THE NEWER RENAL EFFICIENCY TESTS

and THEIR VALUE in DIAGNOSIS and PROGNOSIS

by

ANDREW MELVILLE WATT,

M.B., Ch.B. (Edin.).

Thesis presented for the Degree of

M.D.

University of Edinburgh.

April 1937

The Newer Renal Efficiency Tests and their Value in Diagnosis and Prognosis

It is probable that, at the present day, the function of no organ in the body is being investigated by so many methods in the course of hospital treatment, as that of the kidney.

The large number of methods used is the result of the fact that there is no general consensus of opinion as to any one method being definitely better than the others.

A great deal of work has been done, comparing the relative values of these tests, the one with the other, in different types of nephritis, and in different stages of the disease.

Much investigation has also been carried out to demonstrate how the results of these biochemical tests compare with the clinical findings, such as the presence, and severity of haematura, albuminuria, anaemia and elevation of blood pressure.

One feels that in the multiplication of the kidney function tests, we are getting further and further away from the patient, and our attention is being diverted too much from the ward towards the laboratory.

In/

In the following pages, it is our intention to pick out the salient points from the vast amount of recent investigation into kidney function, and in the light of these findings, to discuss the various kidney function tests in daily use, and their relative merits.

Thereafter we intend to present the records of an investigation carried out by ourselves.

The object of the investigation has been to correlate more closely the results of the kidney function tests to other clinical findings recorded simultaneously. The clinical tests used deal chiefly with changes in the condition of the cardiovascular system, as indicated by the systolic and diastolic blood pressure, the pulse pressure, and the pulse rate.

The justification for such an investigation will be discussed later.

It is hoped that by this co-relating the clinical findings to the kidney function, one might be able, by the clinical findings alone, to assess the kidney function. In this way much time-consuming and expensive laboratory investigation might be eliminated, without loss of efficiency, or insight into the true condition of the patient, and the course of his disease.

SECTION/

In discussing the various kidney function tests, we shall first take those which deal with ability of the kidney to excrete water and urea, since their evolvement is intimately connected with the discovery of nephritis, and the gradually increasing knowledge of the disease.

Bright first described nephritis in 1827.

A year later, in 1828, Dohler performed the synthesis of urea. Not only was this an important event for the urologist, but was unique, in that it was the first time than an organic compound had been synthesised in Vitrio.

Eight years later, in 1836, Bright^I noted that in a case of nephritis, under his care, the concentration of urea in the blood was the same as the concentration of urea in the urine. Also in this case, while the urea in the blood was more then normal, the urea in the urine was only a third of the normal concentration.

Gréhart² in 1904, was the first to use the ratio of the Blood Urea to the Urinary Urea as an expression of the functional ability of the kidney.

His results showed so many discrepancies, however, that the test was dropped for the time being.

It had long been recognised that in the nephritic/

nephritic, the kidney did not excrete water ingested as quickly as in a normal person.

Rowantree and Geraghty3 in 1910, suggested that the ability of the kidney to excrete water, might be used as measurement of kidney efficiency.

Ambard and Weill, in 1912 in an article on the excretion of Urea and sodium chloride by the kidney, were the first to take into account the volume of urine passed along the blood urea and the urinary urea.

In 1919 MacLean and de Wesselow⁵ published their investigations into the effect of War Nephritis on kidney function and in the following year⁶ advocated the "Urea Coefficient" as a means of measuring renal efficiency.

Harrison, two years later, in 1922, followed with investigations similar to those of Gréhart mentioned above.

He showed that the discrepancies in the blood urea, urinary urea ratio found by Gréhart, occurred when the urinary output was high. He pointed out that the results showed no discrepancies as long as the volume of urine passed was between 100 to 150 ccs per hour.

To insure a urinary volume within these limits, he restricted the fluid intake of the patients for some time before the test was performed.

His results, he found, were a useful and delicate/

delicate test for renal function.

In America, a year previous to the publication of Harrison's work, Van Slyke⁸ and his colleagues showed that with a constant blood urea level, when the volume of urine passed exceeds 2 ccs per minute, the urea excretion reaches its maximum level. However much urinary volume increases beyond the 2 ccs per minute, there is no further rise in the total amount of urea excreted. This volume of 2 ccs he called the "Augmentation Limit."

When the volume of urine being passed is less than 2 ccs per minute, the total amount of urea excreted varies as the square root of the volume of urine passed.

These findings were in adults. The figures have to be adjusted in children, according to the size of the patient.

Again in 1934, Van Slyke and his colleagues⁹ published further work, showing that a healthy kidney removes, except in very low rates of urine flow, a constant percentage of urea from the blood, no matter what the concentration of Urea in the blood.

REVIEW/

REVIEW of the KIDNEY FUNCTION TESTS.

6.

We now proceed to the actual kidney function tests in common use, discussing their technique, their advantages, and possible fallacies.

THE WATER ELIMINATION TEST

This test exists in many forms. The form used by Fishberg¹⁰ is as good as any.

During the test the patient is kept in bed. Before the test he has been on ordinary diet with no diurctics.

The patient empties the bladder, and is then made to drink 1200 ccs of water in the course of the following half-hour.

At hourly intervals the bladder is emptied, and the volume of urine passed is measured.

The patient should eliminate practically all the 1200 ccs in 4 hours, the larger part of it being excreted in the first 2 hours. The Specific Gravity of the largest hourly specimen sinks to about 1002.

Discussion of the Test.

If the patient has any cardiac insufficiency, or any oedema, it will interfere with water excretion.

The previous salt content of the diet, the amount/

amount of water stored in the tissue spaces, will influence the excretion of water. 7.

The test has the advantage of simplicity, but is seen to be influenced by too many extra-renal factors.

Thus if the water excretion is impared, the ability of the kidney to concentrate urine will have to be determined.

If the kidney is capable of concentrating urine, then the failure to excrete the water ingested must be due to extra-renal factors.

Giglioli¹¹ in his investigation of cases of malarial nephritis states, that "no information was gained from Martinet's water elimination test. The mode of elimination was most irregular, not only in albuminuric, but also in normal control subjects."

DILUTION and CONCENTRATION TEST.

Volhard in 1918 first applied this test in a simple form, but a more complete test is the form suggested by Johnston.12

The patient comes to his doctor in the morning, having fasted for the 12 hours previously. He brings with him, a specimen of urine which he has passed through the night, or of the urine passed first thing when he wakens in the morning.

On arrival at the surgery, he rests for about/

about half-an-hour. He then empties the bladder, and immediately drinks 500 ccs of water, and half an hour later drinks a second 500 ccs of water.

Beginning from the time of the first dose of 500 ccs, the bladder should be emptied at one hourly intervals for six hours, no further fluid being given.

The specific gravity, and the albumin content of the specimen taken over night, are determined.

The volume, and specific gravity of each of the other specimens are estimated.

There should be a high volume peak during the second hour, with a very low specific gravity. Thereafter, the specific gravity should rise, until by the fifth, or sixth hour, it should be approximately that of the night specimen.

The whole of the 1000 ccs ingested should be excreted by the end of the fourth hour. The difference between the specific gravity of the second hourly specimen and that of the sixth hourly, or night specimen, gives an estimate of the efficiency of the kidney.

Discussion of Dilution and Concentration Test.

This test is liable to the same fallacies as the simple water elimination test.

Priestly13 has shown that if any patient has been taking large quantities of fluid for some time/

time previously, even if no further fluid is given, the diuresis may continue for 24 hours.

Peters considers that the fluid intake must be restricted for 48 hours, before performing the test.

This need for prolonged preparation of the patient, greatly detracts from the usefulness of the test, since formerly, its simplicity was the strongest recommendation. Alving and Van Slyke¹⁴ find that the Concentration and Dilution test for renal function is too much influenced by the water retention in oedematous patients and the salt content of the diet, and further, that although preliminary preparation of the patient makes the test more accurate, the test gives no indication of the extent of the renal damage present.

They also state that as a quantitative test it has little accuracy, the same specific gravity readings being found in cases obviously differing in their degree of renal damage, e.g., one case giving a Blood Urea Clearance of 60% and another of only 10%, but the test does give an idea of the elasticity of the kidney function.

The presence of albumin in the urine alters the specific gravity, and a calculation has to be done to correct this, thus again complicating the test.

THE/

THE UREA CONCENTRATION TEST

The test introduced by Maclean and de Wesselow⁶ in 1920 in their Article "On the testing of Renal Efficiency with Observations on the Urea Coefficient," is the test still in general use to-day.

.During the twelve to eighteen hours before the test, the patient takes as little fluid as possible.

At the beginning of the test the bladder is emptied, and 100 ccs of water in which fifteen grams of urea have been dissolved, is administered.

At the end of each hour the bladder is emptied and each specimen observed.

As a rule the urea causes a diureses during the first hour, and the first specimen is rejected.

The second specimen should contain at least 2% of Urea. If the volume of the second specimen is over 120 ccs, the Urea content of the third specimen is determined.

In one of the specimens at least, there must be 2% or more of Urea. With normal kidneys the second specimen usually contains 4% of Urea.

Discussion of the Test.

Again in this test the advantages are simplicity and a minimum of preliminary preparation of/ of the patient.

If the patient is oedematous, the urea given will cause a marked, and prolonged diuresis, which will vitiate the result.

Riches and Robertson¹⁵ have pointed out that there is great variation in the rate at which Urea is absorbed from the gut in different individuals.

Thirdly, no attention is paid to the Concentration of the Urea in the blood.

Thus the Blood Urea may be very high, and accordingly the concentration of Urea in the urine also high, although severe renal impairment may be present.

THE BLOOD UREA CONTENT

In the previous tests, the amount of urea present in the blood has not been considered.

On the other hand, a simple estimation of the Urea content of the blood is frequently used as a guide to the efficiency of the kidney.

Disadvantages of using Blood Urea as an index of Renal Function.

Patients, suffering from severe diarrhœa, or vomiting or from diabetic coma, are all found to have a high Blood Urea. The dehydration seems to be the cause in these cases.

The/

The influence of dehydration has been demonstrated by Mackay. They found that by stopping the fluid intake the blood urea rose. Further, that the blood urea was greatly increased by giving the subject a concentrated solution of glucose intravenously, which caused rapid dehydration.

In addition he found that with simple starvation, with moderate fluid intake the Blood Urea level rose. This is due to the breakind down of the body proteins. A similar condition is the "toxic destruction" of protein which occurs in febrile conditions, raising the blood urea.¹⁶

Since the liver is the main site of urea formation, it may be expected that the blood urea will be affected by the efficiency of the liver.

Wohl and Brust¹⁷ have produced a series of cases in which there was no nephritis, but each had a high concentration of urea in the blood. They state that in almost all the cases, the cause was either, dehydration, caused by diarrhoea, vomiting, excessive perspiration, etc., or a deficient intake of sale and water.

Wood¹⁸ confirmed the findings of other workers, that the Blood Urea level is raised in severe haemorrhage. Besides the loss of fluid, the anoxaemia present in these cases, is thought to be a factor at work in elevating the Blood Urea.

The Mackays 19 have shown that the functioning/

functioning power of the kidneys must be reduced to a half, other conditions being normal, before the concentration of urea in the blood begins to rise.

In conclusion, Osterberg and Keith²⁰ have brought forward recently a series of 25 cases, drawn from the records of the Mayo Clinic over a period of four years. All the cases had a blood urea of less than 10 mgms %. One case had bilateral pyelonephritis, and another bilateral hydronephrosis.

He considers the low blood urea as due to a low protein diet and large fluid intake.

Thus we have abundant evidence of the unreliability of the Blood Urea alone as an index of Kidney efficiency.

THE BLOOD UREA CLEARANCE TEST.

The Basis of the Test.

The Urea content of the blood and of the urine is influenced by so many factors, that one may wonder why urea should still be used in these experiments on kidney function instead of one of the other waste products eliminated by the kidney.

One advantage of Urea was pointed out by Marshall and Davies²¹ when they stated that Urea, being a small molecule, is diffusible, and being a non-electrolyte, is not affected by those forces which act on electrolytes, causing them to be unequally distributed/

distributed in different cells, as is the case with the chlorides, etc.

Urea is therefore found in all tissues of the body, approximately in proportion to the water content of the tissue.

Peters and Van Slyke²² mention the diffusibility of urea, its equal distribution between plasma and blood cell, along with the finding that Urea shows larger variation in quantity, than any of the other substances in the blood grouped under the name of Nonprotein Nitrogen. This makes it a suitable estimate of the nitrogen retention.

We have already considered the factors which regulate the formation of Urea in the blood.

Let us now consider how the urea is removed from the blood, and the various factors influencing the actual quantity of it, which is removed by the kidney at any one time.

From the investigations of various authors, we have already seen that the amount of urea passed is intimately connected with the volume of the urine being passed, and accordingly, a knowledge of the factors increasing or decreasing the urine flow, should give insight into the mechanism by which the blood is cleared of Urea.

FACTORS/

FACTORS INFLUENCING VOLUME OF URINE EXCRETED.

First examine therefore, what happens when abundant fluid is ingested, and diuresis occurs. Although the quantity be massive, the excess fluid is temporarily accommodated in the tissue spaces, and the blood is not appreciably diluted. Thus it is seen that the diuresis is not due to dilution of the blood causing a fall in the osmotic pressure of the plasma, and making filtration through the glomeruli easier.

Richards and Schmidt²³ have shown that massive intake of water does not increase, or decrease, the blood flow through the kidney constantly. Experiment has shown, however, that excess of fluid does increase the number of glomeruli in action, giving a greatly increased filtration surface.

Meantime, presumably because the body wants to get rid of the excess of fluid ingested, by some mechanism as yet not understood, perhaps by means of excretion from the pituitary, the tubules absorb less water than normal, from the fluid passing through them. The urea, acting practically as a non-threshold substance, also passes down the tubules almost untouched.

The result therefore is a diuresis with increase of urea excreted.

As mentioned before, the urea excreted increases with the volume of urine passed, up to the rate of 2 ccs. per minute, when it reaches a maximum, and/

and no further increase in volume of urine passed, increases the total weight of urea excreted.

When the concentration of urea in the blood is increased, either by giving Urea by mouth, or by giving a high protein diet, the urea passing in the glomerular filtrate, acts as a non-threshold substance, keeping fluid in the tubules, and thus increasing the volume of the urine excreted.

Cardiac Failure, dehydration, or any other factor causing oliguria, will produce a rise in the blood urea.

A particularly good example of this is given by Peters and Van Slyke.²⁴ The patient they mention had Lobar Pneumonia. In spite of the pyrexia and toxic destruction of protein, the blood urea was kept low by suitable diet and a large fluid intake. Auricular Fibrillation supervened. Venous congestion followed, resulting in diguria and a high blood urea. Under the influence of digitalis, the normal rhythm was restored, the flow of urine returned to normal and the blood urea fell.

As we have just seen above, the amount of urea excreted in the urine, depends on the concentration of urea in the blood, and on the volume or urine being passed. The Blood Urea Clearance test is based on these two facts.

Thus/

Thus, if we estimate the concentration of Urea in the urine, and multiply the figure by the square root of the volume of urine passed, if less than 2 ccs per minute (since Van Slyke has shown that the amount of urea passed varies as the square root of the volume of urine, if that volume be less than 2 ccs) we get the weight of urea being excreted per minute.

We estimate the concentration of Urea in the blood. By dividing this figure by the weight of urea being excreted per minute, we get a figure which represents the volume of blood which contains that weight of urea.

This figure is the Blood Urea Clearance and theoretically represents the volume of blood which could be completely cleared of its urea in a minute by the kidneys.

When the volume of urine being passed is more than 2 ccs, the urea excreted has reached a maximum, and no further increase in the volume of urine passed increases the total weight of urine passed. Therefore with volumes of urine over 2 ccs per minute the Urinary Urea Concentration is multiplied by the volume, and not the square root of the volume, as in the previous case.

Thus the formula used when urine flow is over 2 ccs per minute is as follows: -

(Percentage/

(Percentage of Urea in the Urine) x (Volume of Urine passed per Minute) Percentage of Uria in the blood

For cases where the Urine flow is less than 2 ccs per minute, -

(Percentage of Ures in the Urine) x, Volume of Urine passed per Minute Percentage of Ures in the blood.

In the former case the excretion of urea has reached its maximum and is therefore called the "Maximal Blood Urea Clearance," represented by the expression C_m . The average normal reading is $C_m = 75$ ccs.

Where the urine flow is less than 2 ccs per minute, shown in the second case, the result is called the "Standard Blood Urea Clearance," represented by the expression C₈.

The average normal finding is $C_s = 54$ ccs.

As a rule, instead of expressing the Clearance in cos, it is customary to express the Blood Urea Clearance as a percentage of normal values. Thus rather than express our finding as $C_m = 75$ ccs, we express it as $C_m = 100\%$ of normal.

Technique of the Urea Clearance Test.

The test is usually performed 2 hours after a meal.

It is best performed during the period between breakfast and lunch, since Mackay has shown that/ that during that period the Blood Urea Clearance shows less fluctuation than during other periods of the day.

At the commencement of the test, the bladder is emptied, the time accurately noted, and the specimen rejected.

Most authors are in favour of giving four to six ounces of fluid at this moment to ensure a good flow or urine, no tea, coffee, cocoa or other diueretic being permitted.

An hour later the bladder is emptied. The volume of the specimen measured and enough of it kept for the estimate of its urea content.

At the same time, enough venous blood is drawn off to determine the Blood Urea content.

An hour later, the bladder is again emptied, the volume of the specimen measured, and part sent for estimation of its urea content.

These specimens, and a note of the times and volumes mentioned above, we sent to the laboratory where the Urea Concentrations are estimated and the calculations mentioned above, are made.

The "augmentation limit" is in adults 2 ccs, but considerably less in children. Accordingly, the Augmentation limit has, in the case of children, to be adjusted to the size of the patient, before calculating the Blood Urea Clearance. The Disadvantages of the Test are, that the timing and measuring of all specimens have to be accurate, and also that it involves more laboratory work than the other tests, and a rather complicated calculation.

The Advantages are, that account is taken of the state of both the blood and the urine, which none of the other tests do. More important still, is that the Blood Urea Clearance test is not rendered invalid by various extra-renal factors, as are the other kidney function tests we have reviewed.

CREATININE CONTENT OF THE BLOOD AS A KIDNEY FUNCTION TEST

The fact that the percentage of creatinine in the blood is kept extremely constant in health, and that a rise in the percentage always indicates renal damage.

The upper limit of the normal reading is 2 mgms%. A rise to 3 mgms % and over indicates considerable renal damage, while concentrations of over 5 mgms % frequently indicate an early fatal termination.²⁵

It is a Disadvantage that the changes are so minute leaving very little margin for experimental error.

In addition the test for creatinine is a colorimetric one, and accordingly liable to error.

It has been $shown^{26}$ that in a patient the blood urea clearance may fall to C_s 20% before the Blood Creatinine rises to the upper normal limit of 2 mgms %. 21.

BLOOD CREATININE CLEARANCE TEST

In 1926, Rehberg²⁷ published his experiments in which he found that if creatinine output were plotted against the blood creatinine content, a straight line was produced.

Thus it appeared that the ratio between the amount of Creatinine in the blood, and the output of creatinine in the urine, would be an accurate measure of glomerular filtration.

This gave rise to what may be called the blood Creatinine Clearance Test.

However Dominguez and Pomerene²⁸ in 1934 showed that the Blood Creatinine curve, and the Urinary Creatinine Curve did not fall together to zero, but while the blood still contained some creatinine, none was being passed in the urine.

Thus, the ratio of the Blood Creatinine to the urinary creatinine, is not constant, and cannot therefore be used as a measure of glomerular filtration;

Moreover in certain mammals the renal tubules have been shown to excrete creatimine, and there is some question as to whether the human renal tubules

do/

do not do so also.29

This raises the interesting question as to whether the difference between the Blood Urea Clearance and the creatmine Clearance would indicate whether, in any particular case, the tubules or glomeruli was the more damaged by disease.

Cope in 1935, carried out Urea, Creatmine, and Xylore Clearance tests on a series of patients. To all tended to run parallel, the only discrepancy being that the Creatmin Clearance was occasionally much higher than the Urea Clearance, e.g., the former being 70 % on one occasion when the latter was only 50%. Excretion of Creatmine by the tubular may account for this.

PHENOL-SOLPHONE-PHTHALEIN TEST.

This test is a typical example of a series in which the efficiency of the kidney is measured by its ability to excrete a dye introduced into the body.

The following is the method most commonly employed at the present time.

•006 Grams of phenol-sulphone-phthalein dissolved in sterile saline to a volume of one c.c. is injected intravenously. An hour and ten minutes after the injection the bladder is emptied, and again an hour later.

The two specimens together should contain 60% of the dye injected. Usually the first specimen contains/ contains 40% of the dye and the second contains 20% of the total dye injected.

The amount of the dye present in the specimens of urine is measured colorimetrically, the urine being made alkaline with 25% caustic soda, when the phenol-sulphone-phthalein develops a pink colour.

Discussion of the test.

Rowantree and Fitz claimed that this was the best test for distinguishing between true renal failure, from that due to venous conjestion of the kidney caused by circulatory failure. They stated that impairment of renal efficiency caused marked reduction in the phenol-sulphone-phthalein excretion, while in venous congestion of the kidney, the excretion of the dye was only diminished when the congestion was very severe.

On the other hand, Fishberg³¹ finds that in heart failure the diluting power of the kidney is much reduced, and the ability to excrete phenolsulphone-phthalein is early affected. Meanwhile the concentrating power is not affected, and the Blood Urea content only rises when the circulatory failure is severe enough to cause objuria.

Earlier he states that in Essential Hypetension, as a rule, the Urea concentration test gives normal results, but the Phenol-sulphonephthalein test gives values below normal. Fishberg considers the latter result is an indication of cardiac/ cardiac weakness rather than of actual renal inefficiency.

His views are thus diametrically opposed to those of Rowantree and Fritz.

Paolo Blascucci³²discusses a fallacy in the test, due, he says, to the irregular absorption of the phenol-sulphone-phthalein by the different organs, and suggests methods of obtaining more accurate results.

Young^{3,3}five years previously, in 1931, claimed more satisfactory results from the test, when the dye was given in divided doses.

We see, therefore, that much disagreement still prevails regarding the technique of the test to be employed, and also regarding the interpretation of the results obtained.

Van Slyke and others have shown that in glomerular nephritis, the Blood Urea Clearance falls long before the phenol-sulphone-phthalein test indicates renal impairment. But when recovery sets in, the latter is the first to register the improvement.

In a series of cases he and his colleagues found that the Blood Urea Clearance had to fall to below 60% of the normal value before the Phenol-sulphone-phthalein test suggests renal impairment. Even in a few cases with a Blood Urea Clearance of 25% to 30% the test with the dye gave normal readings²⁶

In cases where there is residual urine in the bladder, as so often occurs in prostatic enlargement the dye excreted by the kidney is at once diluted by the residual urine, and the concentration of phenol-sulphone-phthalein in the urine voided, gives little idea of the amount of the dye actually excreted by the kidney.

INDIGO-CARMINE TEST.

This is another of the group of dye excretion tests.

5 ccs of \cdot 03% solution of indigo-carmine is given intra-venously.

The urine should be coloured within 3 to 8 minutes of the injection and the whole of the dye should be excreted in 12 to 24 hours.

To get the moment at which the dye appears in the urine a cystoscope must be passed.

The Test has all the disadvantages of the phenolsulphone-phthalein test, and is only a rough guide to the efficiency of the kidney.

Its usefulness lies in the fact that, by comparing the times at which the dye appears at the orifices of the two ureters, one forms an idea of the excreting power of one kidney compared with the other.

UROSELECTAN/

UROSELECTAN TEST

In this test also the time at which each kidney excretes the drug, gives an idea of the kidney efficiency, and of the power of one kidney compared with the other.

The drug should be excreted by the kidney within a period of from 2 to 10 minutes after it has been given intravenously.

The real value of the test is that by means of the drug, an X-ray can be taken, which visualizes the renal tract, showing up any anatomical and pathological abnormality.

SECTION II.

INTRODUCTION TO THE INVESTIGATION PRESENTED

Rationale of the Investigation.

The function of the kidney is intimately connected with the circulation, and disease of the kidney is so frequently accompanied by disease of the vascular system, that the study of the variations in kidney function naturally leads to the study of the concomitant variations in the other, in order to find if there are any changes in the one constantly reflected in the other. The hope being that from the various clinical findings in the vaso-motor system, we may be able to tell, without laboratory tests, the state of kidney function.

PHYSIOLOGY OF BLOOD PRESSURE AND KIDNEY EXCRETION, AND EXPERIMENTS WHICH HAVE BEEN PUBLISHED.

The Blood Pressure varies as the product of the Cardiac output and the Peripheral resistance34.

The peripheral resistance is chiefly dependent on the tone of the arterioles. The blood pressure is only very slightly reduced by its passage through the capillaries.

It was at one time thought that a rise in the general blood pressure did not affect the capillory blood pressure. Mayo³⁵ has since demonstrated

a/

a case of paroxysmal hypertension, in which "the capillaries of the nail fold became entirely obliterated when the blood pressure reached 170 mm. and did not reappear until the Blood pressure had again descended to that level."

Hayman³⁶ has shown that the blood pressure in the glomeruli if the kidney, runs parallel to the aortic blood pressure, being 54% of the latter.

It seems reasonable therefore, to assume that as a rule, the blood pressure in the glomerular capillaries, runs parallel with the general systemic Blood pressure.

Experiments have shown that rate of blood flow through the kidneys rises with a rise in blood pressure and falls with a fall in blood pressure?

The secretion of urine depends on two main factors, namely, the filtration pressure through the glomeruli and the volume of blood flowing through the kidney.

The filtration pressure is equal to the glomerular blood pressure, minus the osmotic pressure of the blood plasma.

This is demonstrated by the experiment in which the ureter is ligatured and a cannula inserted. The other end of the cannula is connected to a monometer. Urine is excreted up to a pressure 30 mm below that of the glomerular blood pressure. If now the plasma is diluted/ diluted with saline to reduce its osmotic pressure, the pressure of urine being excreted rises.

Wood, 18 confirming the work of other writers, mentions that if in severe haemorrhage the systemic blood pressure falls below 40 mms, anuria results. This is because the osmotic pressure of the blood plasma is sufficient to overcome the glomerular blood pressure, and no filtration of fluid through the glomerulus occurs.

The converse of this is, that many experiments have been done to show that the output of urine tends to rise with an increase in the systemic blood pressure.

That the Flow of Blood through the Kidney is as important as the filtration pressure, is seen by ligaturing the renal vein. This increases the filtration pressure, but the flow of urine decreases owing to the lessened blood-flow through the kidney.

<u>Variations in the Pulse Pressure</u> bear no quantitative relationship to the output of the heart, but they increase and decrease synchronously.³⁴

Thus other things being equal, a considerable rise in pulse pressure probably indicates an increase in cardiac out-put and the kidney, in common with the other organs may be expected to have an increase/

29.

increase in blood flow.

Within limits, that is provided the venous return to the heart is increased, an increase in <u>pulse rate</u> means an increase in the output of the heart.

ALTERATIONS TO THE VASOMOTOR SYSTEM IN CONNECTION WITH KIDNEY DISEASE.

So much for the physiology of normal individuals. Let us turn now to the evidence that in disease of the kidneys the vaso-motor system is more unstable than in normal persons.

In orthostatic albuminuria, where the protein appears apart from excretion, although no organic disease is present, the patient is often subject to fainting attacks with a blood pressure which fluctuates with posture and an irritable heart.³⁸

Fishberg finds that in hypertension the fluctuations in the blood pressure caused by, emotion, rest, etc., are much greater than in the normal individual.

One author⁴⁰ reports 2 cases under observation in hospital with acute tonsillitis, who developed a rise in blood pressure five days before showing signs of nephritis.

In their study of War Nephritis, Dunn and MacNee/

MacNee41 emphasise the presence of marked ischaemia of the glomeruli at the beginning of the disease.

Another author⁴² is reporting of cases of Abruptic Placentae considers that the anuria present in the cases is due to spasm of renal vessels.

These findings suggest that the vaso-motor changes may occur first and contribute to the onset of the nephritis.

EFFECT OF CHANGES OF BLOOD PRESSURE ON FUNCTION

of the KIDNEY

Mackay43 has demonstrated that in normal persons the Blood Urea Clearance fluctuated in definite directions at different times of the day.

He did further work to demonstrate that in patients with nephritis these variations in the Urea Clearance during the day, were greatly exaggerated. The exaggeration was greatest in the patients with an unstable vaso-motor system and maximum in those with a continually high though variable blood pressure.

Thus, the Blood Urea Clearance and the Blood Pressure seem so intimately connected, that one wonders what the effect on the Clearance would be, if, in such a case as the above, the blood pressure could be reduced.

Such experience have been carried out. Page 44 in 1934 took a series of cases with high blood pressure,/

pressure, and reduced the blood pressure by various methods, e.g., in one simply by rest; in another by giving large doses of sodium thiocyanate; another by injections of colloided sulphur and in another by denervation of the kidney. In none of the cases did the fall in blood pressure produce any significant change in the Blood Urea Clearance. Later the blood pressure returned to the original reading in these cases, but the Blood Urea Clearance still remained unchanged.

A year later, Page and Hener⁴⁵ report the case of a girl with a flexible but persistently high blood pressure, in whom, by section of the Anterior roots of the 6th thoracic to the 2nd lumbar nerves, they produced a persistent fall in Blood Pressure. This caused no alteration in the Blood Urea Clearance, or in the Cardiac output, but the ability of the kidney to concentrate urine was reduced.

Later in the same article,⁴⁶ they mention a case where by denervation of the kidney, a fall in diastolic blood pressure only resulted, but this time both the Urea clearance, and the concentrating power of the kidney were unaltered.

The recent work of Kellar and Arnott has shown that in animals, experimental glomerular nephritis causes a rise in blood pressure. If the kidney of the animal be denervated before the nephritis is produced, there is no such rise in blood pressure, that/ that is to say, the rise in blood pressure depends on the integrity of the renal enervation.

Gerbie and Martnelli have repeatedly denervated the kidneys of animals with elevated blood pressure. They report, that as a rule, the denervation causes a fall in blood pressure, but that the finding is not a constant one, some experiments showing no result.

From these experiments, we conclude that in Glomerula Nephritis and Essential Hyperpiesia, the elevation of blood pressure does not assist the kidney function.

That on the other hand, the kidney is in some way, probably through its nervous mechanism, responsible for the raised blood pressure in glomerular nephritis.

That in cases of Essential Hypertension and of glomular nephritis, the blood pressure fluctuates more than in the normal patient, and at the same time the blood urea clearance shows abnormally large fluctuations.

We have therefore taken a series of cases, and have investigated the kidney function simultaneously with the vaso-motor system, to discover how far the two are related, and to see if from the various findings in the vaso-motor system one can assess the condition/ condition of the kidney function.

METHODS EMPLOYED IN THE INVESTIGATION

To measure the efficiency of the kidney function, the Blood Urea Clearance test has been used. We believe it to be the most accurate and reliable for the reasons already discussed. In addition, it has the advantage that the measurement of the Blood Urea, the concentration, and the volume of the Urine passed, are all included in this test and can be referred to when required.

The vaso-motor system has been investigated by taking the Systolic, and Diastolic blood pressures, the pulse pressure, and the pulse rate.

There are 20 cases in the series, and in all, 114 Blood Urea Clearance Tests, Blood Pressures, Pulse Pressures and Pulse rates have been taken, and the readings plotted to form the charts presented.

All tests were carried out under as standard conditions as possible.

Most of the patients were confined to bed over the whole period covered by the investigation. A few had been getting up for a little in the evening towards the end of the period. Since the tests were carried out always in the morning, these patients had been/

been in bed for the 15 hours immediately before the tests.

One patient had slight pyrexia 2 days before the first test. Otherwise all cases were apyrexial during the period under observation.

Each patient was given his usual light breakfast, at 7 a.m., but no tea or coffee was allowed.

Because of the way in which the Blood Urea clearance alters with the time of day, all tests were performed between 9 a.m. and 12 noon, at which time the Blood Urea Clearance shows least variation.

At the beginning of the test each patient was allowed six ounzes of fluid to insure an adequate flow of urine. Tea, cocoa, or coffee being, of course, prohibited.

Occasionally certain patients had difficulty in emptying the bladder exactly at the end of the first and second hours. In these cases the time at which the patient actually did pass water was taken. Thus the test in these circumstances extended a little beyond the 2-hour period, but the output of urine per minute was accurately calculated, and since it has been shown⁴⁸ that under the conditions of the test the Blood Urea does not alter appreciably, the delay in passing urine does not make the result inaccurate.

The Blood Pressure and Pulse Rate were

taken

taken at the end of the first hour of the Urea Clearance test.

The patients were all recumbent. The blood pressure was taken by the ausculatory method. In each case, three to five readings were made, to make sure that any spasm in the brachial artery was overcome.

The very first appearance of sound, after the brachial artery was obliterated, was taken to indicate the systolic pressure. Onset of the fourth phase, i.e., the moment at which the second "banging sound" begins suddenly to fade, was taken as the diastolic pressure, rather than the fifth stage where all sound disappears.

The Pulse rate was taken over a two-minute period. If the reading of each minute was not the same, and if the patient's pulse rate was thought to have been accelerated by the taking of the specimen of blood, etc., the pulse was taken again half-an-hour later. In other cases the patient's four-hourly chart was used, along with the reading taken at the same time as the blood pressure, to assess the average pulse rate during the period covered by the Blood Urea Clearance test.

The cases presented are 20 in number, and consist of a variety of kidney conditions in which it is usually judged advisable to keep a careful watch on/

on the efficiency of the kidney function.

In the series, there is a group 7 nephritics, one case of lardaceous disease of the kidney, and 2 cases of essential hypertension with secondary involvement of the kidney.

A group of 9 cases of prostatic retention of urine, and one case of renal calculus.

Of the 7 cases we may term the primary nephritics, 5 of them (Cases No. 1, 2, 3, 4, and 6) were admitted as suffering from acute nephritis. Some of them did well and the others are apparently drifting into the chronic stage of the disease. Of the remaining two in this group, No. 7, had chronic nephritis with repeated acute exucerbations, and only slight tendency to hypertension. No. 5 was a chronic nephritic with albumenuric retinitis and a very high blood pressure.

Case No. 8 is a patient with amyloid disease of the kidney following on tuberculous disease of the spine, and in contrast to the other cases was given a high protein dict.

Cases No. 9 and 10 are patients with essential hypertension and secondary renal changes. No. 10 had at first, a glycosuria which was not due to "leakage of sugar through a damaged kidney" since the patient's blood sugar was above normal. He had retinal/

retinal changes which may have been due to the diabetic element, rather than the nephritic element.

In the group of prostatic cases, with the exception of case No. 18, the condition was of long standing. The patients had only come for treatment when forced to, because of acute retention. Several of the patients were over 70 years of age. The average of the group was 66 years.

Case No. 5 had a pyelonephritis. Case No. 1 had a very heavy urinary infection and for a long time was extremely ill. No. 4 and 9 also had considerable infection of the urinary tract, but their general condition never caused anxiety.

The last record is of a case of a patient with a stone in the left kidney which was surgically removed.

The tests were applied before operation, and again a short time after the operation.

Short histories are given of each case, giving the notes of the patient's general condition when noteworthy, and any other rebrent observation.

The tests were applied at as regular intervals as possible. In most of the cases the interval between the tests was 7 days, in a few it was 10 days, and in a few, the period was less regular because of particular circumstances connected with the cases.

This/

This case was included not only to make the investigation more comprehensive, but because it is an example of unilateral kidney disease causing nitrogen retention. Several such have been reported.49 & 50

ANALYSIS OF THE CHARTS

It has been shown by numerous experiments, that in the healthy kidney, a rise in blood pressure tends to cause an increase in the volume of urine passed.

That the converse is usual in the early stages of acute nephritis, is obvious. At the beginning of the disease, the Blood Pressure rises, there is oliguria, and perhaps even anuria. The improvement appears, often like a crisis, with a sudden fall in Blood Pressure, accompanied by an increased flow of urine, a rise in Urea clearance, and fall in Blood Urea.

This is demonstrated in cases 1 and 4, but the fall in Blood Pressure is gradual.

A similar sudden fall in blood pressure and blood urea, and a rise in clearance, is frequently seen in cases of urinary obstruction, where the obstruction has been relieved. Fishberg⁵¹ considers that such a sudden improvement cannot be due to improvement of the condition of the kidney tissue. He states that it is due to a functional spasm of the arteries being relaxed/

relaxed. The renal arterioles would partake in this dilitation, and the flow of blood through the kidneys would be increased, with a resultant rise in Blood Urea Clearance.

We do not find this dramatic improvement registered on the charts of our prostate cases. The reason is probably, that, firstly, our cases were all of long standing, the patients not coming for treatment until forced to do so by complete, or almost complete, retention, and frequently not their first attack of retention. Accordingly, one expects to find an unusual degree of renal injury, which would prevent any sudden improvement.

Secondly arterial spasm, lasting during the long period from the onset of the disease until the time these patients came for treatment, in arteries no longer youthful and elastic, is not likely to relax suddenly or completely.

Crichton Bramwell⁵² states that loss of elasticity in the arteries is evidenced by a rise in the pulse pressure, and caused most often by a fall in the diastolic blood pressure.

This rise in pulse pressure throws a strain on the heart, limits the oxygen intake, and lessens the excretion of which the patient is capable.

In the light of this, we would expect to find, in older patients with a high pulse pressure, some/ some alteration in the kidney function, due to the strain in the circulation.

41

We find no evidence of any fall in the Blood Urea Clearance in such cases. In Case No. 3. (Chas. R.), although the patient is aged 76, and the pulse pressure is one of the highest in the series, ranging from 82 mm. to 97 mm., yet his blood urea clearance ranges from 50% to 71%, comparing favourably with any of the other cases in his group.

In younger patients an increase in pulse pressure may be an indication of increased cardiac output. This should be reflected in the Blood Urea Clearance, unless there is constriction of the renal vessels. In none of our cases do alterations in the pulse pressure appear to have any regular influence on the Blood Urea Clearance.

Cardiac Output is also influenced by the Pulse Rate. Within limits, in normal circumstances, particularly as the result of emotion or exercise, the cardiac output is said to increase as the pulse rate rises to 120 beats per minute, and remains stationary as the pulse rate increases from 120 to 240 beats per minute⁵³ These alterations in output should be reflected in the kidney function, although not with tachycardia due to exertion.

Examination/

Examination of the charts will show no regular relationship between the pulse rate alone and the rental function in the nephritic group.

In the prostatic group on 18 occasions a fall in Pulse Rate was recorded, and on 13 of these occasions it was accompanied by a rise in the blood Urea Clearance. On 17 occasions a rise in pulse rate was recorded, and on 14 of these occasions it was accompanied by a fall in the Clearance. Thus out of the 35 records taken in the prostate cases, the pulse rate varied in inverse ratio to the Blood Urea Clearance in 77% of cases.

On more than one occasion, one sees a rise in the pulse rate along with a rise in the blood pressure.

Two possible causes suggest themselves. Firstly it may be due to an increase in the kidney damage (since we see in the acute stage of nephritis a rise like this, both the Blood Pressure and pulse rate falling as recovery sets in). If increased kidney damage is the cause, we shall find a fall in the Blood Urea Clearance.

In case No. 12 (Henry G.) from the prostatic group, there is a fall in the Urea Clearance, in readings 3 and 5 when Blood Pressure and Pulse rate have/ have both risen. In reading 2, however, there is a definite rise in Urea Clearance although the Blood Pressure and Pulse rate have both risen.

On the other hand the rise in blood pressure may be due to the increased pulse rate, which indicates an increased cardiac output. If this were the case, we would expect the kidney to have a larger blood flow, and the Urea Clearance to rise.

Under these circumstances we do get a rise in Case 3 (Walter S.) reading 7., but a fall in Urea Clearance in Case 6 (Joseph R.), reading 6. Both of these cases have nephritis which is running a very similar course.

Where the Pulse rate has fallen with a

rise in the Blood Pressure, a spastic condition of the vaso-motor system is suggested, with a reflex slowing of the heart. Arterial spasm should reduce the flow of blood through the kidneys and reduce the Blood Urea Clearance.

In Case 10 (Chas. P.) this does occur in reading 2, but there is actually a rise in reading 3.

Similarly in Case 6 (Joseph R.), reading 3 shows a fall and reading 4 shows a rise in the Urea Clearance, although in each case the Blood Pressure rises and the Pulse rate falls.

In the prostatic group, the readings are equally/

equally inconsistent.

<u>A rise in the Pulse Rate, along with a</u> <u>Fall in the Blood Pressure</u>, gives quite inconsistent results in the Urea Clearance in the Nephritics.

However, in the group of prostatic cases, unless immediately after an operation, a rise in pulse rate with a falling or stationary blood pressure always causes a fall in the Blood Urea Clearance.

The only explanation which presents itself, is that it may be due to a failing circulation. This is not the case, since it occurred in two of the most robust patients in the group.

<u>A Fall in Pulse Rate with a Fall in Blood</u> <u>Pressure</u>, when it occurs in the group of nephritis gives quite irregular results. In cases 1 and 3, it is accompanied by a rise in Blood Urea Clearance, but in Cases 4 and 5, it is accompanied by a fall in the Clearance.

In case 9 (Alice S. with essential hypertension with secondary nephritis) in reading 2 the clearance rises and in reading 4 it falls, although in each case the pulse rate and blood pressure fall.

In contrast to this are the findings in the Prostatic group. Wherever both the pulse rate, and blood pressure fall, the Blood Urea Clearance rises.

The same occurred in the case of renal calculus.

TO/

To summarize therefore, we find in none except the prostatic cases any recognizable relationship between the changes in the vaso-motor system which we have recorded, and the variations in the Blood Urea Clearance.

In the prostatic cases the Blood Pressure, and pulse pressure, considered alone, are no guide to the state of the kidney function.

The pulse rate alone, but better still, considered along with the blood pressure, is a guide, as to whether the kidney function is improving or deteriorating.

Thus in 72% of the recordings a fall in pulse rate indicated a rise in the Blood Urea Clearance.

In 82% of the recordings a rise in pulse rate indicated a fall in the Blood Urea Clearance.

Unless immediately after operation, a rise in pulse rate with a fall in Blood pressure, always meant a fall in the Clearance.

A fall in pulse rate along with a fall in Blood Pressure, always meant a rise in the Blood Urea Clearance.

The Blood Urea Clearance Test, compared with the Blood Urea Content as a Test for Renal Function, when Patient is under the Standard Conditions of Treatment.

In /

In the discussion of the relative merits of the various Kidney Function Tests, we have seen the unreliability of the Blood Urea content as an indication of renal function. This is because it is profoundly affected by the presence of dehydration or excessive water intake of the patient, etc.

If therefore, we have a patient under standard treatment regarding intake of water, type of diet, etc., the two main disadvantages are eliminated.

Accordingly, let us compare the Blood Urea Content with the Blood Urea Clearance, to see if and under what circumstances the former which is much the simpler test, can be with confidence substituted for the latter.

From the charts we see that in every case of <u>nephritis in the series</u>, with the exception of the 2nd reading in cases 3, 4 and 5, (Walter S., Annie P., and Joseph J.), the Blood Urea figure bears an inverse ratio to that of the Blood Urea Clearance, i.e., the blood urea falls as the clearance rises, and <u>vice versa</u>.

This relationship is not always found at the very beginning of treatment. The reason being presumably, that even although the kidney function does not improve, the treatment given is sufficient to bring down the Blood Urea level.

The ratio between the Clearance and the Blood Urea is not a quantitative one, but merely a qualitative/ qualitative one.

Thus, in case No. 1 (Vincent S.) we see that the Blood Urea Clearance rose from 40% to 68% (i.e., 28%) while the Blood Urea fell from 41 mgms % to 32 mgms% (i.e., 9 mgms). A fortnight later, the Blood Urea Clearance had risen from 68% to 69% (i.e., 1%), while the Blood Urea had fallen from 32 mgms % to 20 mgms % (i.e., 12 mgms %).

In Case No. 3 (Walter S.) Blood Urea Clearance fell from 67% to 60% (i.e., 7%), while Blood Urea rose from 30 mgms % to 54 mgms % (i.e., 24 mgms). By the next week the Blood Urea Clearance had fallen from 60% to 46% (i.e., 14%) while the Blood Urea rose from 54 mgms % to 55 mgms % (i.e., only 1 mgm %).

The danger of regarding the Blood Urea as a quantitative test, is seen in Case 6 (Joseph R.) reading 6, where the Blood Urea (46 mgms %), would lead one to expect a much higher Clearance than the 24% which accompanies it.

In the two Cases of Essential Hypertension, Nos. 9 and 10, there was no relationship found between the Blood Urea Clearance and the Blood Urea.

The same must be said of Case No. 8, where the patient has lardaceous disease involving the kidney. The Blood Urea remaining very low and steady and the Clearance being very high and variable. 47.

Examination/

Examination of the Prostatic group shows

that in several of their charts there is the same definite inverse ratio between the Blood Urea Clearance and the Blood Urea, as has been seen in the cases of nephritis, namely No.'s 11, 14, 15 and 19.

From their histories it will be remembered that these four had heavily infected urine. Case No. 5 had a pyelonephritis and at one time No. 1 was suspected of having the same, but his rapid recovery ruled out such a diagnosis.

Of the other cases of prostatic obstruction, in two of them, namely No.'s 12 and 13, the Blood Urea and the Blood Urea Clearance ran parallel. The remainder showed no relationship between the two.

It would appear that infection of the renal tract is the factor which makes the Blood Urea, Blood Urea Clearance ratio similar to that in nephritis.

In one of his articles Rickes¹⁵ indicates that he considers that infection in the urine reduces the Urea Clearance and such has been our experience in the pyelitis of pregnancy.

This is borne out by what we find in Case No. 20 (Ernest F.) suffering from a Lt. renal calculus. Cystoscopic examination showed that only one kidney was affected. Although one kidney, after removal of the other, can give a normal blood Urea Clearance, yet in this case, the Clearance was at first only 57%. This was apparently reflexly caused by the infection of/ of the kidney with the stone in it. As the infection cleared up, the Blood Urea Clearance rose and did so still further when the stone was removed, and the inverse ratio between the Clearance and the Blood Urea disappeared with the infection.

We must conclude therefore, that taking the prostate cases as a group, the fluctuations in Blood Urea give no indication of the direction taken by the fluctuations of the Blood Urea Clearance.

One important fact does emerge however from a review of the charts in this group. This is, that however much the Blood Urea and Blood Urea Clearance may fluctuate, no case with a Blood Urea Content of 40 mgms % or less, had ever a Blood Urea Clearance taken at the same time, less than 50%.

Patients waiting for prostatectomy are given a low protein diet, and abundant fluids, to eliminate the waste products which have accumulated in the system, during the period of urinary retention.

As this elimination proceeds the patient's general condition improves and the Blood Urea falls. It is feared by some⁵⁴ that the kidney may be efficient enough to do its work only under such very favourable conditions, and the apparent health of the patient, and the low blood Urea, may be obscuring considerable kidney weakness.

In/

In none of our cases was this fear justified. Every case with a blood urea within normal limits, had a Blood Urea Clearance of 50% or more. Most surgeons consider that a Clearance of 50% indicates a kidney efficient enough to stand the strain of the operation for removal of the prostate, and such has been our own experience.

SUMMARY.

We have ennumerated the various kidney function tests, and pointed out the possible fallacies in each.

This has been accompanied by a review of the recent research done on the subject, by many authorities, to show that the Blood Urea Clearance test is the most reliable test of the functional ability of the kidney.

Comparing the Blood Urea Content with the Blood Urea Clearance, we find that in our cases of nephritis under treatment for a few days, the Blood Urea always bears an inverse ratio to Blood Urea Clearance. Changes in the Blood Urea can thus be used as a guide to the changes taking place in the Blood Urea Clearance, but the actual level of the Blood Urea can never be relied upon to give an indication/

. .

indication of the actual Blood Urea Clearance reading.

In cases of prostatic retention recorded, the the same inverse ratio between the Blood Urea and the Blood Urea Clearance seen in the nephritics, was seen only in those cases with a definite infection of the urinary tract. It appears that this infection of the urinary tract affects the Blood Urea Clearance more profoundly than actual damage to the renal tissue itself.

There was no such ratio found in the other cases of prostatic obstruction in the series.

Thus, in the prostate cases in general, the fluctuations in Blood Urea cannot be used as a guide to Blood Urea Clearance.

It was found, however, that when the Blood Urea was 40 mgms % or less, it always indicated a Blood Urea Clearance of 50% or more.

In none of the cases presented, with the exception of the prostatic group, could the variations in Blood Pressure, Pulse Pressure or Pulse rate, taken singly or combined, be used as an indication of the progress of the renal function.

In the Prostatic Cases, the Pulse Rate, and better still the pulse rate and Systolic Blood Pressure taken together, may be used as a reliable guide to the improvement/ improvement or deterioration of the kidney function, as measured by the Blood Urea Clearance test.

In 72% of the occasions on which there was a rise in the pulse rate, it was accompanied by a fall in the Blood Urea Clearance.

In 82% of the times a fall in pulse rate was recorded, it was accompanied by a rise in the Blood Urea Clearance.

Thus, if in our prostatic cases, we consider that a rise in pulse rate indicates a fall in the Urea Clearance, and a fall in pulse rate to indicate a rise in the Urea Clearance, we shall be correct in 82% of cases.

A more accurate guide is found in combining the readings of the Pulse rate with those of the Blood Pressure.

Except during the few days immediately following an operation, a falling pulse rate along with a falling systolic blood pressure, means a rise in the Blood Urea Clearance in 100 % of cases.

A rise in the pulse rate accompanied by a falling, a stationary blood pressure, means a fall in the Blood Urea Clearance.

Thus in dealing with cases of prostatic enlargement/ enlargement under treatment, it appears that by making frequent use of Blood Pressure and Pulse Rate readings, frequent kidney function tests are unnecessary. At the outset, an estimation of the Blood Urea Clearance gives an idea of the state of the kidney. Thereafter, Blood Pressure, and Pulse rate give a sufficiently accurate guide to the improvement or deterioration of the kidney function. Later, the Blood Urea content may be taken, and if below 40 mgms % indicates a Urea Clearance of at least 50%. The Blood Urea Clearance should then be taken for the accurate estimation of the kidney function.

In this way much of the time and expense incurred by the usual frequent Blood Urea Clearance estimations, may be saved and more use made of the clinical findings, without loss of efficiency, or insight into the progress of the patient's disease.

BIBLIOGRAPHY

1.	<u>R. BRIGHT</u> . Guy's Hosp. Reports, 1836, First Series, p. 338.
2.	<u>N. GRÉHART</u> . Journ. Physiol. et Path. gén. 1904, VI, 1.
3.	L. G. ROWANTREE and J. T. GERAGHTY. Journ. Pharm. & Exper. Therapy. 1910, 1. 579.
4 •	L. AMBARD and A. WEILL. Journ. Physiol. et Path. gen. 1912. XIV. 753.
5.	H. MACLEAN and O. L. V. de WESSELOW. Quart. Journ. Med. 1919, XII, 347.
6.	H. MACLEAN and O. L. V. de WESSELOW. Brit. Journ. Exper. Path. 1920. 1. 53.
7.	G. A. HARRISON, Brit. Journ. Exper. Path. 1922. 3. 28.
8.	J. H. AUSTIN, E. STILLMAN, D. D. Van SLYKE. Journ. Biol. Chem. 1921. 46. 91.
9.	D. D. Van SLYKE, C. P. ROADS, HILLIER, A. S. ALVING. Amer. Journ. Physiol. 1934.109.336.
10.	<u>A. M. FISHBERG</u> , Hypertension and Nephritis, 1935, $p \cdot 59$.
n.	<u>G. GIGLIOLI, Malarial Nephritis, London, 1930,</u> Part III, p. 78.
12.	<u>R. L. JOHNSTON</u> , Journ. Lab. & Clin. Med., 1929-30, Vol. XV. p. 943.
13.	PRIESTLY, Journ. Physiol 1921.55.305.
14.	A. S. ALVING, and D. D. Van SLYKE. Journ. Clin. Invest. 1934. XIII. 969.
15.	E. W. RICHES and J. D. ROBERTSON. Brit. Journ. Surg. Vol. XXIII. 89. pp. 128-140.
16.	J. P. PETERS, Bull. New York Academ. Med. 1934. X. 415.
17.	M. G. WOHL and R. W. BRUST, Journ. Lab. & Clin. Med. 1934-5. XX. pp. 1170-1179.
18.	<u>I. J. WOOD</u> , Brit. Med. Journal. 18th July 1936, p. 116.
19./	

19.	E. M. MACKAY and L. L. MACKAY, Journ. Clin. Invest. 1927. IV. 127.
20.	A. E. OSTERBERG and N. M. KEITH, Journ. Lab. & Clin. Med. 1934-5 XX, 141.
21.	E. K. MARSHALL and D. M. DAVIES. Journ. Biol. Chem. 1914 - 18 - 53.
22.	J. P. PETERS and D. D. Van SLYKE. Quant. Clin. Chem. Interpretations, London 1931. p. 286.
23.	A. N. RICHARDS & SCHMIDT. Amer. Journ. Physiol. 1924. 71. 128.
24.	J. P. PETERS and VAN SLYKE. Quant. Clin. Chem., Baltimore 1932 p. 297.
25.	SAMSON WRIGHT'S APPLIED PHYSIOLOGY, 1934. Chap. 8. p. 531.
26.	D. D. Van SLYKE, J. F. McINTOSH, E. MÖLLER, C. JOHNSTON, R. R. HAMMON., Journ. Clin. Invest. 1930. 8. 357.
27.	<u>P. B. REHBERG</u> , Biochem. Journ., 1926. 20. 447- 461.
28.	R. DOMINGUEZ and E. POMERENE, Journ. Biol. Chem. 1934. 104. 449.
29.	W. H. NEWTON. Recent Advances in Physiology. 1936. p. 403.
30.	C. L. COPE. Journ. Clin. Sc. 1935, II, 35.
31.	A. M. FISHBERG. Hypertension & Pephritis, 1935 p. 590.
32.	<u>P. BLASUCCI</u> . Journ. Urol. 1936-36-564.
33.	H. H. YOUNG. Journ. Urol. 1931.26.25.
34.	SAMSON WRIGHT'S APPLIED PHYSIOLOGY, 1934. Chap. 5, p. 332.
35.	MAYO. Journ. Amer. Med. Assoc. 1927; 89. 1057.
36.	HAYMAN, Amer. Journ. Physiol, 1927-79-389.
37.	W. G. BALL and G. EVANS. Diseases of the Kidney, London 1932. Chap. 1, p. 12.
38.	PRICE'S TEXT BOOK of MEDICINE, 1933. Sec. XVI. p. 1262.
39/	

39 •	A. M. FISHBERG. Hypertension and Nephritis, 1935, p. 552.
40.	E. KYLIN, Svenska Handlingar, 1923, XLIX, 130.
41.	J. S. DUNN and J. W. MACNEE. Brit. Med. Journ. 1917-2-745.
42.	<u>W. J. DICKMAN.</u> Amer. Journ. Obst. & Gyn. 1936. XXXI, 741.
43.	E. M. MACKAY. Journ. Clin. Invest. 1928. 6. 505.
44.	I. H. PAGE. Journ. Clin. Invest. 1934. XIII, 909.
45 ·	I. H. PAGE and G. J. HENER. Journ. Clin. Invest. 1935. XIV. 22.
46.	I. H. PAGE and G. J. HENER. Journ. Clin. Invest. 1935. XIV. 27.
47 •	R. J. KELLAR and W. M. ARNOTT. Brit. Med. Jour. 1936. Aug. 15. p. 372.
48.	J. P. PETERS and D. D. Van SLYKE, Quant. Clin. Chem., Baltimore 1932, Vol. II, p. 564.
49.	W. H. OLMSTEAD & J. R. CAULK, Journ. Amer. Med. Assoc. 1922, 79. 1380.
50.	J. B. SQUIER, C. G. BANDLER and V. C. MYERS, Journ. Amer. Med. Assoc., 1922-79-1384.
51.	A. M. FISHBERG, Hypertension & Nephritis, 1935, p. 215.
52.	J. C. BRAMWELL. Quart. Journ. Med., 1934. XVII, 225.
53.	A. M. FISHBERG. Hypertension & Nephritis, 1935. p. 208.
54.	B.W.R.I.URQUHART and J. L. McCOLLUM. Canad. Med. Assoc. Journ. 1935. XXIII. 251.
1	

SCALES USED IN THE CHARTS

Blood Urea Clearance -----. XY = 0., .1 ins = 2 units.

Blood Urea ----- XY = 20, ·l ins = 2 units.

Blood Pressure ---- XY = 60. 1 ins = 2 units.

Pulse Pressure ----- XY = 20. 1 ins = 3 units.

Pulse Rate ----- XY = 60. ·1 ins = 1 unit.

M = "Maximal" Blood Urea Clearance.

CASE NO. 1.

Name: VINCENT S. Act. 15 yrs.

Disease: Subacute Nephritis.

57.

Case History:

- 4 weeks before admission gt. developed. v. severe sore-throat suggestive of diphth., but swab of throat was K.S.B. - ve.
- 2 weeks before admission developed oedema of face and hands.
 - 8.10.35 Admitted to hospital. Pt. very pale. No oedema present. Throat swab gave no culture, Strep. Haeno-Lyt. and S.Viridans.
- 11.10.35 Pt. had attack of vomiting.
- 12.10.35 B.P. 138/85. Pulse Press. 53. Pulse 80.

Blood Urea 20 mgms %. Blood Urea Clearance C_s 16.91% Urine contained Blood and Albumin. Esbach Reading G.4¹/₂.

- 17.10.35 Electro-cardiogram showed. Lt.Dominance.
- 21.10.35 B.P. 140/80. Pulse Press 60. Pulse 80. Blood Urea 40 mgms% B.U.C. (C_s) 27.65% Urine contained Blood and Albumin. Eshach reading $G.\frac{3}{4}$.
- 5.11.37 B.P. 130/80: Pulse Press. 50: Pulse 80. Blood Urea 41 mgms %. B.U.C. (C_g) 40.31% Urine contained Blood and Albumin. Esbach reading. $G.\frac{1}{2}$.

8.11.37 Return of headache and vomiting.

15.11.37 B.P. 110/78. Pulse Press 32. Pulse 85. Blood Urea 32 mgms % B.U.C. (C_s) 67.71%. Urine contained Blood and Albumin. Esbach reading G.¹/₄.

23.11.37 Pt. developed a follicular tonsillitis. Complained of Headache.

 29.11.37 B.P. 130/70: Pulse Press 60. Pulse 88.
 Blood Urea 20 mgm %. Blood Urea Clearance (Cs) 69.14%
 Urine contained Blood and Albumin.
 Esbach gave trace only.

.....

12/

CASE NO. 1. (Contd.).

12.11.37 B.P. 114/70. Pulse Press. 44. Pulse 80. Blood Urea 20 mgms %. Blood Urea Clearance (C_s) 72.15% Urine contained Blood and Albumin. Esbach reading, trace only.

Case II /

CASE NO. 2.

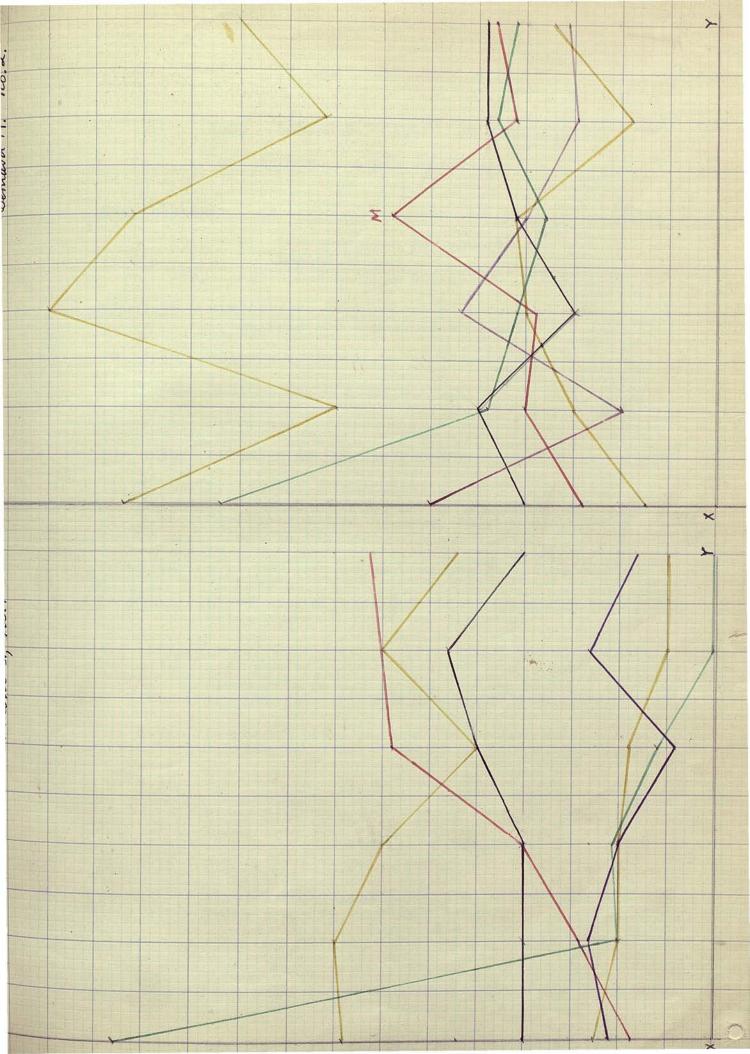
Name: BERNARD M. Act. 44.

Disease: Subacute Nephritis.

Case History:

5 weeks before admission began to complain of "feeling out of sorts," constipation, pain across his back. 3 days later developed "a sore-throat". 2 weeks later noticed swelling of his feet and legs. 28.10.36 Patient admitted. Was vomiting. Complaining of headache. Oedema present in feet and legs. No other abnormality found. 29.10.36 B.P. 184/75: Pulse Press 109: Pulse 80 Blood Urea 123 mgm %. B.U.C. = Cs 28.12% Urine Contains blood and Albumin. Esbach reading G.5. 4.11.36 B.P. 140/90. Pulse Press. 50. Pulse Rate 85. Blood Urea 67 mgm %. B.U.C. = Cs 39.99%. Urine contains blood and albumin. Esbach reading G.4. 11.11.36 B.P. 200/100. Pulse Press 100. Pulse Rate 75. Blood Urea 62 mgm %. B.U.C. = Cg 39.14%. Urine contains Blood and Albumin. Esbach reading G.4. 18.11.36 B.P. 182/102. Pulse Press 80. Pulse Rate, 81. Blood Urea, 55 mgm %. B.U.C. = Cm 68.42 %. Urine contains Blood and Albumin. Esbach reading G. 15. B.P. 142/78, Pulse Press 64, Pulse Rate 84. 25.11.36 Blood Urea 65 mgm %, B.U.C. = C₈ 43.47%. Urine contains Albumin but no blood. Esbach reading G. 2. B.P. 160/94, Pulse Press. 66, Pulse Rate84. 2.12.36 Blood Urea 62 mgm %, B.U.C. = C_B 47.36 %.

During the above period the patient's general condition improved slowly, steadily and uneventfully.



CASE NO. 3.

Name: WALTER S. Act: 38 years.

Disease: Acute Nephritis.

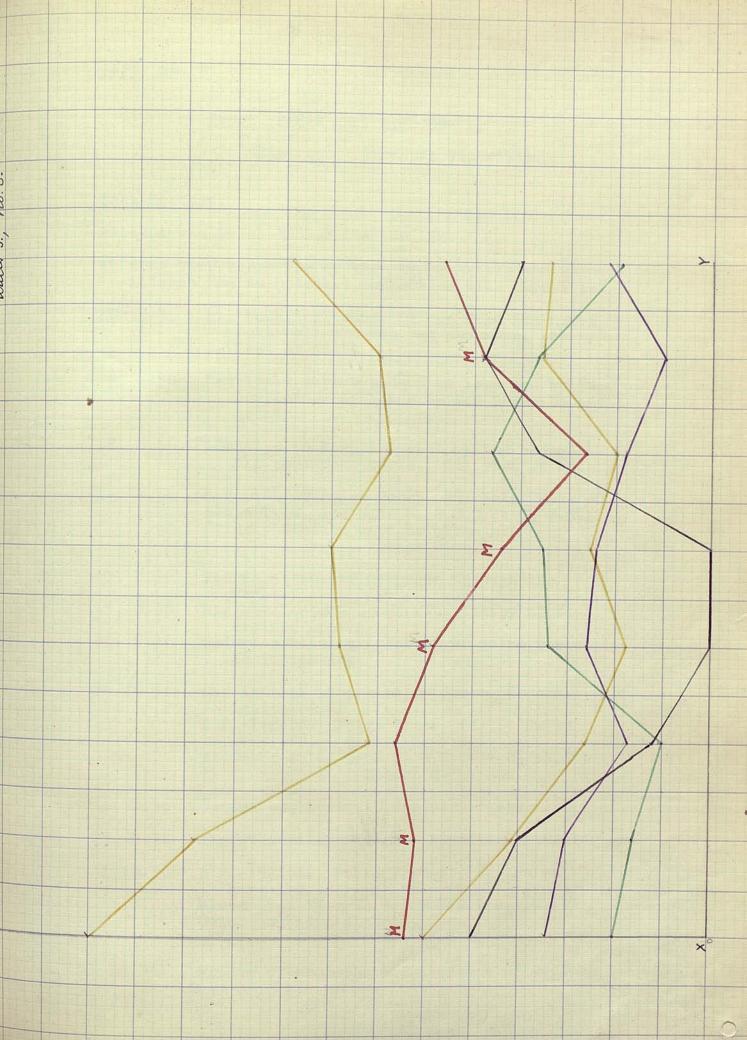
Case History:

	191	8. Pt. had operation for chronic			
	193	mastoiditis. 3. Again had mastoid operation.			
7.	3.36	Admitted to Hospital with otorrhoea, aural polypus and a mastoid abscess.			
10.	3.36	Polypus removed.			
27.	5.36	Radicle Mastoid operation.			
7.	6.36	Developed Erysipelas.			
20.	6.36	All sign of Erysipelas gone.			
9.	7.36	Pt. developed albuminuria.			
11.	7.36	Developed oedema of face, scrotum and sacral region.			
22.	7.36	B.P. 190/120, Pulse Pressure 70, Pulse Rate 85. Blood Urea 40 mgms % B.U.C. = C _m 64.83%. Urine contains Blood and Albumin. Esbach G.3.			
29	7.36	B.P. 168/103, Pulse Press 65, Pulse Rate 80. Blood Urea 36 mgm %, B.U.C. = C_m 63.44% Haematuria. Esbach G.l ¹ / ₄ .			
4.8	8.36	Haematuria still present but oedema has disappeared.			
5. 1	8.36	B.P. $132/86$, Pulse Press. 46, Pulse Rate 66. Blood Urea 30 mgm %. B.U.C. = C_B 67.34%. Haematuria. Esbach G. $\frac{1}{2}$.			
12. (8.36	 B.P. 138/78. Pulse Press. 60. Pulse Rate 60. Blood Urea 54 mgms %. B.U.C. = Cm 59.54%. Urine contains Blood and albumin. Esbach G.¹/₄. 			
19./					

CASE NO. 3 (Contd.).

- 19. 8.36 B.P. 140/85. Pulse Press. 55, Pulse Rate 60.
 Blood Urea 55 mgm %. B.U.C. = Cm 45.734%. Urine contains Blood and Albumen. Esbach G.¹/₄.
- 10. 9.36 B.P. 128/80. Pulse Press. 48. Pulse Rate 78. Blood Urea 67 mgm %. B.U.C. = C_B 26.08%. Urine contains Blood & Albumin. Esbach G. 1/2.
- 17. 9.36 B.P. 130/95. Pulse Press. 35. Pulse Rate84. Blood Urea 56 mgms % B.U.C. = C_m 48.59%. Urine contains Albumin but no Blood. Esbach G. $\frac{1}{4}$.
- 6.10.36 B.P. 148/94. Pulse Press 54. Pulse Rate 80. Blood Urea 39 mgm %. B.U.C. = Cs 56.35%. Urine contains Albumin but no blood. Esbach G.¹/₄.

Case 4 /



CASE NO. 4.

Name: ANNIE P. Act. 50.

Disease: Subacute Nephritis.

Case History:

Patient was twice in hospital in 1931.

- At that time there was no sign of nephritis and the illness had no possible connection with nephritis.
- 22. 7.36 Pt. took influenza. Since then has never felt well and has had oedema of the feet at night.
- 9.8.36 Developed severe oedema of legs and vulva, complained of headache and urine became scanty.
- 12. 8.36 Admitted to hospital with marked oedema of all dependent parts. Nil else of note.
- 14. 8.36 B.P. 168/80 Pulse Press. 88. Pulse Rate 90. Blood Urea. 134 mgms %. B.U.C. = C_s 7.58%. Urine contained copious albumin. Esbach G. 6.
- 17. 8.36 Had an attack of dyspnoea through the night.
- 18. 8.36 Has developed a cough with frothy bloodstained sputum.
- 20. 8.36 Oedema subsiding. Volume of urine passed is increasing.
- 26. 8.36 Had another attack of dyspnoea. Oedema increased. B.P. 148/84. Pulse Press. 64. Pulse Rate 108. Blood Urea 162 mgms %. B.U.C. = Cs 11.43% Esbach G. 5.
- 31. 8.36 Oedema beginning to diminish again.

23. 9.36 Improvement continues. Appetite returning.

1.10.36 B.P. 152/84. Pulse Pressure 68. Pulse Rate 110. Blood Urea 75 mgms %; B.U.C.= C_s 18.13%. Urine contains Albumin. Esbach G.3.

6.10.36. Return of headache and increase of oedema. 8.10.36/

GASE NO. 4 (Contd.).

8.10.36 Electrocardiogram shows St.ventricular dominance.
B.P. 154/85. Pulse Pressure 69. Pulse Rate 10.
Blood urea 47 mgms %. B.U.C. = C_s 34.04%.
Urine contains albumin.

Esbach G.2.

16.10.36 B.P. 148/86. Pulse Pressure 62. Pulse Rate 94.
Blood Urea 53 mgms %. B.U.C. = C_s 24.56%. Urine contains albumin. Esbach G.1¹/₂.

Case 5/

CASE NO. 5.

Name: JOSEPH J. Act: 54.

Disease: Chronic Nephritis.

Case History.

- 1907. Pt. had his first attack of "dropsy" and says he has frequently had mild attacks since.
 1917. While in the army had nephritis.
 10. 6.36. Patient admitted to hospital with oedema of the face and large Enlargement of
- 10. 0.90. Patient admitted to hospital with bedema of the face and legs. Enlargement of heart to the left. Cough, nocturnal dyspnoea, albuminuria retinitis.

11. 6.36 Electrocardiogram shows myocardial damage.

- 15. 6.36 B.P. 205/140. Pulse Pressure 65. Pulse Rate 92. Blood Urea 150 mgms %. B.U.C. = C_m 37.06%. Urine contains albumin. Esbach $G.\frac{3}{4}$.
- 16. 6.36 Pt. has developed a carbuncle.
- 20. 6.36 Return of nocturnal dyspnoea.
- 30. 6.36 Oedema of legs again appearing. Carbuncle healing well.
- 5. 7.36 % nausea in the morning.

17. 7.36 Has developed a furuncle on arm.

- 29. 7.36 B.P. 232/130. Pulse Pressure 102. Pulse Rate 80. Blood Urea 108 myms %. B.U.C. = C_s 24.6%. Albuminuria continues. Esbach. $G.\frac{3}{4}$.
- 6.8. 36 B.P. 220/135. Pulse Pressure 85. Pulse Rate 88. Blood Urea 96 mgms %. B.U.C. = Cm & s 26.6% Albuminuria continues. Esbach. G.¹/₂.
- 12. 8.36 B.P. 212/130. Pulse Pressure 82. Pulse Rate 84. Blood Urea 98 mgms %. B.U.C. = C_s 16.55 %.

13/

CASE NO. 5 (Contd.).

13. 8.36 Furnncle developed on right hand.

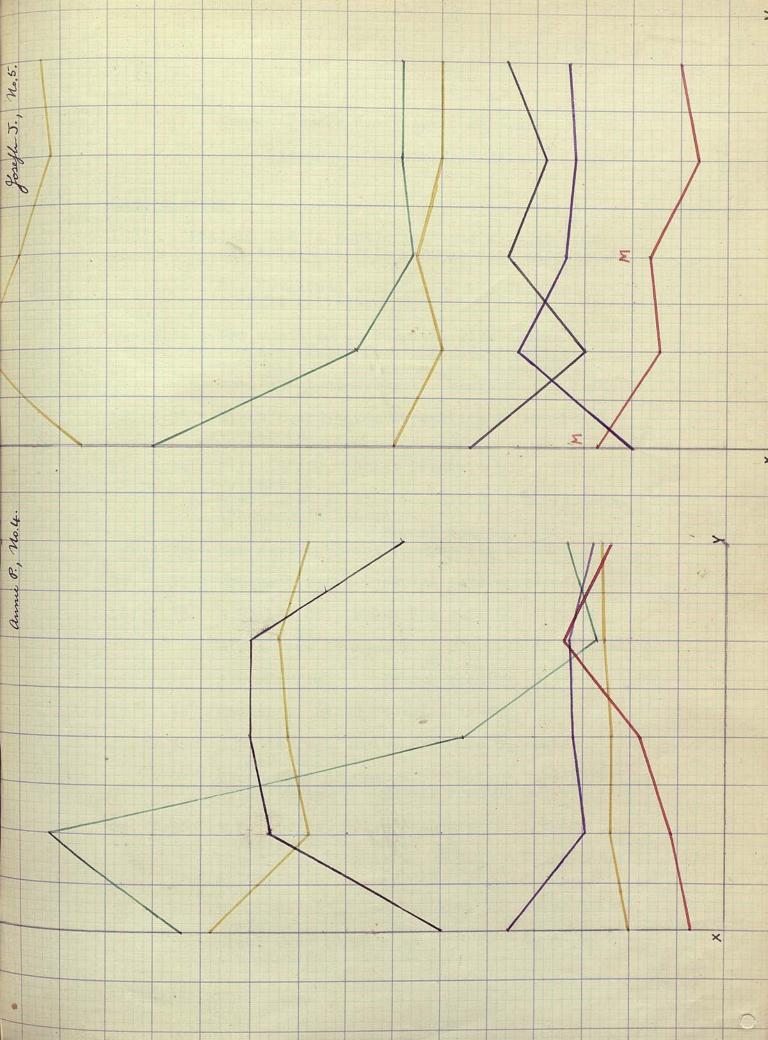
16. 8.36 Cellulitis of right hand developing.

18. 8.36 Hand improving.

19. 8.36 B.P. 214/130. Pulse Pressure 84. Pulse Rate 88. Blood Urea 98 mgms %. B.U.C. = C₈ 19.66%. Albuminuria continues. Esbach. G. ³/₄.

- 1. 9.36 General condition deteriorating.
- 8. 9.36 Patient died.

Case 6/



CASE NO. 6.

Name: JOSEPH R. Aet. 15 years.

Disease: Chronic Nephritis.

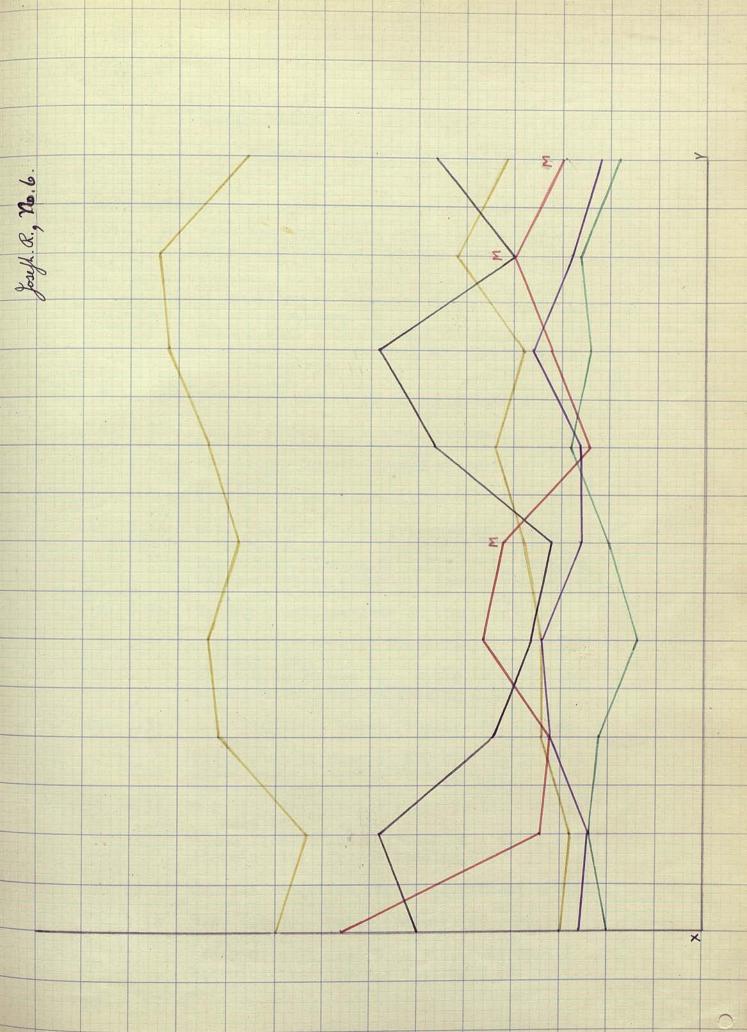
Case History:

1934. Pa	was told that "his kidneys were affected."
2. 2.36	Pt. developed swelling of the face but felt well otherwise.
9. 3.36	Was admitted to hospital with oedema of face, legs, feet and sucral region. Very pale. Has album. and haematuria. Some enlargement of the heart to the left.
10. 3.36	Electrocardiogram suggestive of mycardial damage.
11. 3.36	 B.P. 150/90. Pulse Pressure 60. Pulse Rate 90. Blood Urea 40 mgms %. B.U.C. = C₈ 74.925%. Albuminuria continues.
2. 4.36	Esbach reading G.3. Has developed Aortic Systolic murmur.
29. 5.36	Frequent extra-systoles have appeared.
10. 7.36	 B.P. 144/88. Pulse Pressure 56. Pulse Rate 94. Blood Urea 43 mgms %. B.U.C. = C₈ 33.82%. Albuminuria. Esbach reading, G.3.
17.7.36	Return of blood to urine to-day.
21. 7.36	 B.P. 163/94. Pulse Pressure 69, Pulse Rate 82. Blood Urea 42 mgms %. B.U.C. = C₈ 32.19%. Albuminuria. Esbach reading G.3.
28. 7.36	B.P. 164/94, Pulse Pressure 70. Pulse Rate 78. Blood Urea 35 mgms %. B.U.C. = C _B 46.43%. Albuminuria. Esbach. G.1.
5. 8.36	B.P. 158/98, Pulse Pressure 60, Pulse Rate 76. Blood Urea 40 mgms %. B.U.C. = Cm 42.02 % Albuminuria. Esbach G.4.
11. 8.36/	

CASE NO. 6 (Contd.).

11. 8.36. B.P. 164/104, Pulse Pressure 60, Pulse Rate 88. Blood Urea 47 mgms %. B.U.C.= C_B 23.495 %. Albuminuria. Esbach reading G.3. 12. 8.36 Oedema of feet has returned. Blood in urine to-day. 18. 9.36 24. 9.36 B.P. 172/98. Pulse Pressure 74. Pulse Rate 94. Blood Urea 45 mgms %. B.U.C.= 03,31.09%. Albuminuria. Esbach reading G.52. 27.10.36 Albuminuria gradually increasing. Has loud Aortic and Pulmonary systolic 5.11.36 murmurs. B.P. 174/112, Pulse Pressure 62, Pulse Rate 80. Blood Urea 47 mgms %. B.U.C. = Cm 39.91% Albuminuria. Esbach reading G.7. Murmurs both disappeared. 11.11.36 B.P. 156/102. Pulse Pressure 54, Pulse Rate 88. Blood Urea 38 mgm %. B.U.C. = Cm 30%. Albuminuria. Esbach reading, G.6.

Case 7/



CASE NO. 7.

Name: WALTER M. Aet. 49 yrs.

Disease: Myocarditis and Nephritis.

Case History:

191	6 Pt.	developed pneumonia after having had chronic bronchitis for many years.
8.	2.36	Admitted to hospital, complaining of swelling of feet and hands. Cough and dyspnoea. During next 2 months had intermittent attacks of haematoea and had constant albuminuria.
7.	4.36	Developed oedema of bases of both lungs.
8.	6.36	Had an attack of urgent dyspnoea.
16.	7.36	Electrocardiogram showed myocardial change. Discs show no abnormality.
30.	7.36	 B.P. 138/90. Pulse Pressure 48. Pulse Rate 76. Blood Urea 53 mgms % B.U.C. = Cm 38.64 %. Urine contains albumin. Esbach reading G.3.
4.	8.36	Having breathlessness in the mornings.
5.	8.36	B.P. 128/90. Pulse Pressure 38. Pulse Rate, 88. Blood Urea 30 mgm %. B.U.C. = C _s 56.42%. Albuminuria. Esbach reading G.5.
13.	8.36	 B.P. 160/105. Pulse Pressure 55. Pulse Rate 80. Blood Urea 51 mgms % B.U.C. = C_s 49.32 %. Albuminuria. Esbach reading G.2¹/₂.
19.	8.36	 B.P. 130/96. Pulse Pressure 34. Pulse Rate 80. Blood Urea 36 mgms %. B.U.C. = C_s 72.834%. Albuminuria. Esbach reading G.2.
30.	8.36	General condition improving. Chest clearing
10.	9.36	 B.P. 110/90. Pulse Pressure 20. Pulse Rate 92. Blood Urea 31 mgms %. B.U.C. = C_s 79.53%. Albuminuria. Esbach reading G 12.
18.	9.36/	

CASE NO. 7 (Contd.)

- B.P. 130/96. Pulse Pressure 34. Pulse 18. 9.36 Rate 80. Blood Urea 36 mgms %. B.U.C. = C_s 72.834% Albuminuria. Esbach reading G.2. 30. 8.36 General condition improving. Chest clearing. B.P. 110/90. Pulse Pressure 20. Pulse 10. 9.36 Rate 92. Blood Urea 31 mgms %. B.U.C. = C. 79.53%. Albuminuria. Esbach reading $G.1\frac{5}{2}$. 18. 9.36 Return of rules in chest. 30. 9.36 General condition deteriorating during last 2 weeks. B.P. 130/90. Pulse Pressure 40. Pulse 6.10.36
 - 10.36 B.P. 130/90. Pulse Pressure 40. Pulse Rate 96. Blood Urea 30 mgms %. B.U.C. = Cm 65.21% Albuminuria. Esbach. G.4.

Case 8/

CASE NO. 8.

Name: JOHN B. Act: 15.

Disease: Spinal Caries with Lardaceous kidney.

Case History:

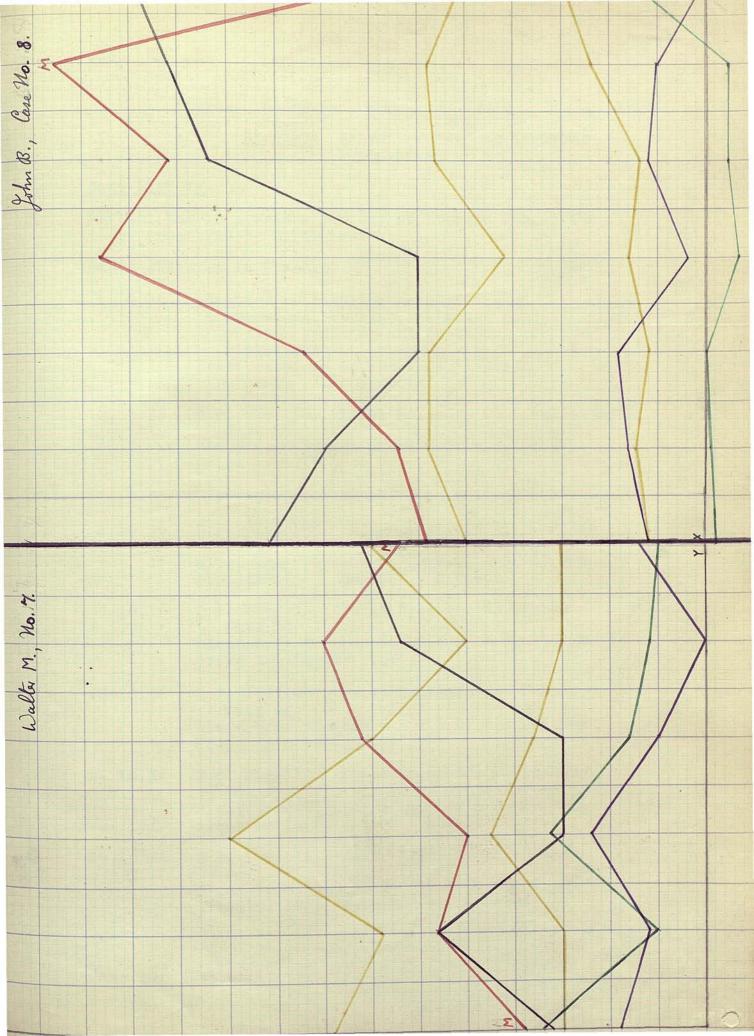
1930. Patient developed tuberculosis of the spine. Was put on a spinal frame, on which he has remained ever since. 14. 9. 36. Albuminuria first appeared. 15. 9.36. B.P. 110/72. Pulse Pressure 38. Pulse Rate 106. Blood Urea 18 mgm %. B.U.C. = C. 58.77% Albuminuria. Esbach reading G.6. 23. 9.36 Pt. becoming oedematous. 2.10.36 B.P. 118/75. Pulse Pressure, 43. Pulse Rate 100. Blood Urea, 19 mgms %. B.U.C. = C. 64.89%. Albuminuria. Esbach reading G.7. 6.10.36 Oedema increasing. 9.10.36 High Protein diet begun. This diet continued from now onwards. B.P. 118/72. Pulse Pressure 46. Pulse 12.10.36 Rate 90. Blood Urea 20 mgms %. B.U.C. = Cs 84.36%. Albuminuria. Esbach reading G. 7. 18.10.36 Some decrease in the oedema. B.P. 102/76. Pulse Pressure 26. Pulse 20.10.36 Rate 90. Blood Urea 13 mgms %. B.U.C. = C_s 126.54%. Esbach reading G.7. Albuminuria. B.P. 116/74. Pulse Pressure 42. Pulse 30.10.36 Rate 112. Blood Urea 15 mgms %. B.U.C.= Cs 112.11%. Esbach reading G.7. Albuminuria. 3.11.36 Oedema again increasing. B.P. 118/84. Pulse Pressure 34. Pulse Rate 6.11.36 116. Blood Urea 15 mgms % B.U.C. = 137.16%. Albuminuria. Esbach. G.51.

12.11.36/

CASE NO. 8 (Contd.).

12. 11. 36 B.P. 110/90. Pulse Pressure 20. Pulse Rate 120. Blood Urea 36 mgms %. B.U.C. = Cs 61.86% Albuminuria. Esbach reading, G.8.

Case 9./



Name: Alice S. Act. 36 years

Disease: Arterio-sclerotic Nephritis-Hyperpiesia.

Case History:

Patient has no previous history of illness suggestive of nephritis or hyperpiesia.

Had 6 children in 9 years.

9.9.36	Admitted to hospital complaining of headaches, vertigo, malaise, cold
	sweats, vomiting and frequency of micturition.
	Heart enlarged to the left. Discs normal. Albuminuria.

10. 9.36 B.P. 220/130. Pulse Pressure 90. Pulse Rate 96. Blood Urea 32 mgm %. B.U.C. = Cs 64.63. Urine Albumin free.

17. 9.36 Electrocardiogram shows no abnormality.

- 1.10.36 B.P. 200/125. Pulse Pressure 75. Pulse Rate 74. Blood Urea 28 mgms %. B.U.C. = Cm 83.89%. Albuminuria. Esbach. A trace.
- 8.10.36 B.P. 195/112. Pulse Pressure 83. Pulse Rate 90.
 Blood Urea 31 mgms %. B.U.C. = Cm 73.81%.
 Urine is albumin free.
- 16.10.36 B.P. 152/108. Pulse Pressure 44. Pulse Rate 64. Blood Urea 30 mgms %. B.U.C. = C_s 44.16%. Urine is albumin free.
- 23.10.36 B.P. 202/120. Pulse Press.82. Pulse Rate 72. Blood Urea 30 mgms % B.U.C. = Cm 49.77% Albuminuria. Esbach. Slight trace.

29.10.36 B.P. 192/124. Pulse Pressure 68. Pulse 86.
Blood Urea 28 mgms % B.U.C. = Cm 39.48%.
Albuminuria. Esbach. Slight trace.

Case 10/

CASE NO. 10.

Name: CHARLES P. Act. 50.

Disease: Hyperpiesia Arterio-sclerotic Nephritis.

Case History.

5. 6.36	Went to his own Doctor complaining of
	swelling of his face and ankles.
	Was found to have albuminuria and
	glycosuria.

- 11. 6.36 Had sudden attack of dysphoea.
- 17. 6.36 Dysphoeic attack. Albuminuria and glycosuria continue.
- 19. 6.36 Admitted to hospital. Ht. increased to Lt. Alb. and Glycosuria.
- 20. 6.36 Blood sugar .271% Given diabetic diet. No insulin.
- 22. 6.36 B.P. 180/110. Glycosuria and albuminuria continue.
- 30. 6.36 Blood Sugar .200%. Pt. given ordinary light diet.
- 6. 7.36 B.P. 156/98. Pulse Pressure 58. Pulse Rate 104.
 Blood Urea 51 mgm % B.U.C. = Cs 55.5%. Urine contains sugar and albumin. Sugar 1.5%. Esbach G. 12.
- 7. 7.36 Blood Sugar .340%.

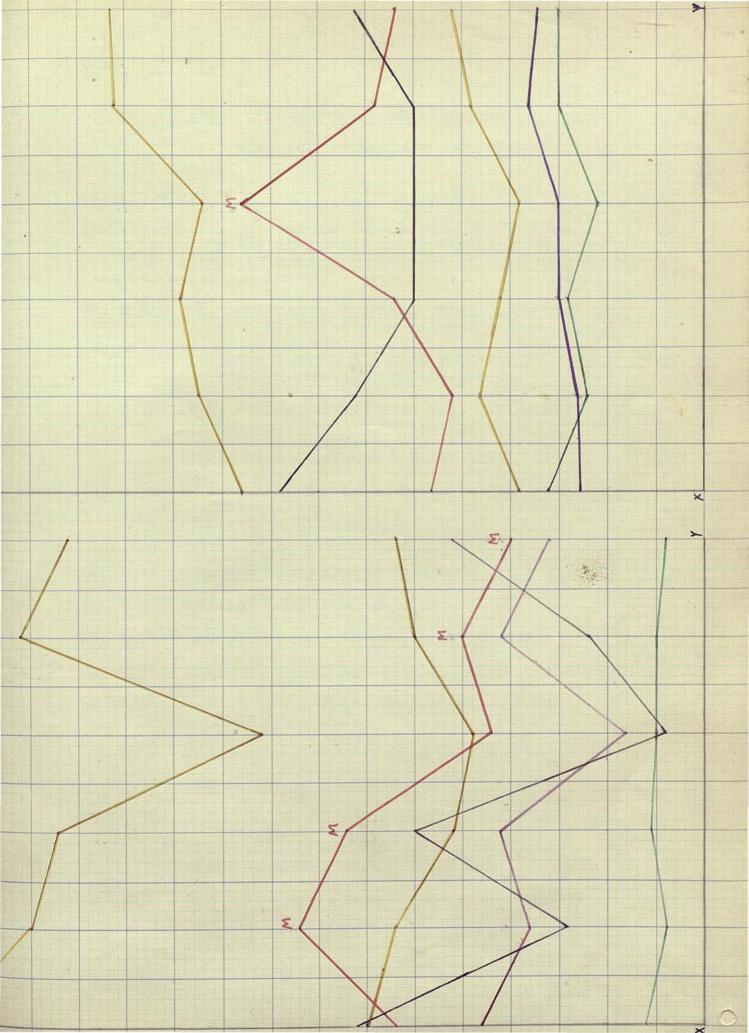
14. 7.36 B.P. 165/106. Pulse Pressure 59. Pulse Rate 96. (Blood Sugar .234%) Blood Urea 43 mgm %. B.U.C. = Cs 51.48%. Urine sugar free. Albuminuria. Esbach G.3.

21. 7.36 B.P. 168/102. Pulse Pressure 66. Pulse Rate 90. Blood Sugar .330%, Blood Urea 49 mgm%, B.U.C. = Cs 64.75%. Urine Sugar free. Albuminuria. Esbach G.2.

28. 7.36/

CASE NO. 10	(Contd.)
	 B.P. 164/98. Pulse Pressure 66. Pulse Rate 90. Blood Sugar .265%) Blood Urea 42 mgms%. B.U.C. = Cm 97.35%. Urine Sugar free. Albuminuria. Esbach G 3.
30. 7.36	Discs show retinal changes.
4. 8.36	(Blood Sugar .400%).
	 B.P. 182/108. Pulse Pressure 74. Pulse Rate 90. Blood Urea 50 mgms %. B.U.C. = C₈ 29.3%. Urine Sugar free. Albuminuria. Esbach. G. 2¹/₂.
8. 8.36	Electrocardiogram shows myocardial change and left dominance.
	 B.P. 183/112. Pulse Press. 71. Pulse Rate 96. Blood Urea 50 mgm %. B.U.C. = Cg 64.528%. Blood Sugar .326%. Urine sugar free. Albuminuria. Esbach G.2.

Case 11./



CASE NO. 11.

Name: GEORGE A. Act. 67 yrs.

Disease: Prostatic retention.

Case History.

- For 4 years the patient has been having increasing frequency and difficulty in micturition with occasional attacks of retention, when he then catheterises himself.
- 30. 8.36 Pt. admitted to hospital, thin, pale and ill, with distended bladder and enlarged prostate. Catheter passed, fixed in position, bladder gradually decompressed.
- 3. 9.36 B.P. 142/82 Pulse Press. 60. Pulse Rate 80.
 Blood Urea 68 mgms %.
 Blood Urea Clearance = C₈ 67.76%.
- 9.9.36 B.P. 102/70. Pulse Pressure 32. Pulse Rate 84.
 Blood Urea 148 mgm %.
 Blood Urea Clearance Cs = 4.71%.
- 12. 9.36 Supra-pubic catheter inserted.
- 15. 9.36 General condition very poor.

23. 9.36 Has slight sore throat. No pyrexia.

- 29. 9.36 Sharp attack of bronchitis. still afebrile.
- 2.10.36 B.P. 110/64. Pulse Pressure 46. Pulse Rate 80. Blood Urea 48 mgms %. Blood Urea Clearance = Cs 51.80%.
- 5.10.36 Has developed Herpes Zoster.
- 15.10.36 B.P. 130/84. Pulse Pressure 46. Pulse Rate 76. Blood Urea 39 mgms %. Blood Urea Clearance = C_B 63.65%.
 - 2.11.36 B.P. 160/94. Pulse Pressure 66. Pulse Rate 78. Blood Urea 39 mgms %. Blood Urea Clearance = Cs 58.09%.

4.11.36/

CASE NO. 11 (Contd.)

4.11.36 Prostatectomy Performed.

10.11.36 B.P. 158/90. Pulse Pressure 68. Pulse Rate 80. Blood Urea 39 mgms %. Blood Urea Clearance= C_s 38.35%.

Case 12/

CASE NO. 12.

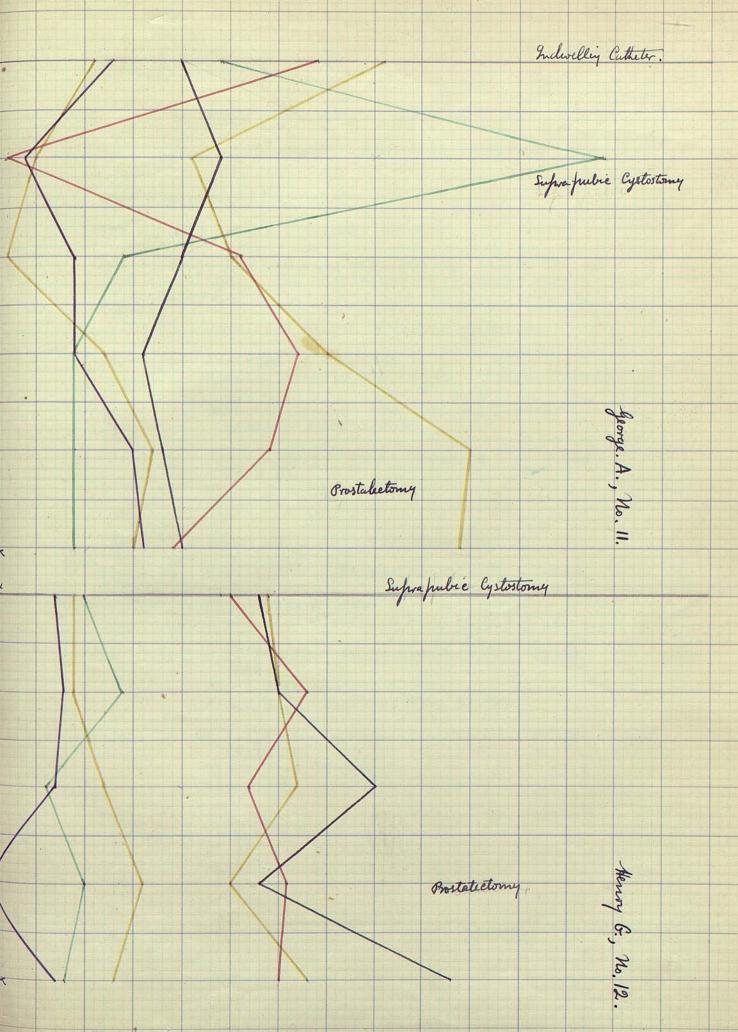
Name: HENRY G. Act. 68.

Disease: Prostatic Retention.

Case History.

193	L. P	atient had been admitted to another hospital with acute retention due to an enlarged prostate.
	A	supra-pubic cystostomy was performed and after some months the pt. was discharged as quite unfit for prostatectomy, with a permanent supra-pubic catheter. The catheter came out 3 weeks after discharge and pt. allowed the wound to close. Since then has had increasing frequency and difficulty in mictur.
1.	7.36	Difficulty became acute and urine dribbled through old wound.
2.	7.36	Admitted to hospital with urine constantly leaking through old supra pubic wound. Small supra-pubic catheter inserted.
13.	7.36	B.P. 118/78. Pulse Pressure 40. Pulse Rate 88. Blood Urea 40 mgm %. B.U.C. = C _B 50.87 %.
21.	7.36	 B.P. 120/78. Pulse Press. 42. Pulse Rate 90. Blood Urea 48 mgm %. B.U.C. 1st. hour = Cs 65.49%. B.U.C. 2nd hour = Cm 87.24%.
28.	7.36	 B.P. 124/84. Pulse Pressure 40. Pulse Rate 100. Blood Urea 32 mgm %. B.U.C. 1st. hour = Cm 116.64%. 2nd. hour = Cs 53.62%.
4.	8.36	88.
19.	8.36	Blood Urea 39 Mgm %. B.U.C. = Cs 61.57 %. Prostalectomy performed.
20.	8.36	Considerable haemorrhage from prostatic bed. General condition very poor.
7.	9.36	Patient improving steadily. Wound closing well.
28.	9.36	

2nd hour = C_m 76.2 %.



CASE NO. 13.

Name: CHARLES R. Act. 76.

Disease: Prostatic Retention.

Case History.

For 2 years has felt vaguely unwell.

For 6 mon mictur drowsin	
1.10.36	Developed almost complete retention.

- 3.10.36 Admitted to hospital.
 Bladder greatly distended. Tongue furred and dry.
 Catheter passed, tied in, and bladder greatly decompressed.
- 8.10.36 B.P. 172/75. Pulse Pressure 97. Pulse Rate 80.
 Blood Urea 71 mgms %.
 Blood Urea Clearance = C_p 49.65%.

10.10.36 Supra pubic catheter inserted.

- 13.10.36 B.P. 150/68. Pulse Pressure 82. Pulse Rate 64.
 Blood Urea 45 mgms %.
 Blood Urea Clearance - Cs 70.91%.
- 26.10.36 B.P. 164/72. Pulse Pressure 92. Pulse Rate 62. Blood Urea 45 mgms %. Blood Urea Clearance = Cs 67.89%.
- 4.11.36 B.P. 151/68. Pulse Pressure 83. Pulse Rate 61.
 Blood Urea 38 mgms %.
 Blood Urea Clearance 55.87%.
- 10.11.36 B.P. 155/72. Pulse Pressure 83. Pulse Rate 58. Blood Urea 35 mgms %. Blood Urea Clearance = C_B 52.05%.

CASE NO. 14.

Name: STEPHEN P. Act. 72.

Disease: Prostatic Obstruction.

Case History:

- For several years patient has had frequency of micturition which has been steadily increasing.
- 26. 4.36 Admitted to hospital with incomplete retention of urine. Bladder distended. Patient not acutely ill.
- 2. 5.36 Supra-pubic catherer inserted.
- 19. 5.36 Blood Urea 49 mgms %. B.U.C. = C_s 103.6%. Pulse rate 90. B.P. not taken.
- 29. 5.36 Blood Urea 41 mgm %. B.U.C. = C_s 76.82%. Pulse Rate 100. B.P. Not taken.
- 10. 6.36 Blood Urea 36 mgm %. B.U.C. = C_s 53.65%. Pulse Rate 90. B.P. not taken.
- 24. 6.36 Urine infected. Slight elevation of temp. and signs of pyelitis.
- 26. 6.36 Temp. subsided.
 B.P. 138/82. Pulse Pressure 56. Pulse Rate 76.
 Blood Urea 36 mgms %. B.U.C. = Cs 50.87%.
- 6. 7.36 B.P. 172/98. Pulse Pressure 74. Pulse Rate 74. Blood Urea 57 mgm %. B.U.C. = Cm 42.00%.
- 18. 7.36 Prostatectomy performed.
- 4. 8.36 Doing well. Wound closing in. B.P. 182/102. Pulse Pressure 80. Pulse Rate 72. Blood Urea. 40 mgms %. B.U.C. = Cs 61.18%.
- 9. 8.36 Has developed acute Orchitis. No pyrexia.
- 20. 8.36 Orchitis subsided.
- 27. 8.36 B.P. 145/84. Pulse Pressure 61. Pulse Rate 74. Blood Urea 42 mgms %. B.U.C. = C_s 55.31 %.

Indwelling Catheter Supra Jubie Cystostomy Chas. R., no. 13. Suprafubie Cystostomy R Prostaclectomy . itephen P. No 14

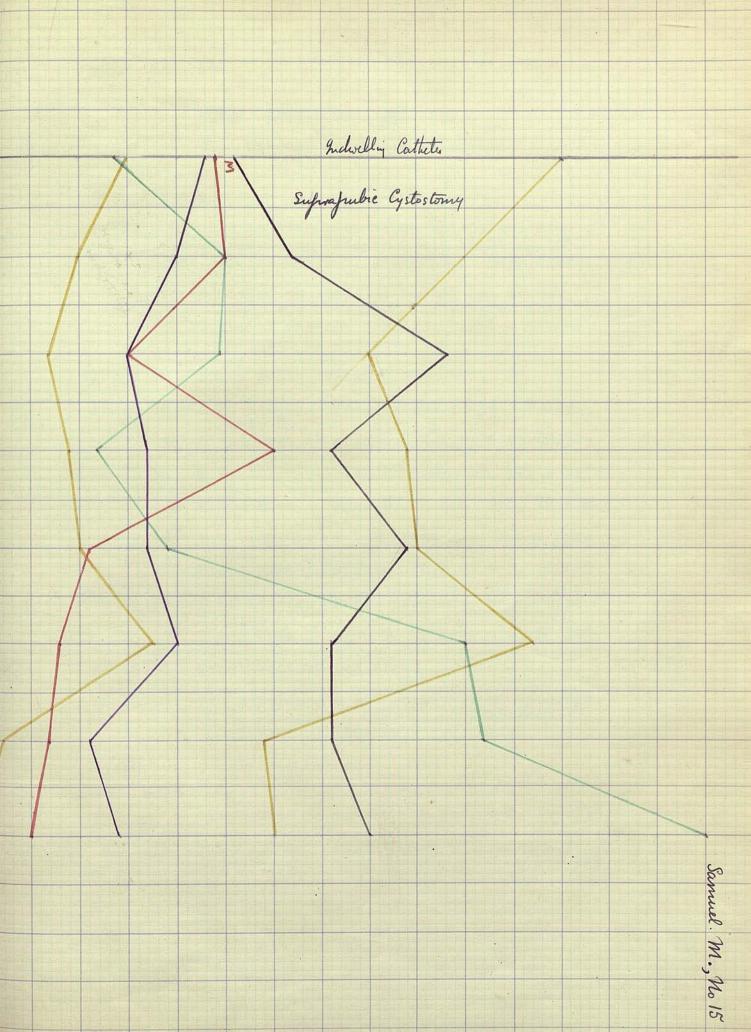
CASE NO. 15.

Name: SAMUEL M. Act. 62.

Disease: Prostatic retention.

Case History.

For 6 months, has had frequency of micturition. 5 months ago, had haematuria. For 1 month, has had dysuria as well as frequency. 7.8.36 Admitted with acute retention. General condition poor. Heart sounds poor quality, rapid with "tic-tac" rhythm. Catheter passed, tied in, bladder decompressed gradually. 11. 8.36 B.P. 180/90. Pulse Pressure 90. Pulse Rate 86. Blood Urea 47 mgms %. B.U.C. = Cm 48.10%. 15. 8.36 Supra pubic drainage established. 18. 8.36 B.P. 160/80. Pulse Pressure 80. Pulse Rate 92. Blood Urea 70 mgms %. B.U.C. = C_B 50.579%. B.P. 140/74. Pulse Pressure 66. Pulse 27. 8.36 Rate 108. Blood Urea 69 mgms %. B.U.C. = C. 30.61 %. B.P. 148/78. Pulse Press. 70. Pulse 3. 9.36 Rate 96. Blood Urea 44 mgms %. B.U.C. = C₈ 61.69%. B.P. 150/80. Pulse Press. 70. Pulse Rate 9. 9.36 104 Blood Urea 58 mgms %. B.U.C. = C_22.38%. B.P. 174/95. Pulse Press. 79. Pulse Rate 28. 9.36 96. Blood Urea 121 mgms %. B.U.C. = 0, 15.13%. 15.10.36 B.P. 118/64. Pulse Press.54. Pulse Rate 96. Blood Urea 125 mgms %. B.U.C. = Cg 14.28%. B.P. 120/58. Pulse Pressure 62, Pulse Rate 24.10.36 100. Blood Urea 170 mgms %. B.U.C. = Cs 11.02%.



CASE NO. 16.

Name: THOMAS L. Act. 59.

Disease. Prostatic Retention

Case History:

- 1932. Pt had an attack of acute retention, was catheterised "and went all right again". Has had frequency of micturition ever since.
- 10. 8.36 Admitted to hospital having had almost complete retention for several days. Catheter passed, tied in, bladder decompressed by degrees.
- 12. 8.36 Supra pubic catheter inserted.
- 18. 8.36 B.P. 148/92. Pulse Pressure 56. Pulse Rate 84. Blood Urea 43 mgms %. B.U.C. = C_s 82.325.
- 27. 8.36 B.P. 148/85, Pulse Pressure 63, Pulse Rate 8. Blood Urea 38 mgms %. B.U.C. = C. 79.18%.
- 3. 9.36 B.P. 140/98. Pulse Press 42. Pulse Rate 84.
 Blood Urea 36 mgms %. B.U.C. = C. 90.65%.
- 9.9.36 B.P. 134/76. Pulse Pressure, 58. Pulse Rate 88. Blood Urea 29 mgms %. B.U.C. = C₈ 63.82%.
- 16. 9.36 Prostatectomy performed.
- 12.10.36 Pt. developed a slight attack of orchitis. No Pyrexia. Says he has had such attacks at intervals for years.
- 23.10.36 Orchitis subsided. B.P. 140/86. Pulse Press. 54. Pulse Rate 84. Blood Urea 34 mgms %. B.U.C. = C_s 102.73%.
- 4.11.36 B.P. 132/82. Pulse Press 50. Pulse Rate 82. Blood Urea 31 mgms %. B.U.C. = Cg 116.21%.

CASE NO. 17.

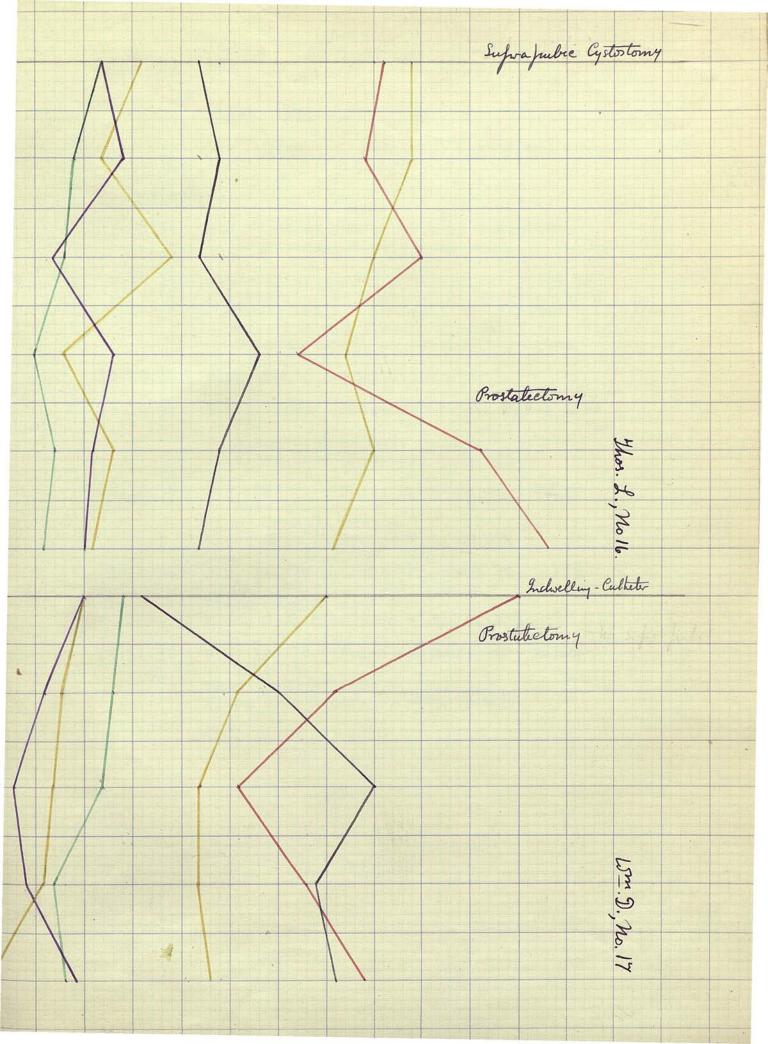
Name: WILLIAM D. Act. 57.

Disease: Prostatic retention.

Case History:

- Several months before admission patient had an attack of acute retention which passed off. Since then has had frequency of micturition.
- 29. 9.36 Sudden attack in the night of complete retention. Admitted to hospital. Catheter passed, tied in. bladder decompressed.
- 2.10.36 Patient's general condition particularly good.
 B.P. 130/80. Pulse Press. 50. Pulse Rate 76.
 Blood Urea 47 mgms %. B.U.C. = C. 110.87%.
- 7.10.36 Prostatectomy Performed.
- 8. 10.36 Considerable haemorrhage from prostatic bed.
- 15.10.36 B.P. 112/75. Pulse Pressure 37. Pulse Rate 90. Blood Urea 46 mgms %. B.U.C. = C. 72.66%.
- 26.10.36 B.P. 104/74. Pulse Pressure 30. Pulse Rate 100.
 Blood Urea 44 mgms %. B.U.C. = C_s 52.03%.
- 4.11.36 B.P. 104/72. Pulse Press. 32. Pulse Rate 94. Blood Urea 35 mgms %. B.U.C. = C. 66.06%.
- 10.11.36 B.P. 106/60. Pulse Pressure 46. Pulse Rate 96. Blood Urea 36 mgms %. B.U.C. = Cs 77.88%.

Case 18/



CASE NO. 18.

	Name: THOMAS McG. Act. 60.
	Disease: Prostatic Retention.
Case Histo	ory:
7. 8.36	Had first attack of urinary retention. Noprevious urinary symptoms.
6.10.36	Suddenly developed complete retention of urine. Was admitted to hospital some hours later. In-dwelling catheter inserted and bladder gradually relieved. General condition particularly good.
10.10.36	Supra-pubic catheter inserted.
13.10.36	B.P.112/60. Pulse Press 52. Pulse Rate 76. Blood Urea 28 mgms %. Blood Urea Clearance = C _s 135.6%.
26.10.36	B.P.122/74. Pulse Pressure 48. Pulse Rate 68. Blood Urea 34 mgms %. Blood Urea Clearance = C _s 80.47%.
4.11.36	B.P. 112/72. Pulse Pressure 40. Pulse Rate 92. Blood Urea 28 mgms %. Blood Urea Clearance = C ₈ 80.23%.
10.11.36	B.P. 118/78. Pulse Pressure 40. Pulse Rate 76. Blood Urea 30 mgms %. Blood Urea Clearance = C _s 75.29%.

Case 19/

CASE NO. 19.

Name:	JACK, M.	Aet:	72 yrs.
all some of the local section in the local section		Constitution of the local division of the lo	

Disease: Prostatic Retention.

Case History.

1934. Began to have difficulty in micturition.

1. 8.36 Developed acute retention.

7. 8.36 Admitted to hospital. Bladder very distended, having overflow incontinence. General condition fair. Irregularity of pulse. Catheter passed and fixed in position. Pressure on bladder relieved by degrees.

11. 8.36 B.P. 130/108. Pulse Pressure 22. Pulse
Rate 70.
Blood Urea, 44 mgms %.
Blood Urea Clearance = C₈ 92.70%.

15. 8.36 Supra-pubic cystostomy perperformed.

18. 8.36 Pulsus Trigeminus.

B.P. 130/80. Pulse Pressure 50. Pulse Rate 88.
Blood Urea 60 mgms %.
Blood Urea Clearance = C_s 55.5%.

25. 8.36 Extra-systole occurring every 12th beat.

27. 8.36 Electrocardiogram shows lt.dominance. B.P. 120/85. Pulse Pressure 35. Pulse Rate 79. Blood Urea 37 mgms %. Blood Urea Clearance = C_B 67.52%.

Case No. 20/

CASE NO. 20.

	Name: ERNEST F. Aet. 36
	Disease: Stone in Lt. Kidney with Hydronephrosis.
Case Histor	cy.
1932. Fi:	rst began to have attacks of Lt. renal colic. Pt. was at that time investigated and no stone found.
15. 6.36	Admitted with typical attack of renal colic
21. 6.36	Pyelography showed stone in Lt. kidney with Hydronephrosis.
29. 6.36	B.P. 152/88.
1. 7.36	Cystoscopy and Ureteral cutheters passed. Urine from rt. kidney contained 1.8 % Urea. Urine from Lt. kidney contained .8% Urea.
6. 7.36	B.P. 142/92. Pulse Pressure 50. Pulse Rate 82. Blood Urea 80 mgms %. Blood Urea.Clearance = 57.44%.
13. 7.36	B.P. 132/76. Pulse Pressure 56. Pulse Rate 82. Blood Urea 35 mgms %. Blood Urea Clearance = C _B 79.18%.
15. 7.36	Operation with removal of stone from Lt. Kidney.
	 B.P. 142/78. Pulse Pressure 64. Pulse Rate 80. Blood Urea. 36 mgms %. Blood Urea Clearance = Cm 118.37%. B.P. 130/86. Pulse Pressure 44. Pulse Rate 84. Blood Urea 35 mgms %. Blood Urea Clearance = Cm 56.16%.

