ANURIA IN ACUTE NEPHRITIS

BY

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New modes of treatment have stimulated interest in the management of patients suffering from acute renal failure. Although uraemia is most often a terminal event resulting from permanent damage to the kidneys, the occurrence of a variety of syndromes associated with acute reversible renal failure establishes the need for an effective method of treatment to sustain life until natural renal function returns. The techniques of peritoneal and intestinal dialysis, exsanguino-transfusion, special dietary regimes, and the artificial kidney have been developed for this purpose. Each method has its special advantages and peculiar dangers. These have not yet been clearly defined. Therefore, whenever these new methods are used detailed biochemical and clinical observations should be made. These contribute not only to the management of the individual case, but will also help to establish the specific indications for each technique, together with its difficulties and hazards.

This paper discusses the significance and treatment of prolonged oliguria occurring in the course of acute nephritis. Two cases are described in detail. Both patients survived, one after peritoneal dialysis and the other with dietary management alone. Full metabolic studies were made, and these provide useful data for a wider discussion of the underlying biochemical patterns of disturbance arising in renal failure, and of the methods by which these may be corrected.

Oliguria in Acute Glomerular Nephritis

Serious oliguria may be said to occur when the daily volume of urine is less than the minimum required for the excretion of the end-products of metabolism. This volume will vary with the rate and character of the metabolism and with the concentrating power of the kidney. Normal subjects on an average diet require a minimum daily urinary volume of 500 ml., but when the kidneys are damaged a larger volume is needed. In this paper a daily urinary volume of 500 ml. or less is taken as a definition of serious oliguria. Earlier reports adopt various arbitrary definitions, so that only a few lend themselves to comparative analysis. A general review of the literature (MacLean, 1919; Richter, 1935-6; Ellis, 1942; Rudebeck, 1946) indicates that in 691 cases of acute nephritis in adults anuria or oliguria lasting more than 48 hours occurred in 35 patients and of these 18 died.

Death in acute glomerular nephritis is often due to disturbances of water balance—namely, pulmonary oedema, cardiac failure, or convulsions associated with cerebral oedema. These changes are sometimes precipitated by the excessive administration of fluids, by mouth or intravenously, in an attempt to force a diuresis. Less frequently a fatal issue is associated with an accumulation of potassium in the extracellular fluid, or with other less well defined causes. Treatment

should therefore be directed towards maintaining the normal composition and volume of the cellular and extracellular fluids. This describes the normal renal function of homoeostasis.

In the temporary absence of renal function, this process may be imitated by the techniques of peritoneal and intestinal dialysis or by the use of the artificial kidney. Such methods are primarily connected with the control of the extracellular fluid and have only an indirect influence on the composition of the cells. This treatment is specially indicated when serious changes have already occurred in the composition and volume of the extracellular fluid, whether arising in the natural course of events or provoked by unsuitable therapy.

A more fundamental procedure is designed to influence cellular metabolism, and thereby to prevent the excessive catabolism of protoplasm which precedes the liberation of potassium, phosphate, and products of protein breakdown into the extracellular fluid. This control is obtained by the use of a suitable diet and by the prevention of serious disturbances of hydration or of acid-base balance. A high calorie diet, rich in carbohydrate, reduces the metabolism of cellular protein. Borst (1948) applied this principle by feeding uraemic patients with a diet of butter and sugar, but this mixture is often nauseating. Bull *et al.* (1949) have modified this idea by the use of an oil, water, and sugar emulsion given by gastric tube. This method of treatment was used in the second patient. Detailed consideration of the water and electrolyte content of the diet and of hormonal and other supplementary measures in this metabolic control will be discussed in the interpretation of the biochemical data.

Case 1. Treatment with Peritoneal Dialysis

A married woman aged 45 developed backache, oliguria, and oedema of the face eight days after a severe attack of tonsillitis, which had been treated with 19 g. of sulphadimidine administered over five days. Red cells, casts, and albumin were found in the urine. After six days of oliguria complete suppression of urine occurred, yet a free intake of fluid was allowed. Three days later the patient was admitted to hospital with nausea, vomiting, anuria, facial and generalized oedema, and ascites. The picture was not that of a sulphonamide nephrosis, and a diagnosis of acute nephritis was made. In view of the possibility that the anuria was due to obstruction by sulphadimidine crystals, ureteric catheterization and bilateral splanchnic block were performed. Both procedures were without effect and no urine was obtained.

After five days' complete anuria the patient's condition was deteriorating and pulmonary oedema gave rise to anxiety. The blood urea had risen to 230 mg. per 100 ml., and the serum potassium, at 7.5 mEq per litre, was rising towards a dangerous level. Peritoneal dialysis was undertaken, using a hypertonic dialysing fluid, low in sodium but balanced with regard to the other main extracellular electrolytes and slightly alkaline in reaction:

Sodium ..	120 mEq per litre	Bicarbonate	26 mEq per litre
Potassium ..	5 " "		
Calcium ..	4 " "	Glucose ..	20 g. per litre
Chloride ..	103 " "	pH ..	8.4

The main technique of peritoneal dialysis was that described by Frank *et al.* (1946). Heparin, 10 mg. per litre, was added to the fluid to prevent obstruction of the tubes by fibrin clot; also penicillin, 10,000 units per litre, to aid bacteriostasis. Streptomycin, 0.3 g. thrice daily, was given intramuscularly. The position of the intraperitoneal tubes was checked by means of radiographs. The composition and rate of flow of the dialysing fluid were adjusted according to the biochemical response as judged by frequent

electrolyte determinations on the plasma and on the fluid removed from the peritoneal cavity.

The dialysis was continued for four days, the patient's general condition improved, and the secretion of urine restarted (Fig. 1). During this period no further significant rise occurred in the levels of the plasma potassium and blood urea, and the alkali reserve was maintained at normal levels. Twenty-five grammes of urea was removed, which if allowed to accumulate would have caused the blood urea to rise from 230 to 330 mg. per 100 ml. The most significant result was the correction of the gross overhydration. By means of overall balances it was found that 597 mEq of chloride was removed from the body. By the use of chloride as a reference ion for changes in the volume of the extracellular compartment it may be computed that the extracellular fluid was reduced by 5.7 litres. The

litis which had been treated with sulphanilamide. After four days of oliguria complete suppression of urine occurred and she was admitted to hospital. Only 4 ml. of urine could be obtained on catheterization, and this contained albumin, casts, and red cells. The blood urea was 57 mg. per 100 ml., plasma potassium 4.7 mEq per litre, and the plasma inorganic phosphorus 3.8 mg. per 100 ml. The face and legs were moderately oedematous, but there was no clinical evidence of ascites or of pulmonary oedema. The indications for treatment were therefore different from those in Case 1. In the absence of dangerous overhydration or of serious biochemical disturbance treatment was primarily designed to regulate body metabolism and thereby maintain the normal chemical composition of the cellular and extracellular fluids, rather than to imitate the excretory function of the kidneys.

FIG. 1.—Effect of peritoneal dialysis in Case 1.

removal of this large volume of salt and water relieved the pulmonary oedema. This was an important factor in improving the patient's general condition and allowed the transfusion of 1,100 ml. of blood to correct a serious pre-existing anaemia.

The main technical difficulties experienced were in balancing the rates of inflow and outflow of fluid. A total of 45 litres of dialysing fluid was used, and when volumes of that magnitude are infused into an oedematous patient fluid balance is clearly of vital importance. Initially the concentration of urea in the peritoneal outflow was 222 mg. per 100 ml., but this level fell progressively, presumably as a channel formed between the two tubes. The multiplicity of the chemical estimations essential for accurate control and the overall complexity of the techniques of peritoneal dialysis required the constant attention of a specialized team of medical and nursing staff. The patient was troubled by increasing abdominal distension due to a mild ileus. Discomfort arising from this cause and the danger of serious ileus or peritonitis are obvious limiting factors in treatment by continuous dialysis. The new method of rapid intermittent peritoneal dialysis introduced by Dérot *et al.* (1950) is likely to avoid these complications.

Case 2. Treatment by Dietary Regime

A single woman aged 45 developed oliguria and generalized oedema eight days after the onset of a severe tonsil-

FIG. 2.—Results of treatment with special diet in Case 2. The gastric drip was given from the third to the tenth day.

An emulsion containing 400 g. of glucose and 100 g. of peanut oil, made up to 1 litre with distilled water (Bull *et al.*, 1949), was given by intragastric drip over each period of 24 hours. Vomiting occurred on one occasion only, and the vomitus was filtered and returned through the intragastric tube. The treatment was continued for eight days, during which time the patient's condition remained very satisfactory. As the urine flow was re-established an additional volume of distilled water equal to the previous day's urinary output was added to the mixture. Fat absorption during the period of intragastric drip therapy was found to be 96%. Despite complete anuria for two days and extreme oliguria for seven days there was no disturbance of the acid-base balance, the plasma potassium rose only to 6.5 mEq per litre, the plasma inorganic phosphorus to 8 mg. per 100 ml., and the blood urea to 150 mg. per 100 ml. (Fig. 2). At no time was there any evidence of pulmonary oedema, neither was there any suggestion of dehydration.

Discussion

When renal homoeostasis ceases two principal factors remain to influence the composition of the body fluids—namely, cellular metabolism and the total intake of food, water, and electrolytes. In renal failure, in the absence of effective treatment, there is an insidious development of changes in the distribution of body

water and electrolytes, progressive acidosis, and the accumulation in the extracellular fluid of potassium phosphate and urea derived from the catabolism of cellular and exogenous proteins. If treatment is begun before these changes in the composition and volume of the body fluids have occurred, it is possible to delay their onset by a process of metabolic control. In the presence of serious biochemical disturbances, methods of imitating renal function should be considered in addition to the dietary treatment. Clearly the metabolic disorders resulting from anuria are complex and closely interrelated. To simplify the discussion they have been artificially separated into extracellular and cellular disturbances.

Changes in Water and Sodium Chloride Equilibrium

Death in acute renal failure is most often associated with overhydration and pulmonary oedema. This is sometimes precipitated by an excessive intake of fluids by mouth or by the administration of saline or sodium sulphate solutions intravenously in an attempt to force a diuresis. There is no convincing evidence that this type of therapy is effective. Water and salt administered in excess of the minimal requirements to replace the daily losses by the skin, lungs, and bowel will remain in the body and add to the existing biochemical disturbances.

In a temperate climate the volume of water required to replace normal extrarenal losses is approximately 1,000 ml. for a 70-kg. patient. Under these conditions the loss of salt by sweating and in the stool seldom exceeds 20 mM a day, and is so small relative to the total body content of sodium chloride that no replacement is required over short periods of treatment. For these reasons the oil, water, and sugar emulsion given to Case 2 is made up in 1 litre of distilled water. When losses of water and electrolytes occurred as urine output was restored, equivalent replacements were added to the intragastric drip. In this patient there was no suggestion of the inability to reabsorb sodium chloride in the renal tubules that has been found in patients recovering from a lower nephron syndrome (Humphrey and Avery Jones, 1947; Strauss, 1948). Vomiting during the period of treatment may lead to loss of water and salt, but this can be most accurately corrected by returning the filtered vomitus through the intragastric tube. The second patient was maintained in this way for eight days; clinical observation, weight change, and chloride balance (as a reference ion for extracellular fluid) gave no indication of disturbance in the distribution of body water and electrolytes.

When additional losses of water and electrolytes have arisen from excessive sweating, or by diarrhoea and vomiting before treatment has begun, these deficits are restored with caution. Dehydration hastens the uraemic syndrome by accelerating cellular catabolism (Black *et al.*, 1944), and may also prolong anuria by reducing plasma volume and renal blood flow. This extrarenal factor needs to be assessed in each patient, whether or not a primary renal lesion is the main cause of the renal failure. Serial determinations of the haematocrit and plasma electrolyte levels are almost essential for the controlled correction of dehydration in the absence of renal function. Peripheral circulatory failure calls for the administration of plasma or whole blood.

Serious overhydration cannot be corrected by dietary treatment alone. Severe water and salt retention is

therefore an indication for combining the dietary regime with the use of the artificial kidney or with peritoneal dialysis. This is illustrated by reference to the patient in Case 1 who was suffering from pulmonary oedema and ascites before treatment was undertaken. By the use of a dialysing fluid low in salt content (120 mEq per litre) yet made hypertonic with glucose, it was possible to remove 5.7 litres of excess extracellular fluid, with the relief of the dangerous pulmonary oedema. A similar correction may be made by the use of hypertonic dialysing fluids in the artificial kidney.

Ion exchange resins in a form suitable for ingestion by mouth are now available in this country. They have already been used with some success for the removal of sodium from the body in the treatment of congestive cardiac failure, hypertension, and nephrosis (Dock and Frank, 1950), and may prove valuable in the treatment of sodium and potassium retention in patients with renal failure.

Cellular Factors

In health there is a dynamic equilibrium in metabolism. Fever, starvation, acidosis, dehydration, and the excessive secretion of adrenal steroids are amongst the factors known to upset the balance by promoting excessive cellular catabolism. When this occurs, potassium and phosphates are transferred from the cells into the extracellular fluid, and with normal renal function are excreted in the urine. In renal failure these ions accumulate in the extracellular fluid together with the end-products of nitrogen metabolism. This leads to a progressive rise in the levels of the blood urea and of the plasma potassium and inorganic phosphorus. A high level of potassium in the plasma is associated with disturbances in the conduction of the cardiac impulse, and may cause heart-block or cardiac arrest (Tarail, 1948). It is not certain that a high blood urea is of itself dangerous. However, the blood urea is a useful guide to the rate of cellular breakdown.

It follows that treatment should in the first instance be directed towards the prevention of excessive cellular catabolism.

When gross disturbances of metabolism have already occurred the use of the artificial kidney, or of similar methods of imitating renal function, is indicated.

The effect of starvation on cellular catabolism is shown in the accompanying table, which compares the rate of increase of the blood urea, plasma potassium, and inorganic phosphorus in two patients with anuria. One was treated with the oil, sugar, and water emulsion (Case 2), while in the other, suffering from chronic nephritis, the intake of food was very small. The rate of increase of the plasma potassium and phosphorus, and of the blood urea, was much slower in the patient who received the high calorie emulsion.

Daily Rise in Levels of Blood Urea, Plasma Potassium, and Phosphorus in Two Patients with Anuria

Treatment	Blood Urea	Plasma Potassium	Plasma Phosphorus
Starvation	42 mg./100 ml.	0.45 mEq/litre	1.25 mg./100 ml.
Oil-sugar emulsion	10 mg./100 ml.	0.16 mEq/litre	0.40 mg./100 ml.

All the calories in the diet are derived from carbohydrate and fat. The addition of protein would necessarily raise the potassium intake, increase the total nitrogen metabolism and the tendency towards acidosis. However, Krauss's (1926) observations suggest that

small amounts of protein added to a high-calorie diet consisting chiefly of fat and carbohydrate may reduce endogenous nitrogen metabolism.

Serious depletion of protein may occur by the loss of albumin in the urine when renal function returns. The second patient was losing 24 g. of protein a day by this route, and as a result in seven days the plasma albumin fell from 3.8 to 2.4 g. per 100 ml. At this stage, provided that the blood urea is falling, the protein intake should be increased.

Failure of renal excretion leads to acidosis from retention of the normal end-products of protein metabolism. Acidosis increases the breakdown of cells and accelerates the transfer of potassium and phosphate into the extracellular fluid. The effect of the protein-free high-calorie emulsion in preventing the development of acidosis is demonstrated in the second patient. During eight days' oliguria there was no significant change in the level of the plasma bicarbonate.

The administration of this diet cannot correct an existing acidosis. In complete renal failure the addition of sodium lactate or bicarbonate in amounts calculated to replace the total deficit of base in the extracellular fluid does little to restore the alkali reserve. Part of the sodium is transferred into the cells, a process which is known to occur in acidosis (Mudge and Vislocky, 1949). When larger amounts of sodium are given, pulmonary oedema is often produced. It therefore appears that serious acidosis in a patient with anuria is an indication for the use of the artificial kidney or of peritoneal dialysis.

The association of a high plasma potassium with disturbances of the conduction of the cardiac impulse is now widely recognized (Tarail, 1948). The most frequent cause of death in nephrectomized animals is the high plasma potassium, leading to ventricular tachycardia and finally to cardiac arrest (Hoff *et al.*, 1941). In man it is not certain that hyperkalaemia is in fact a common cause of death in uraemia, but the few reported cases suggest that the fatal level is about 10 mEq per litre. Present evidence indicates that the potassium intake of patients with anuria should be reduced to a minimum. The emulsion administered to the second patient contained less than 1 mEq per litre. Fruit juices and meat extracts are particularly rich in potassium and should be avoided. In the past potassium salts have often been prescribed in diuretic mixtures. It now seems clear that they should not be given to patients in renal failure.

The inhibitory effect of a high-calorie diet on cellular catabolism and transfer of potassium from the cells has already been discussed. A second important factor in preventing a rise of the plasma potassium is the movement of this ion back into the cells when glycogen is formed in the liver and muscles. Glycogenesis is promoted by the administration of glucose, particularly if insulin injections are given. The metabolic data obtained on the second patient provide evidence that the high glucose content of the emulsion did promote the transfer of potassium into the cells. The rise of plasma potassium was 2 mEq per litre less than would have been expected if cellular components had been catabolized in the normal K:N ratio (Reifenstein *et al.*, 1945), and if no glycogenesis had occurred.

Both peritoneal dialysis and the artificial kidney (Merrill *et al.*, 1950) provide effective means for the removal of excess potassium from the extracellular

fluid. One of these techniques should be used in addition to dietetic control when the plasma potassium is approaching dangerous levels.

Patients in whom dietary control enables biochemical homeostasis to be maintained despite the absence of renal function remain surprisingly comfortable during the acute stage of the illness. When peritoneal dialysis or the artificial kidney is used the patient inevitably suffers some discomfort, and is exposed to additional dangers. The safe application of these therapies requires elaborate biochemical control, and the necessary medical, nursing, and laboratory staffs will not always be available. These techniques for imitating renal function should therefore be employed only if there are clear indications for their use. The fact that they are being used does not allow of any relaxation of the high-calorie protein-free dietary intake. If peritoneal dialysis or haemodialysis is required it should be regarded as a supplement to continued dietary control.

The administration of testosterone promotes protein anabolism (Albright *et al.*, 1941) and produces a transfer of potassium into the cells in the normal K:N ratio. It is not certain that this effect on protein anabolism and potassium storage can be produced in patients with uraemia.

It is possible that one of the factors accelerating protein catabolism in uraemia is an increased secretion of 11-oxysteroids by the adrenal glands. Testosterone inhibits the output of adrenal steroids, and if there is adrenal cortical overactivity in uraemia this would provide an additional reason for its use in treatment (Dérot and Bernier, 1950).

Summary

Oliguria is a serious complication of acute glomerular nephritis. Unsuitable treatment has contributed to the high mortality.

New modes of therapy have been introduced in recent years for the management of cases of severe oliguria or anuria. The application of these measures should be based on an understanding of the biochemical disturbances consequent upon failure of renal function.

Two cases of anuria in acute glomerular nephritis are reported. One was treated with a special diet and the other by peritoneal dialysis. Both patients recovered.

Detailed metabolic studies provide data for a discussion of the biochemical disturbances arising in renal failure and of the means by which they may be treated.

It is considered that patients with severe oliguria should be maintained on a high-calorie diet with restricted intake of protein, water, and electrolyte.

Treatment by peritoneal dialysis or by the artificial kidney is complex and sometimes dangerous. Therefore these techniques should not be applied as a routine to all patients. Indications for their use are overhydration, acidosis, and hyperkalaemia. When these measures are employed the special diets should be continued.

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THE CLINICAL DIAGNOSIS OF DISSECTING ANEURYSM OF THE AORTA

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An ante-mortem diagnosis of dissecting aneurysm of the aorta was rarely made before the publication of Shennan's excellent monograph in 1934. Although described as long ago as 1728 by Nicholls, established as a pathological entity by Peacock in 1843, and correctly diagnosed during life by Swaine and Lathan (1856), the correct ante-mortem diagnosis had been recorded only seven times prior to 1934. By 1939 Rogers was able to collect from the literature 24 cases in which the diagnosis had been made during life, and since then many such reports have appeared. As pointed out by Willius and Cragg, failure to make the diagnosis is due to (1) the relative infrequency of the condition; (2) the absence of a characteristic syndrome; (3) the limitations of special diagnostic adjuncts; and (4) almost universal failure to bear the diagnosis in mind. To this may be added the extremely ill condition of many of the patients, precluding detailed clinical examination. A correct diagnosis is important if only to enable the clinician to give a more accurate prognosis. Furthermore, as the symptoms often suggest a surgical condition, a correct diagnosis will prevent unnecessary surgical procedures.

Because they are fairly representative of the way in which the condition presents, the following cases are considered worth recording. The correct ante-mortem diagnosis was made in two and suspected in three others.

Case 1

A man aged 64 was in good health until the day of admission, when he complained of sudden severe pain in the chest and neck, which came on as he was straining at stool. He described the pain as constricting and very severe. Shortly after the onset of the pain he found that his right leg had become useless and numb. By the time of admission, three hours later, the pain in his chest had improved but the right leg was still numb and painful. There was no previous history of serious illness or any symptoms suggesting hypertension.

On admission the patient, who was shocked, was lying flat in bed and not short of breath. The pulse in the right arm was 60 a minute, regular, and of fair volume. The apex beat was difficult to locate, but the heart sounds appeared normal. The blood pressure in the right arm was 110/70. There was no voluntary movement of the right leg, and the pulse was absent in the right femoral, popliteal, and dorsalis pedis arteries. Patchy areas of anaesthesia could be demonstrated. The pulses in the left leg and left arm were normal.

Eight hours after admission the pain in the chest recurred. At this time, although the pulses in the right leg were still absent, the leg felt normal to the patient, voluntary movement had returned, and the patches of anaesthesia were no longer found. The blood pressure in the right arm was 105/80. Twelve hours after admission it was noted that the left radial pulse was absent. Twenty-four hours after admission the mental condition of the patient was noted to be strange; he failed to recognize people and was in a state of stupor. Detailed neurological examination was not carried out, as he was so ill. The electrocardiogram was normal. He died 36 hours after admission.

Post-mortem examination showed a dissecting aneurysm of the aorta which began a short distance above the aortic valve, where there was a transverse rupture of the intima. It extended throughout the entire length of the aorta and for varying distances into both common carotids, the left subclavian, and the left axillary arteries. The lumen of these smaller vessels was almost obliterated by the accumulation of blood in the wall. The aneurysm had leaked into the mediastinum and also into the pericardium, where there was a large amount of blood clot. Histological examination of the aorta showed no evidence of cystic medionecrosis.

Comment.—The clinical picture was that of a coronary occlusion, associated with loss of voluntary movement in one leg and absent femoral pulsation, which had come on within a few minutes of the chest pain. Dissecting aneurysm appeared the most probable diagnosis, and this was confirmed by the normal E.C.G. and subsequent disappearance of the left radial pulse. Although pulsation was not found in the right femoral artery at any time during life, post-mortem examination showed dissection had stopped at the bifurcation of the aorta and the loss of pulse must have been due to spasm, possibly caused by interference with the sympathetic nerve supply.

Case 2

A woman aged 74 was in good health until the day before admission, when she developed severe pain in the chest and neck while doing housework. At the same time she noticed weakness of the right and slight weakness of the left leg. Sensation in the right leg was absent and she sustained a severe burn from a hot-water bottle, of which she was quite unaware at the time. The severe pain in her chest lasted for one and a half hours and normal power returned to the legs within a few hours. Examination some six hours after the onset revealed slight cardiac enlargement but no murmurs. On admission one day later the patient had no complaints. Examination revealed a difference in the blood pressure in the two arms (right 165/70, left 110/60). The heart was enlarged to the left and a loud diastolic murmur could be heard in the aortic area. All other pulses were present and no abnormal signs could be demonstrated in the legs. The electrocardiogram was normal. A radiograph of the chest showed an enlarged heart. Thirty-six hours after admission the patient collapsed and died. Permission for post-mortem examination was refused.

Comment.—Although post-mortem confirmation was not obtained in this case, the features were so similar to those of Case 1 that a diagnosis of dissecting aneurysm was made with confidence. The appearance