THE importance of adequate fluid and electrolyte therapy is now readily appreciated, and the principles underlying the correction of deficiencies are well established. Everywhere the importance of base conservation by the body is accepted, and measures taken to correct any deficiency of base substance are rigorously applied. We appreciate clearly the difference between a true water deficiency as emphasized by Marriott (1947) and by Darrow and Pratt (1950), and the more serious "hypotonic" dehydration, where electrolyte is lost in amounts at least equivalent to the water loss. Before operation, we realize that the urinary chloride excretion is a more sensitive index of the body chloride state than is the plasma chloride concentration, and we are accustomed to correcting the fluid and electrolyte imbalance before the patient undergoes the additional hazard of the operation itself. The rapid estimation of sodium and potassium by the flame photometer allows us to control therapy from hour to hour and the lag between clinical and biochemical assessment of the patient's condition now scarcely exists. It is important, however, that the routine of replacement therapy should not blind us to the advances which are taking place in this field.

ELECTROLYTE CHANGES RESULTING FROM INJURY

These advances are mainly concerned with the intimate effects of operation upon fluid and electrolyte metabolism, and it should be clearly appreciated that they are not necessarily related to pre-existent deficiencies. Indeed, the changes to be described occur also in normal well-nourished adults who are not suffering from any water or electrolyte imbalance. The earliest accurate studies of the effects of injury on fluid and electrolyte metabolism were carried out by Cuthbertson and his colleagues (1939) on rats subjected to fracture of the femur. As a result of the injury they observed an increased excretion of nitrogen, phosphorus, sulphur and potassium in the urine. Subsequent
studies have shown that a complex series of changes occur which appear to be the result of injury or operation itself. Howard and Carey (1949) noted a post-operative retention of sodium and chloride in amounts greater than theoretical requirements would suggest. Wilkinson, Billing, Nagy and Stewart (1949), in a study of patients after gastric operations, observed this sodium and chloride retention in the first week after operation in patients who otherwise might be considered as stabilized in regard to fluid and electrolyte requirements. In 1949 also, Blixenkrone-Møller showed that, following operation, an increased potassium excretion can be detected within the first six hours after operation. Wilkinson, Billing, Nagy and Stewart (1950) also demonstrated this phase of increased loss of potassium in the urine during the first three days of the post-operative phase. These observations have tended to show that this urinary potassium loss is different in degree from the urinary nitrogen loss, and tends to occur at an earlier phase in the post-operative period.

It has now become clear therefore that the incident of an operation is associated with an increased urinary potassium excretion, and a retention of sodium chloride and water by the body. Operation is used here in the widest sense not only to include the surgical trauma, but the anaesthetic, the pre-medication and the apprehension, all of which may be considered an integral part of an operation. It has become clear also that these changes occur after quite minor operations where no fluid or electrolyte imbalance might occur (MacPhee, 1953), or in more severe operations, where all losses are accurately replaced.

The cause of these metabolic changes remains a matter for debate. Wilkinson and his associates believed that they could be accounted for by the transudation of sodium chloride and water through the capillaries at the site of tissue damage, and by the leakage of potassium from damaged cells. That such changes do occur at the site of operation is certainly correct. These changes have also been shown to occur in association with increased adrenocortical activity as a result of operation (MacPhee, 1953). As similar changes can be produced by the administration of the adrenal cortical steroids, there is something to be said for this theory. Le Quesne and Lewis (1953) consider that the sodium chloride and water retention are indeed due to adrenocortical activity, but in addition, they consider there is an earlier phase of water retention resulting from increased secretion of the pituitary anti-diuretic hormone. Obviously a considerable amount of work remains to be done to clarify these problems, but certain direct conclusions can be drawn at this stage on the management of fluid and electrolyte problems in the early post-operative phase. These conclusions deserve wider recognition because failure to recognise them may lead to some of the catastrophes with which this stage is fraught.

In the first week after operation, it remains a primary consideration to replace such water and salts as are lost. We may expect, however, a considerable sodium chloride and water retention during this period to such an extent that the normal common salt requirements need
not be expected. Provided, indeed, all deficiencies and abnormal loss are corrected, there is no need to supply any further saline during this phase until sodium chloride can be detected again in the urine in appreciable amounts. Such measures will prevent the saline overloading and water-logging of the patient, mistakes which are still too frequently encountered. During the first three days at least there is also an increased urinary potassium loss, as much as the equivalent of three grams of potassium chloride being excreted daily. As this potassium is being mobilised from the cells, the extracellular (and therefore plasma) potassium concentration remains normal or is even elevated for some time. Care must therefore be taken if potassium is supplied in any quantity immediately after operation, as toxic extracellular concentrations can rapidly be reached. In this connection, it is interesting to remember that potassium leaks out of the erythrocytes of stored blood to such an extent that the serum of stored blood may itself be toxic.

POTASSIUM DEFICIENCY

The greatest loss of potassium in the urine takes place in the first three post-operative days, and normally needs no correction if the patient is able to take a normal diet by mouth before or at that time. When, however, a pre-existent potassium depletion is already present, or where oral feeding is not possible after the third post-operative day, potassium deficiency may well occur. Deficiency of potassium leading to symptoms has been reported by Brown, Currens and Marchand (1944) in chronic nephritis, by Govan and Darrow (1946) in diarrhoea, by Lubran and McAllen (1951) in idio-pathic steatorrhoea, and by Hawkins, Hardy and Sampson (1951) in pyloric stenosis. A potassium deficiency may therefore be expected in all cases where vomiting or diarrhoea has been a feature of the disease before operation. Such deficiency, as we have seen, is enhanced by the operation itself, and may indeed be made more severe by the giving of large saline infusions, a factor which Gamble (1942) has shown also increases the potassium excretion rate. It is therefore in the case of operation on the intestinal tract which has been preceded by vomiting or diarrhoea or both, and in which early oral feeding is not possible, that severe potassium depletion is likely to occur.

The treatment of such a deficiency depends partly on suspecting it before muscular weakness, cyanosis, and cardiac enlargement occur, and by treating the patient at an early stage. As has been indicated earlier, the serum potassium concentration is not a good index of cellular deficiency, as the latter may be present when the serum concentration is not markedly reduced. Potassium depletion is best estimated by assessing the potassium loss in the urine and the vomitus or intestinal aspirate, but this is both a troublesome and time-consuming method. Electrocardiographic changes, if recognized, do offer early evidence of severe depletion, and where deficiency is already suspected, prolongation of the Q–T interval, depression or inversion of the T wave, and depression of the S–T segment are progressive signs that potassium replacement therapy is urgently required. Elkinton and Tarail (1950) recommend that, if parenteral replacement is envisaged, the maximum potas-
sium strength of the solution used should not exceed 80 milli-equivalents per litre, and no more than 20 milli-equivalents (i.e. 250 ml.) should be administered in an hour. It will be appreciated that even this strength of potassium is twenty times greater than the potassium concentration in the extracellular fluid. If, therefore, such a concentration of potassium is given, toxic concentrations of potassium may be built up in the extracellular space while cellular potassium depletion is still present. The solution obtained by dissolving 4.5 grams $K_2HPO_4$ and 1 gram of $KH_2PO_4$ in 1 litre of water contains about 60 milli-equivalents of potassium, and if given over a period of 4–6 hours is suitable in severe potassium depletion. Darrow (Darrow and Pratt, 1950), recommends his "K Lactate" solution, which corrects also the frequently associated acidosis. This solution contains 4.0 grams sodium chloride, 2.7 grams potassium chloride and 52 ml. of molar sodium lactate per litre. The potassium content is approximately 36 milli-equivalents per litre, and therefore, even in severe potassium depletion, a litre should not be given in less than 3 hours.

POTASSIUM INTOXICATION

Potassium intoxication is not normally encountered, except in renal failure, or when an overdose of a potassium salt is given. If such an overdose is suspected it is important to be able to treat the condition rapidly. The objects of treatment are to reduce the extracellular potassium concentration by increasing cellular potassium uptake, and by increasing the renal excretion. The latter is best obtained by promoting a simple water diuresis, but is a relatively slow process. Fenn (1939) showed that potassium is deposited in the liver along with glycogen, and it is now known that the extracellular potassium concentration falls during this process. A dramatic, though temporary, fall in the serum potassium can therefore be obtained by giving glucose and insulin by injection, and the response to treatment can be assessed by serial serum potassium determinations.

Clinical and experimental studies leave no doubt that fluid and electrolyte imbalance cannot now be treated by rule of thumb methods. Each patient represents a separate problem in therapy, a problem which can only be solved by adequate data and sufficient knowledge. There is no place for a random choice of inappropriate electrolyte and hit-or-miss methods. Each deficiency must be estimated and corrected as rapidly as is consistent with safety. This is the surest way of increasing the range of operability, and reducing the rate of mortality.

REFERENCES