Peritoneal Dialysis in Children*

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SUMMARY

The principles, technique and complications of peritoneal dialysis are discussed. Some case reports illustrating indications, technical difficulties and complications are given.

Peritoneal dialysis is a safe, efficient therapeutic procedure readily applicable to small infants and children.

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Intermittent peritoneal dialysis is an invaluable therapeutic procedure for severe renal failure and life-threatening intoxications with dialysable drugs and poisons. The early methods, first performed by Ganter in 1923[1] were complicated and hazardous. Technical advances such as the availability of commercially prepared dialysis solutions and equipment and a better understanding of the principles involved, have established peritoneal dialysis as an effective and safe temporary substitute for normal renal function, which may be readily applied to infants and children.

The purpose of this article is to review briefly the principles involved, to outline the method used in our hospital and to discuss the technical difficulties and complications which may be encountered.

PRINCIPLES

The peritoneum behaves as an inert semipermeable membrane and allows diffusion of water and solutes along concentration gradients. The infusion of an 'idealized extracellular fluid' into the peritoneal cavity allows both the removal of excess fluid and solutes and the uptake of needed solutes.

Boen[2] showed that in adults equilibration between the plasma urea and the dialysate occurred in about 120 minutes. Kinetic studies have demonstrated the relative rates of diffusion and clearance to be: urea>K>Cl>Na>creatinine>PO_4>uric acid>HCO_3>Ca>Mg. Irrespective of the osmolality of the dialysate, once equilibration with the plasma has occurred, water and solutes will be absorbed. For efficient dialysis, therefore, the dialysate is drained after allowing a short time for equilibration to occur.

In adults the maximum peritoneal urea clearance is obtained with a dialysis rate of 3·5 litres/hour.[2] For the paediatric age group this is roughly equivalent to 50 ml/kg/hour. This exchange rate and volume may be altered depending on clinical exigencies. More rapid dialysis withdraws more water, but results in increased protein and amino acid loss in the dialysate and costs more. Smaller exchange volumes may be indicated if the abdominal distension aggravates existing respiratory embarrassment.

The young child has a greater peritoneal surface area relative to body weight than the adult and greater clearance values are achieved with peritoneal dialysis in infants and children than in adults.[3]

Warming the dialysis solutions to 37°C in a waterbath causes less discomfort and results in a 35% increase in efficiency due to vasodilatation of the peritoneal vessels.[4]

DIALYSIS SOLUTIONS

The 1·5% dextrose dialysis solution is used for the removal of toxic metabolites and the correction of electrolyte imbalance and metabolic acidosis. It will be seen from Table I that the electrolyte composition of this solution is essentially similar to K-free extracellular fluid. Dextrose is added to increase the total osmolality to 372 mOsm/kg, which is slightly higher than the serum osmolality of uraemic patients and sufficient to counteract the osmotic pressure of the plasma proteins. Potassium is added unless severe hyperkalaemia is the reason for dialysis. Lactate is used as the source of alkali and is converted to bicarbonate by the liver.

The 7% dextrose dialysis solution is markedly hypertonic to plasma and allows the removal of oedema fluid. Peritoneal ultrafiltration is indicated in severe circulatory embarrassment and intractable oedema. This solution is rarely used in the paediatric age group as it may rapidly result in dehydration and shock. Instead, for peritoneal

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TABLE I. PERITONEAL DIALYSIS SOLUTIONS

Peritoneal dialysis solution	Dextrose g/litre	Na^+ mEq/litre	Ca^{++} mEq/litre	Mg^{++} mEq/litre	Cl^- mEq/litre	Lactate mEq/litre	Osmolality mOsm/kg
1·5% Dextrose*	15	141	3·5	1·5	101	45	372·8
2% Dextrose* (low-sodium)	20	130	3·5	1·5	94	41	378·5
7% Dextrose*	70	141	3·5	1·5	101	45	678·3

* Dineal. Baxter Laboratories Inc., Morton Grove, Ill., USA. Supplied in South Africa by Saphar Laboratories Ltd, Stephen Road, Ophirton, Johannesburg.

ultrafiltration, mixtures of the 1·5% and 7% dextrose dialysis solutions are used.

The use of hypertonic solutions enhances the peritoneal clearance of urea by the mechanism of solvent drag and by increased membrane permeability.[5] At the same time it frequently results in hypernatraemia due to the proportionately greater withdrawal of water than sodium.[6]

We have used the 2% dextrose low-sodium dialysis solution with good results for some cases with severe sodium and water retention.

Albumin added to the dialysis fluid enhances the removal of substances normally bound to protein, such as salicylates and barbiturates. Appropriate adjustment of the pH of the dialysate enhances the removal of weak acids and bases.

Recently commercial dialysis solutions* have been introduced which contain acetate and sorbitol instead of lactate and dextrose respectively. Several advantages are claimed for these solutions. Acetate is metabolized to bicarbonate largely by the peripheral tissues and is indicated in the newborn infant and also in liver failure and lactic acidosis. Hyperglycaemia does not occur and as sorbitol is more slowly metabolized than glucose, it tends to maintain an osmotic load in the face of a decreasing blood urea and may thus help to counteract oliguria and the disequilibrium syndrome.

INDICATIONS AND CONTRA-INDICATIONS

The following are indications for dialysis:

1. Acute renal failure:
   - (*i*) uraemic syndrome;
   - (*ii*) blood urea>250 mg/100 ml;
   - (*iii*) uncontrolled hyperkalaemia>7 mEq/litre;
   - (*iv*) intractable metabolic acidosis plasma HCO_3<12 mEq/litre;
   - (*v*) severe hypo- or hypernatraemia;
   - (*vi*) severe circulatory embarrassment.
2. Renal failure of undetermined aetiology to allow evaluation of patient.
3. Chronic renal failure severely symptomatic:
   - (*i*) due to abrupt deterioration during intercurrent infection, heart failure, sodium and volume depletion;
   - (*ii*) pending transplantation.
4. Others:
   - (*i*) intractable lactic acidosis;
   - (*ii*) intractable oedema, e.g. severe heart failure, nephrotic syndrome;
   - (*iii*) hypercalcaemia;
   - (*iv*) hyperuricaemia;
   - (*v*) intoxication with dialysable toxins (see Table II);
   - (*vi*) peritonitis;
   - (*vii*) pancreatitis.

Dialysis is contra-indicated when intra-abdominal disease is suspected; after recent major abdominal surgery; when open abdominal wounds, faecal fistulae and colostomy, extensive adhesions and diaphragmatic defects are present.

The decision to dialyse a child must be made individually for each patient, taking into account all clinical and laboratory findings. Early dialysis, before the child is severely symptomatic is preferable. With the exception of diaphragmatic defects[9] the contra-indications to peritoneal dialysis in children are relative and the risks should be weighed against the expected benefits. Though various devices permitting repeated atraumatic access to the peritoneal cavity have been described, ultimately infection is almost invariable. The quality of life is such that, in our opinion, repeated peritoneal dialysis for chronic renal failure is not a reasonable proposition for children at present, unless kidney transplantation is contemplated.

*McGaw Laboratories, Glendale, California, USA. Not at present available in South Africa.

TABLE II. DIALYSABLE TOXINS AND DRUGS[7,8]

Salicylates	Nitrofurantoin
Barbiturates	Isoniazid
Meprobamate	Kanamycin
Amphetamine	Streptomycin
Bromide	Neomycin
Diphenylhydantoin	Cephalothin
Methanol	Cephaloridine
Boric acid	Amanita phalloides mushroom
Sulphonamides	poisoning

METHOD

The technique in use here is based upon the descriptions of Segar *et al.*,[10] Barry and Schwartz[11] and Maxwell *et al.*[12] with various modifications. Meticulous asepsis during the initiating procedure and during any subsequent handling of the catheter, its connections and the wound, is absolutely essential.

Preparation of the Patient

Unless the patient is comatose he is sedated with intramuscular pethidine 1½ - 2 mg/kg and promethazine 1 mg/kg bodyweight. The bladder is emptied voluntarily or by aseptic catheterization with a small catheter and then irrigated with an antiseptic solution. The stomach contents are aspirated in small infants and unconscious patients.

Procedure

With the child supine, the anterior abdominal wall is prepared as for a surgical procedure. Under local anaesthesia a 2 - 3 mm stab incision is made with a No. 11 Bard-Parker blade in the midline 2 - 5 cm below the umbilicus. The linea alba is relatively avascular and significant bleeding from the wound is unlikely. However, any site below the level of the umbilicus may be used and a site high in the left iliac fossa is the next best choice. The latter is preferred in small infants where the high intra-abdominal position of the bladder, and in the newborn the umbilical arteries, may cause complications. The fixed caecum on the right constitutes a potential hazard. In small infants we prefer to insert the catheter under direct vision in the operating theatre through the smallest incision the surgeon will permit.

Unless considerable ascites is already present one exchange volume of 50 ml/kg of warmed dialysis solution is

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infused into the peritoneal cavity through a short-bevelled 15 - 16 gauge spinal needle. This facilitates insertion of the trocar and cannula and correct placement of the catheter and lessens the risk of perforation of the gut. The needle is supported during the infusion by clamping an artery forceps across it at skin level. At the same time, particularly in small infants, it is advisable to assess the extent to which the respiratory movements of the diaphragm are affected by the abdominal distension. A smaller exchange volume may be indicated.

A paediatric stylet-catheter,*[13] or the smallest trocar and cannula which will admit the selected catheter,† is used. The instrument is introduced with a firm controlled twisting movement; entry into the peritoneal cavity is denoted by a sudden 'give' and confirmed by the gush of dialysis fluid as the trocar or stylet is withdrawn. The catheter is angled towards the pelvis so that the tip will lie in the lowest part of the abdomen. All perforations in the distal 4 cm must be well within the peritoneal cavity. It should glide in smoothly as force will do damage, may kink the catheter and is unlikely to be followed by efficient drainage.

The cannula is removed and the in- and outflow circuit‡ connected (Fig. 1). The inflow tubing may incorporate a warming coil immersed in a waterbath kept at 37°C. This is particularly indicated if volumes of 500 ml or less are used and avoids the need for frequent re-warming of the fluids between inflow periods. The outflow tube, which is connected to the catheter via a Y-connection, drains into a graduated plastic urine drainage bag with a non-return valve § (Fig. 1). This ensures a closed circuit and lessens the risk of infection. The dialysate is then drained by a siphoning action. A deficit in the initial return of about half an exchange volume is acceptable as reservoir.

It is our practice to carry out a further rapid complete exchange before securing the catheter and dressing the wound. Thus, adequate in- and outflow may be ascertained and the position of the catheter is altered, if indicated, while asceptic conditions still prevail. The catheter is secured by strips of non-allergenic adhesive tape; excessive catheter length is cut off. Sutures are avoided as they are painful, risk wound sepsis and leave bigger scars. The wound, surrounding skin and catheter are sprayed with an antibiotic aerosol and covered with a few slotted dry dressings which are secured by broad adhesive plaster applied longitudinally so as to allow abdominal distension. The dialysis circuit is so arranged that only two clamps, clearly labelled 'IN' and 'OUT' need be operated by the nurse (Fig. 1).

Heparin 500 units/litre is added to the dialysis fluid. This does not interfere with the body's clotting mechanisms. Intraperitoneal antibiotics are not used unless peritonitis is present or suspected, considerable leakage is occurring, or recent bowel surgery has been performed. Under these circumstances ampicillin 50 - 100 mg/litre (25% absorbed) or the appropriate antibiotic, as indicated by sensitivity tests, is added to the dialysis fluid in a concentration equivalent to the optimum serum level. Due attention must be paid to absorption and potential toxicity of some antibiotics in renal failure. Tetracycline in particular should be avoided as it increases azotaemia and accumulates progressively in anuric states.[14] We use oral Neomycin 25 mg/kg bodyweight to suppress growth of the intestinal flora before and during dialysis.[15]

An exchange volume of about 50 ml/kg bodyweight cycled at one exchange per hour is used. This allows 10 - 15 minutes for infusion, 30 minutes for equilibration and 15 - 20 minutes for drainage. At the usual blood urea level of about 300 mg/100 ml at which dialysis is commenced, this exchange rate will lower the blood urea by about 150 - 175 mg/100 ml per 24 hours.

In severe respiratory distress and circulatory overload smaller and more rapid exchanges with hypertonic fluid are used. On occasion we have had to pass a naso-endotracheal tube and use intermittent positive pressure ventilation to avoid fatal respiratory embarrassment by the abdominal distension. In some instances we have allowed no time for equilibration as such but have used longer periods for infusion and drainage on the assumption that a reservoir is constantly present and continuous movement of fluid discourages clots and fibrinous obstruction of the catheter.

Not infrequently a deficit in a single return of dialysate occurs, only to be more than regained during the next cycle. It is a waste of time to try and drain the full exchange volume drop by drop, rather commence the next cycle. However, deficits in the return dialysate should not be allowed to accumulate, otherwise absorption and fluid overload will occur. Total dialysis time is limited to 30 - 36 hours which is equivalent to 6 - 8 hours haemodialysis.

In hypercatabolic acute renal failure such as occurs after trauma, operations and with severe infections it may be necessary to continue to dialyse 8 - 12 hours daily in order to maintain reasonable homoeostasis. Daily dialysis increases the risk of infection considerably but has the advantage that it allows a more liberal fluid and dietary intake and extreme biochemical derangements are avoided. In this instance the catheter is removed and reinserted in another site at the first sign of wound or peritoneal infection or in any case every fifth day.

Discontinuation of the dialysis. The catheter is removed after each procedure unless daily dialysis is necessary. Removal is carried out under strict aseptic conditions after complete drainage has been ensured. Should resistance be encountered on withdrawal of the catheter, gentle semicircular rotatory movements may dislodge omentum without twisting it onto the catheter. If omentum prolapses through the wound it should be tied off with catgut, trimmed and allowed to slip back. The need for meticulous asepsis is obvious. Sutures are not usually required. The wound is sprayed with an antibiotic aerosol and a dry dressing applied.

Observations and Records

Pulse and respiration rates, temperature and blood

*Trocath stylet-catheter, paediatric size, McGaw Laboratories, Glendale, California, USA.
†Plexitron dialysis catheter, paediatric size, Travenol Laboratories Inc., Morton Grove, Ill. USA.
‡Plexitron Y-type dialysis solution administration set with drainage tubing. Travenol Laboratories Inc., Morton Grove, Ill. USA.
§Aldon type CV urine drainage bag with non-return valve. Aldington Laboratories Ltd, Mersham, Ashford, Kent, UK.

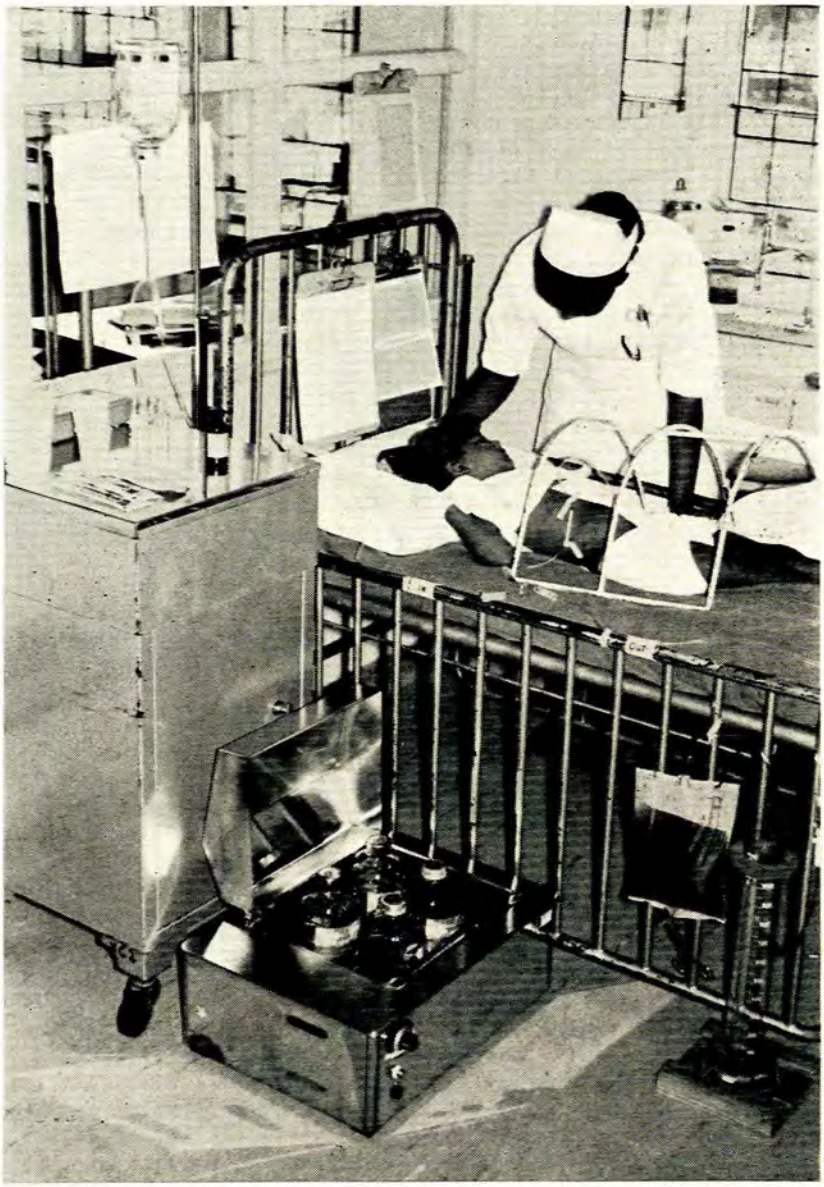

Fig. 1. Peritoneal dialysis in progress. The dialysate is bloodstained.

pressure are recorded hourly, while the patient is weighed before and if possible twice daily during dialysis. A careful watch is kept on the child's state of hydration. The time, the volumes infused and drained and additions to the dialysis fluid are recorded on special charts (Fig. 2). The return dialysate is measured in a tall graduated glass cylinder. Each litre-bottle of dialysis fluid contains an excess of 30 - 50 ml which must be taken into account when calculating the cumulative fluid balance. It is advisable to check the nurses' calculation from time to time. In order that over-all fluid balance may be maintained it is essential to measure and record all intake and output via other routes. Ideally patients should be dialysed on a sensitive bed scale to facilitate accurate assessment of total fluid balance, including insensible losses and metabolic water production.

TECHNICAL DIFFICULTIES AND COMPLICATIONS

The potential difficulties and complications which may be encountered are many: pain; fever; infection; volume depletion and fluid retention; hypoproteinaemia; hypernatraemia; metabolic alkalosis; hyperglycaemic, hyperosmolar coma; hypokalaemia; basal pulmonary atelectasis and pneumonia; perforation or laceration abdominal viscus; acute and chronic hydrothorax; vasovagal reaction and adhesions. However, with experience they become less

PERITONEAL DIALYSIS - FLOW SHEET

R X H RENAL CLINIC

NAME: Willie Smith HOSP. NO. 004789 DATE: 20-3-1970

WEIGHT: 20 kg

INSTRUCTIONS: Volume per Exchange: 1000 ml 1.5% Dextrose Dialysis fluid

Run in: 10 min. To remain in: 30 min. Run out: 20 min.

Additions: 1½ ml 20% KCl/litre

Heparin 500 units/litre

TIME	NO	VOLUME IN	VOLUME OUT	EXCESS+ OUT +	DEFICIT- LEFT IN-	CUMULATIVE FLUID BAL. + or -	ADDITIONS REMARKS
4 p.m	1	1000	800	-	-200	-200	KCl Heparin Blood-stained
4.20	2	1000	1100	+100	-	-100	KCl Heparin
5.00	3	1000	1100	+100	-	0	KCl Heparin
6.00	4	1000	1050	+ 50	-	+ 50	KCl Heparin
7.00	5	1000	700	-	-300	-250	KCl Heparin
8.00	6	1000	1400	+400	-	+150	KCl Heparin
9.00	7	1000	1080	+ 80	-	+230	KCl Heparin

Fig. 2. Peritoneal dialysis record chart.

frequent and are usually easily remedied. Difficulty with insertion of the catheter is overcome by placing it in the peritoneal cavity under direct vision through the smallest incision possible.

The first few exchanges frequently are slightly blood-tinged due to wound haemorrhage. This amounts usually to no more than a few millilitres of blood and requires only Heparin to avoid clotting in the catheter. Significant bleeding is controlled by a generous pursestring suture around the catheter. Leakage of dialysate reduces the efficiency of the dialysis, makes accurate fluid balance difficult and increases the risk of infection. It rarely occurs if the stylet catheter is used. A pursestring suture and smaller exchange volumes will remedy this.

If the catheter has not been inserted deep enough and some proximal perforations are outside the peritoneal cavity, leakage of fluid extraperitoneally or into the subcutaneous tissues will occur. This may also occur if the peritoneum has been pierced more than once. It is troublesome and requires correct replacement of the catheter.

Inadequate return of the dialysate is relatively frequent and may be due to the catheter lying extraperitoneally or in a loculated part of the peritoneal cavity or may be due to obstruction by omentum, fibrin, clots, loops of bowel or an air lock. The former requires re-insertion of another catheter, while obstruction is often relieved by a change in the patient's position, elevating the head end of the bed, flexing the knees, pressure on one or other side of the abdomen, temporarily creating a larger reservoir around the catheter by going on to the next exchange or introducing heparinized dialysis fluid under pressure. Occasionally it is due to exceptionally rapid absorption of isotonic fluid and requires the use of hypertonic dialysis solutions. Failing these manoeuvres, the catheter must be manipulated or replaced with strict aseptic precautions.

Peritonitis is a serious complication but not necessarily a contra-indication to continued dialysis provided appropriate antibiotic therapy, both systemic and intraperitoneal is promptly instituted. The infecting organisms are usually the normal bowel flora. Hypertonic solutions have been shown to promote direct transmural migration of bacteria[16] and the administration of prophylactic small doses of oral Neomycin seems appropriate.[15] The incidence of peritonitis correlates directly with the duration of the dialysis and is yet another reason why early dialysis is preferred. The clinical features of peritonitis are less striking than in other circumstances, and are a low-grade fever, vomiting and moderate abdominal pain and tenderness.

There may be other causes of peritonitis besides dialysis. The presence of leucocytes in the dialysate or a single positive culture in the absence of clinical features may usually be ignored. Twelve-hourly cultures of the dialysate and of the catheter tip at the end of dialysis ensures early bacteriological diagnosis and facilitates the choice of the appropriate antibiotic. Dialysis with a 1·5% dextrose dialysis solution usually results in a negative water balance of about 5%. Fluid retention during dialysis may precipitate pulmonary oedema, congestive cardiac failure and hypertensive encephalopathy. Congestive heart failure may rarely occur without obvious fluid retention and is related to excessive sodium absorption and insufficient withdrawal of excess water in hyponatraemic patients.[17] The use of low-sodium dialysis solutions in patients at risk and careful attention at all times to the cumulative fluid balance and clinical features will diminish the likelihood of this complication.

Half a gram of protein or more may be lost with each litre of dialysate. This consists mainly of albumin and gammaglobulin.[18] Amino acids are also lost in significant quantities.[19] Whereas these losses may be disregarded in one or two dialyses, oral or intravenous replacement is indicated if repeated peritoneal dialysis is required. Water-soluble vitamins are also lost in the dialysate. Hypernatraemia is a frequent finding towards the end of a dialysis, in our experience even with the use of the isotonic solution. Though this is often asymptomatic we now favour the use of low-sodium 2% dextrose dialysis solution if considerable sodium retention is present (see Table I). Metabolic alkalosis is frequently present after 24 - 30 hours of dialysis. It is rarely of any concern, though in one of our cases it resulted in severe respiratory depression.

Conversion of lactate to bicarbonate may be inadequate or delayed in the neonate and in liver failure, resulting in a lactic acidaemia. The use of acetate-containing dialysis solutions avoids this complication.

The disequilibrium syndrome and hyperglycaemic hyperosmolar coma have not been seen in our unit. The former is due to cerebral oedema from a lag in the rate of diffusion of urea out of the brain cells, with consequent movement of water into the cells.[20] It occurs more often in haemodialysis during which the blood urea is rapidly lowered. The disequilibrium syndrome is treated with mannitol infusions to raise the ECF osmolality. Cerebral cellular dehydration may result from the diffusion of glucose from hypertonic dialysate to the ECF.[21] Diabetic patients are particularly prone to the resulting hyperglycaemic, hyperosmolar coma.

The occurrence of progressive mental dulling despite biochemical improvement of the uraemia indicates the need for blood sugar estimations and, if severe hyperglycaemia is present, small doses of soluble insulin. Moderate hyperglycaemia, however, is of no concern and acts as a source of calories. Hypokalaemia is hazardous for patients on Digoxin which is not removed by peritoneal dialysis. Hyperkalaemia may mask digitalis intoxication and the correction of hypocalcaemia, hyponatraemia, metabolic acidosis and glucose loading, all lead to a rapid lowering of the serum potassium. Potassium-free dialysis fluids should not be used for longer than 4 - 6 hours in these circumstances and frequent ECG monitoring is essential.

Deep breathing exercises where possible, postural changes and avoiding excessive abdominal distension by using appropriate exchange volumes will often prevent pulmonary complications.

Perforation of the bowel is recognized by an inadequate dialysate outflow, which may be frankly faeculent and/or profuse watery diarrhoea.[22] Radiographic demonstration of air in the abdomen is of little diagnostic help as some air is often introduced when dialysis is initiated. Adequate distension of the abdomen with fluid before the trocar and catheter are introduced is mandatory, while abdominal scars should be avoided. An acute hydrothorax due to an acquired or congenital diaphragmatic defect is perhaps the only absolute contra-indication to further peritoneal dialysis.[5]

CONCLUSION

Despite the many potential hazards of peritoneal dialysis, in children this method has several advantages over haemodialysis. It may be instituted within an hour, even by less experienced personnel, there is no need for blood transfusion, cannulation of vessels or systemic anticoagulation and it is efficient in the presence of shock. It is readily applicable even in small infants. Rapid alterations in blood volume and the disequilibrium syndrome are much less likely to occur.

On the other hand, haemodialysis is more efficient per unit time than peritoneal dialysis; a fact which may be vital in life-threatening intoxications and hypercatabolic renal failure. Haemodialysis is preferred for children awaiting transplantation in most centres where these are done.

ILLUSTRATIVE CASES

Case 1

A 3-year-old non-White girl was admitted to hospital severely dehydrated and semi-comatose 3 days after the onset of profuse vomiting. Laboratory investigation showed the following: Blood urea 212 mg/100 ml; serum K 6·0 mEq/litre; Astrup: pH 7·36; PCO_2 32·5 mmHg; base excess −5·5 mEq/litre and standard HCO_3 15 mEq/litre. The cerebrospinal fluid was normal except for 11 lymphocytes/mm³. Microscopic haematuria, cylindruria and 2+ proteinuria were present in a urine specimen obtained on admission.

She was rapidly rehydrated with intravenous fluids but passed no more urine during the next 12 hours. Lasix 50 mg and mannitol 10 g were administered intravenously. She remained severely oliguric however and 24 hours later she had a brief generalized convulsion. The blood urea had risen to 300 mg/100 ml.

A predialysis catheter specimen of urine showed an osmolality of 296 mOsm/kg and a urea content of 760 mg/100 ml.

Because of technical difficulties the dialysis catheter was inserted under direct vision through a small incision. After dialysis she was fully conscious and the blood urea was 82 mg/100 ml, serum Na 154 mEq/litre and the serum K 4·8 mEq/litre. She remained oliguric for a further 9 days, whereafter gradual but complete recovery occurred.

Diagnosis: Acute tubular necrosis due to severe dehydration. Presumed viral encephalitis.

Comment: The indications for peritoneal dialysis were uraemia with continuing oligo-anuria. The convulsion may have been due to uraemic encephalopathy or an encephalitis.

This small girl had a scaphoid abdomen and fear of intestinal perforation resulted in some dialysis fluid being infused extraperitoneally. Subsequently it was impossible to introduce the trocar and cannula into the peritoneal cavity due to the extraperitoneal collection of fluid. Insertion of the dialysis catheter was accomplished under direct vision without further difficulty. To avoid leakage, the incision in the peritoneum was closed thoroughly.

Though no change in body weight occurred, post-dialysis hypernatraemia due to absorption of sodium from the dialysate was found.

Early dialysis is preferable before uraemic manifestations appear, particularly if a rising blood urea of >250 mg/100 ml with persisting oligo-anuria is present.

Case 2

A 5-year-old non-White boy was admitted to hospital with severe acute post-streptococcal glomerulonephritis complicated by gross congestive cardiac failure, pulmonary oedema, uraemic convulsions and severe epistaxis. In addition, he had oedema glottidis due to penicillin anaphylaxis and was cyanosed on admission. The blood pressure was normal.

The blood urea was 266 mg/100 ml; the serum K 8·4 mEq/litre with ECG evidence of hyperkalaemia; Astrup: pH 7·02; PCO_2 80 mmHg; base excess −12 mEq/litre and the standard HCO_3 15 mEq/litre.

Naso-endotracheal intubation and 100 mg hydrocortisone intravenously alleviated the respiratory obstruction, but he remained cyanosed out of oxygen. Lasix 100 mg and insulin and dextrose were given intravenously while three-quarters of the digitalizing dose of Digoxin was given intramuscularly over 12 hours.

Peritoneal dialysis with a 4·25% dextrose dialysis solution was instituted, initially using K-free dialysis fluid, small exchange volumes and a rapid rate of exchange to withdraw potassium and fluid. Intermittent positive pressure ventilation was applied in order to avoid fatal respiratory embarrassment.

Twenty-four hours after commencement of the dialysis frequent extrasystoles were noted, soon followed by paroxysmal supraventricular tachycardia with intermittent A-V block confirmed on ECG. The serum K was 3·7 mEq/litre. No further Digoxin was given, the potassium concentration in the dialysis fluid was increased to 5 mEq/litre and intravenous potassium was administered.

Severe hypertension developed after a blood transfusion despite a considerable negative water balance.

At the end of the 40-hour dialysis severe metabolic alkalosis was present with the Astrup: pH 7·61 PCO_2 31·5 mmHg; base excess + 10 mEq/litre and standard HCO_3 33 mEq/litre. Attempts to discontinue IPPR resulted in prolonged apnoea and he could not be detubated till 24 hours later. Complete recovery occurred over the next 3 months.

Comment: The indications for dialysis were uraemia, hyperkalaemia, gross circulatory overloading and severe metabolic acidosis.

Digoxin is not removed by peritoneal dialysis and if thought to be necessary, a digitalizing dose not exceeding one-third and a daily maintenance dose of one-tenth the usual calculated dose must be used. Dialysis with K-free solutions is not continued for longer than 4 hours.

Blood transfusion in acute renal failure is best given when a considerable negative water balance has been achieved by peritoneal ultrafiltration. However, even this precaution does not necessarily prevent severe hypertension from occurring.

Metabolic alkalosis resulting from the conversion of lactate to HCO_3 is rarely of any concern, but in this instance was associated with severe respiratory depression.

Case 3

A 13-month-old White boy was admitted with a 3-day history of vomiting and bloody mucoid diarrhoea. He was mildly dehydrated, acidotic and had clinical evidence of a bleeding tendency and mild jaundice on admission. Laboratory investigations confirmed the clinical diagnosis of the haemolytic-uraemic syndrome. The blood urea was 128 mg/100 ml; serum electrolytes were normal; Astrup: pH 7·30; PCO_2 13 mm/Hg; base excess −18·5 mEq/litre and standard HCO_3 11·3 mEq/litre. The haemoglobin was 10·6 g/100 ml, white cell count 31 800/mm^3; platelet count 15 000/mm^3, reticulocyte count 5%, prothrombin index 52%, partial thromboplastin time 139 seconds and plasma fibrinogen level was 75 mg/100 ml. 'Burr' cells and schistocytes were seen on a bloodsmear.

The boy was rehydrated and an acute renal failure regime was followed. Twenty-four hours after admission he was anuric and had intermittent uraemic twitching of the extremities, soon followed by a brief generalized convulsion. The blood urea had risen to 208 mg/100 ml and continued to rise initially despite the institution of peritoneal dialysis.

During the 32-hour peritoneal dialysis mild dehydration recurred and hypokalaemia developed. This was attributed to the continuing diarrhoea and inadequate replacement of the losses sustained via this route.

Oliguria lasted 14 days but further dialysis was not necessary and ultimate full recovery occurred.

Comment: The indications for dialysis were uraemic syndrome, hypercatabolic renal failure and severe metabolic acidosis.

Despite the severe bleeding tendency no excessive bleeding occurred during the peritoneal dialysis. Dialysis with the 1·5% dextrose dialysis solution usually achieves a negative water balance of about 5%. Many cases of acute renal failure are oedematous and thus withdrawal of excess fluid is indicated, but in cases such as this accurate replacement of all losses becomes very important.

Case 4

An 11-year-old non-White girl had been treated for apparent acute glomerulonephritis for 3 days before being referred for a deteriorating level of consciousness, increasing dyspnoea and persisting oliguria.

On admission she was markedly oedematous, semi-comatose, severely acidotic and there was clinical evidence of pericarditis with a moderate pericardial effusion. Diffuse abdominal tenderness was present; a surgical cause for this was excluded, however.

Laboratory investigations: blood urea 318 mg/100 ml; serum electrolytes normal; capillary blood pH 6·98; PCO_2 38·5 mmHg; base excess> −23 mEq/litre, standard HCO_3 10·4 mEq/litre, haemoglobin, 6·0 g/100 ml and a white cell count of 15 500/mm^3. Examination of a urine specimen revealed 3+ proteinuria, gross pyuria and cylindruria and a growth of Klebsiella and *Proteus morganii* organisms on culture.

Peritoneal dialysis was commenced soon after admission. Inadequate return of the dialysate necessitated re-insertion of a new catheter at a different site. Hypertonic (4·25%) dialysis solution was used for 8 hours by which time a negative water balance of 1 200 ml had been achieved.

Subsequent evaluation revealed chronic pyelonephritis with considerable permanent loss of renal function. The uraemic pericarditis persisted for 2 weeks.

Diagnosis: Acute exacerbation of chronic pyelonephritis.

Comment: The indications for dialysis in this case were the uraemic syndrome with uraemic pericarditis and severe metabolic acidosis.

The metabolic acidosis of renal failure is readily corrected by peritoneal dialysis.

Ascertaining adequate return of the dialysate at the start of a dialysis and the use of Heparin in the dialysis fluid will prevent most difficulties with drainage, but on occasion insertion of a new catheter is nevertheless required.

It is important to exclude intra-abdominal surgical pathology before commencing peritoneal dialysis.

Case 5

A 7-year-old non-White boy was admitted with severe hypertensive encephalopathy, transient blindness, uraemia and hyper-

kalaemia. The presence of impetigo and laboratory evidence of a recent streptococcal infection suggested a diagnosis of acute post-streptococcal glomerulonephritis. The blood pressure was 200/140 mmHg. Pertinent laboratory investigations: blood urea 332 mg/100 ml, serum K 7·7 mEq/litre. The urine specimen showed 2+ proteinuria and on microscopy revealed numerous red blood cells and red-cell and granular casts.

The hypertension was controlled with antihypertensive drug therapy and peritoneal dialysis was commenced soon after admission. Following a 36-hour dialysis the boy remained anuric and evidence appeared for a micro-angiopathic haemolytic anaemia with thrombocytopenia, necessitating 4 blood transfusions.

Two further peritoneal dialyses for the control of uraemia were necessary in the following 2 weeks. An open surgical renal biopsy performed immediately after the third dialysis showed the histopathological features of a rapidly progressive glomerulonephritis.

Diuresis commenced on the 18th day and he ultimately made a good clinical recovery. Clinical and laboratory evidence of progressive glomerulonephritis is present 9 months after discharge.

Comment: The indications for dialysis were uraemia and uncontrolled hyperkalaemia.

In our experience hypertension occurring in renal failure is not much reduced by peritoneal ultrafiltration and anti-hypertensive drug therapy remains necessary.

The boy was given a modified Giovannetti diet following the first dialysis, but his serum albumin remained depressed at about 2 g/100 ml during the next three weeks, though urinary protein losses were minimal due to the oligo-anuria. Protein loss has varied from 6 - 60 g per dialysis on the few occasions we have estimated this. Replacement is indicated if repeated dialysis is necessary. We tend to give oral protein in the form of a modified Giovannetti diet early on during the course of acute renal failure and we usually give additional eggs if repeated dialyses have to be performed.

The dialysis catheter was inserted via a small incision initially. Inadequate closure of the peritoneum probably caused the mild leakage into the subcutaneous tissues which occurred during the second dialysis.

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