THE CIRCULATORY CONDITIONS

IN

ACUTE NEPHRITIS

AND

GRANULAR CONTRACTED KIDNEY

A Thesis presented to the Faculty of Medicine in the University of Glasgow for the Degree of Doctor of Medicine

by

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October, 1904

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C P Thomson.

THE CIRCULATORY CONDITIONS

in

ACUTE NEPHRITIS AND GRANULAR CONTRACTED KIDNEY

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This subject has occasioned much controversy since the days of Bright, and some points connected with it still remain undecided. My remarks are concerned chiefly with the clinical side of the question, and are intended merely as a few obiter dicta.

The literature is very extensive, but perhaps the most interesting of the more recent observations on the question are those made by Sir William Broadbent in his well-known book on the Pulse. There he states certain novel views on the influence of high arterial tension in Acute Nephritis and Granular Kidney, and it is my purpose to instance certain facts which would seem to me to be essentially opposed to some of his theories. And I do this with all the more diffidence when I recall the high terms of eulogy with which this book has been received by medical authorities in general, and by the late Professor of the Practice of Physic in our University in particular.

There is no need to enter into any description of the circulatory changes usually found in the acute and chronic varieties of renal disease. The manner of their production is still a matter of debate.

As I shall illustrate some points by pulse-tracings, some observations on the value of sphygmographic records might not be out of place. In these days the use of the sphygmograph is somewhat neglected, and this perhaps is the natural result of the extravagant expectations formed of it when it first appeared. At first it was expected to revolutionize the study of the pulse, and the diagnosis of all forms of cardiac disease. Now, almost every clinician sneers at it as a mere physiological toy, and an unfailing argument of the anti-sphygmographer is that it will yield any form of tracing desired by the observer.

It is certainly true that a tracing of very fair tension may be got from a pulse which is actually of low tension, and it is even said that a tracing of low tension can be got from a high tension pulse. A tracing of high tension from a low tension pulse is only to be obtained under certain conditions such as occur in Aortic Regurgitation, where the arterial tension is subject to such constant variation. A similarly fallacious result may also be got from a low tension pulse where the arterial wall is calcareous, or where large elastic vibrations occur. The possibility of this fallacy would be obviated, to some extent, by submitting several tracings, taken under different pressures, to illustrate the pulse, instead of selecting that particular one which the observer deems most typical of the pulse.

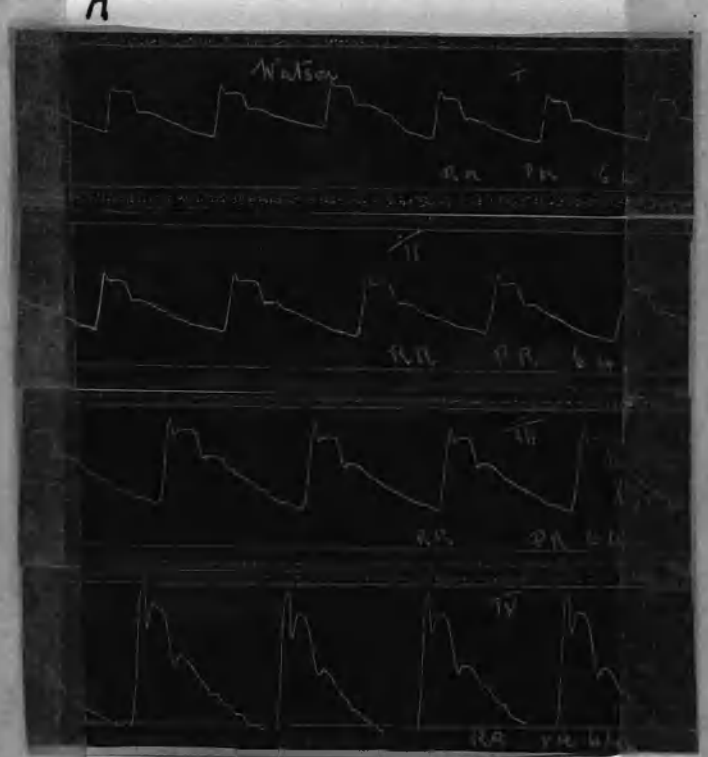
If tracings were taken under varying pressures, from that just sufficient to give a result to that which is just short of extinguishing the pulse wave, under no circumstances would all the tracings show the characters of high arterial tension where the pulse is

actually of low tension.

The influence of calcification of the arterial wall is a matter of importance in sphygmography. It is generally recognised that this calcareous deposition, in the middle coat of the artery, gives a square shaped tidal wave in a tracing, and this is best marked under low pressure on the part of the instrument. If the arterial tension is low, this square shaped tidal wave becomes much less prominent under higher sphygmographic pressure, while the aortic notch becomes more apparent; while, if the arterial tension is high, the square shaped tidal wave becomes more prominent and rounded.

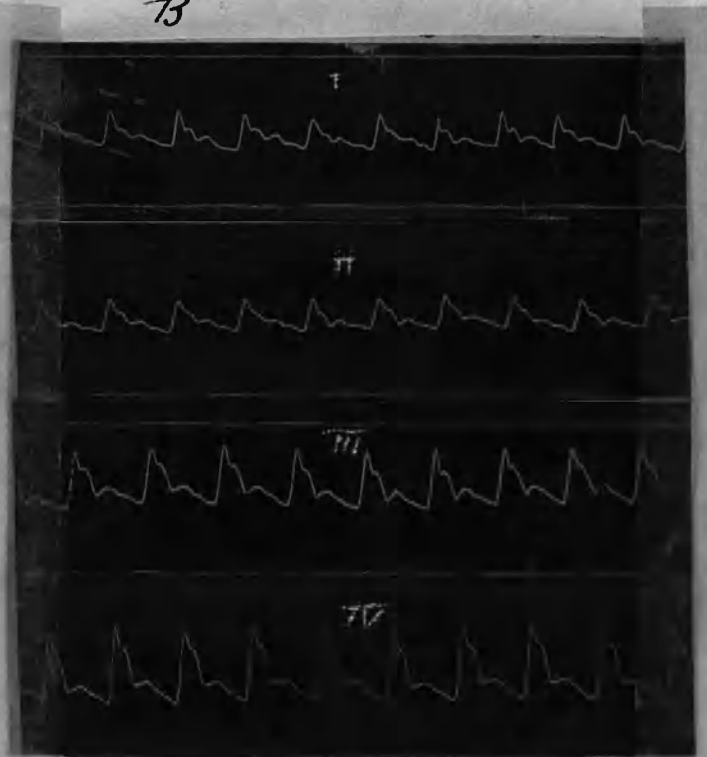
These points are illustrated in the appended tracings; all of these were taken from patients with well marked calcification of the smaller arteries, and they would seem to show that this change has really little effect on the form of the tracing.

A



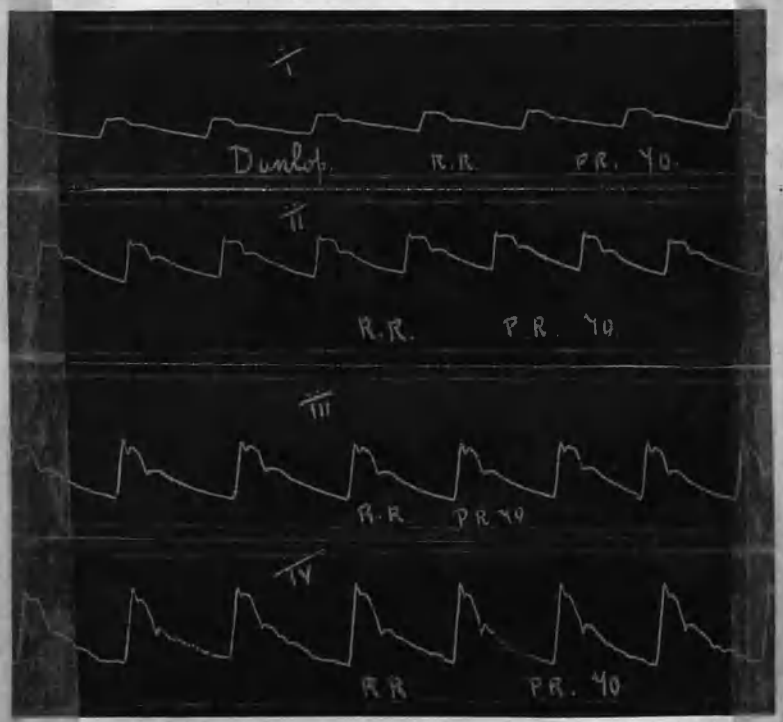
High Arterial Tension

B

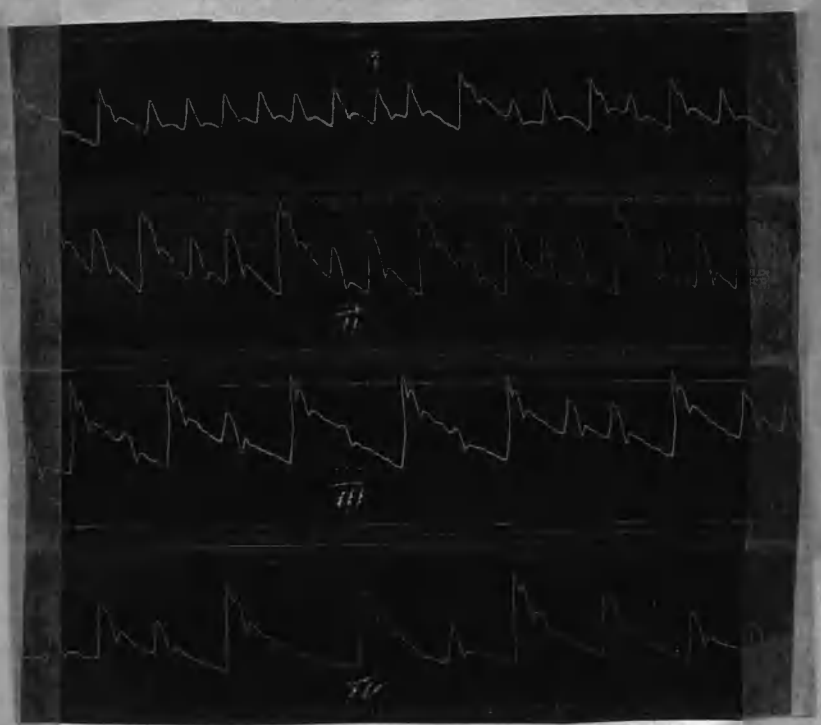


Low Arterial tension
Heart acting efficiently

C.



C.
Moderate Arterial Tension

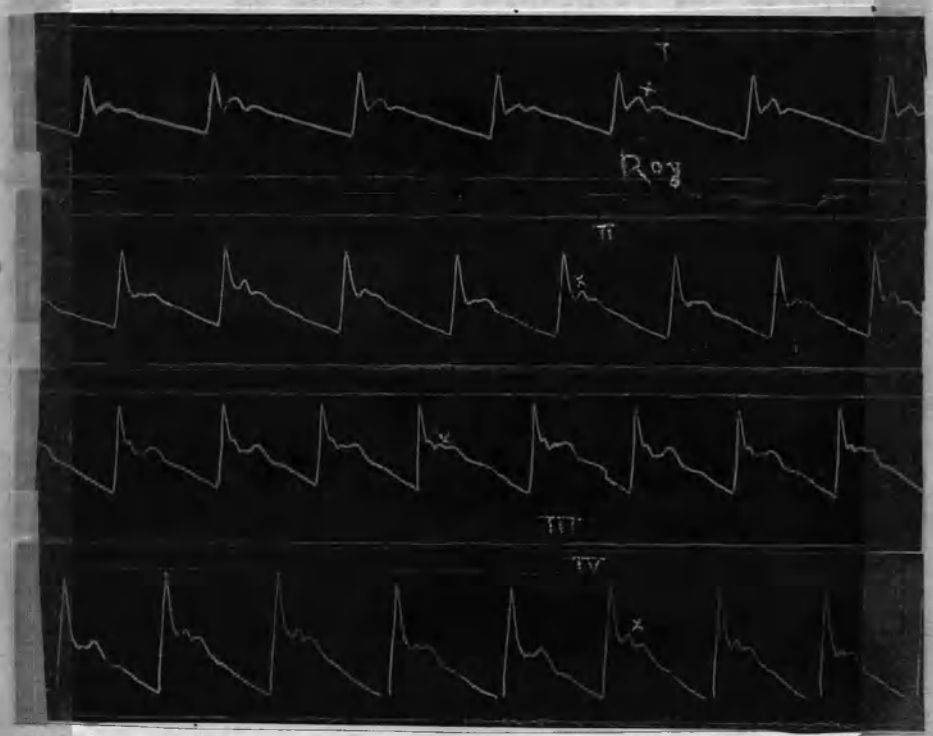


D. Low Arterial Tension
Cardiac "Breakdown".

E

E Low Arterial Tension
Cardiac "Breakdown"

Another factor which occasionally produces a deceptive appearance of enhanced tension in a tracing is the presence of large elastic vibrations on the vessel wall. Accessory waves are common on the tidal wave in tracings of the large high tension pulse of Acute Nephritis, but it is when the vibration gives a wave just before the aortic notch that a deceptive appearance of increased tension is produced. This is not common, and when found it is seen to be best marked in tracings taken with low sphygmographic pressure.



The wave given by the elastic vibration is marked with a cross. In no IV it is seen to be well marked every alternate couple of beats — during inspiration.

It is a point of some importance to determine whether or not a pulse of high arterial tension may give a tracing of low tension. Several authors state that this is so, and Mahomed, in his article on the Clinical Aspects of Bright's Disease, submits a tracing of very low tension, which he says, was taken from a high tension pulse.

He insists that a sphygmogram is only a graphic record of the pulse wave, not of the fulness of the vessel. Now, arterial fulness depends partly on the state of contraction of the arterioles and capillaries, but principally on the vigour of the cardiac action, while the pulse wave also depends most largely on the character of the ventricular systole, though it is influenced to some extent by certain other factors, including the arterial fulness. The relations, therefore, are somewhat complicated and interdependent, but as both the fulness of the artery and the nature of the pulse wave depend so much on the vigour of the cardiac action, it is difficult to see how the arterial tension can remain high in a circulation where the ventricular systole is so feeble that the pulse wave gives a tracing characteristic of low tension.

THE PULSE IN ACUTE NEPHRITIS

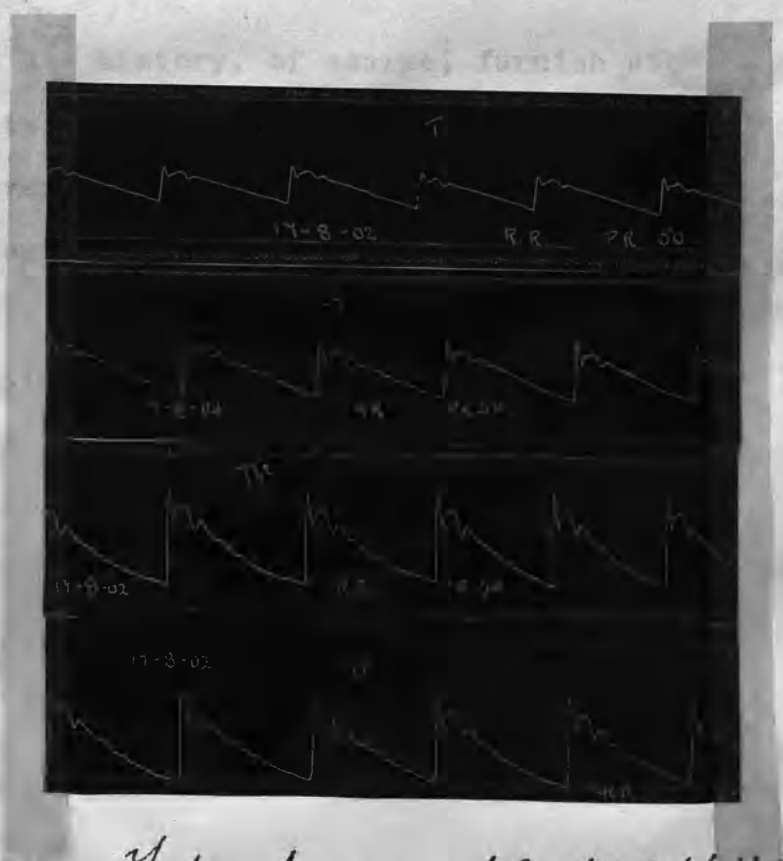
The typical pulse of Acute Nephritis is the large pulse of high tension.

In the period of pyrexia, which is commonly found in the first few days of acute nephritis, the pulse is of low tension, though not extremely so, and it is more full between the beats than is common with a febrile pulse.

From a case of Acute Nephritis: Taken on the third day of the disease. Temperature 100·5°F.

This stage is of short duration, and then the circulatory conditions favourable to the occurrence of oedema are produced. The necessary condition is that of hydraemic plethora, and the main cause of its production is the paralysis of the urinary secretion, a more or less constant feature at the beginning of Acute Nephritis. Broadbent says that "for some time after the oedema appears, the pulse, though full between the beats, is yet compressible and lacking in push, mainly on account of the weakness of the heart" (The Pulse, p. 250). This is so in children, especially in those in whom the disease ensues as a complication of an acute febrile disorder, but when Acute Nephritis occurs in the case of a healthy adult, the pulse from the first appearance of oedema is large, long, and not easily obliterated. The artery itself is large and, as a rule, undegenerate.

From a case of Acute Nephritis
Taken on the 4th day of the disease
Temperature Normal



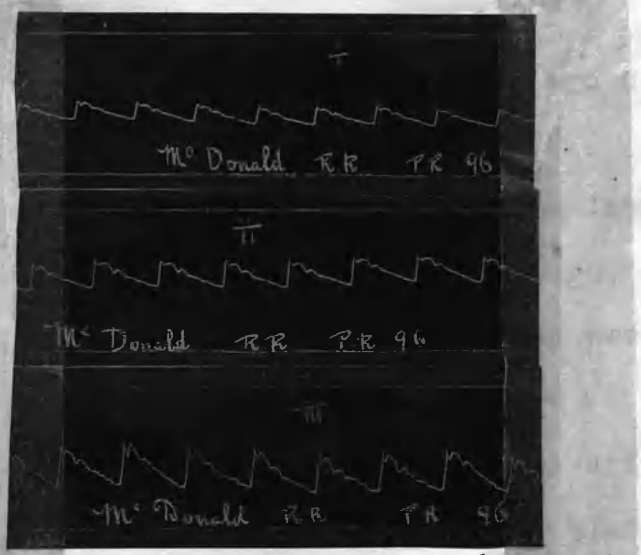
Taken from a case of Acute Nephritis
on the 6th day of the disease
Temperature normal

This large pulse of acute nephritis is found generally during the course of the affection. In the first stage the greater the degree of anuria, the higher is the tension of the pulse, but when once the renal function is re-established, it is impossible to strike any relation between the arterial tension and the urinary output. The amount of albumen in the urine, too, has little discoverable relation to the arterial tension, and the only general rule which can be laid down with regard to these conditions is this, that the grade of albuminuria declines as the urinary secretion is increased.

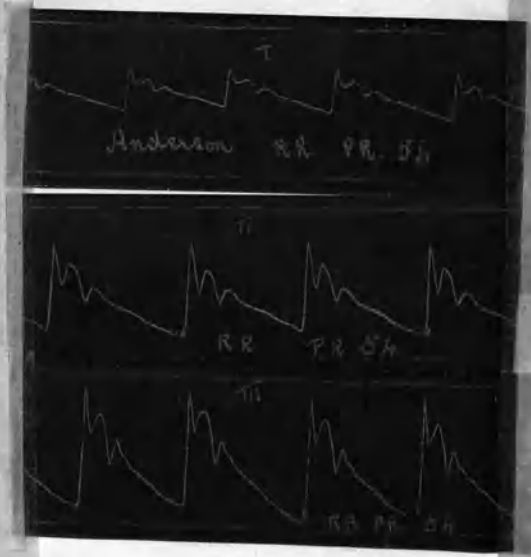
In differentiating Acute Nephritis from Granular Kidney, the pulse furnishes useful indications. This question in diagnosis only arises, of course, when the chronic condition has superimposed on it an acute attack.

The age of the patient and the history, of course, furnish useful information, but there are two factors of great importance to which investigation ought, in every case, to be directed. These are, the state of the circulation, and the appearance of the fundi oculorum.

If much reliance is to be placed on the examination of the circulatory system, it must be made soon after the onset of symptoms, for it is not long before some of the signs become identical. With regard to the pulse itself in acute nephritis, the artery is larger and more compressible, and the pulse wave not so sustained as in renal cirrhosis: in the latter case the artery is small, and its coats usually thickened.



The small Pulse of High Tension
Granular Contracted
Kidney.



The Large Pulse of High Tension
Acute Nephritis

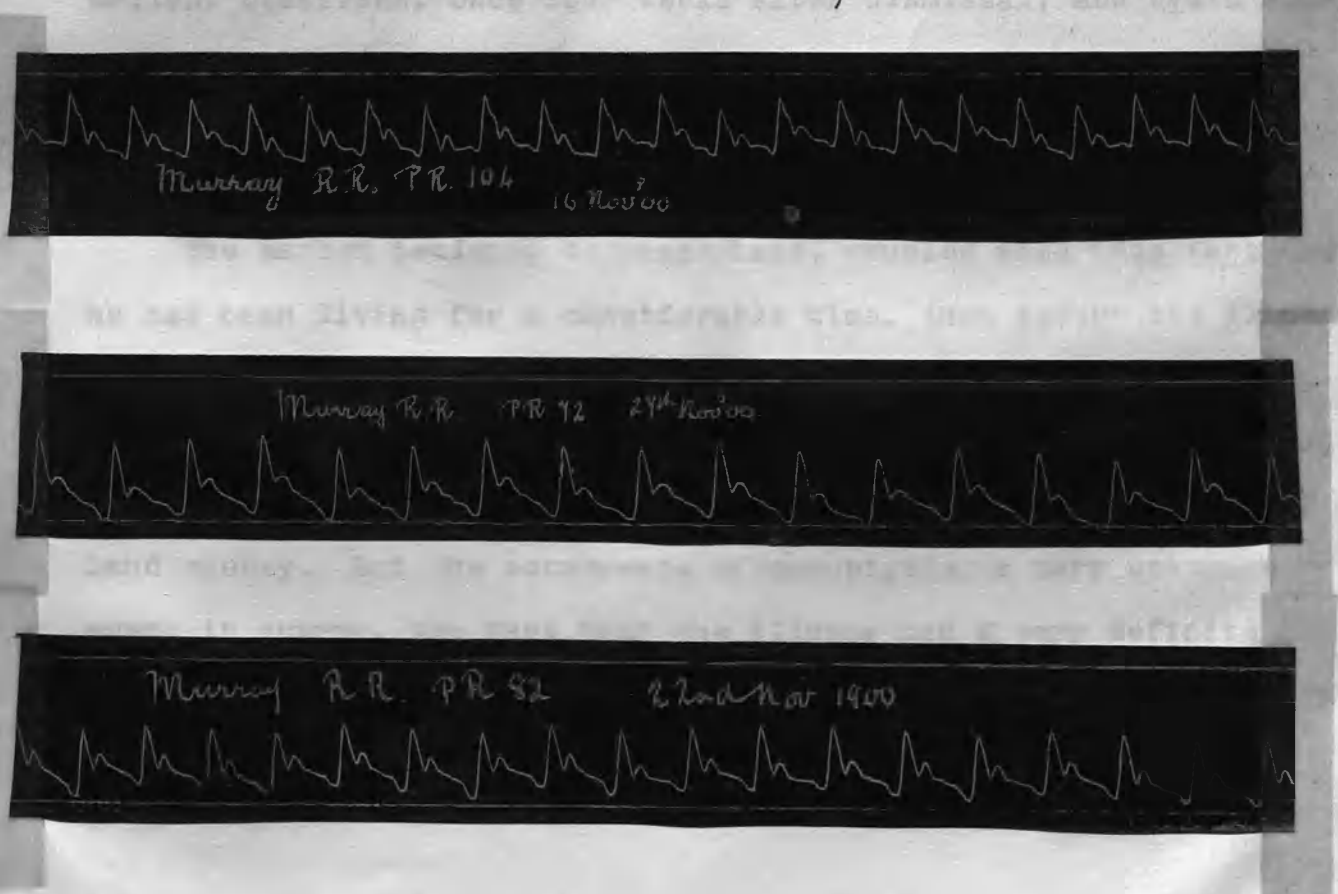
Manifest physical signs of cardiac hypertrophy would be in favour of a diagnosis of Granular Kidney.

In the great majority of cases retinal changes of any sort indicate chronicity of the renal affection; this is invariably true of White Spots and Optic Neuritis, while haemorrhages, if seen alone, are indicative, according to Saundby (Lectures on Bright's Disease, p. 178), of an insidiously beginning or latent chronic condition. Certainly fundus changes occur with extreme rarity in Acute Nephritis; most authors, indeed, state that they never occur. Therefore, any deviation from the normal appearance of the fundus, in a case which may otherwise seem to be acute, must be taken into account, and the settlement as to the chronicity of the disease must be decided by the subsequent course of events. I have seen a small haemorrhage in the eye-ground of a patient in whom the previous history,

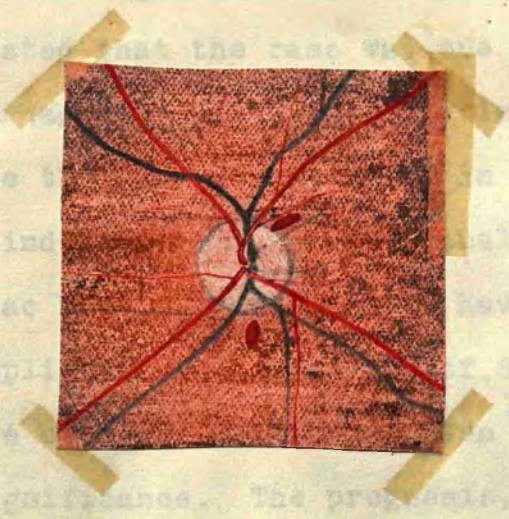
physical examination, and ultimate issue all pointed to renal disease of an acute type.

This patient was a labourer, aged 40, who came under observation complaining of cough, shortness of breath, spitting of blood, headache, weakness, and swelling of the feet and arms of seven weeks' duration.

On examination the urine was found to have a specific gravity of 1022. It contained blood, and albumen was present to the extent of 35% (Esbach's method). Microscopic examination showed the presence of numerous blood and epithelial tube casts. There was some oedema of the wrists and ankles. On auscultation of the lungs, many crepitant rales were heard all over the chest on both sides, front and back. The sputum was frothy, abundant, and uniformly tinged with blood. There was no evidence of cardiac hypertrophy, and the pulse was regular and soft. The temperature was normal.



Examination of the fundi showed the presence of two small flame-shaped haemorrhages close to the right disc.



The case was treated as one of Acute Nephritis, and he was dismissed from hospital, after a stay of two months, practically well. Examination of the urine then showed the presence of blood and albumen in very minute quantities. The urine was examined on two subsequent occasions, once four weeks after dismissal, and again some six weeks later, and on both of these occasions it presented a healthy character, the examination giving negative results.

This case presented several interesting points.

The marked tendency to haemorrhage, coupled with this fact that he had been living for a considerable time, just before his illness began, in primitive quarters on a diet consisting chiefly of tea, bread and butter, and ham, and from which fresh vegetables were entirely absent, suggested the possibility of the case being one of land scurvy. But the occurrence of haemoptysis, a very uncommon event in scurvy, the fact that the illness had a very definite onset, the absence of spongy gums, foetid breath, and subcutaneous haemorrhages all tended to negative this view.

The only circumstance which weakened the diagnosis of Acute Nephritis was the occurrence of retinal haemorrhages. This, of course, suggested that the case was one of Granular Kidney. But retinal haemorrhage in Granular Kidney is due to high arterial tension, and here the tension was low. In addition, the previous history gave no indication of chronic renal trouble, and there was no sign of cardiac hypertrophy. It may have been a case of Acute Nephritis complicated by an element of Scurvy.

The pulse of Acute Nephritis often affords information of some prognostic significance. The prognosis, says Broadbent, is determined "by the response of the left ventricle to the demand for increased contractile energy, and the way in which the pulse develops actual tension" (The Pulse, p. 251). Certainly many of the cases of Acute Nephritis, which show from the first a marked development of arterial tension, ultimately recover, yet many become chronic, and it is no bad sign if the pulse remains throughout of moderate or even low tension so long as this low tension pulse is not the result of dilatation of the left ventricle.

The early development of high tension in Acute Nephritis would seem to be no very desirable thing, for it is found in its greatest degree when the diminution of the urinary secretion is extreme, and then presumably there is excessive congestion of the organs. High arterial pressure will sensibly increase the determination of blood to the inflamed organ by increasing the rate of the blood stream, and the result will be prejudicial.

The patients in my series of cases, who advanced most speedily

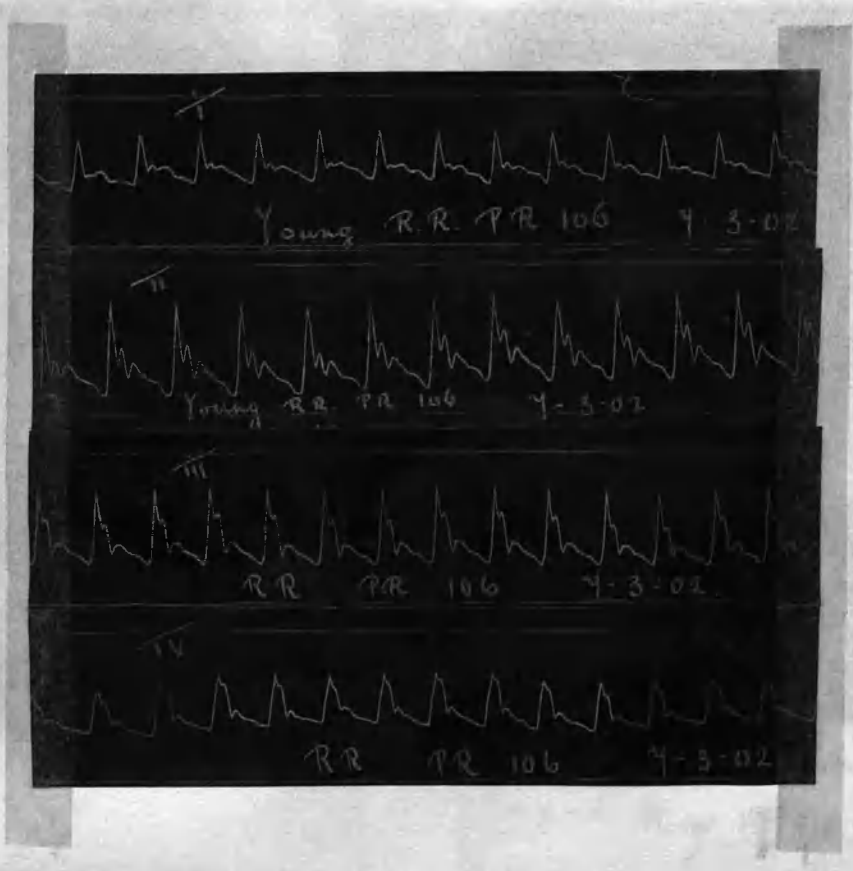
to complete recovery, were those in whom the arterial tension was low during the whole time that they were under observation; and, further, in cases originally showing high arterial tension there was usually a manifest lowering of pressure as the renal function reapproached the normal.

When it is said that cases with low tension pulses speedily improve, it is not meant that the circulatory conditions are the cause of the improvement. The relationship is simply this, that when the kidney function is not seriously interfered with, there is not that accumulation of poisons necessary to cause much capillary contraction and there is not that distension of the circulatory system which results from paralysis of the secretion.

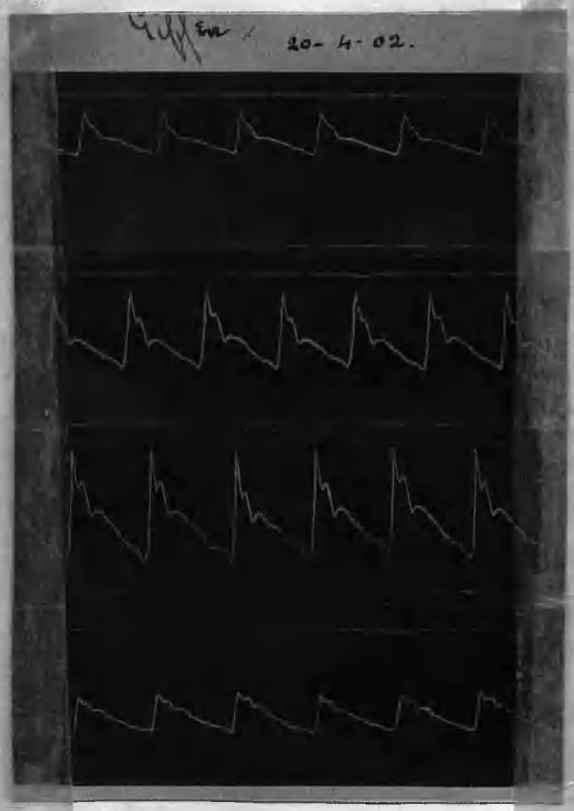
Two clinical facts may be deduced from this:- first - that in such instances the oedema is often trifling and transitory: second - that the urine contains few casts and other renal derivatives.

Clinical observation bears this out and, apart from dilatation of the heart, low arterial tension in Acute Nephritis means a mild attack.

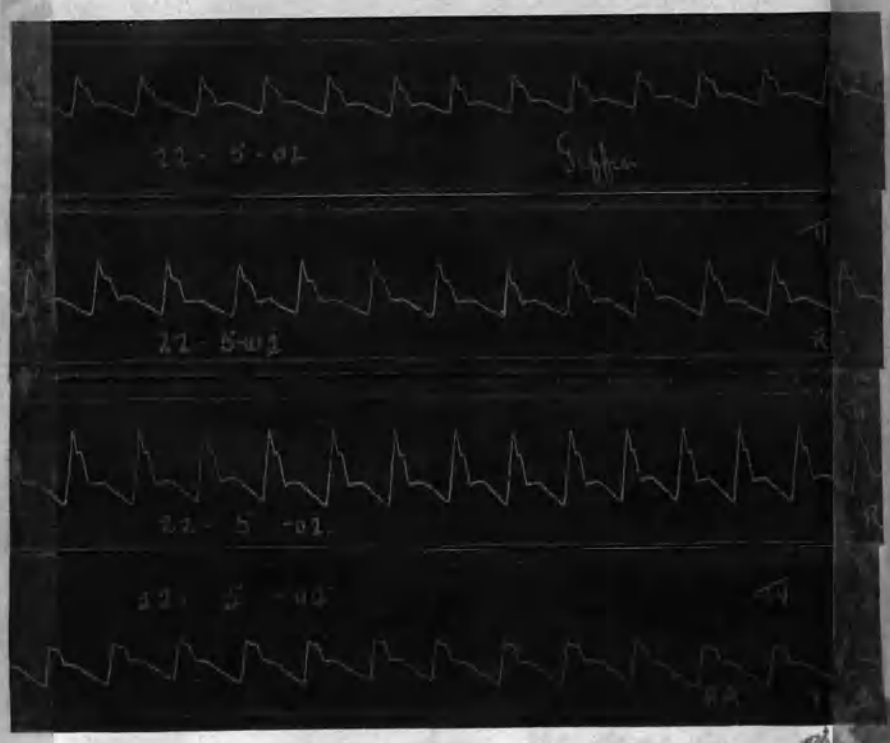
These points are illustrated in the following notes and tracings:-



This set of tracings was taken from a patient, aged 31, at the beginning of an attack of Acute Nephritis; the initial œdema was slight, and the diminution in the quantity of urine was not extreme, the average output for the first three days he was under observation being 42 oz. (he came under observation on the third day of his illness). In the course of four weeks he was perfectly well again, and his urine was absolutely normal without any trace of albumen, blood, or renal derivatives.



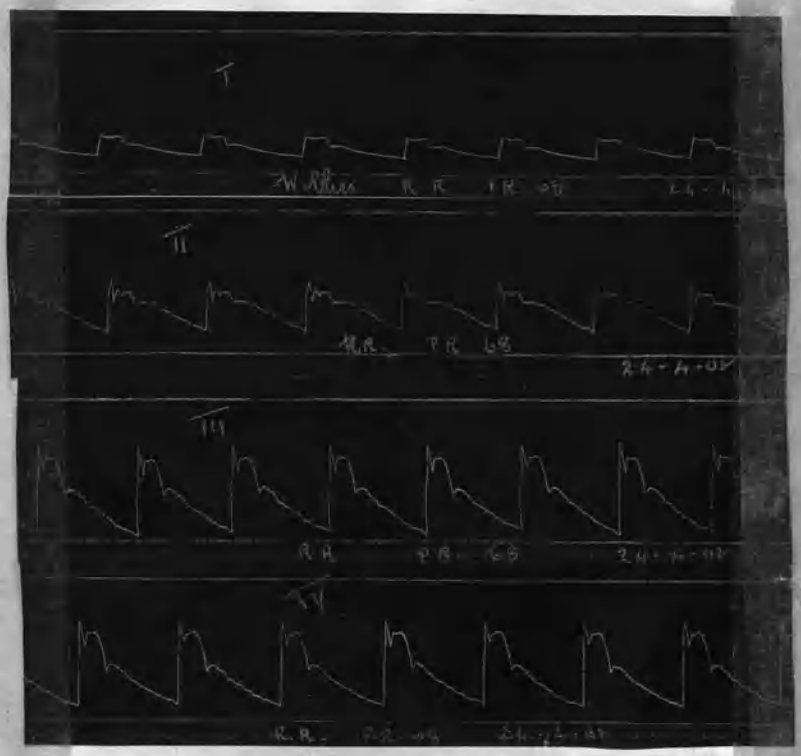
A. Taken on 20th April 1902.



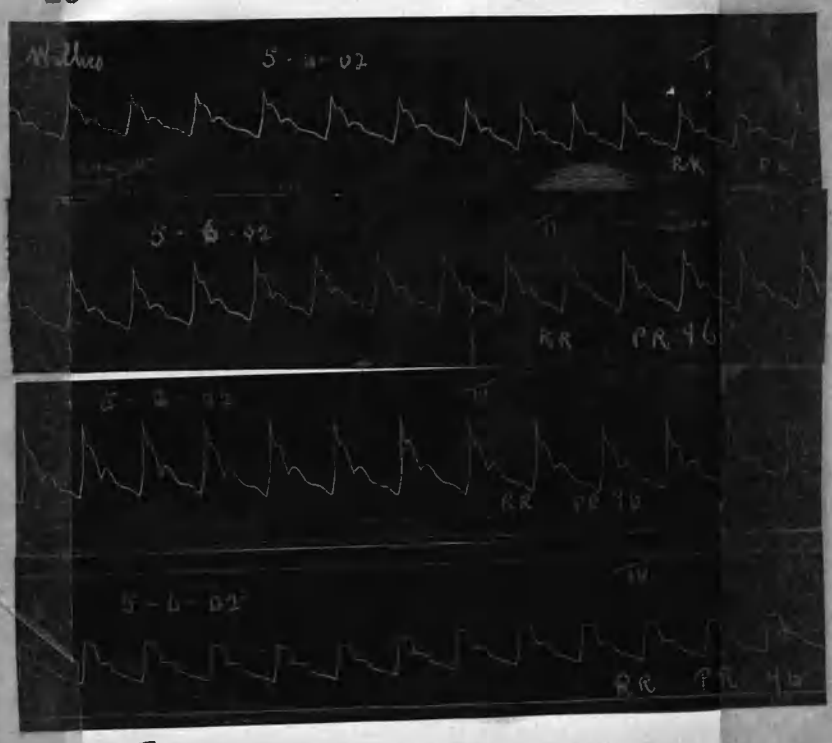
B. Taken from same case 22nd May 1902.

These tracings which show a pulse of low tension, were taken from a patient who recovered in six weeks from Acute Nephritis, the first set being taken on the first week of his illness, the second set a month later.

The initial symptoms in this case, too, were slight, and recovery was speedy.



A. Taken on
24th April 1902.



B Taken from the same case on
5th June 1902.

This patient, a man aged 34, came under observation with a well-marked attack of Acute Nephritis. The symptoms and the examination of the urine pointed to severe inflammation of the kidneys. The pulse tension at first was high: under treatment the case speedily improved and, after a stay of nine weeks in hospital, he was dismissed practically well, albumen and blood being present in the urine in very minute quantity. The tracings show that a marked declension in arterial tension had taken place as he improved.

These remarks suggest another point of some interest in Acute Nephritis. It is well known that convalescence is heralded by a marked increase in the amount of urine excreted. This occurs suddenly, as a rule, and is accompanied by some striking changes in the

appearance of the urine. The amount of blood becomes notably less, the sediment is less dense, loses its chocolate colour, becomes light and flocculent, and contains fewer renal derivatives. These changes in the urine, and the increase in its quantity, are due to a decrease in the renal congestion.

Tirard gives this explanation (Albuminuria and Bright's Disease, p. 111), and from the method of treatment he adopts in these cases he must have had many opportunities of observing its spontaneous occurrence.

Broadbent (The Pulse, p. 251) attributes it to an increase in the degree of pressure in the arterial system, the result of cardiac hypertrophy. But cardiac hypertrophy does not take place in a few days, and this increased urination is always comparatively sudden. In cases of Acute Nephritis, which resist treatment, it is sometimes seen after cardiac hypertrophy is established.

These remarks, as well as those made in connection with the prognostic significance of low arterial tension, do not apply to cases in which there is dilatation of the left ventricle. This is not a common event in Acute Nephritis; at least, it does not often occur at such a time or to such a degree as to influence the course of the disease.

A condition of acute dilatation of the left ventricle is described by some clinicians as occurring commonly at the very outset of the disease, at the same time as the oedema, and as due to the same cause, namely, hydraemic plethora. This is probably seen most frequently in children, and especially in scarlatinal nephritis, where the affection comes on at a time when the heart muscle is

weakened by the stress of the specific fever. But, in the case of adults of sound constitution, it is uncommon and, in any case, if it does occur with any frequency, it is not of much significance, for the heart speedily becomes capable of meeting the increased resistance in the arterial system by the occurrence of hypertrophy.

But dilatation of the heart, taking place during the course of the disease, and after the heart has made some response to the call for increased energy, is a matter of gravity. Some degree of hypertrophy has then probably taken place, and dilatation of the left ventricle is an indication of the heart's inability to continue the struggle against the heightened peripheral resistance.

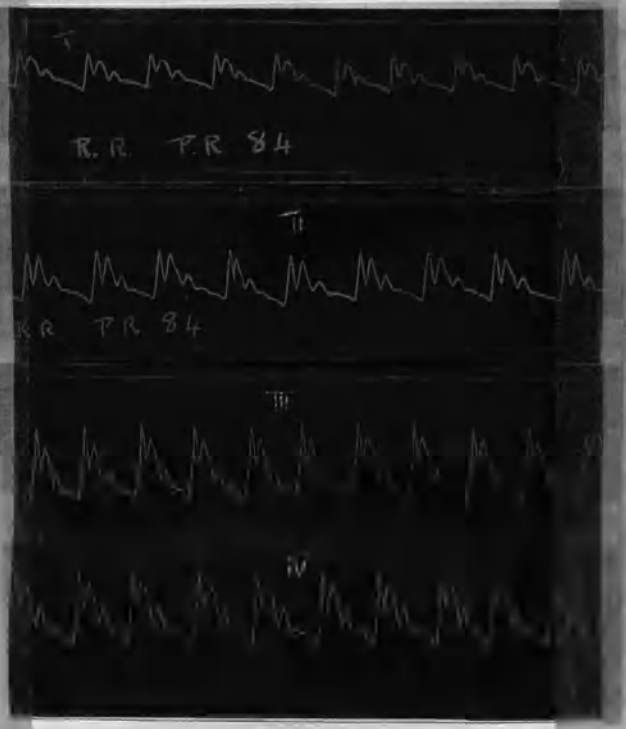
Gibson is of opinion (Diseases of the Heart and Aorta, p. 696) that retention of waste products plays the chief part in causing dilatation of the left ventricle in this disease, and it will be found in most cases of dilatation in Acute Nephritis that there has been a notable diminution of the urinary secretion over a considerable period of time. The presence of the retained waste products is not the only factor. The overdistension of the circulation, which results from diminished urination, plays an important part and, in some cases, it will be found that the heart muscle was either originally feeble, or had become so by the influence of acquired factors, such as over-indulgence in alcohol.

When dilatation of the left ventricle occurs, indications are at once afforded by the urinary chart. The amount of secretion, probably already low, falls still further, and the grade of albuminuria rises.

The physical signs obtained on examination of the heart are just such as would be found in any case of dilatation.

The pulse, however, presents some peculiarities. It rises in rate, and loses in tension. It becomes more vehement than before, is very sudden in onset and disappearance, and is easily obliterated. The sphygmograph yields a peculiar tracing; the upstroke is short and upright, the tidal wave losing that rounded form which is deemed typical of high arterial tension, become sharp pointed and, if anything, more prominent; the dicrotic wave occurs lower in the descent line, is sharper, and much more developed. The peculiarity of the tracing is the presence of a prominent tidal wave in a tracing which has otherwise the characters of low arterial tension.

A.
From a case of Acute Nephritis
Pulse previously of High Tension
The above set of tracings was taken during an attack of Sore-Throat attended by moderate pyrexia
Temperature 100-8.



B. Taken from a case of Acute Nephritis
Pulse previously of High Tension
This set of tracings was taken at the commencement of dilatation of the Left Ventricle
Temperature normal

As will be seen from the above tracings, the record is very similar to that to be obtained when a moderate grade of pyrexia occurs in a case of Acute Nephritis where the arterial tension is high. In the latter case, the arterioles and capillaries are dilated, while the heart continues to act vigorously. In the condition under consideration, the position of affairs is exactly reversed; the peripheral resistance is maintained, while the vigour of the cardiac systole is impaired.

This illustrates a statement made by Foster (Text Book of Physiology, p. 165) that inasmuch as "the pulse is the expression of two sets of conditions, the same features of the pulse may obtain under totally diverse conditions, provided that these conditions effect both factors in a compensating direction".

The prognosis is bad; if the patient does not die of uraemia, he will probably become the subject of chronic parenchymatous nephritis. The condition demands energetic treatment, and this should be directed not to the heart but to the kidneys. The first indication is to establish free diuresis, and I may remark that I have never seen dilatation of the heart occur in this disease where a copious output of urine has been obtained early; this should be regarded as the first and most necessary thing to be obtained in the treatment of Acute Nephritis. And when dilatation of the left ventricle has occurred, this is more necessary than ever. Cardiac tonics, such as digitalis, are very useful in the treatment of the dilated and failing heart of chronic nephritis, and it might appear that the exhibition of digitalis would be indicated here inasmuch as its action is diuretic as well as cardio-stimulant. But, since the diuretic ac-

tion of digitalis depends on its effects on the renal vascular system, either by dilating the renal vessels or by constricting them, and so causing an increased rate in the blood stream through the kidneys, it is inadvisable, theoretically, to appeal to a drug which would cause a greater determination of blood to an already inflamed organ. And actual practice bears this out, for it will usually be found that the most notable result of the use of digitalis in Acute Nephritis is to cause an increase in the amount of albumen excreted.

Alkaline diuretics may be exhibited with advantage, but as Dickinson remarks "Water is the best diuretic". A copious fluid dietary rigidly enforced will, in practically all cases, ensure a profuse output of urine in the course of a few days. The patients, in whom I have seen dilatation of the left ventricle in this disease, have all, for some reason or another, failed to follow out this line of treatment.

Tirard is opposed to the practice of stimulating the kidney in the early stages. He says (Albuminuria and Bright's Disease, p. 109), "During the early stages, it is well to keep the nourishment to the smallest amount possible .... as the diuretic influence of milk is not to be desired so long as there is any large proportion of blood in the urine." And he withholds alkaline diuretics in this stage for the same reason, that their use increases the renal congestion. With regard to the action of alkaline diuretics, most authorities are agreed that that action has no relation to the renal circulation, but is exerted on the renal cells by stimulating their functional activity. Actual practice determines the fact, too, that uræmia is less common when the urine is kept alkaline by their use. While the copious ingestion of liquids flushes out the kidney, and

sweeps clear the choked tubules.

The following is a typical case of Acute Nephritis with dilatation of the left ventricle, and illustrates some of the foregoing remarks.

A. McC., aged 14 years, was admitted into hospital on 30th November 1902, presenting the typical features of an attack of Acute Nephritis of four days' duration. He remained in hospital for one week, but left at the end of that time, as he did not like the restricted diet. Six weeks afterwards he was re-admitted. There was then very extensive oedema involving the whole surface of the body; the peritoneum contained much free fluid, and there was fluid in both pleural sacs. The urine was very scanty, the average daily output of the first week after re-admission being 16 oz. The heart presented obvious physical signs of dilatation. The pulse on the occasion of his first stay in hospital was of fairly high tension (see tracings). On re-admission it was fast, 102 per minute, full between the beats, the pulse waves being sudden in onset and subsidence.

For the next month energetic treatment was carried on. Almost constant purgation was obtained by the use of magnesium sulphate. Digitalis was administered to stimulate the heart. A mixture containing potassium acetate and citrate was given, but there was no improvement in his condition. During this month the abdomen was tapped on three separate occasions, 312 oz. of serous fluid being withdrawn altogether. There was no notable increase in the urinary secretion. The pulse had fallen in tension, and the area of cardiac dulness had increased $\frac{1}{2}$ inch in the transverse diameter. Thus,

after being ill for more than eleven weeks, and after being under treatment for one month, he was still suffering from dilatation of the left ventricle with extensive œdema. During this month he had steadily refused to drink water in any quantity.

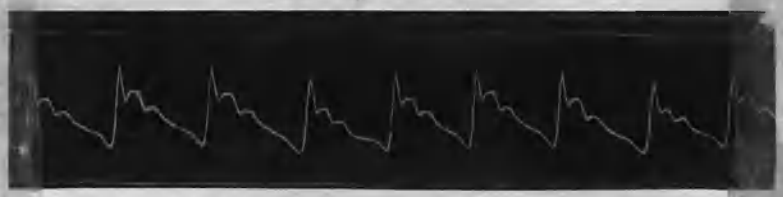
From this time stringent measures were adopted. The hot packs were stopped, the digitalis and alkaline diuretics were omitted, and a copious liquid dietary was enforced. He was made to take $2\frac{1}{2}$ pints of milk, 1 pint of barley water, and 3 pints of potus imperialis in the day. Diuresis was speedily established, and in twelve days all trace of œdema, both subcutaneous and in the serous sacs, was gone. The urine contained much less blood, albumen and renal derivatives; the pulse had gained greatly in tension while falling in rate, and the transverse measurement of cardiac dulness was materially less. Three weeks later he was dismissed almost well.

On dismissal only the faintest trace of blood and albumen could be discovered in the urine, and the cardiac condition was normal.

This case illustrates the effect of paralysis of the urinary secretion, and persistent œdema in causing dilatation of the heart. It shows, too, that dilatation of the heart of this type is best treated by inducing profuse diuresis, and that of all diuretics water is the best.

The improvement in the pulse under treatment is shown in the appended tracings

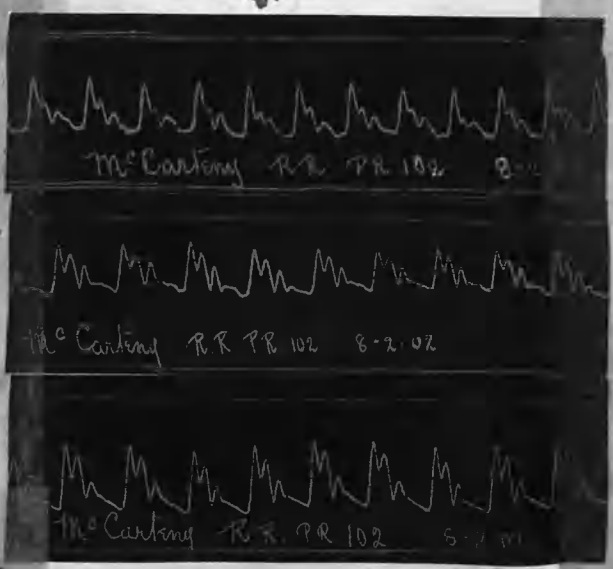
A



24

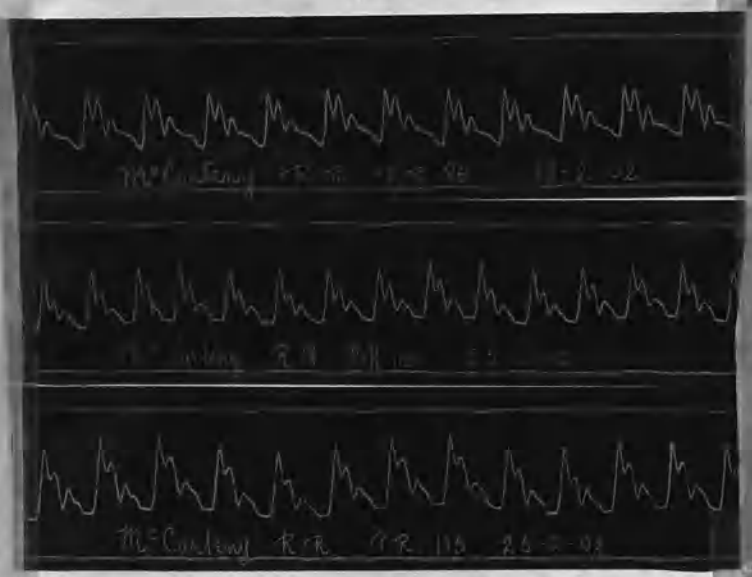
A/ This tracing was taken during his first stay in hospital on 4th December 1901

B



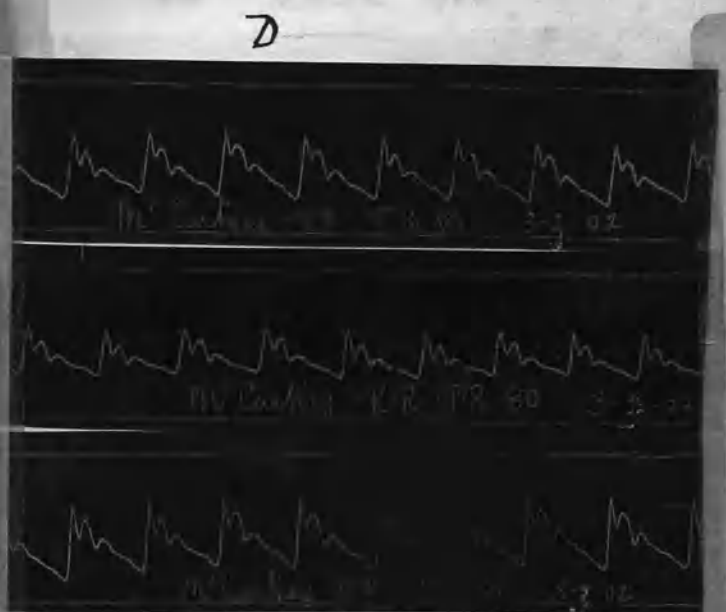
B/ Taken after dilatation of the left ventricle had taken place on 8th February 1902.

C



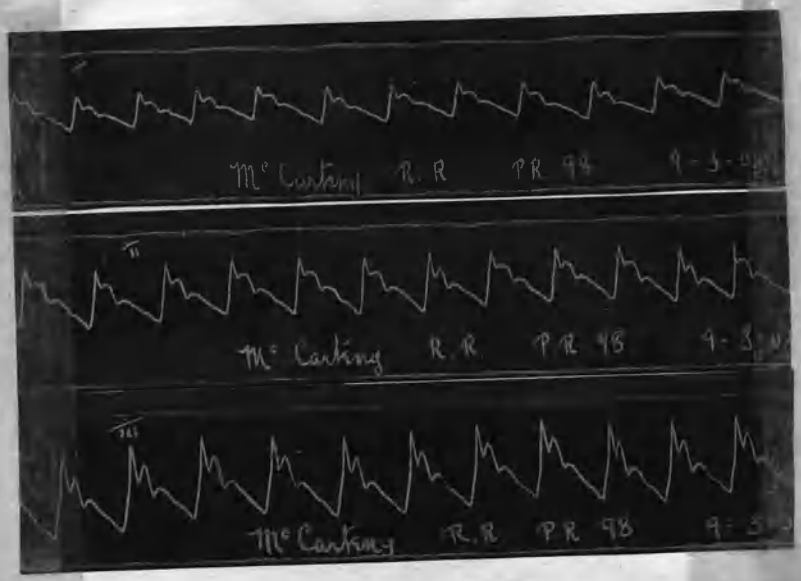
C/ Dilatation of the left ventricle still present in spite of rigorous treatment Taken on 25th February 1902

D



D/ Taken on 3rd March 1902 four days after the "water" treatment, described above, was begun. The improvement in rate and tension is manifest.

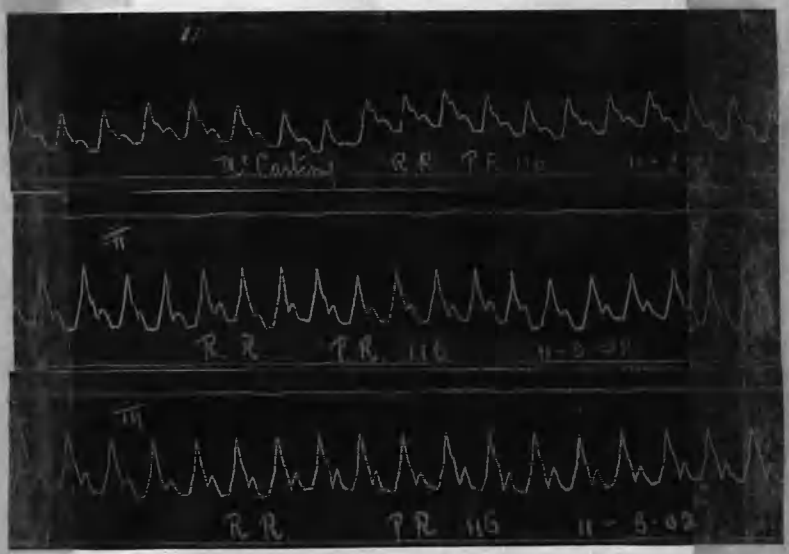
E



Taken on 9th March 1902

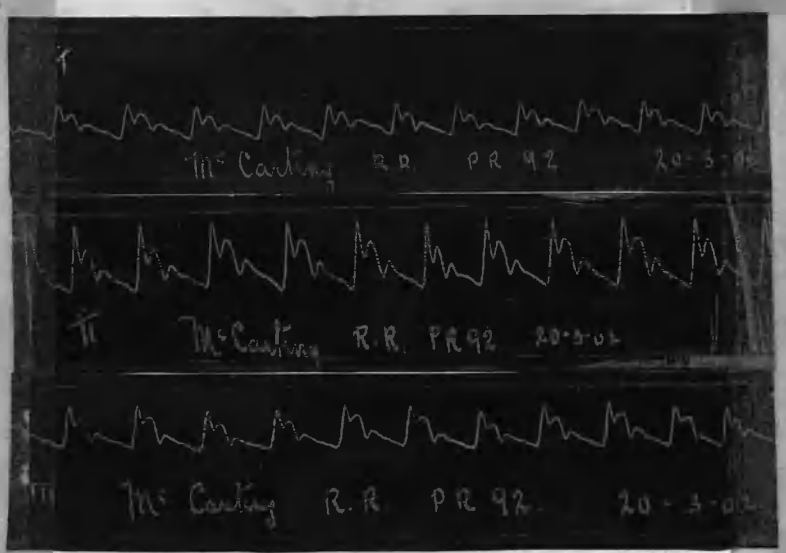
The improvement is maintained

F



F Taken on 11th March '02 on the first day he was allowed to get up. This low tension tracing is indicative of the part played by Cardiac systole in maintaining arterial tension. It also shows how feeble the heart was in this case.

G.



G Taken 20th March 1902, a few days before dismissal

CHRONIC INTERSTITIAL NEPHRITIS

It is in this disease that high arterial tension is most frequently found, and in its greatest degree. Indeed, it is very rarely absent, and in the few instances in which it has been my experience to find it so, the disease has been associated with chronic bronchitis and emphysema.

Mahomed was of opinion that high arterial tension was the essential symptom of the disease, and he contended that the kidney changes and the high blood pressure were both produced by some poison circulating in the blood. Whether or not the relationship is as Mahomed thought, high arterial tension certainly plays a very important part in the clinical history of renal cirrhosis. The course of the disease is prolonged, and its manifestations somewhat protean, but two great types of the disease may be separated:-

First - Those who come under observation suffering from symptoms of defective renal elimination: this we may call the uraemic type

Second - Those whose first symptoms point to cardiac failure.

This latter class might conform to what Osler calls the arterio-sclerotic kidney in distinction to the "primary renal affection"; but it is very doubtful if the renal lesion is ever secondary to arterio-sclerosis. It seems to me that there is no essential difference between these two types, the distinction being simply this, that in the "arterio-sclerotic" type the heart is not able to continue the struggle against the peripheral resistance long enough for uraemic

symptoms to develop.

The essential feature of the arterio-sclerotic type is the presence of calcareous change in the medium and smaller size arteries, and whether this precedes the cirrhotic change in the kidney, or follows it, the symptoms developed will relate at first, at least, to the circulatory system.

For this calcification of the middle coat of the smaller arteries implies several other things. It usually means that the patient is the subject of some chronic intoxication, such as alcohol, lead, or syphilis. Further, it implies almost invariably that there is atheroma in the larger vessels, and that calcareous change rather than fatty degeneration has taken or will take place in these atheromatous patches.

The coronary arteries are probably affected in a similar way with atheroma in the intima, and calcification of the middle coat, and the heart muscle is probably the seat of fatty degeneration or interstitial fibrosis.

Under these circumstances, the heart might well find difficulty in supplying all the needs of the organism, even if no further burden were put upon it. But, with the occurrence of cirrhotic change in the kidneys, its work is immensely increased. The capillaries contract, the internal coat of the arterioles shares in the general thickening which takes place all over the arterial system, and so their lumen is narrowed. A call is thus made on the heart for increased energy of contraction to meet the peripheral resistance. And in its damaged and handicapped condition, it soon gives way, and the first symptom in these cases is oedema. This oedema is

cardiac in type, and is found in the dependent parts.

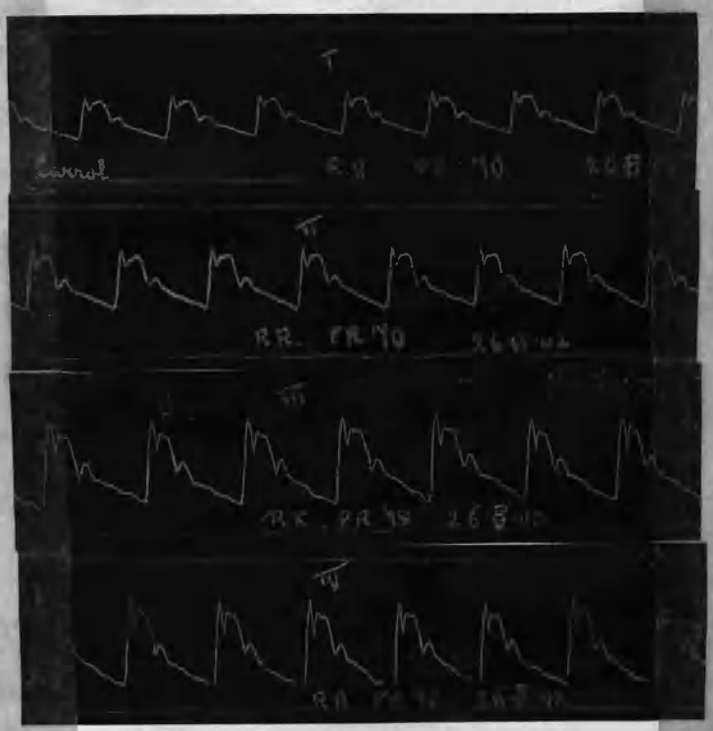
The following case illustrates this stage of the arterio-sclerotic type.

J.C., aged 40, Carter, was admitted complaining of breathlessness and palpitation on exertion, and of swelling of the feet and legs. The breathlessness and palpitation were of ten days' duration; the oedema had been noticed first four days before admission. He made complaint of no other symptoms, and said that till the onset of this illness, ten days before admission, he had enjoyed perfect health. There was no history of headache, vomiting, diarrhoea, or of failure of vision. For years he had indulged in alcohol to excess. The accessible arteries were notoriously degenerate, being thickened, tortuous, uneven, and rigid, with local bulgings. The transverse measurement of cardiac dulness was $3\frac{1}{2}$ inches. The apex beat was visible in the fifth interspace, $\frac{1}{4}$ inch inside the nipple line. On palpation it was diffuse and feeble. In the apical region the first sound was toneless, and was clearly duplicated. There was slight accentuation of the second sound in the aortic region. There was no murmur anywhere. The pulse was irregular in rhythm from the occasional occurrence of missed beats; the pulse wave was fairly long, and subsided slowly, but did not require much pressure to obliterate it. The urine was acid, sp. gr. 1012, contained albumen to the extent of .15% (Esbach). A slight flocculent sediment was deposited, and this contained some hyaline and granular casts.

Under the influence of rest and a fluid dietary, the oedema disappeared in two days. The urinary output rose, and the albuminuria became fractional. The pulse became quite regular after a week, and

a great improvement was noted in the character of the heart's sounds. After a stay of three weeks he was dismissed practically well.

A. Taken on admission 4th August 1902 An abortive beat is marked by a cross.



Taken from the same case three weeks later: There is shown a marked improvement in tension and no irregularity of rhythm is seen.

The absence of symptoms of poisoning with unexcreted products is to be noted here. Oedema is commonly supposed to be seen only as a terminal symptom in Granular Kidney, but where there is much arterial degeneration, it is almost invariably the first complaint. Under the influence of rest, this oedema quickly disappears, and the patient is able to resume the ordinary course of his daily life. But sooner or later he returns with all the symptoms of cardiac breakdown, and in its advanced stage this type of case often presents a condition which closely resembles the failing heart of mitral regurgitation of rheumatic origin. But a case of Granular Kidney with failing heart usually presents some symptoms of chronic

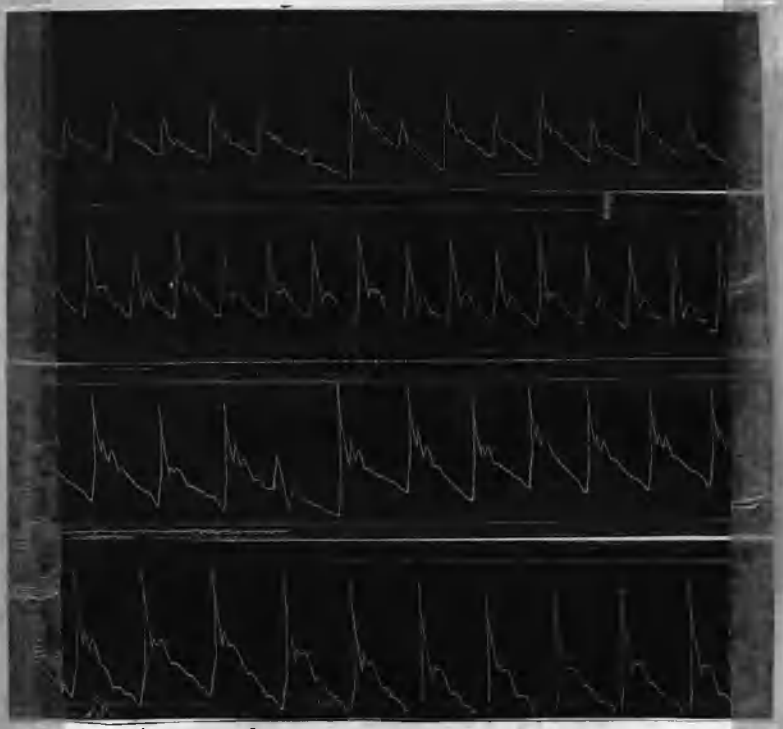
poisoning such as uraemic asthma, Cheyne-Stokes breathing, headache, etc., and these conjoined with the peculiar circulatory conditions are usually sufficient to make the diagnosis clear.

Sansom refers to this point in differential diagnosis as a matter which sometimes perplexes the tyro. But occasionally it is a matter of some difficulty. The points to which attention ought to be directed are laid down by Mahomed in his article on "The Essential Symptoms of Bright's Disease", and subsequent investigation has added little to these. The history must be carefully inquired into, and changes in the fundus of the eye sought for, but it ought to be possible in all cases to make the diagnosis by examination of the circulatory system alone.

The artery of the dilated renal heart is always visible, tortuous, hard, and calcareous. The muscular coat, or what remains of the muscular coat of the artery, is worn out, therefore the pulse is large; the capillaries are still contracted, so the vessel is still somewhat full. The heart is hypertrophied and dilated, and the aorta has lost its elasticity; the ventricular systole is short and sharp; the pulse wave follows quickly on the systole as no time is lost dilating the inelastic aorta; and the pulse beat is vehement and unsustained.

In the case of the dilated heart with mitral regurgitation of rheumatic origin, the coats of the artery are little altered, the vessel is starved, and the pulse wave is small, feeble, and more irregular in rhythm.

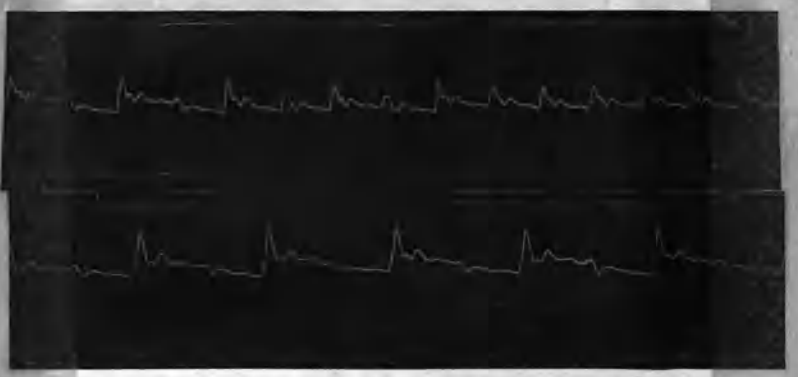
A



(J. W.)

A: Taken from a case of Dilatation of the Left Ventricle succeeding Hypertrophy in a patient with Granular Contracted Kidney.

B



B. Taken from a case of Failure of Compensation in Mitral Regurgitation of Rheumatic origin.

The following is the history of the patient, J.W., whose tracings are shown above (A) He was a joiner, and was 48 years old when he came under observation. His symptoms then had extended over a period of fourteen months, his first symptoms being breathlessness and palpitation on exertion. On one occasion, seven months before admission, he had been laid up for a fortnight with swelling of the feet. This had disappeared on resting, but three weeks before admission it reappeared in his feet, and was of considerable extent in the feet and legs when he came under observation. He had been troubled with headaches for some months, and had had two attacks of breathlessness while lying quietly in bed. His sight had been failing for some months before admission. His previous health before the onset of symptoms (fourteen months before admission) had been

excellent. For years he had drunk alcohol to excess two or three times a week. The arteries were large, dilated, tortuous, and uneven. There were obvious signs of dilatation of the left ventricle. The first sound in the apical region was terminated by a soft systolic murmur. The pulse was large, the pulse beats being sudden, vehement, and unsustained; missed beats were occasionally noted. The breathing was of a typical Cheyne-Stokes character.

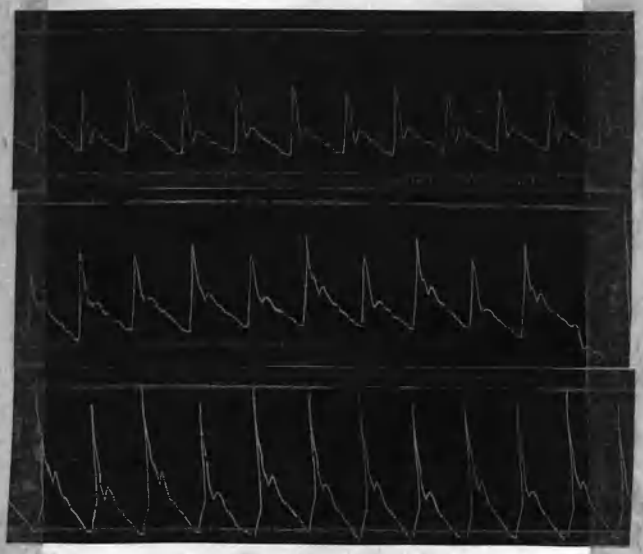
Examination of the fundi showed swelling of the discs in both eyes, with a small flame-shaped haemorrhage in the right retina. The urine was acid, Sp. Gr. 1016. It contained albumen .15% (Esbach). Blood was not present, and no tube casts could be found. Death took place 2½ months later, from cardiac failure.

At the post-mortem examination the kidneys were found to be the seat of cirrhosis, but not in an advanced degree. The left ventricle was hypertrophied and dilated; the heart muscle was fatty; the aorta and coronary arteries were the seat of extensive atheroma and calcification.

The disease ran its course in this case in rather less than a year and a half. This shortness of the course of the disease is a common feature in this type of Granular Kidney.

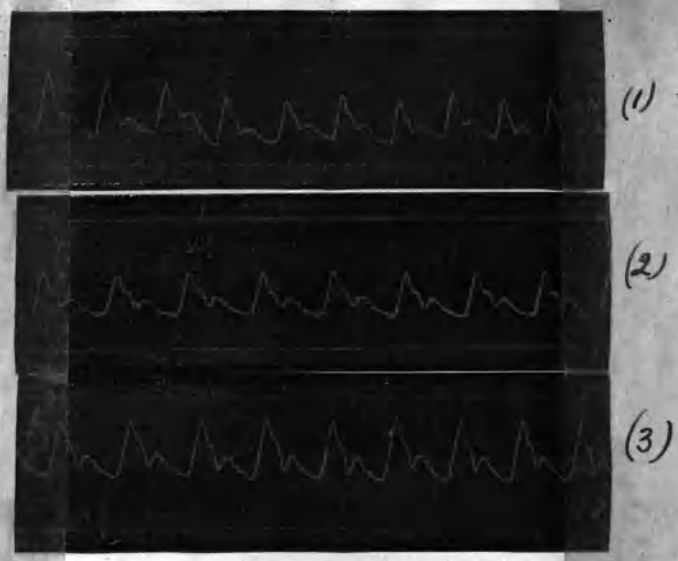
A dilated fatty heart also presents many points of resemblance to the failing heart of Granular Kidney. But careful attention to the history and examination of the circulatory conditions usually make the diagnosis easy.

A.



A. Taken from a case of Granular Contracted Kidney with hypertrophy and dilatation of the left Ventricle

B



(1)

(2)

(3)

From a case of Fatty Heart.

These conditions - renal cirrhosis and fatty heart - possess one occasional symptom in common, which is seldom, if ever, manifested in mitral regurgitation of rheumatic origin, and that is Cheyne-Stokes breathing. This is another symptom which Broadbent attributes to the influence of high arterial tension. It is his opinion that it is due to a disturbance of the relation between the pressure in the systemic and pulmonary systems.

He says that Cheyne-Stokes breathing is most common with uraemic symptoms, especially uraemic coma, so that "the occurrence of uraemic phenomena and Cheyne-Stokes breathing, under like conditions, is a reason for attributing both to the same cause" (The Pulse, p. 171). He also states that there is no evidence of imperfect aeration of the blood, such as lividity of the lips, and that the heart usually "takes no notice of the alterations between breathing and pause, but sometimes the beats slacken in frequency and force towards the end of the pause".

Now the exact method of its production by high arterial tension is not made at all clear by Broadbent, and in addition, his clinical

observations on the phenomenon are somewhat inaccurate.

It is true enough that, in many cases, Cheyne-Stokes breathing and high arterial tension are seen together, but if there is anything in this connection, high arterial tension ought always to be present when Cheyne-Stokes breathing is seen. I have seen four cases of fatty heart with this type of breathing, and in all of them the arterial tension was low; and in tubercular meningitis, in which Cheyne-Stokes breathing, or an approximation to it, is sometimes seen, high arterial tension is very rare.

I have notes of several cases in which lividity of the lips was very marked during apnoea, but disappeared during dyspnoea, thus giving unmistakeable evidence of defective aeration of the blood during the pause.

The various diseases in which Cheyne-Stokes breathing occurs have this in common, that the nerve centres, both high and low, are existing on a supply of blood which is either insufficiently aerated, or contaminated by poisons of some sort.

And Gibson's theory seems to fit in with all the known conditions. The higher centres, he says, are no longer capable of controlling the great organic centres in the medulla when their functional activity becomes lowered by deficiency in blood supply. The lower centres being now uncontrolled, carry on their functions automatically, and these lower centres always show a tendency to periodic action when their activity is lowered in any way. The respiratory centre may be involved alone, or the Vagus and Vasomotor centresas well. This would explain some of the changes in the pulse which are so frequently seen in Cheyne-Stokes breathing. Rhythmical

changes in the blood pressure and in the pulse rate are often to be observed. The only change I have seen in the pulse rate is a slowing in rate and increase in force of the pulse beats towards the end of Apnoea and the beginning of Dyspnoea. From the fact that, when present at all, it is always seen at this one particular part of the cycle it would seem probable that it is due to stimulation of the vagus centre by the accumulation of an excessive amount of CO_2 in the blood.

The changes in the blood pressure have been said to be due to stimulation of the vasomotor centre by the deep breathing of the period of dyspnoea. But it is more probable that they are due to rhythmical periodicity of action of the vasomotor centre for the rise in blood pressure does not always correspond with dyspnoea.

Tracings are appended, taken from the same patient, in which it will be seen that in No(1) A the rise in pressure (indicated by the rise in the base line) takes place during dyspnoea, while in No(2) A. taken a few days afterwards, the rise takes place in apnoea.

Several tracings are shown below taken from patients with Cheyne-Stokes breathing: it will be seen that none of these tracings present the appearances of high arterial tension.

A

(1)

(2) × Apnoea begins Apnoea. Apnoea ends × Dyspnoea begins Dyspnoea Dyspnoea ends × Apnoea begins Apnoea. Apnoea ends ×

(3) × Apnoea begins Apnoea. Apnoea ends × Dyspnoea begins Dyspnoea Dyspnoea ends × Apnoea begins Apnoea Apnoea ends ×

× Apnoea begins Apnoea. Apnoea ends × Dyspnoea begins Dyspnoea Dyspnoea ends × Apnoea begins Apnoea. Apnoea ends ×

A from a case of Granular Contracted Kidney with hypertrophied and dilated heart all the tracings show low tension

In no (1) & (2) rhythmical changes in the blood pressure are seen

in (1) There is a fall of pressure with an increase of the size of the pulse waves in Apnoea

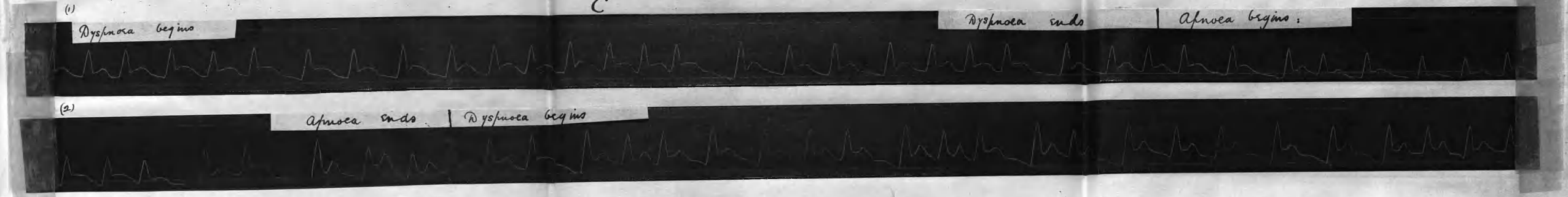
in (2) There is a rise of pressure with a decrease of the size of the pulse waves in Apnoea

in (3) No change is seen at all and when this tracing was taken the Cheyne-Stokes breathing was perfect in type.

B.

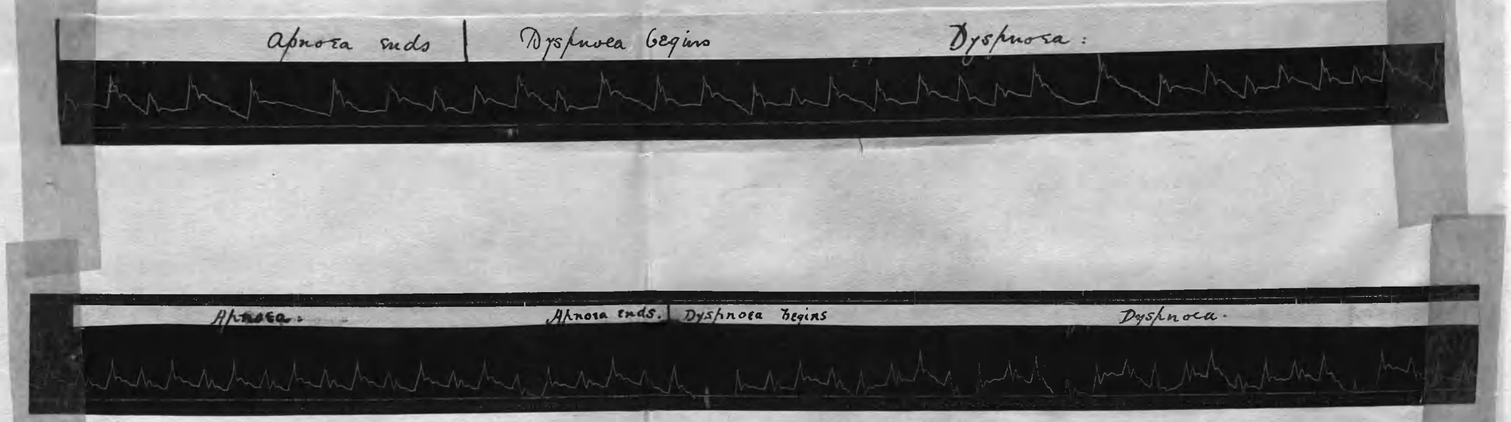
B. This tracing was taken from a patient with Cheyne-Stokes breathing: The case was diagnosed as one of fatty heart and this was confirmed by post mortem examination Here the pulse tension was low and the heart took no notice of the phases in the respiratory cycle.

C



C From a case of fatty heart with Cheyne-Stokes breathing
In both cases a fall in pressure is seen during apnoea
No change in rate is to be observed.
The tension is low.

D.



D From a case with exstensive atheroma of the Aorta
No change in pressure is seen
There is some slowing of the rate at the end of apnoea.
The tension is low.

E From a case of Uraemic Coma
No change in rate or pressure is seen
The respiratory curve is well marked in dyspnoea
The pulse tension is low.

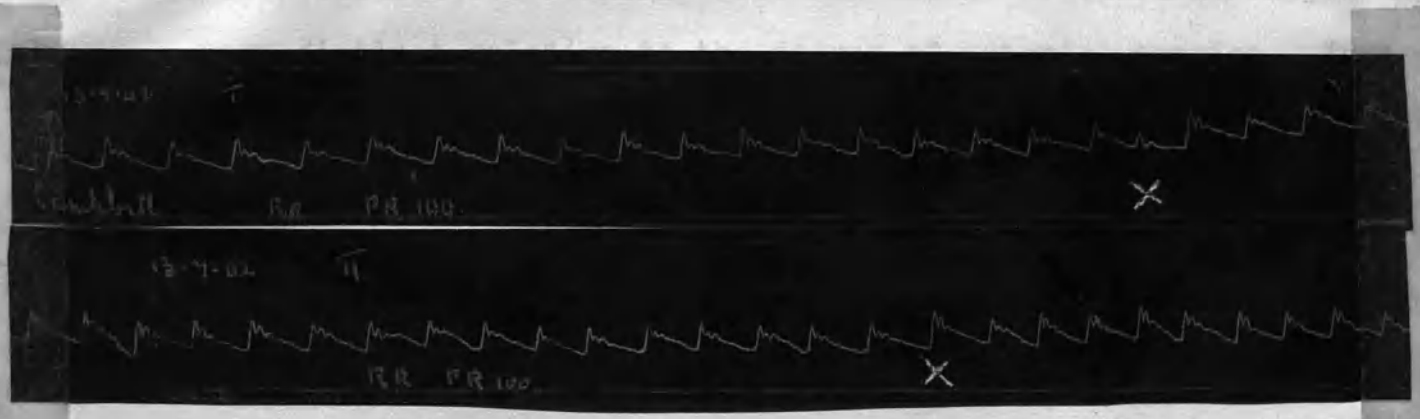
Having discussed the influence of high arterial tension in the arterio-sclerotic type of renal cirrhosis, a few remarks may now be made concerning the effects of high blood pressure in the other variety, which I have already alluded to, as the uræmic type, i.e., that class of case in which the symptoms refer chiefly to the poisonous effects of retained impurities. Here heightened arterial tension does not play such an important part. It is, however, constantly present, and indeed is usually maintained to the very end.

In the very early stages of the disease, it is a symptom of much diagnostic significance, and it is often possible to diagnose the disease from the presence of persistent high pulse tension, and persistent low specific gravity of the urine before albumen and tube casts are present in the urine.

It is at this stage in the course of the affection, while the patient is still able to go about his ordinary duties, that the arterial tension is at its highest. It is heightened by indiscriminate diet and muscular exertion, and, in addition, the hypertrophied heart is still perfectly competent. The possibility of renal cirrhosis ought never to be overlooked, because the urine contains no albumen. This is the time to recognise the disease, for much can be done by lowering the arterial pressure by the use of purgatives, and the selection of a suitable dietary. In this way the danger of the occurrence of cerebral haemorrhage may be lessened, and the period of cardiac efficiency prolonged.

In the terminal stages of this type of the disease, in which the patient dies of uræmia, the pulse is usually found to retain all the characters of high tension to the very end. The actual cause of

death in uraemia is cessation of the heart's action, but this occurs, not because the heart is actually worn out, as in the arterio-sclerotic type, but because it is poisoned like all the other organs by the retained urinous impurities. The patient presents no obtrusive signs of cardiac weakness. Oedema is rarely seen; if present, it is trifling in extent. The arteries are thickened, but not calcareous and irregular. The blood pressure is high. The pulse wave is slow, sustained, and difficult of obliteration. Occasionally a sign of approaching cardiac debility is to be noted in the presence of abortive beats.



Taken from a case of Granular Contracted Kidney which died of Uraemic Coma: These tracings were taken the day before the patient died: They show that the arterial tension was high to the end. Abortive beats and irregularity in the force of the pulse wave are marked by crosses.

Another point in which uraemic patients differ from the arterio-sclerotic type is the greater frequency with which retinal changes are to be met with in the former class. The reason of this is obvi-

ous. White spots and optic neuritis are the results of the chronic poisoning with the products of renal elimination, and in the arterio-sclerotic case cardiac breakdown closes the scene before enough time has elapsed for these changes to be produced.

Broadbent suggests that white spots and choked disc may have their origin in the heightened blood pressure in the circulation of the eye, but it is only necessary to point out that similar changes are found in diabetes and pernicious anaemia, diseases in which the arterial tension is either normal or lowered, to prove that there is no such connection. But the size and number of the haemorrhages have a very distinct relation to the degree of arterial tension. In the arterio-sclerotic type of granular kidney, haemorrhages in the retina are much more common than white spots or optic neuritis, but they are usually few and small. This is noteworthy for the contrary might reasonably have been expected in view of the vascular degeneration which characterises these cases.

But it is in the uræmic type that haemorrhages are seen in the greatest numbers and of the largest size, and this is probably determined by the high arterial tension which is found in this class.

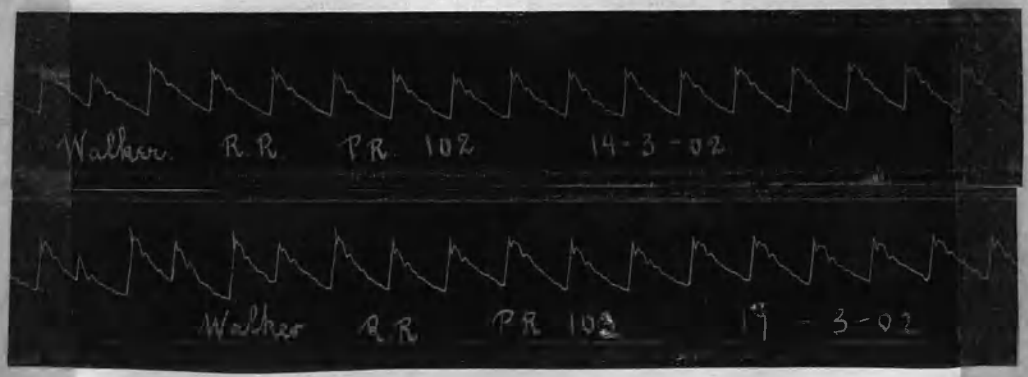
ILLUSTRATIVE CASE

Jessis W., aged 50, came under observation complaining of frontal headache, progressive weakness, and failure of vision of four or five years' duration. She had suffered for two years from dyspepsia, and had had constant vomiting for two months before admission, and, during the same time, breathlessness on exertion, and

palpitation had occasionally troubled her. Two months before admission she had an apoplectic seizure attended by loss of power in the right arm, with loss of speech, both of which symptoms speedily disappeared.

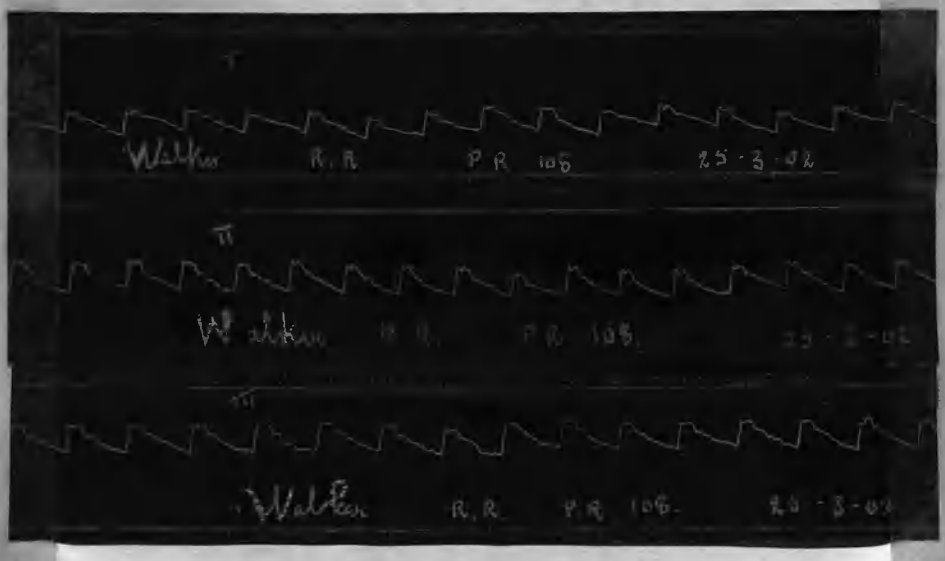
On examination she was found to be mentally dull and listless; the pupils were dilated, the tongue dry, red and glazed. The complexion was pale and yellowish. The respiration was slow, irregular, and sighing. There was no oedema anywhere. Hypertrophy of the heart was manifest; the apex beat was strong and thrust-like. The arteries were nowhere visible, but were slightly thickened and inelastic. The pulse presented all the signs of high tension; it was occasionally irregular from the occurrence of abortive beats.

A.



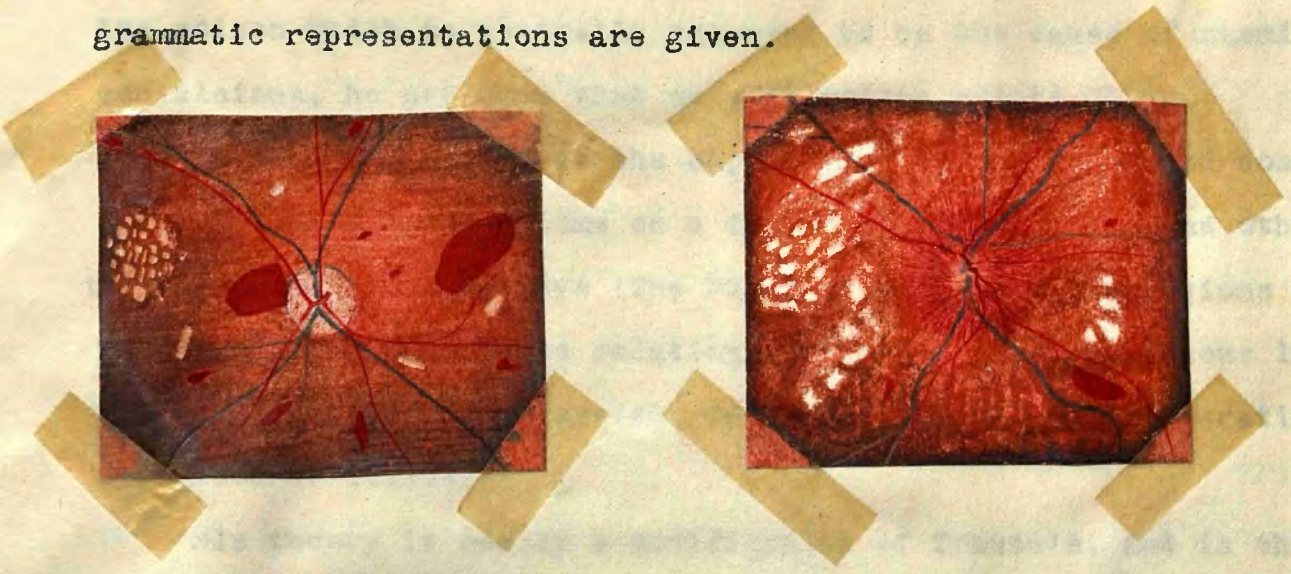
A. Taken on 19th March 1902

B



B. Taken on 25th March 1902 two days before death. These tracings also show the maintenance of high arterial tension in the late stages of the "Uraemic" type

The fundi oculorum presented typical changes, of which diagrammatic representations are given.



She died of uraemic coma after being three weeks in hospital; and during these three weeks she had numerous and severe attacks of epistaxis.

This history is fairly representative of the uraemic type of the disease, and shows several important points of difference from that presented by the case of J. W. (see page 30), which may be taken as more or less typical of the arterio-sclerotic type. The course of the disease is longer; headache, weakness, and vomiting, symptoms of chronic poisoning are prominent, the arterial tension is high to the end, and there is a marked tendency to haemorrhage as the result of this. Signs of cardiac breakdown are practically absent.

It has already been mentioned that Broadbent seeks to establish the dependence of Cheyne-Stokes breathing, and the retinal changes of renal cirrhosis on the circulatory conditions. And he also explains uraemic coma and convulsions on the same grounds. He admits that the headache, vomiting, and diarrhoea of uraemia are due to the action or vicarious elimination of retained products. But, because

experimental research has failed to demonstrate the exact nature of the poison which is generally supposed to be the cause of uraemic convulsions, he presumes that no such poison exists.

He is of opinion that the capricious onset of coma and convulsions places these symptoms on a different footing from the other uraemic phenomena. He says (The Pulse p. 245) "The convulsions appear in effect to have no relation with the amount of urinous impurities present in the system, or with the general deterioration of the blood or tissues."

His theory is really a modification of Traube's, and is this, that the heightened arterial tension leads to increased serous effusion on the surface of the brain, the intra-cranial pressure being thereby increased. Then, when the heart begins to flag in the face of the peripheral resistance, the second factor necessary is supplied and "at some point or another the external pressure (on the cortical vessels of the brain) goes so far as to give rise to stasis in a capillary area of large or small size, it may be by flattening of the convolutions against the vault of the cranium or by compressing cortical veins on their way to the sinus, or by interfering with the outflow of blood at the base of the brain."

This ingenious view is supported by the excellent effect of bleeding in uraemic convulsions, and some countenance might also be thought to be obtained for it from the results recently obtained by Monro in uraemic coma by lumbar puncture.

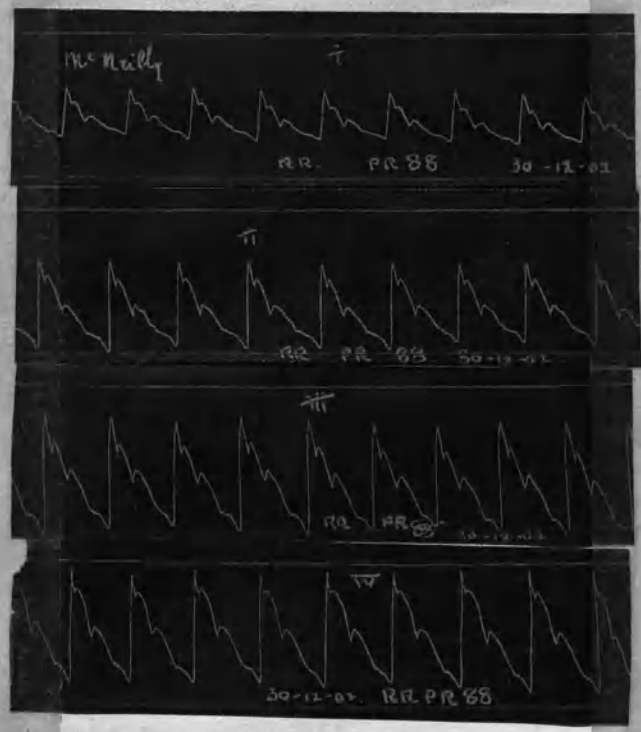
But the mere fact that experimental research has failed to reveal the nature of some suspected poison does not do away with the intoxication theory of uraemic coma and convulsions. Nor does the

capricious incidence of the convulsions negative this view. For, though convulsions sometimes come on when little poison can have accumulated, and do not come on in others where there must be a great deal, one must make allowance for variation in nervous excitability, and, in the latter instance, it is possible that by a process of slow intoxication, the nerve centres have gradually become dulled, and do not respond even to the stimulus of a large quantity of poison.

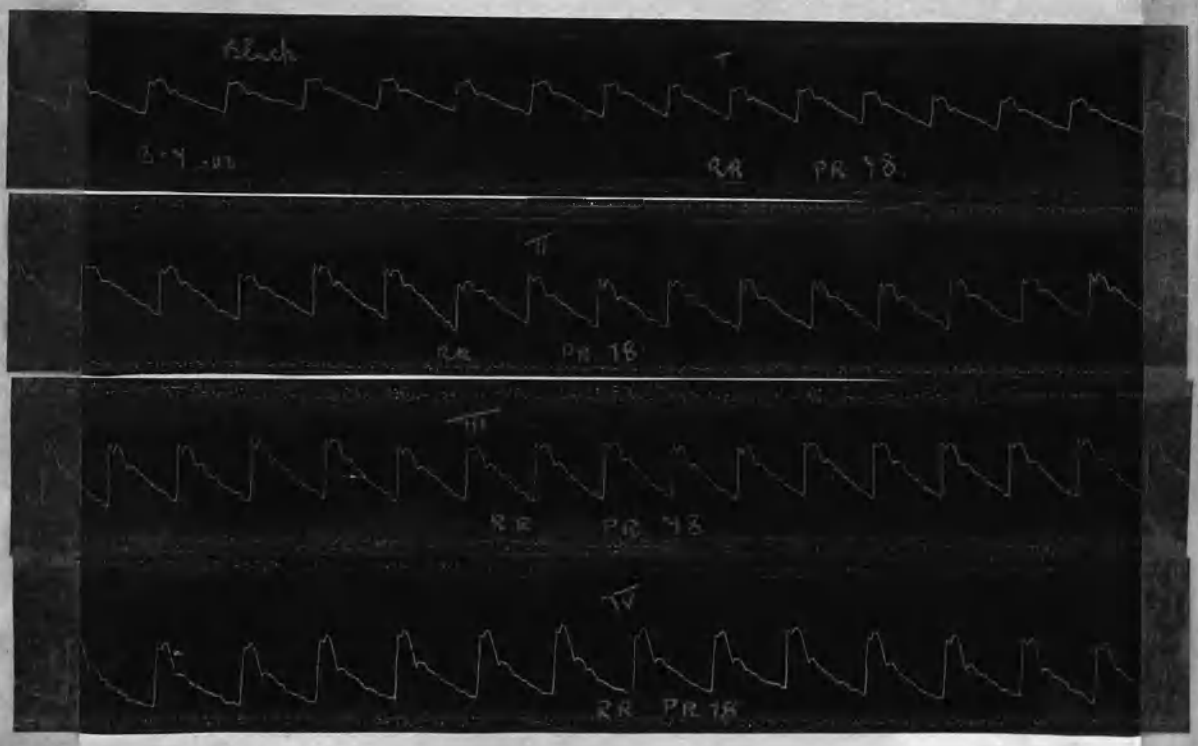
Broadbent appeals to the frequency of convulsions in scarlatinal nephritis, and explains that this is due to the fact that the heart is weakened by the acute disease, is unable to hypertrophy to meet the increased peripheral resistance, and so permits of stasis in the cerebral circulation. But, it is probable that the notorious excitability of the nerve centres in children plays a large part in the frequency of convulsions in scarlatinal nephritis; and, in addition, it is in this condition that anuria is most complete owing to the fact that, in scarlatinal nephritis, the glomeruli are specially involved. Anuria means the retention of a large quantity of poison in the blood. This suggests another point and that is, that uraemic convulsions, occurring in the course of cirrhosis of the kidney, have, in my experience, always been accompanied by a notable fall in the urinary output. And further, though I have seen cases of uraemic convulsions in which there were signs of approaching cardiac failure, yet, in quite a large proportion, there were no clinical signs whatever of this condition; and Broadbent considers weakness of the heart's action an essential to the fulfilment of the conditions necessary to stasis in the cerebral circulation.

Broadbent, in speaking of the evil import of low arterial ten-

sion in Granular Kidney, instances a case in which the arterial pressure was persistently low. The final manifestation was a series of violent one-sided convulsions (The Pulse, p.254.). This case would not seem to give much support to his views on the influence of high arterial tension in causing uraemic convulsions.



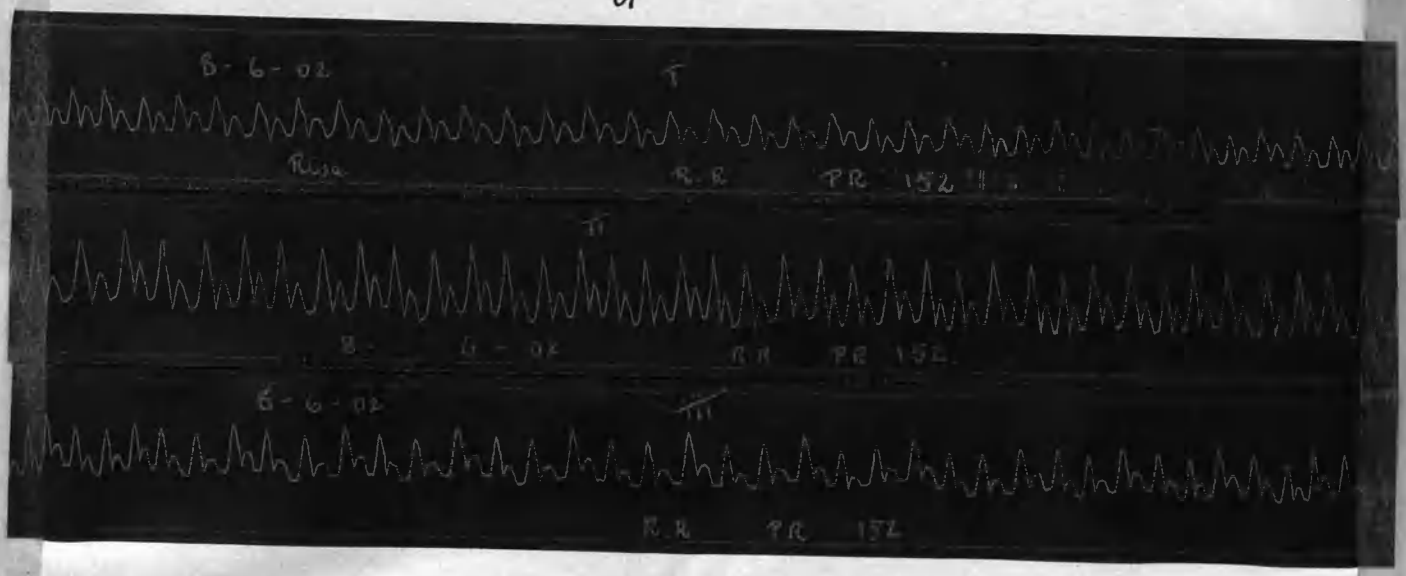
These tracings were taken from a patient, who had violent uraemic convulsions, six hours before their onset. They show no sign of irregularity and the tension is good.
He came under observation a week before he had the Convulsions and during that time the heart gave no clinical sign of flagging. The average amount of urine for the first five days of his stay was 480zs, for the day before the convulsions occurred 20ozs, Average for the week after the convulsions 56ozs.



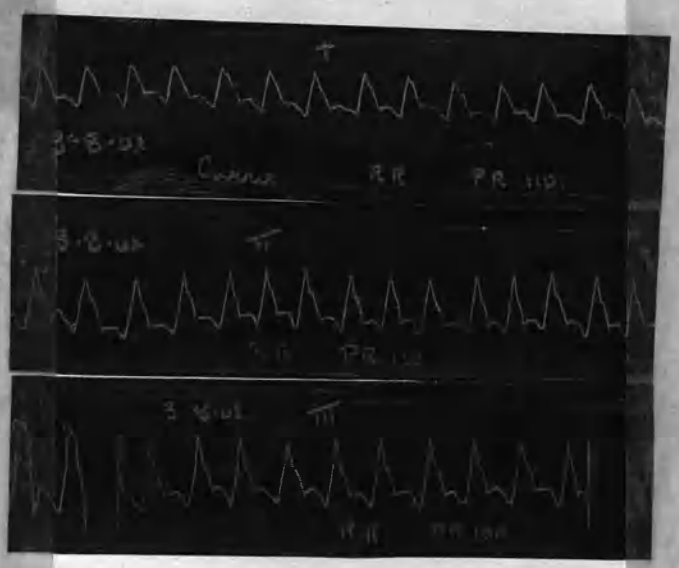
Taken from a patient with uraemic convulsions, in the interval between two fits.

The tension is high but the pulse is regular and the tracings show no sign of approaching failure of the heart. Physical examination showed the heart to be apparently efficient

A



B.



A and B were both taken from patients who suffered from "Large White Kidneys". In both instances the patients died of Uraemic coma and both sets of tracings were taken shortly before death. The pulse in both cases is dicrotous

The symptoms of uraemic coma as found in Large White Kidney are the same as in Granular Kidney with this exception, that the arterial tension is usually low in chronic Parenchymatous Nephritis (Large white Kidney). This seems a strong argument in favour of the view that high arterial tension is not the cause of uraemic coma as found in Granular Contracted Kidney.