POST-TRAUMATIC ACUTE RENAL FAILURE

P. R. ULDALL AND D. N. S. KERR

While in the last few years the incidence of acute renal failure from obstetric causes has steadily declined, that of renal failure attributable to accidental trauma has remained static (Kerr and Elliott, 1970). Presumably the effects of better management of shock have been counterbalanced by the increasing toll from road accidents. Over the same period the mortality of acute renal failure due to obstetric and medical causes has declined but the mortality from surgical and post-traumatic acute renal failure has remained high. No large series has indicated a mortality rate in these groups appreciably below 50 per cent (Flynn, 1970; Kerr and Elliott, 1970; Marshall, 1971). This situation has apparently resisted the impact of improved renal facilities and the widespread development of excellent up-to-date intensive therapy units.

Patients who survive trauma long enough to require treatment for acute renal failure die mainly from non-renal complications such as infection, pulmonary embolism, jaundice and anoxic cerebral injury. Deaths from such preventable causes as hyperkalaemia, overhydration and uraemia are rightly becoming uncommon. None the less there is scope for improvement in renal management.

Resistance to infection is probably impaired by levels of uraemia which have been regarded as acceptable in the recent past, and infection has been the most important cause of death in our experience (Kerr, Rabindranath and Elliott, 1968).

MECHANISMS OF THE RENAL DAMAGE

(1) Severe trauma is nearly always associated with shock and therefore a variable period of poor tissue perfusion. The mechanism by which temporary renal ischaemia produces acute renal failure is still a field for lively dispute. The most popular postulates are

(a) prolonged renal vasoconstriction beyond the period of hypotension;
(b) intrarenal shunting or maldistribution of blood between cortex and medulla;
(c) occlusion of nephrons by raised intrarenal pressure due to oedema;
(d) obstruction of tubules by casts, desquamated epithelium, and cellular swelling;
(e) leakage of tubular fluid into the interstitium through ruptures in the basement membrane;
(f) disseminated intravascular coagulation causing glomerular occlusion and further ischaemic damage in the postglomerular circulation (Schreiner, 1967; Clarkson et al., 1970).

Mechanism (d) has been implicated particularly in acute renal failure following trauma. When there is extensive skeletal muscle necrosis it is usual to observe numerous myoglobin casts in the urine as well as in the tubules in renal biopsy material. Cast obstruction of tubules by certain mucoproteins undoubtedly occurs (Patel, McKenzie and McQueen, 1964), but may be less important than the possible toxic effects on tubules of muscle breakdown products, some of which may be much more damaging than myoglobin. In other situations (e.g. unaccustomed exercise) muscle necrosis can cause acute renal failure without the intervention of shock (Jackson, 1970; Schrier et al., 1970).

(2) Severe injuries may necessitate massive emergency transfusion with blood and blood substitutes. The limited time for cross-matching must increase the risk of infusing incompatible blood; the formation of a complex of antibody with the antigen on the red cell membrane causes shock, widespread vascular damage and acute renal failure (Schmidt and Holland, 1967). If a large incompatible transfusion is recognized early it should probably be treated by exchange transfusions to prevent the prolonged profound shock that often develops and which is not benefited by haemodialysis.

If given in excessive dosage, or in normal doses to oliguric patients, low molecular weight dextran may occasionally cause additional renal damage. Renal biopsy shows dramatic swelling of the proximal tubular cells—an "osmotic nephrosis" (Mailloux et al., 1967; Kerr and Elliott, 1970). Whether this lesion is important in precipitating or prolonging acute renal failure in shocked patients remains in
some doubt but there is general agreement that low molecular weight dextran should now be avoided in the presence of oliguria (Matheson and Diomi, 1970).

(3) Many cases of severe trauma are associated at some stage with gram-negative septicemia (Marshall, 1971), in which endotoxins inflict renal damage by way of diffuse intravascular coagulation (Corrigan, Ray and May, 1968) as well as by way of their sympathomimetic effect in producing vasoconstriction in intrarenal arterioles (Gilbert, 1960; Lillehei, Longerbeam and Bloch, 1963; Martin, Carriissi and Picken, 1965).

(4) Massive trauma is often associated with the development of jaundice which appears from biochemical indications to be mainly obstructive in nature, but is rarely found to have an obstructive cause (Nunes, Blaisdell and Margaretten, 1970). This liver injury can itself induce an impairment of renal function by way of intense renal vasoconstriction (Kew et al., 1971) which may be temporary and reversible provided that the hepatic injury recovers. Obstructive jaundice has also been shown experimentally to render an animal highly susceptible to widespread intravascular coagulation when exposed to a single dose of bacterial endotoxin (Wardle and Wright, 1970).

Clinically, Dawson (1965) demonstrated a greatly increased incidence of acute renal failure following operations performed in the presence of obstructive jaundice and showed that this could be largely prevented by the use of mannitol during the operation. It therefore seems logical to administer mannitol to the traumatized patient who is still passing urine at the time when jaundice develops.

(5) Fat embolism is often associated with acute renal failure. Whether this is a coincidence reflecting the fact that both follow severe trauma, or whether the fat embolism causes the renal failure is uncertain. However, there is suggestive evidence that fat embolism may precipitate disseminated intravascular coagulation and thus produce renal failure (Svane, 1971).

A striking change has occurred in the clinical picture of post-traumatic acute renal failure seen in battle casualties (Whelton and Donadio, 1969). The majority of patients in the Korean war had acute renal failure starting a few hours after injury and due to untreated shock. Rapid evacuation and early transfusion in the Vietnam war has largely eliminated this problem; the patients seen in recent years have acute renal failure starting some days or weeks after trauma as a result of infection and other complications, with a more prolonged course and a poorer prognosis. The same trends can be discerned in civilian practice in the 1950s, '60s and '70s.

**DIAGNOSIS OF ACUTE RENAL FAILURE**

Diagnosis of renal failure depends mainly on remaining alert to the possible conditions in which it may occur. Any patient whose injuries are severe enough to warrant hospital admission should remain under suspicion, whether or not there has been an episode of shock or hypotension. Careful recording of fluid intake and output is a basic essential, but an apparently adequate urine volume may disguise the small proportion of cases of acute tubular necrosis which are non-oliguric. The other essential screening tests are daily estimation of blood urea and electrolytes (Bates, Pigott and Stableforth, 1970) and examination of the urine for protein.

Oliguria from prerenal causes will be associated with a high urine osmolality (>700 mOsm/kg) and high urine urea concentration (>1000 mg per 100 ml) as well as a low urine sodium concentration (<30 m.equiv/l). It should respond to appropriate and adequate fluid replacement guided by central venous pressure monitoring, perhaps assisted by intravenous mannitol infusion, 25 g being a standard dose for an average adult (Luke and Kennedy, 1967).

Conversely established intrinsic renal failure will be associated in most cases with a low urine osmolality and urea concentration and a high sodium concentration. It is, however, unwise to place too much reliance on urine/blood urea ratios in distinguishing between prerenal and intrinsic renal failure (Chisholm, Charlton and Orr, 1966). If after adequate volume replacement and a diagnostic trial of mannitol there is no increase in urine output, and if at the same time the urine contains protein and numerous casts, established renal failure can be assumed.

The presence of complete anuria as opposed to oliguria should make one think of vascular accidents to the kidneys (Grabowsky et al., 1970; Ross, Ackerman and Pierce, 1970; Kerr, 1971) or obstruction in the urinary tract. An isotope renogram is capable of detecting the presence or absence of a vascularized kidney on either side but cannot be relied on to distinguish between obstruction and acute tubular necrosis (Mayo et al., 1971). High-dose intravenous pyelograms, on the other hand, can quite frequently and reliably make this distinction (Brown et al., 1970). In cases of doubt one may have to resort to the special techniques of renal
biopsy, retrograde pyelography and renal angiography.

PREVENTION OF RENAL FAILURE

The anaesthetist, casualty officer, or anyone involved in the emergency treatment of injured patients has a crucial role in trying to prevent the onset of renal failure by quick and effective resuscitation procedures. Provision of an adequate airway and early as well as adequate blood volume replacement are the first essentials. Insertion of a central venous pressure cannula is an early priority as a guide to volume replacement (Riordan, McLay and Walters, 1969). Volume expansion is more urgent than provision of red cells. Of the various forms of plasma, pasteurized plasma protein solution is the only one completely free of the risk of inducing serum hepatitis. Of the plasma substitutes dextran 70 has the advantage of producing a reliable volume-expanding effect for many hours and it also has marked antithrombotic properties (Gruber, 1970). Since little of the larger molecular weight dextran is filtered at the glomerulus it is much less likely to cause osmotic nephrosis than is low molecular weight dextran.

Arterial oxygen desaturation is a notoriously difficult finding to be sure of by clinical examination. Blood-gas analysis should be routinely performed at an early stage in all seriously injured patients so that assisted ventilation can be provided if necessary. The use of sympathomimetic or vasopressor drugs has been seriously brought into question in the last few years because in shock states they will further reduce already critically inadequate tissue (and kidney) perfusion. At slow flow rates blood is more viscous and hypercoagulable so that intravascular clotting will tend to occur in the microcirculation.

Since the mechanisms of shock have become better understood it has been realized that it has been more logical to reduce peripheral resistance than to increase it. For this purpose phenoxybenzamine has been found most effective by producing an adrenergic blockade. When administered intravenously in a dose of 1 mg/kg body weight over about an hour its effect becomes maximal about an hour later and lasts for several hours. Administration is probably unwise in patients who are already vasodilated (Fromm and Wilson, 1969), or in patients whose blood volume has not been adequately replaced.

Digitalis may be indicated in cases where the c.v.p. is high in spite of hypotension—indicating primary myocardial failure. But once renal failure is established digitalis is regarded by most authorities as an unacceptable risk. This is partly because of the danger of toxicity from inadequate renal excretion but mainly because fluctuations in serum potassium in renal failure may cause fatal arrhythmias in digitalized patients.

Finally it should be a golden rule to suspect the presence of gram-negative septicaemia when the cause of shock is obscure. Blood cultures should always be taken before starting antibiotics.

MANAGEMENT OF THE RENAL FAILURE

Preparation for transfer.

Once renal failure is established, or even strongly suspected, any delay in contacting a renal unit with haemodialysis facilities is dangerous. Dialysis may be needed quickly because of the highly catabolic state of seriously injured patients. It also helps the renal unit to plan its work ahead if the members are informed about patients early enough to allow treatment to be elective rather than on an emergency basis. Peritoneal dialysis can often control the plasma urea level in patients with a high rate of urea production (Cameron, Ogg and Trounce, 1967) and is useful as a temporary alternative to haemodialysis if there is a particular risk of haemorrhage. However, it does not control the serum creatinine level, which may be a better index of clinical uraemia, has not been shown to be as effective as haemodialysis in prolonging survival in hypercatabolic patients, and it is contraindicated in the presence of recent abdominal injuries including retroperitoneal haematoma. If peritoneal dialysis is used, therefore, it should be in a unit where haemodialysis can be substituted at any time.

The commonest biochemical danger requiring correction, before transfer of the patient, is hyperkalaemia. If the patient has a non-respiratory acidosis, administration of an appropriate dose of sodium bicarbonate (usually 100–300 milliequivalents over 2–4 hours) is effective in correcting the hyperkalaemia by causing potassium to move into cells. Glucose (50 g i.v.) and insulin (10–20 units i.v.) similarly produce a rapid lowering of plasma potassium by transfer into cells. Perhaps surprisingly, potassium has less effect on the myocardium when intracellular and immediate improvement in the e.g. follows these manoeuvres (Douglas and Kerr, 1977). An additional immediate safeguard is the administration of calcium gluconate (10 ml of a 10 per cent solution) intravenously, since calcium
has antagonistic effects on the myocardium to those of potassium. However, these three emergency procedures all have shortlived effects and should be backed up at once by the use of an ion exchange resin. The sodium phase (Resonium A) is most rapidly effective and should be given rectally in a dose of 30–60 g. Oral administration is often dangerous in the seriously injured because of the risk of vomiting. Inhalation of vomitus is a prominent cause of death in these patients and in many cases could have been prevented by earlier use of nasogastric intubation. If rectal administration is impossible because of bowel injury, 30 g Resonium A can be mixed with 40 ml 20 per cent sorbitol and fed down the nasogastric tube in small aliquots over a couple of hours.

The emergency treatment of hyperkalaemia buys time for rapid transfer of the patient and the institution of haemodialysis. This is now performed, almost invariably, through a subcutaneous shunt placed in the forearm which facilitates frequent dialysis. However, in traumatized patients, with their hypercoagulable state, these shunts do not remain patent as long as they do in the well managed patient in chronic renal failure. It is quite often necessary to move the shunt site at least once in a prolonged episode of acute renal failure, when some of the extremities may be covered in plaster. The preservation of peripheral veins is therefore vital; “cutting down” should be avoided at all costs. Subclavian or internal jugular cannulae (Jernigan et al., 1970; Latimer, 1971) are ideal since they can be used for c.v.p. measurement, they leave the forearms and legs unencumbered and if peripheral veins are not available they can be used temporarily as the return site for haemodialysis. However, cannulae should be removed as soon as they cease to be essential, to avoid the risk of bacterial colonization and septicaemia. Once daily haemodialysis has been established it is sometimes possible to give all the fluid requirements into the circuit of the dialyzer.

**General medical care.**

The ideal place to manage the patient with multiple injuries is in an intensive therapy unit with facilities for haemodialysis, where one can call on the skill of several disciplines simultaneously. One of the difficulties is in avoiding treatment by committee. Adequate communication between different members of the medical staff becomes very important and is usually best achieved by having an agreed time (or times) each day for visiting the patients. One member of the junior hospital staff should be designated the “final common path” for all recommendations on treatment; in a large ITU the obvious choice is the SHO/Registrar on standby duty, who must therefore be well instructed in the management of renal failure even if his initial loyalty is to anaesthetics or surgery.

The combination of chest injury and acute renal failure is a particularly difficult challenge (Kennedy et al., 1963). Uraemia causes increased pulmonary capillary permeability (Gibson, 1966) and this is probably a factor in the causation of pulmonary oedema in patients with renal failure, though the more important cause is overhydration during the oliguric and early diuretic phases. The reduced resistance to infection produced by acute renal failure calls for particularly scrupulous asepsis in managing a tracheostomy. Acute uraemia produces a striking reduction in salivary flow which is probably mirrored by a reduction in bronchial secretions. Accumulation of inspissated mucus in the respiratory passages has led to fatal obstruction (Holmes et al., 1960). Humidification of the inspired air is therefore mandatory.

Extra care is required in renal failure patients who require a general anaesthetic. If such a person has a non-respiratory acidosis the compensatory hyperventilation should be maintained or a rise in $P_{CO_2}$ may cause a fall in blood pH with a consequent shift of potassium from the cells to the extracellular fluid; a fatal rise in serum potassium may occur (Goggin and Joekes, 1971). Prior correction of the non-respiratory acidosis is clearly desirable if possible.

Uraemia predisposes to infection and nearly all severely injured patients develop some form of infection. Strenuous efforts should be made to protect such patients from resident hospital organisms of which the two most notorious are Staphylococcus aureus and Pseudomonas aeruginosa. Strict barrier nursing facilities should be made available including the use of cubicles with separate filtered ventilation under positive pressure.

Regular prophylactic bacteriological examination of wounds, sputum, excreta, blood, and other sites should provide in-vitro sensitivity information about invading organisms before infections become clinically manifest. Antibiotics should be used precisely for specific infections and never as a prophylactic umbrella. Doses must be tailored to the renal function of the individual patient in order to avoid toxicity. Good information is now available.
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on predicted safe dosage for patients on dialysis (Linton and Lawson, 1970; Kerr and Walls, 1971). Antibiotic blood levels can be obtained in cases of doubt.

The use of a urinary catheter may be justified at the onset of acute renal failure for diagnostic purposes and particularly while assessing the response to mannitol. Once renal failure is established catheters are dangerous from an infection point of view and serve no useful purpose.

Dialysis and diet.

Up to the early 1960s haemodialysis was performed for symptoms of uraemia and on specific biochemical indications such as a plasma urea of 400 mg/100 ml or a plasma bicarbonate of 12 m.equiv/l. It is now customary to dialyze with a high efficiency machine (e.g. a UF100 or Ex-03 coil), daily at first and on alternate days when the catabolic rate slows down, maintaining the plasma urea at about 180 mg/100 ml pre-dialysis and 80 mg/100 ml post-dialysis. There has been an improvement in survival rate for post-traumatic renal failure contemporaneously with this change from about 25 per cent to about 50 per cent (Kerr and Elliott, 1970), but much of this must be attributed to better management of shock, respiratory failure and antibiotic therapy. No controlled trial has ever been carried out to test the value of intensive dialysis but the universal impression of nephrologists that it is beneficial is so strong that no such trial is ever likely to be undertaken. Whatever it does for survival, daily dialysis commends itself because it frees the patient from most of his dietary restrictions and permits intravenous feeding when necessary. However, it carries a risk of excessive dehydration which may prolong the renal failure; bed weighing to estimate fluid loss during dialysis is not easy when the patient is tied to a Balkan beam. A nomogram showing the expected fluid loss at varying blood flow rates and venous bubble trap pressures should therefore be affixed to the dialyzer so that fluid is given prophylactically during dialysis. This rough calculation must be checked by frequent observation of the patient for clinical signs of overhydration or dehydration and by c.v.p. monitoring as long as the catheter is in situ. Once the c.v.p. catheter is removed reliance must be placed on observation of the jugular veins and the discrepancy between c.v.p. and j.v.p. measurements should be kept in mind (Riordan, McLay and Walters, 1969).

Intake output charts should be kept, within the limits imposed by numerous dressings, pyrexia, intestinal ileus or diarrhoea, etc., but undue reliance should not be placed upon them. The poorly estimated fluid shifts during dialysis often outweigh other losses and make accurate assessment of external balance impossible. Observation of the patient takes precedence over observation of the charts.

Calorie and protein intake should be maintained from the start. Weight loss of 10–20 kg is not uncommon in the course of post-traumatic acute renal failure and such massive catabolism must contribute to the high mortality. Feeding must often be by vein initially and the aim should be to provide total calorie replacement plus 40 g of protein equivalent to replace the considerable losses through the dialyzer. Intravenous aminoacid solutions can be given into the return line of the dialyzer in the last 2 hours of dialysis without an unacceptable loss into the dialysis fluid (Norée et al., 1971).

Calorie intake can be increased by adding glucose to the bath of the dialyzer in a concentration of 0.5–1.5 per cent. This results in a dextrose uptake by the patient of the order of 100–200 g in a 6-hour dialysis. Blood glucose levels should be monitored if the higher concentrations are used, at least on the first one or two occasions, in view of the impaired glucose tolerance found in uraemia.

As long as oral feeding is withheld, mouth toilet requires scrupulous attention. Hourly mouthwashes should be given during waking hours as well as regular attention to crusted teeth. As soon as the surgical condition permits, oral feeding should be resumed with as high a calorie intake as the patient can tolerate and at least 40 g of protein per day. In practice these patients are usually anorexic and are best fed with a free diet, within reason; supplementary calories are often required intravenously during dialysis to bring their oral intake up to their requirements.

Dialysis should be continued until the patient can maintain his own plasma urea and creatinine at a stable, acceptable level (e.g. a plasma urea below 150 and a creatinine below 8 mg/100 ml). This may not happen until the diuretic phase has been established for 1–2 weeks and the urine volume is well above 2 litres per 24 hours, if the patient is still hypercatabolic. Diuresis usually begins within 3 weeks of the onset of oliguria unless renal failure is prolonged by infection, haemorrhage, hypoprotein-æmia or dehydration. If oliguria persists beyond 4 weeks one should suspect bilateral cortical necrosis.
The diagnosis is confirmed by the demonstration of renal calcification on radiographs of the abdomen or by renal biopsy.

Anabolic steroids reduce the production of urea (and by inference the breakdown of tissue protein) in patients with chronic renal failure and postobstetric acute renal failure (Robson, Kerr and Ashcroft, 1968). Their contribution to the management of post-traumatic renal failure is difficult to assess because of the other factors influencing catabolism, but is probably small. However, a single injection of a non-icterogenic steroid, e.g. nandrolone decanoate 50 mg, at the onset of oliguria does no harm and may do a little good.

MANAGEMENT OF COMPLICATIONS

Fat embolism.

Fat embolism is a well-known complication of all major fractures. The classical physical sign is the appearance of small haemorrhages in skin, mucous membranes, and retina. Ross (1970) has shown by blood-gas analysis that the most important effect of fat embolism is hypoxia. This in turn is responsible for the early cerebral signs such as restlessness, anxiety, and drowsiness, and ultimately may be the cause of death. The petechiae mentioned above may appear later. Ross suggests that hypoxia is brought about by two mechanisms, firstly defective gas transfer across the alveolar-capillary membrane as the result of the severe degree of alveolar oedema, and secondly, the shunting of deoxygenated blood through the lungs. O'Driscoll and Powell (1967) reported benefit from the use of clofibrate in reducing lipaemia and the incidence of fat embolism after trauma. A carefully designed double-blind prospective trial of clofibrate by Cole (1971) revealed no advantage over a placebo. Unfortunately there are no entirely effective measures for dealing with fat embolism once it has occurred. Brain damage can be reduced by induction of hypothermia (Larson, 1968; Ross, 1970). Improvement in blood flow and perfusion of tissues can be achieved by use of phenoxybenzamine which by producing adrenergic blockade relieves vasoconstriction and improves cardiac function. Positive pressure ventilation may improve arterial oxygen saturation (Larson, 1968). Early recognition of hypoxia is perhaps the most important single aspect. It is wise to be alert to the possibility of fat embolism in the first 48 hours in any patient showing signs of restlessness and confusion progressing to coma.

Gram-negative shock.

Early recognition of gram-negative septicaemia, allowing prompt antibiotic therapy after blood cultures have been obtained, will usually prevent progress to overt shock. Once shock has occurred diffuse intravascular coagulation is likely to be present (Corrigan, Ray and May, 1968). The most frequently useful sign of this is thrombocytopenia, but other confirmatory tests are a low level of factor V in the plasma and the presence of fibrin degradation products in serum. Heparin is beneficial in this situation if given early but if disseminated intravascular coagulation has already occurred heparin is without much effect. According to Lasch (1969) streptokinase-induced fibrinolysis has experimental and clinical justification in the established case.

Jaundice following shock and trauma.

The onset of jaundice following shock and trauma is an ominous sign which those who take care of severely injured patients learn to dread. Nunes and co-workers (1970) have studied a number of these patients and believe that the primary mechanism is a hepatic cellular excretory defect resembling intra-hepatic cholestasis and probably due to hepatic anoxia. The increased bilirubin load provided by transfused and extravasated blood, which in the normal liver would cause only mild jaundice, results in more severe and prolonged jaundice in the patient with an anoxia-damaged liver. Histology of the liver at autopsy shows centrilobular congestion. Engorgement of sinusoids and compression of the central cells with or without necrosis is the common pattern. True centrilobular necrosis is unusual. Although liver function tests suggest obstructive jaundice, obstruction is rarely found. If the patient survives, the jaundice does not recur and liver function tests return to normal. There is as yet no known effective treatment apart from supportive therapy until spontaneous recovery occurs.

PROGNOSIS

Patients with bilateral cortical necrosis (about 1–2 per cent of the total) will occasionally recover enough renal function to maintain life if dialysis is carried on for long enough (Walls, Schorr and Kerr, 1968; Deutsch et al., 1971). Even if he remains oliguric the young survivor of accidental trauma is often a good candidate for regular dialysis or transplantation. In uncomplicated acute tubular necrosis about 70 per cent of patients are left with a defect in renal function after one year; however, this is of minor
degree in over four-fifths of these and even the more severely affected are usually asymptomatic and do not appear to develop progressive renal failure or hypertension as late complications (Briggs et al., 1967). The rewards for successful management of acute renal failure are therefore worth the considerable effort.

REFERENCES


CORRESPONDENCE

PROPANIDID VERSUS THIOPENTONE FOR INDUCTION OF GENERAL ANAESTHESIA IN ELECTIVE CAESAREAN SECTION

Sir,—I read the paper published by Dr Baraka and his colleagues with interest (Brit. J. Anaesth. (1971), 43, 609). The authors mention that the incidence of neonatal depression was higher with thiopentone, muscle relaxant, and nitrous oxide anaesthesia. I have anaesthetized sixty-two patients for Caesarean section in the last two years. In ten, epidural analgesia was used, and general anaesthesia in the remainder. In the latter group, after premedication with atropine 0.6 mg followed by preoxygenation, anaesthesia was induced with thiopentone 125–175 mg. Suxamethonium 75–100 mg was injected. Anaesthesia was maintained in thirty-nine patients using nitrous oxide, oxygen, pethidine 50–75 mg and a trace of ether. The latter two drugs were administered after delivery. Suxamethonium was injected intermittently as required.

In thirteen patients, thiopentone 125–175 mg, nitrous oxide, oxygen and tubocurarine 15–20 mg was used, pethidine 50–75 mg being given intravenously after delivery. Neonatal respiratory depression was observed in seven patients. Six of these were judged to be depressed to a mild degree in the opinion of the neonatal physician and the Appgar score. Resuscitation was not a major problem. The seventh baby had severe respiratory depression which responded after prolonged treatment. In all seven cases there was evidence of cephalo-pelvic disproportion, prolonged labour, or early placental separation.

I strongly believe that neonatal respiratory depression associated with thiopentone can be avoided if it is used in a sleep dose and not in a full induction dose.

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THE OCCURRENCE OF UNBLOCKED SEGMENTS DURING CONTINUOUS LUMBAR EPIDURAL ANALGESIA FOR PAIN RELIEF IN LABOUR

Sir,—We were interested to read the article under this title by Dr Mary Ducrow (Brit. J. Anaesth. (1971) 43, 1172). Her observations on the incidence of unblocked segments and unilateral blockade prompt us to make three points.

(1) Spread of block is in part dependent on the volume of solution injected. There should therefore be some difference in the extent of blockade depending on whether air or saline is used as the "loss of resistance" medium. For instance, if 10 ml of saline is injected as the medium, and this is followed by 8 ml 0.25\% bupivacaine, then in fact the patient receives 18 ml of 0.1\% solution.

(2) It is not clear whether the epidural catheter is inserted 5 cm beyond the tip of the needle (i.e. 5 cm inside the epidural space) or whether the catheter is inserted 5 cm from the skin. In the case of the former there is enough catheter free for it to pass into a root sheath; in the case of the latter, in large or obese patients the tip of the catheter would barely reach the epidural space.

(3) The predominance of missed L1 segments could be accounted for by the fact that this is frequently the most painful area during labour. In the context of extreme dilution of local anaesthetic, only partial blockade of all segments may be achieved, but because of greater stimulus in this area, it may appear that the L1 segment is still sensitive although in fact it is anaesthetized to the same degree as segments above or below it.

Roger Fletcher
J. C. Richardson
G. A. Meadows
Liverpool

SIR,—I am grateful to Drs Fletcher, Richardson and Meadows for their observations regarding unblocked segments and unilateral blockade during lumbar epidural analgesia, and for the opportunity to reply. To take each of the points in turn.

(1) The method of identifying the epidural space in all cases was loss of resistance using a Macintosh balloon. This utilizes 2 ml of air, so dilution of local anaesthetic solution was not a factor.

(2) The epidural catheter was inserted, usually 5 cm beyond the tip of the needle. Unfortunately this fact is not noted in the records and may have varied from case to case and with different operators. If the catheter passed into a root sheath I would have expected a small area only to become analgesic rather than a large area analgesic with a small area missed. However, too much catheter inserted may indeed have accounted for unilateral blockade.

(3) I agree that the predominance of missed L1 segments may be due to the greater stimulus in this area. As dilution of local anaesthetic solution was not a factor then presumably uneven spread of solution must be responsible.

Mary Ducrow
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