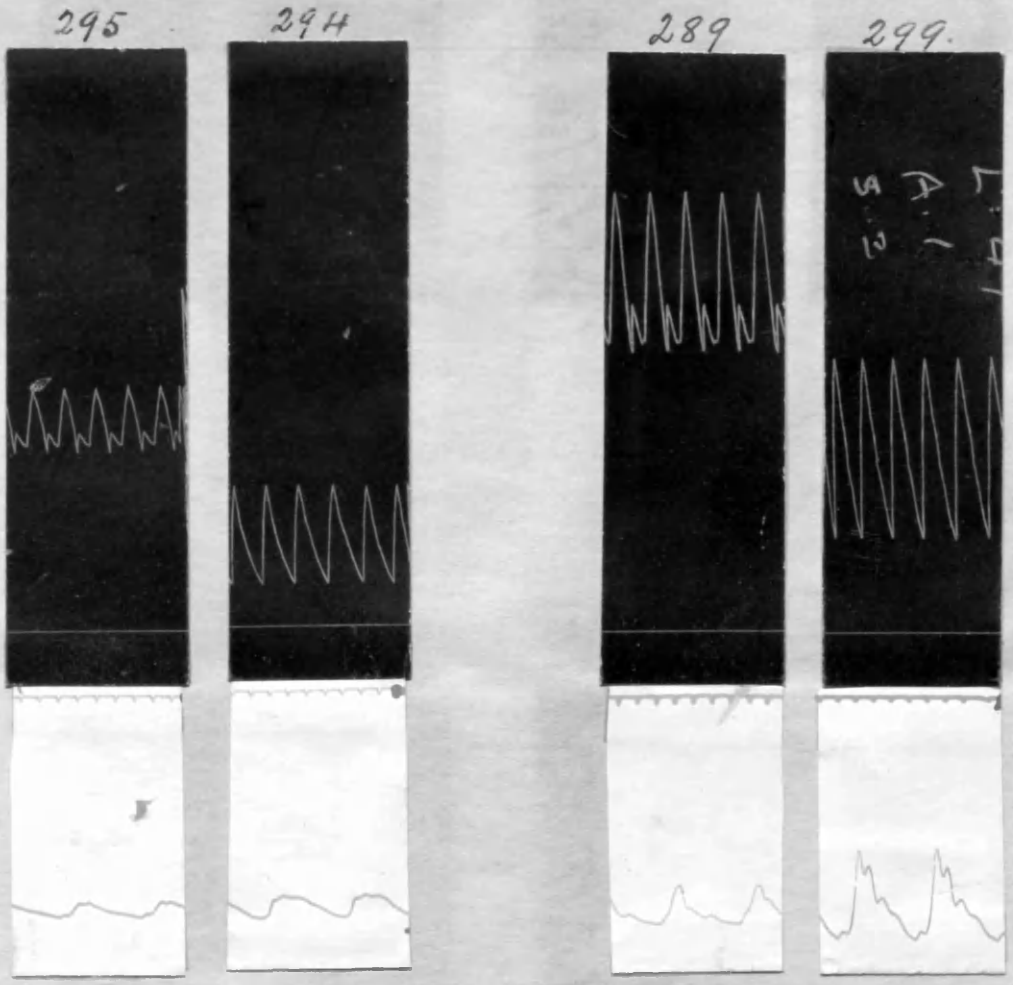
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Series J. MITRAL INCOMPETENCE.

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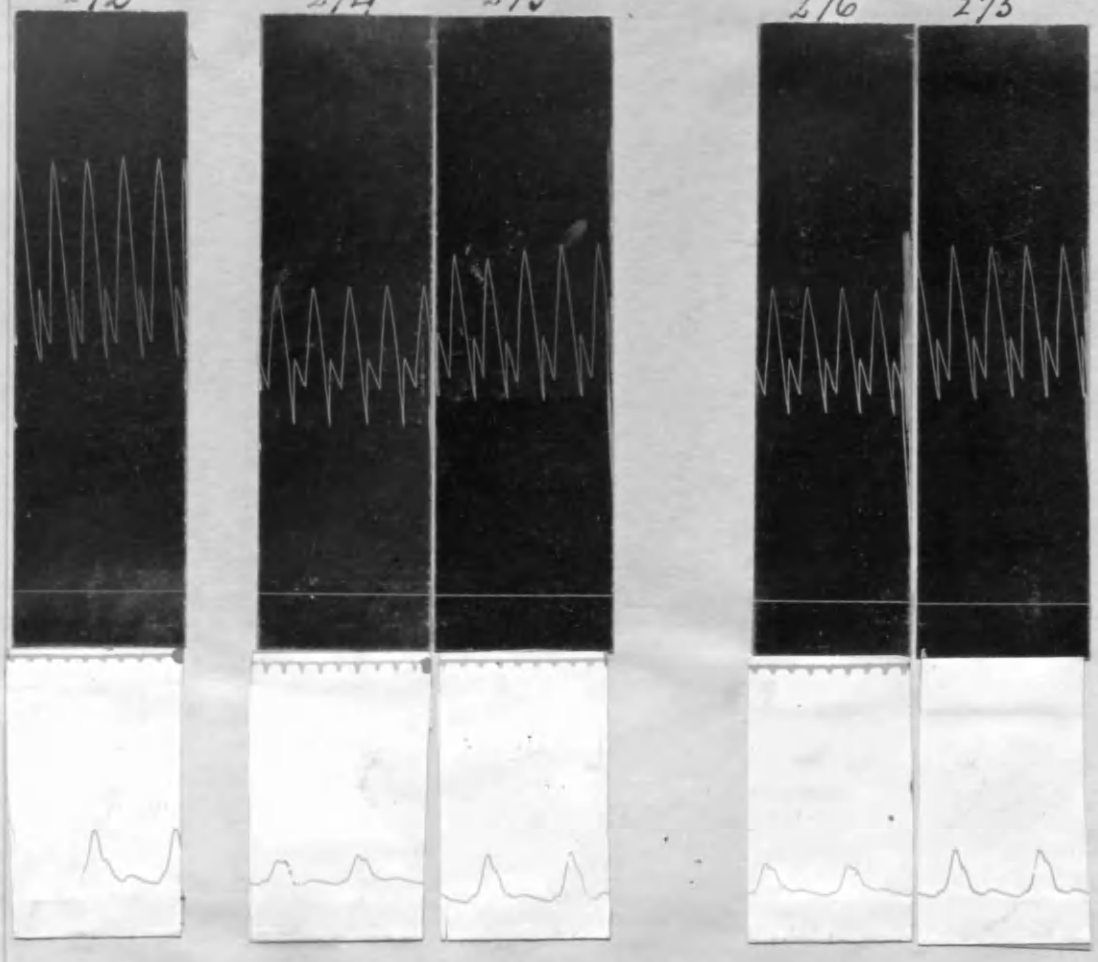

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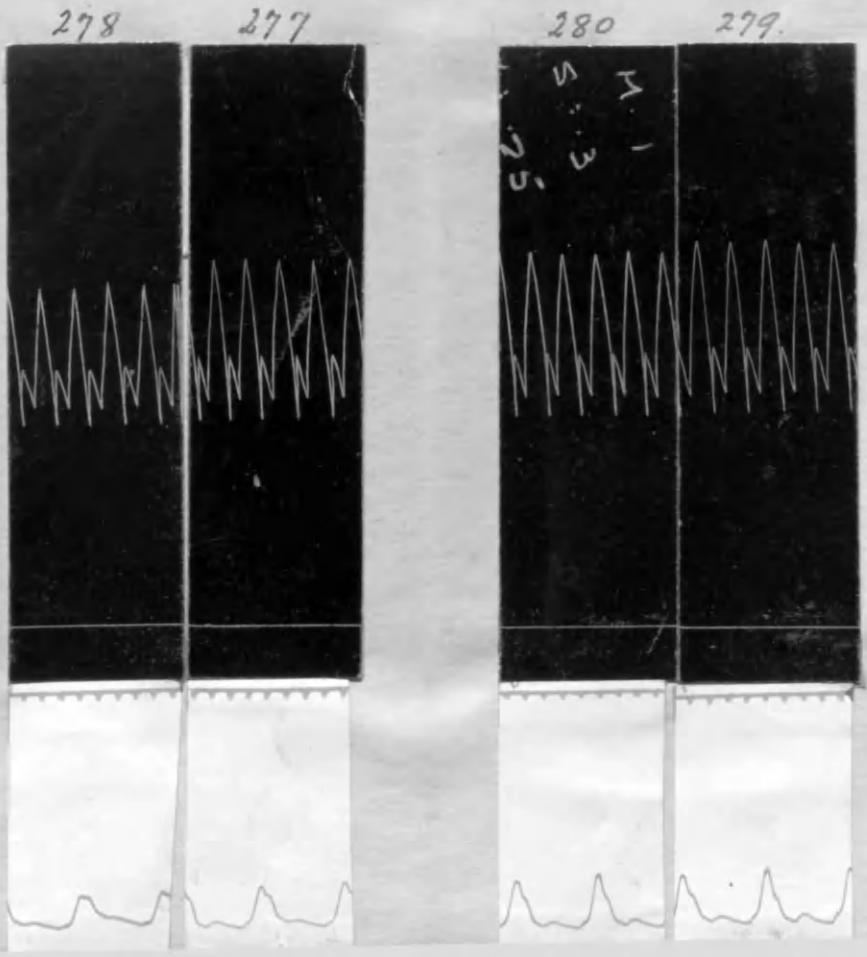

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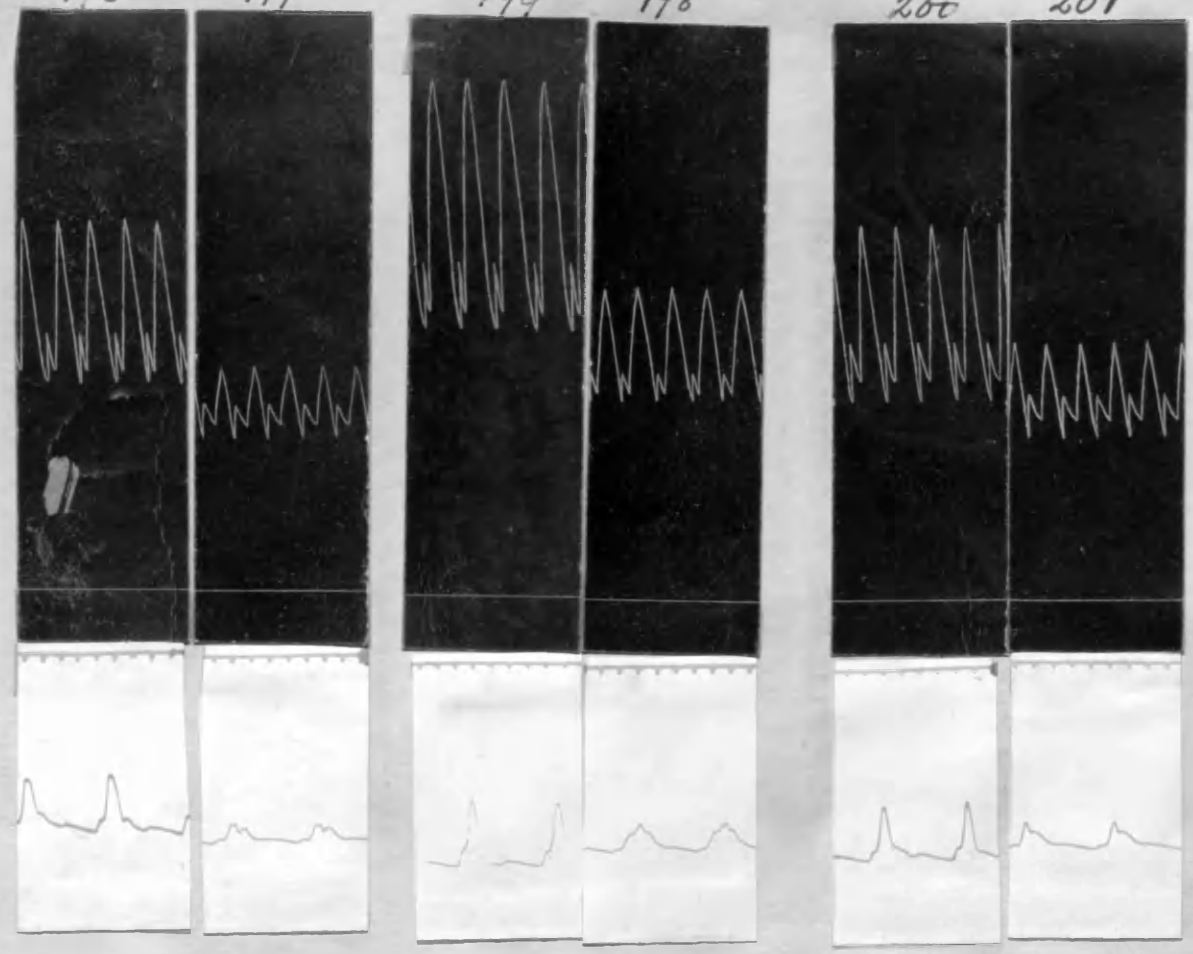

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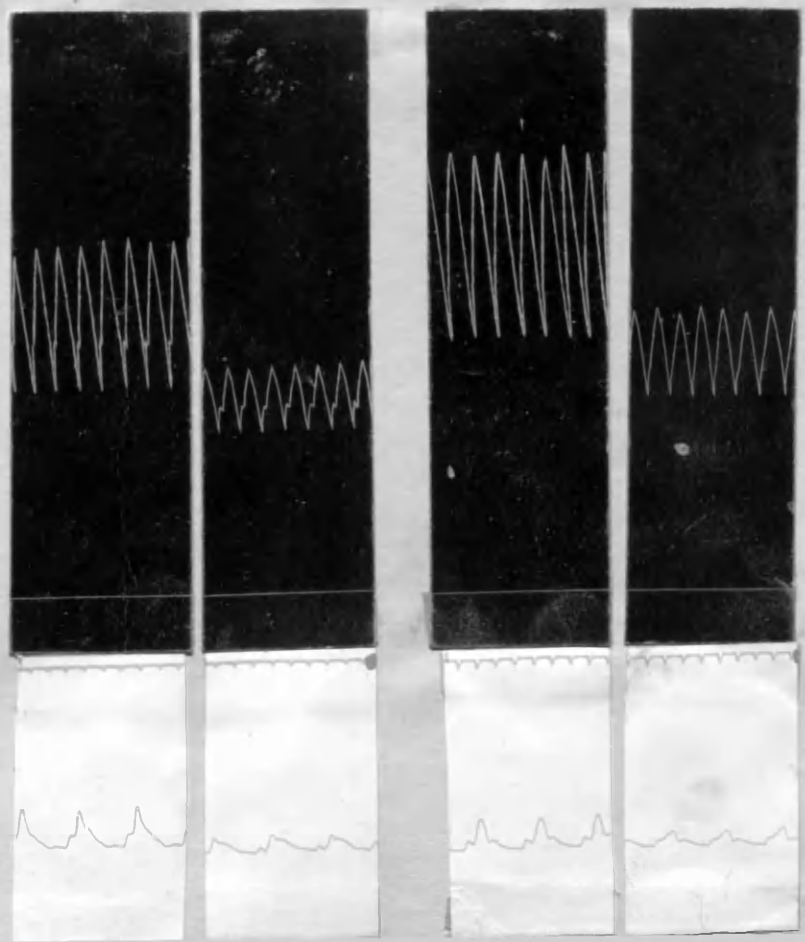

MITRAL INCOMPETENCE.

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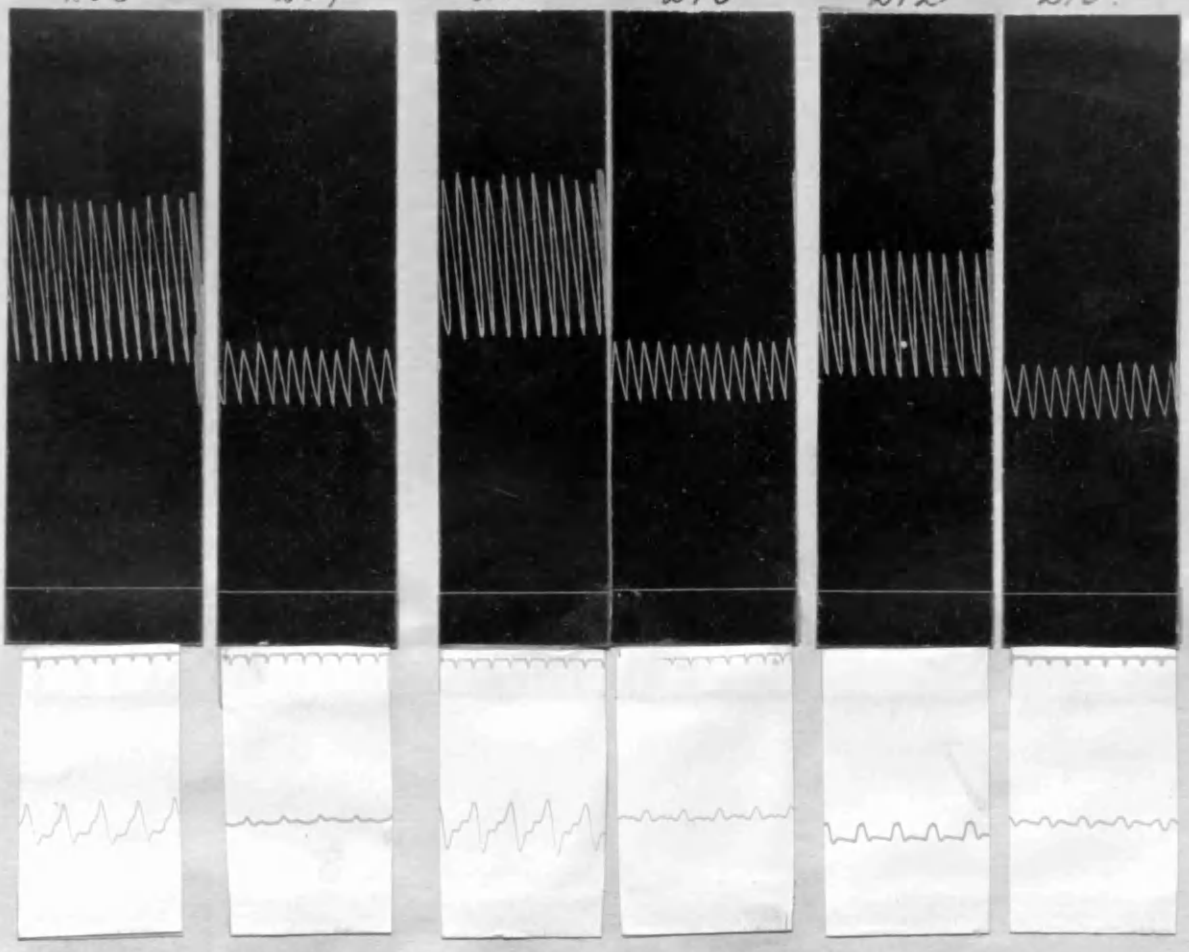

Series L. MITRAL INCOMPETENCE.

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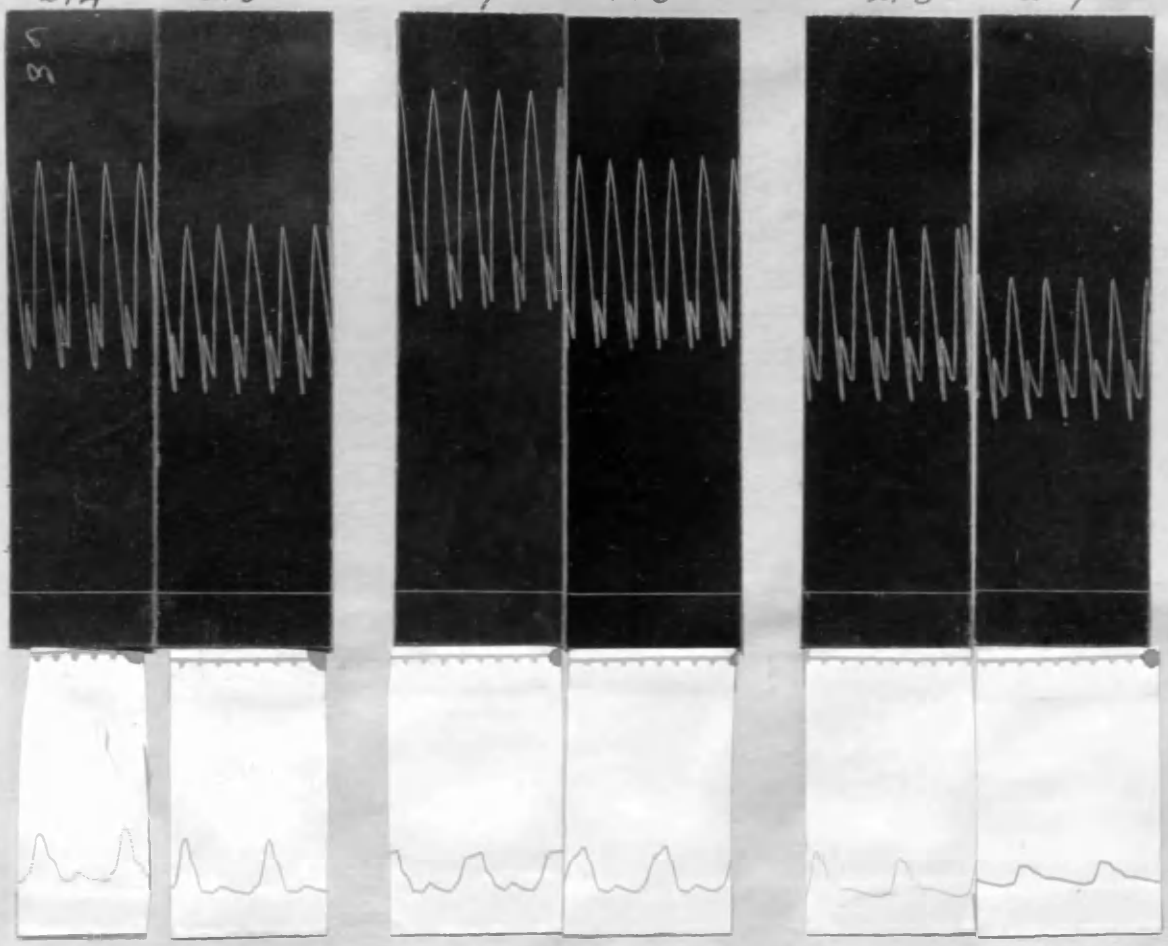

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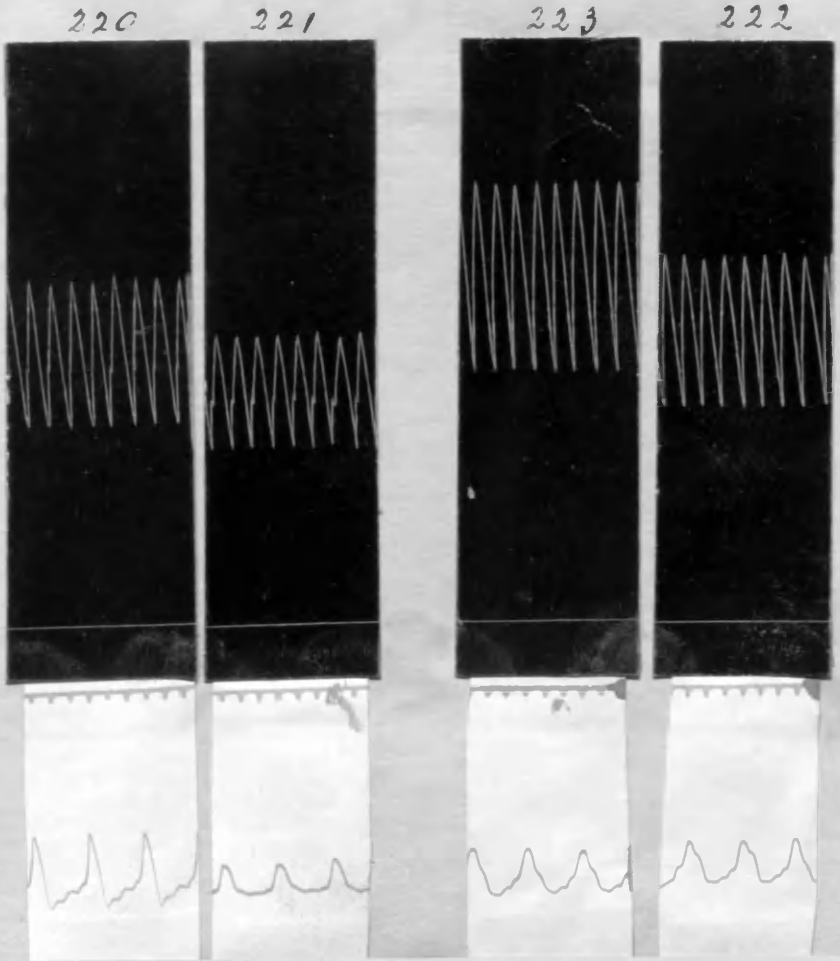

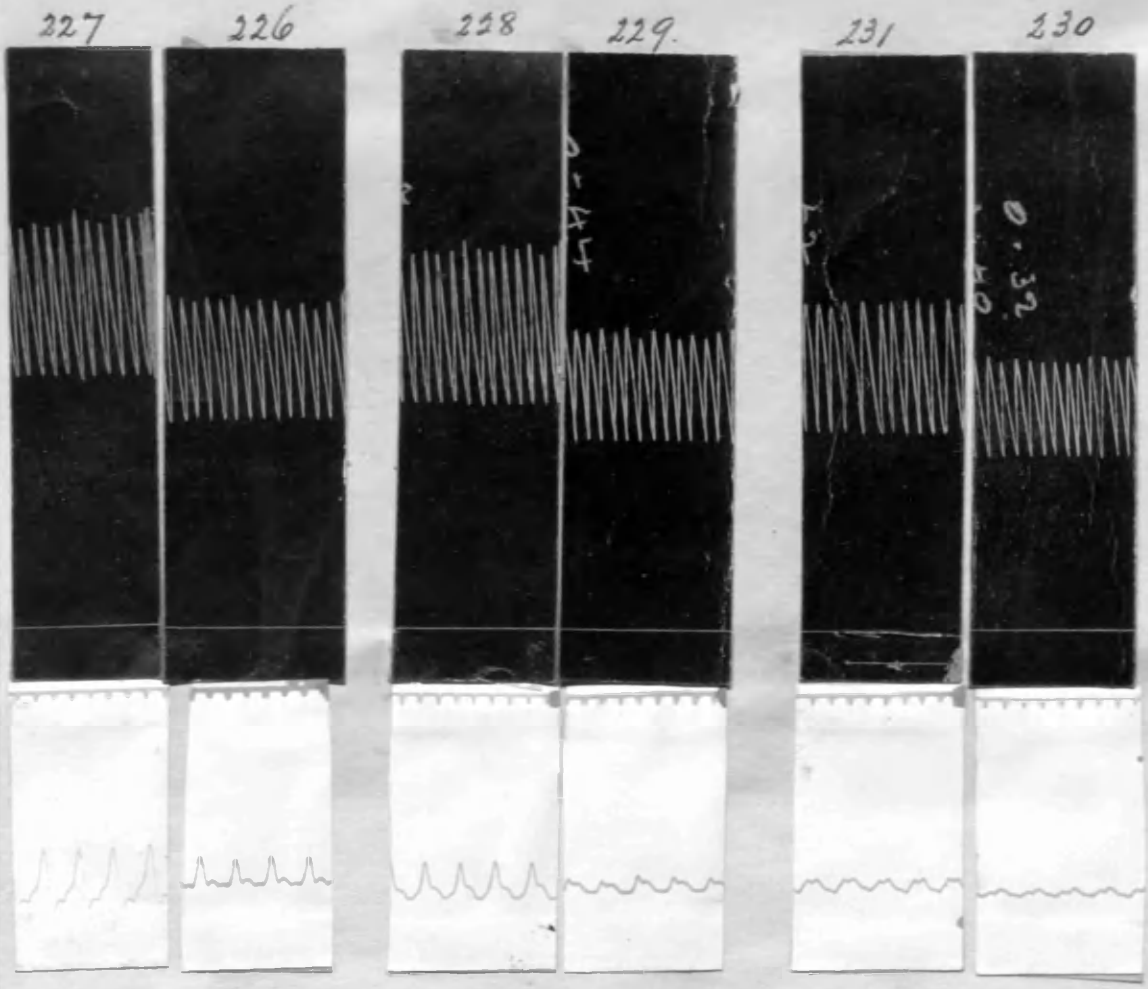
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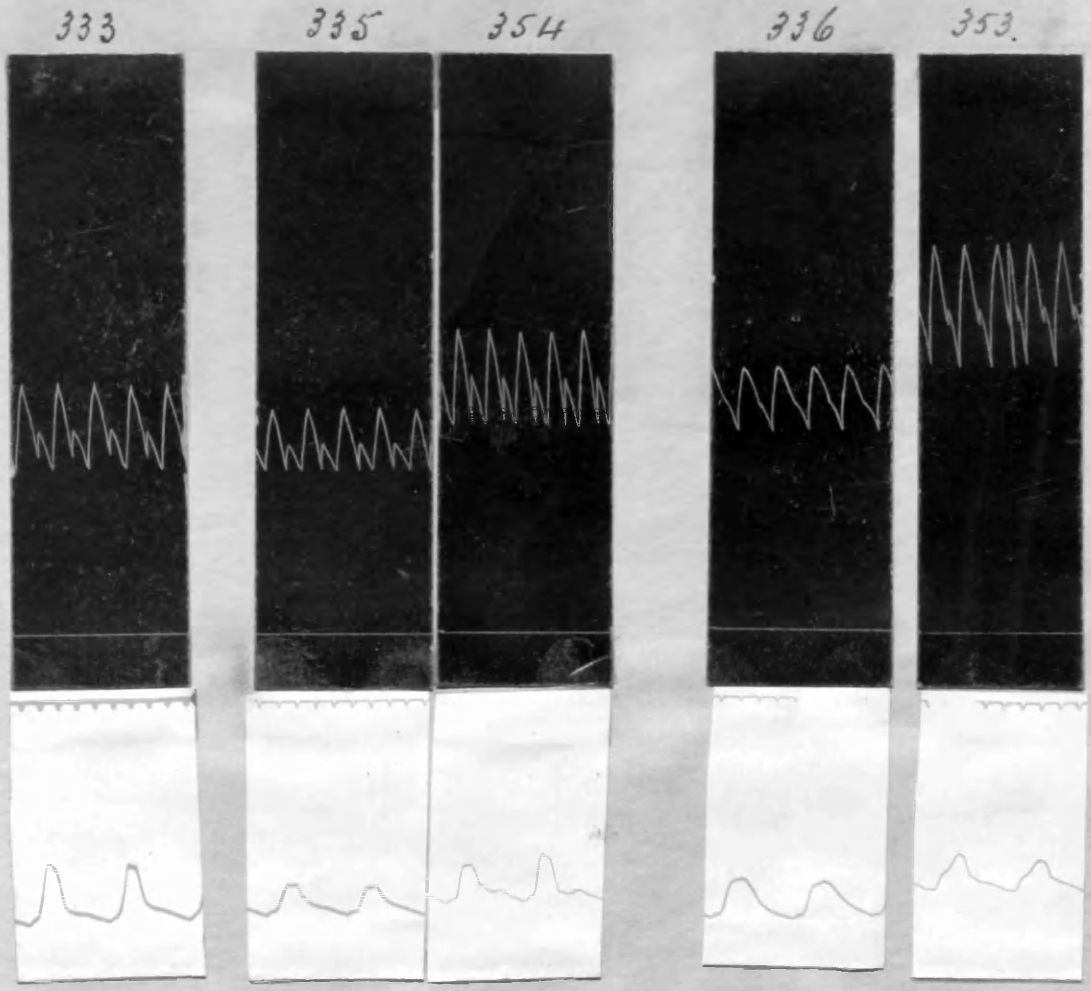
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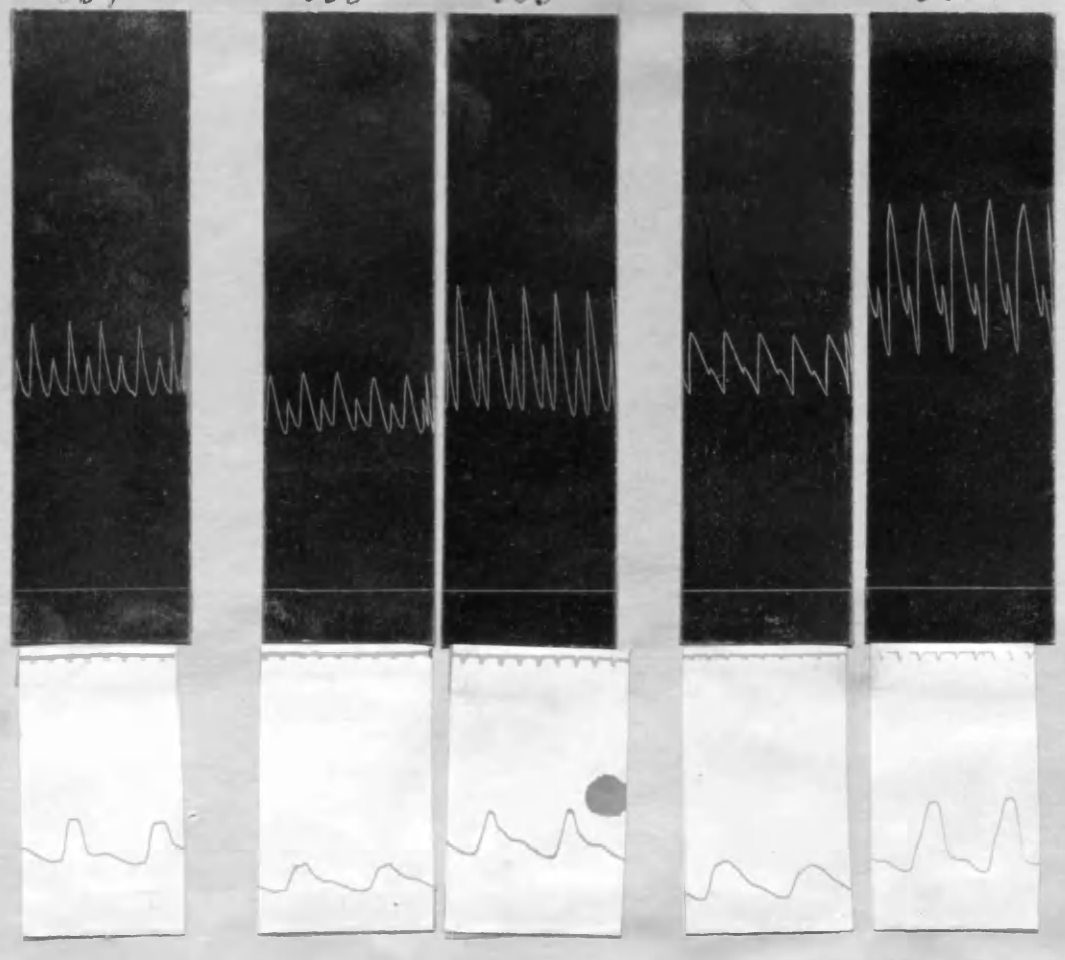

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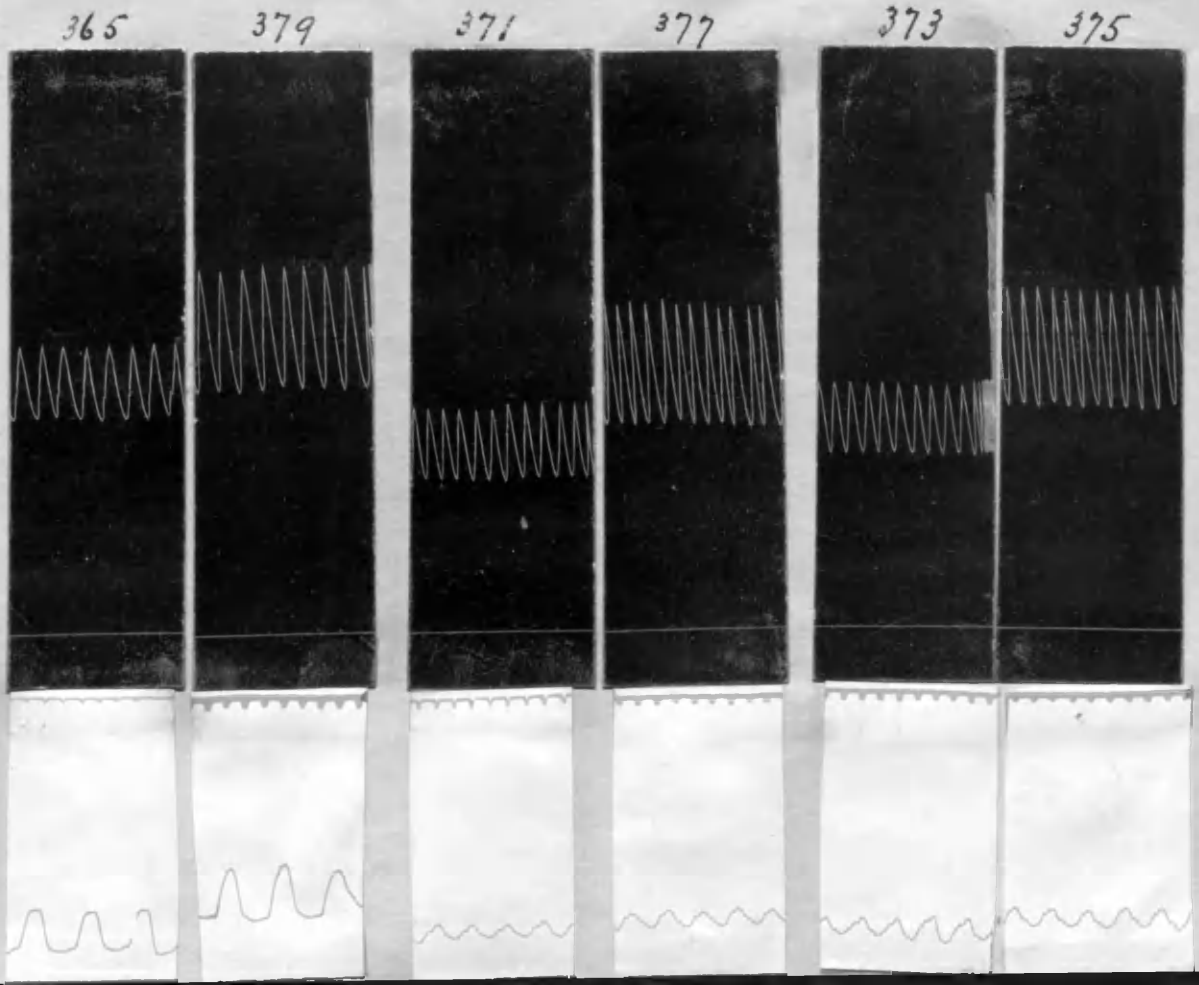

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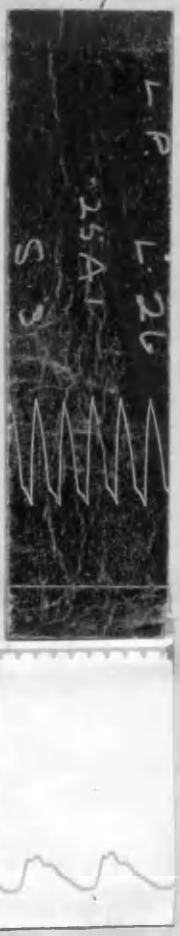

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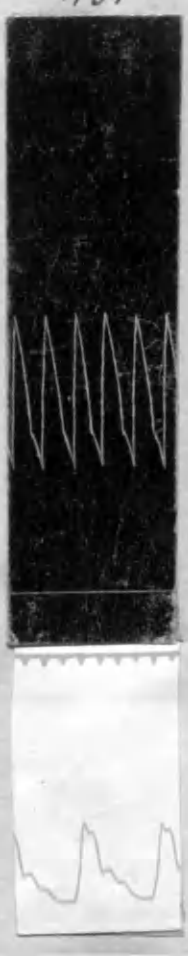

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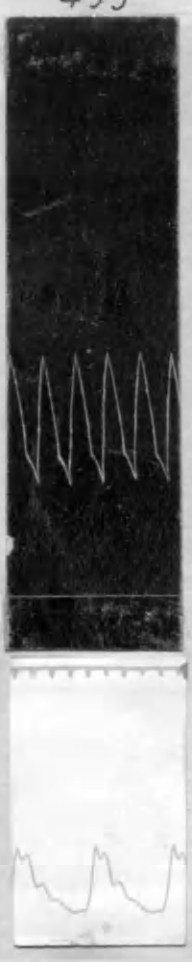

Series S. COMBINED LESIONS

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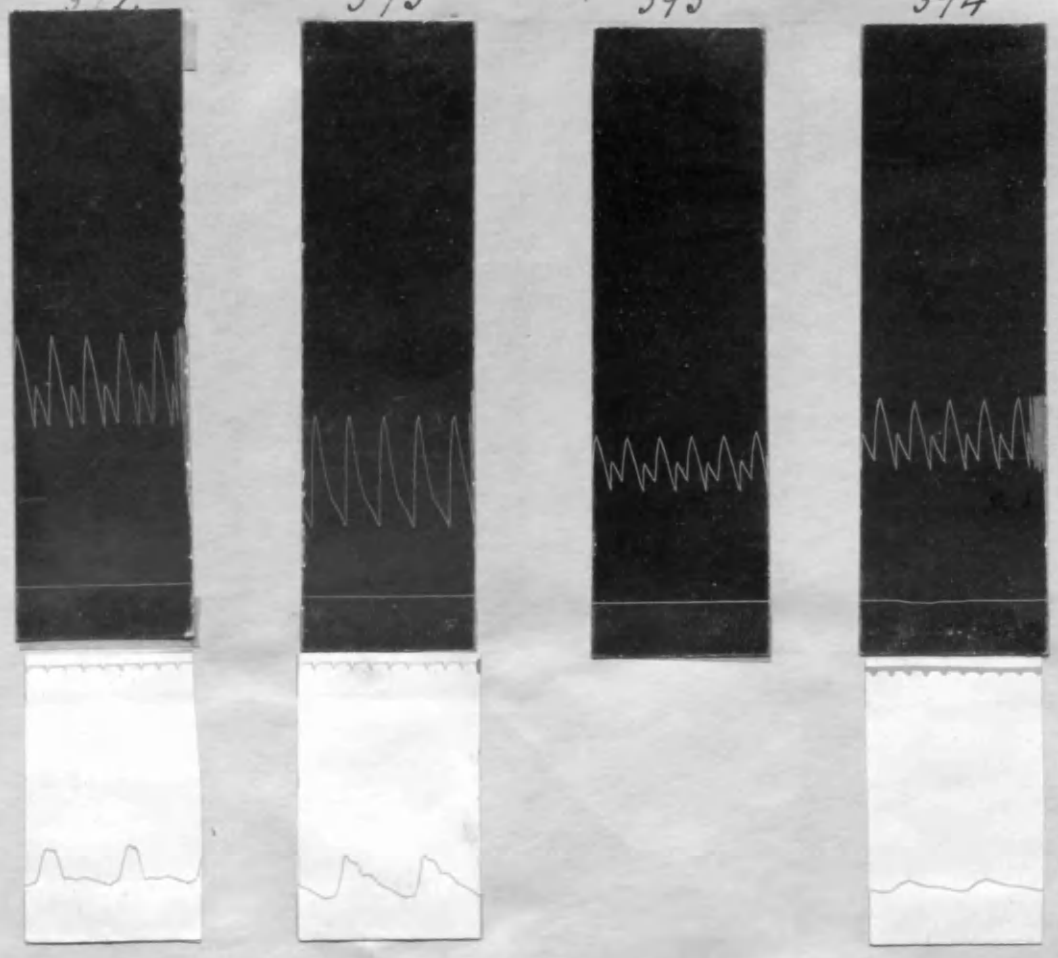

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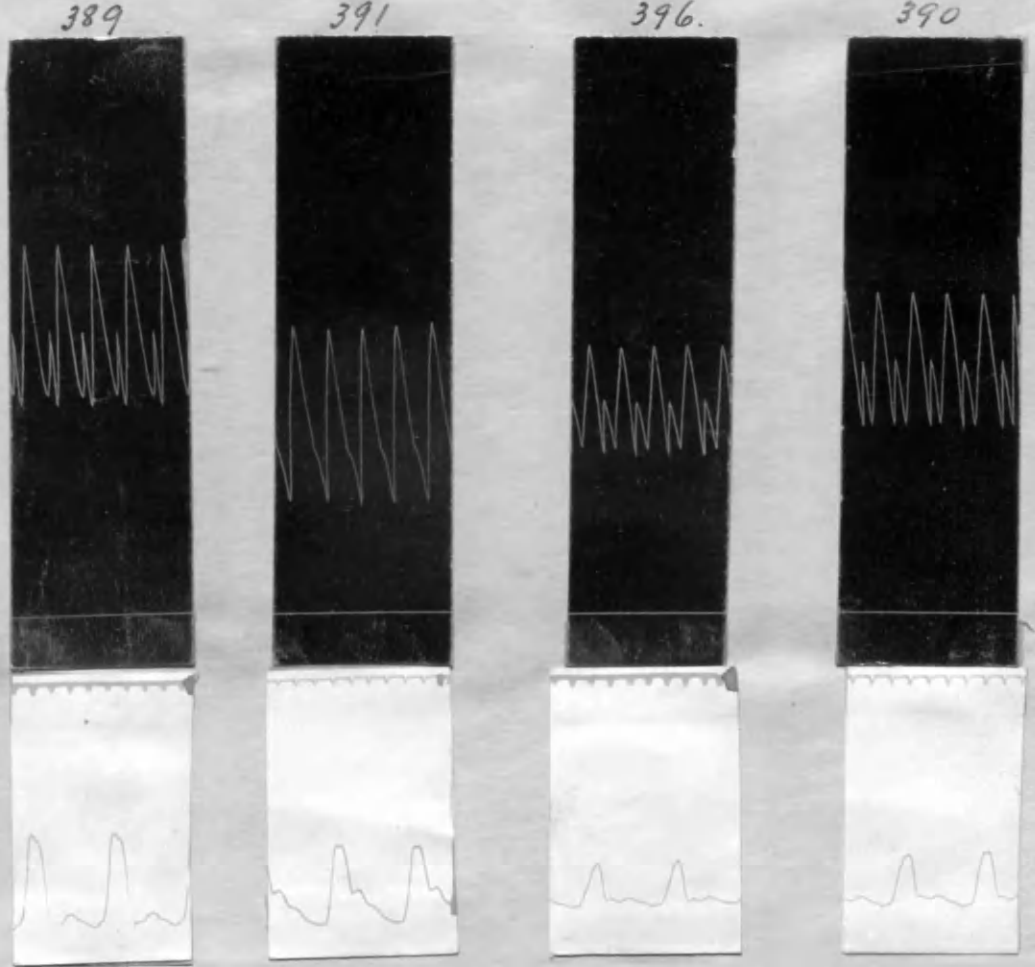

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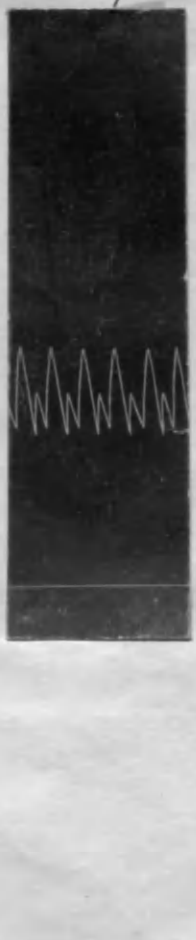

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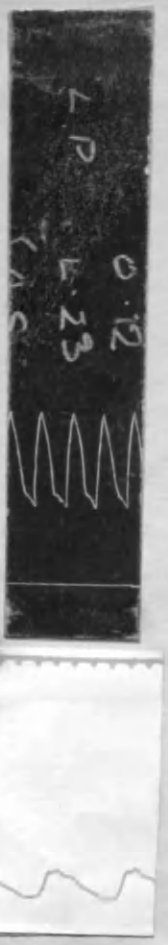

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