REFRESHER COURSE FOR GENERAL PRACTITIONERS

TREATMENT OF WATER AND ELECTROLYTE DEPLETION

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In a previous article in this series entitled "Modern Views on Uraemia and Dehydration" (*British Medical Journal*, 1950, 1, 893) I gave a general description of the treatment of dehydration. The present article aims at a more detailed exposition of the treatment of water and salt depletion, and includes an account of potassium depletion.

Water Depletion

Here the water content of the body is reduced both absolutely and relative to the salt content, and the concentration of electrolytes in body fluids, as estimated by the serum-sodium level, is increased. The syndrome arises when the intake of water is impeded by dysphagia, apathy, or coma, and is aggravated by any increase in sweating, as in the febrile patient. Hyperpnoea increases the loss of water in the expired air.

The characteristic subjective symptom of water depletion is thirst, which is related to general tissue dehydration and is not relieved by moistening of the tongue and palate. Oliguria is an important sign of an inadequate water intake, but must be distinguished from the oliguria of advanced salt depletion or primary renal disease. The urine in water depletion is of high specific gravity and chloride concentration. General signs of "dehydration," such as wrinkling of the skin, collapsed veins, and low intraocular tension, are less obvious in water depletion than in salt depletion of comparable degree, for the fluid loss is spread over the whole body fluid, not limited to the extracellular fluid compartment as in salt depletion. Water depletion requires treatment with non-saline fluid; suitable fluids are discussed later.

Salt Depletion

The primary change in salt depletion is decrease in the total salt content of the body. Salt is the definitive electrolyte of extracellular fluid, and a deficiency of salt causes shrinkage of the extracellular compartment, though the absolute fall in extracellular fluid volume is minimized by some lowering of the concentration of salt in the remaining fluid.

In contrast to water depletion, which arises mainly from defective fluid intake, salt depletion is usually due to abnormal losses of salt, though it may on occasion be aggravated by defective intake of salt in patients with anorexia. Vomiting, diarrhoea, intestinal aspiration, and exudation from extensive burns are all obvious causes of loss of salt from the body; the urine may be a less obvious channel of salt loss, as in Addison's disease or diabetic pre-coma, in both of which severe salt depletion can be traced to salt loss in the urine. Copious sweating is another cause of salt loss from the body, but water is lost to a greater extent than salt, and symptoms of salt depletion arise only when thirst is satisfied by water without added salt.

Review of the causes of the two syndromes indicates that salt depletion, as a syndrome demanding active

treatment, is commoner than water depletion, or, at least, that in the mixed syndrome of clinical dehydration salt deficiency usually predominates over water deficiency.

General signs of dehydration are prominent in salt depletion, and it may be very difficult to get into the collapsed veins, either for sampling or for venoclysis. Thirst is often rather strikingly absent. The urine volume may be quite high in mild salt depletion, but oliguria supervenes when the plasma volume has shrunk and the blood supply to the kidneys has decreased. The urine is usually chloride-free, except in Addison's disease or acidosis, and the plasma sodium is low; these points serve to differentiate the oliguria of advanced salt depletion from that of water depletion. Vomiting and anorexia may occur in salt depletion, and add to the difficulty of treatment with saline fluids.

Acidosis and Alkalosis

Disturbances of acid-base balance are often found in association with depletion of body fluid. Gastric juice contains more chloride than sodium, and its loss promotes alkalosis; intestinal juices are alkaline, and their loss by aspiration or diarrhoea leads to acidosis. Excessive breakdown of body fat, as in starvation or uncontrolled diabetes, promotes an organic acidosis. Acidosis may also be produced when salt depletion is treated by sodium chloride alone; such treatment dilutes the plasma bicarbonate, giving rise to a "dilution acidosis." Clinically, acidosis is associated with air-hunger, and the urine may contain chloride even when there is clear evidence of salt depletion. A low alkali reserve is found in this type of acidosis. Alkalosis is often quite silent clinically, but unusual drowsiness or mental aberration may suggest it, and polyuria is not uncommon; the alkali reserve is high.

Biochemical Tests in Diagnosis

Unfortunately, biochemical tests do not give direct estimates of the total amount of salt and water in the body, but only of concentration of electrolytes in body fluid. A further limitation is that only the extracellular fluid, as represented by plasma, is directly accessible to analysis. As a general rule, however, salt depletion is attended by a lowered concentration of sodium and chloride in the plasma, and water depletion by an increased plasma sodium. But it sometimes happens that concentrations of electrolytes give a false picture of electrolyte balance in the body, usually because the kidneys have failed to respond by appropriate changes in salt and water output. For this reason among others biochemical results must form only a part of the evidence on which clinical decisions on salt and water depletion must be based; a history of the type and amount of fluid lost is just as important as the biochemical results. When a general diagnosis has been arrived at on the sum of evidence, biochemical results are valuable in assessing the degree of depletion, with a view to replacement therapy.

The plasma sodium is the most satisfactory single estimation, especially when it can be done quickly and accurately by a flame photometer. The normal range is 137–147 mEq per litre. When a flame photometer is not available the plasma chloride and plasma bicarbonate should be estimated on the same sample. The plasma chloride is normally 100–105 mEq per litre, and the plasma bicarbonate 25–30 mEq per litre. When the sum of chloride and bicarbonate is outside the range of 125–135 mEq per litre an abnormality in plasma sodium can be inferred. Deficit of plasma sodium is confirmatory of salt depletion and gives an estimate of its severity. For a patient of average size a deficit of 10 mEq per litre of plasma sodium is equivalent to a total body deficit of 500 mEq of sodium, equivalent to about 30 g. of sodium chloride. The plasma sodium (or chloride + bicarbonate) gives information on the "water/salt ratio" in the body.

It is often desirable to know whether acidosis or alkalosis is present, and the plasma bicarbonate is the critical investigation here—a low figure suggesting acidosis and a high one alkalosis. Respiratory acidosis or alkalosis must be excluded clinically before basing an assessment of acid-base balance on the alkali reserve, but they are uncommon by comparison with metabolic acidosis and alkalosis.

The blood urea also gives some information on the severity of salt or water depletion if primary renal disease can be excluded; this may be useful in those few patients in whom salt depletion is associated with normal electrolyte levels in the plasma—a raised blood urea is then valuable supporting evidence for a clinical suspicion of salt depletion.

The significance of the plasma potassium will be discussed in relation to potassium depletion.

Choice of Fluid for Treatment

When it has been decided that body water is depleted to a greater extent than salt, the appropriate fluids for use in treatment are water by mouth or 5% glucose intravenously or subcutaneously. In the alert conscious patient who is not vomiting, the water intake by mouth should be pushed until thirst is relieved; thereafter it should be adjusted to some 1,500 ml. a day, or 2,500 ml. in the presence of fever or hyperpnoea. When the patient cannot be roused to drink adequately, or when he is vomiting, 5% glucose can be given parenterally, or a rectal drip of tap-water can be set up. When a patient who has previously been thirsty no longer complains of thirst, it is a sign that the water depletion has been substantially corrected, and the fluid intake should be adjusted to basal requirements.

When the patient's kidneys are healthy no real harm is done by giving some excess of water, as the excess will promote a corrective water diuresis; but the situation is quite different when the kidneys are faulty, so that they cannot respond by water diuresis to excessive water intake. Water intoxication is then a real danger, to be avoided by giving just enough fluid to control thirst.

For a few days after surgical operation water given to the patient is excreted abnormally slowly, and apathy, retching, or convulsions may then be signs of excessive water ingestion. A low serum sodium, in the absence of dehydration, is biochemical evidence of some degree of water intoxication. The intake of water should be restricted, and sweating encouraged by radiant heat cradle. Actual convulsions can be controlled by hypertonic

saline injected intravenously—10–20 ml. of 5% saline is given by syringe.

Correction of salt depletion has to be done with saline fluids. Unfortunately, only fluids of quite low salt content can be tolerated by mouth in any quantity; patients with salt depletion, moreover, do not have a craving for salt comparable to the thirst of a patient with water depletion. Something can be done by adding salt to the food over a period of time, and by using drinking water, milk, fruit drinks, or meat extracts made up to a salinity of about 0.45% sodium chloride ("half normal"). But it is not possible to correct severe salt depletion rapidly by oral saline fluids, as rapid correction of salt depletion demands fluids of a higher salinity than body fluid.

The size of the salt deficit in clinical salt depletion has not always been realized, but it constitutes a real difficulty in replacement therapy. For example, a deficit of 20 mEq per litre in the plasma sodium, such as is by no means uncommon in clinical salt depletion, corresponds to a total body salt deficit of 1 gramme-equivalent—that is, no less than 58.5 g. of salt or about 7 litres of "normal saline." It is obvious that a deficit of this order cannot be quickly corrected by mouth, and, further, that its correction by normal saline involves the giving of very large amounts of fluid by vein.

In patients with unequivocal salt depletion we have found it advisable to correct the greater part of the initial deficit with 2% or 5% saline, allowing the patient to drink at will during the infusion; we have even given as much as 500 ml. of 10% saline in this way, without untoward effect, and there is much less risk of overloading the circulation when this comparatively small amount of fluid is given. An infusion of this type should be spread over about three hours. After gross salt deficit has been partly corrected, and the kidneys have been assured of an adequate circulation, it is possible to complete correction of the deficit and to cover basal requirements of salt by the use of normal or half normal saline. At this stage it is important to watch for signs of overdosage with salt; such overdosage leads to expansion of the extracellular compartment, including the plasma volume. Engorgement of the veins or moist sounds at the lung bases are clear indications for stopping intravenous saline.

In patients who cannot take or retain water by mouth, and in whom therefore hypertonic saline therapy may carry some risk, the best course is to aim at a more gradual replacement of the salt deficit with normal saline, by giving 3–5 litres a day for two or three days and watching closely for any signs of saline overloading.

Special caution must be exercised in treating salt depletion in infants and children, since the absolute salt deficit is small because of the low body weight and also because of the inability of the infant kidney to attain a high concentration of urinary chloride; in the adult the ability to form a hypertonic urine is a partial protection against overdosage with salt. On the other hand, the daily turnover of water in infants is high in relation to their weight, by comparison with adults, and 0.2% or 0.45% saline should be used in the treatment even of salt depletion.

No reference has so far been made to the control of the acid-base balance during treatment with intravenous fluids. If water and salt balance is restored and maintained, healthy kidneys can usually correct acidosis or alkalosis of moderate degree. As has already been men-

tioned, when large amounts of sodium chloride are given there is a tendency to acidosis; for this reason, when the patient has no antecedent alkalosis it is wise to give a fifth part of the sodium replacement in the form of bicarbonate by mouth, or lactate by vein. Sodium lactate is usually supplied in ampoules of molar concentration, and should be diluted one part in six to make M/6 sodium lactate, which is isotonic with plasma.

In the treatment of primary severe acidosis M/6 sodium lactate should be used; the alkali reserve should be estimated frequently, as kidney function may be impaired, and with it the defence against alkalosis from overinfusion of lactate.

The appropriate treatment for primary metabolic alkalosis depends largely on the cause. When vomiting is paramount the alkalosis is associated with severe salt depletion and can be corrected by replacement of the salt deficit by sodium chloride without lactate. When the alkalosis is related to long-continued alkali treatment of peptic ulcer there is no salt deficit and ammonium chloride can be used by mouth. The use of M/6 ammonium chloride intravenously has been recommended for the treatment of alkalosis, but it should be limited to patients with a severe primary alkalosis and with no salt depletion.

In treating electrolyte imbalance we must always remember that the agents used are themselves capable, when given in excess, of causing a syndrome opposite to what is being treated. It is not enough merely to make even a careful initial assessment of the original water or salt deficit and to restore it quantitatively; not only is the error of any such assessment considerable, but the body also adapts itself to water or salt depletion, and simple restoration of a calculated deficit will not restore normality but will leave the body economy distorted by such adaptations as have taken place during the period of uncorrected depletion. Fortunately, with each approach to normality the natural homeostatic capacities of the kidney are liberated from the constraints imposed on them by the severer degrees of fluid depletion and they can correct considerable deviations from the optimum of fluid given. This does not absolve us from the responsibility of making a careful initial assessment and of keeping constant observation and undertaking biochemical control so long as fluid is being given artificially.

When the patient has been adequately treated for water or salt depletion his weight should be normal, and there should be no oedema or venous plethora, no haemoconcentration, and no abnormality in the plasma concentration of sodium, potassium, chloride, or bicarbonate. The blood urea should be falling, though it often takes some days for an accumulated nitrogen load to be dissipated.

Potassium Depletion

The widespread recognition of potassium depletion as a clinical problem is a matter of the past five years, and springs from increasing appreciation and extension of Darrow's fundamental observations on potassium depletion in infantile gastro-enteritis. Although potassium depletion is probably as common as sodium depletion, it is much less easy to demonstrate, as potassium is mainly an electrolyte of intracellular fluid; severe potassium depletion may be present without any clear change in the potassium concentration in plasma. It is true that potassium depletion can be shown by analysis of muscle

biopsy material or by subsequent retention of potassium in a balance experiment, but these methods are limited in their application and to some extent retrospective. It is the more important to appreciate the usual mechanisms by which potassium depletion is produced, so that treatment may be started early.

The body depends for its potassium intake on ingested food—notably bread, vegetables, and meat—and any restricted diet is likely to be a low-potassium diet; anorexia likewise lowers the potassium intake. The renal defence of body potassium is relatively inefficient, in that potassium is excreted to some extent even on a low-potassium diet, in rather striking contrast to the disappearance of sodium from the urine on a low-sodium diet. The renal loss of potassium is increased by dehydration, by massive saline infusion, and by osmotic diuresis such as is produced by the glucose excretion in uncontrolled diabetes mellitus. Trauma to tissue raises the plasma potassium and augments urinary potassium excretion. Urinary output of potassium is also increased under the influence of adrenocorticotrophic hormone, cortisone, or deoxycortone acetate.

Gastro-intestinal secretions contain rather more potassium than is present in plasma, and loss of these secretions in sufficient amount depletes the body potassium. Even when the total body potassium is not depleted, movement of additional potassium into the cells may lower the plasma potassium. For example, when glycogen is being formed under the influence of insulin the plasma potassium may fall to half its initial value; and in attacks of familial periodic palsy the plasma potassium is low, although there is no net loss of potassium from the body.

Clinical Aspects of Potassium Depletion

Potassium depletion has been recognized in patients with severe diarrhoea or vomiting, during the recovery phase of diabetic coma, and during convalescence from surgical operations. Less common syndromes associated with potassium depletion are Cushing's syndrome, deoxycortone overdosage, familial periodic palsy, and massive saline infusion.

In evaluating the clinical effects of potassium depletion it is important to differentiate between the effects of intracellular and of extracellular potassium deficit. Although the intracellular potassium is more commonly and more extensively depleted than the comparatively small moiety of potassium which is extracellular, the effects attributed to intracellular potassium deficit are somewhat vague. They include apathy, muscular weakness, abdominal distension, and mental disorientation. Such symptoms are obviously difficult to ascribe to any one cause in patients who commonly have depletion of protein and of other electrolytes as well as of potassium, but they have sometimes been found to improve with potassium treatment alone. It should be frankly admitted, however, that the clinical significance of intracellular potassium deficit rests at present more on the proved frequency of such a deficit than on any characteristic syndrome of effects.

No such uncertainty surrounds the effects of extracellular potassium deficit, for a fall in the concentration of plasma potassium is easy to detect and the corresponding clinical syndrome is well defined. Paresis of muscles and generalized weakness are the outstanding clinical effects of lowered extracellular potassium concentration; the paresis may be extensive, and simulate

an ascending Landry's paralysis. The electrocardiogram commonly shows a low flat T wave, prolonged Q-T intervals, and depressed ST segment, the degree of change being dependent on the extent to which plasma potassium is lowered. The low plasma potassium is often associated with a metabolic alkalosis, the plasma bicarbonate being raised; this change may be related to the entry of sodium into the cells to replace a potassium deficit therein.

In the diagnosis of potassium depletion most importance should be attached to a clinical assessment of the possibilities of potassium loss by diarrhoea or vomiting, or by polyuria. A test dose of 10 g. of potassium chloride is normally excreted within 24 hours, and retention of such a dose indicates a potassium deficit of some magnitude. A plasma-potassium level of less than 4 mEq per litre demonstrates depletion of extracellular potassium; but a normal plasma potassium cannot be taken as excluding potassium deficiency, as this may be confined to the cells; moreover, even if the total extracellular potassium is low, this may be masked by loss of extracellular fluid due to concurrent sodium depletion and hence potassium concentration. A rise in the plasma bicarbonate may precede any fall in plasma potassium, since sodium enters the cells at a stage of potassium depletion before the extracellular potassium is involved; but the diagnostic value of this change is lessened by its lack of specificity. Appropriate electrocardiographic changes are useful evidence of potassium depletion, but their absence does not exclude it.

Treatment of Potassium Depletion

An ordinary diet supplies 2-5 g. of potassium daily, and this gives some guide to what must be supplied in treatment. A pint (570 ml.) of milk supplies about a gramme of potassium, and undiluted fruit juices supply a similar amount. Strong coffee has about half this potassium content, and drinks made from meat extracts rather less than coffee. It is therefore possible to give about a normal amount of potassium in a fluid diet, but not to give any great surplus to correct depletion. Potassium chloride can be given in doses of 5 g. at a time without provoking nausea, and the taste can be partly disguised with sweetened milk. About 10-15 g. a day by mouth is a suitable dose for a patient who is recovering from diabetic coma, and should make up the probable deficit in a few days.

When the renal excretory function is normal potassium can be given by mouth without risk, as the amount tolerated orally is well within the excretory powers of normal kidneys. In patients with renal impairment potassium salts should not be given unless the level of plasma potassium can be frequently checked. Oral potassium is the treatment of choice for patients with no gastro-intestinal illness, and intravenous potassium should be limited to those who have a low plasma potassium and who cannot take adequate quantities by mouth.

The risks of giving intravenous potassium are quite definite, and fatal accidents have been reported. The danger lies not so much in the total amount of potassium given as in the speed with which the plasma potassium concentration is increased. The margin of safety is small, as an increase to 10 mEq per litre of plasma potassium produces definite electrocardiographic changes and the risk of cardiac arrest. The concentration of potassium in fluids for intravenous use should not exceed 70 mEq per litre, corresponding to 0.5% potas-

sium chloride, and the rate of giving such a solution should not exceed 4 ml. a minute. Too high a concentration of potassium in plasma causes peaking of the T wave in the E.C.G., cardiac irregularity and arrest, and may also cause paralysis. If any of these signs appear during potassium infusion the potassium should be stopped, a glucose infusion begun, and 5 units of insulin given intravenously. A conservative attitude towards intravenous potassium therapy is justified, and at present it should be limited to patients with actual paresis and a low plasma potassium. Increasing knowledge of the effects of intracellular potassium deficit may broaden the indications for intravenous potassium therapy, but at present it seems best to limit it to patients with an extracellular potassium deficit causing definite clinical symptoms.

LEGAL IMPLICATIONS OF MEDICAL PRACTICE IN U.S.A. AND GREAT BRITAIN

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In considering any legal question regarding the U.S.A. one has to bear constantly in mind the fact that it is not one country, in the British sense of the term, but is in fact the "United States" of America. This aggregate of 48 states is governed by two sets of statute law—the federal law enacted by Congress and applicable to all states, and the state law which is peculiar to, and applicable only in, the state in which it is enacted. While many of the legal aspects of medical practice are fairly uniform throughout the whole country, there are many more which vary from state to state. It would be clearly impossible in one short paper to deal with all the minutiae of variation from state to state, and, in any case, this would be of little interest to British medical readers. It is proposed, therefore, to handle the matter in a general rather than in a detailed fashion.

The Lawful Practice of Medicine

At common law any person who wished could practise medicine. This is precisely the position which existed in Great Britain before the passing of the Medical Act of 1858. In Great Britain any unregistered person may still lawfully practise medicine if he wishes to do so, subject to certain well-known limitations, and provided he uses no title which would lead a member of the public to believe that he is a registered medical practitioner. The American Congress has no power under the Federal Constitution to regulate the practice of medicine. It is a matter over which each individual state has jurisdiction. In all the states medical practice is regulated by statute, and it is provided generally that before undertaking the practice of medicine one must first secure a licence or certificate from a designated body or board.

The practice of medicine by an unlicensed person, or the aiding and abetting of an unlicensed person to practise medicine, constitutes a criminal offence. As in Britain, therefore, the possession of a degree in medicine