"A Clinical study of pure Aortic Regurgitation, with special reference to the prognosis in the condition, and an analysis of one hundred cases."

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A clinical study of pure Aortic Regurgitation, with a special reference to the prognosis of the condition, and an analysis of one hundred cases.

Prognosis must, at all times, and in the most favourable circumstances, be a very weighty matter, because so very many issues may depend on our pronouncements. In making a prognosis, we must sum up and most carefully weigh and consider every factor which has any bearing on our case - whether it be the physical signs - the individual's past history, his domestic and working surroundings, and a whole host of other material points. In my own experience, and in that of many of my colleagues, the question which troubles our patients most is not so much "What is the matter with me?" or "How ill am I?" but "Shall I ever be well and fit for work again?", and it is in cardiac lesions that this question becomes of very special importance, because so very much depends upon our answer.

Our answer may involve the re-arrangement of the patient's whole life for the future, and also of his work, if his life is to be prolonged. In cardiac affections, the question which is foremost in the minds of our patients is that of sudden death, because the man in the street has got an idea that sudden death and "heart disease" go hand in hand. Assuredly /
Assuredly in the lesion under our consideration, aortic regurgitation, sudden death is a frequent occurrence, and in it, of all the cardiac valvular lesions, our prognosis must be of a grave nature, for the reasons I shall, later, detail.

As part of our prognosis and of our treatment in this condition, we must eliminate every factor which produces any sudden strain on the heart, either physical effort or mental emotion. We may have to suggest, therefore, change of occupation for example, to one which involves the minimum of strain both mental and physical, one in which the surroundings are suitable, involving no change of temperature or exposure to climatic changes. This, of course, also involves the means of getting to work, as obviously any hurrying for bus, tram or train, with the exertion entailed therein, may cause far more disastrous results than the work itself.

Again, we may have to give a prognosis in the case of a lady, with this serious condition, who wishes to get married. Should she be married, and can she with any safety undertake the ordeals of pregnancy and parturition?

In children, whose hearts are affected as the result of acute rheumatism, or of one of the rheumatic manifestations, e.g. chorea, growing pains, tonsillitis, we may have to decide whether they can go to schools where they may not have all the comforts and cares of home - where the child may take liberties
with itself in the matter of exercise and games, and where of course the mental strain of lessons will have some effect. In children we must be specially careful and guarded in our prognosis, because any cardiac lesion in them, is so prone to involve the whole cardiac structure, endocardium, myocardium, pericardium to cause in short a pericarditis. In such cases the outlook must be grave indeed.

Finally, our advice will be sought as to the chances of an individual with the lesion, who has to undergo an operation involving general anaesthesia. What are the chances of sudden cardiac failure in such case?

So, then, in all these conditions, it behoves us to employ every means of investigation in making our prognosis. It will not be sufficient just to rely on the stethoscope and finger. Polygraph, electro-cardiograph, sphygmonanometer, must all be used. Our patient must be examined, lying down, standing up and after exercise, and moreover, more than one examination must be made if necessary, because the very fact that the examination being made is of such grave import may so upset the individual's nervous system, that we may get a totally erroneous, idea of the state of affairs.

Prognosis /
Prognosis in valvular lesions, then, especially in Aortic Incompetence should not be attempted until every factor has been most carefully weighed up, until to every sign and symptom, individually and collectively its due significance has been given and until every means of examination has been employed. Then and not till then, should our pronouncement be made.

INCIDENCE OF AORTIC REGURGITATION.

GENERAL INCIDENCE.

Of all the valvular lesions, those of the Aortic valve are the most serious, and at the same time, are fortunately, the least common. A short examination of some figures will show this. During the years 1893-1897 inclusive, 1914 cases of valvular lesions were admitted to the Royal Infirmary, Edinburgh. Of these 411, or 21.4% were pure aortic lesions, whilst 1120 or 58.5% were affections of the mitral valve, giving a proportion of mitral affections to aortic of almost three to one \[1\]. During the year 1921, the most recent year of which I can get complete statistics, only 34 cases of pure aortic lesion were admitted to the same institution.

Again, in the John Hopkin Hospital, Baltimore, of 209 consecutive cases of valvular affections, 53 were pure aortic, 77 were pure mitral, and 41 were combined aortic and mitral lesions \[2\].
Perhaps the most striking figures of all, are those given by the physicians of the National Heart Hospital, London, in a report on the examination of 10,000 recruits with doubtful hearts who were examined at that Institution on behalf of the War Office, during the years 1915-1918. Of this large number only 124 cases of pure aortic lesion were found, and 183 cases where an aortic lesion was combined with some other valvular lesion.

The lesion under consideration, that of Incompetence of or regurgitation at, the aortic orifice is by far the commoner of aortic lesions, and it must be admitted that it is, of all the cardiac valvular lesions the most grave - and, as I have stated before, the one in which sudden death is most likely to occur.

SEX INCIDENCE.

With regard to sex incidence, it is only to be expected that the sex which has had to bear the heavier cares and stresses of life should suffer more from the grosser cardiac lesions. If we lay Lockhart Gillespie's figures under contribution again, we find that the proportion of males to females suffering from aortic incompetence is as seven to one - a very significant statement when we consider the etiology of the condition. In the hundred cases I examined - statistics of which are given at the end of the thesis, 87 cases were males and 13 were females - an almost identical proportion. In other
statistics the proportions are not quite so high perhaps, but taking an average through several sets of figures the proportion works out about six males to one female.

Women are more liable to mitral affections, because for some reason or another - in them, any endocardial affection, any sclerotic degeneration would seem to affect the mitral orifice more than the aortic.

AGE INCIDENCE.

Further examination of Gillespie's figures, show that of his 411 cases of aortic lesions, 139 occurred during the age period 50-69; 108 during the period 40-49; 80 during 30-39; 58 during 20-29; 15 during 10-19 and 11 occurred over the age of 69.

In the National Heart Hospital report already referred to, the largest number of aortic regurgitants were found in men between the ages of 27 and 31 years, and the smallest number - 38 - between 37 and 41 years of age. These two sets of figures might at first sight seem contradictory, but it must be borne in mind that Lockhart Gillespie's figures are for patients actually under treatment for their cardiac condition which had probably existed for some years previously whilst the latter set of figures is for young men examined fairly early in life.
in whom the lesion had occasioned no definite symptoms, in whom the lesion had, as it were - been accidentally discovered. The two sets of figures would rather suggest a cause which had acted in early life, and had ceased to operate as age advanced. This strongly supports the theory of the rheumatic origin of the lesion.

THE ETIOLOGY OF AORTIC INCOMPETENCE.

I think we may take it, that, speaking broadly, primary Aortic incompetence, is due to one of the five following causes: -

1. Rheumatic Fever.
2. Syphilis.
3. Trauma or Strain.
4. Degenerative changes other than Syphilitic.
5. "Functional."

Secondary lesions of the aortic valve arise in cases long standing Infective Endocarditis of a sub acute type, where the infection spreads eventually to the aortic valve. There may, or may not, have been a previous lesion at one of the other valves, notably the mitral valve.

No matter what be the cause, however, the end result is the same, a deformity with a puckering and adhesion of the segments. This leads in time to one of two things namely, either a faulty closing of the valve segments, or the valve does not close at all. Though the systemic results are identically the same, each of the above causes produces local effects which are /
are totally different when examined postmortem.

1. RHEUMATIC FEVER.

The Aortic orifice is affected more by the poison of acute rheumatism than by any of the other constitutional affections. Parrott has shown that of 1100 cases of endocarditis, which is the form rheumatic fever takes in infecting the heart, 380 cases of aortic valvular trouble followed. Sperling of the Berlin Pathological Institute gives 40 aortic valvular cases out of 200 cases, and in the John Hopkins Hospital 330 cases of rheumatic fever gave rise to 110 cases of endocarditis.

Undoubtedly, rheumatic fever is the chief cause therefore, of aortic incompetence. It affects the valve in the form of endocarditis, a sequela of acute rheumatism.

Of 307 cases of aortic incompetence examined at the National Heart Hospital, London during the years 1915-1918, 162 or nearly 60% gave a definite history of acute rheumatism, and a further 55 or just inside 18% gave a history of "rheumatism" in one or other of its various manifestations, e.g. chorea, tonsillitis, growing pains, etc.

Of the hundred cases I examined at the same institution, and whose records were taken at random 44 gave a definite history of acute rheumatism and 43 of "rheumatism."

Cowan /
Cowan and Poynton and Paine have shown very concisely the effect of the poison of rheumatic fever on the cardiac mechanism. They have shown that the precise site of the primary affection of the endocardium by the organism is the base of the valve. Following very quickly on this primary affection the margins of the valve segment become infiltrated and swollen. The next thing that may happen is that the swollen and infiltrated edges may ulcerate with a very marked loss of substance. Alternatively, we may find vegetative growths forming on the segments. These in due course lead to ulceration of the segments and their destruction. We may therefore get all stages and conditions in this lesion up to and including total destruction of the valve.

Coexistent with these endocardial changes, we may get, indeed frequently do get degenerative changes in the heart muscle itself; and it is on these changes that the individual's future life so much depends, for if the myocardial changes be extensive, then the outlook is anything but promising. One of the most prominent changes— and one in which the outlook is extremely grave—is that of a fatty degeneration.

As a result of the poisoning of the acute rheumatism—a poisoning which has a very marked effect on the myocardium—an acute degree of dilatation of the left ventricle frequently occurs, and when /
when it does occur is a sign of very grave import. So great at times has been this ventricular dilatation, that it has actually been diagnosed as a pericardial effusion.

Of course not every case of rheumatic fever presents indication of cardiac involvement, during or soon after the acute condition, but there are very few people who do not show sooner or later, some cardiac weakness, as the result of the acute rheumatic illness.

What has been said of acute rheumatism holds good of the subacute rheumatic affections - growing pains, chorea, tonsillitis. Of the ten thousand recruits with doubtful hearts examined during the war at the Heart Hospital, many giving a history of one or other (or a continuation of them) were found to be suffering from aortic incompetence either as a single lesion or accompanied by some other cardiac complication.

(II) SYPHILIS.

It is now definitely recognised that Syphilis plays a very important part in the production of aortic incompetence. The changes produced in the valves are those of a degenerative or sclerotic type. The poison of rheumatism attacks the valves from within the heart cavity - endocardially; but syphilis on the other hand attacks the valve from without /
without - or exocardially. The poison first attacks the aorta, and thence travels backwards to the valve guarding its origin, that is to say the condition commences as an aortitis, not an endocarditis.

The first effect is a loss of the pliancy of the segments, then follows a gradual thickening and shrinking of these. There is no actual loss of tissue as in the rheumatic variety, but the segments are shrunken, and contracted, resulting in the incompetence of the valve.

It is of great importance, both from the prognostic and therapeutic points of view, especially in those cases where a luetic history has been denied - to distinguish between cases of aortic insufficiency caused by rheumatism and those caused by syphilis. Many authorities have sought to lay stress on the difference in the qualities of the murmurs as distinguishing features, but in my opinion, and that of others, this is by no means a reliable guide. We must have recourse to other methods. Naturally one would try the Wassermann reaction, but, here again this is not always a certain sign. Even electrocardiograms are of little help. We get a certain amount of help from the appearance of our patient. In the syphilitic variety, the complexion is pallid, 'tis true, but the pallor is not /
not that of a typical aortic incompetence. It is more putty coloured, or dirty ashen. The age again sometimes helps, the luetic being usually of later life than the rheumatic, or infective fever type. Then the duration of the cardiac illness is of some service. The illness in the endocardial variety is of longer duration than in the luetic variety. This is evidently due to the fact that the endocardial type usually occurs in younger people where the myocardium-coronaries, etc. are more healthy than in the exocardial patient, and also to the fact that the rheumatic variety becomes stationary in many cases fairly early in its progress, whereas the syphilitic tends to progress steadily, unless treatment has been undertaken early. Of course a definite history of rheumatic fever or of one of its subacute manifestations or on the other hand a definite luetic history, clears up things at once. But if we have a case of aortic regurgitation in an individual aged from 35 up to 45 or 50 years, who gives no history of rheumatism in its broadest meaning or of any of the infective fevers - then we are entitled to suspect a syphilitic origin.

The point on which most authorities lay stress, however, is the fact that in Syphilitic aortic regurgitation that lesion exists by itself, whereas in the endocardial type, the lesion is often accompanied by aortic stenosis.

III.
III. STRAIN OR TRAUMA.

Aortic Incompetence due to strain may be (1) a temporary affair, where the valve has become incompetent under some sudden unaccustomed strain, and where with rest and abstention from all hard physical exertion the condition has passed off. Many such cases were seen in both sexes during the late war, where individuals who had hitherto led sedentary lives without a great deal of exercise were suddenly called upon to undergo hard physical training, route marching, motor driving, etc. This class of case is that which certain authorities label as "functional", and in which all the symptoms and physical signs passed off under rest and quiet.

Then (II) the "functional" incompetence may become permanent, through persistence in the same work which caused the original trouble, with damage to one or other of the valvular segments. When one of the segments ruptures it has been shown that it is, in the greater proportion of cases, the left posterior cusp which suffers. The reason for this is not apparent but the rupture does occur during diastole when the aorta is full, and when the blood cannot escape except by some abnormal channel.

Traumatic rupture of the valve may be due to direct violence - a very hard blow or kick on the sternum has caused rupture of an aortic valvular segment, or it may be due to indirect violence, some /
some sudden effort on the part of the individual himself. In this connection it has been shown that comparatively short and light work involving muscular effort, and to which the individual has not been accustomed, is sufficient to raise the intra arterial blood pressure and put a very severe strain on all the cardiac valves.

IV. DEGENERATION NOT NECESSARILY SYPHILITIC.

This is by no means an uncommon cause of aortic incompetence. It is seen in individuals on the wrong side of middle-life who have lived a life of ease and pleasure - no stinting of alcohol or tobacco, and who have generally "enjoyed life". It is also seen at the other end of the social scale in workman who have been exposed to certain metallic poisons e.g. lead, mercury, etc. Gout, Bright's disease, also bring aortic incompetence in their train in many cases. The condition produced here is closely allied to the sclerosis produced by Syphilis which has already been discussed. Here, however, the change in the aortic valve is part and parcel of a general cardio vascular change with calcareous thickening of the arterial walls. The segments undergo similar changes, becoming hard, rigid and contracted with resulting failure to close the opening.

It is /
It is in this variety more than in any other, except perhaps cases arising from Syphilis, in which angina pectoris makes its appearance fairly early. The small openings of the coronary arteries get blocked up gradually, the vessels themselves become sclerosed and angina results. This as a matter of course makes the prognosis extremely grave.

V. THE SO-CALLED FUNCTIONAL AORTIC REGURGITATION.

Certain authorities, both at home and abroad, have recorded cases where there was abundant evidence of aortic regurgitation during life, yet, on post mortem examination, no definite lesion of that valve was found. Stolkind quotes such a case in which a definite diastolic murmur was audible during life, but at the post mortem examination, the patient died of cerebral tumour - no lesion of the aortic valve was found. G. Duncan Whyte of Swatow China records a case of a Chinaman suffering from ankylostomiasis, who had definite signs of aortic regurgitation which disappeared on anthelmintic treatment. He also records, in the same article, cases which had a definite diastolic murmur, which however, disappeared on treating the haemic condition. These and similar cases are undoubtedly due to some relaxation of the aortic ring, sufficient to /
to produce a leakage, with a possible dilatation of the first part of the aorta.

The word functional is I think, unfortunately used often to cloak our ignorance. Every organic lesion starts as a functional lesion, that is some small derangement in which the normal function or action of the organ concerned is upset, and eventually lead on to something more grave. I think if these so-called "functional" disorders of any organ were followed up carefully, permanent lesions would be found to have developed in later years. It behoves us to be very careful in dealing with such cases, and not give a hasty opinion. A re-examination six or nine months later may alter all our opinions, and save us, perhaps, unfortunate ridicule.

SECONDARY AORTIC REGURGITATION OR SUBACUTE SEPTIC ENDOCARDITIS.

This form of aortic incompetence deserves special mention. It is part of a generalised septic endocarditis in which according to some authorities there is a primary mitral lesion on to which a septic infection of some kind becomes implanted. This becomes conveyed, in time, to other valves. Others say again such a previous lesion is not necessary, but that the septic condition starts "per se."

When /
When the aortic valve becomes infected, it is, as in the case of strain or trauma, the posterior left cusp which is affected, and results in a soft blowing murmur, diastolic in time.

The individuals who suffer from this form, are usually pallid, cheerful, complain of nothing or at the most slight breathlessness on exertion. On examining them we find an anaemia, with clubbing of the fingers and toes. There are always at least two valvular lesions, and the murmurs are always well-marked, indicative of a gross lesion. Petechias occur in the skin, particularly on the legs and feet. At the points of the digits small nodes - Osler's nodes' form and these are nothing but small emboli.

The temperature in such cases is swinging with a very marked evening rise, and this is usually accompanied by profuse sweating. There is usually albuminuria of a persistent type.

We should always suspect such a condition when a preexisting valvular lesion develops a sudden swinging temperature accompanied by a steadily progressive anaemia, and when above all the pulse becomes suddenly of a collapsing type and where we have no definite signs of aortic incompetence. I shall deal with the prognosis of this type later on.

Therefore /
Therefore, whether the cause be endocardial or exo-cardial, sclerosis, strain, trauma, or so-called functional, the end result is the same, namely, a patency of the aortic orifice, a failure of the valve segments to close tightly. This, going on for any length of time produces certain well-marked, definite effects on the general bodily system, and these I shall now proceed to discuss.

THE SIGNS AND SYMPTOMS OF AORTIC REGURGITATION.

Although the signs and symptoms of Aortic Regurgitation are very definite when taken collectively there is no single one, which can be called diagnostically distinctive, of a definite lesion of the valve.

Many cases of the condition exist, in which compensation is so well established and so perfectly adjusted, that the individual carries on his daily avocations for many years, it may be without any difficulty, and without any suspicion of the existence of anything abnormal. Then one day he does something which calls for a little extra strain, physical or mental. This upsets the compensatory balance, and then the individual finds out that something has gone wrong. He finds perhaps a little pain in the chest on some exertion, or that he cannot get upstairs without being breathless and having a good deal /
deal of palpitation. That is a very common state of affairs, but we often find that, although compensation is well and adequately maintained there may be occasionally no sign or symptom present which indicates that all is not well with the cardiac mechanism. This may again be some slight breathlessness, palpitation or exertion, and so on, which are often put down to attacks of indigestion or a little "bronchial catarrh". Taking a broad view of the facts then, in the endocardial and exocardial forms the symptoms come on very gradually, and almost imperceptibly progress.

Our patient finds that there is pain in his chest on any exertion, a pain which gets worse and which arrives at length independent of the exertion and which stays longer than it did at first. Associated with this pain, there is palpitation occurring frequently independent of exercise often in bed at night, breathlessness, cough, vertigo, fainting, and in the later stages insomnia of a very painful and distressing type, and also diuresis. This last is rather an important sign, and one which I don't think has been quite appreciated. The "raison d'être" I shall discuss later. Associated with all these signs and symptoms, there is a feeling of oppression and depression, in marked contrast /
contrast to the subacute infective endocarditis type, when the individual is nearly always most cheery and optimistic. There is the constant feeling of "pumping going on inside" as one patient graphically described his sensation, and often an intense throbbing headache. The appearance of the individual is characteristic, pallid in colour, suggestive of a profound degree of anaemia, more apparent than real. There is the typical capillary pulsation, elicited in the usual way by drawing a finger nail rapidly and firmly across the forehead or by pressure on the finger nails.

On stripping the patient, the eye is at once arrested by the marked pulsations present in the regions of the main arteries - carotids, brachials, femorals, and frequently a marked epigastric pulsation. The examination digitally of the radial pulses reveals all those phenomena so well and graphically described by Corrigan in his original work on the subject.

There is no definite rate of pulsation in this condition, it may be fast or slow according to circumstances. The rhythm shows no marked alteration, and the pulsations are all nearly equal to each other. Each individual pulsation is characteristic-presenting the typical "water hammer" type. The rise is quick and full, is very badly maintained, with a quick and complete fall.

Corrigan /
Corrigan originally explained this pulsation as being caused by a large output of blood during systole causing a wave of high pressure in the arteries. Now as soon as systole comes to an end and the discharge of blood from the ventricle ceases, the patency of the aortic valve allows of a regurgitation of the blood, into the ventricle with a resultant fall in the diastolic pressure. This is not all however. The collapse after the wave occurs whilst blood is actually flowing from the ventricle into the aorta and not when regurgitation from aorta to left ventricle is taking place.

It is important to observe, however, that a pulse - very similar to a Corrigan - and also a similar capillary pulsation - is frequently found in cases of arterio-sclerosis without any damage to the aortic valvular segments. They are also found in conditions, like Graves disease, in which there is a distinct lack of tone in the arterial walls.

PHYSICAL EXAMINATION.

PALPATION. If we place our hands on the praecordia we are at once struck with the violence of the cardiac pulsations, and we notice that the diastolic recoil is very marked. The apex beat is also very pronounced and it is as a rule displaced somewhat downwards and outwards. In addition to these - a very marked thrill, diastolic in time
may be felt perhaps over the front of the chest generally - but perhaps most markedly over the upper part of the chest - corresponding to the area of the cardiac base. This thrill is caused by the sudden back flow of blood from the aorta, through the patent aortic valve into the left ventricle, and, naturally, the more patent the orifice, the more marked and distinct the thrill.

PERCUSSION.

This reveals a marked increase in the area of the cardiac dulness. This increase is in the longitudinal diameter as well as in the transverse. The left border of the heart is, as might be expected, much more displaced than is the right.

AUSCULTATION.

This reveals at once the typical diastolic murmur, which may vary greatly in character from a soft blowing murmur, to one which is harsh and almost metallic. This wide range is due, perhaps, to the degree of patency of the valve, and Gibson points out that in his experience the harshest murmurs are those which are due to a ruptured cusp, or where there is loss of substance in a segment due to vegetations eating it away. There is another theory which is now being advanced with a great deal of confirmation, and that is that the soft blowing murmur indicates an incompetence due to a posterior cusp lesion /
lesion - whilst the harsher murmurs are those due to lesions of the anterior segment. In connection with this it is pointed out that the first variety soft murmurs are associated with precordial pain to the left of the upper part of the sternum towards the Pulmonary area, whilst the harsher murmurs are associated with pain and are found on the right side of the sternum. The maximum intensity of the murmur is best heard about the level of the fourth right chondro-ster nal articulation. This point of maximum intensity is, however, a very variable one - and may be found anywhere over the cardiac area - even as far out as the left nipple. The murmur is conducted downwards towards the Xiphisternum.

(13)

Austin Flint in 1862, and many other writers both at home and abroad, have described a presystolic murmur in the mitral area which accompanied the aortic diastolic murmur. Different theories have been propounded for this, but none can be accounted satisfactory except those of Bramwell and Guitéras. They say the cause of the murmur may be that the aortic lesion affects the posterior coronary segment of the aortic valve, - with the result that the full force of the regurgitating current falls, as it were - on the great anterior segment of the mitral valve, forcing it into the position which Flint has described as being essential for the production of the murmur.

In /
In addition to these cardiac murmurs - Durozies\(^{(16)}\) described two murmurs heard on ausculting the arteries in a case of aortic incompetence. One murmur follows immediately on the ventricular systole and the other immediately on ventricular diastole. The murmurs are entirely of local origin, and are produced by pressure of the stethoscope narrowing the lumen of the vessel. The murmur is diagnostic of the lesion, but on the other hand Balfour and Gibson \(^{(17)}\) are at one in stating that many cases of aortic regurgitation exist without these murmurs being audible. When they are present I have found that they can be heard most clearly in the femoral arteries in Scarpa's triangle - or in the popliteal arteries.

**BLOOD PRESSURE.**

In Aortic Regurgitation the standard picture in blood pressure is a high systolic pressure and a low diastolic. This of course results in a high pulse pressure. The systolic pressure in the arm may be as high as 300 mm of Hg. but the average is about 180 mm.

In this condition, it is interesting to observe, the blood pressure in the leg is always higher than that in the arm. This is due to over compensation, a hypertonic condition of the blood vessels. This this is so can be probed by immersing the legs in warm water, and then taking the pressure, when it will be found to equal that in the arm.

It is a matter of grave moment when we find the systolic /
systolic blood pressure in the arm, in a case of Aortic regurgitation beginning to fall. This is a certain sign of a failing myocardium. Similarly a low Blood Pressure from the start is bad. The only time when we would expect a low systolic pressure in this condition is when we have a small leak in the aortic valve as in a posterior cusp lesion. The pressure in the leg should always be taken in this condition as it is an excellent guide to the degree of damage.

SYSTEMIC EFFECTS OF AORTIC REGURGITATION.

Such then are the physical signs and a few of the symptoms of aortic insufficiency; what are the effects of the lesion on the body generally?

We may divide these effects broadly into two classes:

1. Cardio-vascular effects.
2. Systemic effects.

1. CARDIOVASCULAR EFFECTS.

Almost the first effect is cardiac pain, varying very much in degree. It has this peculiarity, that the degree of pain does not bear any relation to the severity of the lesion, for we find that slight lesions have very severe pain and vice versa. The severity would seem to depend on the degree of involvement of the coronary arteries.

Another early symptom is tachycardia, which is often most distressing, and which does not bear any relation to the amount of bodily strain undergone at the time for it is frequently most severe when the individual is quiet in bed, and not noticeable when he is doing any hard work.
The effects of aortic incompetence, on the heart itself come on early in the condition. As might be expected the constant regurgitation of blood into the left ventricle causes that chamber to dilate, in order to accommodate the increased volume of blood. To meet the strain thus imposed on it, the walls of the ventricle hypertrophy and continue to do so as the lesion progresses, and become more pronounced. The left ventricle thus growing and hypertrophying, begins to encroach on its fellow ventricle, causing trouble there. Finally as the result of all this, the mitral orifice becomes strained and gradually the cusps fail to close completely and we get a condition of mitral incompetence supervening.

With all this hypertrophy of muscle in the heart, and a reduced supply of blood it is only natural that changes will take place in the myocardium itself, and in these changes in the myocardium, lies the great danger in aortic incompetence. They lead on to impaired contractibility, impaired conductivity and a host of other changes. Those which are to be viewed with the gravest concern are undoubtedly auricular fibrillation and heart block in all its varieties, and a generalised fatty degeneration of the myocardium.

Other cardiovascular changes to be met with are throbbing carotids, and later generalised increased arterial pulsation. This is at times so very pronounced the pulsations are visible at some distance away, and as in a case I saw in the Royal Infirmary, Edinburgh, the throbbing /
throbbing carotids, give a perceptible movement to the head, as the patient lay with it on the pillow.

II. SYSTEMIC EFFECTS.

These, as may be imagined, are many and varied, and all can be traced to the increased arterial tension and the reduced blood supply. Their order of onset is inconstant, but speaking broadly, those which affect the nervous system are usually first in onset. We find often complaints of a most painful distressing temporal headache of a throbbing nature, which is often accompanied by vertigo, especially on change of posture. Insomnia is a frequent and trying complaint, and when sleep does come, the unfortunate sufferer is tortured by obnoxious and terrifying dreams. This insomnia is a very difficult symptom to combat, and unless energetic measures are taken to relieve it, it brings more serious mental troubles in its train. Dyspnoea did not used to be considered a frequent symptom, but I have found it so, both in hospital and private practice, and is, in the late stages, not associated with exertion. Cough is frequently complained of - a nasty hard cough - in the advanced stage accompanied by frothy blood stained sputum.

As frequently happens as the result of the regurgitation if there be dilatation of the first part of the aorta, or of its arch, we may get pupillary inequality as the result of interference with the sympathetic and also inequality in the radial pulses.

Vicarious haemorrhages are often a feature of this
this condition - epistaxis, haemoptysis - uterine haemorrhage being of relatively frequent occurrence, and these haemorrhages are often the first indication of anything seriously wrong. In my own practice, a few years ago, I had an instance of this. Miss M. D. aged 67 came complaining of frequent uterine haemorrhage; which had existed for two years previously, but which were increasing in frequency and amount. Investigation revealed a history of rheumatic fever in early middle life - and the pulse was suggestive of aortic incompetence, which was confirmed by auscultation. The systolic blood pressure was 165 mm. of Hg. and the diastolic about 80. Pelvic examination revealed no neoplasm of uterus or adnexa, nor was there any factor accompanying the haemorrhage nor was there any discharge.

Rest and cardiac tonics improved the cardiac condition, and the uterine condition also improved. After a heavy spring cleaning the haemorrhage started again, and became more profuse, and the cardiac condition markedly worse, signs of heart block supervening. The end came after three profuse uterine haemorrhages in twenty-four hours.

Diuresis is a fairly common complaint in this condition, and like the haemorrhage may be the thing that brings the individual to seek medical advice. It is due to the high arterial pressure in the kidney /
kidney, and may cause the sufferer considerable inconvenience especially at night. Albuminuria is not found as a rule traceable to the lesion. If it exists, then there is usually some other cause of it.

Neuritis of a very painful and obstinate variety is of commoner occurrence, particularly in the cervico-brachial region.

Finally in advanced cases, mental symptoms of melancholic type - supervene, giving rise to much anxiety on the part of the unfortunate individual's relatives - and frequently ending in suicide of the sufferer himself.

THE PROGNOSIS.

Of all the valvular lesions aortic regurgitation is the one in which, as I have said, there is the greatest risk of sudden death. The cardiac musculature is unable to stand up to the ever increasing strain caused by the constantly increasing regurgitation of the aorta with all its sequelae of hypertrophy and dilatation, and eventually the ventricle becomes paralysed as it were, a condition of asystole. Death may also ensue from rupture of the heart, not an uncommon ending in this condition, embolism of the pulmonary circulation - thrombosis of the coronary arteries - or finally from apoplexy. When death occurs from asystole, post-mortem /
mortem examination reveals the ventricle to be full of blood and in diastole.

In aortic regurgitation, we must be clear as to the real cause before we give any definite prognosis. If the original cause was an uncomplicated rheumatism the outlook of the individual is quite good, because the damage is not likely to spread after energetic antirheumatic measures have been taken. It is much more likely to become stationary. But it must be noted this does not hold good in children. In children, unfortunately, the outlook is grave in the extreme, because in these young subjects there is great liability to a pancarditis, myocardium and pericardium both being involved, with close adhesions. The young cardiac muscle, cannot meet the strain thus imposed upon it, and very quickly gives up the struggle.

In adults the rheumatic cases respond very well as a rule, to treatment and rest, with later, graduated exercises to tone up the heart muscle, and in time the individual can carry on a fairly active life with care.

If the lesion be due to syphilis, syphilitic arterio-sclerotic changes, our prognosis must be guarded. Although on the whole, the type of case is more amenable to alleviation, there is great risk always of sudden death from myocardial failure owing to invasion of the muscle by the luetic poison.

Whatever /
Whatever be the causal factor the outlook is enhanced, if the individual can be placed in the best possible surroundings, both as regards occupation and rest, with a complete freedom from worry, a therapeutic Utopia difficult of attainment. Cases may remain fully compensated for years, and the individuals do not suffer any great inconvenience. If the cardiac hypertrophy equalises the valvular defects, no urgent or untoward symptoms may arise, even though moderately heavy physical exertion be undertaken.

In making our prognosis in this condition, there are certain very important factors to be taken into consideration, and they must, individually and collectively, be most carefully investigated, in order to arrive at a true conception of the case.

I. THE PULSE, AND ITS CONDITION. This is undoubtedly our surest guide to the condition of affairs if other signs of cardiac failure be wanting. The points for which one looks are alterations in its rate and its rhythm. If the rate should go up to a point above 90, and remain there, then danger threatens; but the outlook is better if the rate remains anywhere around 70 - 80. We must remember however, that in this condition, patients are apt to be subjects of a nervous tachycardia, and therefore before /
Electrocardiogram - showing Rt. Ventricular Extrasystoles complicating Aortic Regurgitation.

Dec. 8th, 19—

Feet and legs show marked improvement.

Outlook is good.

The patient was discharged on Dec. 10th with instructions to take a change of climate and food, and to return in January.
before coming to a conclusion on this head - repeated examinations of the pulse should be made, counts being made at all hours of the day, and even during the patient's sleep.

Any alteration of the rhythm should also be regarded with apprehension, because these alterations are all indicative of myocardial damage.

The most important alterations we may meet with in their order of frequency of occurrence are (a) Extra Systoles (b) Auricular Fibrillation (c) Sirio-auricular heart block (d) Pulsus Alternans. The order of their gravity is exactly reversed, Pulsus Alternans being the gravest whilst Extra systoles are the least grave.

(a) Extra Systoles. These are, as I have said, the most frequent irregularity in Aortic Regurgitation. If they are of left ventricular origin, which can be determined readily by means of the electrocardiograph, they are indicative of fatigue of the left ventricle. This, as may be imagined is of grave significance, because the left ventricle is the main compensatory chamber.

The extra systoles are very apparent both to physician and patient. To the former, the type of pulse, character of the apex beat, and the auscultating sounds make them manifest. Whilst to the patient himself they occur as sudden stoppage of the heart - "flopping" of the heart etc.

Extra systoles are now definitely understood to be a sign of myocardial hyper-irritability. When
Electrocardiogram - showing Atrial Fibrillation complicating Aortic Regurgitation.
When they occur they produce sudden increased intravascular strain, and therefore the heart cannot withstand them for any length of time, without serious results following.

(b) AURICULAR FIBRILLATION.

This condition, now well known, is more commonly associated with Mitral Stenosis, but it may occur in Aortic Regurgitation, under two conditions, (1) when the aortic lesion is of a sclerosing type, with associated general myocardial damage. (2) When the Aortic lesion is secondary to an associated Mitral lesion.

The musculature of the auricle gets into an agitated condition, through some unknown source of stimulation arising at innumerable foci, and the more fibrillation there is the graver is the import.

(c) HEART BLOCK.

This takes the form of $S_{70}$-auricular Block, where the whole heart remains silent periodically and is/course indicative of impairment of conductivity.
Electrocardiogram - Showing Right Bundle lesion, complicating Aortic Regurgitation.
(d) PULSUS ALTERNANS.

This is the gravest form of pulse irregularity and its presence in a case of Aortic Regurgitation is of the gravest significance, because it is a certain sign of the failure of the myocardium to respond to stimuli especially in the ventricle - and is a prelude to myocardial failure.

These are the chief points in the pulse - to which attention must be given, in making a prognosis, and I would point out - the more collapsing the pulse is, the greater is the damage to the valve.

II. BLOOD PRESSURE.

This, after the pulse, is the most important guide in our prognosis in aortic regurgitation.

If the systolic pressure be maintained at a fairly high figure - say 180 mm. of Hg, and if the diastolic pressure bears to it a relation similar to that in health, then the outlook will be good.

As I indicated before the blood pressure should always be taken in arm and leg in aortic incompetence because the relation between the two pressures is an excellent guide for us as to the progress or otherwise of the case. It has been found that, in the healthy normal individual the blood pressure in the arm or leg are nearly equal or more frequently the pressure /
pressure in the leg is less than that in the arm. Now in aortic incompetence it is otherwise, the pressure in the leg is always greater than that in the arm, and the greater the difference between them, the graver the outlook.

If we have, then, a systolic pressure in the arm which shows a pronounced tendency to fall, and which is not increased by any exercise, and if this arm pressure be divorced from the diastolic arm pressure, and also from the systolic leg pressure then the case has passed into the region of myocardial failure and therefore the outlook is bad in the extreme.

III. Now I come to a point on which I would lay great emphasis, as being one of great importance, when we make our prognosis. I refer to that of VASOMOTOR TONE. It is one which seems to be almost neglected by many, and yet it is a most important guide for us.

We have often seen cases of aortic regurgitation who have died suddenly, after retiring from an occupation which has involved a certain amount of physical effort. Why should this be so?

In aortic incompetence, the vasomotor system is in a state of great instability, and is extremely hypersensitive, and unless we keep up the tone and prevent muscle fatigue by suitable treatment, we shall get a collapse and sudden death. Muscular exertion supplies the necessary stimulation to keep
up the blood pressure, and supply the heart with the necessary amount of blood during diastole, which in this way maintains a certain degree of tone in the vasomotor system. Should anything then occur, which interferes with the maintenance of the blood pressure at a proper level, and with the proper supply of blood to the heart, the vasomotor tone falls, fatigue sets in, and if this be extreme syncope ensues.

This vasomotor fatigue explains also—why we get the history of "fainting turns" in so many cases of aortic regurgitation. These attacks are not due as one might suppose—to any failure of the myocardium itself but are entirely due to excessive vasomotor fatigue.

IV. MYOCARDIAL CHANGES.

The myocardium demands special attention and care for even valvular lesion is loss of myocardial tone more dangerous, and more difficult to treat than in aortic incompetence.

A great deal of stress used to be laid on the amount of ventricular hypertrophy as a factor in prognosis in this condition, but investigation, not yet completed, tend to show that this hypertrophy is not entirely due to the mechanical response to the regurgitation, but that certain other factors are involved. We cannot gauge with any certainty the degree of incompetence by the amount of
of ventricular hypertrophy.

Any anasarca arising in this condition cannot be attributed to myocardial degeneration or failure entirely. If the oedema does occur, then we must look for some other lesion in the heart, or outside of it - in kidney or liver - as being the cause.

V. Finally there are two symptoms, in addition to those due to cardiac failure, the persistence of which must give rise to a good deal of apprehension in our prognosis. I refer to PAIN and INSOMNIA.

It is astonishing how many people come complaining of these two symptoms alone, in aortic incompetence, and it is not surprising when we consider that they are both due to myocardial degeneration. Pain is not an invariable symptom though a very frequent one in this lesion. In the hundred cases I examined I found that 77 had had "pain in the chest" as one of their symptoms, and that 45 came for advice because of the same kind of pain. Whilst in many cases it is undoubtedly a sign of myocardial degeneration, pain in the praecordia may sometimes be caused by a very sudden rise in the blood pressure or of the balance of an already badly balanced vasomotor system. Insomnia is due to a combined cardiovascular and nervous disturbance.

The myocardial weakness, results in weak contractions and /
and thus the nerve cells in the brain are deprived of their sustenance and a state of irritable weakness results.

If the insomnia is allowed to persist for any length of time, it becomes indeed a sign of grave significance inasmuch as the individual becomes exhausted, both physically and mentally especially the latter. This mental exhaustion results in the melancholia and suicidal tendencies already referred to, with eventually dire consequences.

Angina is of grave significance, both from the point of view of the blockage of the coronary arteries, and from that of danger from inhibition of the vagus, a very common sequel.

These then are the main points to be taken into consideration in making a prognosis in aortic incompetence. There are many others of lesser import, but all the same they require careful consideration. These are, the patient's age, his surroundings both at home and at work, his mode of life, past and present; his former illnesses and powers of recuperation.

SECONDARY AORTIC REGURGITATION.

The prognosis in this condition can be summed up in two words - absolutely hopeless. It is, here, indeed, merely a question of time. As I have indicated /
indicated, the individuals present few or no signs or symptoms until a very short time before the end. Prognosis may be made more difficult, because there may be a few periods of remission, which, however, do not last for long, nor does each remission last as long as its predecessor. Text books, when they deal with the matter at all, give a time limit of two years, but those cases which have come under my notice have died within a much shorter period after seeking advice - a few months perhaps. We are working in the dark, as it were, as we cannot get at the seat of infection; the type of infection is not known until too late to avail ourselves of vaccine therapy. In short these subacute infectious endocarditic cases must be classed as the absolutely rapidly fatal cases of aortic regurgitation.

THE TREATMENT of AORTIC REGURGITATION.

In discussing the treatment of aortic regurgitation, I do not propose to deal with the prophylactic side of the question in so far as it applies to cases of acute rheumatic fever. The measure to be adopted in such cases are many and varied. But I want to deal with the procedure to be adopted when a patient comes with a well established lesion.

The first step is, of course, a thorough investigation into the probable cause. If this is found to be specific, then anti specific measures must be adopted along with the remedial measures to be de-
detailed later. Similarly with rheumatic cases, anti-rheumatic measures should be adopted.

In deciding upon any course of treatment in aortic lesions - or indeed in any other cardiac lesion, our first care must be to ascertain the state of the myocardium. For if the myocardium be undamaged then our objective is to make the individuals life a fairly useful one - both to himself and his dependents, but if on the other hand, the myocardium be damaged then our efforts are directed towards prevention of sudden death - a vastly different and more difficult objective. The easiest and simplest way of ascertaining the condition of the myocardium is by means of (1) Myocardial Efficiency Tests. These have been clearly defined by Strickland Goodall in his work on the subject. Briefly they consist in making the individual perform certain exercises, going upstairs a certain number of times, etc., estimating the amount of foot-pounds of work done - and carefully comparing pulse - respiration, blood pressure before and after the exercises.

(2) The electrocardiograph.

The electrocardiograph does not, of course, delineate the exact lesion present, but it gives us an excellent picture of the condition of the various parts of the cardiac mechanism - as a result of any one lesion.

These two preliminary investigations completed comes /
comes the key note of successful treatment of aortic regurgitation with intact myocardium. The patient should be seen very frequently, certainly not less frequently than once in ten days, and at each consultation the pulse rate should be carefully taken, and the question asked, "How are you sleeping"? Any alteration in pulse rate, any change in sleeping in the directions indicated before, are for the worse. An individual with increasing pulse rate and increasing insomnia is going the wrong way.

If we have a case of aortic regurgitation with a healthy myocardium, our efforts must be directed to the prevention of vassomotor fatigue. All the individuals muscular efforts should of course be within his cardiac ability, and should be directed towards a definite purpose. There is nothing so conducive to vassomotor fatigue - with the attendant danger of sudden death - than aimless "pottering" about. The rest of such an individual should be thorough, ten or twelve hours per noctem, and if there be any tendency to insomnia prompt remedial measure. Morphia and its derivatives should be avoided, because it has been found frequently, that any effort on the patients part, say sitting up in bed when the effects of the drug are in the "passing off" stage, may be attended with fatal consequences. The drugs which are safest to employ are Bromides and chloralamid, a combination of the two, ten to twenty grains of each, according to individual needs being a very suitable and safe remedy. The individual must be warned against doing anything which will
will involve a sudden and excessive strain on his heart, and he and his relatives should be warned of the danger of sudden nervous and emotional shocks. Needless to say, the daily life—food and habits if need be, must be most carefully regulated to each individual case.

Any alteration in pulse rate and increasing insomnia must be met with promptly and efficiently. For the pulse rate increase, complete rest in bed is absolutely imperative. This increased pulse rate is due to one cause and one cause only, namely, myocardial irritability, a sure forerunner of myocardial failure. Partial rest and restricted movement may be all very well for reducing myocardial pain and oppression in slight degrees. But here nothing short of absolute rest will do. This should be accompanied by the exhibition of digitalis in some form—the tincture 10 minim thrice daily being perhaps the best. Its effects should be carefully watched and if a fall in the pulse rate occurs the quantity should be reduced by one dose per diem. If, however, no effect takes place on the pulse rate after a week or ten days administration, then the quantity should be cut down by half or more, and the small does /
dose given systematically, because, we now know that digitalis has a most beneficial tonic action on cardiac muscle. The combination of rest and digitalis has often a most magical effect on the pulse in these cases.

As regards acute specific measures, the combination of iodides with inunction of mercurial ointment will do best. The modern arsenical preparations, for some unknown reason, do not act so well in these cases.

Such, then, are a few clinical observations on Aortic Regurgitation, embodying the most modern views on the subject. It will be noted that I have said nothing regarding congenital aortic incompetence. Cases are rare, and when they do occur, the individuals rarely
rarely survive more than a few days, or at most a few months. The outlook is bad in the extreme, and very little can be done for them, except as a well known Edinburgh Professor used to say in similar circumstances, "Treat them, gentlemen, secundem artem."

I shall now pass to the analysis of one hundred cases of aortic regurgitation, whose records I have examined for this thesis. Before I do so, I should mention they were all patients of Dr J. Strickland-Goodall at the National Heart Hospital London, to whom I wish to tender my grateful thanks for his kindness and courtesy in placing the material at my disposal.

The cases were taken at random, and an analysing them, I shall endeavour to bring out the points embodied in the former part of this treatise.

Analysis of ONE HUNDRED CASES OF AORTIC REGURGITATION.

SEX INCIDENCE:

Of these hundred cases, 87 were males and 13 females, shewing a proportion corresponding most remarkably close to Lockhart Gillespie's figures mentioned at the beginning of this treatise - namely seven to one.

AGE INCIDENCE.

<table>
<thead>
<tr>
<th>MALE</th>
<th>Females</th>
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<tbody>
<tr>
<td>Between 10 &amp; 15 years old - 4 cases:</td>
<td>0 cases</td>
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<tr>
<td>&quot; 16 &amp; 20 &quot;</td>
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<td>&quot; 21 &amp; 30 &quot;</td>
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<td>&quot; 31 &amp; 40 &quot;</td>
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<td>&quot; 41 &amp; 50 &quot;</td>
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<td>&quot; 51 &amp; 60 &quot;</td>
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<td>&quot; 61 &amp; over &quot;</td>
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<tr>
<td><strong>87</strong></td>
<td><strong>13</strong></td>
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These
These figures are also approximate to those quoted in the beginning, and they show that the condition of Aortic Regurgitation is, manifestly, a disease of the prime of life, from 25 to 50 years of age, in both sexes. It is interesting to see the fall that takes place after the age of 40, which is undoubtedly accounted for, by the fact that rheumatism, the great casual factor of this lesion, is a disease of early life, not being common after the age of 40.

PREVIOUS ILLNESSES:

Turning now to the previous illnesses from which the hundred suffered, and classifying them — as they are classified at the Heart Hospital, we get some most interesting figures.

Specific Fevers (Scarlet, Measles Typhus, Typhoid) - 27

Rheumatic Fever (Chorea - 9) 44
Rheumatism (Tonsillitis - 18) - 43
(Growing Pains - 16)

Diphtheria - 5
Influenza - 42
Syphilis - 14
Traumatic or Strain - 2
No previous illnesses - 9

These figures shew very clearly, indeed, the great place rheumatism in some form takes in the causation of this grave lesion. No less than 87 cases giving a history of it.

Treading closely on the heels of rheumatism we have influenza with 42 cases, but, though given as "influenza", the probabilities are that many were mild cases /
cases of rheumatism. This is the more evident when I enquired more closely into the matter because the previous disease, with which influenza was most frequently coupled was rheumatism.

The specific fevers come next with 27 cases either alone or accompanied by some of the conditions. Of the specific fevers, Scarlet fever preponderated. It is interesting to note, in estimating the part scarlet fever plays in the causation of cardiac lesions generally, that, in many cases, patients give a history of a sudden fibrile attack following the scarlet fever proper, which they describe as being very similar to rheumatic fever itself. This is a well known fact, and should be borne in mind.

After the specific fevers in point of numbers comes Syphilis in 14 histories. This includes all those cases where a luetic history was denied, but where a positive Wasserman was found on investigation.

Two cases gave no definite history of any antecedent illness classified above, but on investigation, one case, a milkman aged 62, gave a history of a fall with a heavy milk can on his chest some six or seven years previously, from which he dated his troubles. Undoubtedly, a traumatic case. The other was a clerk, who had Malaria twice in Mesopotamia.

URGENT SYMPTOMS:

If we examine these: and by "urgent symptoms", I mean those symptoms which compelled the sufferer to seek advice, we find as follows:-
45 cases - pain in chest, either alone or with another symptom.
51 cases - shortness of breath, ditto.
16 cases - giddiness ditto.
2 cases - headache ditto.
26 cases - palpitation ditto.
3 cases - dyspepsia ditto.
7 cases - fainting attacks ditto.
5 cases - Tiredness or weariness ditto.
1 case - Tachycardia.
2 cases - "heart missed beats."
3 cases - Insomnia accompanying other symptoms.
3 cases - Oedema of legs accompanying other symptoms.
2 cases - Exhaustion.
2 cases - Nervousness.
1 case - Oppression and lightness in chest.
1 case - pain in legs only (boy of 12 years of age.)
3 cases - Nothing definite. "Out of sorts" "not feeling well," "Not up to the mark."

From the formidable list the favourite combination of symptom was pain and shortness of breath in 19 of the 100 cases, next in order being shortness of breath and palpitation in 13 cases, pracordial pain alone, 14 cases. Tiredness occurred in five cases, and its close allied friend, physical exhaustion in two. Certain observers point to these two symptoms as being very suggestive of aortic regurgitation, but I have not found it so in this instance, nor in any of the patients attending the clinic.

On questioning the patients as to their symptoms from time to time since they began to feel out of sorts, the following interesting table was made:

Cough /
Cough - 34 cases.
Dyspnoea - 95 "
Fainting - 32 "
Vertigo - 60 "
Palpitation - 82 "
Pain - 77 "
Insomnia - 47 "
Dueresis - 30 "

In this table as in the one immediately preceding, dyspnoea, palpitation and pain are the prominent features, as we would expect. Insomnia, that distressing symptom, and one of grave import, was given in 47 instances, whilst fainting, that sure sign of vasomotor fatigue figured in 22 cases, and its very close neighbour vertigo in 60.

THE OCCUPATIONS were very interesting study. Of the 87 males, 36 had no occupation, out of work, and of these 36, 33 were ex soldiers or sailors, who had at one time or another suffered from rheumatism in some of its forms; syphilis or influenza, men who had undergone great hardships in trenches or at sea. The class contributing the next largest number was curiously enough, clerks, a class of men with a more or less sedentary occupation. Ten of these suffered from the lesion. Investigation, however, shewed that seven had been in the army previously, and the other three had had rheumatic fever at some antecedent time.

The other male occupations comprised many classes from engineers and stevedores men with hard lives to laboratory assistants and dental students.
The female occupations were confined to housewife duties (eleven cases) and two dressmakers. Dressmaking can hardly be classed as a labouring occupation, yet one of the worst cases of the 100 was a dressmaker. Another very bad female case was one who had done very heavy munition work during the war, following a severe attack of rheumatic fever, and since the munition work influenza, obviously a strain on a damaged myocardium.

OTHER VALVULAR LESIONS ACCOMPANYING the Aortic incompetence:

- Aortic Stenosis - three cases.
- Mitral incompetence - sixteen cases.
- Mitral Stenosis - ten cases.
- Double Mitral - five cases.

All of these extra valvular lesions developed subsequent to the aortic regurgitation with the possible exception of these cases of double mitral lesion.

BLOOD PRESSURE:

These were carefully recorded, both from arm and from leg, and showed a very wide range. The lowest systolic pressure in the arm was 125 mm of Hg. with a diastolic of 70. The highest arm recorded was 230 mm of Hg. as the systolic pressure, with a diastolic of 100.

In the leg the lowest systolic pressure was 135 with a diastolic of 80, and the highest (recorded in the same individual with the highest arm pressure) was 250 mm of Hg. and the diastolic 120.

The average systolic arm pressure was about 160 mm, the average systolic leg pressure was 180.

ELECTROCARDIOGRAMS.
ELECTROCARDIOGRAMS:

As I have pointed out before, the electrocardiogram affords us most valuable information, not only in Aortic Regurgitation, but in all other cardiac affections, for it gives us a graphic record indicative of the functional state of the heart muscles, and clearly shows the changes which have occurred in the musculative, not only as the result of any original infection, or distinctive process, but also of the compensatory changes which have resulted from the valve lesions.

I was fortunate in securing 116 electrocardiograms for 85 of the hundred cases, and the analysis of these is particularly interesting.

The most constant electrocardiographic change met with in the series was some modification in the ventricular complex, only twelve of the whole series could be regarded as more or less normal. If we consider where falls the greatest strain in Aortic Regurgitation, which part of the heart is most affected, we can see why this should be so. Now if one proceeds further - and analysis these defects, this striking feature presents itself, namely: - that the 2nd ventricular complex - the T. wave, was most affected, and that the commonest variation or aberration in the complex was delay or inversion, the actual figures being 34 delayed T. waves and 34 inversion in some form or other. Only eleven showed an approximately normal wave.
If the inversion be taken and analysed, we find

Inverted in leads $\text{II}$ & $\text{III}$ - 10 cases

" " " $\text{I}$ & $\text{II}$ - 1 case.

... " lead $\text{III}$ - 15 cases.

" " " $\text{I}$ - 1 case

whilst three cases showed inversion in all three leads as the illness proceeded.

What exactly is the significance of these aberrations of the T. wave? At present much difference of opinion exists on this point, but Strickland Goodall whose experience in electrocardiography is very great, points out that the T wave in the most conspicuous part of the electrocardiogram of the young healthy adult, and that as age advances, the wave diminishes in proportion. The wave is most markedly increased by exercise when the heart is healthy. Now if we consider the sequence of events in contraction of the heart, it is very evident that the T wave must represent the last part of the ventricle to contract, and especially so after any stress or strain. Gotch, Goodall and others, think that in lead $\text{II}$ at any rate, the T wave is indicative of activity in the region of the Aortic Base. Figures from the Mayo clinic support conducively Goodall's finding that persons with defective T waves are liable to sudden and early death.

Therefore /
Therefore, in view of these figures, and the work and evidence of these authorities, it can, I think, be rightly assumed; (1) that any delay in the T wave must mean some impairment in the conductivity of the myocardium in the region of the Aortic Base; (2) Any diminution of altitude of the wave indicates impaired autoactivity or tone; (3) An inverted or absent T wave means definite myocardial changes in that region which is the main area of distribution of the left coronary artery. Therefore, no matter from which point of view we approach the T wave, we have in it a most valuable help in making a prognosis in all cardiac condition.

The First Ventricular Complex. The QRS wave was approximately physiological in 75 cases and aberrant in 10; and the feature of them was the constant alteration of the R wave, in the form of a notch or split.

Next in importance to the abnormalities of the 2nd ventricular complex, a constant feature of the series was a marked left sided preponderance 49 cases showing this whilst only seven showed a right-sided preponderance.

AURICULAR COMPLEX or P wave was normal in about 42 cases. It was flattened in eleven, delayed in fifteen, increased in eight, inverted in five, flat and inverted in fifteen, and the waves were abnormally small in three.
EXTRA SYSTOLES were mostly of left ventricular origin, and, as I have previously indicated, they are suggestive of left ventricular fatigue.

BUNDLE LESIONS.

These were present in four cases, and were indicative of obvious extensive damage to the ventricular myocardium.

The P.R. interval was increased in five cases showing that there was damage in the main bundles as well as in the branches.

This is a short analysis of the hundred cases whose records I examined, and I put it forward in support of the theories I have advanced in the earlier part of the thesis and they lead me to form the following conclusions:

1. Aortic Regurgitation is due in the majority of cases to rheumatic infection at an early period of life; through syphilis, influenza, and the specific fevers, especially scarlet fever, are potent causal factors.

2. It is commonest in males from 21 to 40 years of age.

3. The signs and symptoms are many and varied, though the constant one is the "water hammer" or Corrigan pulse.

4. /
4. The prognosis is always grave, and depends on:

(I) The nature of the lesion, whether it be rheumatic with a tendency therefore to quiescence or a sclerosing or progressive lesion.

(II) The extent of the valve lesion, e.g., a localised cusp lesion causes little disturbance, therefore the outlook is fairly good. On the other hand, extensive lesion of all cusps with increasing ventricular strain is very bad.

(III) The extent of myocardial damage to be estimated by testing the liability of the heart muscle to fatigue, and by the electrocardiograph.

(IV) The efficiency of the vaso motor compensation.

In connection with III. and IV. we must remember more cases of aortic regurgitation die from one or other of these things myocardial failure or vaso-motor fatigue. Of course in addition one must take into consideration certain general considerations—such as the patient's age; his or her occupation, the liability to secondary infection; general surroundings both at home and at work.

5. The treatment consists mainly of rest and complete avoidance of any physical or mental fatigue or emotion shocks. Vasomotor fatigue to be particularly /
particularly avoided. All exercise etc. to be done with a definite object in view. Tachy cardia to be treated by rest and digitalis until rate approaches normal again. Above all constant and frequent supervision of the case and examination at least weekly. Insomnia to be treated at once or appearance.

6. If the myocardium be involved to any degree - the risk of sudden death is greatly increased, and a careful watch must be preserved.

7. Subacute infective Endocarditis, is a hopeless variety. The patient at first presents few or no symptoms, but the course is rapid, with all the signs of an acute general toxaemia, and death ensues within a few months of the onset.

8. Congenital aortic regurgitation, rarely survives birth more than a few days or weeks. When it does, all the signs and symptoms of the acquired variety are present but much aggravated.
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