

Peritoneal dialysis in acute kidney injury

Brett Cullis

Correspondence Email: brett.cullis@gmail.com

INTRODUCTION

Renal failure requiring renal replacement therapy (RRT) is common and occurs in approximately 6% of hospital admissions, but that number is closer to 35% in intensive care units (ICU). Very little is published about the use of peritoneal dialysis (PD) in acute kidney injury (AKI) due largely to the predominance of publications from high-resource countries, where the high cost of continuous veno-venous therapies is not prohibitive. However, many practitioners from poorly resourced settings have used peritoneal dialysis for a number of years and are well aware of its value. There are a number of reasons why PD may be as good as, and more appropriate than, other forms of RRT for treating patients with acute kidney injury in the ICU (Table 1). In developing countries the cost of PD is often significantly lower than that of haemofiltration, thus allowing more efficient use of precious resources.

BASIC PRINCIPLES OF PERITONEAL DIALYSIS

The peritoneal space is filled with approximately 100ml fluid in normal states and is lined by both visceral and parietal peritoneum. This peritoneal membrane is made up of three components – the mesothelium, interstitium and capillaries.

The mesothelium acts as a protective barrier, but also

increases the surface area of the membrane through villous processes (finger-like projections from the mesothelium). This means the average peritoneal membrane surface area is approximately 20m². It has no role in regulating flow of solute. The interstitium is the matrix which holds the membrane together. The capillaries are the site of selective movement of solutes and water - i.e. the capillary walls **ARE** the semipermeable membrane.

Solute removal

Dialysis occurs through both diffusion and convection. Diffusion is the selective movement of a solute down a concentration gradient through a semipermeable membrane. As an example consider a tea bag from which tea moves into the surrounding water. Convection is the movement of solute with water through a semipermeable membrane, also called solute drag – squeeze a tea bag and more tea comes out with the water. Unlike diffusion this is not a selective process and there is no control over which solutes are removed (as in haemofiltration). Diffusion is responsible for movement of small solutes (K⁺, urea, creatinine etc), convection is the predominant mechanism for movement of proteins and large molecules.

Table 1. Relative benefits of peritoneal dialysis and haemofiltration.

Peritoneal dialysis	Haemofiltration
Cheaper	More ability to control ultrafiltration rate
Biocompatible membrane	Suitable for patients post laparotomy
Cardiovascular stability	
No need for vascular access	
No anticoagulation	
No specialized equipment or nursing required	
More rapid recovery of renal function than haemodialysis	
Easy transition to long term PD	

Summary

The author describes their first-hand experience with this technique in a resource poor setting. Although published trials of its efficacy and safety are few, the significantly lower cost of PD make it the ideal option in a low resource setting.

Brett Cullis

Consultant Nephrologist and Intensive Care Physician
Royal Devon and Exeter NHS Trust
Exeter
UK
and
Greys Hospital
Pietermaritzburg
South Africa

How do we achieve these two methods of solute removal?

The PD solution is instilled into the abdominal cavity, causing potassium, urea and creatinine to move from the serum, where levels are high, to the peritoneal fluid where levels are low. Water also moves into the PD fluid by osmosis (see later) and this drags larger molecules with it. Therefore, in order to increase the amount of solute removed, we need to continually replace the fluid that has equilibrated with serum, with new solution to reset the concentration gradient.

A very important factor in achieving clearance of solutes is the time the fluid is in contact with the peritoneal membrane. Therefore time spent draining the dialysis fluid in and out is not effective dialysis time and therefore this needs to be kept to a minimum (see 'Dialysis prescription').

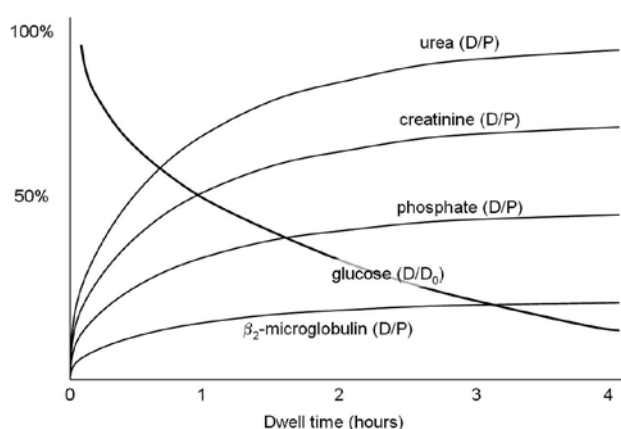


Figure 1. Time taken by various solutes to equilibrate with the serum; D/P = dialysate/plasma ratio; D/D_0 = the ratio of dialysate glucose concentration compared to concentration at time zero.

Fluid removal

Fluid is removed by osmosis. This requires a crystalloid osmotic pressure gradient and this is achieved through the addition of glucose to the PD fluid. Other agents such as amino-acids and icodextrin are also used but not in PD for acute kidney injury.

The higher the concentration of glucose, the more water will be transported into the peritoneal space by osmosis. Unfortunately glucose also diffuses down its concentration gradient in the opposite direction (into the patient) and so, over time, the osmotic gradient falls. If left long enough the glucose level will be so low that there will be a net absorption of fluid by the patient.

Increased fluid removal requires a dialysate with a higher glucose concentration, or reduction in the cycle time, in order to ensure no reabsorption has occurred.

PERITONEAL ACCESS

Originally acute peritoneal dialysis was achieved through various designs of rigid and flexible catheters. The desired features of an acute PD access device are that there is maximal hydraulic flow and minimal interaction with the peritoneal space. It should be simple to insert and reduce the chances of leaks and peritonitis.

There are two commonly used devices used today. The rigid 'stick'

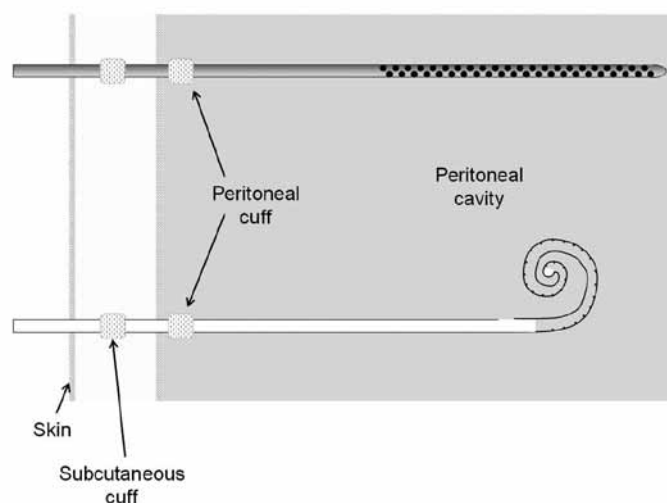


Figure 2. Tenckhoff catheters.

catheter is a plastic catheter, mounted on a stylet that is introduced through a subumbilical incision and directed into the pelvis before the stylet is removed. These catheters are prone to complications and, as they have a narrow lumen, flow of dialysate is sluggish. They also frequently get blocked with fibrin and require flushing. This has the potential to facilitate introduction of bacteria and cause peritonitis. The rigidity of the tube also means that leakage and hemorrhage, due to vessel erosion, are relatively common.

An advantage of these catheters is that they are very easy to insert and the technique can be performed by unskilled staff. They are also cheaper than the more flexible Tenckhoff catheters (see below) but are much less efficient. It is the author's belief that these catheters should only be used if there is no option to use a flexible catheter.

The flexible Tenckhoff catheter is a silastic catheter with two Dacron cuffs and either a straight or coiled end (see Figure 3). It can be inserted by a percutaneous approach at the bedside if there are no contraindications, such as:

- Midline surgical scar
- Previous abdominal TB
- Vertical incision for Caesarean section
- Complicated appendicitis/cholecystectomy.

If contraindications are present, insertion should be done by a surgeon to ensure there is no bowel adherent to the anterior abdominal wall. The percutaneous approach is through a sub-umbilical incision using a guidewire and 'peelaway' sheath, through which the catheter is introduced. The catheter is then tunneled under the skin to facilitate patient care and prevent leaks (Figure 3).

DIALYSIS PRESCRIPTION

There has been concern over the ability of PD to provide adequate clearances in patients with AKI, especially when compared to haemodialysis. This may be true in those cases where a rigid catheter is used and the amount of PD fluid instilled is small. However clinicians in Brazil and India have shown that it is possible to get clearances of urea in excess of those seen with intermittent haemodialysis.^{1,2} Gabriel

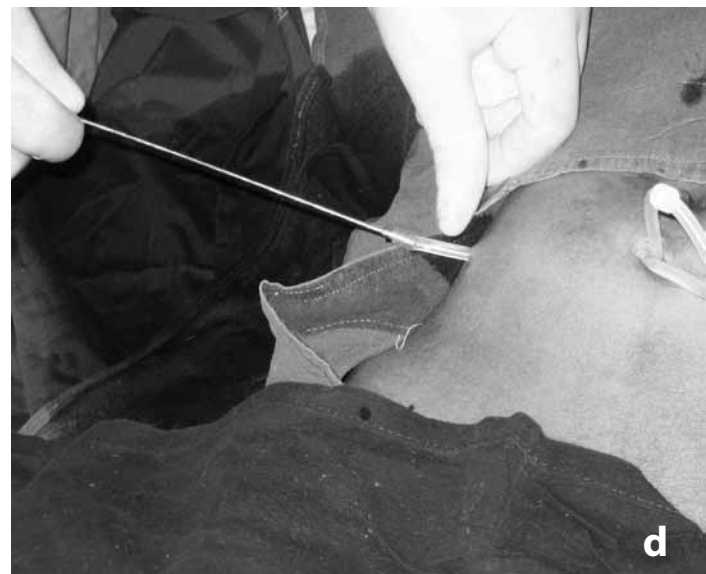
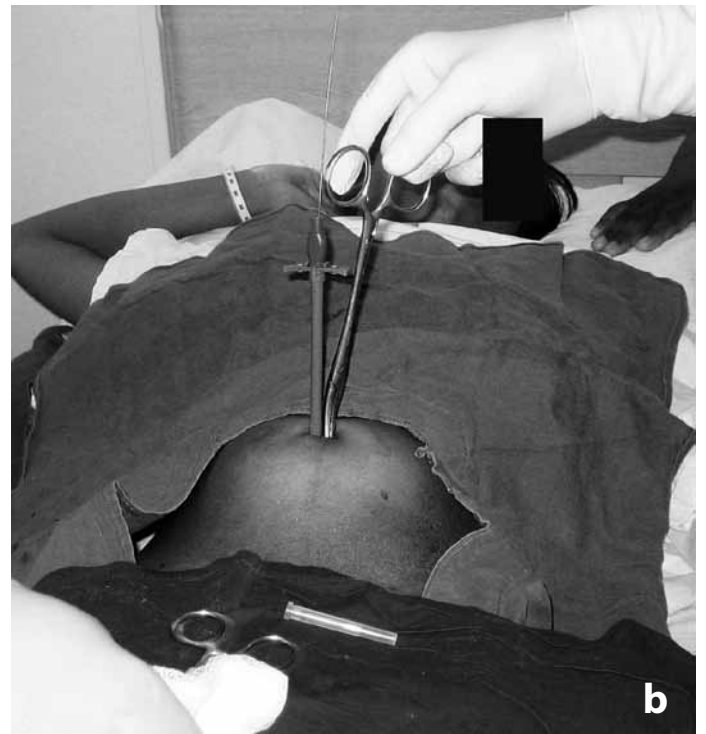


Figure 3. Percutaneous insertion of a Tenckhoff catheter; (a) guidewire inserted through needle or cannula into peritoneal cavity; (b) 'peelaway' sheath inserted with dilator over guidewire; (c) dilator removed and catheter inserted through sheath that is then peeled away; (d) catheter is tunneled under skin.

and Balbi in Brazil randomized critically ill patients with AKI to daily haemodialysis or PD. The mortality was no different and patients recovered renal function 3 days earlier when using PD.¹ It is also possible that clearances of cytokines from the blood may be greater due to the large pores, but this has not yet been confirmed.

The dialysis prescription needs to take into account two factors:

1. Is the patient fluid overloaded?
2. Do they need rapid correction of hyperkalaemia or acidosis?

The dialysis fluid comes in three strengths – 1.36% (1.5%), 2.27% (2.5%), and 3.86% (4.25%) glucose (with the equivalent dextrose

figures in brackets). The higher the concentration of glucose the more fluid is removed into the dialysate. Therefore in a patient with pulmonary oedema due to fluid overload, 3.86% fluid should be used. If the patient is dehydrated, then 1.36% fluid is indicated. It is not only the strength of the fluid, but also how rapidly the cycles are performed, that determines the rate of fluid removal. With increased frequency of fluid exchanges, more fluid is removed. This cycle time should not be less than one hour as the time spent draining in and out becomes greater than the time spent dialysing.

For patients with hyperkalaemia or acidosis, again it is the frequency of fluid exchanges that determines correction of the abnormality. In life

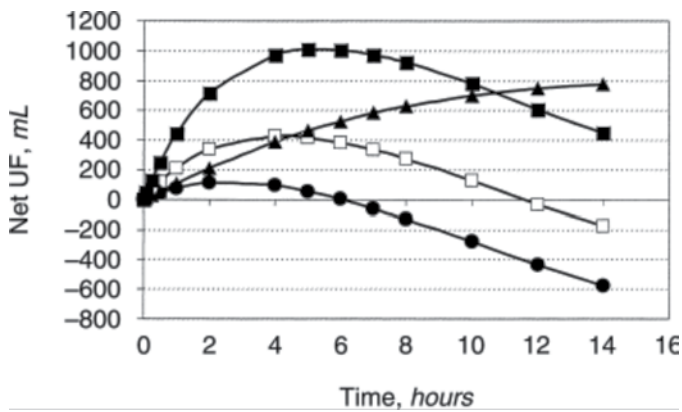


Figure 4. Ultrafiltration profiles of 1.35% (closed circles), 2.27% (open circles) and 3.86% (closed squares) glucose (from reference 3)

threatening disease hourly exchanges can be done until the potassium and pH levels are within a safe range. If hyperkalaemia or acidosis is not severe then two-hourly exchanges are preferable. Usual practice is to continue two-hourly exchanges until the potassium and pH are in the normal range (usually 24 hours) then change to 4 hourly exchanges. This facilitates the clearance of larger molecules and cytokines. It also reduces the cost of dialysis. Remember dialysis will NOT correct a lactic acidosis; this requires management of the precipitating cause.

The volume of fluid is dependant on the size of the patient. The following are recommended:

Table 2. Recommended volume exchanges.

Weight (kg)	Exchange volume (ml)
<50	1000
51-80	1500
>80	2000

Table 3. Suggested dialysis prescription.

	Fluid overload	No fluid overload
K+ > 6.5	Hourly 3.86%	Hourly 1.36%
pH < 7.1	Hourly 3.86%	Hourly 1.36%
K+ < 6.5 and pH > 7.1	Hourly 3.86%	2 Hourly 1.36%
Day 2 onwards	4 hourly 3.86%	4 hourly 1.36%

COMPLICATIONS

Haemoperitoneum is common after the catheter has been inserted, but should clear after a few exchanges. Peritonitis occurs mainly due to touch contamination of the end of the catheter. Bacteria are then

flushed into the peritoneal space. The pH of the PD fluid impairs macrophage function and makes infection more likely. It is therefore imperative that nursing staff understand the importance of cleaning their hands well and not allowing the tip of the catheter to touch anything unsterile. Peritonitis is diagnosed by cloudy PD effluent and a white cell count on the PD fluid of greater than 100ml^{-1} . In acute PD it can often be detected early by doing daily urine dipstix on the fluid. If dipstix shows 2+ leukocytes then 10ml effluent fluid should be sent to the laboratory in blood culture bottles, in order to isolate the causative organism.

If over 100 white blood cell per ml are seen then start empiric antibiotics using vancomycin or a 1st generation cephalosporin (to cover gram positive organisms) and either ceftazidime or an aminoglycoside to cover gram negative organisms including pseudomonas. They should be added to each bag using a sterile technique to inject them. If the range of antibiotics available is limited, then gentamicin is probably the best choice to cover *Staphylococci* (usually coagulase negative) and Gram negative organisms. Some sites find that amikacin provides better cover.

If the bags have not become clear by day 3, or if the patient develops sepsis with no other evident source, or if the culture reveals a fungal infection, the catheter should be removed.

Fibrin is commonly found in the fluid of patients on acute PD and

Table 4. Antibiotic dosing for peritonitis.

Vancomycin	Load 1g.L^{-1} then 25mg.L^{-1}
Cefazolin	Load 500mg.L^{-1} then 125mg.L^{-1}
Ceftazidime	Load 500mg.L^{-1} then 125mg.L^{-1}
Amoxicillin	125mg.L^{-1} in all bags
Oxacillin	125mg.L^{-1} in all bags
Chloramphenicol	500mg - 1g 6hrly IV
Gentamicin	Load 8mg.L^{-1} then 4mg.L^{-1}

often contributes to blocking of catheters and poor drainage. This can be prevented by adding 500 units of heparin to each litre of PD fluid.

If the catheter blocks try flushing with 20ml 0.9% saline, using a sterile pack and gloves. If that does not work then passing a central venous catheter guidewire down the catheter may work. It may also be that the catheter is wrapped in omentum in which case it will need to be removed, but a new catheter can be inserted through the same incision.

Hypokalaemia can occur with rapid exchanges used to treat pulmonary oedema. If the potassium falls, add 4mmol KCl to each litre of fluid. This will not result in hyperkalaemia as the potassium will only equilibrate and extra potassium will not be absorbed. Hypernatraemia may occur with rapid cycling. If the patient is not fluid overloaded, 5% glucose can be infused intravenously to maintain normal sodium levels.

CONCLUSION

Peritoneal dialysis offers significant advantages over haemodialysis and haemofiltration in its simplicity, cost effectiveness, lack of need for expensive machinery and more rapid recovery of renal function. There is evidence of similar outcomes when compared to haemodialysis and filtration, although larger trials are needed.

It should be considered in all centres where haemofiltration cannot be offered or costs are prohibitive.

REFERENCES

1. Gabriel DP, Balbi AL. Response to high-volume peritoneal dialysis in acute kidney injury. *Kidney Int* 2009; **76**: 1117.
2. George J, Varma S, Kumar S et al. Comparing continuous venovenous hemodiafiltration and peritoneal dialysis in critically ill patients with acute kidney injury: A pilot study. *Perit Dial Int* 2011; **31**: Epub before print.
3. Mujais S, Vonesh E. Profiling of peritoneal ultrafiltration. *Kidney International* 2002; **46**: 496-503.