CHAPTER 179

Indications, Contraindications, and Complications of Peritoneal Dialysis in Acute Renal Failure

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OBJECTIVES

This chapter will:

1. Present data on the clinical effectiveness of peritoneal dialysis (PD) in treatment of acute renal failure.
2. Compare clinical benefits of acute PD with those of other modalities of dialysis for support of patients with acute renal failure.
3. Explain some particular benefits of PD, especially in removal of middle-molecular-weight toxins, protein-bound toxins, and fluid.
4. Detail the clearance benefits of continuous-flow PD and methods by which it can be performed currently in patients with acute renal failure.
5. Discuss unique problems, risks, and limitations of PD in the treatment of acute renal failure.
6. Define special training needed in the hospital for staff to provide PD, and also how the training overlaps with that for "urgent start" PD therapy for patients with end-stage renal disease.

When choosing treatment for an adult with acute kidney injury (AKI), nephrologists generally think of continuous renal replacement therapy (CRRT) or intermittent hemodialysis (HD), but not peritoneal dialysis (PD).[1] This tendency makes acute PD a considerably *underused* therapy.[2] Stated another way, given the risks and universal side effects of HD and the failure of continuous venovenous hemodialysis (CVVHD) to be truly "continuous," perhaps these therapies are *overused* in treatment of AKI. Recently, "urgent start" PD with almost immediate use of a Tenckhoff catheter has been implemented as a safe, efficient, and effective therapy in treatment of patients with end-stage renal disease (ESRD) starting unplanned dialysis in a hospital or outpatient clinic.[1] In AKI, the continuous fluid removal of PD makes it appropriate for many of the same patients for which CRRT is chosen: those with heart failure, hypotension, and low cardiac index who cannot tolerate the rapid fluid removal rate of standard HD. If "urgent start" PD is effective initial and long-term therapy for patients with advanced ESRD, should it not also be effective for many patients with AKI?

As with all dialysis procedures, PD first was used in therapy of AKI.[3] PD was once a common choice for treatment of AKI in adults even after HD was also an option in most hospitals.[4,5] PD is still the mainstay in treatment of AKI in infants and children.[6,7] As with acute HD, the access type is critical, and the safest and most effective PD is done with chronic tunneled dialysis catheters, in AKI and ESRD. In this chapter, we compare PD with HD and CRRT in treatment of AKI, analyzing differences in outcomes, incidence of renal recovery, complications and risks, chemical efficiency (with potential improvements), and training needs for a PD program in the hospital. We also discuss the importance and efficiency of using only tunneled and cuffed catheters for PD in the hospital, whether for treatment of AKI or ESRD.

OUTCOMES

Regarding patient outcomes, a number of prospective and retrospective studies and some randomized studies have compared mortality rates of patients with AKI when treated with PD versus with HD or CVVHD. Although the patient populations in the various studies and treated groups are not exactly the same, and there is some selection bias in patients that may favor PD, most studies have shown outcomes to be at least as good for PD as for HD, as shown in Table 179.1.[8–15]

Except for the study by Phu et al.,[12] all studies summarized in Table 179.1 have shown that in patients with AKI treated with PD, mortality and incidence of renal recovery are roughly equivalent to those in similar patients treated with HD. Firmat and Zucchini,[9] reviewing literature reports involving more than 1100 patients, concluded that the mortality rate was identical for patients with AKI receiving PD and those receiving HD. Most of these studies were performed in the 1970s and 1980s and many used acute PD catheters rather than chronic tunneled catheters and open systems for draining the dialysate. However, Struijk continued analyzing patients treated with each modality at the Academic Free Hospital in Amsterdam from 1986 through 1999, using tunneled catheters and closed drainage systems (DG Struijk, personal communication, 2000). In his studies, mortality in patients treated with PD was identical to that of patients treated with HD.[11] In the study by Phu et al.,[12] 70 adult patients with AKI, because of severe falciparum malaria in 48 and sepsis in 22, were assigned randomly to treatment by PD or by continuous venovenous hemofiltration (CVVH). The mortality was significantly higher in the group treated with PD (47%), as well as a lower rate of renal recovery than in the group treated with hemofiltration (mortality 15%). The PD schedule was very aggressive (70 L of fluid per day) and was performed using acute PD catheters and open drainage containers. Urea clearance of PD was about equal to that with CVVH, but creatinine clearance with PD was about half that with CVVH.[12] What is most unusual about the Phu study is the exceedingly low mortality of the group treated with CVVH, rather than an unusually high mortality in the group treated with PD. As Daugirdas[16] pointed out, it is possible that the heparin anticoagulation of CVVH was of benefit to the many patients with malaria in this study. Also, hyperglycemia accompanying PD may have stimulated malarial growth in the liver or red cells, or high osmolality may have diminished white cell function. Failure to correct acidosis, a serious problem in the PD group, may have been due to use of acetate as buffer in the PD solution, as opposed to lactate in the CVVH infusion fluid.

Gabriel performed a randomized study of patients with AKI, treating 60 patients with daily HD and 60 with PD.[13] Patients were excluded if they had absolute contraindications to either therapy. Peritoneal access was a Tenckhoff catheter placed by trocar by a nephrologist. Mortality over a 30-day period was almost identical in the two groups. George randomized 50 patients with AKI, 25 to PD and 25 to CVVHD.[14] Access was by a rigid, acute PD catheter. Mortality was statistically the same in both groups. Ponce randomized 143 patients with AKI to treatment by PD or extended daily HD.[15] In-hospital mortality was identical in the two groups and in fact odds-ratio of mortality favored patients treated with PD.

Several of the studies summarized in Table 179.1 were randomized and prospectively controlled, and yet there was still some bias in patient selection. However, this bias worked for and against patients treated by PD. Patients with AKI who have abdominal trauma, are awaiting abdominal surgery, or have abdominal drains or severe ileus cannot be treated by PD. In general, however, patients with AKI after surgery have a higher rate of recovery from AKI than patients with other causes of AKI, such as sepsis and shock. In these studies, PD often was chosen for patients with hypotension or cardiovascular instability that would make HD dangerous, a practice that also selected a group with a potentially worse outcome. The techniques used for PD in most of the studies were antiquated by today's standards. In many studies including that by Phu, semirigid acute PD catheters were used rather than Tenckhoff catheters. Acute PD catheters have irregular outflow characteristics, and most are removed and re-inserted every 3 days. Each insertion increases the risk of bowel puncture and outflow failure. PD fluid was infused from bottles in many of the studies, and there were no Y-sets to allow drainage and infusion of PD fluid though a single catheter connection. In spite of the use of rather crude techniques in many of these studies, PD patients recovered renal function and survived at least as frequently as patients treated with HD, with the notable exception of the study by Phu et al.[12]

TABLE 179.1

Comparison of Mortality in Acute Renal Failure: PD Versus HD or CVVHD

STUDY[a]	YEAR	NO. OF PATIENTS	MORTALITY (%)	
			PD	HD or CVVH
Orofino et al.[8]	1976	82	52	62
Firmat[9]	1979	1101	50	50
Ash[2]	1983	97	38	48
Swartz[10]	1980	77	44	60
Struijk[11]	1980	45	45	(same)
Struijk[a]	1986–1999	50	78	(same)
Phu[12b]	2002	70	47	15 (CVVH)

[a]Personal communication, 2000.
[b]All hypercatabolic patients.
Superscript numbers indicate references.

RENAL RECOVERY

In many of the studies comparing PD with HD for AKI, the improved survival in the PD group correlated with a higher rate of renal recovery. Of all patients with AKI few recover general health but fail to recover renal function, leaving

the hospital with ESRD to be supported by dialysis (less than 10%). In patients with ESRD, treatment by continuous ambulatory PD (CAPD) results in better preservation of intrinsic renal function than treatment with HD.[17–20] This preservation of renal function is important in ESRD because it maintains the endocrine function of the kidneys, diminishes the clearance requirements for adequate dialysis, minimizes required ultrafiltration during dialysis, and thus diminishes physiologic stress during dialysis. Intermittent HD is known to have the following nephrotoxic effects: (1) generation of inflammatory mediators by the extracorporeal circuit, (2) concomitant and rapid decrease in osmolality and vascular volume, which diminish renal perfusion, and (3) hypotensive episodes, which result in fresh ischemic lesions in the kidneys.[21]

By contrast, the effects of CAPD therapy help maintain renal perfusion, because of the following: (1) smaller daily variation in body weight, (2) more constant blood pressure, (3) continued mild overhydration, with higher mean pulmonary arterial pressure,[22] (4) persistent high blood osmolality, partly because of glucose, and (5) continued removal of proteins from the blood (including β_2-microglobulin, albumin with associated uremic toxins, plasminogen activator inhibitor type 1, and immunoglobulins).[16,23,24]

Given the beneficial effects of PD, it is not surprising that some patients started on PD for what appears to be ESRD recover intrinsic renal function and no longer need dialysis (3.3%). Given the negative physiologic effects of HD, it is also not surprising that very few patients with ESRD who are treated with HD recover renal function (0.8%).[22] Recovery of renal function is most common in patients whose renal failure was caused by uncontrolled hypertension, cardiac failure, nephrotic syndrome, rapidly progressive renal failure, analgesic nephropathy, urinary obstruction, or cholesterol emboli.[22] Many of these underlying conditions are better corrected by CAPD than by HD, because of continuous chemical removal, better preservation of renal perfusion and glomerular filtration rate, and slow removal of immunoglobulins. These same physiologic and chemical benefits may account for the higher recovery of renal function, in most studies, in patients with AKI treated with PD than with HD.[16–19,25]

COMPLICATIONS

There is general consensus that continuous dialysis therapies such as CVVH and CVVHD are the most efficient therapies for AKI, with the fewest adverse physiologic effects. These "gentle" forms of therapy remove fluid at a slow rate and cause less decrease in cardiac output than HD, especially when HD is performed for only 3 to 4 hours daily or every other day. CVVH and CVVHD do not affect pulmonary function adversely or significantly activate the complement cascade.[7] Continuous arteriovenous hemofiltration (CAVH) was described first in 1967.[26] Pump-assisted CVVH and CVVHD were developed to make the rate of blood flow through the hemofilter more consistent, improve clearances, eliminate problems and risks of arterial access, and render the therapy more nearly continuous. Continuous extracorporeal blood therapies also have a number of disadvantages in comparison with HD and PD. CVVH and CVVHD require considerable attention from nurses to ensure adequate blood flow, monitor anticoagulation status, adjust ultrafiltration rate, and calculate patient fluid balance. The patient is immobilized during the treatment. Continuous heparin administration increases risk of bleeding. Central venous or femoral catheters often provide insufficient blood flow and carry risks for infection and sepsis.[5–7] In spite of the name and intent, the average duration of individual treatments with "continuous" blood therapies is 20 hours before clotting of the system or need to transport the patient for diagnostic or therapeutic procedures. Some centers previously performing CVVH or CVVHD for acute dialysis have begun using sustained low-efficiency dialysis with extended duration (SLED, 6–8 hours/day) to avoid many of the problems of CVVH or CVVHD, limiting heparin use and immobility of the patient while keeping the advantages of vascular stability and improved clearances.[27] By contrast, PD is a truly continuous dialysis therapy associated with potentially fewer risks. It requires less nursing effort than CVVH, CVVHD, or SLED and allows more patient mobility during therapy.

CLEARANCE OF UREMIC TOXINS

Standard Single-Access Peritoneal Dialysis

The major criticism of PD, of course, is a low clearance of uremic toxins, and clearance of low-molecular-weight uremic toxins is generally lower than that with other therapies. For continuous PD therapies, the time-averaged clearance is the same as the immediate clearance. For intermittent therapies, the time-averaged clearance is diminished in proportion to the time between dialysis sessions. Although blood urea nitrogen (BUN) clearance is lower with PD than with either CVVH or HD, creatinine clearance with PD can be close to that with CVVH if the peritoneal fluid volume flow is high enough. Figure 179.1 compares blood urea nitrogen and creatinine levels in a patient treated with high-volume PD, then CVVH, then HD.[28] Although the blood urea nitrogen level rose to approximately 80 mg/dL during days of PD, the serum creatinine values during PD and CVVHD were comparable and both were lower than the creatinine value during intermittent HD.

The clearance of PD appears more effective when considering removal of middle-size uremic toxins. Clearance of phosphorus on PD is close to that for creatinine. PD is effective in removing various protein-bound anionic organic compounds that function as middle molecules (Fig. 179.2).[23] In AKI, clinical experience confirms that PD results in

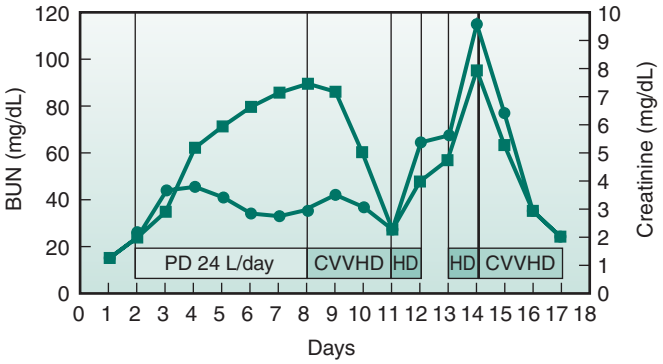

FIGURE 179.1 Blood urea nitrogen (BUN; *circles*) and serum creatinine *(squares)* concentrations in a single patient with acute renal failure treated successively with high-volume PD, continuous venovenous HD (CVVHD), and hemodialysis (HD). Rights were not granted to include this figure in electronic media. Please refer to the printed book. (From Amerling R. PD in treatment of acute renal failure. Presented at CRRT Conference, San Diego, 2005.)

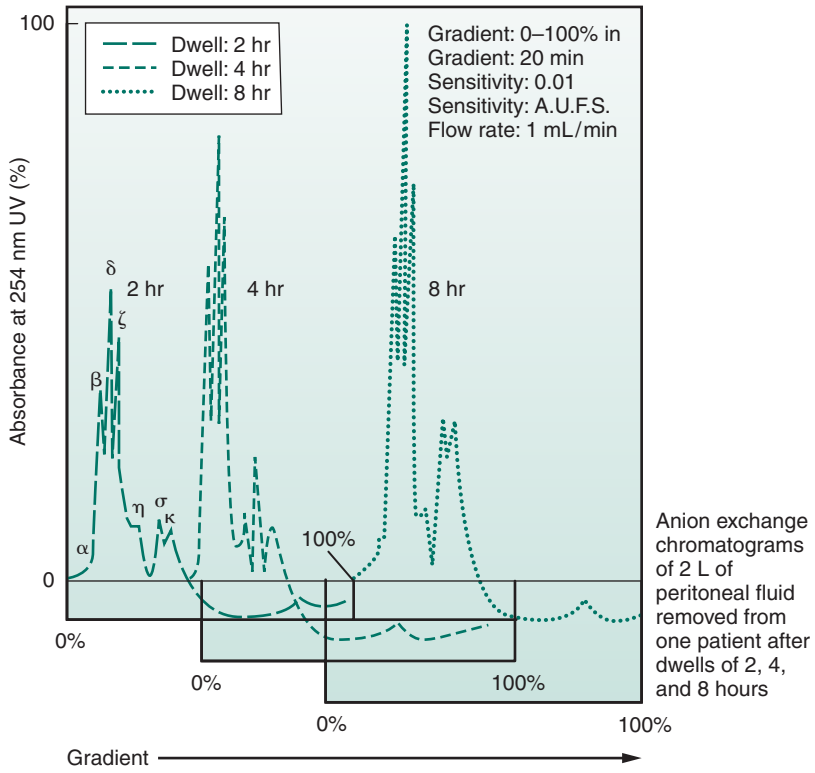

FIGURE 179.2 Increase in anionic organic compounds within peritoneal dialysis fluid, over periods of 2 hours, 4 hours, and 8 hours, in a patient with end-stage renal disease. Chromatograms generated by direct anion exchange chromatography, without protein removal. Rights were not granted to include this figure in electronic media. Please refer to the printed book. (From Ash SR, Bungu ATJ, Regnier FE. Dependence of middle molecular clearance on protein concentration of peritoneal fluid. In Maher JF, Winchester JF. eds. *Frontiers in PD.* New York: Field, Rich and Associates; 1986:56–63.)

equal or higher resolution of uremic symptoms, recovery of renal function, and patient survival in comparison with the other therapies (discussed). This clinical benefit may be due to effective removal of numerous middle-size organic molecules in PD.

In addition to removal of uremic toxins, of course, dialysis for AKI must remove fluid and salt from the patient. With a properly functioning PD catheter, exchanges of 2 L of dialysate with 2.5% or 4.25% glucose concentration provides daily fluid removal at a rate the same as or greater than that of HD or CVVHD, without causing hypotension in most patients. In patients with refractory congestive heart failure, fluid removal is the main goal, and with PD therapy and hypertonic dialysate, improvement in clinical symptomatology and left ventricular function is routine. In one study of 20 patients with resistant congestive heart failure, all improved after PD with only 12 inflow/outflow cycles.[29] As demonstrated by Gotch,[30] fluid removal rates with 2-L PD exchanges are initially 0.2 to 1.0 L/hr, depending upon the glucose concentration of the fluid (Fig. 179.3). With continuous-flow PD (CFPD; discussed later), fluid removal can be maintained at the initial rate of batch exchanges.

Just as with CVVHD, the small molecule clearance of PD can be increased greatly by raising the flow rate of dialysate to 1.5 to 2 L/hr or more. Tidal PD (TPD) easily can deliver 2 L/hr into and out of the peritoneum, and with use of a cycler, automated TPD is only a little more complicated than manual in/out exchanges. In one study of patients with AKI, a peritoneal dialysate flow rate of 2 L/hr produced an average normalized creatinine clearance of 68.5 L per week per 1.73 m^2 of body surface area (BSA) and a urea clearance value (Kt/V) of 2.43, versus average values for "equilibrium" PD, 58.9 L/wk/1.73 m^2 BSA and a Kt/V of 1.80. TPD and manual-exchange PD were adequate for treatment of patients with AKI and mild to moderate catabolism but insufficient in some patients with hypercatabolism.[31]

The fact that PD results in significant protein loss (5–20 g/day) generally is considered a nutritional problem.

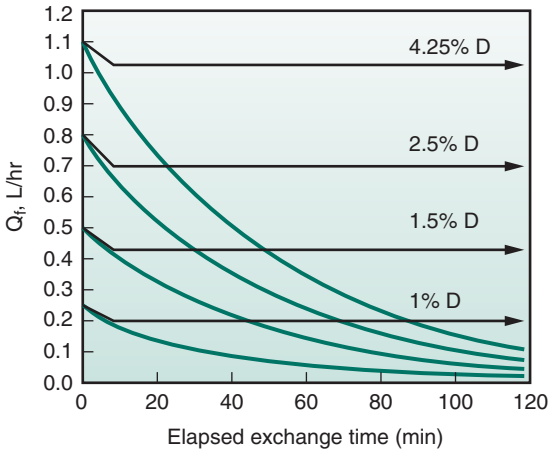

FIGURE 179.3 Fluid removal rate (Q_f) with batch exchanges of PD fluid *(green lines)* and continuous-flow PD (*black arrows*). (From Gotch FA. Kinetic modeling of continuous flow PD. *Semin Dial.* 2001;14:378–383.)

However, this loss of protein also contributes to the chemical effectiveness of the procedure. In patients with hemolytic uremic syndrome, PD significantly reduces plasminogen activator inhibitor type 1, which inhibits fibrinolysis in hemolytic uremic syndrome.[32] Most of the organic anions removed by PD in uremic patients in fact are bound strongly to protein, so protein output or "loss" increases their clearance.[23] These protein-bound organic anions act like middle molecules, because the protein binding restricts their passage across dialysis membranes; they are still accumulating in peritoneal dialysate at 8 hours of dwell time (see Fig. 179.2). The presence of protein within the dialysate facilitates the transfer of these compounds into the peritoneum. The peritoneal transfer of protein can be increased by use of hypertonicity/hypotonic exchanges and

pharmacotherapy. With this treatment the globulin removal by PD on a daily basis could equal or exceed that of daily therapeutic plasmapheresis.[24]

Continuous-Flow Peritoneal Dialysis

The chemical efficiency of PD can be greatly increased by implementation of an "old but good" idea, continuous-flow peritoneal dialysis (CFPD).[33] This modality uses two access points to the peritoneum, one for inflow of dialysate and the other for outflow. Flow rates are determined only by the rate at which the draining catheter can drain reproducibly the abdomen, and the peritoneum is filled continually with fluid. With CFPD, dialysate flow rates of up to 300 mL/min can be maintained through the peritoneum with clearances comparable to those of CVVHD.[34] With use of an external dialyzer to "regenerate" the dialysate, clearances for urea, creatinine, and urate average 57, 35, and 39 mL/min, respectively, in adult patients.[35] At 170 mL/min of dialysate flow, urea and creatinine clearances have averaged 31 and 23 mL/min, respectively.[25] Studies using dialyzer-regenerated PD fluid and dual Tenckhoff catheters have confirmed urea clearances of 50 mL/min or more in several patients with AKI.[36]

CFPD also has been used effectively for treating massive fluid overload, much like isolated ultrafiltration. In six pediatric patients with acute respiratory distress syndrome (ARDS) resulting from sepsis or systemic inflammatory response syndrome, CFPD at 10 to 30 mL/kg/hr with two Tenckhoff catheters resulted in an average 33% decrease in body weight and an improvement in alveolar-arterial oxygen gradient.[37] With CFPD, the time-averaged clearance of urea with PD theoretically can exceed that with daily 4-hour HD and come close to those with CVVH or CVVHD—approaching the K_oA or maximal clearance theoretically obtainable from the peritoneum, as shown in Figure 179.4.[30] Dialysate flow rates of 200 to 300 mL/min seem unrealistic today only because nephrologists are accustomed to using expensive, prepackaged dialysate and gravity flow. However, if PD machines reappear that proportion fluid on site, or if sorbent-based regenerative systems are commercialized[38–40] peritoneal dialysate will be available at just about any flow rate desired. Clearances of urea by CFPD may approach the theoretical limit of the peritoneal membrane and far exceed those of TPD (as in Fig. 179.4). However, as shown in this figure, there is a considerable variation in clearance from day to day with CFPD, especially at lower dialysate flow rates. This variation probably relates to changes in intraperitoneal (IP) volume during the therapy. In CFPD the volume of fluid infused is known and the volume drained, but not the ultrafiltration rate of the patient, so the actual IP volume is not known or controlled. Low IP volume results in diminished area of peritoneum recruited for chemical transfer, and high IP volume leads to channeling between catheters.[41] In an animal study, our laboratory demonstrated that there is an optimal peritoneal volume for highest efficiency of the peritoneum in CFPD, about 1.5 L in the dog.[42] During CFPD the IP volume can be determined intermittently by draining the peritoneum as is done with TPD. However, this requires cessation of inflow during the drain period, thus diminishing overall clearance. In a study of pediatric patients on CFPD the abdomen was drained every 4 hours to determine the UF rate. With this approach, CFPD was three to five times more effective for urea and creatinine clearance and ultrafiltration than conventional PD.[43] For CFPD to be a reliable and easy therapy, however, convenient methods must be found to estimate IP volume frequently.

One approach to measuring IP volume is to measure the IP pressure, which is related directly to IP volume. For individual patients the compliance of the peritoneum is relatively constant, especially when the patient is supine. For populations of patients there is a linear relationship between IP pressure and volume when expressed in mL/kg body weight but also a huge scatter of points (Fig. 179.5). The compliance curve can be measured for a single patient at the start of PD by using a sterile manometer to measure pressure as the abdomen is filled (stopping the flow to

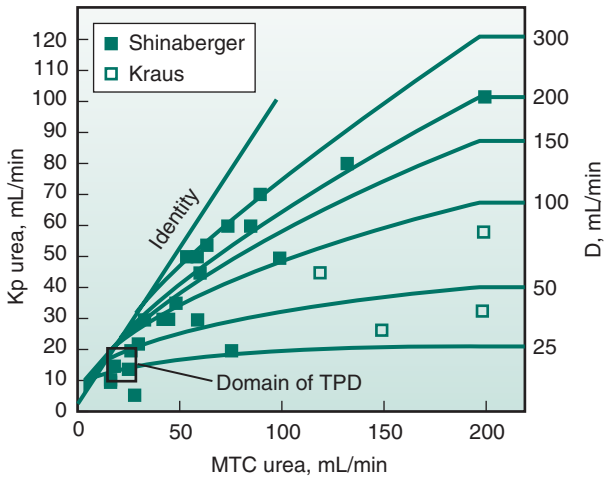

FIGURE 179.4 Analysis of published urea clearances (K_p) and peritoneal mass transfer coefficient (MTC) at varying peritoneal dialysate flow rates (D) during continuous-flow PD. (Data from Kraus; and Shinaberger et al.[33]) (From Gotch FA. Kinetic modeling of continuous flow PD. *Semin Dial.* 2001;14:378–383.)

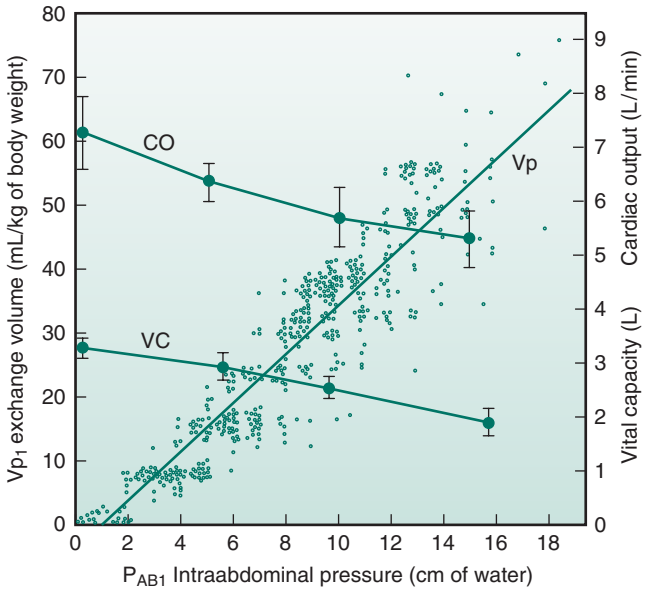

FIGURE 179.5 Relationship among IP pressure (P_{AB1}; measured versus umbilicus, with patient reclining), IP volume (Vp) vital capacity, and cardiac output. Rights were not granted to include this figure in electronic media. Please refer to the printed book. (From Ash SR, Carr DJ, Diaz-Buxo JA, Crabtree JH. Peritoneal access devices: Design, function, and placement techniques. In Nissenson AR, Fine RN. eds. *Clinical Dialysis.* 4th ed. New York: McGraw-Hill; 2005.)

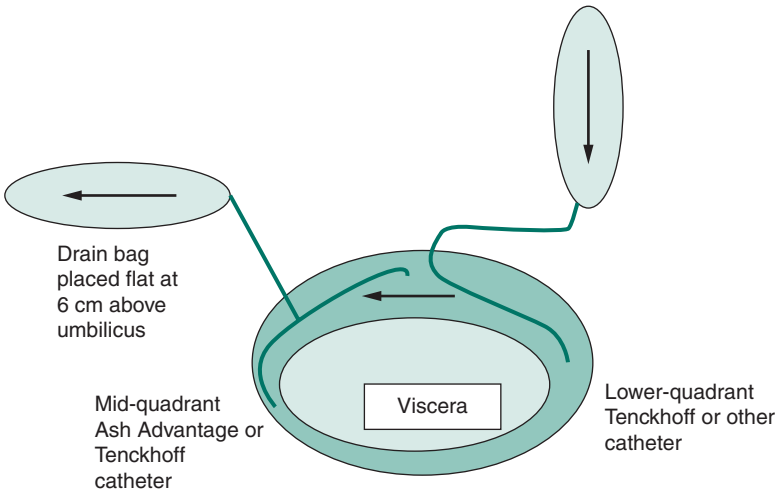

FIGURE 179.6 Simplified method for controlling intraperitoneal pressure and volume during continuous-flow peritoneal dialysis by placing the drain bag above the umbilicus.

measure the IP pressure). The patient should be in the same supine position as will be used during PD, and the "0" point of the manometer should be placed at the level of the umbilicus. The resulting compliance curve will define a goal pressure for each desired IP volume. Symptoms of overfilling relate to IP pressure rather than volume. At a goal IP pressure of 10 cm H_2O, there is only a modest decrease in cardiac output and vital capacity (see Fig. 179.5).[44,45] Problems with the "pressure" approach are that fluid flow must be stopped to make it completely accurate, and extra connections must be made in the PD circuit to accommodate the manometer.

In spite of problems with pressure as a measure of volume, it is reasonably easy to create a constant IP peritoneal pressure during CFPD in a supine and relaxed patient, as long as the infusion rate is modest (such as 15–50 mL/min) and the outflow PD catheter functions well. For access, one tunneled PD catheter is placed through the lateral border of the rectus and pointed laterally. Another is placed on the lateral border of the rectus on the opposite side and also pointed laterally. Infusion of peritoneal fluid through one catheter is performed at 15 to 50 mL/min (1 to 3 L/hr), and the outflow catheter is attached to a drain bag that lies flat upon a bedside stand placed 6 cm above the umbilicus (Fig. 179.6). This arrangement provides an IP pressure of 6 to 8 cm with a well-functioning outflow catheter. Fluid obtained by ultrafiltration collects in the drainage bag, and the ultrafiltration rate can be determined by intermittently weighing the PD drain bag and emptying it, then and subtracting the known weight of infused bags. This method is relatively simple to perform manually, and calculations are similar to those used for ultrafiltration measurement during CAPD.

Pressure-controlled CFPD using this system has been performed in a patient with acute renal failure.[33] CFPD has also been used in a full-grown horse with nonoliguric AKI because of myoglobinuria and a creatinine level of 16 mg/dL.[46] The horse recovered from renal failure and remained healthy long after discharge.

A more sophisticated method for determining IP volume is to use bioimpedance measurement. In 2003, Zhu, Levin et al. performed a study using eight skin surface electrodes and a meter to measure segmental bioimpedance analysis in patients on PD and correlate changes in the signal. Bioimpedance measurements using the resistance term alone (R) were able to measure IP volumes very accurately, within +/– 50 mL for IP volumes up to 3 L.[47]

IMPORTANCE AND PRACTICALITY OF TUNNELED CATHETERS

In AKI the success and risks of every dialysis therapy relate in part to the access devices.[48] If acute catheters are used in PD, each catheter can be used for only 3 days without high risk of peritonitis or bowel perforation, and each successive catheter placement carries a higher risk of these complications. For acute PD to be performed effectively and safely, a chronic tunneled PD catheter must be the access device. In pediatric patients with the use of a surgically placed Tenckhoff catheter, incidence of all types of complications during PD treatment of acute renal failure was only 9%, compared with 49% for patients in whom catheters were placed percutaneously at the bedside.[49] In the study by Phu showing high mortality in PD patients (Table 179.1), all PD therapy was done with acute PD catheters.[12] Chronic PD catheters were used in the studies by Ash et al. and Struijk et al. and other studies in which acute PD had outcomes comparable to acute HD (see Table 179.1).

Tunneled PD catheters today are placed using one of six techniques: peritoneoscopy, blind (Seldinger technique), fluoroscopy (Seldinger technique with radiocontrast injection), surgery (dissection) laparoscopy, and the original Tenckhoff Trocar (rarely used in the United States).[50] All of these techniques except laparoscopy can be performed at the bedside in the ICU or in procedure rooms, with proper preparation and equipment. With proper care, the deep cuff of the Tenckhoff catheter can be placed within the rectus muscle with any of these techniques (which greatly diminishes the risk of pericatheter leaks). Peritoneoscopically placed catheters are placed with a single puncture technique that automatically places the deep cuff within the lateral or medial border of the rectus muscle.[51] Under vision a guide can be directed to lie against the parietal peritoneum in a direction that avoids adhesions and bowel loops, and the catheter then follows the course of the guide. With the peritoneoscopic placement of PD catheters it is relatively easy to place another tunneled catheter to perform CFPD, if desired. Peritoneoscopically placed catheters have a higher rate of successful hydraulic function in the first few weeks of use as well as over years of use than surgically placed catheters.[52] Fluoroscopically placed PD catheters also have fewer complications than surgically placed catheters and a similar longevity.[53,54] With a properly functioning chronic peritoneal access, the effectiveness of PD is increased and

risks are considerably diminished because one catheter is used for the duration of AKI therapy. Tenckhoff catheters allow peritoneal access for years, rather than the days of safe use for acute PD catheters. Thus patients who recover from their acute illness but still have renal failure may be able to continue PD as treatment for ESRD, using the same access device that supported them through the acute illness.

COMPARISON OF RISKS

Among therapies for AKI, PD has the unique risk of causing peritonitis. However, in patients in whom infection is suspected as a cause of AKI, performing PD can be helpful in ensuring that peritonitis is not present. In other patients, if peritonitis is detected, diagnostic tests can be implemented to determine the source, and IP antibiotic therapy begun to effectively treat the infection. The onset of peritonitis in PD therapy of AKI is much different from that in CAPD therapy. If peritonitis is detected during therapy of AKI with PD, it usually is noticed within 2 or 3 days of the start of therapy.[18,55,56] Therefore peritonitis detected early in PD actually may be due to contamination of the peritoneum that preceded the implementation of PD. The organisms causing peritonitis in patients with AKI may be different from those causing peritonitis in patients undergoing CAPD. Older studies showed a predominance of *Staphylococcus epidermidis* and *Candida* species in AKI patients treated with PD, organisms that are not usually seen in peritonitis in patients undergoing CAPD, and mixed infection was common.[57] More recent studies have demonstrated equal overall rates of peritonitis in patients on urgent-start PD and planned PD therapy, with roughly the same organism distribution.[58] If peritonitis occurs during acute PD therapy, it causes cloudy dialysate and sometimes local symptoms but does not usually result in septicemia. This is a much different outcome from that of catheter infection during HD or CVVH, which always results in septicemia.

The complications of PD and HD for AKI have been compared in centers providing both types of therapy. In a study reported by Swartz et al.,[10] the patients treated by HD had a high incidence of severe hypotension and severe hemorrhage, acidosis, and shunt clotting. Patients undergoing PD had a high incidence of hyperglycemia, poor catheter drainage, and asymptomatic peritonitis. The major causes of death in patients with AKI were also different for patients treated by HD and with PD. Death from sepsis unrelated to dialysis was higher for the HD group, but cardiac deaths were higher in the PD group, owing to the more frequent implementation of this therapy in patients with underlying heart disease.

When one compares the overall risks of each type of therapy for AKI, there are marked differences among CVVH, CVVHD, HD, and PD (Table 179.2). The blood treatment therapies have a significant risk of septicemia, low flow from the blood access, hypotension, membrane clotting, and bleeding. PD therapy involves risks of PD catheter outflow failure, hyperglycemia, and peritonitis as described below. Peritonitis is obviously the most serious complication of PD. However, if the initiation of PD reveals a preexisting peritonitis, antibiotic or surgical therapy may resolve the infection that caused the AKI in the first place. In patients treated with PD during AKI, recognition and therapy of preexisting peritonitis contribute to the improved outcome of these patients.

TABLE 179.2

Risks of Various Dialysis Therapies for Acute Renal Failure[a]

RISK	CONTINUOUS ARTERIOVENOUS HEMOFILTRATION	CONTINUOUS VENOVENOUS HD	HD	PD
Septicemia	+	+	+	–
Vascular occlusion	+	+	+	–
Hypotension	–	–	+	–
Membrane clotting	+	+	+	–
Bleeding resulting from anticoagulant	+	+	+	–
Peritoneal dialysis catheter outflow failure				+
Hyperglycemia				+
Asymptomatic peritonitis, often preexisting				+

[a]Plus sign indicates that risk applies to modality; minus sign indicates that risk does not apply to modality.
HD, Hemodialysis; *PD,* peritoneal dialysis.

ADVANTAGES AND DISADVANTAGES

One advantage of PD is that it is relatively simple to perform and has less labor costs than either HD or CVVHD. Nurses who perform acute PD by manual techniques will confirm that this therapy is simple; every 2 to 4 hours, a clamp is opened to drain the peritoneum, a new bag is attached to the inflow line, and the inflow clamp is opened. The costs of the therapy are only the cost of 6 to 12 bags of peritoneal dialysate each day, plus that of the labor of an ICU nurse to open a clamp to drain the peritoneum, then attach and infuse the volume of a new bag. Data collection is simple; the outflow volume is measured and recorded, and the fluid is inspected to determine whether it is clear or cloudy. Outflow problems with PD catheters do require extra time to address and resolve; however, much more nursing time is required for procedures and measurements related to CVVH, CVVHD, and HD treatments. In addition, the patient is unattached to any machine and free to transfer to other rooms or departments for procedures during manual acute PD. Automated PD cycler machines are also relatively easy to set up and use and have the added advantage that specially trained staff can be used to set them up. Of course, mobility is limited while the patient is attached to a cycler machine, but the cycler therapy can be discontinued and restarted whenever needed. The disadvantage with PD cyclers is that nurses must receive extra training in how to operate the machine and how to respond to a number of potential alarms.

Supply costs for PD are moderate but highly dependent upon the cost of bags of dialysate. The cost of the PD solution varies greatly from country to country. One study in India found that supplies for PD were much less costly than those for CVVHDF,[14] whereas the study from Vietnam showed that PD supplies were more expensive than supplies for HD.[12] Labor costs were not included in either evaluation, although total hospital costs were compared in the study in Vietnam. PD for AKI has a number of other patient and staff advantages, summarized by Golper.[59]

The greatest disadvantage of PD for acute therapy is that it is a therapy performed in the patient room by nurses,

primarily the patient's primary nurse. This means that an entire staff of a hospital ward must be trained in how to perform PD, either with manual techniques or automated cycler techniques. Although all nurses have some passing familiarity with how PD is performed, especially the older ones, they still need training in the necessary steps to ensure sterile connections and proper analysis of problems. From the primary nurse's perspective, it may be easier to have HD staff come to the room to perform the HD for a few hours daily and then leave than to perform PD themselves. However, CVVHD, which usually is performed by ICU nurses, is not an easy choice for nurses. The workload increase for CVVHD is much greater than for PD, and nurses performing CVVHD often are restricted to caring for only one patient while performing this therapy.

In the past few years there has been increasing recognition that PD can be used in an "urgent start" as a safe, efficient, and effective therapy in treatment of ESRD patients starting dialysis in a hospital or outpatient clinic.[60–63] "Urgent start" means that the patient needs dialysis to correct uremic symptoms but does not have life-threatening illness or laboratory values that would require "emergent" dialysis therapy within 48 hours. A tunneled PD catheter is placed promptly and dialysis started very shortly in an outpatient or inpatient setting. Urgent start therapy became much more practical and easier to implement when percutaneous, nonsurgical methods for placing tunneled PD catheters became more popular among radiologists and nephrologists (avoiding delays of surgery consultation and scheduling). Wherever urgent start programs have been set up to treat patients with PD promptly in outpatient settings, an impetus has evolved to treat patients with more severe symptoms within the hospital shortly after catheter placement.[64] This avoids need for acute HD and acute central venous catheters for dialysis. When urgent start PD programs are implemented, nursing staff within the hospital are usually trained to perform the PD procedure at least on certain units or wards.[65]

Several randomized studies have shown that with a properly placed PD catheter, exchanges can be started almost immediately in patients as long as they are inactive when the abdomen is full of fluid.[59,60] There may be an increase in risk of pericatheter leak or outflow failure when using surgical placement,[60] but this is resolved primarily by improved surgical technique[59] or percutaneous catheter placement (fluoroscopic, peritoneoscopic, or blind) with special attention to placing the deep cuff in the rectus muscle.[61] Many patients with ESRD have symptoms that are not so severe as to require HD procedures before PD is initiated.[62,63] For patients who have uremic symptoms but are not severely ill, clinical improvement can be expected within a day or two of starting PD. Overall mortality does not differ between patients receiving urgent start PD versus urgent start HD, over 6 months of follow up.[66] A cost analysis of unplanned dialysis for hospitalized ESRD patients indicated that the overall per-patient cost for urgent start PD programs (using outpatient dialysis centers) was about 15% less than those of urgent start HD.[67] Many patients with acute renal failure who do not have life-threatening symptoms also can be managed successfully with acute PD, and therefore, in a hospital with urgent start PD, no extra staff training is needed.

CONCLUSION

When a properly functioning chronic peritoneal access device is placed, PD is a safe, effective, and inexpensive modality for the treatment of AKI. This modality is underused greatly for treatment of AKI in the United States. In hospitals using urgent-start PD for initial treatment of patients with ESRD, resources and training are already available to implement an acute PD program. This program also will enhance the use of PD to treat patients with AKI, especially those patients without severe adverse symptoms. With improvements in efficiency and simplicity of PD, such as with CFPD, PD will become a much more widely used therapy for AKI.

Key Points

1. Clinical outcomes of acute renal failure, such as recovery of renal function and patient survival, are similar for peritoneal dialysis (PD) and hemodialysis (HD).
2. The chemical function of PD is equivalent to or better than that of intermittent acute HD, though less effective than that with continuous venovenous HD or continuous HD.
3. Fluid removal by PD is continuous, offering clinical advantage over intermittent therapies.
4. Continuous-flow PD, which offers the potential to improve the chemical effectiveness greatly of acute PD, can be performed with the use of currently available catheters and bagged solutions.
5. The unique adverse effects of PD include peritonitis and increased IP pressure; to minimize risk of these complications, certain patients with acute renal failure should not be treated with this modality.

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