

A THESIS ON URAEMIA.

With a series of five cases successfully treated  
by Venesection combined with the Intravenous Injection  
of Normal Saline Solution at a Temperature of  
110° Fahrenheit.

by

JAMES FREDERICK SELKIRK, M.B., Ch.B. (Edin).



Uraemia is the name applied to a series of manifestations arising in the course of many renal diseases and due to the retention within the body, of excrementitious material, which should normally be eliminated by the kidneys.

The theories concerning the production of uraemia may, broadly speaking, be resolved into two :  
 (a) the mechanical theory, according to which nerve structures are excited or paralysed by the condition known as cerebral oedema or cerebral anaemia and  
 (b) the chemical theory, according to which the same results are brought about by some toxin or toxins circulating in the blood.

The mechanical theory, supported by Traube and others, is, that the watery condition of the blood occurring in Bright's disease predisposes to transudation of serum from the blood vessels; that the hypertrophy of the left ventricle, which occurs in Bright's disease, greatly increases the pressure in the arterial system, and that, from any cause still further increasing the tenuity of the blood serum, serous exudation takes place through the cerebral capillaries and the oedema thus produced compresses the capillaries of the cerebral vessels causing anaemia of the brain, convulsions, and coma.

This theory taken alone does not however to my mind explain satisfactorily the symptoms of uraemic/

uraemic poisoning as seen at the bed side.

The treatment of uraemia viz: venesection combined with the injection of a normal saline solution into a vein, which I propose to discuss later, would appear to be the worst form of treatment if this theory were correct, as the saline infusion combined with the venesection would still further increase the hydraemic condition of the blood and therefore, the oedema of the brain .

This treatment, on the contrary, has proved very beneficial in cases of uraemia.

There is no doubt that cerebral oedema is seen in many cases of fatal uraemia and that it may produce many of the symptoms, such as convulsions or Cheyne-Stokes breathing, but it is not a constant lesion.

This mechanical theory was first of all put forward to explain the localised uraemic convulsions; but a poison circulating in the blood may pick out only a portion of the nervous system, and lead and arsenic offer us examples of this fact.

I turn now to the chemical theory which is undoubtedly the more tenable, though unfortunately the exact nature of the toxic agent in the blood is not yet known.

Three theories have been put forward to account for the presence of this poison in the system:

(a)/

(a) the retention in the blood of a body or bodies which ought normally to be excreted:

(b) the abnormal decomposition in the blood and tissues of such a body or bodies:

(c) the products of tissue degeneration.

The first theory is the simplest explanation of uraemia and it was formerly supposed that urea was the offending substance, and there is no doubt that it has been found in considerable quantities in the blood up to thirty times the normal, or even more, whereas the normal is about .015 per cent, in acute uraemia the urea may be as much as .4 or .5 per cent.

The toxic effects of urea are now known to be less than was formerly supposed, and it can be injected into the veins of animals, if the kidneys are healthy, without any appreciable result, excepting an increase of urine and in the amount of urea.

Some have stated that the potash salts are the cause of the uraemia because they found that the potash salts increased in the blood of dogs after death following ligature of the ureters.

Other workers on the subject have not been able to discover any increase in the potash salts in the blood.

Certainly some of the symptoms of uraemic dogs closely/

closely simulate those of poisoning by potassium. In any case, however, potassium poisoning cannot be accepted as the only cause of the symptoms in uraemia.

Bouchard asserts that normal urine is itself toxic and that the toxic action is of a complex nature.

He states that he has found seven toxic substances in normal urine including a diuretic, (urea) a narcotic, an antipyretic, and a myotic.

If this is so it might explain the different symptoms of uraemia as due to difference in the amounts of the toxins which fail to be excreted. The difficulties in the way of explaining uraemia as dependent simply on the retention of some normal constituents of the urine are, in the first place, that in granular disease of the kidneys patients often develop alarming uraemic symptoms, even though they may be passing considerable quantities of urine containing, it is true, less urea than normal, but not necessarily less than many patients suffering from other diseases and taking less food, and secondly, in cases of suppression of urine, as by calculi in the ureters, the symptoms are not those characteristic of uraemia.

In view of these facts many observers have put forward the theory that these retained urinary constituents/

constituents have undergone decomposition in the blood or tissues, that the urea has been changed into ammonium carbonate, and that the symptoms of uraemia are due to the action of this salt.

Certainly in advanced kidney disease ammonia can frequently be detected in the breath and the injection of the salt into the vein of a dog causes convulsions; but it is doubtful whether ammonia is ever found in the blood, and, in fact, the alkalinity of the blood is even diminished in uraemia.

Finally, seeing the difficulties in the way of the retention and decomposition theories, it has been suggested that the symptoms of uraemia are due to the products of tissue degeneration; and it has been urged in favour of this that, in cases where the kidney substance is reduced in amount, the blood and tissues contain large quantities of urea and other nitrogenous extractives, that these must have arisen from tissue degeneration, for no retention of urine occurred as considerable quantities of it were being passed.

On examining these three theories. it appears to be reasonable to suppose that they may all, in a greater or less degree, lend support to the total cause in the production of uraemia, and the more so when we remember the extreme variety of the symptoms and the different/

different conditions under which it may be produced.

Uraemia may arise in congestion of the kidneys, in acute and in chronic nephritis, in granular kidney, in lardaceous, tuberculous, calculous and cystic disease of the kidney, in obstruction of the ureters, and in those cases of complete suppression sometimes seen after operation on the kidney or urinary tract.

Dr. Oliver, in the British Medical Journal of November 1899, relates a case which illustrates clearly the dangers of hydronephrosis when both kidneys are affected.

The patient had been suffering, for the last eight or nine months, from a tumour in the abdomen, which had increased rapidly in size during the last four or five weeks.

The tumour reached as high as the ensiform cartilage.

A catheter was passed and five ounces of urine were withdrawn, the tumour was then aspirated and ten pints of urine were removed.

The patient died six hours later in uraemic convulsions which had come on suddenly and for the first time.

At the post mortem examination the bladder was found to be very large and thickened, reaching above the/

the umbilicus, the ureters were dilated and there was hydronephrosis of both kidneys.

It has been mentioned above that uraemia occurs after operations on the kidney or urinary tract.

It may follow operations in cases where albumen is present in the urine. The following case will show the danger of operating under such conditions.

A patient aged 58 suffered from a large tumour which reached to three inches above the umbilicus.

The tumour was found to be an ovarian cyst.

Albumen was present in the urine, though not in any great quantity.

She was dieted and treated with suitable medicinal remedies until only a faint trace of albumen was present.

The operation was then performed and the tumour removed.

Afterwards the urine at once began to decrease and the albumen in it to increase. The patient became restless, sleepless and vomited, and death from uraemia occurred on the tenth day after the operation.

Only eight ounces of urine, loaded with albumen, had been passed during the last forty-eight hours.

At the post mortem examination there was no peritonitis/

peritonitis present but there was double nephritis.

In the nephritis secondary to valvular disease of the heart, and due to mechanical congestion,

Uraemia seldom declares itself; but, should pregnancy occur, the nephritis if present is at first acute and diffuse; then rapidly changes into the granular variety and uraemia is by no means uncommon.

It has been suggested that these symptoms may be due to some toxic agent, viz., the excrementitious result of foetal nutrition.

Dr. Swindell, in the Lancet of 1893, page 496, relates a case of uraemia complicating pregnancy.

The patient when five months pregnant, had a fit in the garden.

Her urine was found to be loaded with albumen. She recovered from this and pregnancy went on to full term.

Labour was normal up to the passage of the head over the perinaeum when she suddenly became comatose, with stertorous breathing and twitchings of the hands and arms. The pulse was full and of high tension.

Labour was quickly completed but for two days she remained unconscious; on the third day venesection was attempted but no blood flowed from the vein.

She/

She died the following day, her condition being unchanged.

I will now indicate the symptoms of uraemia.

They may be classified in two ways:

- (1) according to the nature of the most prominent symptoms, into the cerebral, dyspnoeic, and gastrointestinal varieties
- (2) according to the mode of onset, into the acute and chronic varieties.

The cerebral manifestations may be

(a) CONVULSIONS.

These simulate epilepsy, though the initial cry is not often present.

The fits are violent and frequent and in the interval between the fits the patient is in a state of coma.

They may come on suddenly and without any warning, or their onset may be heralded by headache and increasing restlessness.

Sometimes the convulsions are general and may be preceded by a tonic spasm lasting some seconds, or they may be localised and of the type known as Jacksonian Epilepsy.

The pupils are generally moderately dilated, and the knee jerks exaggerated. The temperature is usually subnormal but there may be hyperpyrexia.

Uraemic amaurosis occurs frequently just before the/

the seizure, in which no ophthalmoscopic changes can be detected.

(b) MANIA.

This is not common and is most frequently seen in the young. It comes on abruptly in a patient who may not have shown any previous indication of mental trouble and who may not be known to suffer from kidney disease.

The patient is excited and very violent but after a time quiets down, becomes drowsy and finally comatose.

(c) COMA.

Although unconsciousness always accompanies the convulsions, still, a coma may develop gradually or suddenly without any convulsive seizures.

It may be preceded by headache, cramps in the muscles and twitchings, nausea and vomiting.

Cases may be so insidious in their onset that the condition is not suspected until the urine has been examined.

(d) LOCAL PARALYSIS.

A hemiplegia, or even a monoplegia, may occur and no lesion in the brain may be found after death to account for it.

Other cerebral symptoms may be, intense headache, insomnia, and extreme restlessness, indistinct articulation and clipping of words.

A curious condition of uraemia may be mentioned here which is known as latent uraemia and is usually the result of obstructive suppression in both kidneys.

The symptoms are very slight, the patient may lie for a week or ten days without passing any urine, some headache or twitching of the limbs may be present, the pupils are contracted and the patient usually dies from respiratory failure with little or no mental disturbance.

#### DYSPNOEIC VARIETY.

This is usually very severe in type, the patient sitting up and gasping for breath.

The attacks, at first, usually come on at night, but afterwards may be present during the day time.

The breathing is hissing and noisy but there may not be any cyanosis.

A peculiar feature of this dyspnoea is that the respiratory rhythm may often assume the form known as Cheyne - Stokes breathing.

These attacks are not the same as the ordinary bronchial asthma, for the patient need never have shown any tendency to asthma and the lungs, on auscultation, reveal nothing abnormal, though some pulmonary oedema may be present in the later stages.

The dyspnoea is probably due to a constriction of the pulmonary vessels, which, cutting off to a certain/

certain extent the supply of blood to the lungs, thus produce intense dyspnoea, a condition analogous to that produced by a pulmonary embolus, and also to the dyspnoea produced by muscarine poisoning in which there is spasm of the pulmonary and other arteries up to death.

The following case, related by Dr. Elgood, in the British Medical Journal of September 1899, gives a good example of dyspnoea accompanied by Cheyne-Stokes respirations.

A plumber had been, for the last five weeks, attended for chronic nephritis. His arteries were thickened and tortuous, his heart was hypertrophied but with no valvular lesion. The urine contained a quantity of albumen. At this time Cheyne-Stokes breathing was noticed during sleep.

Each cycle had eleven respirations increasing up to the fourth and diminishing after the eighth.

On the following day he had great distress in breathing, the respirations were regular and their frequency not markedly increased.

He panted when he tried to speak and talked like a man who had been running a race. Air freely entered the lungs and he had no cyanosis. The apnoeic intervals of the Cheyne - Stokes respirations, which lasted about sixteen seconds, were his only respite, for the rhythm came on during the daytime as/

as well, and was present till death occurred.

His distress was great and he used to fling himself about in agony.

He died in uraemic convulsions.

THE GASTRO-INTESTINAL MANIFESTATIONS. are nausea intense and persistent vomiting, hiccough and sometimes diarrhoea. The vomiting may be so severe as to suggest the possibility of intestinal obstruction.

A special uraemic stomatitis has been described in which the lips, gums and tongue are swollen and red.

Various skin eruptions may be present, conditions brought about, it is supposed, by the saturation of the skin with uraemic products.

In the Lancet of November 1891, Dr. Lancaster speaks of eight different eruptions due to uraemia.

The special sense organs may be affected in uraemia, the eye being the most commonly affected.

We may have dimness of vision or complete blindness. A seizure is frequently accompanied by blindness which may remain for some time after the seizure.

A form of uraemic deafness has been described but it is very rare.

Some words may also be said about the classification of uraemia into the acute and chronic forms.

This/

This is best done by giving a few clinical pictures of cases illustrating these two conditions.

In the acute form the patient may have been going about in apparently good health when he is suddenly seized with violent convulsions and unconsciousness.

He afterwards remains unconscious.

On examination one finds his arteries are thickened and his pulse of high tension. His heart is hypertrophied and albumen is found in his urine.

From the symptoms and signs we diagnose the convulsions as due to uraemia.

Take another case. A child may be recovering from scarlet fever when it develops an attack of acute nephritis. After a few days the child suddenly becomes worse, the oedema increases and unconsciousness supervenes, followed quickly by a series of convulsions with intervals of coma.

Turning to the chronic form, a patient who is suffering from chronic nephritis is gradually noticed to be getting somewhat torpid. His speech is slow and hesitating, his tongue is furred, he has loss of appetite and occasional fits of vomiting.

As time goes on he gets more apathetic and drowsy, till, finally, he becomes unconscious altogether and has some twitchings of the muscles and, it may be, general convulsions.

DIAGNOSIS.

DIAGNOSIS.

Having regard to the great variety of symptoms of uraemia it is evident that the diagnosis, in some cases, is attended with difficulty.

The chief points which should be remembered are the condition of the heart and the arteries, and the condition of the urine.

Uraemia may be confounded with :-

(1) CEREBRAL LESIONS such as haemorrhage and meningitis, and in the chronic forms with cerebral tumour.

It is especially difficult when the uraemia takes on a paralytic type as may be seen from the following case which was admitted, into the General Infirmary, Chichester, under my care in February 1902. A.T., aged 68, was found by the police lying unconscious in the road.

He was brought to the Infirmary where he was found to be in a comatose condition. His pulse was 98 per minute and of high tension and his arteries were thickened and tortuous.

His breathing was laboured but there was no cyanosis. His pupils were equal and reacted to light and were moderately dilated. His heart was greatly hypertrophied and on auscultation a loud systolic murmur was heard at the apex which was propagated/

propagated into the axilla.

The lungs were healthy. He could move his left arm and leg but was unable to move at all the right side. The knee jerks were normal. The mouth was drawn to the left side.

A Catheter was passed and four ounces of urine were drawn off, which was acid in reaction and contained a large amount of albumen.

Two and a half hours after admission he had twitchings in the right arm and leg - the left side being unaffected. He remained comatose for two days, then consciousness gradually returned and he was now able for the first time to answer questions.

Under treatment he gradually improved and the albumen disappeared from the urine.

He was discharged in a month, apparently quite restored to health, the paralysis having entirely disappeared.

(2) ENTERIC FEVER, or GENERAL TUBERCULOSIS., may be confounded with uraemia where the uraemic patient lies in a torpid condition for many weeks, together with a furred tongue, rapid feeble pulse, and muscular twitchings.

(3) ALCOHOL or OPIUM POISONING may be confounded with uraemic coma.

In these conditions, besides the special signs of uraemia mentioned above, the state of the pupils may help in the diagnosis, these being contracted in opium and generally dilated in alcoholic/

alcoholic poisoning.

In uraemia they are not constant.

Albuminuric retinitis may be present in uraemia and the odour of the breath would also afford a clue.

(4) DIABETIC COMA might be confounded with uraemic coma.

The examination of the urine would, however, remove any doubt as to the cause of the coma.

#### PROGNOSIS.

The prognosis of uraemia is, as a rule grave.

Uraemia occurring in an attack of acute nephritis is not, however, so serious as an attack of uraemia occurring in chronic nephritis.

It may be taken as a broad rule that the acute attacks, which come on suddenly and violently, are more likely to recover than the chronic attacks, which come on slowly and insidiously, and, in which the patient gradually sinks into a torpid and finally into a comatose condition.

#### TREATMENT.

The main indications in the treatment of uraemia are as follows :-

- (1) To prevent as much as possible the further formation of the poisons.
- (2) To counteract the actions of the poisons.
- (3) To eliminate the poisons from the system.

In/

In order to hinder the further formation of the poisons the patient should be carefully dieted and, as long as the uraemic symptoms last, the nitrogenous elements in the diet should be eliminated.

Meat should be forbidden and it is best that the patient should be confined as much as possible to a purely milk diet, and afterwards go on to bread and butter, tea and coffee, fish and chicken, and fruits such as oranges and grapes.

After the attack is over the return to a meat diet should be gradual and, in any case, meat should not be taken more than once a day. Alcohol should be strictly forbidden.

These latter regulations apply more to cases of uraemia occurring in the course of chronic nephritis.

In cases following acute nephritis, where the kidneys have completely recovered, more latitude in diet afterwards would naturally be allowed.

The second indication in the treatment, viz., to counteract the poison, must be looked upon as purely a palliative method as opposed to a curative.

This treatment, in fact, may be summed up in these words that it is simply adding another poison to those already present.

But we must not lose sight of the fact that, although this method of counteracting the poison is unable actually to cure, still, the relief that it frequently gives enables the sufferer to gain strength for the time being and to resist the/

the action of the poisons within him, which otherwise might have proved fatal before the effects of the more curative remedies could have been felt.

As examples of these let us take chloroform, bromide of potash, chloral hydrate and morphia.

In violent uraemic convulsions the inhalation of chloroform generally proves of great value, stopping the convulsions for a time and thereby affording the patient relief from the intense strain to which he has been subjected.

In the same way chloral hydrate and bromide of potash may be used when there is much restlessness and insomnia.

The most important of the above mentioned drugs is, I think, morphia as this drug frequently acts when chloral hydrate has not the slightest effect.

It is of special use in those cases of uraemic asthma with intense dyspnoea, and it also gives great relief to the headache and restlessness.

One sixth of a grain of morphia hydrochloride may be injected hypodermically, or thirty to forty minims of liquor morphinae hydrochloride may be given by the mouth if the patient is able to swallow.

It acts chiefly by arresting muscular spasm, counteracting the effects of the uraemic poison.

Thus we see how it is especially valuable in cases of uraemic asthma, which, as previously mentioned, is/

is caused by the spasm of the pulmonary vessels.

Nitrite of amyl may be given in these cases as an inhalation, or one minim by the mouth, and often proves most useful, similarly nitroglycerine.

The third indication in the treatment, viz., to eliminate the poison from the system is the most important.

It may be done in a variety of ways.

Since the kidneys are unable to do their work properly it is necessary to help them, as much as possible, by utilizing the skin and the bowels for the elimination of the poisons circulating in the body, and thus purify the blood and reduce arterial tension.

For this purpose we use cathartic purgatives such as calomel, elaterium and jalap, and saline purgatives such as the sulphate of soda or magnesia.

We may use diuretics such as digitalis, diuretin, citrate of potash and theocin. This latter drug, which has come lately into use and which I have tried frequently, I have found of the greatest value where one wishes to establish diuresis.

It is a most powerful diuretic and frequently produces diuresis when all other drugs have failed.

We may stimulate the skin by giving diaphoretics such as the hot air bath, or the hypodermic injection of pilocarpin, though this last drug must not be given/

given indiscriminately in every case.

For instance, if a patient had much oedema of the lungs, it would be jeopardising the patient's life to give such a drug.

But all these measures would be naturally adopted in cases of ordinary nephritis uncomplicated by uraemia and therefore I do not propose to discuss them at any length but turn rather to a most valuable remedy which, in addition to those enumerated above, may be used when symptoms of uraemia appear on the scene and cause further complications.

This remedy which we possess is venesection combined with the injection into a vein of hot normal saline solution, and, since this is the most important agent which, in my opinion, we possess for the treatment of uraemia, I propose to discuss it at greater length, and, at the same time, illustrate my remarks by a series of five cases of uraemia which I have personally treated, with most favourable results, by the combined method of venesection and the injection of hot normal saline solution into a vein. I will now give briefly the history and treatment of the 5 cases of uraemia.

#### CASE I.

J.C., aged 40, was under my care, at the Worcester General/

General Infirmary, from July 10th 1903 to September 18th 1903.

For the last six weeks he had ~~had~~ attacks of coughing, with dyspnoea on exertion. Three days before admission his legs began to swell and he passed a very little dark coloured urine.

On admission, on July 10th, his condition was as follows :- Patient was a thin spare man with pale face, lips and conjunctivæ, <sup>and</sup> some puffiness about the lower eye-lids. His tongue was covered with a thick white fur and was dry to the touch.

There was considerable oedema of the legs and feet.

His pulse was 74 beats to the minute and very incompressible and the artery wall was thickened. The heart was not enlarged and there was no valvular mischief detected. His breathing was laboured but nothing abnormal was detected in his lungs.

The urine was acid in reaction, its specific gravity was 1024, and it contained a large quantity of albumen, and some blood and epithelial tube casts.

His temperature was 98.4°.

He was confined to bed and put on a strictly milk diet, and was prescribed ten grains of diuretin every four hours and one drachm of Magnesium Sulphate in hot water, every two hours, until the bowels acted. On July 14th his legs were noticed to be getting more swollen and oedematous and the patient/

patient complained of feeling sick. He was passing on an average sixteen ounces of urine in the twenty-four hours.

On July 15th he commenced to vomit, and he had passed only ten ounces of urine during the last twenty-four hours which contained blood and albumen.

He was given a hot air bath with a subcutaneous injection of one-sixth of a grain of pilocarpin: but without any good result.

On July 16th the vomiting became incessant. He complained of severe headache and became delirious and attempted to get out of bed.

On July 17th the patient had sunk into a condition of coma and had six convulsions, each lasting for about a minute and consisting, for the most part, of tonic spasms of the muscles of the whole body. His pupils were dilated and insensitive to light. His temperature was  $97.4^{\circ}$  and the pulse was 104 and the tension high.

His respirations were non-stertorous.

During the last twenty-four hours he had passed no urine at all.

In the evening of July 17th venesection of the right median basilic vein was performed and twenty ounces of dark coloured blood were withdrawn and immediately afterwards two and a half pints of normal saline solution, at a temperature of  $110^{\circ}\text{F.}$ , were injected/

injected into the right median basilic vein.

His breathing became, almost at once, quieter and his pulse was 110 and was more compressible than before the operation. His temperature, an hour after the operation was 99.2° F.

He commenced to perspire profusely three hours after the operation and passed some urine in bed involuntarily. He had no more convulsions and gradually became conscious, and, on the morning of July 18th, he was able to talk quite rationally. He was again put on ten grains of diuretin every four hours and three drachms of sulphate of magnesia in hot water every morning. He was given a milk diet, consisting of milk and milk puddings.

The amount of urine rapidly increased in quantity and the blood and albumen gradually disappeared. He was discharged on September 18th as cured, his urine being then quite free from blood and albumen and he was passing, on an average, 52 ounces per diem.

Here we have a case of uraemia following acute nephritis in which hot air baths and the injection of pilocarpin did not appear to give relief, but which showed a marked and rapid change for the better after venesection combined with the intravenous injection of saline solution had been performed, and the improvement was afterwards maintained.

CASE II.

A.E., aged 50, was under my care at the Worcester General Infirmary, from March 21st 1903 until April 7th 1903.

The patient was brought into Hospital at 11 p.m. on March 21st with the history that he had been seen to fall down in a fit in the street.

While he was being removed to hospital he had two more fits.

On admission he was quite unconscious, his breathing was stertorous and his pupils were equal and reacted sluggishly to light and were moderately dilated. His knee jerks were normal and there was no paralysis detected.

His temperature was 97° F.

His pulse was 89 and was of high tension and the artery wall was much thickened. The heart was greatly hypertrophied and the second sound in the aortic area was accentuated but no murmurs were present. The respiratory system was normal. The tongue was furred, and his breath had a faint urinous odour. There was no oedema present. A catheter was passed, with due aseptic precautions, and sixteen ounces of urine were drawn off. The urine was acid in reaction with a specific gravity of 1016 and gave on testing a thick cloud of albumen. There was no blood/

blood present or other abnormal constituent. He had two more fits after admission; each fit lasting about two minutes altogether in which all the muscles were first thrown into tonic contractions and then became clonic.

The case was diagnosed as one of uraemic convulsions and he was given two minims of croton oil, a hypodermic injection of one sixth of a grain of pilocarpin , and a hot air bath. After this he had three more fits and then remained unconscious for five hours. He was now very cyanosed.

In the morning of March 22nd, no improvement having taken place, venesection was performed and 24 ounces of blood were abstracted from the left median basilic vein and immediately afterwards three pints of normal saline solution, at a temperature of 110° F., were injected into the left median basilic vein.

His pulse shortly after the operation was 100 and the tension was less.

The temperature was now 100° F.

The patient rapidly became of a better colour and the breathing was less noisy. He commenced to perspire freely a few hours after the operation.

Eight hours after the venesection and saline injection he became conscious and told one of the nurses/

nurses he had been in hospital before for "kidney disease". In the evening of March 22nd he passed 30 ounces of highly albuminous urine and of a specific gravity of 1010.

He was given 4 grains of theocin every six hours and a saline purgative every morning and kept on a milk diet. The urine rapidly increased in quantity and on March 31st he was passing on an average 60 ounces of urine in the 24 hours.

He was gradually placed on a more liberal diet of fish and chicken and was allowed up on April 2nd. He was discharged at his own request on April 7th his urine still, however, containing some albumen.

Here we have an attack of uraemia occurring in a case of probably chronic interstitial nephritis.

Here also are seen the marked benefits which accrued from treatment by venesection and saline injection. In this case albuminuric retinitis was present.

### CASE III.

E.L. aged 23, married, nullipara, was admitted, into the Worcester General Infirmary, on April 30th 1903 with the following history.

On the morning of April 29th she complained of severe occipital headache and dimness of vision which continued during the whole day.

In/

In the evening she was suddenly seized with a convulsion which affected the muscles of the whole body. These fits recurring at frequent intervals a medical man was called in who gave her a hypodermic injection of morphia and an enema of chloral hydrate.

On admission into hospital the next morning, she was quite unconscious with general clonic convulsions occurring at frequent intervals.

Her face was puffy, and there was slight general oedema of the whole body. The breathing was laboured but there was no cyanosis present.

The pupils were equal and dilated and did not react to light. The knee jerks of both legs were exaggerated but there was no paralysis observed.

The pulse was 94 and the tension was increased, the artery wall was not thickened. The heart was slightly hypertrophied but otherwise was healthy.

The respiratory and alimentary systems were healthy. Her temperature was  $101.2^{\circ}$  F. On passing a catheter only two ounces of urine were withdrawn which was alkaline in reaction and almost went solid on boiling.

On examining the abdomen the uterus was felt reaching nearly to the umbilicus.

It was resolved to empty the uterus without delay and for this purpose chloroform was administered. The/

The os uteri having been dilated up with instruments a five months foetus was removed.

The convulsions ceased for six hours after this, but began again and she had fifteen fits in four hours, remaining comatose in the intervals between the fits.

Her pulse now was 106 and her temperature  $100^{\circ}$  F. She had passed no urine since her admission.

Venesection was now performed and fifteen ounces of blood were abstracted from the right median basilic vein and immediately afterwards three and a half pints of normal saline solution, at a temperature of  $110^{\circ}$  F. were injected intravenously. She had two more fits after this operation and then they ceased completely. An hour after the operation her pulse was 96 and the tension was still increased, and her temperature was  $101^{\circ}.4$  F. She was given a hot air bath and she commenced to perspire profusely five hours after the operation. The patient slowly regained consciousness and on the following day, May 1st, she could answer "yes" or "no" to questions put to her. In the afternoon of this day she passed nine ounces of highly albuminous urine.

Her temperature became normal on May 2nd and remained so.

She was placed on light diet and given a mixture/

mixture of citrate, bicarbonate and acetate of potash every four hours.

Her urine rapidly increased in quantity and on May 9th she was passing on an average 52 ounces of urine per day.

The albumen rapidly disappeared from the urine and when discharged on May 29th no albumen could be detected.

#### CASE IV.

M.H. aged 21 was admitted into the Worcester General Infirmary, on January 9th 1904 suffering from severe and frequent convulsions.

The day before admission she felt quite well until the evening when she complained of a throbbing sensation in the head, she had no headache or sickness. On going to bed that night she suddenly became blind and immediately afterwards fell down in a convulsion. During the night she had twelve similar convulsions.

On admission she was comatose with stertorous breathing. Her legs, feet and eye-lids were oedematous and the tongue was furred and dry to the touch. The pupils were equal, dilated and did not react to light. Her pulse was 99 and very incompressible, the artery wall was not thickened.

The heart was slightly enlarged but there was no valvular disease. Nothing abnormal was detected in/

in the lungs. There was no paralysis present. Her temperature was 99.6° F. On palpating the abdomen the uterus was felt reaching up to the umbilicus and foetal heart sounds were detected on auscultation. On passing a catheter only about one ounce of urine was obtained which on testing proved to be loaded with albumen.

She was put to bed and a hot air bath along with a hypodermic injection of one sixth of a grain of pilocarpin administered.

The fits increased in severity and frequency, each fit consisting of a tonic stage in which the whole body became rigid and respirations were arrested. This was followed by a clonic stage which commenced with twitchings of the muscles of the face and spreading quickly to the rest of the muscles of the body. Her face was cyanosed and foam issued from the mouth during the fits. She had eighteen fits in four hours.

Five hours after admission, chloroform was administered and the uterus emptied of a six months foetus.

She had no more fits for one and a half hours after the operation, when they commenced again and she had ten fits in two hours.

Venesection was now performed and twenty ounces of blood were withdrawn from the left median basilic vein/

vein and  $3\frac{1}{2}$  pints of normal saline solution at a temperature of  $110^{\circ}$  F. were immediately afterwards injected intravenously.

Her pulse after the operation was 110 and the tension was considerably reduced.

Her temperature was  $102^{\circ}$  F.

Three hours after the operation she commenced to perspire freely and although having no more fits she remained deeply comatose. It was decided to inject more saline solution and two pints at a temperature of  $110^{\circ}$  F. were injected, with due aseptic precautions, subcutaneously in the region of the left mamma.

Consciousness gradually returned and in the afternoon of January 10th she was able to answer "yes" or "no" to questions put to her, but still seemed dazed and confused. On January 11th she had completely regained consciousness and passed urine for the first time since her admission. The progress of the case was uneventful, the urine steadily increasing in quantity and the albumen in it diminishing.

She was discharged at her own request on January 27th feeling quite well, the urine however still containing a faint trace of albumen.

We have here two cases of puerperal eclampsia or uraemia - the latter being sudden in onset - in which/

which morphia and chloral hydrate in the first case had been given, and in the second case hot air baths and pilocarpin, without any beneficial result, and the convulsions still persisting after the uterus had been emptied and marked improvement setting in after venesection and the intravenous injection of normal saline solution.

I would call attention to the recovery of these two patients, both of whom had had over thirty fits.

Fothergill states that when a case of puerperal eclampsia has had over twenty convulsions the chances of saving the patient are very small.

#### CASE V.

T.B. aged 32, first noticed that his eyes were puffy on the morning of February 29th 1904. He came up to the York County Hospital the same day and was admitted.

He was a well nourished man with a pale and puffy appearance of his face. His lower eye-lids were slightly oedematous and there was slight oedema of the ankles.

His tongue was dry and furred otherwise the alimentary system was healthy.

His heart was considerably hypertrophied and there was a systolic murmur heard in the mitral area which was propagated into the axilla. His pulse was/

was 75 and very incompressible, and the artery wall was thickened. On examining his lungs there was slight dulness at both bases and numerous fine crackling rales were heard on auscultation. On examining the urine it was found to be acid in reaction, specific gravity was 1012, and contained a considerable quantity of albumen and a few granular tube casts.

His pupils were equal and reacted to light and accomodation. On examining his eyes with the ophthalmoscope albuminuric retinitis was well marked.

He was put on a strictly milk diet and given a saline purgative every morning, and five grains of theocin three times a day. He was passing on an average about 40 ounces of urine per diem, which contained much albumen and a few granular tube casts.

On March 4th he was suddenly seized with a clonic convulsion which lasted about two minutes and he had conjugate deviation of both eyes to the left.

He became comatose after the convulsion and had nine more fits in six hours. He was given a hot air bath; but the skin remained very dry and the convulsions became more frequent.

His temperature was  $97.4^{\circ}$  F and his pulse was 85.

In the evening of March 4th venesection was performed and nineteen ounces of dark coloured blood/

blood were abstracted and three pints of normal saline solution, at a temperature of  $110^{\circ}$  F, were injected into the right median basilic vein. About three hours after the operation the skin reacted and he sweated freely. His pulse now was 101 and the tension was slightly lower than before the operation. The temperature was  $98.4^{\circ}$  F. He had no more fits and gradually became conscious and on the evening of March 5th he was quite conscious.

He was now given five grains of theocin every six hours and kept on a milk diet.

The oedema of the eye-lids and ankles disappeared. On March 7th he was passing, on an average, 80 ounces of urine per diem.

He steadily improved and was discharged four weeks after admission with still, however, albumen present in the urine.

Here we have another case of uraemia arising in a case of probably granular kidney disease, and coming on suddenly and without warning.

Pilocarpin was not administered to this patient on account of the oedema of the bases of both lungs as it was considered that the oedema would be greatly increased, and the patient's life put in danger if it had been given.

Since, in uraemia, there is always the question of/

of a toxic agent being present in the blood, it is manifestly reasonable to suppose, that by withdrawing a portion of the blood the amount of toxin would be reduced and the blood would be rendered more watery, and still more so when combined with the injection of saline solution into the blood stream which would render the blood still more hydraemic and dilute the toxins still further.

The injection of the saline solution further increases these beneficial conditions and counteracts the bad effects which might follow the loss of a quantity of blood.

At the same time the hot solution stimulates the patient generally, and excites in particular the action of the skin and kidneys.

A brief description of the operation will not be out of place.

The saline solution is first prepared by adding one drachm of common salt to a pint of sterilized water at a temperature of 110° F.

The skin, in the region of the median basilic vein, having been carefully prepared, an incision, about one inch in length, is made over the vein. When the vein has been exposed a small oblique incision is made into it and the required amount of blood withdrawn.

The canula of the transfusion apparatus is now inserted/

inserted through the opening and tied into the vein.

Great care must be taken here to prevent any air being injected into the blood stream, and this is best done by allowing the saline solution to flow through the canula while it is being inserted.

The saline solution is now allowed to run slowly into the vein and the effects on the patient carefully observed.

The required amount of saline solution having been injected, two ligatures are passed beneath the vein - one above the opening in the vein and one below it.

The lowermost ligature is now tied and the canula removed and then the upper ligature is tied.

The skin incision is sutured with some silk-worm catgut and a pad of aseptic gauze bandaged over the wound.

It has been stated that alarming and even fatal symptoms may suddenly arise whilst injecting saline solution intravenously.

I have never experienced any such symptoms and, I think, that, if due care be taken, to prevent air being thrown into the blood stream, and to watch carefully the effects on the circulatory system, the operation is practically without danger.

I have frequently used the intravenous injection of /

of normal saline solution, at a temperature of  $110^{\circ}$  F, in cases of shock and collapse after operation or severe injury, and in cases suffering from the effects of severe hæmorrhage.

In such cases the effects are often marvellous, the whole condition of the patient being markedly improved.

I would suggest that the intravenous injection of normal saline solution, at a temperature of  $110^{\circ}$  F., might be tried with benefit in other toxaemic conditions such as, diabetic coma, sapsraemia, opium poisoning etc.

Dr. Oliver, of Newcastle, recounts a case of diabetic coma treated successfully by intravenous injections of normal saline solution.

In private practice, where one could not get assistance, I should recommend the trial of hot saline enemata at a temperature of  $110^{\circ}$  F., retaining them, if necessary, by pressure with a towel over the anus.

Another simple method, for injecting the saline solution subcutaneously, has recently been suggested in the "Lancet" viz., attaching a large exploring needle to an ordinary Higginson's syringe and gently pumping the fluid under the skin by this means.

This method would prove of great utility in private practice also, as it is very simple, requires no/

no assistance, and the apparatus is always at hand.

I do not recommend that venesection combined with the intravenous injection of saline solution should be performed in every case of uraemia; but where the case is seen early, and the pulse is good and the tension increased, I would strongly recommend it.

In using this treatment I would lay stress on the fact that time should not be wasted in treating the symptoms with morphia, chloral hydrate, pilocarpin, etc.

If the symptoms did not improve rapidly under treatment with drugs one should at once proceed to perform venesection combined with the intravenous injection of normal saline solution at a temperature of not less than 110° F.

Reviewing this method in the treatment of uraemia we can judge by these cases that it is of the greatest value and should be employed without delay.

If this rational treatment - treatment which aims at removing the cause of uraemia or eclampsia - was more generally adopted, instead of time being wasted in treating the symptoms with sedatives, many valuable lives would be saved.