Acute renal failure following trauma may be divided into two categories: incipient acute renal failure (IARF) and established acute renal failure. The first section of this paper will deal with the principles of diagnosis and reversal of incipient acute renal failure. The second section will deal with the total metabolic management of patients with acute renal failure inclusive of circulatory and respiratory problems. Since the mortality of acute renal failure still remains alarmingly high at greater than 50% in those patients aged more than 50 years (Lindsay, 1974) any improvement in the early reversal of incipient acute renal failure or of the management of established acute renal failure is highly desirable.

**INCIPIENT ACUTE RENAL FAILURE**

**Diagnosis**

The most important consideration here is an awareness of the possibility. Without this alertness, appropriate tests will not be undertaken or their interpretation will be misdirected. In general, it is well to consider the possibility of acute renal failure complicating trauma where there has been significant blood or plasma loss, hypotension, considerable tissue damage or complicating infection.

There are many definitions of acute renal failure, but no one single measurement is sufficient. The following profile of parameters would constitute the best definition of acute renal failure: (a) a urine/plasma (U/P) urea ratio of less than 10 to 1 (Perlmutter et al., 1959); (b) a U/P osmolality of less than 1.1 (Eliahou, 1964); (c) a urine volume of less than 20 ml/h; (d) a urinary sodium of greater than 10 mmol/litre; (e) the exclusion of chronic renal failure or of obstructive uropathy. If these criteria are fulfilled then the clinician should act on the assumption that acute renal failure may already have occurred or will occur unless appropriate measures are taken. Most definitions of incipient acute renal failure refer to this condition occurring within 48 h of the onset incident. It is usually agreed that any attempts at positive reversal after the 48-h interval meet with little success.

It may be difficult to know whether the current situation in a patient represents de novo renal insufficiency or is an acute-on-chronic problem. Attention to the following points will help resolve the problem. If the patient is known to be receiving hypotensive treatment, or to have proteinuria, or have a history of loin pain, dysuria, haematuria, micturition difficulties, nocturia, nausea or vomiting and lethargy, these will suggest previous renal tract pathology. The finding of small renal outlines on nephrotomography or changes suggestive of renal osteodystrophy on a chest x-ray or an e.c.g. showing hypertensive changes or a normochromic normocytic anaemic picture will lead to a similar conclusion. Total anuria always calls for early cystoscopy and ureterograms to ensure patency of the post-renal tract.

However, it must be stressed that where doubts exist it is mandatory that an approach be adopted on the basis that the renal failure is reversible and there should be no delay in obtaining investigations to prove to the contrary. Such delays can lose the opportunity of successfully reversing acute incipient renal failure.

**Factors influencing development of acute renal failure**

The heterogeneity of clinical acute renal failure is highlighted if one considers the following factors which may be involved: water depletion, plasma depletion, blood loss, electrolyte imbalance, the influence of anaesthesia, nephrotoxic agents such as myoglobin after crush injuries or drugs such as cephaloridine and sulphonamides, disseminated intravascular coagulation following post-partum haemorrhage or the haemolytic uraemic syndrome, cardiac arrhythmias and sepsis. Each of these individual problems requires special attention and correction before any specific measures are adopted to reverse incipient acute renal failure.

It may be very difficult to distinguish between the normal oliguric response to anaesthesia and that resulting from circulatory insufficiency related to trauma. Anaesthesia may lead to oliguria by physiological responses such as diminished renal perfusion,
diminished glomerular filtration rate, increased tubular reabsorption and increased activity of ADH and aldosterone, both of which reduce urine volume (Mielke and Kirklin, 1966). The two conditions can merge imperceptibly, but corrections of deficits and adequate monitoring will help distinguish between circulatory inadequacy and intrinsic renal changes. Enthusiasm at ensuring adequate repletion should not lead to overcorrection and its attendant dangers. Monitoring should include observations of arterial pressure, heart rate, jugular venous pressure (or central venous pressure measurements), auscultation of the lungs, measurements of electrolytes, total proteins and haematocrit.

It must be recognized that in acute renal failure not all nephrons are equally involved or ablated. In the surviving nephrons normal pharmacological responses, such as an increased urine flow following a given challenge (for example a diuretic), will continue. Although the urine flow may increase compensatorily in some individual nephrons, this will not prevent the increase in blood urea nitrogen as a result of an overall diminished urinary concentrating ability. One factor which is often overlooked is that even with active repletion measures in a patient considered to have incipient acute renal failure, there may be a delay of up to 6 h before a diuresis occurs following such repletion. Therefore any responses following other specific measures adopted within this interval, for example use of diuretics, must be interpreted with caution with respect to their activity in reversing incipient acute renal failure.

**Treatment of IARF**

If all the repletion measures have been completed, the time interval allowed, and there is still no increase in urine volume, it is reasonable to try the specific use of diuretic agents to promote a urine flow. It is emphasized that the use of any of the diuretics has as its main objective simply an increased urine flow. Whatever the results of earlier measurements which indicate incipient acute renal failure, they will not be immediately reversed by the exhibition of diuretics. The urine flow alone changes (Luke and Kennedy, 1967). The diuretics most favoured are the osmotic diuretic, mannitol, and the loop diuretics, frusemide and ethacrynic acid. In my opinion mannitol alone should be considered for this purpose. Investigations with mannitol have shown it to have the following properties in both animals and man: (1) it acts as an osmotic diuretic, (2) the para-amino-hippurate clearance is increased, (3) there is a transitory increase in circulating blood volume and (4) there are cumulative effects in the extracellular fluid compartment if excessive doses are given and not excreted. Frusemide has been shown to be a naturietic, to dilate the renal vasculature, to increase blood flow through the kidney, to increase renal cortical blood flow, possibly to stimulate the intrinsic renin-angiotensin system of the kidney and finally to have a possible synergism with some aminoglycosides such as gentamycin which can augment the possible serious side-effects, for example ototoxicity.

The effects of frusemide may be dependent upon whether the acute renal failure has a toxic or a circulatory aetiology. In the former there is some evidence to show that it may have a beneficial effect, whilst in the latter it does not (Bailey et al., 1973). Ethacrynic acid shares many of the same properties as frusemide in this situation (Kjellstrand, 1973). Some of the earlier reports claiming success for frusemide suffer from the disadvantages that incipient and established acute renal failure patients were not clearly demarcated and a comparison between patients treated with frusemide and others was made in successive groups often spanning several years (Cantarovich et al., 1973). It is well known that the pattern of admission of patients with acute renal failure has changed considerably over the past 10 years compared with the previous decade (Stott et al., 1972).

When mannitol is to be used, it is recommended to use it in the following way (Barry, Mazze and Schwartz, 1964; Eliahou and Bata, 1965). First of all an i.v. bolus of mannitol 25 g (preferably 25% solution) is infused. For this protocol it is necessary to catheterize the bladder under strict asepsis. If, during the next 2 h, a urine volume of greater than 50 ml/h is achieved, this can be considered as a positive response and the usual management of circulatory volume, fluid and electrolytes maintenance adopted. If after 2 h no response has been obtained, a further 25-g bolus of mannitol may be given and a further 2-h period monitored to see if a urine volume of greater than 50 ml can be achieved. These mannitol loads should not be exceeded and the use of hypertonic mannitol solutions as recommended prevents the danger of fluid overload. Mannitol is not metabolized, so if an excess is given and renal function not re-established it will accumulate in the extracellular fluid compartment (e.c.f.) and cause water to move from the intracellular fluid compartment into the e.c.f. (Mawer and Lee, 1968). This e.c.f. expansion
RENAL FAILURE FOLLOWING TRAUMA

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can cause congestive cardiac failure in elderly patients with an already compromised renal function.

If after a total of 4 h no positive response has been achieved with mannitol, then one dose of frusemide 0.25-0.5 g in 100 ml of 5% dextrose infused over 20 min may be tried. Alternatively, ethacrynic acid 50 mg i.v. may be given. Such trials with these diuretic agents must only be undertaken after full repletion protocols have been completed. The role of beta blockade in the management of incipient acute renal failure in man awaits further evaluation, although there is early evidence to suggest that propranolol may inhibit the renin–angiotensin II mechanism (Iaina, Solomon and Eliahou, 1975).

The prophylactic use of mannitol may need to be considered in patients treated for trauma. Its use in this way has been found valuable in preventing the incidence of acute renal failure in patients undergoing cross-clamping operations of the abdominal aorta (Barry et al., 1961) and in patients undergoing surgery on the biliary tract or who are already jaundiced (Dawson, 1964, 1968). It is recommended that 5% mannitol infusion is begun before operation and continued for a period of 12 h after the operation period and that the infusion rate is adjusted to maintain a urine flow of more than 40 ml/h.

Conditions that may be confused with acute renal failure

Elderly patients often become azotaemic following trauma or operations, whilst not becoming oliguric. They may have a degree of associated acidemia although they are rarely severely hyperkalaemic. In such patients it must be appreciated that, with the ageing process of the kidney, the solute excretory capacity diminishes (that is, there is a diminished urinary concentrating ability) and thus any sudden increase in metabolic load must be associated with retention and altered blood concentrations unless there is a dramatic increase in urine volume. If the usual solute load presented to the kidneys is doubled, then double the usual urine volume is required at the same urine concentration if no evidence of retention is to occur. Where the urine volume does increase appropriately, unless adequate measures are taken to maintain fluid balance, dehydration will occur because of the urea-induced diuresis.

Other conditions may lead to an increased plasma urea to creatinine ratio (normal range 20-40) without necessarily implying acute renal failure. These include conditions with increased protein catabolism such as the high metabolic rate associated with trauma or fever, the effect of steroids, or the increased ureagenesis associated with gastrointestinal bleeding or tetracycline therapy. Salt and water depletion in cases of salt wasting nephropathy, or with large gastrointestinal losses or following over-zealous use of diuretics may lead to a similar divergence of the urea/creatinine ratio. Finally, over-enthusiastic use of high protein feeds with inadequate water allowance can lead to a similar result in elderly patients.

Established acute renal failure

Metabolic response

The metabolic response to trauma, infection and surgery in acute renal failure patients is exactly the same as in their non-renal failure counterparts (Moore and Ball, 1952; Moore, 1959). Thus, trauma is followed by a catabolic response characterized by ureagenesis and production of excess potassium and hydrogen ions from dissolution of cell mass and early retention of salt and water as a result of an altered hormonal environment. Whereas the non-renal failure patient can excrete the products of this metabolic response to trauma, in patients with renal failure these accumulate with the occurrence of azotaemia, hyperkalaemia and metabolic acidosis, together with fluid retention and consequent pulmonary oedema if allowed to progress far enough.

The commonest cause of death in acute renal failure patients remains sepsis (Montgomerie, Kalmanson and Guze, 1968). This is particularly relevant to intra-abdominal operations and in the elderly. The relationship between malnutrition and impaired cellular and humoral immunocompetence has long been recognized (Bistrian et al., 1975). It is perhaps not surprising, therefore, that much of the morbidity and mortality of patients suffering from acute renal failure may result from inadequate metabolic care and, in particular, poor nutrition in their management. Whilst attention to haemoglobin and fluid requirements, respiratory, bowel and skin care and other management may be excellent, often the nutritional requirements of these patients are not met.

The consequences of negative nitrogen balance are the same in patients with acute renal failure as in others and are associated with a decrease in body weight, increased susceptibility to infection, decreased immunocompetence, failed rate of wound healing, hypoproteinaemic oedema, apathy and a prolonged convalescence. It should be customary in such patients to assess not only their biochemical and fluid environment, their circulatory and respiratory status, but also their nutritional status (Mickelsen, Shier and Narins, 1975; Blackburn et al., 1976). The latter can
be best done by: clinical assessment, recording body weight, measuring skin fold thickness as an indication of energy reserves, measuring mid-arm circumference as an indicator of muscle protein mass, measuring the serum short half-life proteins, such as transferrin and complement C₃ (Schaeffer et al., 1975), assessing skin anergy, for example to candida antigen, a lymphocyte count (although this is not very reliable in this setting), and, when possible, amino acid profiles (which are probably a better indicator in chronic renal failure). The nutrition requirements of patients with acute renal failure can be met by oral, nasogastric or i.v. feeding. Undoubtedly, where possible, the oral route is the favoured method, but if a patient is seen not to be willing or able to eat, there should be no hesitation in reverting to the other routes. It must be emphasized that any attempt at controlling the nutritional self-cannibalism of acute renal failure and the disintegration of the patient is not just an attempt to achieve a normal blood urea concentration.

Metabolic and nutritional requirements

It is helpful to think of patients with acute renal failure as falling into different categories of catabolism which will give some indication of the metabolic and dialysis management. Patients in group 1 may be considered as those with modest catabolism associated with a blood urea increase of 4–6 mmol/day (10–14 g nitrogen breakdown); group 2 patients are those with moderately severe catabolism associated with a daily blood urea increase of 6–10 mmol (breakdown of 14–24 g nitrogen) and group 3 comprises those with severely catabolic (hypercatabolic—Silva et al., 1964) renal failure, who have blood urea increases of 10–12 mmol/day related to a daily nitrogen breakdown of 24 g or more. For group 1 patients it is reasonable to adopt a protein-restrictive regimen, whereas for groups 2 and 3 it is preferable to feed normally and to dialyse accordingly (Lee et al., 1968; Lee, 1975, 1976). Protein-restricted regimens provide 0.3 g of high biological value (egg or meat based) protein/kg body weight per day together with 45 kcal (189 kJ). Hycal (Beecham’s products), a liquid dextrose concentrate (1 bottle contains 475 kcal (2 MJ)/106 ml water), and Caloreen (Hospital Scientific Supplies Ltd), a glucose polymer, are useful energy sources. The sodium content should be kept around 20 mmol/day and the potassium content below 40 mmol. Fluid restriction should be 500 ml daily plus the addition of any previous day’s water losses, such as vomit, diarrhoea, urine, nasogastric aspiration, entero-cutaneous fistula losses. An allowance for the water of metabolism which amounts to 0.5 litre/day must be made in calculating the requirements of such patients otherwise fluid retention can rapidly occur over the course of 1 week. In group 1 patients treated by low protein diets there is no evidence that any nutritionally significant urea nitrogen recycling occurs (Walser, 1974; Richards, 1975; Varcoe et al., 1975). This is even debatable in chronic renal failure. Any decrease in the rate of increase of blood urea is not caused by recycling but by a diminished catabolic rate. Daily biochemical investigations are important (for example blood urea, sodium, potassium, bicarbonate, phosphate, creatinine, albumin, haemoglobin, packed cell volume, WBC), for in this way one can predict in the absence of increasing urine volume when the patient’s blood parameters will reach certain concentrations and hence when dialysis will be required. Careful daily fluid balance and weight charts must be kept so that appropriate adjustments can be made for alterations in the patient’s metabolism.

For patients in groups 2 and 3 there should be no hesitation in implementing a full nutritional regimen inclusive of vitamins, essential biological elements and water and using dialysis as required. There seems little reason to adopt the reverse attitude of trying to adapt the patient’s management to dialysis requirements.

Use of diuretics

Earlier reports (Cantarovich et al., 1973) recommended the use of diuretics in the management of established acute renal failure as a way of improving the overall management and diminishing the need for dialysis. Such early claims have not been confirmed by more recent reports (Kleinknecht et al., 1976). Any such consideration must take into account the daily urine volume, the urine urea concentration, the rate of increase of blood urea, the incidence of hyperkalaemia, what other supportive therapies are required and the risk of any toxic effects from other associated drug therapy in the face of compromised renal function. It is relatively simple to gauge the efficacy of possible diuretic-managed acute renal failure by consideration of the osmotic excretory requirements. If, for example, a 70-kg patient has a daily blood urea increase of 6 mmol/litre this would give a total daily blood urea increment of 252 mmol. This arises because the blood urea is equally distributed throughout the total body water which can be taken to be 60% of total body weight. If, in such a patient, the urine volume was 0.5 litre/day with a urine urea con-
centration of 100 mmol/litre then only 50 mmol/day would be excreted. In such a patient on diuretic therapy there would need to be an increased urine volume of 2.5 litre/day to hold down the rate of increase of blood urea. It is readily appreciated how impossible such tasks would be when treating patients with hypercatabolic acute renal failure, who may have blood urea increases of 12-15 mmol/day. Furthermore, the use of large doses of diuretics on a daily basis in these patients is not without the risk of toxic effects such as ototoxicity (Kleinknecht et al., 1976).

**Dialysis**

The indications for dialysis are shown in table I. Once patients are being dialysed these techniques impose further nutritional losses on them. With peritoneal dialysis daily losses of 20-30 g of protein per 40-litre dialysis and 13-15 g of amino acids are incurred (Berlyne et al., 1967). With haemodialysis 2-3 g of amino acids are lost per hour of dialysis (Heidland and Kult, 1975). With both methods, losses of water-soluble vitamins and essential biological elements (for example copper and zinc) are incurred. Compensation for the amino acid dialysis losses can be made either by adding 10 ml/litre of peritoneal dialysis fluid of Vamin N (Kabi Pharmaceuticals), which is equivalent to 94 mg of amino acid nitrogen/litre or washing back with Vamin N 500 ml (4.7 g nitrogen or 35 g amino acids) over the last 3 h of haemodialysis.

Which method of dialysis is used will depend on both facilities available and individual patient requirements. For the more severely catabolic patients, for those with abdominal injuries and those requiring urgent biochemical correction there can be little doubt that frequent haemodialysis is the most advantageous (Silva et al., 1964). With this technique dialysis time is foreshortened, rapid fluid adjustments can be made and less interference with a patient’s physiotherapy occurs. Furthermore, not only are the nutritional losses increased less by dialysis but the risk of pulmonary complication is decreased (Berlyne et al., 1966). For paediatric practice, peritoneal dialysis has much to recommend it as it requires far less expertise (Lee, 1967). Peritoneal dialysis can also be set up immediately and occasionally a situation will arise where, for expediency, peritoneal dialysis will be used first, for example treatment of hyperkalaemia and subsequently haemodialysis. Peritoneal dialysis may be specifically indicated in patients with heart failure or those with haemorrhage and when no blood is immediately available. Both techniques have their complications (Lee, 1967; Jones, 1971); some are shared by both techniques, for example amino acid losses, and some are specific to haemodialysis, such as the disequilibrium syndrome affecting brain and heart function. Others more relevant to peritoneal dialysis are respiratory difficulties, dissection of fluid into abdominal wall layers and patient discomfort. Sudden changes in circulating blood volume may occur with haemodialysis and one must be wary of the dialysance of therapeutic agents such as antibiotics and therefore loss of adequate blood concentrations as well as potential toxic effects arising from the particular drugs used with the change in biochemical environment, for example digoxin toxicity following hypokalaemia after dialysis.

It is important to start dialysis early and not delay until values become too great, for example blood urea 80 mmol/litre, serum potassium 7 mmol/litre and serum bicarbonate 10 mmol/litre. The emphasis must be on prophylactic haemodialysis or peritoneal dialysis which yields much better results (Whelton and Donadio, 1969).

**Hyperkalaemia**

This is the most serious immediate life-threatening metabolic consequence of acute renal failure. Its occurrence demands immediate treatment whilst the more specific approach of dialysis is being prepared. The emergency measures include glucose–insulin regimens (glucose 50 g i.v. with 15 units of soluble insulin); 8.4% sodium bicarbonate solution 100 ml i.v. may also be given. Glucose–insulin regimens are temporary measures only and are not a substitute for
dialysis. Calcium resins 30–60 g daily in divided doses orally or rectally may be given to control hyperkalaemia between dialyses. They are not effective as emergency measures.

**Intravenous nutrition**

Since many hypercatabolic patients with acute renal failure have associated gastrointestinal failure, complete parenteral nutrition using a crystalline full profile (essential and non-essential) amino acid solution with glucose and insulin or glucose, insulin and a soya bean oil emulsion (Intralipid) should be used. There is no evidence to show that the use of fat emulsions as part of the energy substrate interferes with dialysis membrane characteristics (Lee, Sharpstone and Ames, 1967). An example of such a regimen in a modestly hypercatabolic patient is shown in table II. Such regimens have been shown to be associated with increased patient survival, increased sense of well-being, less cardiotoxicity, decreased rate of increase of blood urea, diminished incidence of hyperkalaemia and possibly a decreased duration of acute renal failure (Abel et al., 1973; Abel, 1976).

Glucose-insulin-amino acid regimens may be particularly valuable by increasing the interval between dialyses. Large doses of soluble insulin (200–400 U./day) may be required initially. Later, as the metabolic state becomes more stable, the daily insulin requirement decreases. Initially with such regimes frequent blood-sugar checks are required. This can be conveniently done by using a bedside Ames meter (which can be operated by nurses) and sufficient insulin used to keep the blood-sugar at 10 mmol/litre. It is usual to use a 50% glucose solution with a basic addition of 120 units of soluble insulin, with potassium (depending on potassium status) of 40–80 mmol/litre. This is then usually given as 1-hourly, 50-ml boluses or as a constant infusion if a pump is available. A "metriset" is required for the bolus method. In the interest of keeping the i.v. volume to a minimum and similarly the insulin requirements, it is convenient to use energy derived on a 50–50 basis from glucose and a fat emulsion (Intralipid 20%, Kabi Pharmaceuticals). Checks should be made that the fat emulsion is being utilized (Lee, 1976). By frequent dialysis which keeps the internal environment near normal the energy substrates are normally metabolized. If parenteral nutrition is to be used in such patients the utmost care with catheter management is mandatory. The percutaneous infraclavicular subclavian vein technique is the preferred route.

**Other considerations**

Patients with acute renal failure often have more than one end-organ failure, for example lung (shock lung) (Parsons, 1974; Stoddart, 1974), gastrointestinal failure and liver failure or both. Furthermore, many of these patients may require interim surgery such as amputation of a gangrenous leg or general debridement. The attitude should never prevail that operation should be delayed in such cases until renal function has been re-established. On the contrary, it has been shown many times that removal of a potential source of toxins or infectious focus is imperative before renal function will return. Many of these patients have complications such as disseminated intravascular coagulation (DIC). Here it is important to try to define what is the cause of the DIC, such as infection (Wardle, 1975), and to seek a possible focus of infection and eradicate it either by surgical or antibiotic means. Secondary endotoxaemia may be the link between infection and DIC and although endotoxaemia is difficult to diagnose (for example the Limulus test) and to obviate (because there is no specific antiserum), the search for and eradication of infection is mandatory.

Many drugs (O'Grady, 1971) used in severely injured patients or after major operations will have to be modified in the patient with acute renal failure because the route of excretion is impaired. Some of the more important drugs which require modification are shown in table III.

### Table II. Suggested i.v. regimen in a patient with acute renal failure and modest catabolism and gastrointestinal failure

<table>
<thead>
<tr>
<th>Solution</th>
<th>Volume (litre)</th>
<th>Energy (kcal)</th>
<th>Carbo</th>
<th>Fat</th>
<th>Nitrogen*</th>
</tr>
</thead>
<tbody>
<tr>
<td>50% Glucose†</td>
<td>0.5</td>
<td>1000</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>20% Intralipid</td>
<td>0.5</td>
<td>—</td>
<td>1000</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Vamin-glucose</td>
<td>1.0</td>
<td>400</td>
<td>—</td>
<td>—</td>
<td>9.4</td>
</tr>
<tr>
<td>Boots phosphate§</td>
<td>0.25</td>
<td></td>
<td></td>
<td>—</td>
<td>9.4†</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>2.25</strong></td>
<td><strong>2400</strong></td>
<td></td>
<td></td>
<td><strong>9.4†</strong></td>
</tr>
</tbody>
</table>

Notes: (i) Provides 90 mmol sodium and 25 mmol potassium; (ii) provides 32.5 mmol phosphorous (7.5 mmol from Intralipid); (iii) add water-soluble vitamins and essential biological elements (Addam solution, Kabi Pharmaceuticals). * May require considerably more if associated large entero-cutaneous fistula losses. † May need to add soluble insulin; if all energy derived from glucose, patients will need insulin. ‡ Equivalent to 58.8 g protein. § Requirements increase as patient becomes more anabolic.
### TABLE III. Modification of some drug dose schedules in patients with acute renal failure

<table>
<thead>
<tr>
<th>(1) Antibiotics</th>
<th>Maintenance</th>
</tr>
</thead>
<tbody>
<tr>
<td>(a) Gentamycin, kanamycin, streptomycin, colistin.</td>
<td>Large reduction; check serum concns</td>
</tr>
<tr>
<td>Normal loading dose</td>
<td></td>
</tr>
<tr>
<td>(b) Ampicillin, flucloxacillin, lincomycin, clindamycin, benzylpenicillin.</td>
<td>Usually half normal; depends on frequency of dialysis</td>
</tr>
<tr>
<td>Normal loading dose</td>
<td></td>
</tr>
<tr>
<td>(c) Fusidic acid, nalidixic acid, erythromycin, carbenicillin, doxycycline, cotrimoxazole</td>
<td>Normal</td>
</tr>
</tbody>
</table>

(Nitrofurantoin, tetracycline, chloramphenicol, amphotericin, the ceporins and sulphonamides are best avoided)

| (2) Immunosuppressants | (a) Steroids | Normal (note effects on urea) |
|------------------------|--------------|
| antimitabolites        | (b) Azathioprine | Half normal |
|                        | cyclophosphamide | |

| (3) Cardiovascular drugs | (a) Digoxin—standard initial dose | ½-¾ normal |
|--------------------------|----------------------------------|
| (b) Methyl dopa         | ½ normal                        |

(β-blockers (e.g. propranolol), diazoxide, hydralazine normal doses)

| (4) Hypnotic and anticonvulsants | Phenobarbitone | ¼ normal |
|----------------------------------|----------------|
|                                  | Butobarbitone |
|                                  | Epanutin       |

(Short-acting barbiturates, diazepam, phenothiazine derivatives no change)

Where patients experience respiratory difficulties there should be no hesitation in putting them onto a ventilator. The management of patients with acute renal failure demands a knowledge of ventilator care and the associated respiratory problems and it is important that either a physician with experience of respiratory disorders or an anaesthetist are co-opted to the management team. There is a similar heterogeneity to the causes of respiratory insufficiency related to renal failure as to the primary condition itself (Moore, 1969). These include effusions, pulmonary infections, excess water and sodium values, myocardial decompensation and impairment of neuromuscular ventilatory function such as may occur after anaesthesia (Smith, 1973). Many of these problems can be corrected by adequate and appropriate dialysis techniques, by the use of antibiotics and by positive end-expiratory pressure ventilation. For the "shock lung syndrome" extra measures inclusive of heparinization and large doses of steroids may be required. These additional treatments will have their own impact on dialysis requirements.

The combination of acute renal failure and rapidly developing jaundice is of grave prognostic significance. There is little merit in the term "hepato-renal syndrome" for it does not define a distinct entity (Sherlock, 1958). It merely indicates (in the absence of actual intervention on the biliary tree) that the severity of the initial ischaemic or anoxic insult following trauma affected two organs. Frequent haemodialysis is indicated in such cases and management becomes compounded by trying to match metabolic requirements to the failure of two end-organs. However, the best chance of survival in these patients is by trying to maintain as near normal a metabolic environment as possible whilst other general measures are undertaken. The use of a specific amino acid solution in this condition (Fischer et al., 1975; Aguirre et al., 1976) is of particular interest.

It cannot be over-emphasized that adequate monitoring is mandatory throughout the management of all patients with acute renal failure so that many of the potential metabolic complications can be avoided, such as hyperglycaemia (corrected by extra insulin), hypophosphataemia during i.v. nutrition or hypalbuminaemia which may require correction by human plasma protein fraction.
The diuretic phase

This is characterized by the daily urine volume exceeding 1000 ml. In the early stages of the diuretic phase the urine is virtually an ultra-filtrate of the plasma. Therefore, dialysis may still be required during this time. There is a variable delay before urine concentrating and acidifying abilities return. Thus, in severely injured patients it may take up to 10 days before the blood urea concentration spontaneously decreases in patients during the diuretic phase. The degree of diuresis varies considerably but may be as high as 5–6 litre/day. This can be a danger period for the patient because either inappropriate attempts are made to replace the fluid losses, or inadequate repletion measures are taken. This diuretic phase, which usually signifies an improvement in renal blood flow and glomerular filtration rate, results from a combination of variable factors. These are (1) the excretion of retained excess fluid and electrolytes, (2) the inability of the renal tubule to concentrate and (3) an osmotic diuresis caused by the persistently high blood urea concentration. Thus if factor (1) is the principal cause no replacement is required, whereas with factors (2) and (3) adequate repletion is mandatory. The duration of the diuretic phase is variable but lasts roughly as long as the preceding oliguric phase.

Prognosis

Most patients who survive the complications of acute renal failure have a return of renal function after 2–4 weeks. When a situation arises in which a patient is now 6 weeks from the onset incident and there has been no resumption of normal renal function, a renal biopsy is indicated. There is no clear-cut division between so-called acute tubular necrosis and bilateral cortical necrosis and one may merge into the other. Renal calcification on a plain x-ray of abdomen may be seen at about 6 weeks, but a biopsy is most important at this stage to arrive at decisions about future management.

It is readily seen that the management of acute renal failure is a team approach and attention must not be focused simply on the kidney. Once acute renal failure is established then the whole of the cardio–respiratory–physiology problems must be considered together with full metabolic management. Dialysis is only part of the overall treatment and not necessarily central to it. It is for this reason that I believe that such patients are best managed in acute renal failure centres or in intensive care units with dialysis facilities which can best cater for the overall needs. Too often the mistake is made that “all will be corrected by peritoneal dialysis”. Only those centres which have considerable experience of acute renal failure should attempt haemodialysis.

CONCLUSION

It is still a great disappointment that, after the efforts of teams working in this field, so many patients still die (Stott et al., 1972; Lindsay, 1974). However, if all the circumstances are considered and all the permutations of treatment adopted, it seems reasonable to assume that an increased survival can be anticipated. However, since the criteria for the treatment of acute renal failure have widened and since the indications for aggressive surgery have enlarged, particularly in the elderly, it seems inevitable that there will always be a certain hardcore of patients beyond salvation by modern means.

REFERENCES

RENAL FAILURE FOLLOWING TRAUMA


