

CARDIAC PULMONARY INCOMPETENCE:

With Special Reference to Difficulties of Diagnosis.

A Thesis for the Degree of

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E d i n b u r g h.

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In presenting a consideration of this subject in the form of a Thesis for the degree of M.D. I am recording my observations in a few unique cases of Pulmonary Incompetence, which for some weeks have been under my daily notice at the Edinburgh Royal Infirmary.

In the following pages it is my intention to consider the subject generally, and then to refer to the difficulties that present themselves in the diagnosis of this interesting cardiac affection.

In general practice, in which I have been engaged for the past six years, my attention has frequently been directed to a bruit occurring at the base of the heart and where, after a consideration of all the symptoms present, one was in doubt as to whether it was produced at the Aortic or Pulmonary orifice.

Considering the comparative rarity of lesions of the right side of the heart to those of the left it is not often that cases present themselves illustrative of this affection.

In the following pages I beg to record two cases of Pulmonary insufficiency where the signs and symptoms present are compared with those of a case of functional Aortic reflux, and from a consideration of these I hope to record, from a clinical standpoint, the points of

interest as well as the difficulties that occur in its diagnosis. In the general routine of a busy practice the pulmonary area is possibly not looked at with the same practical interest as are the other orifices of the heart, and so one is probably apt to dismiss its lesions with lessened consideration.

It is undoubtedly true that the left side of the heart is far more liable to valvular affections than the right side, and so in practice the great majority of cases of cardiac disease that call for treatment, present lesions of the mitral or Aortic valves.

Nevertheless the affections of the pulmonary orifice although of much less frequency are none the less interesting, often none the less grave, and accordingly merit attention.

Historical.

This subject has been given much attention by many writers and observers not only in Britain but also in America and on the continent.

The earliest, I find, who has dealt with it is Chevers⁽¹⁾ in his articles on the affections of the Pulmonary artery.

He has recorded numerous cases, many of which however are only interesting from a congenital point of view. From his time onward one meets with such names as Whitley⁽²⁾, Stokes⁽¹⁰⁾, Hope⁽⁹⁾, Duckworth⁽⁵⁾ more recently with many other writers in this country and abroad, whose views we shall consider later.

I shall endeavour to deal with:-

1. A few anatomical and physiological points relevant to the Pulmonary Artery and Aorta.
2. A consideration of the Etiology, Morbid Anatomy and symptoms of the disease.
3. A record of the cases under my observation.
4. Special reference to the difficulties of Diagnosis.
5. Other remarks, photographs, tracings, etc.

Section 1.

Let me recall very shortly some anatomical facts which bear upon this subject. I shall confine these

only to a short comparison of the build of the Pulmonary artery and the Aorta.

The two openings resemble each other in being guarded by valves having three cusps, triangular in shape and attached by their base to the wall of the vessel their free margins meeting to close the orifice against the column of blood in the arteries.

In the case of the Pulmonary artery two of the cusps are anterior and one posterior, the opposite being the arrangement in the Aorta. The cusps are strengthened by bands along their margins, forming the lunules. Anatomically, so far, their structure is the same.

When, however, we come to compare them as to their relative strengths and the thickness of their walls we find they differ.

The structure of the minute anatomy of the Pulmonary Artery does not in the main differ from that of the Aorta. Still in many respects it is a vessel "sui generis."

Its resemblances to veins, in that it has thin walls compared to the Aorta and in that it is more elastic and dilatable is to be explained by the functions it has to perform. These duties are at once seen to be much less onerous than are those of the

Aorta. In the first place the Pulmonary blood circle is one which offers much less resistance than does the systemic circle. In the next place it is to be noted that the Pulmonary circulation is much aided by the suction action of the movements of the lungs, and so it is not necessary that the Pulmonary Artery should be so strong in its walls as the Aorta is.

In view of these facts we consequently find that the right ventricle has walls much thinner than the left ventricle. Nevertheless, the blood is projected with considerable force into the comparatively thin-walled Pulmonary Artery and it would undoubtedly soon become permanently damaged were it not owing to its ready dilatability and its power to resume its former capacity after temporary dilatation - a state of things which occurs in certain Pulmonary and Cardiac affections. In connection with this property of physiological dilatability in these vessels it is probable that while the expansion in the Pulmonary Artery is of a lateral kind, that of the Aorta, on account of the greater resistance present in the systemic circulation may be more in its line of axis.

In the succeeding pages I shall have to refer to a stretching of the Pulmonary artery and orifice from an increased blood pressure in the Pulmonary circle,

and also a similar condition at the Aortic orifice (although much rarer) in which the blood pressure evidently plays an important part.

This question therefore is of importance in the above connection.

With regard to the size of the Pulmonary and aortic orifices, and thickness of cardiac walls

(6)
Woodhead gives:-

Walls.

Aortic .9-1 inch

Left Ventricle $\frac{1}{2}$ - $\frac{3}{4}$ inch

Pulmonary 1.1-1.2 inch

Right Ventricle $\frac{1}{2}$ inch

Landmarks.

The relative positions of the Pulmonary artery and Aorta to one another and the position of their valves on the chest wall are important in their examination, and I will refer to them shortly.

(11)

In Cunningham's Text-Book of Anatomy I find "That the Pulmonary artery lies behind the left⁺ border of the Sternum, opposite the second interspace and second costal Cartilage, whereas the Aorta lies behind the Sternum opposite the second and third ribs and unless dilated does not project beyond the right border: the upper border of the Aortic arch lying at or a little above the centre of the manubrium sterni."

Also, "That the Pulmonary orifice directed upward and slightly backward and to the left lies opposite the upper part of the third left Chondro-Sternal junction, whereas the Aortic orifice directed upward and backward to the right lies further from the surface behind the left half of the sternum opposite the lower border of the third costal cartilage."

The point of nearest approximation of the Pulmonary artery to the chest wall is the second left interspace and the second right costal cartilage in the

case of the aorta. The above are also the Pulmonary and Aortic areas where the sounds produced by the respective valves are best heard. 9.

I may just mention that systolic Aortic and Pulmonary bruits are usually heard best in the Aortic and Pulmonary area, the former being propagated with the blood streams.

It is worthy of note that although Aortic diastolic murmurs are usually best heard in the Aortic area, they are not unfrequently most audible to the left of the sternum in the Pulmonary area, and even a space lower. They may be also audible only at the apex. Aortic diastolic bruits are propagated to the lower end of the sternum and might be more so to the left, a Pulmonary diastolic more to the right. The latter are generally localised to the Pulmonary Area.

The Etiology.Section 11.

The causes at work in the production of Pulmonary Incompetence are evidently somewhat varied when compared with the etiology of other Heart lesions.

Many of the earlier cases which have been described by Whitley⁽¹⁾ and Chevers⁽²⁾ were, some of them at any rate, only discovered after death.

It is not intended in this paper to consider the cases of a congenital character which contribute a large proportion of the diseased conditions of the right side of the heart: but those cases due to actual involvement of the valves and also those cases where the Pulmonary orifice is affected secondarily as regards its competency.

I therefore propose to divide cases of Pulmonary Incompetence so far as their Etiology is concerned into two large groups.

1. Those arising from Congenital Malformation.
11. Those manifesting actual disease acquired during life.

Under group 1, would come to be considered such causes as Abnormalities in the number of cusps, patent ductus arteriosus, narrowing of the Pulmonary orifice

etc. which are not within the scope of this thesis.

II The Second Class I have divided into two groups:-

1. Those displaying valvular disease caused by Rheumatism and Acute Infective processes.

11. Those cases of Pulmonary Incompetence which are secondary to other cardiac or Pulmonary lesions present, and where the valves are not necessarily diseased.

Under this head would fall those cases described as Relative or Functional.

Class 1.

From a study of the literature on the subject I find that Endocarditis of the Pulmonary valve from whatever cause is not of common occurrence. It is even seldom found affected where the other valves of the heart show an Endocarditis.

In the great majority of cases the cause is undoubtedly some acute infective process such as the specific fevers, pyoemia or saproemia, or Rheumatism.

Of the three cases recorded by Whitley one only⁽²⁾ had a history of Rheumatism. In one there is a history of traumatism. In a large number of reported cases where disease of the Pulmonary valve was suspected and was found to be present, no cause had been assigned. In one case which I shall record later

I am of opinion that the Pulmonary valves are damaged and incompetent. In this case there is a well marked history of rheumatic attacks.

The Etiological factors in this disease may thus be tabulated.

1. Damage to the valves (Causing vegetations) from some congenital condition of the Right heart,
2. Septic ouerperal conditions.
3. Pyoemia.
4. Septic Phneunonia.
5. Gonorrhrea.
6. Rheumatism.

A history of Rheumatism is evidently not obtainable in very many cases and it is probable that acute infective processes as detailed above are as important a factor in its causation as is Rheumatism.

Period of Greatest Frequency.

With respect, however, to those rare cases of Incompetence caused by an endocarditis the period seems to be early and middle life rather than later life.

(8)
Barie gives the age period of 13 - 35 as that of greatest incidence, and Pitt⁽⁷⁾ in his series of cases collected from Guy's Hospital Reports puts the greatest number of cases as occurring between the ages of 21 & 30.

These two observations therefore correspond somewhat closely.

Roughly speaking it may be taken that about 86-90 % of cases occur under the age of 50 years, and about 59-60 % under 30.

In searching the literature on the subject one observes its non-occurrence during the very early years of life: that is, apart from the congenital cases at that period. The conditions, no doubt, which give rise to it determine its occurrence at a later period of life. The two sexes are about equally affected. (Barie)⁽³⁾

Class 11.

I come next to Secondary or Relative Pulmonary Incompetence.

This variety has been described as occurring where there was a high blood pressure in the Pulmonary Circulation from whatever cause. Probably the one most commonly seen is that in association with mitral stenosis where, towards the later stages of the malady a leakage at the Pulmonary orifice occurs.

I have had under observation for many weeks lately an interesting case of this kind and have recorded it fully later on in these pages.

This incompetency of the valves is often associated with a dilated condition of the vessel beyond them

The other class of cases predisposing to this reflux are morbid conditions of the lungs themselves which increase the resistance to the flow of blood through them: such as Fibroid and emphysematous involvement and probably also long standing bronchitis etc. With regard to this stretching of the orifice (which shall be more fully referred to later) I believe it possible that such may occur, the conditions for its occurrence being present, and yet no evidence of it be seen post-mortem.

(12)

Adams even puts it as a natural provision of nature in saving the weaker structures of the Right Heart in such cases.

It is also very probable that a somewhat similar stretching of the Aortic orifice can take place without any organic disease of the valves.

I shall also detail a case later where this was possibly present.

The important point, therefore, in the causation of such cases seems to be an increased resistance in blood current beyond the orifice, and occurring in such cases as mentioned above.


The majority of cases of this description may, I believe, give no signs of dilataion or incompetency after death, although one may be perfectly satisfied from

signs before death of its presence.

This, I think, may be accounted for by the Pulmonary artery and orifice being capable of a considerable degree of dilatation and yet of recovering to a point which after death would give little or no evidence of dilatation having existed.

Under this head may be mentioned as a cause of Pulmonary reflux any tumour pressing on the Pulmonary artery from without, such as an aortic aneurism arising from the first part of the arch. In such a case the pressure gradually causes distortion and stretching of the valves, with resulting incompetence.

The periods of occurrence in this form is necessarily that of the conditions of the heart and lungs to which it is secondary.



Morbid Anatomy.

In considering a few of the Pathological changes found I shall first refer to Group 1.

In such cases one generally has signs of an Endocarditis either of an acute or chronic kind and variable in position and degree. Vegetations may be found on the valves themselves, which may also shew some degeneration and thickening. The changes in the valves sometimes indicate old endocarditic processes in that they may be found thickened and puckered: consequently rendering the valves incompetent.

Cases are on record where large parts of the valves were absent from disintegration. A considerable number of the recorded cases which I have looked into while shewing vegetations on the Pulmonary valves, presented at the same time lesions in a greater or less degree at other valves: notably the mitral and aortic.

It seems, however, possible to have the condition confined to the Pulmonary orifice. The tricuspid may also be more or less affected. The vegetations may be of a gross character as in a case reported by Whitley⁽²⁾ where there were found vegetations on the ventricular side of the valves of large size. The valves may shew perforations through their substance where the

process has been of an extreme kind.

The Artery beyond the valves is also sometimes affected and has been found dilated, and with thrombi some little distance into the lung substance. ⁽³⁾ (Yeo) specially in those cases where other heart lesions are a primary cause.

The orifice, where there has been destruction and shrinking of the valves, is as a rule incompetent. The same may be found where the artery beyond is permanently dilated.

There may be a contraction of the orifice and most-ly so in congenital cases.

Where there is evidence of incompetency at the orifice the right ventricular wall is found more or less hypertrophied and the cavity dilated. Where this has been present for some time, and the tricuspid valves have also become incompetent the auricular wall and cavity may shew similar changes.

Where a lesion of the Pulmonary artery alone has been present with consequent marked hypertrophy of the right ventricle, the walls of the left ventricle and aorta have been found to be thinner than normal

⁽²⁾
(Whitley).

Incompetence at the Pulmonary orifice, therefore, from whatever cause, can give rise to a hypertrophy of

the right ventricular wall as extreme as may be met with on the left side of the Heart.

In such cases, fatty degeneration is to be found in the cardiac muscle, and where the effects have through lapse of time passed backward changes will be found in other organs.

11. There are undoubtedly I think cases which, during life present signs of a dilated Pulmonary artery and incompetence of the orifice, and where after death no pathological signs may be present. There may be even no evidence of the valves having been incompetent. Such cases as I have mentioned previously are those which are caused by a high pressure in the Pulmonary artery from the causes before stated.

(1)
Chevers however says that, in cases of old bronchitis and extensive emphysema of the lungs as well as in cases where obstructive lesions exist on the left side of the heart, you may find the Pulmonary artery with its valves more or less dilated, but in addition coarse in structure, irregularly thickened, and opaque. I am of opinion ~~however~~ that, where you get a genuine Pulmonary escape in the ~~course~~ course of a left sided valvular obstruction of not very long standing, you may find post-mortem, in many cases at any rate, little or no evidence of it.

I shall now pass to the

Signs and Symptoms.

These present a somewhat varied picture according to the kind of case under consideration and no doubt also depending on the stage at which it comes under observation.

They are also varied much, I think, by the presence as concomitants of other lesions of the heart or lungs. Under this head I intend to deal shortly with the symptoms as occurring in cases of single and mixed lesions. Later on under "Diagnosis" I shall have reason to refer to views as to the relative values of the physical signs and symptoms in this disease. I shall deal with the physical signs presently.

I believe that a real incompetence of the Pulmonary artery may exist and yet the case may present few signs of it beyond those found on auscultation.

Pulmonary regurgitation, per se, is extremely rare and consequently one has to consider to what extent the symptoms present are attributable to the other lesion or lesions.

This is a real difficulty and is present to some extent in one of my cases. Anyhow in nearly all cases there are present a few fairly constant symptoms, and generally the first to be noticed is:-

Dyspnoea.

This may be present in extreme degree or it may only be noticeable on exertion. At perfect rest in the recumbent position the patient may be quite comfortable, but on the slightest exertion becomes breathless.

One would naturally expect this symptom to be present where there was Pulmonary reflux in any marked degree. In functional incompetence there may be dyspnoea to be accounted for by the other lesions, in which case it might be extreme.

Cyanosis as a rule accompanies the dyspnoea and may be present in any degree from a slight tinge of blueness to a deep lividity. When present it is to be well seen in the lips, lobes of the ears, and the extremities as well as the conjunctivae and mucous membrane of the mouth. The entire skin may partake of this blueness.

Pain is a symptom which may be present. It was present in case A. W. which I have recorded later on. Since he came under my notice I find he has no pain when at rest in bed, but that it is apt to return on exertion. As his is a case where there is more than a Pulmonary lesion it is difficult to know to which it may be attributed.

Such pain seems to vary from a sense of discomfort to that of a shooting character across the upper sternal region. It may be absent altogether, or may radiate.

Secondary Effects.

As the disease progresses the circulation of the organs immediately concerned becomes affected.

1. As a result of the incompetence the walls of the Right ventricle become hypertrophied and the cavity dilated. If it goes on for a lengthened period we get dilatation of the Right Auricle and some hypertrophy of its wall, owing to the tricuspid valve becoming incompetent. Signs of venous engorgement in the Vessels of the neck and also of other organs such as the liver etc. follow.

Respiratory System.

2. Where there is much reflux at the Pulmonary orifice signs of imperfect circulation in the lungs appear and in an aggravated degree if the lesion is associated with obstructive lesions on left side of the heart.

Cough therefore may be present accompanied by expectoration which may become at times haemorrhagic.

"Yeo"⁽³⁾ mentions a case where there was in the presence of a mitral stenosis degenerative changes and dilataion of the Pulmonary artery with thrombus causing infarets in the lung and resulting haemoptysis. The

variations in the pressure of the Pulmonary circulation would predispose to such a symptom.

In such a Pulmonary circulation there will be present other signs, as, areas with moist rales and also of lessened resonance.

In the latest stages where there is also a left sided stenosis, effusion may take place into the pleural cavity. Where there is an extreme case of reflux at the Pulmonary artery alone or complicated as above the whole train of symptoms in abdominal viscera etc. follow on account of the imperfect venous return.

These effects are best seen in a mitral stenosis followed by leakage at the Pulmonary artery, giving way of the tricuspid, and dilatation and hypertrophy of the right heart. The case A.G. is one of this nature.

Viewing such a case as the above we get gradually involved in many of the other systems and I shall just refer to them shortly.

Alimentary.

Gastric disturbances follow of which possibly the most marked is vomiting. This may come on at any time, but is as a rule aggravated by the taking of food. There may be a variable presence of other gastric disturbances such as acidity, water brash, feelings of distention and even pain and discomfort over

the epigastrium - all due no doubt to the disturbed secretion from the engorged stomach wall.

The vomit may be the food swallowed some little time before mixed with mucous and bilious or even
 matter.

sanguous. The secretion is mostly acid in reaction.

In the case A.G. gastric disturbances were a marked feature in the case.

The tongue may be slightly furred and where much cyanosis is present it is found to share in this symptom.

Appetite. At such a stage the appetite is capricious and much impaired. The Bowels may be inclined to be loose and are often rendered so in the course of treatment.

Abdomen. The liver becomes affected secondarily and is found enlarged in its vertical dulness, reaching often below the costal margin. It may be tender on pressure. There are consequently disturbances in the bilious secretion as before stated. Another symptom is effusion into the serous cavities, and therefore one may find that ascites has developed. The intestines also may partake of a catarrh similar to that present in the stomach, with disturbances in their secretions.

Urinary. The kidneys suffer from the general

stasis and consequently their secretion becomes altered in quantity and composition. The urine may contain albumen, blood and casts.

Haemopoietic. The spleen and other blood glands may be affected also and somewhat enlarged. There may be a certain amount of anaemia and altered blood count.

Integumentary. A marked anasarca is generally gradually developed, especially in the legs and feet, gradually spreading to other parts of the body in the final stages of such a case. Skin rashes may appear. In case A.G. there developed a well-marked rash haemorrhagic in character and distributed mainly over the backs of the hands, elbows, and on the eyelids:- evidently purpuric in nature.

Clubbing of the fingers and arching of the nails, a symptom occurring in congenital pulmonary stenosis, also occurs in cases of incompetence. It may also however be found in long continued mitral stenosis and in the above mentioned case (A.G.) where both lesions were present it was slightly observable. The arching of the nails was marked.

I have detailed the series of symptoms which present themselves in a case of Pulmonary Incompetence of an entirely functional character secondary to and complicated with a mitral stenosis.

In the cases however of incompetence at the Pulmonary orifice alone from an organic cause the lung symptoms would be the most prominent and the other secondary signs described might be present in varying degree. In their cases the physical signs of the heart itself would often constitute the main symptoms.

Physical Signs.

Subjective phenomena. There may be few present. Pain as before mentioned may be a symptom, and may be referred to the praecordia. It may only be present on exertion. In advanced cases with mixed lesions there is generally marked discomfort over the praecordia and epigastrium, due to a failing of circulation.

Palpitation may be complained of at times.

Dyspnoea as I have said is a common symptom and especially in a case of Pulmonary reflux alone. It is also of course found in the cases of a functional character. It may be extreme in degree or only provoked on exertion.

Faintness may be present.

Pulse. The condition of the pulse is not so valuable a symptom in Pulmonary cases as in other heart lesions. It may be regular or irregular (as where there is mitral stenosis.) It is often

accelerated and of small or irregular volume. As a rule the pulse is soft, compressible and ^{the vessel} not too well filled. The arterial wall may not be thickened. It is in cases of Pulmonary reflux complicated with aortic regurgitation that it is of most value. I shall refer to this fully under "Diagnosis" and also in connection with sphygmographic tracing taken.

The veins of the neck after some time shew pulsation and this may be present along with a carotid pulsation where there is also aortic incompetence. A vesicular murmur due to a capillary pulse in the Pulmonary circulation heard specially during expiration and best over the angle of the right scapula is said to be present. (Allbutt's Medicine)

Inspection. In a marked case of Pulmonary Incompetence the first thing one notices is that the apex beat is beyond the mammary line to the left, This is mainly due in this case to great enlargement of the right heart.

The real apex beat may not be well felt: the left ventricle being somewhat displaced. There is often pulsation in the epigastrium, and such a movement can also often be seen to the left of the sternum about the level of the second and third inter-

:spaces. The pulsations in the neck are also seen. With mixed lesions signs are visible according the the orifice affected. The form and appearance of the praecordia may be unaltered, or there may be an undue prominence or bulging.

Palpation will give corroboration to inspection. The apex beat will be felt and position verified as well as the pulsations mentioned above. Sometimes thrills can be felt, systolic or dastolic in time, - Well felt at the apex in cases where a mitral lesion also exists: and sometimes well marked over the pulsa- :ting area, nearer the base as described, and to the left of the sternum.

Percussion reveals the cardiac dulness to be extended to the right as well as to the left and if there is much reflux at the Pulmonary orifice this dulness may extend mostly to the right of the sternal margin. According to the other lesions present the dulness may be much extended to the left and downward.

Auscultation: A bruit, diastolic in time is heard at the base of the heart. It may be audible over a considerable area. Its point of maximum intensity is, one might say, always in the second left interspace. It may, however, be well heard along the interspace for some distance to the left, and even in the inter-

:space below.

This bruit is generally conducted down the left side of the sternum and probably more to the right than a similar aortic bruit. It is not propagated into the neck. Such a bruit may occur along with a similar aortic one and may render the case very difficult of diagnosis. Some authorities believe it possible to distinguish between them from the tone of the respective bruits. (Hope.)

This may be rendered easier possibly when one is of a musical character as in the case of A.W.

Undoubtedly it may often be soft and blowing in character rather than rough or musical. I shall refer to this later. Such a bruit, then is generally to be heard and along with other symptoms is often of the the greatest help in diagnosis. In secondary or functional cases this bruit is heard as a soft blowing sound coming immediately after the closure of the valves. In other cases the bruit may be the only thing heard.

Where a systolic bruit existed we should expect some stenosis present.

Aortic area. One may find associated an aortic diastolic bruit, giving rise to great difficulties in diagnosis. A diastolic aortic bruit may be best heard in

the Pulmonary area (Duckworth) or even at the apex as in case W.W.

Mitral area. Where the Pulmonary reflux is secondary to mitral disease we find at this area systolic, presystolic or diastolic bruits. Their lines of Propagation however settle as a rule their points of origin.

Tricuspid. The above would also apply to this area.

The Pulmonary diastolic is generally fairly localised to the area I have mentioned.

Section 111.

I shall now, before considering the diagnosis, detail a few cases which I have for some considerable time had under observation, and after I have set forth these observations we shall consider what is to be learned from them.

Case 1.

A.G., Age 16, Occupation, clerk, Born in Edinburgh. Complaint, Heart Disease and vomiting. Date, February 14, 1904. With regard to History, no evidence of Syphilis in family. Mother had one miscarriage 14 years or so after his birth, and one before his birth. No history of skin rashes, or diseases, teeth or bone affections, or mental troubles. When about 3½ years of age he fell into a pool and was nearly drowned. Thinks that since that time he has always had a "stomach" cough. Temperature was subnormal.

Family History. Father alive and well, and so was his mother. Two brothers alive and in good health. One dead who had a badly swollen neck from Scarlet Fever "not coming out!" One had died at the age of six months, said to be blood-poisoning. Sisters, -one dead, whooping cough. No rheumatism on either side of family for generations and no history of Chorea.

Personal. Home was comfortable and circumstances good.

Previous Health. No illnesses of any consequence till about 6 years of age when he had Rheumatic Fever. No history of temperature. Pains everywhere. Legs and feet swollen and feet turned inwards. No special swelling of the joints or special sweatings were noticed. Got quickly better. Had whooping cough four months later - recovered completely. At 7 years of age had measles - recovery was complete. At 8 years took scarlet fever and when he had been ill a week he took rheumatic pains again. No swelling of joints, and no special temperature, and the pains and scarlet fever went off together. Got entirely better. Since then has had an attack of Rheumatism every second year, and for the last two years has had an attack both years. Said to have had true Rheumatic fever three times in all: the other attacks being simply pains in various regions. From account never seems to have had a typical Acute Rheumatism until last attack: the other attacks being characterised by general pains in limbs, with no special localisation in joints and no particular temperature.

Present illness. This last attack began in October last with pains threatening. In a week he

became so bad that he took to bed. Pains soon left. No swelling of knees or ankles. Slight swelling in feet. Had profuse sweatings (but was of course under treatment.) Temperature soon fell. Remained weakly but resumed work December following. Was back to bed in ten days. Since then has been continually in bed. Did not complain of pain. Merely weakness and palpitation and beating at the chest. Subjective symptoms had only been felt since last illness, since then had complained of above symptoms and considerable cardiac distress. Heart had been known to be affected for 4 years before.

Had no attacks of vomiting between December and February. Was sick after eating and generally so after everything - but not as a rule putting up food. Vomited matter mostly bilious looking, often quite green. Would vomit two or three times in an hour after food. Had interval of rest between meals. This was really the chief complaint together with shortness of breath on exertion, palpitation, and occasional pain over the praecordia.

Circulatory System.

Subjective Phenomena:- Patient had often pain over the cardiac area, mostly of a darting kind. At other times a dull uneasiness. Palpitation was

frequent and so were the Dyspnoeic attacks. Had frequently attacks of giddiness without actual syncope.

Pulse Frequency was 110 per minute, very irregular in time as well as volume. The movement was somewhat small. Vascular pressure moderate. Arterial wall slightly thickened. The Pressure taken by Sphygmometer was 105-110 m m. Hg. - obliterated at 150 m m. (vide Section on Tracings.)

Inspection The praecordia was well formed: the bony thorax however being long. Skin shewed marks of recent chicken-pox.

Pulsations. Apex beat was visible in the sixth left Interspace and was diffused over a considerable part of the praecordia. Its character was forcible, and its time irregular.

Over the Pulmonary Area was seen a distinct pulsation most marked on expiration.

Pulsations outside the Praecordia. Pulsations were visible in the episternal notch and root of neck also markedly in epigastrium:- systolic in time.

Palpation. Apex beat was palpable in sixth Interspace of left side outside the mammary line. Above mentioned diffuse pulsation was also felt giving a rocking feeling to the praecordia.

Apex beat was very irregular, sharp and forcible

in character.

Thrills. Two thrills were felt over the mitral area - diastolic and presystolic. A systolic thrill was also felt over the pulsation in the Pulmonary area followed by closure of cusps. The other pulsations seen were verified.

Percussion Upper Border was at lower border of 2nd rib in the left parasternal line.

Left Border.

In 3rd interspace two inches from left sternal margin

In 4th " 3½ " " " " "

In 5th " sound masked by stomach note.

Right Border

Upper border of liver dullness in mammary line was lower border of 5th rib.

In 3rd Interspace right border was 1½ inches from right sternal border.

Auscultation Mitral Area:- There was audible a blowing systolic bruit and rougher diastolic and pre-systolic. They had their maximum intensity over the mitral area. The systolic alone was propagated into the axilla.

Pulmonary Area. There was audible a distinct diastolic murmur. This was of a blowing character and was heard loudest in the Pulmonary area fading off round about as in chart. It was quite different in

character of note from the other bruits heard. It

Was heard distinctly all over area of pulsation.

Triscupid. Both the systolic and diastolic bruits were heard here, and were more musical and twanging in character than heard in the mitral area.

Aortic. Sounds were closed.

Haemopoietic System.

Red Blood Corpuscles	-	4,5000,000
White " "	-	6,500
Haemoglobin	-	68%

Film showed nothing special.

Count made before and after the administration of oxygen.

Before	{	R.B.C.	-	5,670,000
		W.B.C.	-	20,000
		H.b.	-	68%

After	{	R.B.C.	-	4,800,000
		W.B.C.	-	11,000
		Hb.	-	60%

Alimentary System. Lips were dry, tongue furred, teeth were good. Bad taste in mouth was constant as also was dryness of mouth. No pain on swallowing. Appetite at first for solids was good, but became gradually impaired. Had great thirst. Had marked discomfort over stomach - even to pain and local tender-

ness. Pain was not specially aggravated after food. Flatulent eructations and water brash were markedly present and gave him pain. Vomiting was a marked symptom and got more urgent as case went on. Had difficulty to retain any food latterly.

Vomited Matter. No free acid.
Total acidity .109.

Abdomen.

Palpation. Abdomen was tense and presented extreme tenderness over epigastric and both hypochondriac regions.

Percussion. Lower border of stomach was a little above umbilicus.

Liver. Upper border in mammary line was lower border of 5th rib - deep dulness. Lower border - costal margin. Bowels fairly regular.

Respiratory System. Breathing was Costo- Abnormal. Had some cough at times and his history referred to a "stomach cough" having been present for years.

Inspection. Chest was full and emphysematous in form. Expansion was somewhat under the average. Palpation Vocal fremitus was lessened slightly on both sides.

Percussion. Shewed increased resonance all over chest.

Auscultation. Vocal resonance somewhat lessened specially on right side. Breath sounds all over chest

were coarse vesicular, with marked prolongation of expiration and occasionally accompanied by sibilant rhonchi - especially over left lung.

Integumentary System. Skin was very dry all over specially that of hands. No oedema was present until about three weeks before death. (vide progress). Over chest were some marks of recent chicken-pox.

Urinary System. Sp. Gr. averaged about 1023. Was acid. Albumen at first a trace but gradually increased in amount. No blood, bile, or sugar was found.

Urea was 5gr. per oz.

There was nothing to note in other symptoms.

The progress of this case during the time it was under my observation was as follows:-

This case came under my notice about the beginning of February 1904. His history as detailed above, and the general symptoms present led me without any hesitation to consider the case to be one of mitral stenosis and incompetence. The cardiac irregularity, cyanosis and progressive oedema led me to regard his condition as grave. The interest in this case was not so much (so far as this paper is concerned) confined to the mitral condition as to the somewhat rare condition at the Pulmonary orifice.

From the above description of the case it will be seen that so far as physical signs go there was clear evidence of a leakage at the Pulmonary orifice. The diastolic bruit indicative of this was during the whole time I watched the case invariably heard - in the same area, with more or less distinctness. It will be seen from the diagram that it was heard over an egg-shaped area from about the level of the upper border of 2nd rib to nearly the upper part of fourth interspace. The right margin of this area corresponded to the left lateral sternal margin. The left margin was $\frac{3}{4}$ " to the left of the right margin. The length of the area was about $2\frac{1}{2}$ ". Within this area the bruit had its maximum intensity in the 2nd interspace and slightly higher about $\frac{3}{4}$ " from the sternal margin. It was entirely confined to this area and although with fluctuations in the condition of the patient owing to treatment it varied slightly in intensity and quality at times, yet it was always audible there. It was diastolic in time: the closing of the cusps could be heard, and this murmur of escape after it. It was slightly more rough at times but could always be distinctly heard in the same area. This bruit was not heard anywhere else. The aortic sounds were quite closed, and there was no bruit conducted to the vessels.

It could not be mistaken for the bruits heard at the mitral area, and I am convinced it was produced at the Pulmonary area.

The further history of the case was one of progressive heart failure. There were one or two interesting points which I may merely refer to. He was throughout restless and often much concerned as to his progress. The oedema commencing in the feet kept on increasing. He developed over abdomen, chest, and part of the back a punctiform erythema, which disappeared on pressure. At this time his temperature rose slightly above normal. The rash was not itchy, it became papular and then gradually passed off. The face gradually became puffy, and the general oedema increased:- the skin pitting readily. Some crepitations then appeared at both bases, and the abdomen became fuller although no thrill was felt. The cyanosis came and went, being extreme at times. The vomiting was also persistent at intervals. About ten days before death there appeared on the backs of the hands, feet, and elbows a rash similar to Peliosis Rheumatica. The spots were bright red, haemorrhagic, and were inclined to coalesce. There was under this an erythematous blush which faded on pressure. It was, in short, evidently a variety of Purpura. The

oedema gradually extended up the back. No fluid gathered in the pleura at this stage.

Face and eyelids became more puffy, vomiting and the cyanosis continued. The lower eyelid (as he was lying) showed haemorrhagic swelling. He became now much weaker, the pulse gradually failing in force and regularity in spite of treatment. Until the time when he passed away (27th April) his further history was one of progression downwards, with aggravation of most of the symptoms.

The report of the section made by Dr. Stuart McDonald is as follows:-

Alexr. G.

Rigor mortis passing off.

General anasarca.

Large collection of fluid in peritoneum, and both Pleural cavities.

Three ounces of clear fluid in pericardial sac.

Heart weighed 1 lb.

Diffuse thickening of Epecardium.

Some chronic old adhesions at reflection of pericardium posteriorly.

Right Auricle:- distended with fluid, blood and Post-Morten thrombus.

Right Auricular Appendix:- Some ante mortem thrombus.

Tricuspid orifice:- normal size.

Pulmonary Valve:- competent.

Right ventricle:- contains some Post-Mortem thrombus.

Distinct vegetations (recent) on tricuspid valve.

Pulmonary cusps and Aortic cusps nothing abnormal.

Left Auricle:- enormously dilated and contains Post-Mortem thrombus.

Endocardium much thickened.

Mitral:- stenosed.

Left ventricle nothing of note, save a little Post-Mortem thrombus.

Aortic valve shews marked chronic thickening of cusps. Edges of valve distinctly rigid.

Some recent minute vegetations on both cusps.

Aortic valve shews chronic thickening, and the aortic segment (anterior) is adhered to margin of right posterior cusp.

Numerous recent vegetations and some points of haemorrhage along line of vegetations. Aorta is slightly atheromatous.

Coronary Arteries are not very atheromatous although thickened.

Myocardium firm, showing glistening points in muscle, and white spots indicative of chronic myocarditis.

Left Lung:- Glands at root enlarged and pigmented. Nothing else abnormal.

Left bronchi:- congested.

Pulmonary Artery walls very distinctly thickened. Chronic Pleurisy over upper lobe anteriorly and inferiorly.

Minute petechial haemorrhages.

On section marked congestion and irregular dark areas of increased resistance.

Condition is one of typical chronic venous congestion. Fibrous tissue in lower lobe increased.

Vessels thickened. No consolidation or tubercle.

Right Lung:- Glands at root enlarged and pigmented. No tubercle. Larger bronchi congested

and contains muco-pus and blood-stained fluid.

Chronic Pleurisy over upper lobe. On section

there is chronic venous congestion. Large

infarct in upper part of lower lobe shewing a red appearance only at surface. The rest of the con-

solidated area is partly decolourised. Athrom-

bosed branch of the Pulmonary Artery is well

seen. Infarct in anterior edge of lower lobe

also shews partial decolouration. Vessels thickened throughout.

Liver:- Gall bladder has thick oedematous walls, and contains dark viscid bile. No stones. Surface is smooth and mottled. On section there is typical chronic venous congestion.

Spleen:- Increased consistence on section, chronic venous congestion. Malpighian bodies not prominent.

Kidneys:- Chronic venous congestion. Capsule strips fairly readily, except at one or two places. Foetal lobulation well seen.

On section:- Chronic venous congestion. Cortex a pink red colour.

Malpighian bodies prominent as pale shining points, very minute and thickened.

Pelvis shews haemorrhage in mucosa.

The other organs shewed nothing special.

The heart after being placed in 20% formalin for a few days was again carefully examined by myself and Dr. McDonald, specially with regard to measurements. They were taken both by calipers and tape, and were as follows:-

Pulmonary orifice - 2.6 c m. or 1.1 inch nearly.

Aortic orifice - 2.6. c m. or 1.1 inch nearly.

Thickness of walls.

Right Ventricle	-	.75 c m.	or	$\frac{1}{2}$ inch	nearly.
Left Ventricle	-	1 c m.	or	$\frac{1}{3}$ inch	"
Right Auricle	-	.25	or	$\frac{1}{12}$ inch	"
Left Auricle	-	.25	or	$\frac{1}{12}$ inch	"

Note

From the above it would appear that the Pulmonary orifice was little, if anything, dilated, the measurement of the aorta being rather under normal. The right ventricular wall is a little under the average thickness.

Case of A. W. Age 8. School boy.

Complaint was pain in left side of chest occasionally felt in shoulder and left arm, as far as elbow.

His Former History was as follows. Father's age was 53, who had suffered from fever and ague, but was otherwise healthy. Mother age 40, quite healthy.

Had three sisters and one brother, who were all alive and healthy. Four children had died of children's diseases - two of convulsions, two of Whooping Cough, and one of Measles. History was therefore good.

Home was only a two-roomed house, and was inhabited by seven occupants. The environments were unhealthy.

Home influences were not of the highest kind - parents being given to alcoholic excesses. Appetite had always been fair. Food had not been very abundant.

Previous Illnesses. Had had four years ago an operation for rupture. Had measles, and had Rheumatic Fever August 1902, which had kept him in bed during August and September.

In September 1903 he had twice severe attacks of pain in left side of chest which radiated into shoulder and left arm. Returned to school in following October. Since then had often complained of the pain in chest with varying breathlessness, but never was severe enough to keep him from school for more than a day or

two. On February 12th, 1904 had a severe attack of pain in chest and arm. This attack caused him to scream, and his eyes would become fixed as if on some object above him on the wall. Was frightened, and pointed to spot he was looking at. Turned stiff and pale. Did not sweat or sleep afterwards, but rather complained of the persistence of pain. The attack would last for over an hour.

Patient was a small, thin child. Wore a serious and anxious expression. Skin was mottled and marked. Looked as if not too well cared for. He shewed some slight cyanosis specially on exertion. When at rest in bed it passed away.

Circulatory System.

Subjective Phenomena. Pain as described in history was occasionally felt, but improved with rest. No other subjective phenomena.

Pulse Frequency 95 per minute. Fairly regular in time and force. Movement moderately large. Vascular pressure fair, and as taken by Sphygmometer was 110 m m. - obliterated at 140. (vide tracings.)

Inspection. Chest was well formed. Walls thin, slightly more prominent on left side over praecordia than right side. No indrawing or bulging of inter-spaces. No pulsations except apex beat. This was

seen in 5th left interspace in mammary line. Pulsation was present in episternal notch, and slightly in carotids. Also a slight venous pulsation.

Palpation. Apex beat was 2½ inches from midsternal line in 5th interspace, and was of a heaving character. Other pulsations above were verified. Thrills felt in 3rd and 4th interspace with each systole.

Percussion Liver. Upper Border Fourth Interspace or upper border of 5th rib.

Right border of Heart. In 3rd interspace 1½ inches from midsternal line.

Left border - 5th Interspace 3 inches from midsternal line.

4th	"	2½	"	"	"	"
3rd	"	2	"	"	"	"

Auscultation

Mitral Area. A soft systolic murmur was heard and was conducted into axilla, and heard over a wide area, as seen in diagram, second sound was closed.

Tricuspid Area. A systolic bruit and diastolic musical bruit was heard accompanying the second sound.

Pulmonary Area. Rough systolic was heard here also a musical diastolic bruit overlying a blowing diastolic murmur with its maximum intensity in the 2nd left interspace near the sternum and well up and down the sternum and towards the apex.

Aortic. Rough loud systolic bruit heard also in the vessels of the neck. The musical diastolic was heard as described.

Capillary Pulsation was present, although not very marked at times.

Alimentary System. Teeth fairly good, Tongue clean, Appetite was fairly good. No pain or other symptoms present. Bowels were regular. Abdomen rather full. Liver was not enlarged.

Haemopoietic System Spleen and other glands were not enlarged. Blood count:-

Red Blood Corpuscles	3,830,000.
White Blood Corpuscles	7,000
Haemoglobin	60%

Film shewed nothing special.

Urinary System. No subjective phenomena.

Urine Sp.Gr.1015, Reaction was acid, Mucous deposit, No albumin, blood, bile, or sugar present.

Case of W.M.

Age 37, butcher and married. Before seen by me had been under treatment for "bloodlessness."

Family History. Father was dead: cause not known but was advanced in years. Mother was alive and well. Had three brothers alive and well. Two had died young from diphtheria. Wife was alive and well. Had three children, two were alive and well - one was dull, backward and delicate. This child was second in series. There had been one miscarriage after the second birth. There was not evident any special disease tendency in the family.

Personal. His work had been of a manual kind and fairly heavy. Hours worked being 7 a.m.- 7-30 p.m. All indoor - not specially exposed, but always standing.

Home had been quite comfortable - food good, regular, and well cooked. House was sanitary. Had been in the habit of hurrying to work in mornings without proper food. Had not been an abstainer, had drunk to excess, but only rarely. Had been on the whole fairly temperate.

Had Gonorrhoea about 15 years ago. Not had syphilis. Usually weighed about 10 stone 8 pounds up to 11 stones, but had lost a good deal since then. His

temperature was about 99.8 generally and his pulse averaged 112. Respirations 22.

Frequently got cuts on hands, and once had a bad wound on knee. He never had any trouble with these, either as to bleeding or poisoning.

Previous Illnesses. Had measles. Was healthy when a boy at school. Was subject to sore throats and abscesses about the mouth and throat. Had one in throat recently which burst internally - evidently Quinsy. He had an attack of Rheumatism of a Sub-Acute kind at 25 years of age. Still had attacks of pain in shoulder and wrist at times. Had at age of 36 a ruptured varicose vein, from which he lost a small amount of blood.

Had been troubled for years with piles, and two months before I saw him he had had a bad attack, and had lost a large quantity of blood thereby. Health all over therefore for a few years had not been good. Had at times also attacks of vomiting generally of a bilious character.

Present Illness. For six months previous to the beginning of this year he had been getting weaker without any definite commencement. Had first noticed a difficulty in mounting a hill. Had not special shortness of breath, rather general weakness and

incapacity for exertion. Had gradually thus become unable for work. Complained at times of a beating or pulsation in his back on exertion, located to each side of middle line above the sacro iliac synchondrosis. Complained of frequent giddy attacks, not actual fainting. Could only recall having once fallen from giddiness and collapse.

General facts. This patient was a fair haired, pale man having marked anaemia of conjunctivae and mucosae. His sclerotics shewed a mixed tinge, not yellow. Nor was his skin definitely yellow. Shewed no pigmentation bands and no spots on tongue or buccal mucosa. There was present some scars on hands, result of cuts. Small cuts resulted in him in a fair amount of haemorrhage, but was easily enough stopped. Scars on knee as above mentioned. A junction of lower and middle third of left leg was a dark pigmented area - stated to result from the bursting of the varicose vein before mentioned. The bleeding had been stopped by means of the cautery. He had two scars on left side of neck the relics of small abscesses which had burst.

Circulatory System.

Thorax was long, the upper ribs sloping downwards, thus producing an unusual width of the upper inter-

:spaces. Pulsation visible in the episternal notch and vessels of the neck throb markedly, but there was no venous pulse.

Apex beat was palpable in 4th interspace internal to mammary line, it was feeble and slightly diffuse and was not palpable in the 5th interspace.

Heart. Right border at level of the 4th rib was:-
1½ inches from midsternum.

Left Border.

At level of 4th rib was 4½ inches from midsternum
" " " 3rd " " 3½ " " "
" " " 2nd " " 2½ " " "

Upper border was at the level of the 2nd rib.

Auscultation. The sounds were all closed at the time when he came under my observation. The mitral first sound was only possibly a shade roughened. All over the sounds were rather feeble.

Pulse. was regular in time and force. The volume was fair. Frequency was 112.

Haemopoietic System. The right submaxillary and the axillary glands were slightly enlarged.

The thyroid was not enlarged.

The spleen was not enlarged.

Blood Count. When first examined it was as follows:-

Red Blood Corpuscles	1,450,000.
White " "	3,300
Hoemoglobin	20%
Index -	.68

53.

On careful examination of Film it shewed considerable poikilocytosis. The red blood corpuscles were of normal size. There were several normoblasts and one megaloblast in the film examined. An occasional rare megalocyte was found.

Respiratory System. There was nothing special here to note.

Alimentary System. Tongue clean. Functions all healthy. Bowels somewhat irregular.

Patient had three large pale external and mucocutaneous piles protruding from and surrounding the anus. They were not painful then, but had some little time previous been much inflamed and painful. They bled frequently.

Urinary System. Urine acid. No albumen, blood, bile, or sugar. Nothing in centrifuged deposit. Urea gr.vii per ounce.

The other symptoms shewed nothing abnormal.

This case as far as I have yet recorded, therefore, shewed no cardiac condition of any movement. It is because of the development of cardiac symptoms later,

that I have recorded his case somewhat fully. It will be necessary for me to shortly state the series of changes that took place in the patient's unfortunate condition until the time of his death.

As the patient was making no progress he had the haemorrhoids removed. He suffered no bad effects from the operation. It was found, however, that he continued gradually to grow worse. After operation the pulse stood about 90.

As will be seen from the following table his blood count steadily fell.

	R.B.C.	W.B.C.	Hb.
10 : 2 : 04	1,320,000	4,400	23%
19 : 2 : 04	1,220,000	5,000	26%
21 : 2 : 04	1,110,000	-	24%
29 : 2 : 04	800,000	-	18%
9 : 3 : 04	500,000	3,000	16%

(Death)

His pulse gradually became more rapid and his skin shewed a yellowish tint. In short, the case seemed to be one of pernicious anaemia. The blood displayed an occasional megaloblast. The colour index was plus. Notwithstanding various modes of treatment into which I need not enter he went steadily downhill. The pulse continued of fair volume. The right brachial gave a pressure of 135 m.m. Hg. His temperature rose to 101⁰ but fell again later, before

death.

Five days before death there developed a soft murmur diastolic in time and limited strictly to the region of the apex. In the accompanying diagram I have endeavoured to shew the extent of this bruit, taken somewhat rapidly owing to the extremely weak state of the patient. It was evidently a diastolic aortic bruit produced in some manner at that opening. It was soft and blowing in character, and was constantly present till the time of death.

Commentary on Cases.

1. In case of A.G. there is in my opinion ground for considering that the Pulmonary Artery allowed of an escape. There was no doubt as to the mitral stenotic lesion. The aorta was closed and there was present all the conditions to give a high pressure in the Pulmonary Artery.

(15) (7)

Steell, Pitt, and others are of opinion that a diastolic bruit audible down the left side of sternum, and heard loudest in the Pulmonary area is to be observed in many cases of mitral stenosis, and that in such cases the cusps Post-Mortem may be healthy, and shew no evidence of regurgitation.

A careful examination of all the symptoms present, leads me to think that this condition obtained in my Case A.G. From the report of the Autopsy appended however there was not much evidence of the valve being incompetent.

Stokes⁽¹⁾ was of opinion "that when the Pulmonary Artery was dilated there was as a rule no change in the cusps, nor any notable symptom during life." Such may be the case occasionally, I am of opinion a diastolic Pulmonary bruit will generally be heard.

I also believe that you say Post-Mortem have some

sign of this dilatation, but that very often no sign of it exists - although all the symptoms pointed to it being present during life.

Probably the first to refer to this subject of relative incompetence at the Pulmonary artery was Hunter⁽¹⁴⁾, and Adams⁽¹²⁾ later held that it had not been sufficiently emphasised. Let me quote his statement: This difference between the right and left sides of the heart he looks on as a natural provision of nature to allow of a partial reflux into the right auricle on occasions, when from any cause the arterial flow is impeded.

⁽¹⁵⁾ Steell also considers that a murmur of incompetence at the Pulmonary artery can occur when there is much obstruction to the Pulmonary circulation with a dilated Pulmonary artery, just as a diastolic murmur is not infrequently heard from slight incompetence of the aortic valves when that vessel is dilated.

The reflux in case I have recorded (A.G.) I think can fairly and honestly be attributed to this cause - the increased pressure in the Pulmonary circulation secondary to the mitral lesion present giving rise to some dilatation of the artery and also of the orifice itself.

11. In this connection and by the way of comparison I have recorded the case W.M. where in a case of Extreme Anaemia a diastolic aortic bruit developed some days before death. This bruit was confined to an Area practically round the apex, as is found occasionally and undoubtedly indicated a relaxation of the aortic ring as a result of the gradual dilatation of the Aorta, and also probably the Left Ventricle itself. When this development occurred I decided that it was an example of a functional incompetence without any organic lesion in the valves. I was not able unfortunately to verify this idea by an autopsy - none being allowed.

It was interesting for me while observing a case of relative incompetence at the Pulmonary Artery to watch this case of reflux at the Aortic orifice.

We are prepared for the statement that this can be of less frequency than at the Pulmonary Artery owing to the much firmer structure of the aorta.

It undoubtedly occurs, and I may only refer to cases recorded by Renvers⁽¹⁵⁾, Gairdner⁽¹⁶⁾, Chevers⁽¹⁾, Besnier⁽¹⁷⁾, Dombrowski⁽¹⁸⁾, Cabot⁽¹⁹⁾ and Locke.

Such bruits are not unfrequently heard best in the Pulmonary area to the left of the sternum. In the case I observed it was heard best at the apex, and also

towards lower end of the sternum. Such bruits may be heard loudest as I said in the Pulmonary area, and when this is the case a consideration of the other symptoms would only decide whether it was not the result of a Pulmonary reflux.

The pulse tracing which I took corroborated the diagnosis of aortic reflux. It would be interesting to know what were the real causes in the production of this reflux, whether it was due to the condition of the blood or to an increased blood pressure.

As this is somewhat outside the subject of this paper I will just merely refer to it.

(20) (21)
Klein, Sahli, and others record cases of this kind, the latter of whom is inclined to think that for the stretching of the aortic ring a high pressure is necessary. These conditions were to some extent present in the case W.M. recorded.

The blood pressure, as carefully taken by the Sphygmometer, stood at 140 m.m. Mercury at the time the diastolic bruit developed. I should say therefore that this blood pressure coupled with some dilatation of the ventricle, in this case of extreme anaemia, were factors in the production of the stretching of the aortic ring, and consequently of the regurgitation.

111. With regard to the case A.W. it is recorded as 51.
remarkable example of a combination of well marked
diastolic bruits indicating most probably some reguri-
:tation at the Pulmonary orifice as a result of an
organic lesion there. The aortic valves are also
evidently incompetent. I am of opinion that the
loud musical bruit is of aortic origin, and that the
Soft blowing diastolic is produced at the Pulmonary
area.

The systolic heard over a wide area is probably
produced at both .

I should say that the valves of both the basal
orifices are organically affected. An important ques-
:tion to decide was whether there was mitral incompet-
:ence. The systolic at the mitral area was not loud
and was very difficult to hear propagated into the
axilla. I should say that if present the reflux
could only be very slight.

The condition at the base was different. The
loud systolic at the aortic area could be well heard
in the vessels of the neck. There was also the musi-
:cal diastolic heard as described. Then the distinct
blowing diastolic in the Pulmonary area distinct from
all, together with the other symptoms favoured some
leakage at that opening.

The examination of the pulse and pulse tracings did not help much. It is neither indicative of aortic or mitral lesions. This leads one more confidently to believe that the Pulmonary orifice is incompetent. The history did not give any leaning to the idea of there possibly being a congenital lesion present, and after a consideration of all the symptoms and the wonderful combination of bruits heard, I am of the belief that (although a rare lesion) there is some incompetence at the Pulmonary orifice, and that most likely it is complicated with an aortic lesion.

Diagnosis.

Section IV.

I am of opinion that the diagnosis is often a matter of considerable difficulty. This difficulty has several times been apparent to me in practice where the physical signs suggested to one the probability that there might be a leakage at the Pulmonary valve.

The cases which I have had under observation for many weeks and which have been recorded at some length, give examples of this affection, and can be usefully compared with each other in reference to the difficulties of diagnosis.

First, therefore, in the case A.W. I consider as I have said that the incompetence at the Pulmonary artery is not secondary to other lesions, but that the Pulmonary cusps themselves being probably thickened and diseased are allowing of an escape. There is here also evidently an aortic lesion which gives rise to difficulty.

In case A.G. on the other hand, I have brought forward a typical example of a functional incompetence of the Pulmonary artery, the result of a high pressure in the lesser circulation, and intimately associated with the mitral stenotic condition present.

In the third case (W.M.) I have recorded an example of functional incompetence of the aortic orifice by way of comparison. This case is an interesting one, and as the great difficulties of diagnosis are mainly concerned with these two basal orifices it is of value here.

In the diagnosis we will be more likely to arrive at the truth if we do not confine our attention too much to one set of symptoms alone, but be guided by them all as a whole.

1. Inspection and Palpation of the patient generally, and the condition of the præcordia and vessels of the neck in particular are essential to a diagnosis. Pulsation in the second and third interspaces of left side is commonly present in those cases where there is actual dilatation of the Pulmonary artery. This I think may be of value mainly in those cases which result from a high pressure in the Pulmonary Artery, and where there may be even some permanent dilatation of the artery. Such cases as the former are recorded by Hope, ⁽⁹⁾ Steell, ⁽¹³⁾ Gibson, ⁽²²⁾ and others.

⁽⁹⁾ Hope refers in his work to one case where with the presence of a thrill, dulness, and pulsation between the second and third left cartilages an actual aneurismal condition of the Pulmonary Artery existed. The

presence of an area of pulsation with thrill would point therefore to some possible dilatation. Aneurism of the Aorta would have to be considered where there was a pulsating area. The pulsation which was present in my case and which I consider was caused by a dilated state of the Pulmonary artery could not be mistaken for a pulsating aneurism. The other lesions present led one to a conclusion as to its nature. The pulsation was not so expansile or forcible as one would expect to find in aneurism under similar conditions.

The fullness and pulsation of the veins in the neck although present in many cases of Pulmonary incompetence and of value where you are dealing with a simple case of organic lesions at that orifice are unfortunately also to be met with in cases of ordinary heart failure from other valvular lesions. They, therefore, must be considered only in conjunction with other symptoms. The same may be said with regard to presence of clubbing of the finger nails, cyanosis, etc.

Auscultatory Evidence. With regard to the bruits heard on auscultation Stokes⁽¹⁾ is of opinion that the great difficulty lies in the fact that you have the sounds at the Pulmonary orifice masked by those of the Aorta when there is a bruit present. That is undoubtedly so. Lesions "per se" of the Pulmonary artery

are comparatively rare and therefore the above difficulty in most cases presents itself. What is still worse: there may be a lesion at both orifices and then it is increasingly difficult to arrive at a diagnosis with some degree of certainty. There is ~~little~~ evidence that a diastolic bruit may be heard at the base of the heart, and produced at the Pulmonary valve when it was considered to be aortic. Let us consider the value of the presence of a diastolic bruit and this generally confined to the left of the sternum in the Pulmonary area:- 2nd left interspace. All writers on this subject describe this murmur as being heard here. In my cases, as seen from the charts, it was heard as a rule in the 2nd left interspace, and loudest there, but was well heard, but with diminishing intensity, down the left side of the sternum. It could be heard as low as the 4th interspace or even the fifth rib. The bruit was inclined to be propagated more in the direction of the apex, but not beyond the limits described. The diastolic bruit was not heard in the vessels of the neck. We have, however, to face the difficulty that in not a few cases an aortic diastolic bruit may have its maximum region of intensity to the left of the sternum, i.e. practically in the Pulmonary area, as pointed out in ^{works} many of the

heart.

In one of my cases, (one of extreme anaemia) which I have brought forward there was developed some days before death a diastolic murmur confined mostly to the region of the apex beat - undoubtedly of aortic origin. ⁽³⁾ Barie in his consideration of this subject lays considerable stress on the Auscultatory signs, and believes that Pulmonary Incompetence has most often, probably been mistaken for Aneurism and Aortic Incompetence.

I referred to aneurism pressing on the Pulmonary artery as a cause of functional reflux. In such a case it most likely would be impossible to make a diagnosis in most cases, as the signs would be masked by those of the aneurism.

With regard to the character of the bruit I am not sure that one can lean too much on any difference of tone or pitch which might be supposed to exist.

⁽²⁾ Hope was of opinion that bruits produced at the Pulmonary area were of a higher note than those produced at the Aortic orifice. ⁽¹⁾ Stokes, however, thinks that such is not to be at all depended upon, and that a diagnosis on such grounds is entirely fallacious.

The bruits I have heard were all of a soft blowing character and certainly more so than one generally

hears at the aortic orifice.

We thus see that it would appear somewhat risky to arrive at a definite conclusion so far as auscultation is concerned: and yet it is often, so far as I can see, the main symptom.

When percussion is performed. One finds as a rule the signs of Hypertrophy and dilatation of the right side of the heart. The right border may be found to be well beyond the right sternal margin, whereas in aortic cases we would find the signs of hypertrophy of the left ventricle. In cases, nevertheless, where there is great enlargement of the right heart alone the apex is often displaced downward and to the left, although the left ventricle may not be hypertrophied. In the cases, however, where both lesions exist or where there is a mitral lesion this sign is only of value when considered with the other symptoms as for example, the respiratory signs, and the help given by the pulse. In the case of A.G. the increased dullness to the right was not of much help in itself because of the mitral lesion present. If much regurgitation is present at the Pulmonary orifice it is a constant factor. If slight, only, it may not be well marked. (Case A.W.)

The next point I mean to refer to is the one I think of greatest importance as a differential

diagnostic between Pulmonary and aortic reflux, viz:
the condition of the pulse. (9) Hope, (5) Duckworth, (3) Barie,
(22) and Gibson, have all laid great value on whether there
was present a Corrigan pulse or not. (10) Stokes has point-
ed out however that in the early stages of aortic
reflux this symptom might not be so prominent as to be
of real value in a case of difficulty. I think this,
although not likely as a rule to detract from such a
valuable differential symptom, is a point of some little
importance. It shews that such a case might arise
where it would be of little or no help. It is not
likely that some of the other signs of Aortic regurgi-
tation such as pain, faintness, capillary pulsation,
auscultation of vessels of neck etc. would not lead one
to make a diagnosis. Where the pulse is felt by the
finger to be of a bounding character, and with rapid
collapse of low tension and the vessel somewhat empty,
coupled with a tracing corroborative of such a pulse,
one can have no doubt, whatever other lesion may be
present, that there is incompetence at the Aortic valve.
In case A.W. I have referred to this question. Here
the pulse gave no indication of Aortic incompetence,
although the venous pulse in the neck shewed an inter-
esting condition (vide section on tracings) rather in
favour of Pulmonary reflux.

In cases where diastolic bruits exist at both orifices such a pulse would prove aortic incompetence to be present. It would not, however, prove that there was not also present Pulmonary reflux, and undoubtedly both conditions may be present in same case. In the event of such a combined case arising, and where the pulse did not give help, one would have to be guided by the percussion signs, Pulmonary condition, and the general signs of backward pressure.

The difficulty arises I think from the fact that in the possible presence of Pulmonary Incompetence one is thrown back on a reliance more on the Auscultatory signs than in the case of Aortic and mitral lesions, because the pulse, of great value in proving the existence of aortic disease, would give no help in determining whether there was Pulmonary reflux or not.

The signs of backward pressure, Pulmonary signs, and even the condition of the cervical veins and finger nails to be referred to shortly may to some extent be met with in mitral cases or cases of heart failure even from Aortic disease. Accordingly in such an eventuality one has to fall back on the Auscultatory signs and a careful balancing of the other symptoms.

(10)

The position is put very well by Stokes when he says that "The elements of probability on the one hand

and association of symptoms and signs on the other must be the chief guides in determining the seat of valvular disease."

In Pulmonary Incompetence pure and simple as I said the pulse does not tell you much. It may be regular or irregular. It is as a rule of small volume, and not at all forcible in movement. The condition of the arterial wall may not be at all altered.

From the respiratory symptoms we may be able to glean facts and signs of importance, and these placed to other details may help one to the diagnosis. From an anatomical and physiological point of view when there is reflux at the Pulmonary orifice it would be naturally expected that the lungs would suffer in their functions. This is so. Respiratory embarrassment is constantly present in a more or less pronounced degree in the form of dyspnoea, cough, haemoptysis, cyanosis, due to the imperfect and unsteady supply of blood to that organ. This symptom (dyspnoea and cyanosis) in one of my cases was only to be noticed on exertion. In another it was present, but exemplifies its value as a diagnostic agent because in this case (A.G.) there was also a mitral lesion which might have accounted at least partly for its presence. Is it then any guide in such cases? I think it is of

the greatest importance if no other lesion beyond Pulmonary Incompetence is suspected to exist, and I should say in a combined lesion it is decidedly more helpful where the second lesion is aortic, than where you have the Pulmonary Incompetence complicated by a mitral lesion. An extreme degree of cyanosis and dyspnoea etc. present along with auscultatory and other signs of Pulmonary reflux such as hypertrophy of right heart even in the presence of a mitral lesion would lead one to the conclusion that there might be an escape at the Pulmonary valve:- of a functional kind. This class of symptoms would undoubtedly have to be considered in the light of others present owing to the fact that they are found in mitral cases and even in general cardiac failure.

Haemoptysis may occur where the Pulmonary circulation is affected. When it occurs as the result of emboli in the lung the Pulmonary orifice is usually the seat of an endocarditic process. This again not being altogether peculiar to Pulmonary reflux must be taken along with the other symptoms. It is however a fairly constant symptom in cases of direct endocarditic infection of the Pulmonary valves, the result of acute infective processes. A symptom referred to by Pitt^(?) as a capillary pulsation in the Pulmonary circulation

is sometimes present. It seems to be best heard at the angle of the right scapula and is described as a waxing and waning of the vesicular sound. Theoretically this is likely to occur but I should say its presence or absence would depend greatly on the amount of regurgitation at the Pulmonary orifice. It was not heard in the cases I have recorded.

In the previous pages I have enumerated many signs and symptoms of this malady, yet it is my belief, that in many of the cases especially in those where other heart lesions exist, one has only a small margin for a diagnosis. To arrive at such, it appears to me, that the utmost scrutiny of all the signs and symptoms must be made.

Some writers on this subject have put much reliance on the position and character of the auscultatory signs and no doubt in many cases these are of a very convincing kind. It is possible, however, too much reliance may be attached to them. Yet in the light of the absence or presence of other symptoms they may go a far way in leading one to come to a conclusion in the diagnosis of this disease.

Prognosis and Treatment.Section V.

I do not intend in this thesis to dilate on the above headings.

For the sake of completeness I merely refer to them.

The prognosis must in the majority of cases be grave. This holds I think whatever may be the etiology in the case.

Where it occurs along with other lesions it adds to the gravity of the condition on account of the Pulmonary involvement. In endocarditic cases the lung condition makes the case more anxious.

Sieveking⁽⁴⁾ was of opinion that there was a close relationship between this disease and Pulmonary tuberculosis.

This may be so in relation to the cases he has described, but I am not able to say it has relations to the variety of Pulmonary affection I have considered here.

Treatment would be pursued on the lines of restoring compensation (as in aortic cases) when that was not established.

In the complicated cases the treatment would be

Tracings. Vide p. 79.Sub Section.

As previously mentioned, the condition of the pulse, in Pulmonary Incompetence, is not generally prominent as a guiding symptom. As a means of help however, in making a differential diagnosis between Aortic and Pulmonary insufficiency it is of great value in the majority of cases.

It may also, I think, shew (by sphygmographic tracings) points suggestive of the condition of the venous return to the right side of the heart, as well as other interesting minutiae. I shall therefore add a word or two to the tracings I have taken from my cases.

The tracings (1-9) are taken from case A.G.

No 1-4 are tracings taken after the commencement of treatment with strophanthus and other heart tonics. They, however, shew the irregularity and small amplitude of a mitral case. No.1 shews an abortive ventricular contraction following a more perfect contraction with fair regularity.

No. 2 - 3. Shew an irregular pulse and is indicative of a more irritable state of the ventricle. The respiratory record does not seem to affect the

No. 5 shews combined cardiographic and pulse tracings, and is I think fairly typical of an "inverted cardiogram."

This fall corresponding to the systolic pulse wave, probably due to "the supposed sucking in" action of the chest wall is most likely here caused by the great dilatation of the right side of the heart which was present. From the measurements I found the fall to exactly correspond with the systolic wave.

No 6 shew same taken by itself.

No 8 shews same.

No 9 shews comparative regularity and improvement from treatment.

No 10 - 19 are tracings from case W.M. (Functional Aortic Incompetence)

They show a fairly typical Corrigan pulse tracing with abrupt up and down stroke and obliteration more or less of the dicrotic wave. There is also some irregularity due to respiration.

No 20 - 23 are from case A.W.

From them there is neither much evidence of a mitral nor aortic lesion.

It is an interesting example, however, of the irregular duration of the diastolic periods - the

irregularity found in youth.

No. 22 - 23 are two tracings which I took with an instrument more suited to a child, and the last shews the venous tracing from neck along with that of the pulse. This shews well the beginning of a small auricular wave just in front of what is most likely the carotid wave. It occurs with fair regularity.

I think it is interesting in this case as pointing to a corroboration of some possible dilatation of the Right Heart.

This tracing also shews some irregularity of the diastolic period.

The three photographs are those of heart of A.G.

From a study of this subject, therefore, there occurs to me the following ideas:-

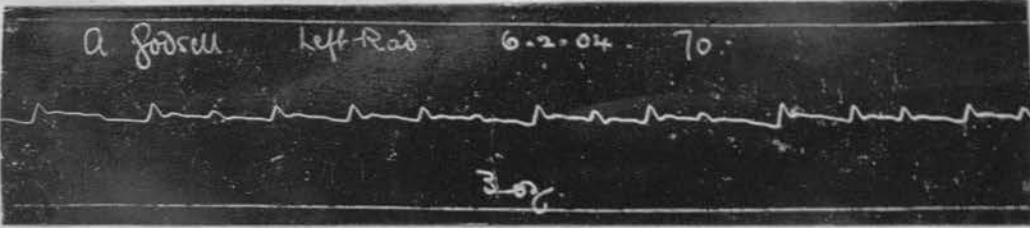
1. That an incompetence from organic disease at the Pulmonary orifice is very rare.
2. That it is difficult to diagnose with certainty (the number of reliable symptoms being small.)
3. That its diagnosis is still more obscure if there is present at the same time an Aortic lesion.
4. That functional incompetence is probably more common.

5. That it is also difficult of diagnosis.

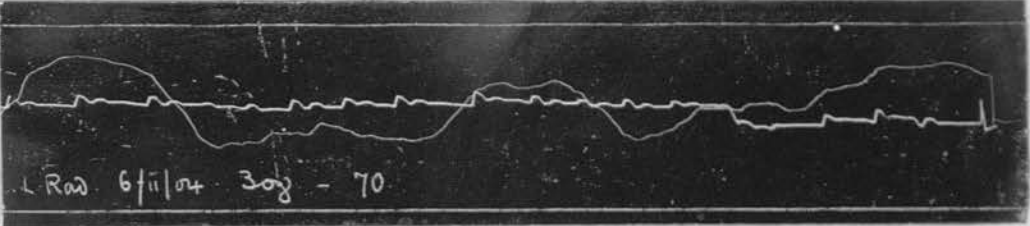
6. That a Post-Mortem may give no evidence of it - although it had been present in life. .

In conclusion, I have considered a cardiac lesion which is at least in one of its forms, comparatively rare. Nevertheless, its interest and in addition its gravity, shall cause me in future to give it my further attention.

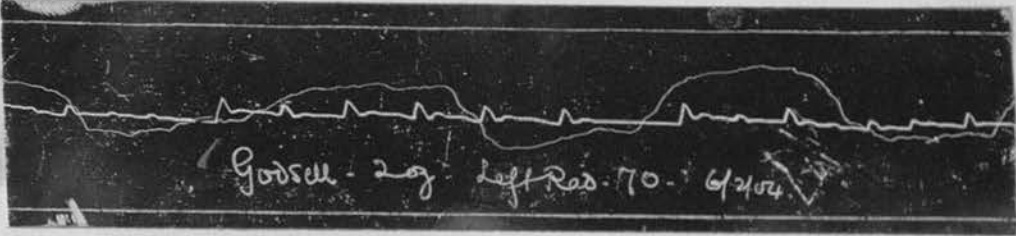
Sphygmographic Tracings.



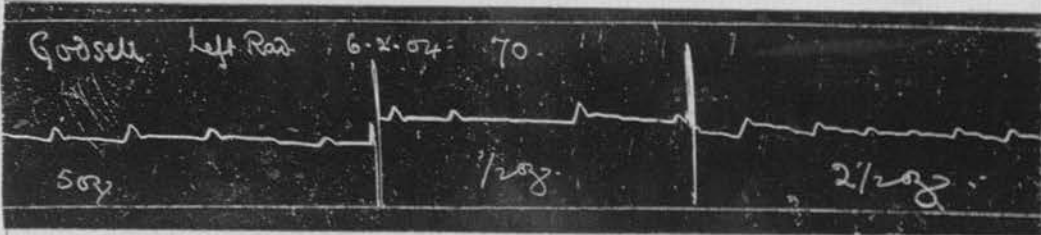
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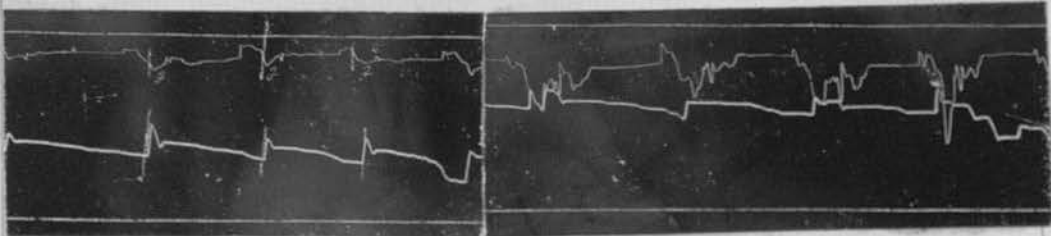
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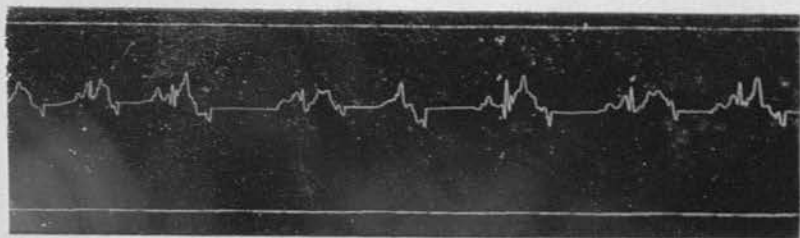
(3) Case A.S.



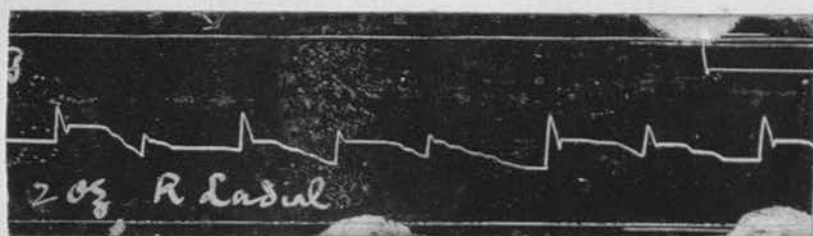
(4) Case A.S.



(5) Case A.S.



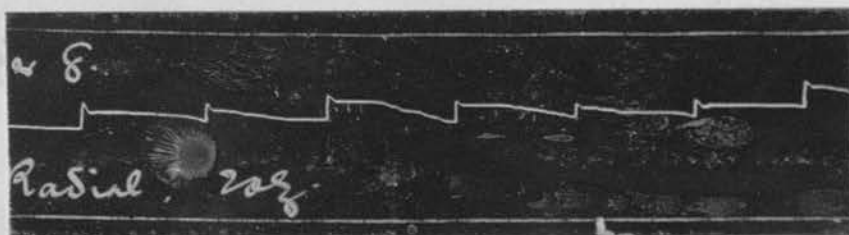
(6) Case a.f.



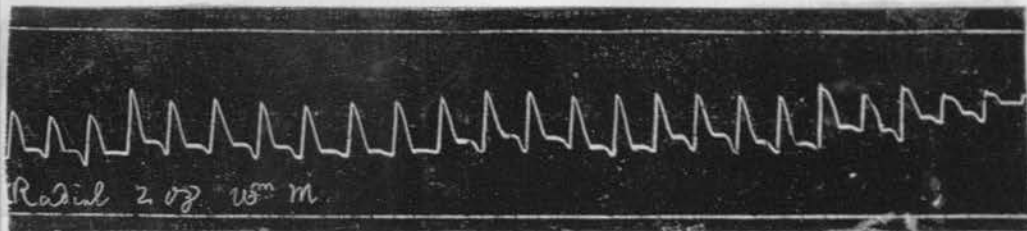
(7) Case a.f.



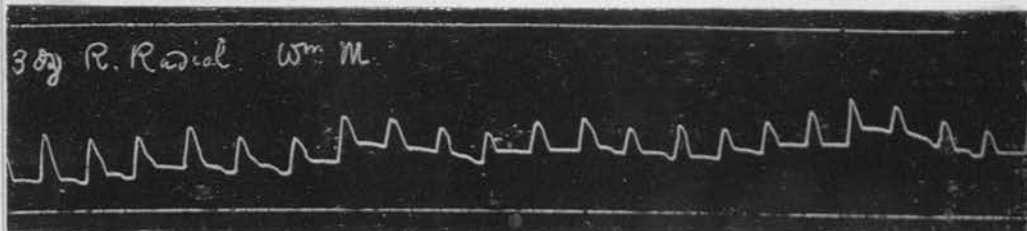
(8) Case a.f.



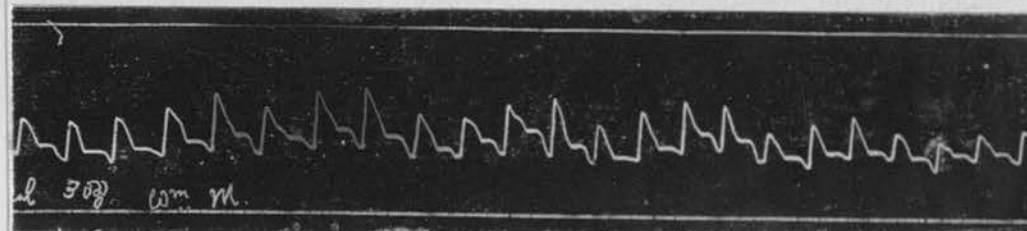
(9) Case a.f.



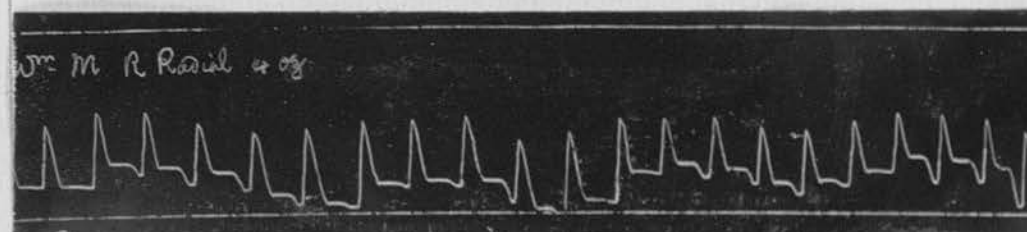
(10) Case W.M.



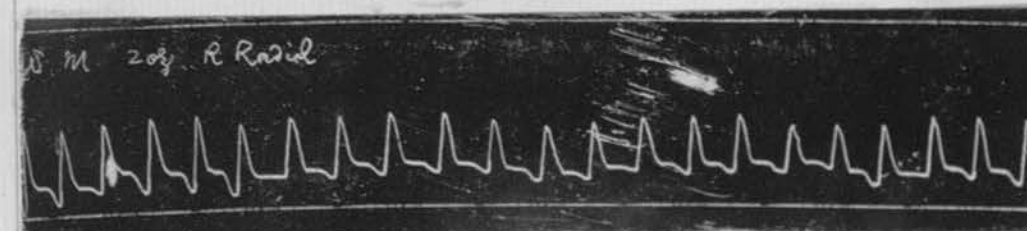
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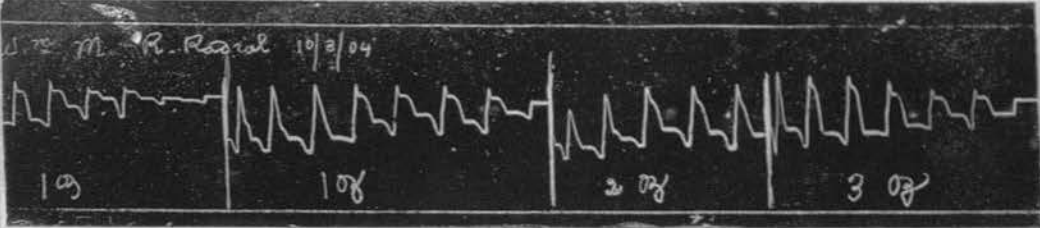
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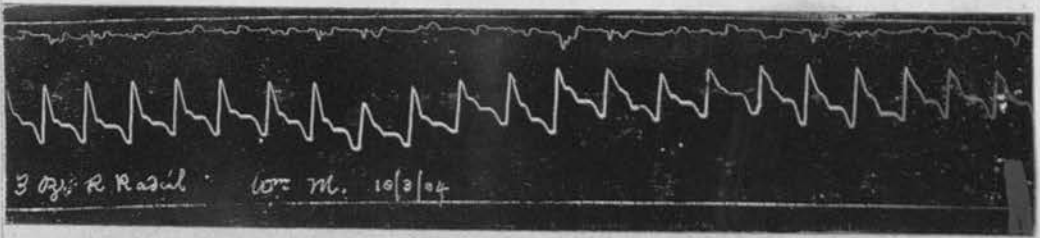
(13) Case W.M.



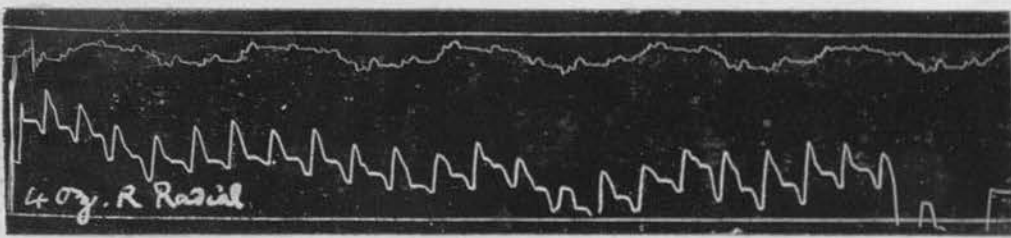
(14) Case W.M.



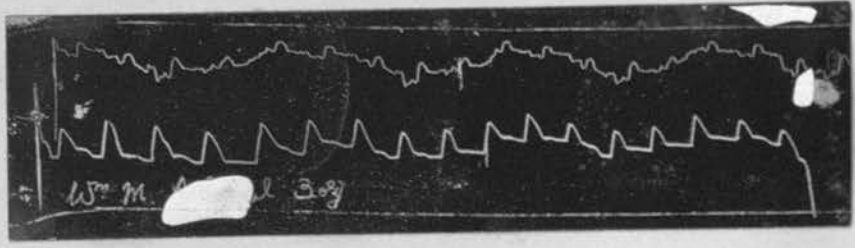
(15) Case W.M.



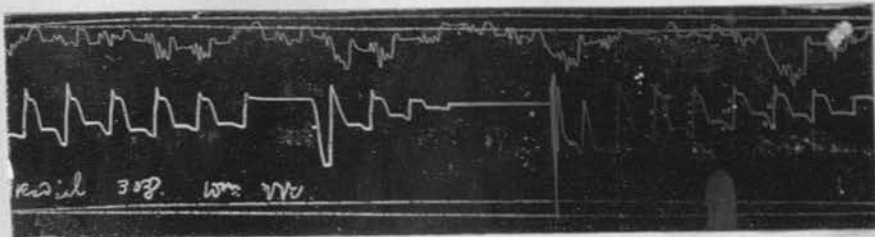
(16) Case W.M.



(17) Case W.M.



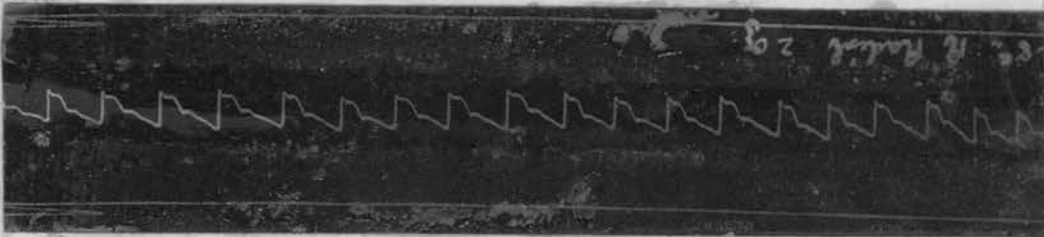
(18) Case W.M.



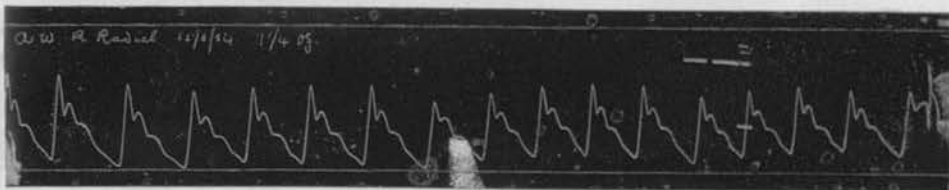
(19.) Case W.M.



(20.) Case a.w.



(21.) Case a.w.



(22.) Case a.w.



(23.) Case a.w.

I. Photograph of Heart of case Alex: G.,



Showing.

(α) The Pulmonary Orifice opened, also valves.

(β) Cavity of Right Ventricle and hypertrophy of its wall.

II Photograph of Heart of Case Alex: G. -



Showing. (a). The Aortic Valves, Aorta and Left Ventricle.
 (B). Ventricular Wall.

III Photograph of Heart of Case Alex: G. -



- Showing: (α) Dilatation of the Left Auricle
and stenosis of Mitral orifice.
(β) Extreme dilatation of the Right
Auricle
(γ) Hypertrophy of Auricular Walls.

Works &c consulted.

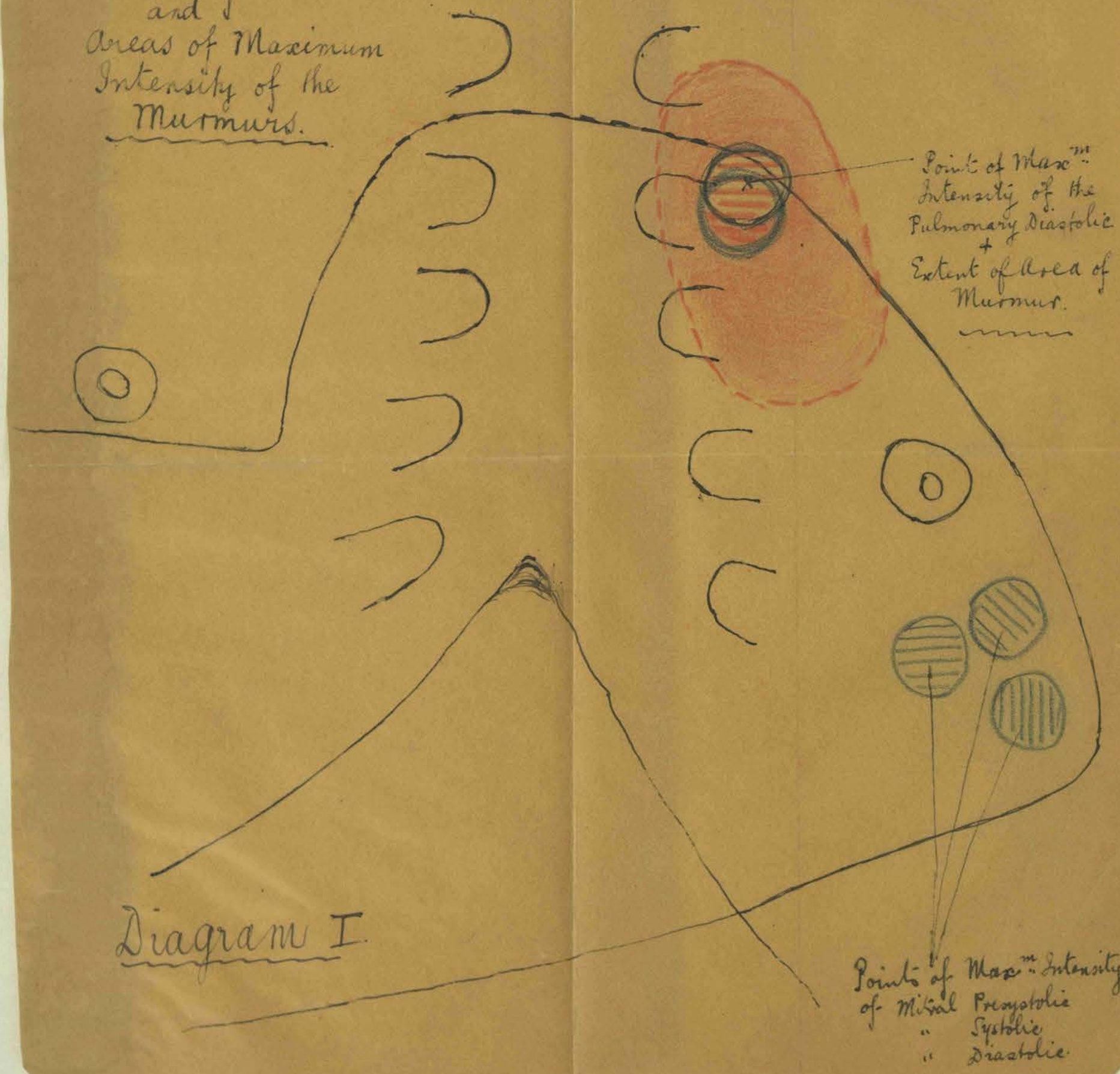
87.

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Case of Aec: G.

Outline of Heart
and
Areas of Maximum
Intensity of the
Murmurs.



Point of Max^m
Intensity of the
Pulmonary Diastolic
+
Extent of Area of
Murmur.

Diagram I

Points of Max^m Intensity
of Mitral Pre-systolic
" Systolic
" Diastolic.

Case of Arthur W.
Showing distribution
of
Murmurs.

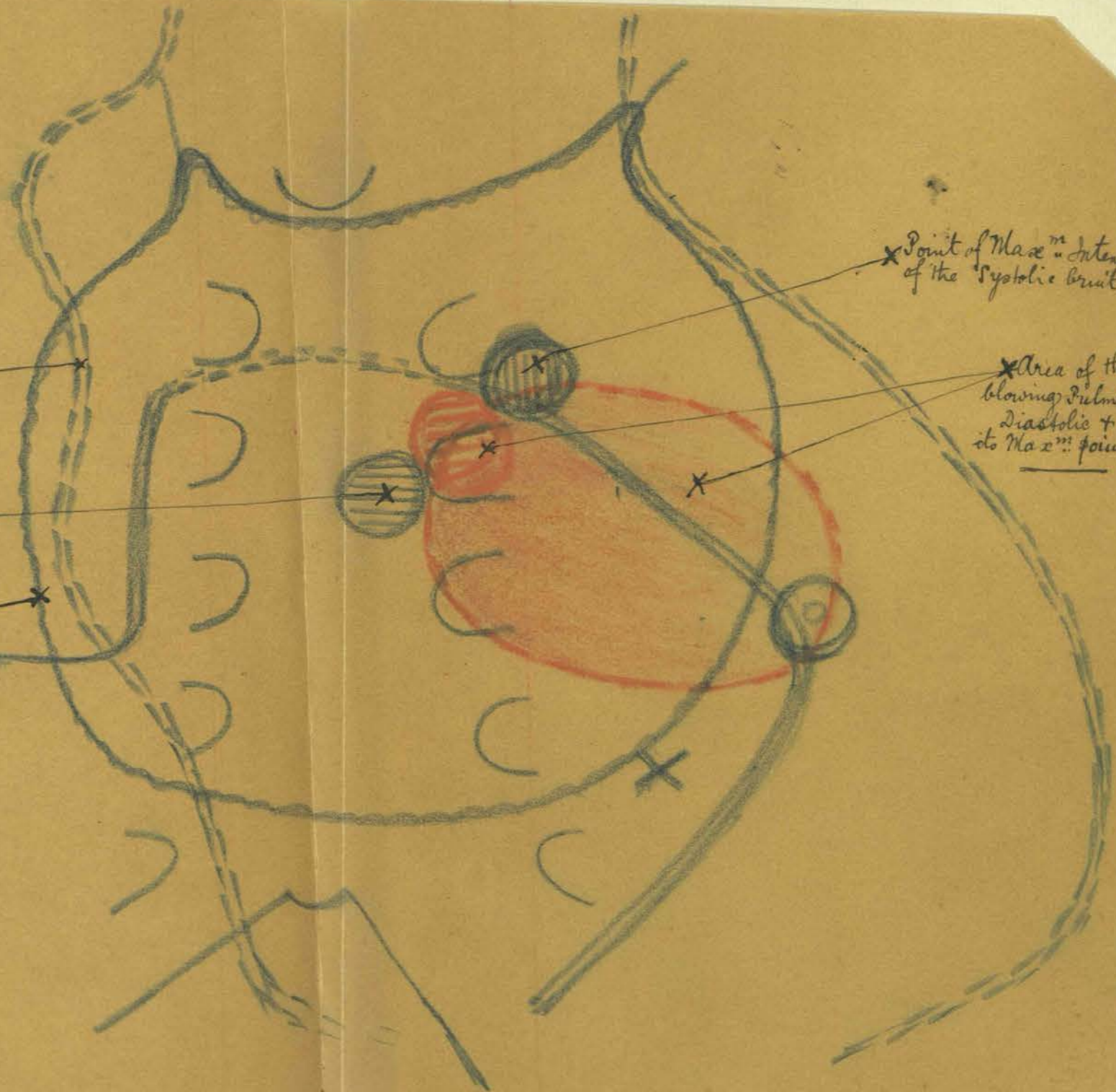
Outline of area of the
Systolic bruit.

Point of Max^m
Intensity of Musical diastolic
of
Area of audibility of
this bruit.

Point of Max^m Intensity
of the Systolic bruit.

Area of the
blowing Pulm.
Diastolic +
its Max^m point.

Diagram II.



Case of W. M.
Shewing point of the
Aortic Diastolic bruit.

Max^m point and distribution
of a systolic (haemic) bruit also
to be heard before death.

Point of Max^m
intensity of the
Aortic Diastolic & the
area to which it was
restricted.

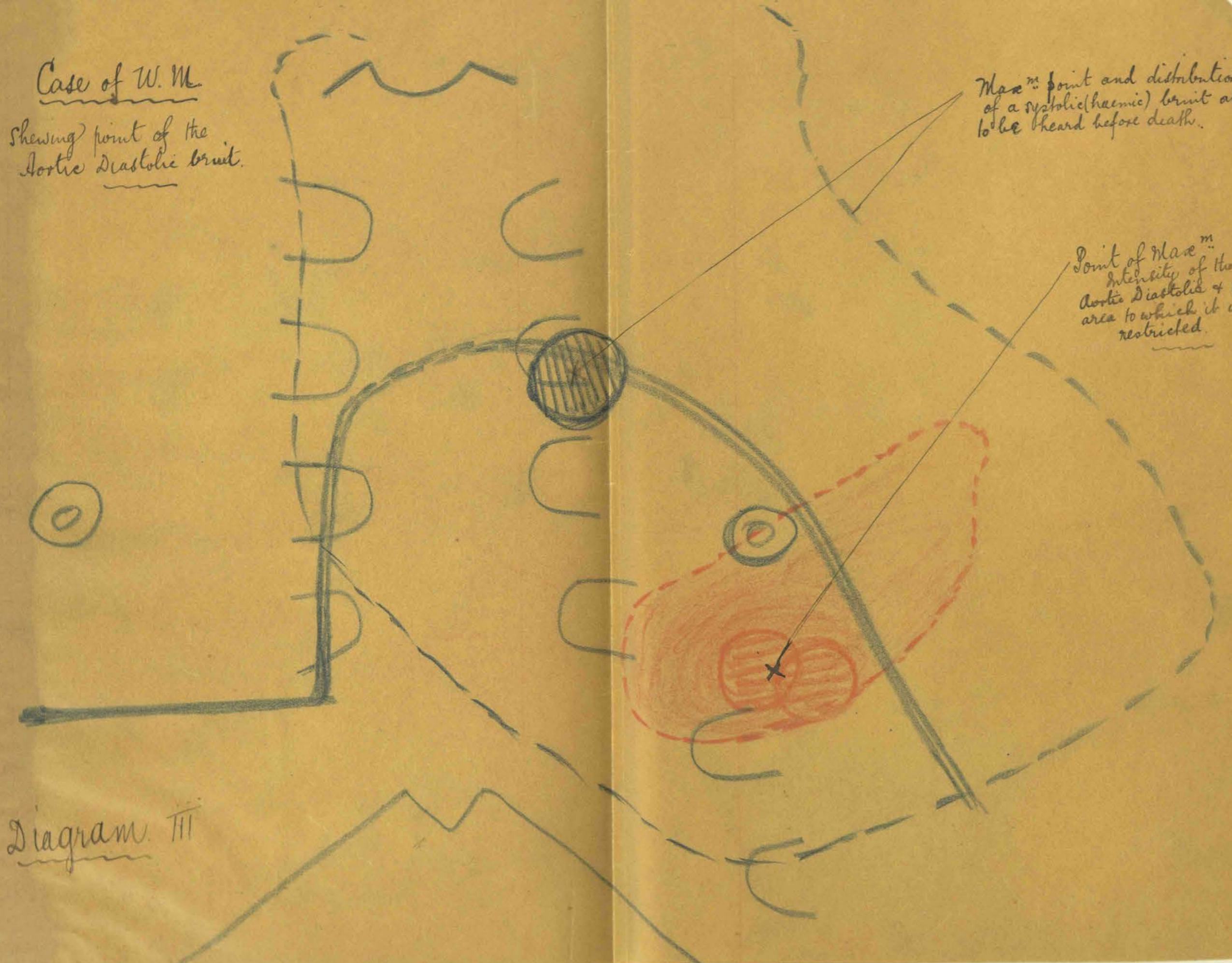


Diagram III