Patients admitted to intensive care units may suffer from the failure of more than one organ system. Good management therefore consists of the correction not only of cardiorespiratory problems, but also of gastrointestinal dysfunction, nutritional and metabolic defects, water and electrolyte balance and degrees of renal failure. Whilst all these aspects have to be considered separately, they relate to each other, and neglect of one factor may reflect adversely upon the others. It is the purpose of this paper to discuss first the nutritional and metabolic problems and second some related aspects of the water, electrolyte and renal pathophysiology of critically ill patients. In order to give logical treatment it is first necessary to analyse the physiological abnormalities.

PATHOPHYSIOLOGY

Nutrition and metabolism

The twin problems of starvation and of the metabolic response to injury often occur together. Their clinical importance is highlighted when one considers that a 30–40% weight loss as a result of acute starvation is often fatal, but that in the presence of illness the fatal weight loss may be as low as 25%.

Starvation

Levenson, Crowley and Seifter (1975) have reviewed the work in this field by authors such as Lehmann, Dubois, Benedict, Keys and Cahill, which has led to our present knowledge of the exquisitely appropriate adaptations to conserve essential tissues which take place in starved man. Fat stores provide 80–90% of the energy requirements, with the remainder being obtained from protein as soon as the small stores of glycogen (approximately 400 g) are exhausted. Whilst fat is the chief fuel store, muscle protein is the main reserve, not only of nitrogen, but also of carbohydrate which is essential for the energy metabolism of nervous and blood tissues and for the supply of oxaloacetate and other important intermediary metabolites. Since fat, except for its glycerol moiety, cannot be reconverted to glucose, amino acids released from muscle are taken up by the liver, where the amino groups are used for urea synthesis and the carbon fragments enter the gluconeogenic pathway. Alternatively, the liver can use this supply of amino acids for synthesis of plasma and essential proteins. After 3–5 days of starvation, lipolysis and the consequent high concentrations of acetyl CoA lead to the formation of ketone bodies from which the brain can derive a substantial proportion of its energy. Muscle can also oxidize ketones instead of the branch-chain amino acids, which again spares protein. Added to these two changes is an overall decrease in metabolic rate which is greater than would be expected from the loss of lean body mass. There is, therefore, less demand for glucose and the rate of gluconeogenesis decreases by 50%. These factors have been described as the metabolic "adaptation to starvation", the net result of which is to conserve body protein and create the best conditions for survival.

Response to illness or injury

In contrast to starvation alone, injury and illness are associated with accelerated tissue catabolism without the limitations imposed by the adaptation to starvation. The changes are proportional to the severity of injury and are both prolonged and exacerbated by complications such as sepsis. Following uncomplicated abdominal surgery the catabolic response may be small and transient, whereas after a major burn it may be large and persist for several weeks.

Cuthbertson (1930) described the increased metabolic rate and the accelerated nitrogen losses after injury. He proposed that muscle protein is mobilized to meet the demands of healing and showed that the increase in net protein catabolism could be partly offset, but not abolished, by feeding. The work of Kinney and his colleagues (1970) has shown that the function of protein catabolism after injury is not primarily to meet the increased demand for energy but to supply the liver with precursors for gluconeogenesis. They showed further that the acceleration of hepatic gluconeogenesis was not inhibited by artificial increase of the blood glucose. The necessity for such an unrestrained increase in glucose supply is only partly understandable in terms of the requirements.
of specialized tissues such as the nervous system. Recently, Wilmore and his colleagues (reviewed by Wilmore, 1977) have studied patients where a burn injury involved one leg and spared the other. They found that glucose uptake in the injured limb was much greater than in the control and suggested that the increase in gluconeogenesis was partly to meet the demand for glucose of injured tissues. The metabolic response to acute illness or injury may therefore be summarized as follows:

**Metabolic rate** is increased in proportion to the severity of the illness and its complications. Following an uncomplicated operation for vagotomy and pyloroplasty the minimal increase in the metabolic rate may be more than offset by the associated bed rest so that the energy requirements may lie between 1500 and 2000 calories (6.3–8.4 MJ) per day. With major surgery, particularly that associated with intra-abdominal sepsis, the increase may be greater, giving an energy requirement of 2000–3000 calories (8.4–12.6 MJ) per day. With major burns there may be an increase of 50–100% with an energy requirement of 2500–4000 calories (10.3–16.8 MJ) daily.

**Protein metabolism.** There is a net breakdown of muscle protein to supply amino acids to the liver as precursors for both gluconeogenesis and visceral protein synthesis. This phenomenon is again proportional to the extent of injury and its complications.

**Carbohydrate metabolism.** Glycogen stores are mobilized and there is an increase in gluconeogenesis. There is glucose intolerance in respect of both ingested and infused glucose solutions. This has important therapeutic implications. An increased glucose supply is essential to meet not only the demands of the nervous system, blood cells and intermediary metabolism, but also those of injured tissues.

**Fat metabolism.** Fat stores provide 80–90% of the energy requirements in the injured fasted patient. Triglyceride is mobilized by lipolysis, leading to an increase of plasma free fatty acids and glycerol which are then available for cell oxidation.

**Mediation of the metabolic response to injury**

Although there may be direct effects on metabolism by the injured tissues and by peripheral nervous control, nevertheless it seems likely that the major mediating factors are endocrine. Albright, in his Harveian oration of 1943, drew attention to the similarity between the changes after injury and those seen in Cushing's syndrome. When, in 1943, Cope and his colleagues demonstrated the increased production of corticosteroids from the adrenal gland after injury, it was postulated by Selye (1946) that cortisol may be the important mediating factor. Since that time more details of the endocrine response to injury have been described. The importance of increased catecholamine secretion, demonstrated by Birke and colleagues (1957) and Goodall, Stone and Haynes (1957) following burn injury, has been underlined by Wilmore, Long and colleagues (1976). In the acute phase of injury, Allison, Prowse and Chamberlain (1967) and Allison, Hinton and Chamberlain (1968) described the catecholamine-mediated suppression of insulin secretion. This was followed by a period of insulin resistance where high insulin concentrations were found in the presence of continuing glucose intolerance. Glucagon concentrations are also increased (Wilmore et al., 1974). The metabolic changes which might be expected when insulin action is relatively depressed in the presence of increased secretion of catecholamines, glucagon and cortisol bear a remarkable resemblance to those actually observed (Allison, 1974) (fig. 1). Studies carried out on patients in whom denervated limbs have been injured, or where injury is below the site of a high spinal lesion, have shown that denervation of the injured site produces very little modification in the endocrine or metabolic response (Wilmore, Taylor et al., 1976). The effects of injury are therefore determined at a central level and are presumably under thalamic and hypothalamic control.

**Modifying factors**

**Nutrition.** Although the metabolic response to injury is proportional to the severity of the initial
injury and its subsequent complications there are other important factors which also determine the response. The nutritional state of the patient at the time of illness is one of these. If a patient has already undergone a period of starvation causing wasting of muscle protein, the negative nitrogen balance after any subsequent injury is diminished. This does not represent a beneficial effect of starvation, but rather represents a labile protein reserve inadequate to meet the demands of injury. It is paradoxical that a response such as mobilization of muscle protein is essential for survival and yet, when carried to excess, contains the seeds of destruction of the patient. It serves to emphasize that the aim of treatment should be not to block all the metabolic responses to injury, but to steer them into paths which ensure subsequent survival. For example, it may be undesirable to have a diminished protein reserve, or to block the patient's muscle catabolism with glucose, unless at the same time sufficient protein is supplied from feeding to make the mobilization of the patient's own reserves unnecessary.

Environmental temperature. The increase in metabolic rate after injury is, of course, associated with heat production. It has been postulated that the major stimuli for this response are first a requirement to maintain a high core temperature and second the increased heat losses associated with conditions such as burn injury. It seemed possible, therefore, that nursing patients at the thermoneutral zone (30–33 °C) would diminish the metabolic stimulus. Davies and Liljedahl (1970) reported a significant reduction in nitrogen and potassium losses in burned patients nursed at 32 °C when compared with normal room temperature. Wilmore (1977) found less dramatic changes and concluded that although a high environmental temperature removes the additional stress of maintaining body heat in cold surroundings, it does not abolish the fundamental response to injury. In practical terms it would seem that critically ill patients, particularly those with burns, require warm surroundings to remove the additional stress thereby minimizing their metabolic requirements.

Exercise. Paralysed or inactive muscles tend to waste. Patients with motor dysfunction following head injury or other neurological illness, however well managed their nutritional treatment, suffer wasting of limbs. Conversely, early mobilization and exercise in surgical patients can have anabolic effects. Of particular consideration to anaesthetists is the wasting of diaphragmatic and intercostal muscles which may take place when respiration is assisted for long periods and which may contribute to the difficulties of weaning patients from ventilators.

Treatment. Cure of underlying disease such as infection restores metabolic normality. Sometimes however, as in patients with burns, this process is prolonged and the metabolic changes must be modified by an active nutritional programme until such time as the underlying disease has resolved.

CLINICAL ASPECTS OF NUTRITION

A 2- or 3-day fast following uncomplicated abdominal surgery may be of little clinical consequence, but in severely injured patients such as those with extensive burns, the catabolic rate may be so high that it is important to start an adequate feeding regime as soon as possible after the initial stage of resuscitation. The oral route should be used whenever possible and particular attention paid to the palatability and nutritional value of the food supplied. Even with inflation, steak and chips is still a cheap drug. High-protein, high-calorie ice creams are particularly useful in children. It should be a rule in burned patients that all fluids ingested should have at least some calorie or protein content. Only in this way is it possible to keep up with the enormous metabolic demands imposed by such an illness. Where the gastrointestinal tract is functioning normally but the patient is unable to swallow (for example patients on ventilators), nasogastric tube feeding is indicated. Only where it is impossible to supply sufficient food via the gastrointestinal tract should there be recourse to i.v. feeding. The principles of design of any feeding regime are similar whichever route is to be used. Energy requirements under different conditions have been discussed above. A nitrogen intake of 0.2 g/kg body weight with a calorie-to-nitrogen ratio of 180 : 1 (0.75 MJ/g N) will suffice for a non-catabolic patient. In the most severely ill patients, however, the nitrogen intake may need to be double this quantity and, contrary to previous belief, should probably be increased in greater proportion than the energy input, giving a calorie-to-nitrogen ratio of 150 : 1 (0.63 MJ/g) or less (Wilmore, 1977; Woolfson, Heatley and Allison, 1977).

Tube and supplementary feeding

Technique

Where swallowing is impossible, but gastrointestinal function is adequate, this method is easy, safe, effective and cheap, provided that it is carried out properly. The feed may be delivered via a fine bore
(Ryles tube) passed into the stomach. Alternatively, a long central venous catheter line can be introduced into the stomach by means of a guide wire or endoscope. Another simple way of introducing such a line is by clipping the catheter and Ryles tube tips together with a gelatin capsule (obtained from an antibiotic capsule). The two tubes are introduced side by side, the capsule blown off by syringing water down and the Ryles tube withdrawn, leaving the catheter in place. Our own method of administration is to place the feed in a standard 0.5-1-litre screw-top Winchester bottle and allow it to drip slowly into the nasogastric tube via a "Nottingham" drip feed system (adaptation of bladder wash out set: Avon Plastics Ltd). The screw-top at one end of the set allows a water-tight connection with the feed bottle. The connector at the other end fits into a Ryles tube only. There is no chance therefore that the set will be inadvertently connected with an i.v. cannula. When fine nasogastric catheters are employed there is no alternative to using i.v. type bottle and drip sets. Initially, milk and water is given at a rate of 30–100 ml/h and the stomach aspirated every 4 h. As soon as it is apparent that the stomach is emptying normally, the appropriate feed is given at half concentration for 24 h and thereafter at full strength. The feed is prescribed in detail by a doctor or dietitian. One day's supply is made up accordingly by the diet kitchen cooks or the nursing staff on the ward and stored at 4 °C. Administration of nasogastric tube feeds by continuous drip has two main advantages over the technique by which a bolus of feed was syringed down the tube by a nurse every 1–2 h. First it saves much valuable nursing time (Woolfson et al., 1976) and second it causes much less diarrhoea. The feed may be given throughout the 24 h or just as a supplement during the night.

Materials
I see very little case for the use of so-called "elemental diets" which are prepacked combinations of amino acids with calories, vitamins and minerals. They are extremely expensive and there is no proof that the giving of amino acids has any advantage over the giving of whole protein. Indeed, since half the nitrogen absorbed is usually in the form of di- and tri-peptides, the administration of amino acids cuts out half the route of absorption of nitrogen and may well be a positive disadvantage. The myth has also been created that these materials somehow contain a lower residue than whole protein such as milk powder or eggs. The additional disadvantage of prepacked diets is that their electrolyte content is fixed and in clinical situations of salt retention or hyperkalaemia it is difficult to give a low sodium or potassium feed.

In our unit we use artificial tube feeds made up from individual materials so that the feed can be altered to suit a particular patient's requirements. Such feeds are less viscous than liquidized ward diets and therefore can be more easily administered by the drip technique. Furthermore, they also have a known content, enabling balances to be kept more easily.

Our main energy source (Caloreen, Scientific Hospital Supplies Ltd) is a glucose polymer of average length 5 molecules. It is absorbed as glucose

\[
\text{Table I. Designs for tube feeding}
\]

(1) Caloreen–Complan. For ordinary maintenance feeding on general wards. Nitrogen is given as whole protein (Complan)

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Wt (g)</th>
<th>Non-protein energy kcal</th>
<th>Protein (g)</th>
<th>(N_2) (g)</th>
<th>(Na) (mmol)</th>
<th>(K) (mmol)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Caloreen</td>
<td>250</td>
<td>1000</td>
<td>—</td>
<td>—</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Complan</td>
<td>300</td>
<td>1070</td>
<td>60</td>
<td>9.6</td>
<td>46</td>
<td>53</td>
</tr>
<tr>
<td>Total</td>
<td>500</td>
<td>2070</td>
<td>60</td>
<td>9.6</td>
<td>46</td>
<td>53</td>
</tr>
</tbody>
</table>

The feed is made up to 3 litre with water. More sodium or potassium may be added as indicated clinically. Hypercatabolic patients (e.g. burns) require both higher energy and nitrogen intakes. The amounts of Caloreen and Complan may be increased to the limit of tolerance of the patient. There is sufficient fat in Complan to prevent fatty acid deficiency. The vitamin, mineral and trace element content are probably sufficient for most patients. Cost: £0.59.

(2) Caloreen–Albumaid. Low electrolyte feed

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Wt (g)</th>
<th>Non-protein energy kcal</th>
<th>(N_2) (g)</th>
<th>(Na) (mmol)</th>
<th>(K) (mmol)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Caloreen</td>
<td>600</td>
<td>2400</td>
<td>10.0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Albumaid</td>
<td>60</td>
<td>—</td>
<td>—</td>
<td>8</td>
<td>26</td>
</tr>
<tr>
<td>Mineral mixture (Ca, P, Mg trace elements)</td>
<td>8</td>
<td>—</td>
<td>—</td>
<td>14</td>
<td>17</td>
</tr>
<tr>
<td>Ketovite tabs</td>
<td>2</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Ketovite syrup 5 ml</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Total</td>
<td>—</td>
<td>2400</td>
<td>10.0</td>
<td>8</td>
<td>40</td>
</tr>
</tbody>
</table>

If the feeding period exceeds 7 days, essential fatty acids provided by 1 egg yolk daily. Water is added as appropriate —usually 1500–2500 ml. The electrolyte content may be diminished by omitting the mineral mixture, or increased by addition of sodium or potassium chloride. Cost: £2.60 daily.
but is one-fifth of the osmolarity of glucose in solution. In most cases Complan (Glaxo Ltd) is used as the nitrogen source, but the beef serum hydrolysate Albumaid (Scientific Hospital Supplies Ltd) is particularly useful where a low electrolyte feed is required, as this product is virtually electrolyte-free. The basic design of our tube feeds is outlined in table I.

Complications and precautions

Hyperosmolarity is most commonly caused by hyperglycaemia since sick patients are intolerant of glucose. The large amounts of Caloreen administered may give rise to this problem in a few critically ill patients. Since a diabetic urine chart is kept for all our patients, glycosuria can be observed early and insulin can be administered on a sliding scale. If renal function is poor, or if an inadequate water intake is maintained, or if insufficient calories are administered in relation to nitrogen, the blood urea may increase. Twice weekly, urea and electrolyte measurements allow this complication to be observed early and the appropriate counter-measures taken.

Diarrhoea. With the drip method of feed we have found this uncommon except in patients who are receiving broad-spectrum antibiotics. The symptom has responded immediately to the administration of codeine phosphate syrup 30–120 mg daily down the nasogastric tube. In no case has the feed had to be withdrawn.

Nausea. This may be present at the early stages and be caused by too rapid introduction of feed. It can be helped with the use of metoclopramide syrup 10 mg three times daily.

Supplementary feeding

All these materials may be used as supplementary feeds by mouth. Caloreen, in particular, is very much less sweet than glucose and large amounts can be administered without causing nausea. Any amino acid preparation is relatively unpalatable and we prefer to use whole protein in the form of either Complan or eggs.

Nitrogen source

This should be in the form of L-amino acids with an adequate proportion of essential and an even distribution of the non-essential amino acids (Wretlind, 1974). For the majority of patients Aminosol 10% (Kabi Pharmaceuticals Ltd) is still the cheapest effective nitrogen source. It should not be used in paediatric practice because of its peptide and sodium content. There are several synthetic L-amino acid preparations such as Vamin or FreAmine which are more expensive but have a more balanced electrolyte content. Optimum protein sparing is obtained when calorie and nitrogen sources are given simultaneously.

Much confusion has been created by the proposal of Blackburn, Flatt and Clowes (1973) that isotonic amino acid infusions should be used in the feeding of surgical patients. They argued that 5% dextrose infusion excites an insulin response. This in turn inhibits lipolysis and the resultant ketosis associated with the adaptation to starvation. If isotonic amino acid solutions were substituted for dextrose, then ketosis would develop and allow the administered nitrogen to be used anabolically. They were in fact able to show that a better nitrogen balance was obtained when amino acids were used alone than when dextrose was used alone. In further studies they argued that the addition of dextrose to the amino acid infusion produced a worse nitrogen balance than when the amino acids were given on their own. However, this conclusion was unwarranted and has been refuted by others (Greenberg et al., 1976). In practical terms, therefore, there is little place for isotonic amino acids, save in the non-catabolic patient with adequate fat stores. Since such patients are usually able to eat within a few days of operation and injury, the marginal advantage in terms of nitrogen balance obtained by giving fluid in the form of isotonic amino acid solutions instead of 5% dextrose seems hardly worth the considerable extra expense.

Energy sources

Glucose (4 cal/g, 16.7 kJ/g). This is the most satisfactory carbohydrate source. Since one of the main roles of protein catabolism after injury is to provide gluconeogenic precursors it is no surprise to find that glucose is a more protein-sparing energy source than fat in severely catabolic patients. Wilmore (1977) has shown that it inhibits endogenous protein catabolism by its ability to excite an insulin response. Woolfson, Heatley and Allison (1977) have confirmed the earlier observations of Hinton and colleagues (1971), that
the administration of exogenous insulin with glucose results in greater insulin concentrations and an even greater protein-sparing effect. This has proved particularly useful in burned patients and also in patients with acute renal failure associated with catabolic illness. These therapeutic results are compatible with the hypothesis that one of the main factors mediating the response to injury is the imbalance between the action of the anabolic hormone insulin and the catabolic hormones (see fig. 1). For these reasons we give virtually all the energy in such patients in the form of glucose, giving fat only to prevent fatty acid deficiency and as a vehicle for fat-soluble vitamins. In critically ill patients where insulin resistance and protein catabolism are high it may be necessary to add insulin to maintain the blood glucose between 5 and 10 mmol/litre. The insulin may be either added to the glucose bottle, in which case allowance should be made for a loss of 20% on the glass-ware and tubing, or administered via a separate line using a syringe pump. If 25 g of glucose is being administered per hour, the insulin requirement may vary from zero in non-catabolic patients to 10–20 u./h in severely ill patients. Insulin requirements are determined by repeated urine testing for sugar and by finger-prick blood-sugar estimations using glucose oxidase sticks. This latter technique gives accurate blood-sugar readings between 3 and 20 mmol/litre. It is important to give a high nitrogen intake (12–24 g/24 h) with such a regime, since the potent inhibition of muscle protein catabolism may starve the liver of nitrogen and induce a kwashiorkor type of picture with low serum albumin and oedema. Hypokalaemia and hypophosphataemia may be induced similarly when less than adequate supplements are given (see below).

**Vitamins**

Recent work has emphasized the rapidity with which folate stores may be depleted unless an intake of at least 500 µg daily is maintained (Wardrop et al., 1975). Vitamins C and B may also be used up rapidly. Parenterovite (Bencard Pharmaceuticals Ltd) has been used in the past but it is incomplete and should be superseded by the water-soluble preparation Soluvit and the fat-soluble vitamin additive Lipovit (Kabi Pharmaceuticals Ltd) which will soon be available commercially. The low folate content of Soluvit (200 µg) requires further supplementation.

**Minerals**

With a high carbohydrate intake and the consequent increase in glucose phosphorylation, inorganic phosphate concentrations in the plasma may decrease very rapidly, causing non-specific symptoms of deterioration in the patient which may be ascribed mistakenly to the underlying disease process. Sufficient phosphate is present in Aminosol (18 mmol/litre) and Intralipid (15 mmol/litre) to provide an adequate phosphate intake. The new amino acid preparations do not all contain phosphate, and supplements should therefore be added in the form of sodium or potassium dihydrogen phosphate 20–30 mmol daily. Magnesium depletion may occur, particularly where gastric aspirate or fistula losses are high, and this mineral should be replaced with magnesium chloride or sulphate (maximum 12 mmol/day). Zinc may also be important and the role of other minerals remains to be determined. There are commercially available mineral supplements (for example Addam electrolyte solution, Kabi Pharmaceuticals Ltd) for i.v. use and these should be employed wherever long-term feeding is required.

**Measurements**

**Weight.** Daily weighing gives invaluable information, chiefly about water balance, but is also useful over long periods for assessing the progress of nutrition. In most patients the information derived from weight and plasma sodium concentration gives sufficient information to assess both water and salt balance.

**Inspection of the patient.** This is often forgotten and leads to such errors as the giving of excess salt to an already oedematous patient. In the absence of venous or lymphatic obstruction, dependent oedema must mean an expansion of the interstitial space.

**Haemoglobin and blood film.** These should be examined twice weekly. Marrow depression may occur under the influence of continuing sepsis and a decreasing haemoglobin concentration may be a signal for blood transfusion. On the other hand, low haemoglobin and white counts with thrombocytopenia may signal the advent of folate deficiency. Requirements for iron may also be assessed.
**METABOLIC MANAGEMENT**

Plasma urea and electrolytes. In critical situations these may be required daily, remembering that the figures obtained give information about concentration and, on their own, give little indication of the total body balance of electrolytes. A low serum potassium concentration however, is a good reflection of the inadequacy of potassium supplementation.

**Twenty-four-hour urine collections.** The urine volume may give some indication of hydration although, in the presence of plasma or blood volume deficiency, the patient may be in excessive water and salt balance and yet have a small urine volume. In the presence of oliguria (less than 400 ml/24 h) the urine-to-plasma urea ratio gives useful information about renal concentrating ability. Below a figure of 14 : 1 it may represent intrinsic renal damage (Luke and Kennedy, 1967). However, many critically ill patients have a substantial impairment of concentrating ability and therefore require to produce a greater urine volume to excrete a given urea load.

Urea production rate reflects protein catabolism and allows a crude nitrogen balance to be constructed which is sufficient for most clinical purposes (Lee, 1975). The following formula may be used:

\[
\text{Urine urea (g/24 h) } \times \frac{28}{60} \times \frac{5}{4} \\
\text{(or mol/24 h } \times \frac{28}{5} \times \frac{5}{4})
\]

= nitrogen output (g)

A correction is made for any change in plasma urea as follows:

\[
\text{Change in plasma urea (g/litre) } \times \text{ whole body water (60% body weight) } \times \frac{28}{60}. 
\]

(60 = molecular weight of urea; 28 = weight of two atoms of nitrogen; 5/4 is a correction factor to allow for the fact that urea = 4/5 of the nitrogen excreted.) Alternatively, the change in urea (mmol/litre) × whole body water × 0.028 is the correction needed.

Measurements of urinary sodium and potassium excretion allow balances to be kept of these electrolytes. The ability of the patient to excrete a sodium load may also reflect his clinical status (see below). In patients with magnesium deficiency or hypophosphataemia, the urinary excretion of these minerals gives a useful reflection of the adequacy of replacement, since the kidney controls their plasma concentrations and any spill-over into the urine represents an excessive replacement and hence a safety margin.

Serum albumin. Twice-weekly measurements suffice. Decreasing values must raise the question of whether nutrition is adequate, although it takes up to 3 weeks to increase a low albumin even by intensive feeding. Excessive losses of protein, for example from fistulae, may necessitate plasma transfusion although the effects of this treatment are transitory.

Plasma calcium, phosphate and magnesium. These should be measured twice weekly and with urinary measurements allow assessment of requirements.

Fluid balance. A comprehensive review of this subject is outside the scope of this paper, but one or two aspects are particularly relevant.

Associated with illness there is a tendency to retain salt and water and to excrete potassium (Wilkinson et al., 1949, 1950; Moore, 1959). This is a complex response. Both antidiuretic hormone and aldosterone are involved, but these may not be the most important factors. There may be a redistribution of blood flow between the cortex and medulla of the kidney contributing to sodium retention. This effect is exacerbated by any decrease in renal plasma flow as a result of either deficits of blood, plasma or extracellular fluid volume or any impairment of cardiac output from disease of the heart itself. An increase in potassium excretion is partly related to a mineralocorticoid effect but is largely caused by the release of potassium from cells as protein is catabolized. Nitrogen and potassium excretion therefore tend to run in parallel with each other. Since the provision of water and electrolytes is inseparable from any feeding regime, due consideration should be given to such changes in the patient’s ability to handle these substances.

Extensive discussion has taken place in the literature over the relative advantages of colloid and crystalloid fluids in the management of shock. It is probable that a judicious combination of the two may be correct, consisting for example of plasma and Hartmann’s solution or of blood and saline. However, in prolonged surgical illness, one is sometimes presented with the patient in whom diminished perfusion occurs owing to a decrease in plasma volume (Hinton et al., 1973). Such patients may be oliguric and also oedematous from retained saline, suggesting that the interstitial space is expanded while the plasma volume is depressed. This is analogous to the situation pertaining in the nephrotic syndrome. Such patients require repeated transfusions of plasma proteins or whole blood until sufficient recovery has taken place for the patient to sustain his own plasma volume. A central venous pressure measurement is most useful in diagnosing this problem and monitoring the response to treatment.
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