CHAPTER

83 Peritoneal Dialysis

Seth Furgeson • Rajnish Mehrotra • John M. Burkart • Isaac Teitelbaum

Since the first description of continuous ambulatory peritoneal dialysis (CAPD) in 1976, peritoneal dialysis (PD) has become the dominant modality for home dialysis across the globe. Over the last decade, the patterns of utilization rates for PD have changed. Although the proportion of end-stage renal disease (ESRD) patients treated with PD remains low in many Western countries, as well as developing countries in the Middle East and South Asia, the utilization rates are increasing in several Eastern European, South Pacific, and East Asian countries.[1,2] In the United States, the percentage of dialysis patients on PD has decreased over the past decade with a slight increase in 2008; during that year, fewer than 7% of dialysis patients received PD.[3] Lack of adequate patient and physician education regarding PD likely contributes to this pattern of underutilization.[4] Given the absence of a randomized, controlled clinical trial comparing PD and conventional hemodialysis (HD), observational studies comparing incident PD and HD patients provide the best comparisons of the two modalities. Based on these studies, a few key observations can be made. First, patients commencing treatment with PD are younger and have a lower comorbidity burden than those that are treated with HD.[5] Second, there appears to be a modality by time interaction, such that patients commencing PD have a higher probability of survival during the first 2 to 3 years of renal replacement therapy when compared to HD patients; this advantage diminishes over time.[6–8] Third, the relative outcomes of patients treated by HD or PD seem to be modified by their age and diabetic status and the presence or absence of comorbidities. Thus, among individuals with no baseline comorbidity, treatment with PD appears to be associated with a survival advantage among nondiabetic patients (all age groups) and young diabetic patients (age <45 years).[9,10] It remains unclear if these differences in survival reflect a "modality effect" or are due to selection bias undescribed by known comorbidities. Nonetheless, PD treatment is used by thousands of patients around the world and appears poised to remain an important modality for renal replacement therapy. Furthermore, virtually all studies of contemporary cohorts of dialysis patients have demonstrated a similar overall survival from different parts of the world with different levels of PD utilization.[6,9,11,12]

Space precludes us from describing an extensive physiologic basis for PD. We discuss the current major issues of concern for PD in this chapter—the definition of "adequate" therapy and the control or management of therapy-related complications.

PERITONEAL DIALYSIS MODALITIES

PD may be performed manually and/or with the assistance of an automated device, commonly referred to as a "cycler." Similarly, PD therapy may be either continuous or intermittent. In most patients, selection of the PD modality hinges upon which therapy better suits the patient's lifestyle. However, in the absence of residual renal function, it is probably always desirable to use a continuous therapy.

Peritoneal Dialysis Techniques: Continuous Therapies

Continuous Ambulatory Peritoneal Dialysis

Until recently, CAPD was the most commonly used form of PD. Since its original description, there have been few changes in the basic therapy, although there have been many changes in the connection devices or "connectology" used to make the exchange. CAPD is a manual therapy and usually uses less dialysate than automated PD. The usual dialysis prescription for patients on this technique is four exchanges per day using 2.0 to 2.5 L of dialysate. However, in many developing countries, patients are treated using three exchanges with lower fill volumes with similar results. The equivalent results despite a lower dialysate use may, in part, be secondary to smaller body size in these countries.

Continuous-Cycling Peritoneal Dialysis

The utilization of automated peritoneal dialysis (APD), of which continuous-cycling peritoneal dialysis (CCPD) is the most common, is rapidly increasing in many parts of the world, like the United States.[3] Most often, patients

undergoing CCPD use an automated cycler to perform exchanges while they sleep with a subsequent "last fill" and single daytime dwell until the following evening; therefore, this is a continuous therapy. Some patients also require a daytime exchange, either to maximize solute clearances or to enhance fluid removal. Although it may be done manually, this exchange is more commonly performed using the cycler as a "docking" station for drain of the last fill instilled in the morning and subsequent instillation of fresh dialysis fluid. APD performed in this fashion is commonly referred to as CCPD with a midday exchange or as "high-dose" CCPD (a misnomer, as the volume of fluid used may well be less than that used by another patient performing "low" dose CCPD).

Peritoneal Dialysis Techniques: Intermittent Therapies

Because intermittent therapies typically use multiple short dwells, they tend to be automated, although they can be done manually. Intermittent PD (IPD) therapies are best suited for patients who are found to be high transporters based on the peritoneal equilibration test (PET). However, they should rarely be used once the patient loses residual renal function. These therapies also may be transiently indicated during peritonitis for some patients experiencing problems with ultrafiltration, or if PD therapy needs to be initiated within 2 weeks of implantation of the PD catheter (early "break-in").

Intermittent Peritoneal Dialysis

By definition, IPD implies that therapy periods alternate with periods when the peritoneum has been drained ("dry abdomen"). As classically performed the patient uses multiple short-dwell exchanges three or four times a week. Techniques include manual IPD, cycler IPD, reverse osmosis machine IPD, intermittent reciprocating dialysis with an extracorporeal reconstituting circuit, and others. In recognition of the importance of small and possibly middle-molecule clearances, IPD is now rarely used.

Nonetheless, classic IPD therapies continue to have their uses. Cycler IPD has been used in areas where technical, social, and economic limitations restrict the use of CAPD. Cycler IPD has been used immediately after abdominal surgery, for elderly patients, patients with refractory heart failure, or for those who are on CAPD and have developed hernias or leaks.[13]

Nightly Intermittent Peritoneal Dialysis

Nightly IPD (NIPD) utilizes a cycler overnight with a subsequent dry day. It is best employed by patients who still have residual renal function regardless of their transport type. Daytime ambulatory peritoneal dialysis (DAPD) is based on the same concept as NIPD, but DAPD is a manual technique, and the patient typically has a "dry time" during the night. The lower the peritoneal membrane transfer rates, the lower the 8-hour NIPD or DAPD clearances, and, in some patients, time spent on NIPD or DAPD has to be prolonged by 10% to 40% to achieve minimal target clearances.[16,17] Like IPD, NIPD can be used for management of patients with heart failure, as transient therapy for postoperative patients, or patients treated with CAPD or CCPD, who have developed hernias or leaks,[18] or for women with rectal or vaginal prolapse.

Tidal Peritoneal Dialysis

Tidal peritoneal dialysis (TPD) is best performed nightly by the use of an automated cycler. It involves the maintenance of an intraperitoneal reservoir of dialysate, which is achieved by incomplete drainage of the fluid at the end of each dwell. Additional amounts of fluid are instilled with each exchange to maintain an optimal intraperitoneal volume. By maintaining an intraperitoneal reservoir of dialysate, it is assumed that tidal dialysis may maintain more continuous contact of dialysate with the peritoneal membrane. Furthermore, the more rapid cycling of dialysis may increase mixing and prevent formation of stagnant intraperitoneal fluid layers. Although preliminary studies suggested that small solute clearances are augmented[14,15] subsequent studies have failed to confirm the ability of TPD to enhance clearances.[16–18] TPD is useful, however, for patients who have pain with either infusion or draining; the reservoir of dialysate minimizes pain during drainage and upon instillation of fresh dialysate.[19] In prescribing tidal peritoneal dialysis, variables to be chosen include reserve volume, tidal outflow volume, tidal replacement volume, flow rates, and frequency of the exchanges. Although TPD may have clinical benefits, the treatment cost will be increased due to the additional dialysate fluid.

DEFINING ADEQUACY OF DIALYSIS USING SMALL SOLUTE CLEARANCES

Minimal Versus Optimal Dialysis

The native kidneys perform excretory and endocrine functions and are pivotal in the maintenance of euvolemia. The loss of these functions in patients with progressive renal failure results in numerous metabolic and vascular abnormalities. In order to return the individual to complete health, some of the goals of optimal renal replacement therapy are summarized in Table 83.1. The concept of "optimal" renal replace-

83.1 Goals of End-Stage Renal Disease Replacement Therapy

Improve duration and quality of life
Reverse uremic signs and symptoms
Control acid-base abnormalities
Improve dyslipidemia and cardiovascular risk
Stabilize nutritional status
Remove small and middle sized uremic toxins
Improve abnormalities in mineral and bone metabolism
Minimize patient inconvenience factors
Control blood pressure and maintain euvolemia

83.2 Solute Removal by Dialysis and the Natural Kidney

Solute Clearance	Natural Kidney	HD Low Flux	HD High Flux	CAPD
Urea (L/wk)	750	130	130	70
Vitamin B_{12} (L/wk)	1,200	30	60	40
Inulin (L/wk)	1,200	10	40	20
B_2-microglobulin (mg/wk)	1,000	0	300	250

HD, hemodialysis; CAPD, continuous ambulatory peritoneal dialysis.
Modified from Keshaviah P. Adequacy of CAPD: a quantitative approach. Kidney Int Suppl. 1992;38:S160.

ment therapy, as applied to dialytic therapies, entails that the amount of dialysis delivered is not the rate-limiting step that determines patient outcome. In other words, an "optimal" dialysis prescription eliminates uremia as a potential variable, allows patients to achieve euvolemia, and maximizes quality of life. Given the continuing high risk for morbidity and mortality and poor rehabilitation among the ESRD population, it is clear that the current renal replacement therapies are far from achieving the goal of "optimal" therapy.[20] One of the reasons for this may be that, for a large number of solutes, the dialytic clearances typically replace <10% of the native renal excretory function (Table 83.2).[21] Based on these considerations and the current state of knowledge relating small solute clearances to outcome, it is more reasonable to define clearance goals of dialytic therapy in terms of "minimal acceptable," rather than "optimal," dialysis.

After the widespread introduction of hemodialysis, studies of patients undergoing HD attempted to define a "dose" of hemodialysis sufficient to prevent malnutrition, uremia, and premature death. Based on the initial results and subsequent reanalysis of the National Cooperative Dialysis Study, the concept of urea kinetic modeling was developed to monitor the dose of HD.[22,23] Shortly thereafter, the concept of monitoring the dose of dialysis using urea (and, subsequently, creatinine) kinetic modeling was extended to patients undergoing PD. Thus, over the last two decades, the adequacy of dialysis dose has been based on an assessment of achieved clearances of small solutes.

However, as is clear from Table 83.2, small-solute clearance is substantially lower for PD than HD. Yet, as discussed in the preceding section, the overall outcome is similar between HD and PD patients. It is also clear from Table 83.2 that solute clearances in CAPD exceed those of standard HD for all but the small-molecular-weight solutes. Is the reason that survival rates on CAPD and HD are similar because of comparable "middle-molecule" clearance? Should middle-molecule clearance be measured as the "PD yardstick?" At this time, there are no interventional studies to support such a change in the "PD yardstick." However, it is important to note that strategies that maximize small-solute clearances do not necessarily enhance the clearance of larger-molecular-weight toxins as clearance of the latter is time-dependent (Fig. 83.1).[24]

Small-Solute Clearances and Mortality

Dialysis dose among patients undergoing PD had historically been measured using both urea and creatinine clearances; however, as renal creatinine clearance in ESRD patients is largely a function of creatinine secretion, urea clearance alone is now more commonly used. Because the urea clearance is expressed as a sum of renal and peritoneal clearance, studies evaluating mortality risk and dialysis dose must clearly differentiate between renal and peritoneal Kt/V. Most large, observational studies examining mortality demonstrated that although renal urea clearance is strongly associated with a variety of patient outcomes, peritoneal clearances within the range achieved in clinical practice is substantially less so.[25–35] Furthermore, two randomized, controlled, clinical trials[36,37] have now provided the final confirmatory evidence that increases in peritoneal

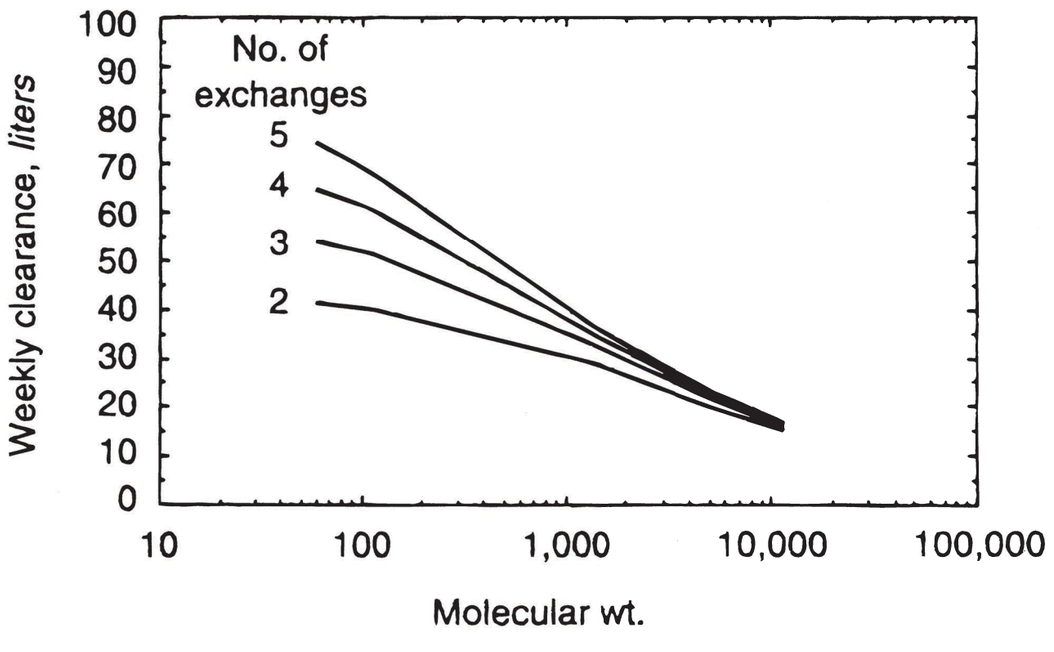

FIGURE 83.1 The influence of the number of exchanges on the weekly solute clearance for solutes with a range of molecular weights derived from a computerized model of peritoneal transport. (From Keshaviah P. Adequacy of CAPD: a quantitative approach. *Kidney Int Suppl.* 1992;38:S160, with permission.)

clearance, within the range achieved in clinical practice, do not result in significant improvement in patient morbidity or mortality (Table 83.3).[52,53] This accumulating body of data should not be taken to mean that peritoneal clearances are biologically irrelevant or that providing peritoneal clearances do not have a survival benefit—an anuric patient would die in the absence of peritoneal clearances. However, these data clearly suggest that within the range of clearances currently achieved in clinical practice, higher peritoneal clearances are unlikely to result in significant improvement in patient survival.

Small-Solute Clearances and Morbidity

ESRD patients suffer considerable morbidity, have impairments in the quality of life, and patients treated with PD continue to have a high rate of transfer off the therapy ("technique failure"). In observational studies, it appears that a low level of small solute clearance is associated with morbid outcome.[28,30,35] Two of the three randomized, controlled trials were unable to demonstrate any beneficial effect of increasing peritoneal clearances on the risk for hospitalization or the number of hospital days or technique survival (Table 83.3).[36,37] In the study by Mak et al., the intervention group had a higher hospitalization rate at the time of entry into the study when compared to the control group. Upon follow-up over 12 months, the hospitalization rate remained unchanged in the intervention group but increased in the control group, such that there were no significant differences in the hospitalization rates between the two groups over the study period.[38]

Furthermore, observational studies have been unable to demonstrate any relationship between small-solute clearances and the quality of life of PD patients.[39,40] These findings have now been confirmed by ADEMEX—a randomized, controlled, clinical trial.[41]

Thus, the existing body of evidence suggests that within the range of clearances currently achieved in clinical practice, higher peritoneal clearances are unlikely to result in significant improvements in hospitalization rate, technique failure, or quality of life of PD patients.

Small-Solute Clearances, Nutritional Status, and Patient Outcome

Due to the high prevalence of protein-energy wasting (PEW) in PD patients and the deleterious long-term consequences of wasting, the impact of small solute clearance on the nutritional status of PD patients has been an actively studied area. As with HD patients, in PD patients there are multiple, imperfect clinical measures of PEW as well as difficult-to-obtain research techniques. Importantly, there is poor correlation amongst the different measurements.[42–44] Low serum albumin and prealbumin levels, poor subjective global assessment (SGA), low fat-free edema-free mass, low dietary protein intake, and diminished hand grip strength are associated with higher morbidity and mortality.[42,43,45–49] Notwithstanding the evidence that the etiology of nutritional decline among ESRD patients is multifactorial (including an important role of inflammation), inadequate dietary intakes are probably important and independent contributors to the high prevalence of PEW among the dialysis population.[50] It follows, then, that if enhancing the dose of dialysis can result in an increase in dietary intakes, the higher dose would have the potential of improving their nutritional status; this, in turn, would be expected to have a salutary effect on patient outcome.

Based on multiple, small studies, there is evidence that increasing dialysis dose can improve nutritional status. Studies that show a relationship between Kt/V_{urea} and nPNA (normalized protein equivalent of nitrogen appearance) are problematic since both equations share common variables.[51,52] However, enhanced solute removal has been associated with improvement in other nutritional parameters: protein intake (as measured by dietary records,[53,54] mid-arm circumference and weight gain,[55] SGA,[53] and albumin[56]). Given data that a factor in uremic serum can induce anorexia in rats, it is plausible that dialytic removal of such a factor would increase appetite.[57] However, notwithstanding the increase in dietary protein intake, recent randomized controlled trials have been unable to demonstrate an improvement in nutritional status with increasing peritoneal clearances (Table 83.3).[37,38]

Minimal Total Solute Clearance Goals

Several organizations around the world have developed clinical practice guidelines to define the target level of small-solute clearances required to optimize the health of patients undergoing PD. As would be expected, these guidelines have evolved with our understanding (as discussed previously), particularly with the availability of the results of two large randomized controlled clinical trials.[37,38] The updated guidelines by organizations in United States, Canada, and Europe are summarized in Table 83.4.[58–62] When compared to guidelines published earlier, the current recommendations differ in several important respects. First, most of the guidelines recommend only one measure of adequacy to define the minimum dose of dialysis (Kt/V_{urea}). Early studies, including the CANUSA study, suggested that patient outcome was more dependent upon total (renal + peritoneal) creatinine clearances rather than total urea clearances.[63] However, the contribution of renal creatinine clearance to total (renal + peritoneal) creatinine clearances is substantially greater than of native renal urea clearances. Because creatinine is secreted and urea is reabsorbed by renal tubules, renal creatinine clearance is always higher than renal urea clearance; on the other hand, peritoneal clearances are dependent on the molecular weight of the solute in question. Thus, creatinine clearance (molecular weight, 113) is always lower than peritoneal urea clearance (molecular weight, 60). Consequently, the expected weekly creatinine clearance is different in a patient who is just starting PD with a residual renal Kt/V_{urea} of 2.0 per week than in an anuric patient with a peritoneal Kt/V_{urea} of 2.0 per week. Thus, although both markers of solute clearance may be predictors of outcome, the target or goal for creatinine clearance may have to change over time as residual renal function decreases and is replaced by peritoneal clearance.

83.4 Targets for Small Solute Clearances Recommended by Various Organizations for Patients Undergoing Chronic Peritoneal Dialysis

Committee	Nature of Clearances	Kt/V	Creatinine Clearance
United States—NKF-K/DOQI[a]	Renal + peritoneal	1.7	—
European Best Practice Guidelines	Peritoneal	1.7	—
Canadian Society of Nephrology	Renal + peritoneal	1.7[a]	—
International Society of Peritoneal Dialysis	Renal + peritoneal	1.7	—
CARI (Australia)	Renal	>1.6	> 60 L/week (high and high-average transporters) > 50 L/week (low and low-average transporters)

[a]For patients with residual renal function >4 mL/min, a peritoneal Kt/V between 1.0 and 1.7 is recommended.
NKF-K/DOQI, National Kidney Foundation's Kidney Disease Outcome Quality Initiative.

On the other hand, it appears from outcome studies that the Kt/V_{urea} target may not need to change. Furthermore, it is now recognized that the stronger relationship of creatinine clearances to patient outcome was a result of the effect of the confounding effect of residual renal function. There is no evidence that peritoneal creatinine clearances are superior in predicting outcome, when compared to peritoneal urea clearance. In light of these considerations, the various expert groups recommend the use of Kt/V_{urea} alone to determine the dose of dialysis (Table 83.4). Second, the targets for Kt/V_{urea} have been changed, such that Kt/V_{urea} of 1.7 at all times is now considered to be the minimum dose necessary needed for patient well-being. Based on the results of the two recent randomized controlled trials, it is also recognized that some patients may require a higher dose of dialysis to manage uremic symptoms or to achieve euvolemia.[36,37] Third, except in the CARI guidelines, there are no differences in the definition of minimum dose of dialysis based upon the patients' transport type (see below). Fourth, some expert groups (Europe and Australia) have defined the adequacy of dialysis based only on peritoneal clearances, whereas others (Canada and the United States) define it based upon total clearances. Fifth, the targets are the same, irrespective of PD modality (CAPD or APD). Finally, volume control is recognized as an additional dimension to define adequate dialysis (see below).

MONITORING AND ADJUSTING SMALL-SOLUTE CLEARANCES

Determination of Peritoneal Transport

In its function as a dialysis membrane, the peritoneum performs two important processes: diffusion (movement of solute down a concentration gradient) and convection (movement of solute along with water, ultrafiltration [UF]). There is interpatient variation in peritoneal membrane transport characteristics. A variety of methods have been suggested, standardized, and studied to assess the peritoneal membrane function (Table 83.5).[64–68] The most precise method to evaluate diffusive function of the peritoneum is to determine the mass transfer area coefficients (MTAC) of solutes like creatinine.[69] These define transport independent of ultrafiltration (convection-related solute removal) and, consequently, are not influenced by dwell volume or glucose concentration. In order to determine the MTAC, additional laboratory measurements and computer models are necessary, but, once these are obtained, MTAC can be used easily in the clinical setting.[67,69,70]

However, of these various assessments of membrane transport characteristics, the peritoneal equilibration test (PET) is the most widely used.[65] All patients commencing PD therapy should undergo a PET. The first PET should be performed after at least 4 weeks of commencing peritoneal dialysis therapy.[71] Although some centers choose to repeat a PET only if clinically indicated, others perform the test periodically to monitor peritoneal membrane function.

In order to enhance the reproducibility of the test, several steps of the PET are standardized: (1) long (8 to 12 hours) preceding exchange; (2) drain the preceding exchange as completely as possible over 20 minutes; (3) infuse 2 L of 2.5% dextrose dialysate over 10 minutes (time 0); (4) take samples of dialysate of times 0, 120, and 240 minutes; (5) in order to take samples, 200 mL of dialysate is drained into a bag, 10 mL is drawn for testing, and 190 mL is reinfused; (6) a blood sample is taken at 120 minutes; and (7) the dialysate is drained completely at 240 minutes and the drain volume is measured. Dialysate and serum urea, glucose, and creatinine

83.5 Tests to Evaluate Peritoneal Membrane Function

Test	Parameter Used to Evaluate Solute Removal Function	Parameter Used to Evaluate Fluid Removal Function
Peritoneal equilibration test (4-hour)	D/P creatinine, D/Do glucose	Drain volume
Dialysis adequacy and transport test (24-hour)	D/P creatinine	Drain volume
Standard peritoneal analysis	MTAC creatinine	Drain volume, D/P sodium, and others
Personal dialysis capacity	Area parameter	Ultrafiltration coefficient
Apex	Purification phosphate time	Apex time

are measured. For each dwell time (0, 120, and 240 minutes), dialysate to plasma ratios (D/P) of creatinine and urea are determined, as is the ratio of glucose at the drain time (120 and 240 minutes) to the initial dialysate glucose concentration (D/D_0). These results are plotted against time and compared to known standard curves (Fig. 83.2). Based on the values of D/P creatinine or D/D_0 glucose, patients are classified into one of four categories: low, low average, high average, and high transporters. It should be noted that there is a significant discordance between the categorization of patients' transport type, based upon whether D/P creatinine or D/D_0 glucose is used (Fig. 83.3).[72] Studies suggest that abbreviating the preceding exchange to 2 to 3 hours does not significantly influence the values of D/P creatinine or D/D_0 glucose; thus, patients being treated with APD do not have to change their treatment schedule on the day prior to the PET.[73,74]

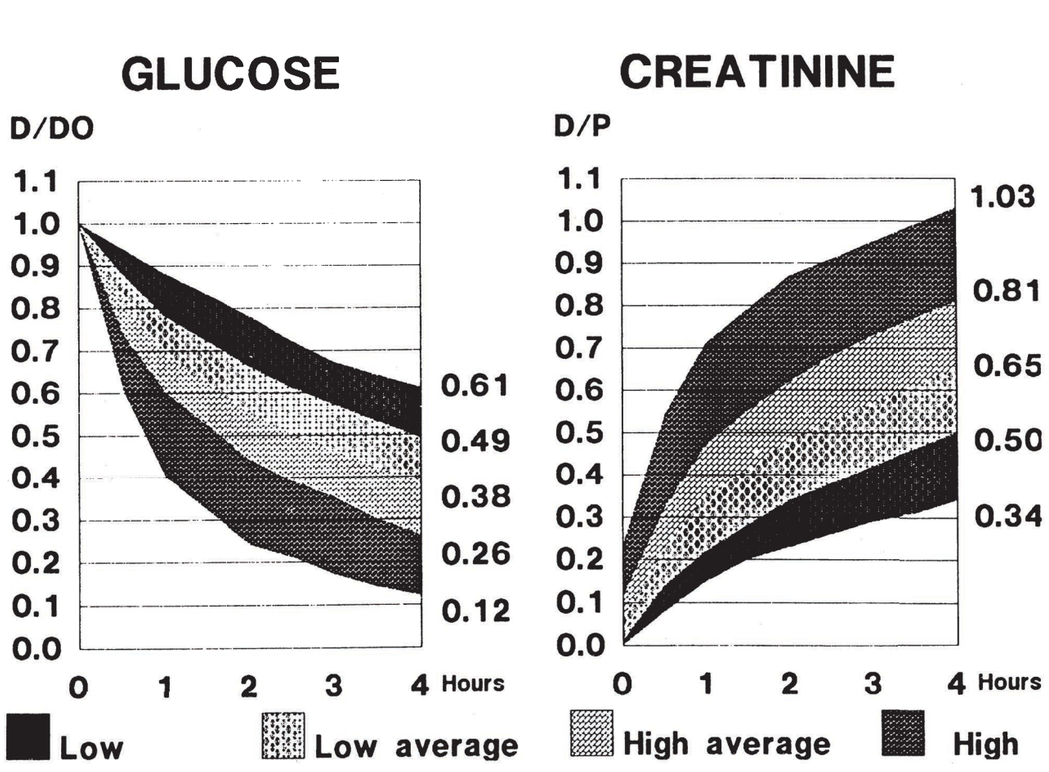

FIGURE 83.2 Dialysate to plasma ratios (D/P) for creatinine and drain time to initial dialysis concentration (D/D_0) ratios for glucose, generated from standard peritoneal equilibration testing. (From Twardowski ZJ. Clinical value of standardized equilibrium tests in CAPD patients. *Blood Purif.* 1989;7:95, with permission.)

As more has become known about ultrafiltration and water transport across the peritoneal membrane, it has been recommended that a 4.25% dextrose PET be used to characterize the ultrafiltration capacity of the peritoneum, including aquaporin-mediated water transport and solute transport.[75] The 4.25% PET has been compared to the 2.5% PET in a cohort of chronic PD patients and no clinically relevant difference in classifying the patients into different transport types was noted, suggesting that the 4.25% PET may be as clinically useful in prescription management as is the 2.5% PET.[76] The 4.25% PET has the added advantage of directly assessing the adequacy of ultrafiltration as well; ultrafiltration is defined as failure of a 4-hour dwell with 4.25% dextrose to yield at least 400 mL of net ultrafiltration.[75]

Clinical Relevance of Characterizing Peritoneal Membrane Function

The PET is used specifically to characterize the patient's peritoneal membrane transport properties. Knowledge of the peritoneal transport allows a physician to choose an appropriate prescription for a patient; this is particularly useful when using computerized, kinetic modeling for prescription management.

In general, rapid transporters of creatinine and urea also tend to be rapid absorbers of dialysate glucose (high D/P creatinine and low D/D_0 glucose). Therefore, although the D/P creatinine ratios for a 4-hour dwell tend to be close to 1, drain volumes tend to be small. Rapid transporters maximize their D/P ratios and intraperitoneal volumes early during the dwell. Once the osmotic gradient dissipates, UF ceases, followed thereafter by net fluid reabsorption. With standard CAPD, these patients may have drain volumes that are actually less than instilled volumes. Short dwell times often are needed to optimize clearance.[77]

On the other hand, in slow transporters, peak UF occurs late during the dwell, and net UF can be obtained even after prolonged dwells because glucose absorption is slow (low

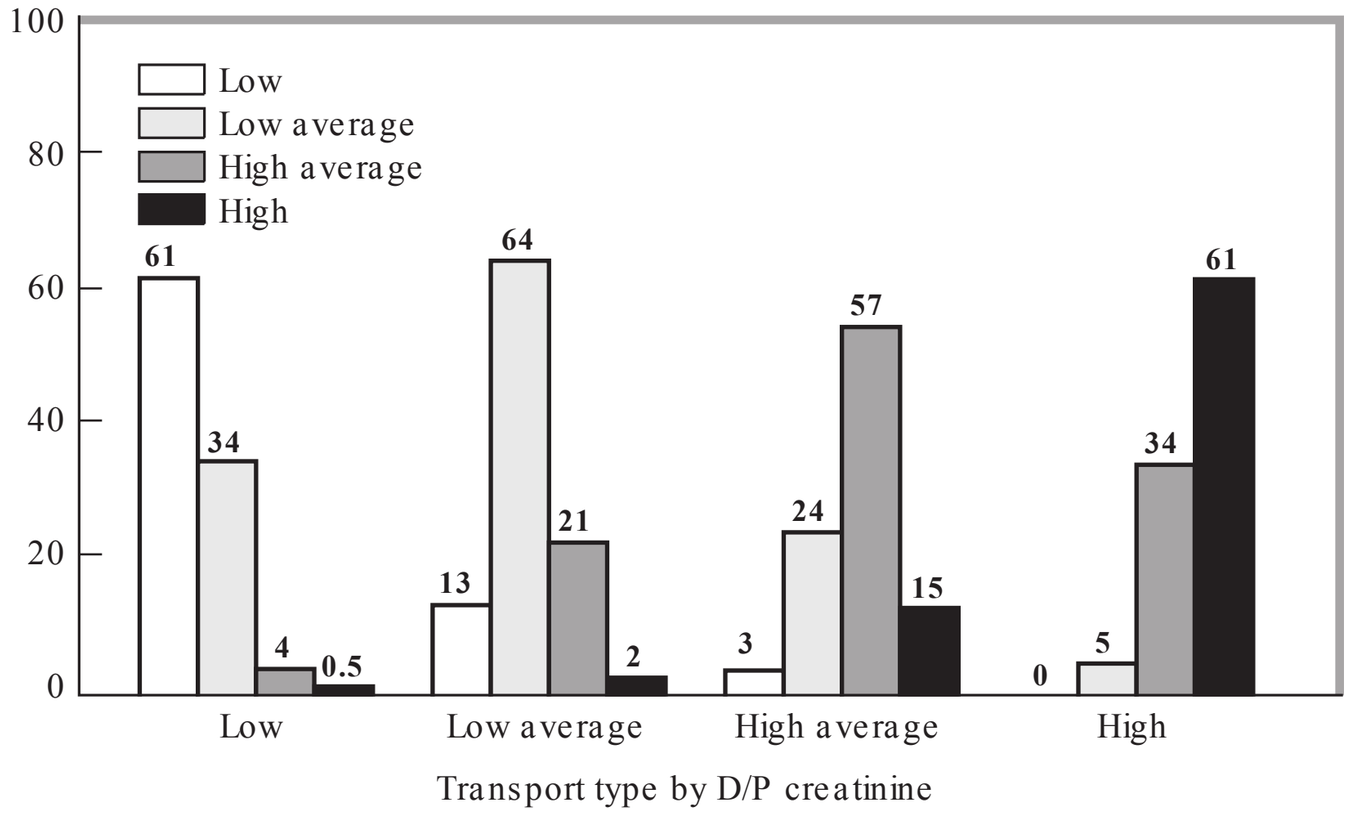

FIGURE 83.3 Discordance between categorization of patients' transport type by D/P creatinine or D/D_0 glucose. Thus, of the patients categorized low transporter by D/P creatinine, 61% of them will be classified as a low transporter by D/D_0 glucose; of the patients classified as low average transporter, 64% will be classified as low average transporter by D/D_0 glucose; of the patients classified as high average transporter, 57% will be classified as high average transporter by D/D_0 glucose; and of the patients classified as high transporter by D/P creatinine, 61% will be classified as high transporter by D/D_0 glucose. (Modified from Mujais S, Vonesh E. Profiling of peritoneal ultrafiltration. *Kidney Int Suppl.* 2002;81:S17, with permission.)

D/P creatinine and high D/D_0 glucose). The D/P ratios for creatinine and urea increase almost linearly during the dwell. For these patients, dwell time is the crucial determinant of overall clearance. They do best with continuous therapies, such as standard CAPD or CCPD. Notwithstanding these considerations, the vast majority of patients have an "average" transport type and they can be successfully treated with either PD modality. Two recent, large studies have demonstrated that there is not a difference in mortality among patients treated with CAPD or APD.[78,79] Furthermore, either PD modality (CAPD or CCPD) can be successfully adapted to even patients at the extreme of transport type (rapid or slow).

The original studies of the PET demonstrated associations between clinical variables and transport status.[65] Diabetes has been commonly linked to high transport status.[80] More recent, larger studies have not demonstrated a firm association with many clinical variables (e.g., diabetes, inflammation, and volume status) and transport status.[81–83] Accordingly, peritoneal membrane function can only be determined by an actual, standardized measurement rather than predicting transport rate from clinical variables. Furthermore, the PET cannot be used as a substitute to measure the dose of dialysis. Although it is possible to estimate daily clearances from PET studies, these estimates can significantly over- or underestimate actual daily clearances.[84]

The PET also provides useful prognostic information for patients treated with CAPD. Brimble et al. performed a meta-analysis of studies examining the consequences of high transport status.[85] Twenty studies representing distinct populations throughout the world were included in the analysis. Increases in D/P Cr were associated with higher mortality risk and treatment failure. Due to rapid dissipation of an osmotic gradient for ultrafiltration, high transporters on CAPD would be expected to more commonly have volume overload. Indeed, within the meta-analysis, the association between high transport and mortality was much diminished in CCPD patients and other data demonstrate that once patients with high transport status transfer to HD, mortality rates equalize.[86] Thus, the present state of knowledge would suggest a careful evaluation and aggressive management of nutritional and volume status and comorbidities among individuals with a higher transport type.

Measurements to Monitor Dialysis Dose

It is recommended that monitoring should include both dialysis dose and nutritional parameters because outcomes correlate with both. In light of emerging data favoring urea clearances over creatinine clearances, however, the consensus of the various expert groups seems to be to use only urea kinetics to monitor the dose of PD (Fig. 83.4). The only major difference appears to be with regard to defining the clearance targets based upon peritoneal or total (renal + peritoneal) Kt/V_{urea}. This is an important consideration since, for a 70-kg man, each 1 mL per minute of renal urea clearance adds approximately 0.25 to the total weekly Kt/V_{urea}.

Collections of dialysate and urine over 24 hours are relatively easy to obtain and can provide most of the clinically relevant data one needs to individualize a patient's prescription and monitor progress. These collections also can be used to calculate PNA, fat-free, edema-free mass (FFEFM), and other variables. The data obtained from 24-hour collections is complementary to that obtained from PET and are routinely used together for developing a patient's dialysis prescription and problem solving.

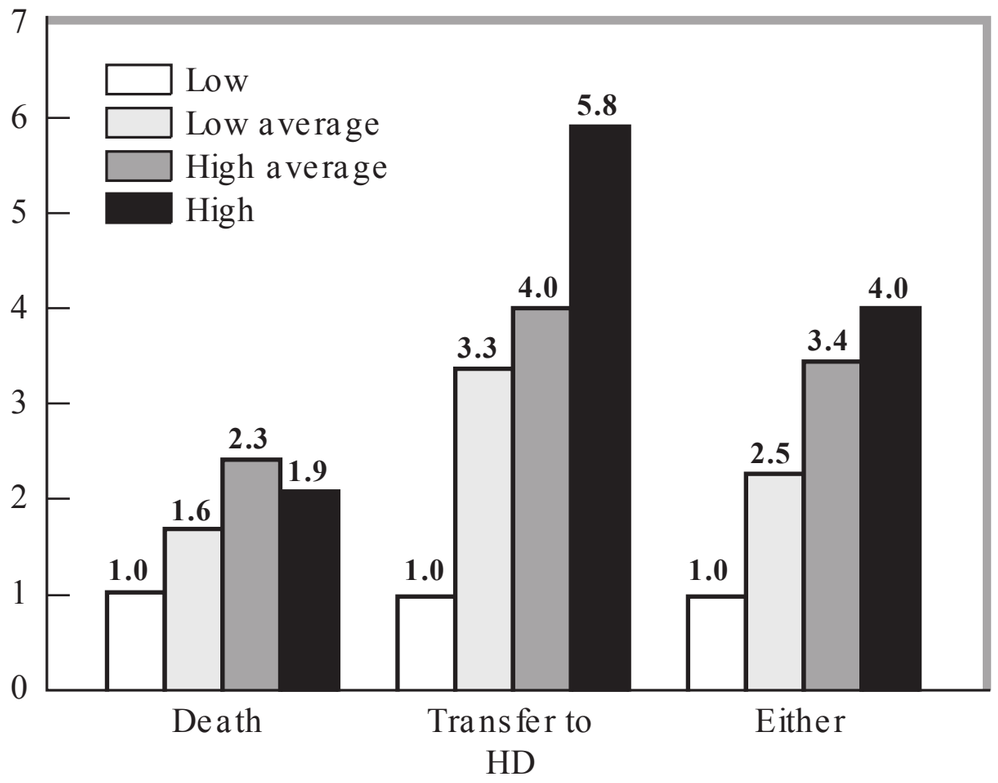

FIGURE 83.4 Relationship between transport type and patient outcome. With increasing permeability of the peritoneum, as defined by the peritoneal equilibration test, there is an increasing risk for death and/or technique failure. (Modified from Churchill DN, Thorpe KE, Nolph KD, et al. Increased peritoneal transport is associated with poor patient and technique survival on continuous ambulatory peritoneal dialysis. *J Am Soc Nephrol.* 1998;9:1285.)

Calculation of Dialysis Dose

To individualize dialysis dose and make comparisons of dose between patients, the solute clearances are typically normalized. If urea kinetics (Kt/V) are used, the sum of the daily dialysate and residual renal urea clearances are then divided by the volume of distribution for urea (V).[29] The urea V can be estimated to be 60% (males) or 55% (females) of the patient's weight in kilograms. More accurate estimations of V can be obtained using standardized nomograms, such as Watson[87] or Hume and Weyers.[88]

Calculation of the urea volume of distribution (V) is complicated by numerous pitfalls.[89] Weight has a different effect on normalization for men or women and, therefore, will affect Kt/V measurements. These differences are most marked when a patient's weight differs significantly from the norm in patients with the same height and frame size. The actual V is different in a patient with the same body weight if the increase in body weight from desirable is owing to overhydration or increase in adipose tissue. The same is true if loss of weight is due to protein energy wasting (PEW) versus amputation.

Calculation of Dietary Protein Intake

Dietary protein intake can be directly measured in metabolic wards, by dietary histories, or food recall records. An advantage of using food records is that they also evaluate total energy intake. Unfortunately, food records are time consuming and difficult to obtain because they require trained dietitians. Therefore, most reports relating dialysis dose to protein intake use estimations, based on urinary and dialysate nitrogen appearances and expressed as the protein equivalent of nitrogen appearance (PNA).[90] The most commonly used formulas to estimate PNA are summarized in Table 83.6. [91–94]

83.6 Commonly Used Formulas for Protein Nitrogen Appearance

$PNA = 10.76\ (G_{un} + 1.46)$[91]
$PNA = 9.35\ G_{un} + 0.294\ V +$ protein losses[90]
$PNA = 6.25\ (UN_{loss} + 1.81 + 0.031 \times$ body weight$)$[94]
$PNA = 6.25 \times$ N loss[92]
PNA (g/24 h) = 15.1 + (6.95 × urea nitrogen appearance in g/24 h) + dialysate and urine protein in g/24 h (Bergstrom)[a]
In the absence of direct measurement of urinary and dialysate protein losses, this following less accurate formula may be used: PNA (g/24 hours) = 20.1 + (7.50 × urea nitrogen appearance in g/24 h (Bergstrom)

[a]Bergstrom J, Heimburger O, Lindholm B. Calculation of the protein equivalent of total nitrogen appearance from urea appearance, which formulas should be used? Perit Dial Int 1998;18:467. (Modified from Keshaviah P, Nolph K. Protein catabolic rate calculations in CAPD patients. Trans Am Soc Artif Intern Org. 1991;37:M400.)
PNA, protein nitrogen appearance; G_{un}, urea nitrogen generation rate; V, volume of urea distribution; UN_{loss}, urea nitrogen loss; N, nitrogen.

The total PNA is then divided by the patient's body weight to determine the "normalized" PNA (nPNA), expressed in grams per kilogram of body weight per day. This term does not take into account differences in frame size and fat-free, edema-free mass (FFEFM). If a patient is markedly obese, the aforementioned calculations give a falsely low nPNA for the patient's actual FFEFM. Conversely, if a patient has PEW and has a less than expected FFEFM, these equations yield a falsely elevated nPNA. Various attempts to avoid this problem have been investigated, but corrections have not been standardized. One modification uses actual measurements of V or data from nomograms that more accurately estimate V. This V is then "normalized" by dividing it by 0.58 kg per L to determine normalized body weight. The PNA is then divided by normalized body weight to get nPNA. An extension of these principles is utilized to determine FFEFM from creatinine kinetics.[95] Finally, there is early evidence that bioimpedance measurements can assist with identifying both the "dry weight" and relative contribution of muscle mass, adipose mass, and fluid.[96]

Adjusting Dialysis Dose and Recognizing Pitfalls in Prescribing Peritoneal Dialysis

The initial PD prescription should be based upon a knowledge of the patient's transport type (determined using a PET), body size, and presence or absence of residual renal function. This can be done by using published algorithms (e.g., K/DOQI guidelines, data from EAPOS) or using computerized kinetic modeling.[97,98] The clearances achieved with the initial prescription should be confirmed with 24-hour collections of urine and dialysate. If the patient is not at goal, the prescription should be adjusted. This adjustment can also be done either empirically or using computerized kinetic modeling programs. There are two general changes that can be made to maximize clearances in an individual patient—either increase the drain volume or increase the D/P ratio in the dialysate effluent. Increasing the instilled volume increases the total drain volume and thus, the convective clearance. By altering dwell time, one can change both the D/P ratio at the end of prescribed dwell and the drain volume. The strategies to maximize the drain volume in patients undergoing PD are summarized in Table 83.7.[4,73,99–104] If a patient does not have a continuously wet abdomen, providing 24-hour dialysis should be the first step to enhance clearances. In a patient with a continuously wet abdomen, increasing the dwell volume should be the first step to enhance clearances. Most patients are able to tolerate the increased fill volumes without any discomfort and, if blinded to the fill volume, many are unable to correctly identify the amount of fluid instilled.[105,106] In order to improve tolerance, the fill volumes may be increased when the patient is lying supine (i.e., for the nighttime exchanges). Furthermore, cycler therapy allows increases in fill volumes in increments of 100 mL and improves tolerance of increasing the volume of instilled dialysate.

Some issues to consider in patients on standard CAPD are: (1) inappropriate dwell times (a rapid transporter would do better with short dwells); (2) failure to increase dialysis dose to compensate for loss of residual renal function; (3) inappropriate instilled volume (patient may only infuse 2 L of a 2.5-L bag); (4) multiple rapid exchanges and one very long dwell (patient may do three exchanges between 9 AM and 5 PM, and a long dwell from 5 PM to 9 AM,

83.7 Strategies to Enhance the Peritoneal Small Solute Clearances

Continuous Ambulatory Peritoneal Dialysis	Automated Peritoneal Dialysis
Daytime exchanges	Daytime exchanges
Increase dwell volume[100]	Add daytime dwell (if dry day)
Increase ultrafiltration (tonicity of dialysate)	Add midday exchange[101] Increase dwell volume Increase ultrafiltration (tonicity of dialysate or alternative osmotic agents like icodextrin)
Nighttime exchanges	Nighttime exchanges
Increase dwell volume[99,100]	Volume of each dwell[100]
Increase number of exchanges (nighttime exchange device)	Number of nighttime exchanges[104]
Increase ultrafiltration (tonicity of dialysate or alternative osmotic agent like icodextrin)	Number of hours of cycling (8–10 hours)[101] Increase ultrafiltration (tonicity of dialysate)

limiting overall clearances); and (5) inappropriate selection of dialysate glucose for long dwells that may not maximize UF and clearance.

Other problems are specific for those patients on cycler therapy: (1) the drain time may be inappropriately long (more than 20 minutes), (2) inappropriately short dwell times may not maximize clearances, (3) failure to augment total dialysis dose with a daytime dwell ("wet" day versus "dry" day), and (4) inappropriate selection of dialysate glucose may not allow maximization of UF, resulting in less total clearance. One may be able to achieve weekly urea clearance targets, but not creatinine or middle-molecule clearance targets, with short dwell times, as in NIPD. A shortened time with fluid in the peritoneum is accompanied by decreased middle-molecule clearances and this may have an adverse effect on outcomes. It appears reasonable to state that when patients become anuric, they must maximize their "time" (most of day) on dialysis to maintain middle-molecular-weight clearances.

83.8 Reported Benefits of Residual Renal Function in Patients Undergoing Peritoneal Dialysis

Greater probability of survival[26–29]
Lower morbidity Hospitalizations[108] Peritonitis rates[108] Technique survival
Cardiovascular Lower total body and extracellular water[107] Better control of blood pressure[109] Lower left ventricular mass index[1]
Nutritional status Higher dietary nutrient intakes[53,112] Higher serum albumin[1,27,113] Better nutritional status[2,111,113]
Anemia management Higher hemoglobin[1,113]
Divalent ion metabolism Better control of serum phosphorus[111,113]
Lower levels of circulating putative uremic toxins Low molecular weight like α1-microglobulin, alkaline RNAse[3] "Middle molecules"[115] Advanced glycosylation end-products like carboxymethyllysine[114]
Better quality of life[40,41,116]

RESIDUAL RENAL FUNCTION AND PERITONEAL DIALYSIS

Over the last decade, the centrality of residual renal function (RRF) in maintaining the health and welfare of patients undergoing PD has been established. Multiple studies mentioned previously demonstrated the superiority of renal urea clearance over peritoneal urea clearance. Over the same time period, data has accumulated that patients with significant RRF have a lower morbidity, have a lower severity of numerous complications associated with uremia, and have a better health-related quality of life (Table 83.8).[32,40,41,53,71,107–119] Thus, an understanding of the determinants of and strategies that retard the rate of loss of RRF are critical to the success of PD.

Notwithstanding the beneficial effect of treatment with PD on rate of decline of RRF, residual renal function inexorably declines over time (Table 83.9). Although many comorbidities have been linked to loss of RRF, to date, only one intervention has shown a beneficial effect on RRF. Randomized, controlled trials have shown that treatment with angiotensin-converting enzyme inhibitors (ACEIs, ramipril) or angiotensin-receptor blockers (ARBs, valsartan) significantly slows the rate of decline of RRF.[120,121] Finally, it appears that use of ACEIs and ARBs in patients undergoing PD is safe and does not result in significant elevations in serum potassium.[122] This may be related to the use of PD fluids without any potassium, as currently practiced. An initial, short-term, randomized, cross-over study demonstrated that patients had a higher urine volume when treated with PD solutions with low concentration of glucose degradation products.[123] However, three subsequent clinical trials, with follow-up for up to 12 months, have been unable to substantiate the benefits of these PD solutions on preserving residual renal function.[124–126]

ULTRAFILTRATION

A certain minimal amount of daily UF is necessary to maintain water balance in patients with ESRD. This is achieved by an osmotic pressure gradient between blood and the dialysate using glucose (predominantly) as the effective osmotic agent. During UF, retained solutes are swept along with the bulk solvent flow even in the absence of a concentration difference for diffusion. This contribution to net solute clearance has been termed solvent drag or convection; therefore, overall solute clearance is the sum of that owing to diffusion and convection.

Clinical Physiology

Simultaneous with UF of fluid from the bloodstream into the peritoneal cavity, there also occurs absorption of fluid from the peritoneal cavity, largely across tissue beds and partially via lymphatics.[127] Intraperitoneal volume at any time is determined by the relative magnitudes of transcapillary UF and tissue reabsorption and lymphatic reabsorption. Net

83.9 Summary Results of Some of the Studies That Have Compared the Rate of Decline of Residual Renal Function among Hemodialysis and Peritoneal Dialysis Patients

Author	Patient no. HD/PD	Study Design	Baseline Measure	Index of Renal Function	%Decline/Month (HD/PD)	PD Decline Rate, % of HD Rate
Rottembourg[4]	25/25	Prospective	Predialysis	C_{cr}	6.0/1.2	80
Cancarini[5]	75/86	Retrospective	Pre- and postdialysis	C_{cr}	5.8/2.9	50
Lysaght[6]	57/48	Retrospective	Pre- and postdialysis	C_{cr}	7.0/2.2	69
Misra[2]	40/103	Retrospective	Postdialysis	Mean	7.0/2.2	69
Lang[7]	30/15	Prospective	Dialysis start	C_{cr}	9.4/5.0	47
Jansen[8]	279/243	Prospective	Predialysis	Mean	10.7/8.1	24
McKane[9] [d]	300/175	Retrospective	Pre- or postdialysis	Urea Cl	Rate of decline similar in HD and PD	

[a]C_{cr}, timed creatinine clearance
[b]Mean, mean of timed urea and creatinine clearances.
[c]Urea Cl, timed urea clearance.
[d]All HD patients dialyzed with high flux dialyzers and ultrapure water.

UF at the end of any dwell is defined as the difference between drained volume and instilled volume. This definition assumes that the residual intraperitoneal volume is constant, which is often not the case. This variation is insignificant for day-to-day clinical practice.[128,129]

Ultrafiltration rates are highest at the beginning of the dwell. As glucose is absorbed and its concentration is diluted by influx of ultrafiltrate, UF decreases as osmotic equilibrium is approached. Depending on the concentration of instilled glucose, osmotic equilibrium is reached at different times in the dwell cycle. For 2-L solutions containing 1.5% dextrose, osmotic equilibrium and maximal drain volume are reached after about 2 hours of dwell time in patients with average peritoneal membrane transport characteristics. For 4.25% dextrose solutions, peak intraperitoneal volumes are not likely to occur until after 3 or 4 hours.[77] As osmotic equilibrium is approached, intraperitoneal volume and ultimate drain volume decrease owing to isosmotic absorption of fluids. In CAPD patients, this absorption rate ranges from 40 to 60 mL per hour and is attributable both to bulk absorption of fluid across the peritoneal membrane and lymphatic drainage of the peritoneum.[130]

Ultrafiltration Failure

UF failure represents a failure to maintain volume homeostasis. This definition implies that clinically, UF failure can result from either loss of residual renal function, inadequate fluid removal by PD, patient nonadherence to the therapy, or excessive salt and water intake. Furthermore, failure to remove adequate amounts of salt and water by the current PD prescription does not necessarily imply that there must be a pathologic alteration of the peritoneal membrane itself. Other possible causes include catheter malfunction, inadequate selection of tonicity of dextrose or icodextrin, inappropriately long dwell times, fluid sequestration, including, as recently described, retroperitoneal leakage,[131] and failure to match dwell time to peritoneal membrane transport status.[75]

Among CAPD patients, ultrafiltration failure may be defined as clinical evidence of fluid overload despite restriction of fluid intake and the use of three or more hypertonic (4.25% dextrose) exchanges per day.[132] However, other definitions have been used in various publications; therefore, the exact incidence of UF failure is unknown. At one center, UF failure was observed in 6.2% of 227 CAPD patients over 10 years and the risk increased with time on PD.[133] The prevalence was 2.6% after 1 year on PD and 30.9% after 6 years. If one considers a more rigid definition, such as one defined by the ability to generate an UF volume of at least 400 mL after 4 hours of dwell with 4.25% dextrose, the true incidence is rather low. As discussed previously, clinical symptoms consistent with UF failure are not always caused by an actual loss of peritoneal UF capacity. The first steps in the evaluation of a patient with suspected UF failure are to rule out dietary indiscretions, determine urine volume, and establish whether net effluent drain volume and/or peritoneal transport have changed. Apparent loss of UF is potentially reversible if caused by catheter malposition, dialysate leak, or recent peritonitis, but usually is permanent if kinetic studies sug-

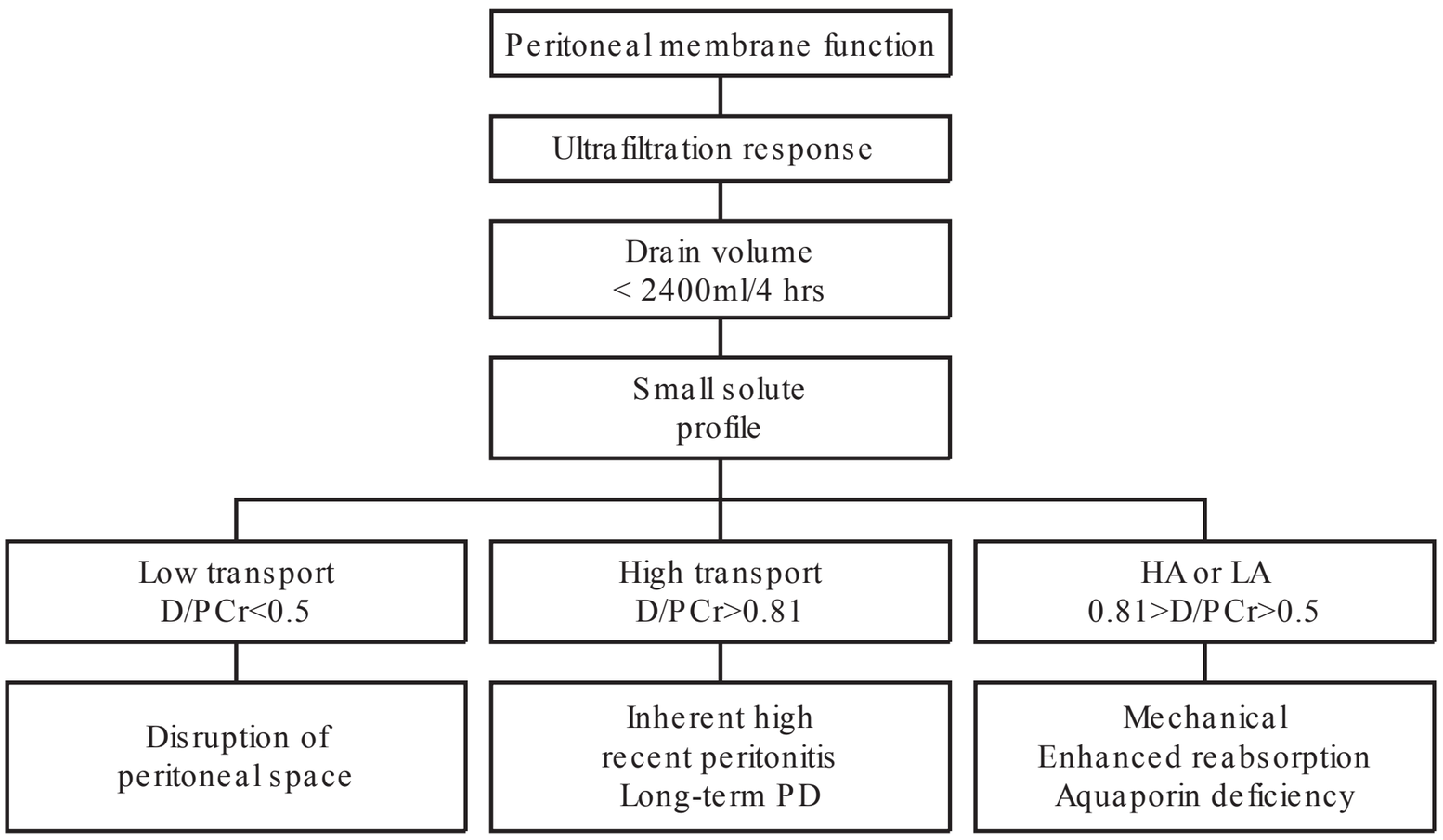

FIGURE 83.5 Algorithm for loss of ultrafiltration in continuous ambulatory peritoneal dialysis (CAPD) patients. *D/PCR*, dialysis:plasma creatinine ratio. (From Mujais S, Nolph K, Gokal R, et al. Evaluation and management of ultrafiltration problems in peritoneal dialysis. International Society for Peritoneal Dialysis Ad Hoc Committee on Ultrafiltration Management in Peritoneal Dialysis. *Perit Dial Int*. 2000;20 Suppl 4:S5–21.)

gest a reduction in UF capacity of the membrane. A rational approach to the patient with suspected UF failure is found in Figure 83.5.

Ultrafiltration Failure and High Solute Transport

Patients with loss of UF and current 4-hour PET ratios of D/D_0 glucose of less than 0.3 and D/P creatinine of greater than 0.81 are characterized as high solute transporters (see Fig. 83.2). These patients tend to have rapid small-molecular-weight solute transport and poor UF owing to high (rapid) glucose absorption and resultant rapid dissipation of the osmotic gradient. These patients are the largest group with true UF failure. Some patients have these transport characteristics at baseline and, if their dwell times are mismatched for their membrane transport characteristics, they often appear to have UF failure as they lose residual renal function and no longer have urine flow to supplement dialysate daily fluid losses. In other patients, the loss of UF is owing to an increase in membrane transport. This increase is caused by either an acquired increase in transport (formerly called type I membrane failure) or membrane changes associated with a recent episode of peritonitis.

Recent Peritonitis

It is a common clinical experience for PD patients to experience fluid retention during peritonitis. Compared to baseline, a PET performed during peritonitis reveals an increase in the D/P ratio for creatinine and a decrease in the D/D_0 ratio for glucose. There is also an increase in dialysate protein losses and a significant decrease in net UF.[134] In order to maintain UF during episodes of peritonitis, patients often need a temporary change in their standard dialysis prescription (shorter dwell times or increased tonicity) to maintain UF. Several studies have indicated that UF during an episode of peritonitis can be maintained with alternative osmotic agents such as icodextrin.[135]

Acquired Increase in Membrane Transport

An acquired increase in peritoneal transport over time on PD (formally called type I membrane failure) can cause chronic UF problems in CAPD. PET confirms high or high average transport rates with resultant rapid glucose absorption, loss of the osmotic gradient, and a decrease in net transcapillary UF. In contrast to the situation seen with peritonitis, where transport changes and protein losses usually are transient, small-solute transport changes and protein losses are more permanent with acquired loss of UF capacity.[136] There also tends to be less of a decline in dialysate sodium owing to the sodium sieving with convective transport. These changes are thought to result from an increase in effective surface area of the peritoneal membrane, supported by biopsy data showing an increase in vascular density in the membranes of long-standing PD patients.[137]

Risk factors for developing membrane changes are not firmly established but the incidence of an increase in membrane transport seems to increase with time on PD, implicating prolonged exposure of the peritoneum to dialysate as a possible cause. Davies et al. retrospectively analyzed glucose exposure in two patient cohorts: those whose transport characteristics were stable over 5 years and those who exhibited an increase in membrane transport over the same length of time. They found that the patients who were destined to exhibit increased transport over time had been exposed to a greater glucose load from the inception of peritoneal dialysis, strongly suggesting a relationship.[138] Similarly, among patients who were followed in the EAPOS study, those on icodextrin were less likely to have ultrafiltration problems than those on glucose.[139] This limited clinical evidence is consistent with a substantially more robust body of laboratory data linking the high glucose degradation products, glucose degradation products, and advanced glycosylation products to the

long-term anatomic and functional changes seen in the peritoneal membrane.

Most cases can be managed by shortening dwell times and using icodextrin solution for the long dwell. Because these patients have high (rapid) transport of small solutes, they have adequate urea and creatinine clearances even with short dwell exchanges. Occasionally, resting the peritoneum for at least 4 weeks through temporary transfer to HD has been associated with an improvement.[140]

Patients with apparent ultrafiltration failure due to rapid solute transport should be strongly considered for treatment with icodextrin, a slowly metabolized glucose polymer which acts as a colloid osmotic agent. Clinical studies have shown icodextrin to be a safe and effective alternative to glucose.[141] Although the rate of UF with icodextrin is slower than that with dextrose, the effect of icodextrin persists much longer, making it suitable for ultrafiltration during long dwells of up to 14 to 16 hours. In both CAPD and APD patients, icodextrin has been shown to provide ultrafiltration superior to that with either 2.5% or 4.25% dextrose.[103,142] This is associated with decreases in total body and extracellular fluid water, lower blood pressure, and, possibly, regression of left ventricular hypertrophy (LVH).[143] Although icodextrin is approved for only a single dwell in the United States, it has recently been reported that the use of icodextrin in two daily exchanges in CAPD patients improved volume status and LVH.[144] With the use of a combination of short dwells with dextrose-based solutions and icodextrin for the long dwell, it is usually possible to achieve adequate small-solute clearances and fluid removal in these patients; yet, a minority may require transfer to HD for volume and blood pressure control.

Ultrafiltration Failure and No Change or Average Solute Transport

Loss of UF in patients with no change or average transport characteristics tends to result from catheter malfunction, fluid leaks, excessive lymphatic reabsorption (formerly type III membrane failure), or aquaporin dysfunction. If loss of UF is owing to catheter malfunction or fluid leaks, the patients do not have a functional change in their membrane and usually can be maintained on PD after the problem has been resolved.

Excessive Lymphatic and Tissue Absorption

Excessive lymphatic absorption is a very uncommon cause of membrane failure related to excessive rates of lymphatic and tissue absorption of fluid from the peritoneal cavity.[132] Although these patients may not have a significant change in D/P values when compared to baseline, they do have drain volumes after 4 hours of dwell that are less than baseline values or that which would be expected based on standard therapy. A further diagnostic clue is that these patients tend to have higher dialysate sodium concentration during the dwell than controls (Fig. 83.6).

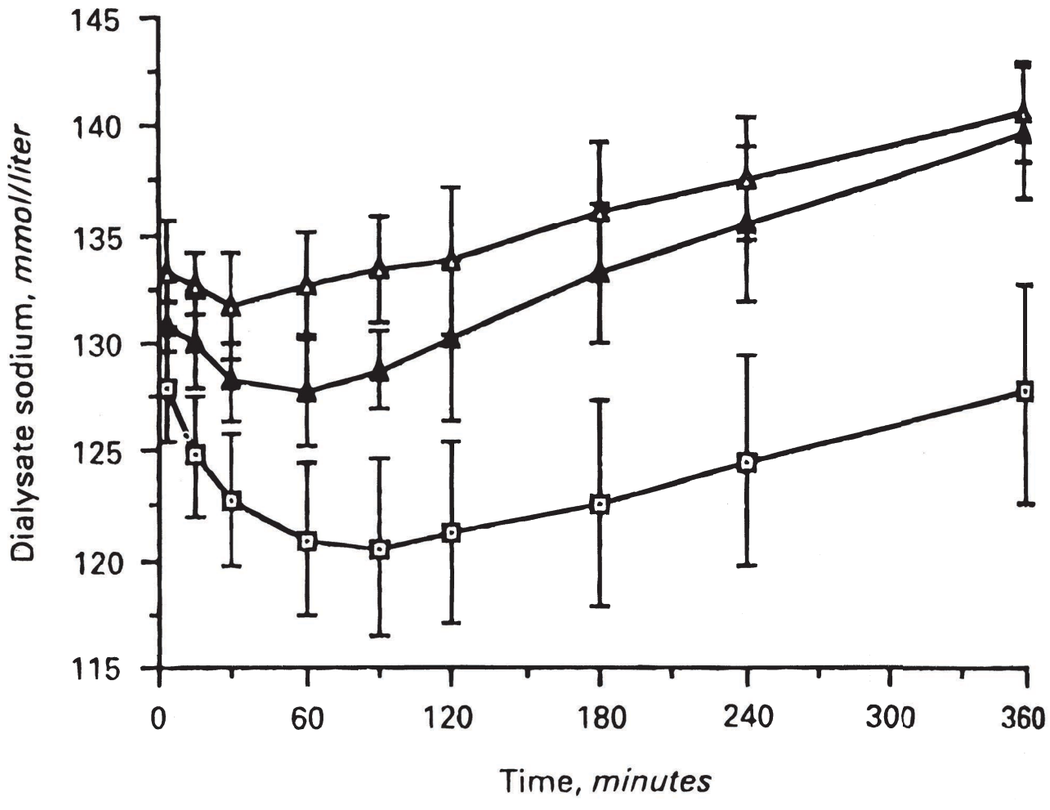

FIGURE 83.6 Dialysate sodium concentrations as a function of time in patients using 4.25% dextrose exchanges over 6-hour dwells. Results are compared in patients with normal ultrafiltration kinetics (*hollow squares*), those with high lymphatic absorption rates (*solid triangles*), and those with high glucose absorption rates (*hollow triangles*). (From Heimburger O, Waniewski J, Werynski A, et al. Peritoneal transport in CAPD patients with permanent loss of ultrafiltration capacity. *Kidney Int*. 1990;38:495, with permission.)

Aquaporin Dysfunction

Aquaporin dysfunction is a very rare condition.[145] Patients with suspected aquaporin dysfunction have damage to, decreased number of, or no water channels (ultra-small pores) that can lead to deficient crystalloid-induced UF.[146] These patients are diagnosed clinically by finding less than 400 mL of UF with a 4.25% PET and lack of sodium sieving early in the dwell. However, one must be careful to exclude patients who are very rapid transporters. Rapid transporters do exhibit sodium sieving, but it occurs so early in the dwell that if looked for after 60 to 90 minutes of dwell time it may be masked. These patients should respond clinically to use of colloid osmotic agents (such as icodextrin), which achieve ultrafiltration through a different mechanism and are not dependent on the water channels for UF.

Ultrafiltration Failure and Low Solute Transport

Patients with UF failure and low (slow) solute transport (D/D_0 glucose of more than 0.5 and a D/P creatinine of less than 0.5) tend to have inadequate small-solute clearances as well. Poor UF occurs despite the maintenance of adequate osmotic gradients. These patients are found to have a loss of functional peritoneum and the differential should include: peritoneal sclerosis (formally called type II membrane failure) or multiple peritoneal adhesions and, at times, patients with high lymphatic absorption rates. These patients often require transfer to HD.

DIALYSATE SOLUTIONS

Over the last three decades, a large number of patients have been successfully treated with conventional peritoneal dialysis solutions (Table 83.10) for long periods of time. However, several concerns have been identified with these solutions. First, the solutions are unphysiologic in that they are hyperosmolar, contain very high concentrations of glucose, and heat-sterilization generates toxic glucose degradation products. A large body of laboratory data and several observational clinical studies suggest that long-term use of conventional solutions results in structural and functional changes in the peritoneal membrane that limits its use as a long-term dialysis membrane. Peritoneal biopsies from patients on long-term peritoneal dialysis demonstrate mesothelial cell denudation, submesothelial fibrosis, neovascularization, and vasculopathy that primarily affects the postcapillary venule. This is associated with an increase in peritoneal solute transport rate and results in ultrafiltration failure in up to 30% of patients after 6 years of the therapy. Second, there are limitations with the use of glucose as an osmotic agent, particularly in the long dwells—these are the overnight dwell in a CAPD patient, and the day dwell in an APD patient. Absorption of glucose across the peritoneal membrane and dilution by the ultrafiltrate results in a progressive decline in glucose concentration, and hence, the ultrafiltration gradient during long dwells. In some patients this can result in net fluid reabsorption during long dwells and make it difficult to achieve euvolemia. Third, systemic glucose absorption can result in unwanted weight gain and is associated with a more atherogenic lipid profile. Fourth, the low pH of the fluid can cause infusion pain, particularly in the presence of peritonitis. Finally, concern has been raised that the unphysiologic peritoneal dialysis solutions can impair neutrophil and phagocyte function and increase the risk for and/or severity of peritonitis.

In order to overcome some of these limitations, several advanced peritoneal dialysis solutions have been introduced and are commercially available in different parts of the world. Glucose-based, lactate-buffered low glucose-degradation product peritoneal dialysis solution has been offered as a solution that is potentially more biocompatible. This claim is supported by animal studies and surrogate measures of peritoneal health in humans (higher concentrations of CA-125 and lower concentrations of profibrotic biomarkers). However, there are no data on the long-term effect of these solutions on either the structural or functional characteristics of the peritoneal membrane. Furthermore, the hope for a better preservation of residual renal function with these solutions has not been confirmed in three randomized, controlled trials.[124–126] Although previous studies showed no evidence for reduced episodes of peritonitis with these solutions, the recently published balANZ study reported longer time to the first episode of peritonitis in patients using a neutral pH, low GDP dialysate. Note however, that this study too demonstrated no beneficial effect on residual kidney function.[126a] There is no evidence for lower peritonitis rates with these solutions. Finally, an observational study from Korea demonstrated a lower risk for death in patients treated with low glucose-degradation product solutions.[147] These findings, although provocative, cannot be considered definitive. Thus, there are limited data that support a widespread use of low glucose-degradation product solutions.

Lactate is the most commonly used base but neutrophil function is better preserved with bicarbonate-containing dialysate compared to lactate-containing dialysate although bicarbonate with a high glucose concentration remains cytotoxic.[148] Lactate-containing dialysate with neutral pH is much less inhibitory of superoxide generation by neutrophils compared to standard lactate dialysate and is almost similar to bicarbonate-containing dialysate. Bicarbonate containing dialysate is feasible, in that the bicarbonate and dextrose can be kept in separate compartments and combined prior to infusion.[149] Two-chambered bicarbonate lactate-buffered PD fluid confers better phagocytosis and is associated with lower glucose degradation products compared to standard dialysate.[150] Use of bicarbonate-containing dialysate has been shown to improve peritoneal macrophage function.[151,152] Despite the in vitro data, bicarbonate solutions have not shown protection against peritonitis in patients. In a randomized controlled trial, bicarbonate-based peritoneal dialysis solutions were associated with significant lower severity of infusion pain.[153] Furthermore, a recent observational study from Korea has demonstrated a lower risk for death in patients treated with bicarbonate-based peritoneal dialysis solutions.[147] However, given the non-random assignment of patients to the different PD solutions, these findings cannot be considered definitive.

As mentioned previously, the glucose polymer icodextrin is an alternative to dextrose, particularly in high transporters. Dextrose is rapidly absorbed during a dwell, thus decreasing the osmotic gradient and leading to considerable caloric load. Glucose polymers are isosmolar; UF is obtained through colloid osmosis. Several randomized controlled trials have now demonstrated a higher UF volume, and lower extracellular water in patients treated with

83.10 Dialysate Composition

Dextrose, measured in g/dL (%) as the hydrous dextrose, available as 1.5%, 2.5%, and 4.25%
Sodium, measured as mEq/L, available at 132
Chloride, measured as mEq/L, available at 102, 96, and 95
Lactate, measured as mEq/L, available at 35 and 40
Calcium, measured as mEq/L, available at 2.5 and 3.5
Magnesium, measured as mEq/L, available at 0.5 and 1.5
Bag volumes, measured in L, available at 0.25, 0.5, 0.75, 1.0, 1.5, 2.0, 2.5, 3.0, 5.0, and 6

icodextrin.[102,103,142,143] This reduction in extracellular volume has been shown to be associated with regression of left ventricular hypertrophy in CAPD patients using icodextrin for the long overnight exchange. Peritonitis results in increased degradation of icodextrin, an increase in dialysate osmolality, and, therefore, increased ultrafiltration, in striking contrast to the changes seen with glucose dialysate in peritonitis.[154] Observational studies demonstrate a better preservation of peritoneal membrane function and lower mortality in patients treated with icodextrin.[139,147] However, none of these findings can be considered conclusive.

Amino acid–containing dialysate has been proposed as an alternative to glucose-containing dialysate. Polymorphonuclear cell function is not impaired by amino acid dialysate in contrast to dextrose-containing dialysate.[155] Amino acid dialysate has similar small- and large-molecular-weight solute transport and UF to equimolar dextrose dialysate.[156] The use of one exchange each day of a 1% amino acid dialysate for 6 months improved nitrogen balance, but did not result in a rise in the serum albumin. Disadvantages of amino acid dialysate include a rise in the blood urea nitrogen level and a fall in the bicarbonate; therefore, close attention must be paid to urea nitrogen clearance to prevent uremia and oral sodium bicarbonate often is necessary during use of amino acid dialysate.[157] Amino acid–containing dialysate is not available in the United States.

CATHETERS

Types of Peritoneal Catheters

The Tenckhoff catheter originally designed by Palmer and modified by Tenckhoff continues to be used in the majority of PD patients.[158–160] A number of variations are available. The straight or curved subcutaneous portion may have one or two cuffs. Double-cuffed catheters are used in the majority of patients. The intra-abdominal portion of the catheter may be straight or coiled. Coiled catheters were designed to decrease outflow problems and infusion pain but appear to have similar complication rates as straight Tenckhoff catheters.[160] Although there are plausible benefits to coiled and double-cuffed catheters, prospective studies comparing different catheter designs have not shown a difference in infections or need for catheter replacement.[161]

To decrease migration of the intra-abdominal portion and exit-site infections, Twardowski and associates[162] designed a catheter with a curved subcutaneous pathway in which both the internal and external exit sites are downward (swan-neck catheters). Prospective comparisons of swan-neck and straight catheters have consisted of small trials. From the trials, it appears that swan-neck catheters have a lower incidence of catheter migration than straight catheters although there is no clear difference in infectious complications or other mechanical complications.[163,164] Given the overall equivalency of outcomes, the choice of a straight or swan-neck catheter often is predicated upon the location of the exit site. An exit site in the upper abdomen that is directly lateral is best achieved with a straight catheter whereas an exit site in the lower abdomen that is pointed downward is best obtained with a swan-neck catheter. A modification of the swan-neck catheter with a presternal exit site had excellent 2-year survival of 95% in the hands of an experienced team.[165] Placement of a 5- to 10-g weight at the tip of the intra-abdominal portion of the catheter has also been shown to decrease catheter migration. Use of this "self-locating" catheter was shown to result in significant decreases in catheter dislocation, peritonitis, tunnel infections, cuff extrusion, leakage, and obstruction with a concomitant improvement in overall catheter survival.[166,167]

Peritoneal Catheter Placement

The location of the exit site should be discussed with the patient prior to catheter placement to avoid the beltline. Preoperative laxatives are indicated for constipation, commonly present in patients because of phosphate binders, and an important cause of catheter malfunction.[168] The patient should void prior to the procedure; if the patient has a neurogenic bladder, then urethral catheterization is performed. Placement can be done either with local anesthesia and sedation or general anesthesia. The patient usually does not require overnight admission. Prophylactic antibiotics (generally a cephalosporin) for catheter placement, given before the skin incision, decrease the risk of catheter-related peritonitis.[169]

Most PD catheters are inserted by a surgeon using a dissection technique. A small paramedian incision is made overlying the rectus sheath down through the muscle to the peritoneum. The catheter is inserted so that the deep cuff is within the rectus muscle and the tip is in the deep pelvis. A purse string of nonresorbable suture (to decrease the risk of subsequent leaks) is placed where the catheter enters the peritoneum. Catheter function is assessed intraoperatively by infusing and draining fluid. The subcutaneous tunnel is formed such that the superficial cuff is 3 cm from the skin surface and is directed downward or pointed laterally. A small exit site wound formed by a tapered tunneling device of the same diameter as the catheter is best for minimizing trauma and decreasing the risk of subsequent exit site infection and catheter-related peritonitis.[170] If needed, the exit site should be closed with Steri-Strips (3M, St. Paul, MN); sutures should be avoided in order to decrease the risk of wound infection with a foreign body.

Placement via a laparoscopy is increasingly commonly used. This technique allows direct intra-abdominal visualization.[171] Adhesions can be avoided and the tip of the catheter placed to allow optimal catheter function. In a randomized comparison of laparoscopic versus conventional dialysis catheter insertion (both done by surgeons) outcomes were not different except that the conventional placement was faster (14 vs. 22 minutes, $P < .0001$).[172] However, a recent report studied the outcomes of over 400 laparoscopically placed catheters in which adjunctive procedures (e.g., rectus sheath tunneling, omentopexy, adhesiolysis, or resection of epiploic appendices) were employed as well. The catheter survival rate

was 99% at an average of 21 months follow-up; the revision free survival was 96%.[171] The laparoscope may be particularly useful in patients with previous surgery or when placement by dissection results in a nonfunctioning catheter.

Blind percutaneous catheter placement may be used for placement of a catheter for acute renal failure to be used for a short time. However, for chronic dialysis patients, it does not allow a peritoneal examination nor does it allow surgical repair of intra-abdominal abnormalities, such as herniorrhaphy or omentopexy.[170] Additionally, the risk of bowel perforation makes percutaneous placement a less desirable technique. On the other hand, percutaneous placement under fluoroscopic guidance can be successfully used in a large proportion of uncomplicated cases.[173]

To decrease the risk of peritonitis from the formation of a biofilm, Moncrief and coworkers[174] developed a new insertion technique (Moncrief-Popovich technique). At insertion, the entire external portion of the catheter is buried in abdominal wall subcutaneous tissue. Three to 5 weeks later, the catheter is externalized via a small incision, which becomes the exit site. Burying the external portion of the catheter for up to 2 years does not change technique survival.[174a] Data regarding a possible decrease in infectious rates with this technique are conflicting.[175,176] A recent, large nonrandomized study did show a decrease in infections and leaks and an increase in catheter survival.[177] This technique has been employed for use with presternal as well as abdominal catheters.[178] Of note, however, use of the Moncrief-Popovich technique does appear to allow for earlier patient acceptance of catheter placement, analogous to the early placement of arteriovenous fistulas in patients planning to perform hemodialysis. Furthermore, because of the previous healing period, no "break in" period is required and full dose peritoneal dialysis may be started immediately upon externalization of the catheter.

Children require special consideration. In infants, the exit site is located above the diaper area to prevent contamination. Partial omentectomy is useful to prevent outflow problems. In boys, herniotomy and ligation of patent processus vaginalis at the time of catheter placement decrease the risk of subsequent inguinal hernia and hydrocele.[179]

Perforation of the bladder or bowel or laceration of the spleen is an uncommon occurrence, but adhesions increase the risk. Perforation of a hollow viscus should be considered if the effluent is feculent or when watery diarrhea, polyuria, or watery vaginal discharge occurs with infusion of dialysate. Minor bleeding frequently occurs after catheter insertion, but generally stops quickly and spontaneously.[180] Flushing the catheter with heparinized dialysate (500 U/L) is useful to clear the catheter and prevent blockage by clots.

Postoperative Management and Exit Site Care

If possible, initiation of PD is postponed by about 2 weeks from the time of catheter placement to allow healing and prevent leaks ("break-in"). The break-in period may need to be longer in patients who may have problems with wound healing, patients with a failing kidney transplant, or other conditions for which long-term corticosteroids are prescribed. During the break-in period, the catheter should be flushed several times with 1 L of dialysate or saline until the effluent is clear and then capped until training begins.[160] However, PD can be started within hours of placement of the catheter, if clinically indicated. Under such circumstances, the patient may initiate low-volume supine PD—best achieved with a cycler.[181]

Postoperative sterile dressing changes until healing takes place may help reduce infection risk. The surgical dressing should be left intact for 1 week unless there is bleeding. The exit site should be kept dry until well healed—this may require up to 2 weeks. During this interval, patients should not shower or bathe in tubs—personal hygiene should be performed with sponge baths. Once healed, many centers advise washing the exit site with bactericidal soap and water during routine bathing. Once the exit site is well healed, swimming in chlorinated pools or the ocean is permitted, but swimming in creeks or ponds or the use of hot tubs should be avoided, because this may result in infection.[182]

Mechanical Complications

Early inadequate outflow occurs after 7% of catheter insertions, requiring replacement in one half of these patients.[183,184] Constipation may lead to shifting of the catheter position, drainage failure, but only rarely catheter loss. Ideally, the catheter tip should be in a pelvic gutter, because this location ensures good hydraulic function of the catheter and minimizes risk of omental entrapment.[185] Tip migration to the epigastric or hypochondrial regions is generally associated with dysfunction.[183,184] Poor drainage owing to catheter malposition in the upper quadrants may be corrected by surgical repair (either open or laparoscopically).

There are other causes of catheter dysfunction in addition to catheter malposition. One- or two-way obstruction may result from clots or fibrin within the lumen. Forcibly flushing with heparinized saline may resolve this problem, but fibrinolytic agents may be effective if this fails.[186,187] Omental obstruction may necessitate omentectomy, especially in children.[179,183] Omentopexy or partial omentectomy at catheter placement improves catheter survival; the latter is performed routinely in children.[188]

Peritoneal dialysate leaks, which may occur at several different locations,[189]develop in 5% to 10% of catheters in the immediate postoperative period[183,184] and in 2% to 4%[184] of catheters later in the course of CAPD. Dialysate leaking from the exit site presents as clear fluid that is strongly test strip–positive for glucose. Leaks at the internal cuff may present as abdominal wall edema. These leaks may result from the use of resorbable suture material at the deep cuff, placement in a median rather than paramedian site, early initiation of CAPD, or hernia formation.[190,191] Computed tomography (CT) scan peritoneography (using Omnipaque, 50 mL/L of dialysate) is the best way to evaluate leaks and hernias.[192] A dialysate leak may resolve with PD in the supine position or temporary

cessation of PD (using HD).[189] Dialysate leaks from the exit site often are associated with infection; thus prophylactic antibiotics should be given.[193] If a leak occurring more than 1 month after catheter insertion does not resolve within 4 days of reduced dialysate volumes, or if it recurs after full volumes are resumed, surgical correction generally is required.[194]

Diagnosis of Peritoneal Dialysis-Related Infections

A diagnosis of peritonitis is made when a patient has two of the following three: (1) cloudy peritoneal effluent and abdominal pain, (2) white blood cell count ≥100 cells/L with more than 50% polymorphonuclear cells, and (3) positive Gram stain or culture.[195] The patient usually does not have a fever. The effluent white blood cell concentration is a less sensitive indicator of peritonitis if the patient is already on antibiotics or if the patient is on automated PD who has either a dry abdominal cavity or has had fluid for a short period of time at the time of presentation. In these circumstances, the percentage of neutrophils (more than 50%) is more useful than is total white blood cell concentration.

The optimum technique for culture of peritoneal dialysate consists of centrifugation of 50 mL of peritoneal effluent at 3,000 g for 15 minutes followed by resuspension of the sediment in 3 to 5 mL of sterile saline and culture on both solid and liquid media.[195] More commonly, however, 5 to 10 mL of dialysis effluent is injected directly into blood culture bottles. When processed in this fashion the culture is "sterile" in approximately 14% to 20% of episodes that meet the criteria for peritonitis based on cell count.[196] A fastidious microorganism that has not grown in culture probably causes most of these episodes. When subsequently recultured, a microorganism is identified in one third.[197] Also, recent antibiotic exposure can render dialysate "sterile," despite active peritonitis. Mycobacteria always should be considered in peritonitis that is culture negative. Such patients have cloudy effluent, abdominal pain, and fever. Extraperitoneal TB is not necessarily present. Polymorphonuclear cells predominate in the effluent and, thus, do not distinguish Mycobacterium peritonitis from bacterial peritonitis. Acid-fast bacillus (AFB) smears of the effluent, even examining three concentrated specimens, are seldom positive; therefore, the diagnosis is generally made on culture, delaying treatment for weeks. Peritoneal tissue cultures are more optimal than culture of peritoneal fluid.

Approximately 6% of patients with culture-positive effluent present with abdominal pain and clear effluent.[198] A delayed effluent cell reaction occurs in two thirds of these patients, but one third never develop an appropriate cellular response to infection. When not experiencing peritonitis, such patients have a lower dialysate cell count (particularly macrophages and CD4 lymphocytes) and a delayed production of interleukin -6 and -8, compared to other patients. Though there are truly noninfectious causes of cloudy dialysate (see later) any patient on PD who presents with abdominal pain should be considered to have peritonitis until proved otherwise.

An exit site infection is defined by the presence of purulent drainage, with or without erythema, at the catheter exit site.[195] Induration and tenderness at the exit site are abnormal and may indicate infection. In the absence of drainage, erythema of the exit site (which is normally flesh colored) does not necessarily indicate the presence of infection; erythema may result from irritation or trauma to the exit site,[199] but is seldom associated with catheter loss unless drainage also is present.[168] Nonpurulent drainage and crusting of the exit site do not necessarily represent infection, nor does a positive culture of a normal-appearing exit site.

An infection of the subcutaneous catheter (or "tunnel infection") is present when there is pain, tenderness, erythema, or induration over the subcutaneous pathway. Tunnel infections most often occur in the presence of an exit site infection.[195] Tunnel infections may be clinically occult. This has been shown by numerous studies using sonography of the subcutaneous tunnel in patients with exit site infections.[200] When peritonitis occurs in conjunction with an exit site infection owing to the same microorganism (particularly Staphylococcus aureus or Pseudomonas aeruginosa), the presumption should be that there is a tunnel infection.[201]

PD-related infections remain a major problem. Such infections are responsible for the majority of catheter loss and contribute to transfer of the patient to HD.[202,203] Peritonitis is a major cause of hospitalization (Fig. 83.7).[204] Peritonitis occasionally results in death, either directly from sepsis or indirectly from ensuing complications such as cardiovascular disease.[205,206]

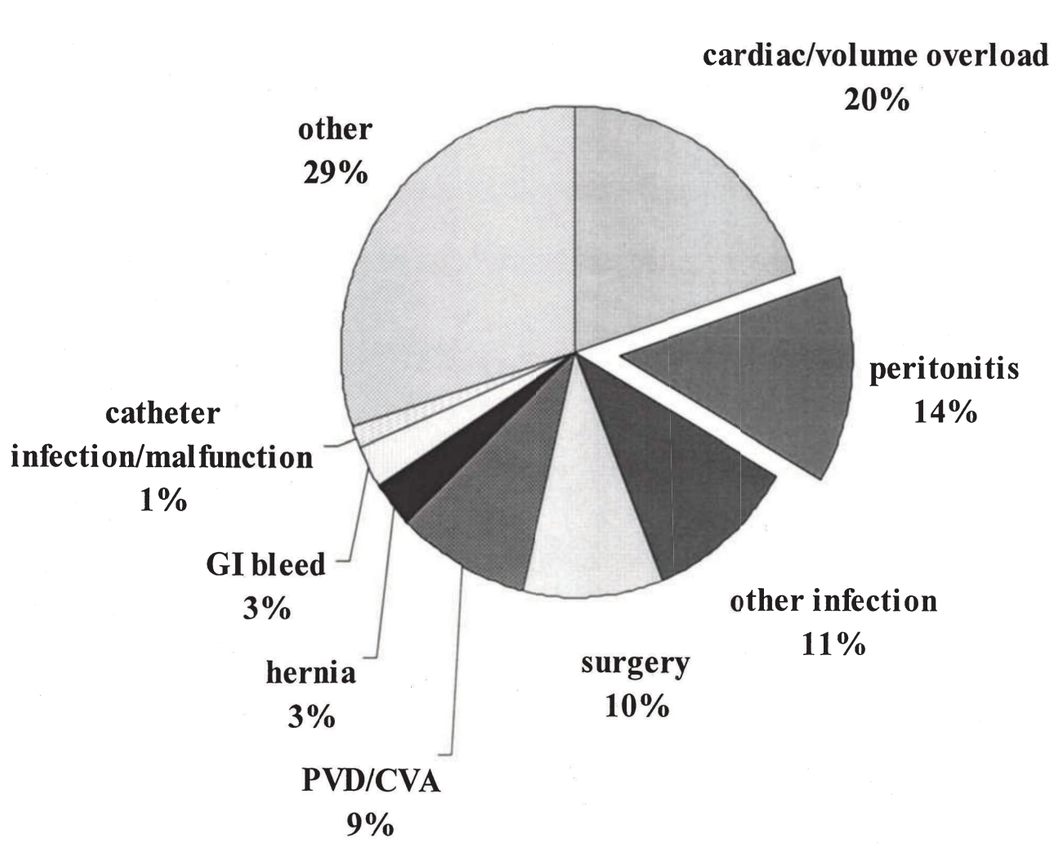

FIGURE 83.7 Causes of 274 hospitalizations for 126 peritoneal dialysis patients, as percentages. (From Fried L, Abidi S, Bernardini J, et al. Hospitalization in peritoneal dialysis patients. *Am J Kidney Dis.* 1999;33:927, with permission.)

Connection Devices

Evolution of connection techniques over time have resulted in a dramatic lowering of peritonitis rates, particularly those owing to organisms such as coagulase-negative Staphylococcus. For many years, the standard connection system was a straight line with an empty dialysate bag attached to the patient between dialysis exchanges. The exchange was performed manually. The straight line system has been replaced with safer connection systems, such as the Y-set and double-bag system. With the Y-set, the patient connects the catheter to a Y-set of tubing attached to a full dialysate bag and an empty bag. The patient sequentially flushes dialysate through the line into the drain bag to clear air, then drains the effluent from the peritoneum, infuses the fresh dialysate, and disconnects the Y tubing, either capping the catheter or snapping off the tubing. This strategy is known as "flush before fill" and was initially brought into practice by Buoncristiani.[215] The double-bag system is a further improvement in technology, because both the drain and fill bags are already attached to the Y tubing; therefore, the only possible site of contamination is during the connection the patient makes to the exchange tubing attached to the catheter. Peritonitis rates are significantly lower with the double-bag system compared to the Y-set in high-risk populations; however, there does not appear to be a difference in exit site infections or catheter survival.[216–218] There are lower rates of gram-positive peritonitis with the double-bag as compared to the Y-set, suggesting that this method further reduces the risk of contamination (Fig. 83.8) and, hence, the double-bag system has become the community standard of care in the United States.

The data are conflicting on whether peritonitis rates are lower on the cycler compared to CAPD, although a number of studies suggest that is the case (Fig. 83.9). A recent meta-analysis compared randomized trials of APD versus CAPD. In two out of three trials, peritonitis was significantly less common in APD patients (relative risk 0.75 vs, CAPD).[219] Two out of the three trials were quite small and the meta-analysis was heavily

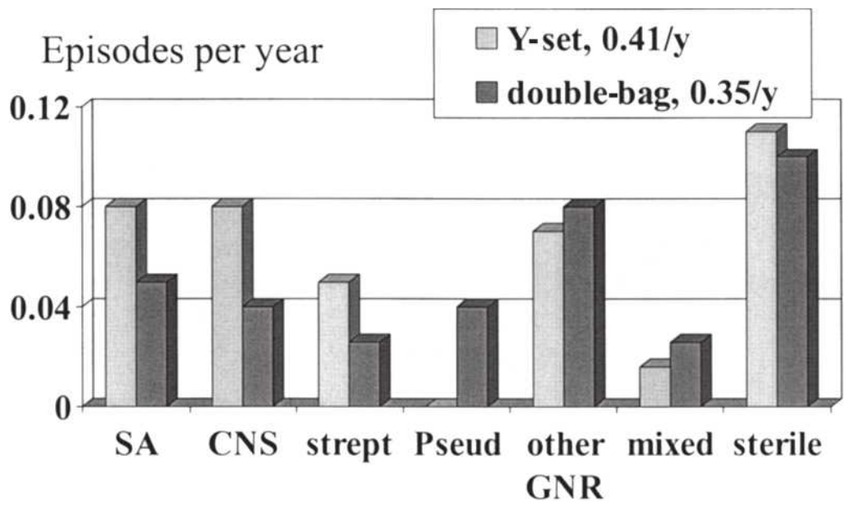

FIGURE 83.8 Episodes of peritonitis per dialysis year at risk in patients randomly assigned to the Y-set or the double-bag system for CAPD. (Modified from Li PK, Szeto CC, Law MC, et al. Comparison of double bag and Y set disconnect systems in continuous ambulatory peritoneal dialysis: a randomized prospective multicenter study. *Am J Kidney Dis*. 1999;33:535.)

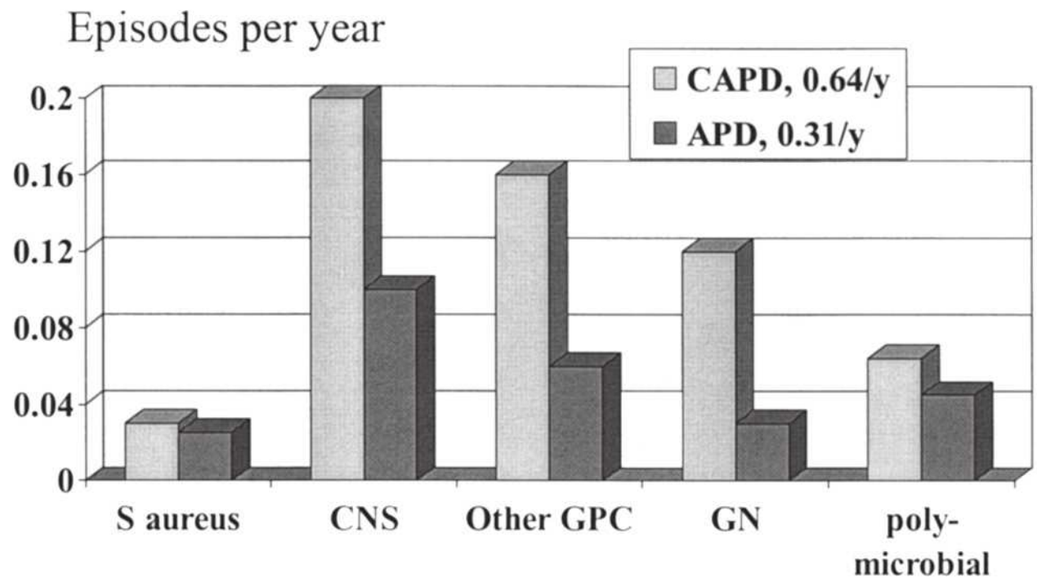

FIGURE 83.9 Episodes of peritonitis per dialysis year at risk in patients on CAPD versus APD. The center uses prophylaxis for *S. aureus* nasal carriers; therefore, *S. aureus* peritonitis rates are very low in both groups. (Modified from Rodriguez-Carmona A, Perez Fontan M, Garcia Falcon T, et al. A comparative analysis on the incidence of peritonitis and exit-site infection in CAPD and automated peritoneal dialysis. *Perit Dial Int*. 1999;19:253, with permission.)

weighted toward the trial by de Fijter et al.[220] In this study, 82 patients were randomized to APD or CAPD. Patients on APD had close to a 50% reduction in peritonitis. However, in an analysis of the USDRS database, patients on CAPD appeared to have a lower peritonitis rate than patients on APD.[221] A small study suggests that NIPD may be associated with lower infection rates, perhaps due to enhanced peritoneal immune function as a consequence of the abdomen being kept dry for a portion of the day.[222] However, most of these studies were undertaken prior to the widespread use of the double-bag system for CAPD; with use of contemporary connectology, the difference in peritonitis rates between CAPD and APD, if any, is small and not clinically significant.

Catheter Infections

The International Society for Peritoneal Dialysis has published comprehensive reviews of the approach to infectious complications in the PD patients.[195,223] The reader is referred to those articles for details; the following text is a broad overview.

There is marked variation in reported rates of exit site infections, in part because of differing definitions and because exit and tunnel infections are not always reported separately. Furthermore, much of the infection data precedes recent innovations in connector technology and prophylactic treatment. In a randomized controlled trial of mupirocin versus gentamicin prophylaxis, the rate of exit site infections in the mupirocin group was 0.54, similar to previous studies.[224] The rate of clinically obvious tunnel infection is 0.19 per year[225]; however, when an exit site infection is present, fluid collections along the subcutaneous pathway can be frequently demonstrated by ultrasound examination. Tunnel involvement is common when an exit site infection is concurrent.[200,226]

Microorganisms causing exit site infections are shown in Table 83.11.[227,228] The most common organism causing exit site and tunnel infections is S. aureus, which may be difficult

83.11 Pathogens Causing Exit Site Infections

Pathogens
Staphylococcus aureus
Staphylococcus epidermidis
Gram-negative
Culture-negative
Total

[a]Much lower in programs using S. aureus prophylaxis.[H1]
Modified from Flanigan MJ, Hochstetler LA, Langholdt D, et al. Continuous ambulatory peritoneal dialysis catheter infections: diagnosis and management. Perit Dial Int. 1994;14:248; Holley JL, Bernardini J, Piraino B. Infecting organisms in continuous ambulatory peritoneal dialysis patients on the Y-set. Am J Kidney Dis. 1994;23:569.

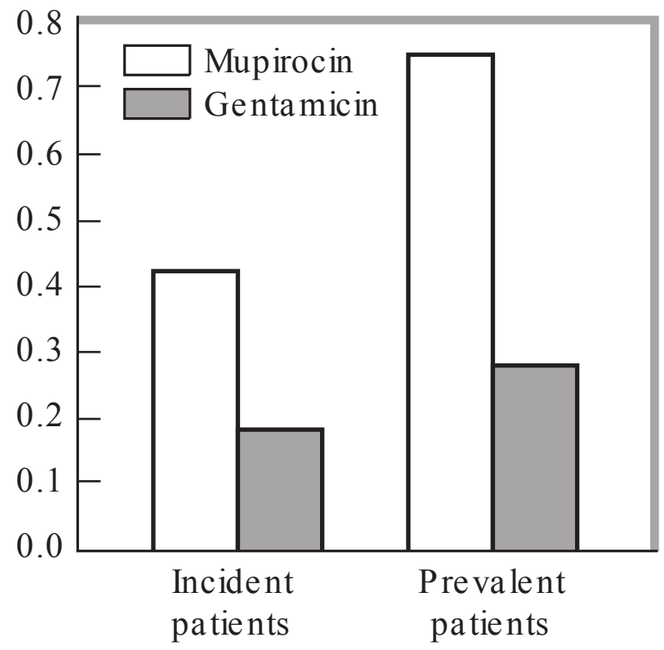

FIGURE 83.10 Rates of exit site infections in incident (on ≤3 months) and prevalent patients (on >3 months). In both groups, those who were using gentamicin exit site cream had significantly lower ($P < .01$) rates than those who were using mupirocin.

to resolve and can lead to peritonitis and catheter loss.[201,203] P. aeruginosa is the second most common cause of exit site and tunnel infections and frequently recurs or is refractory to antibiotic therapy and tunnel revision.[229,230] Therefore, early catheter removal is appropriate if the patient does not respond to a course of antibiotics. Staphylococcus epidermidis and culture-negative exit site infections are generally nonpurulent, and only infrequently do they cause peritonitis.[231]

The peritonitis rate in patients who have catheter infections is more than twice that of patients who do not.[231] Involvement of the tunnel, especially the inner cuff as demonstrated by ultrasound, predicts subsequent peritonitis.[200] Even in the absence of a clinical tunnel infection and with resolution of exit site infection with therapy, the deep cuff may harbor S. aureus or P. aeruginosa, resulting in recurrent peritonitis.[232]

A number of studies have demonstrated the efficacy of local antibiotics applied to the exit site to prevent infections. Daily exit site mupirocin is highly effective in reducing S. aureus exit site infections.[224,233–235] It must be noted, however, that organisms with low level mupirocin resistance have begun to emerge.[236,237] Although this is not yet a clinical concern, should the organisms acquire high-level resistance (minimum inhibitory concentration [MIC] ≥512 μg per mL), increased infection and/or relapse rates may ensue. Ciprofloxacin otologic solution, 0.5 mL single-dose vial, applied daily as part of routine care, reduced both S. aureus and P. aeruginosa exit site infections compared to historical controls.[238] However, the use of ciprofloxacin may be prohibitively expensive for many patients. A double-blind study compared the effects of 0.1% gentamicin sulfate versus 2% mupirocin applied to the exit site daily (Fig. 83.10).[224] Use of gentamicin resulted in significantly decreased rates of exit site infections and peritonitis. Gram-negative infections were markedly diminished by the use of gentamicin and there were no infections with P. aeruginosa; the frequency of S. aureus infections was unchanged. Gentamicin has the added advantage of being far less expensive than is mupirocin. This regimen is likely to become the preferred mode of prophylaxis for catheter-related infections.

Peritonitis

Peritonitis rates appear to be decreasing over the last two decades. In a recent analysis of a large cohort of PD patients (over 40,000), there was a 2% to 3% decline in peritonitis from 2000 to 2003.[239] In an analysis of peritonitis rates in the United States and Canada, Mujais reported peritonitis rates of one per 32.7 months in the United States and one per 27.6 months in Canada.[240] The organisms that most commonly cause peritonitis are listed in Table 83.12.[228,241,242] Szeto et al. have documented that the percentage of S. epidermidis peritonitis is significantly decreasing with the use of the disconnect systems and this increases the relative proportion of all peritonitis episodes that are caused by gram-negative organisms.[243] Many other organisms in addition to those listed have been identified in episodes of peritonitis, including those caused by fungi, protozoans, algae, viruses, and mycobacteria.[195] The outcome of peritonitis is highly organism-specific. Etiologies of peritonitis are shown in Figure 83.11.

A number of demographic features are associated with an increased risk for peritonitis. White, nondiabetic patients aged 20 to 59 years have the lowest risk of peritonitis. The reason for the increased risk seen in blacks is not understood.[244,245] Conflicting data exist on whether diabetic patients have an increased risk of peritonitis.[246,247] Age greater than 60 years was a risk factor for peritonitis in some reports, but most studies indicate that elderly patients have similar peritonitis rates as younger patients.[248] Peritonitis rates in children are higher than those of adults.[249] Immunosuppressed patients are also at increased risk, especially for

83.12 Pathogens Causing Peritonitis Using Disconnect Systems

Pathogens	Episodes/Year
Staphylococcus epidermidis	0.1–0.2
Staphylococcus aureus	0.15[a]
Other gram-positive	0.1–0.2
Gram-negative	0.1
Polymicrobial	0.01
Fungi	0.01
Culture-negative	0.01–0.1
Total	0.4–0.6

[a]Approximately one third of this is in programs using S. aureus prophylaxis.
Modified from Holey JL, Bernardini J, Piraino B. Infecting organisms in continuous ambulatory peritoneal dialysis patients on the Y-set. Am J Kidney Dis. 1994;23:569; Tofte-Jensen P, Klem S, Nielson PK, et al. PD-related infections of standard and different disconnect systems. Adv Perit Dial. 1994;10:214; Lupo A, Tarchini R, Carcarini G, et al. Long-term outcome in continuous ambulatory peritoneal dialysis: a 10 year survey by the Italian cooperative peritoneal dialysis study group. Am J Kidney Dis. 1994;24:826.

infections owing to S. aureus and fungi.[250] Peritonitis risk is also increased after an episode of peritonitis.[251]

Contamination at the time of an exchange, usually but not invariably resulting in coagulase-negative staphylococcal peritonitis, remains a leading cause of peritonitis. S. epidermidis peritonitis is not usually caused by a catheter infection or

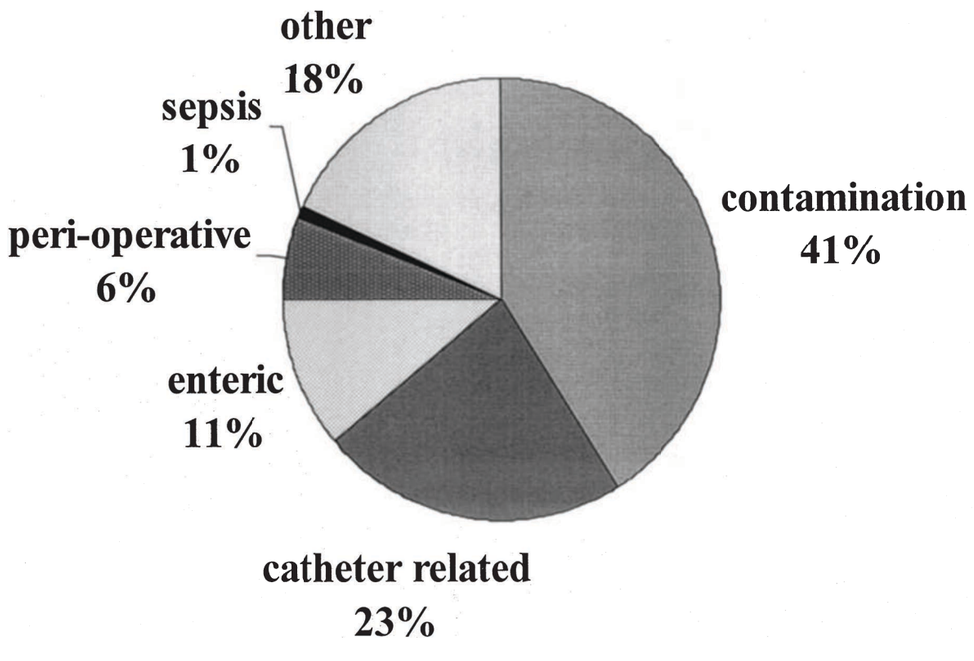

FIGURE 83.11 Etiologies of peritonitis. (Modified from Harwell CM, Newman LN, Cacho CP, et al. Abdominal catastrophe: visceral injury as a cause of peritonitis in patients treated by peritoneal dialysis. *Perit Dial Int*. 1997;17:586, with permission.)

colonization of the skin, nose, or exit site; however, S. epidermidis can colonize the peritoneal catheter, producing a slime layer (or biofilm) that can extend from the exit site through the cuff(s) into the peritoneal cavity.[252,253] The rate of bacterial colonization of the catheter is related to the degree of bacterial contamination of the exit site at the time of catheter insertion, but, within 3 weeks of insertion, most catheters are colonized.[254] The relationship of biofilm to peritonitis is unclear. Recurrent or relapsing peritonitis (defined as a second episode owing to the same organism within 4 weeks of stopping antibiotics) is generally caused by Staphylococcus and may be related to the presence of biofilm, which shields bacteria from antibiotics.[195] The coagulase-negative staphylococci isolated from patients with peritonitis are more likely to be producers of biofilm than are isolates not associated with peritonitis; however, biofilm formation does not invariably lead to peritonitis.[255,256] The keys to preventing peritonitis caused by coagulase-negative staphylococcal peritonitis are avoidance of connection techniques requiring spiking of bags and extensive training of the patient in aseptic technique. Miller and Findon have demonstrated that proper hand washing and drying prior to performance of an exchange sharply reduces bacterial numbers on the spike connection and in the peritoneal space after touch contamination.[257] Furthermore, patients should be trained how to identify contamination and report to the dialysis unit; prophylactic antibiotics should be administered under appropriate circumstances.[182]

S. aureus carriage and catheter infections are another source of peritonitis. S. aureus in the nares, at the exit site, or on the skin is associated with S. aureus catheter infection and peritonitis. Prevention of S. aureus peritonitis is critical, because the outcome is worse compared to that of other staphylococcal infections. Several antibiotic protocols have been shown to decrease the risk of S. aureus infection in PD patients.[216] These predominantly use intranasal mupirocin cream, twice a day for 5 days monthly for carriers, or daily at the exit site. These protocols are uniformly effective in reducing exit site infections but not peritonitis. Exit site mupirocin is also effective in reducing S. aureus peritonitis.[233,234] As discussed previously, low-level mupirocin resistance has been reported. Gentamicin is equally efficacious as mupirocin for prophylaxis against S. aureus and is superior for the prevention of gram-negative infections.[224]

Gram-negative peritonitis, which is associated with considerable morbidity, is not well understood.[258–260] The bowel may be a source, through translocation of bacteria across the bowel wall or secondary to organ pathology. Constipation and enteritis have both been associated with peritonitis due to enteric organisms.[261,262] Possibly due to the effects on colonic motility, hypokalemia has been associated with an increase in enterobacterial peritonitis as well.[263] Peritonitis can rarely be caused by intra-abdominal pathology and under those circumstances is associated with severe symptoms and commonly results in transfer of the patient to HD or death, especially if surgery is delayed.[260,264] Examples of primary

intra-abdominal diseases that can present as PD peritonitis include ischemic bowel, cholecystitis, appendicitis, perforated ulcers, colonic polypectomy, and diverticula (see section on intra-abdominal catastrophes). An elevated amylase level in the dialysate effluent is a clue to the presence of enteric peritonitis.[265]

Procedures, such as colonoscopy, endoscopy with sclerotherapy, dental manipulation, endometrial biopsy, liver biopsy, and laparoscopic cholecystectomy can result in peritonitis; thus, antibiotic prophylaxis is indicated.[266–268] Other unusual causes of peritonitis are vaginal leak of dialysate and the use of intrauterine devices.[269] It is recommended that the abdomen be emptied of fluid prior to procedures involving the abdomen or pelvis.[195]

Fungal peritonitis accounts for 2% to 3% of all peritonitis episodes.[270,271] Abdominal pain may be severe and associated with fever. Patients may be acutely ill and appear to have a surgical abdomen; death may result, particularly if catheter removal is delayed.[272] A recent observational study of Australian patients suggests that mortality from fungal peritonitis may be decreasing. Previous small series described a mortality rate greater than 20%. In the largest series to date (162 patients), mortality rate was 9%.[270] Prior antibiotic therapy and frequent bacterial peritonitis are predisposing causes. Prophylaxis, mainly using nystatin during antibiotic therapy, appears to be most effective in programs with high fungal peritonitis rates (Table 83.13).[273–278] Programs with a low fungal peritonitis rate do not appear to benefit from prophylaxis.

The differential diagnosis for truly sterile cloudy fluid is broad and is best approached by considering whether turbidity is due to cellular or acellular elements.[279] Acellular causes include fibrin and triglycerides; the latter may be due to lymphatic obstruction, superior vena cava syndrome, pancreatitis, or certain dihydropyridine calcium channel blockers. Cellular elements may include polymorphonuclear leukocytes, eosinophils, red blood cells, or malignant cells. Intraperitoneal generic vancomycin and amphotericin may cause chemical peritonitis.[280,281] Icodextrin had been previously reported to cause sterile peritonitis with the number of cases peaking in 2002.[282,283] This was determined to be a consequence of contamination with a bacterial peptidoglycan that was introduced during the manufacturing process.[284] Correction of the manufacturing process has virtually eliminated this problem (incidence now 0.01%).

Treatment of Peritoneal Dialysis-Related Infections

Exit Site Infections

The initial antibiotic for an exit site infection must be active against staphylococci, with subsequent therapy dependent on the specific organism identified. Oral antibiotics are as effective as intraperitoneal antibiotics with the exception of infections with methicillin-resistant Staphylococcus aureus (MRSA); these will usually require treatment with vancomycin. Sonography of the tunnel may be useful, although not always necessary, to determine length of therapy (Fig. 83.12).[195] Infections limited to the exit site require an average of 2 weeks of antibiotic therapy, whereas involvement of the superficial tunnel lengthens average therapy to 3 weeks or more. Involvement of the deep cuff requires 2 months or more of antibiotic therapy and may require removal of the catheter to prevent peritonitis.[285] Local care of the exit site is generally intensified and, in mild or equivocal exit site infection when the tunnel is not involved, this may suffice as therapy. If prolonged antibiotic therapy fails to resolve the exit site infection, revision of the tunnel with removal of the external cuff (in two cuffed catheters) may help to prolong the life of the catheter in a select group of patients but there is limited long-term data.[286] An incision is made over the tunnel to expose the cuff, which is carefully shaved

83.13 Fungal Peritonitis without and with Prophylaxis

Reference	Prophylaxis	Incidence[a]
Zaruba[278]	Nystatin tid	0.20 vs. 0.03
Robitaille[273]	Nystatin or ketoconazole	0.14 vs. 0
Wadhwa[277]	Fluconazole qod	0.08 vs. 0.01
Lo[276]	Nystatin qid	0.02 vs. 0.01
Thodis[275]	Nystatin qid	0.02 vs. 0.02
Williams[274]	Nystatin qid	0.01 vs. 0.01

[a]Antibiotic associated fungal peritonitis, in episodes per year. Rate without prophylaxis given first.

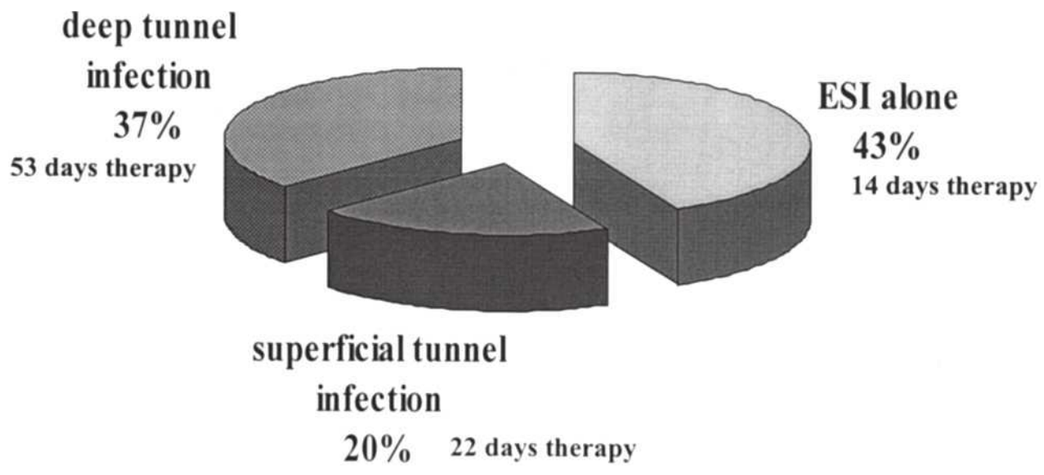

FIGURE 83.12 Extent of *S. aureus* catheter infection ($n = 49$) using sonography with mean days of therapy also shown. (From Vychytil A, Lilaj T, Lorenz M, et al. Ultrasonography of the catheter tunnel in peritoneal dialysis patients: what are the indications? *Am J Kidney Dis.* 1999;33:722, with permission.)

from the catheter. The area of granulation tissue and cellulitis may also be débrided. Cuff shaving and tunnel revision are never effective if catheter-related peritonitis is present.

Pseudomonas exit site infections are particularly prone to recurrence and often lead to peritonitis, which is a devastating complication.[287] Therefore, if the patient has a history of prior exit site infection with Pseudomonas, the antibiotic chosen for empiric therapy should be efficacious against that organism (e.g., oral quinolone). Dual therapy is sometimes needed and the duration of therapy may need to be extended to as long as 6 weeks. Recurrent and refractory exit site infections might be best managed with catheter replacement. In such high-risk patients, to prevent recurrence in a new catheter, consideration should be given to using gentamicin or ciprofloxacin otic solution at the exit site, as previously described.

Peritonitis

As discussed previously, not all patients who present with cloudy dialysate will prove to have peritonitis. Nevertheless, to avoid delay in treatment, empiric antibiotic therapy should be started upon presentation with cloudy dialysate.[288,289] Initial therapy should include coverage for both gram-positive and gram-negative organisms.[195] This should be guided by knowledge of both the patient's history and the individual program's pattern of microorganisms responsible for peritonitis and their antibiotic sensitivities.

A first-generation cephalosporin or vancomycin should be used to provide gram-positive coverage. A single daily dose of cefazolin, 15 mg per kg, results in dialysate concentration levels above the MIC over 24 hours for sensitive organisms, allowing once a day dosing (for those without residual renal function).[290] In CCPD patients treated with intermittent dosing with cefazolin, the antibiotic should be administered in a long day exchange (at least 4–6 hours of dwell) immediately preceding the overnight cycling. Alternatively, cefazolin could be administered in every bag for either CAPD or APD patients (500 mg per L loading dose followed by 125 mg per L in each subsequent bag). Antibiotics could be administered with intermittent therapy. Vancomycin is needed in patients with a cephalosporin allergy and could be used in centers with a high incidence of infection with methicillin-resistant organisms. Centers with a high prevalence of methicillin-resistant organisms, however, still may use cefazolin empirically since the relatively high concentration of cefazolin in the dialysate can be effective even in the presence of resistant organisms. Patients on vancomycin generally require dosing at 3- to 5-day intervals—the more frequent dosing is important particularly in patients with significant residual renal function or with frequent cycling at night. The frequency of dosing can be individualized by checking plasma vancomycin levels and levels maintained ≥ 15 μg per mL.

Gram-negative coverage may be provided by a third-generation cephalosporin (e.g., cefepime or ceftazidime) or an aminoglycoside agent (Table 83.14). A single dose of ceftazidime, 15 mg per kg IP, results in serum and dialysate concentrations above the MIC (for susceptible organisms) for more than 24 hours, because the serum elimination half-life is 22 hours.[291] Hence, ceftazidime can be dosed intermittently for both CAPD and APD patients as described above for cefazolin. Alternatively, the drug could be dosed continuously with the antibiotic added to each bag (500 mg per L loading dose followed by 125 mg per L in each subsequent bag). Although a retrospective study suggested long-term aminoglycoside therapy should be avoided when possible to preserve residual renal function a short course of empiric therapy appears to be safe.[292,293] Lui et al. randomized patients with peritonitis to either netilmicin or ceftazidime for 14 days. Both regimens were equally effective and associated with a transient loss of residual function. More importantly, netilmicin did not harm long-term residual renal function.[292,293] Quinolones may be used by centers with documented local sensitivities of gram-negative organisms to this class of drugs. A meta-analysis has confirmed that initial monotherapy with quinolones can be effective.[294] However, the included trials were old and, given the frequent emergence of quinolone resistance, quinolone monotherapy is not recommended.[295]

Subsequent therapy after antibiotic loading depends on the organism isolated. S. aureus or S. epidermidis may be treated with a first-generation cephalosporin alone, if methicillin-sensitive. Fifty percent or more of S. epidermidis causing peritonitis is resistant to cephalosporins.[296,297] These patients should be treated with vancomycin, as should patients with MRSA peritonitis.[298] MRSA peritonitis has a failure rate of 60% when treated with vancomycin alone and, frequently, results not only in catheter loss but also in peritoneal adhesions precluding further PD.[299] Therefore, rifampin should be added to vancomycin therapy. Peritonitis caused by vancomycin intermediate-resistant S. aureus has been reported and was successfully treated with rifampin and trimethoprim–sulfamethoxazole.[300]

Streptococcal or enterococcal peritonitis is best treated with ampicillin; an aminoglycoside may be added for synergy in enterococcal infections. Vancomycin-resistant enterococcus (VRE) as a cause of peritonitis in PD patients is still rare

but there are many published case reports.[301,302] Colonization had been considered rare although there is data that VRE may be increasing in dialysis units.[303] VRE should be treated with linezolid, quinupristin/dalfopristin, or daptomycin.

Infections due to S. aureus or enterococci require 3 weeks of therapy; 2 weeks is generally sufficient for other gram-positive cocci. For any peritonitis, failure to achieve clear dialysis effluent after 5 days of appropriate antibiotic therapy defines refractory peritonitis and is an indication for catheter removal.[195]

The subsequent therapy of gram-negative organisms is dependent on sensitivities. Aminoglycoside therapy should generally be reserved for those infections in which sensitivities dictate the use of these drugs. Once a day dosing of intraperitoneal aminoglycoside, shown to be effective, provides high local levels of the antibiotic, while avoiding systemic toxicity.[304]

P. aeruginosa peritonitis should always be treated with two drugs for a minimum of 3 weeks.[195] Peritonitis caused by P. aeruginosa is difficult to treat and can sometimes result in the death of the patient.[230,259,305–307] Aminoglycosides may be used if the isolate is sensitive to the drugs but long courses may result in vestibular toxicity. Ceftazidime, cefepime, piperacillin, or oral quinolones are generally effective. Antibiotic therapy is much more likely to be effective if a catheter infection is not present, although long courses of therapy may be required to prevent relapse. If a Pseudomonas catheter infection is present in conjunction with peritonitis, catheter removal is necessary.

Peritonitis owing to Stenotrophomonas maltophilia (formerly Xanthomonas maltophilia) is difficult to resolve as the organism displays very limited antimicrobial sensitivities. Despite treatment with multiple antibiotics, catheter removal may be necessary.[308,309] Immunosuppression is a risk factor.

Antimicrobial therapy of fungal peritonitis is not generally successful unless the catheter is removed. Amphotericin B has poor diffusion from blood into the peritoneum, whereas intraperitoneal administration results in chemical peritonitis.[281] Flucytosine, ketoconazole, and fluconazole diffuse readily from blood to the peritoneum and are more effective than amphotericin, although catheter removal still is often necessary.[272,310,311] Fluconazole is particularly well tolerated when administered intraperitoneally. Chan and colleagues found a cure rate of 9.5% using fluconazole therapy alone without catheter removal.[312] Fluconazole plus catheter removal cured 67%, whereas 14% required addition of amphotericin. The ISPD recommends prompt catheter removal for fungal peritonitis.

Temporary cessation of PD, which improves peritoneal immune function, has been successfully utilized to assist in resolving peritonitis, in conjunction with antibiotics.[313,314] This approach has been useful in recurrent peritonitis episodes owing to coagulase-negative staphylococcus, but has also been helpful in resolving refractory S. aureus.[315,316] It is effective only if catheter infection is absent.

Catheter removal is necessary to resolve the infection in some cases. Peritonitis owing to S. aureus, P. aeruginosa, or enteric peritonitis with an intra-abdominal source often requires catheter removal.[195] Catheter-related peritonitis accounts for approximately one third of the catheters removed, although the proportion and rate of catheter removal for isolated peritonitis have decreased with use of improved connection systems.[184,228] Recent data shows that persistence of peritoneal white blood cell (WBC) count $\geq$1090 cells per mm^3 after 3 days of therapy portends treatment failure and catheter removal should be considered.[317]

Simultaneous catheter removal and replacement are quite successful for recurring peritonitis and tunnel infections.[318,319] This eliminates an interim period on hemodialysis. This approach should be used only when the effluent leukocyte cell count is under 100 per μL. This approach is not recommended for fungal, mycobacterial, or P. aeruginosa peritonitis, or when peritonitis is a consequence of intra-abdominal pathology—these episodes require that the patient spend a period of time off PD.

83.14 Antibiotic Doses for Intermittent Therapy for Peritonitis

Antibiotic	Dose intraperitoneally
Cefazolin or cephalothin	15–20[a] mg/kg once daily
Vancomycin	30 mg/kg once, then 15 mg/kg every 5 d
Ceftazidime	15–20[a] mg/kg once daily
Gentamicin, tobramycin, or netilmicin	0.6 mg/kg once daily

[a]Higher dose for patients with residual renal function.
Modified from Piraino B, Bailie GR, Bernardini J, et al. Peritoneal dialysis related infections recommendations: 2005 update. Perit Dial Int. 2005;25:107.

OTHER COMPLICATIONS

Pancreatitis

Pancreatic abnormalities including pancreatitis occur with a higher frequency in uremic patients.[320] The highest incidence of pancreatitis among ESRD patients is in transplant recipients, but within dialysis populations, it is not clear that PD patients have a higher incidence than do HD patients.[321–323] Reports from the mid-1980s suggested a greater incidence in PD due to higher uremic solute concentrations in PD patients or even to the potentially direct toxic effects of dialysate, which bathes a portion of the pancreas. The dialysate dextrose concentration, hypertonicity, hypercalcemia, foreign particulate debris, bacteria, or antibiotics may induce inflammation in the sensitive pancreas.[322,324] That the direct toxicity of dialysate may be causative is supported by the recurrence of pancreatitis after reinstitution of PD after initial resolution.[325] The relevance of those reports from 1980s to the contemporary practice of peritoneal dialysis, however, is unclear and there is no convincing evidence for a higher incidence of pancreatitis in PD than HD today. Hyperlipidemia is both a risk factor for and complication of pancreatitis. The hyperlipidemia seen more frequently in PD patients is low-density lipoprotein hypercholesterolemia, which is not particularly toxic to the pancreas. On the other hand, HD patients are more likely to suffer from hypertriglyceridemia, which is a predisposing factor for pancreatitis when severe enough to be associated with hyperchylomicronemia. The high intake of simple carbohydrates in PD patients may be a factor in inducing hyperlipidemia.

Even though pancreatitis is an infrequent complication of PD, older reports suggested a high mortality rate.[320] The typical clinical presentation for acute pancreatitis in a PD patient is characterized by abdominal pain with normal bowel sounds, nausea, vomiting, absence of fever, hyperamylasemia (more than three times normal), elevated effluent dialysate amylase concentration (more than 100 U per L), and a variable appearance of effluent dialysate, including being clear, hemorrhagic, tea-colored, or even cloudy.[320–322] Amylase levels may be spuriously decreased in patients using icodextrin.

Hyperlipidemia and/or hypercalcemia are frequently present and may be predisposing metabolic abnormalities. Pancreatitis should be strongly considered if appropriately treated "peritonitis" does not resolve because this presentation is quite similar to PD-associated microbial peritonitis. The effluent in pancreatitis is usually sterile, even if hemorrhagic, cloudy, or tea-colored. Burkart and associates have suggested that dialysate effluent amylase concentration is low in bacterial peritonitis, even if slow to resolve, whereas it is more than 100 U per L with pancreatitis or other intra-abdominal pathologies.[265] If a diagnosis of pancreatitis is uncertain, CT is the preferred imaging study. In addition to demonstration of an engorged pancreas, CT may be particularly useful to identify the ominous finding of a pseudocyst.

The principles of management do not differ from those in patients without ESRD. Offending agents should be discontinued and, if that includes dialysate, PD should be halted. However, peritoneal lavage can be helpful in removing inflammatory mediators, especially if the dialysate was not the culprit. There is no evidence to support a recommendation to halt PD in all patients with acute pancreatitis, and discontinuing PD probably does not alter the prognosis.[326] Percutaneous pseudocyst drainage may be preferable to internal (jejunal) drainage and this may preclude continuation of PD. Hyperlipidemia and hypercalcemia should be corrected. The role of lower concentrations of calcium in dialysate is unknown.

Chyloperitoneum

There have been a few scattered case reports of chylous fluid leaking into the peritoneum and draining with effluent dialysate. This topic was recently reviewed.[327] The dialysate is cloudy but, on more careful examination, looks milky, reflecting the lipid rich content of chyle. The most common cause is trauma to intraperitoneal lymph vessels, either catheter-induced or from external trauma. Rocklin and Teitelbaum have recently reported a case of chyloperitoneum due to the superior vena cava syndrome.[328] Certain dihydropyridine calcium channel blockers have also been associated with chyloperitoneum, perhaps due to impaired lymphatic peristalsis.[329] Patients are usually asymptomatic. Treatment initially is conservative, to decrease abdominal lymph production by a low-fat, high-calorie diet supplemented with medium-chain triglycerides. The next step is discontinuation of PD, because the presence of dialysate may retard closure of the leak. If this is unsuccessful, a trial of total parenteral alimentation may be considered. Should these steps fail to resolve the leak, catheter removal is indicated. Lymphangiography may identify the source of the leak should surgery be considered.

Hemoperitoneum

As little as 1 mL of blood in 2 L of dialysate results in readily evident visual hemoperitoneum, and 7 mL results in effluent dialysate that looks like red fruit juice. Fortunately, this is an uncommon occurrence, but, when it does occur, it is often very frightening to the patient.[330] Fortunately, however, hemoperitoneum is almost always benign.[330,331] In Table 83.15 are listed causes of hemoperitoneum in PD patients. Hemoperitoneum occurs in 3.8% to 10% of PD patients and is twice as common in women as in men. When it occurs in women of childbearing age, 64% of the causes are related to ovulation or menses. In one series, this population experienced an almost 90% incidence rate.[332] There does not appear to be a correlation with PD-associated peritonitis, nor does hemoperitoneum adversely impact long-term outcomes on PD.

Menstrual and surgical histories are informative (Table 83.15). If the patient is asymptomatic and the bleeding stops spontaneously, no evaluation is absolutely necessary.

83.15 Causes of Hemoperitoneum in Peritoneal Dialysis Patients

Retrograde menstruation
Ovulation
Catheter-induced trauma (omental abrasion, repositioning, constipation)
Bowel disease (ischemic, inflammatory)
Peritonitis
Cysts (ovarian, polycystic kidney, acquired cystic kidney)
Abdominal trauma
Strenuous exercise (including sexual activity)
Systemic bleeding (thrombocytopenia, anticoagulants)
Hypertonic exchanges (hyperemia)
Pancreatitis
Vasculitis (systemic lupus erythematosus)
Sclerosing peritonitis
Adhesions
Granulosa cell tumor
Ectopic pregnancy
Cholecystitis
Colonoscopy
Dissection from adjacent sites (femoral hematoma, spleen, colon)
Previous hepatitis
Enema
Extracorporeal lithotripsy
Splenic infarction

In the absence of active menses, bloody dialysate should be evaluated by effluent cell count and differential, and only if clinically appropriate, Gram stain and culture, and effluent fluid amylase concentration. An abdominal ultrasound may occasionally be informative. Obviously, symptoms referable to the abdomen prompt further evaluation, which ultimately could include a laparotomy. Treatment is directed at the specific cause. However, because patients often are asymptomatic, precluding an extensive evaluation, treatment generally is supportive. Heparin administered intraperitoneally may protect from subsequent catheter occlusion from clots. Three rapid flushes with room-temperature dialysate may induce peritoneal vasoconstriction and stop the bleeding.[333] Dialysate that is significantly cooler than room temperature could precipitate cardiac dysrhythmias. Furthermore, cool dialysate should be avoided where mesenteric perfusion is compromised, because it could exacerbate ischemia of the bowel. This therapy is probably only effective in cases where the bleeding is secondary to a peritoneal membrane bleed. Gynecologic hormone therapy may be indicated in women who demonstrate hemoperitoneum during menses or ovulation.[334]

Defects of the Peritoneal Cavity Boundary

Hernias and Genital and Abdominal Wall Edema

Intra-abdominal pressure rises with increasing intraperitoneal volume, sitting, straining at stool, coughing, and strenuous physical activity. Combined with the extremes of age, debilitation, and poor wound healing from uremia, it is no surprise that hernias are common. A recent study documented a hernia prevalence near 20%.[335] Over 13% of the hernias present are strangulated. Teitelbaum and colleagues reported the largest series of patients with defects of the peritoneal cavity boundary. The overall frequency of hernia in this population of nearly 1900 patients was 6.7%. The most common sites were: inguinal, 25% of total; umbilical, 19%, and ventral, 14.[336] They found hernias to be more common in men than women, although another study demonstrated the converse.[337] Patients with cystic disease as the etiology of ESRD are at higher risk for the development of hernias.[336,338] One-half of the hernias become clinically evident within the first year of PD,[339] but many probably go undetected unless special scintigraphic studies are performed.[340] Most of the scintigraphically diagnosed asymptomatic cases never progress to clinically appreciable disease. Many PD patients have hernias diagnosed prior to initiating PD and herniorrhaphies are performed at the time of catheter insertion. Bargman and colleagues recently documented the outcomes of 50 patients undergoing hernia repair. PD was stopped for 48 hours perioperatively followed by gradual increase in dialysate volume. No hernia recurrences were noted and patients did not require temporary hemodialysis.[341] Other centers have also shown that discontinuation of PD is usually not necessary after hernia surgery.[342,343]

There probably is no benefit from routine screening scintigraphy in adults because clinical manifestations alone dictate the need to repair. Increased intraperitoneal pressure alone is not sufficient to cause hernias—a preexisting anatomic abnormality is often present.[344] In children, some programs routinely perform intraoperative peritoneograms (and herniorrhaphies if positive) at the time of catheter placement. To ensure prompt strength postoperatively, especially for large hernias, supporting prosthetic overlay mesh is inserted at the time of herniorrhaphy.[345] Placement of catheters through the rectus muscle in a paramedian approach probably reduces the incidence of subsequent incisional or catheter site hernias. Postinsertion leakage increases the likelihood of subsequent hernias.

Abdominal wall edema or genital edema is caused by either dialysate leakage through acquired peritoneal defects, such as at the catheter insertion site, traumatic rents such as previous hernias or incisions, or congenital defects that go undetected until PD raises intraperitoneal pressures, opening them (patent processus vaginalis). Thus, the fluid could dissect between tissue layers or through natural pathways. In the study by Teitelbaum and colleagues pericatheter or subcutaneous leaks were present in 3% of the total population.[336] Edema of the scrotum or perineal area is usually owing to a patent processus vaginalis. Scrotal edema may develop in up

to 10% of men on CAPD.[346] This can be managed temporarily by supine PD, but surgical correction is generally required, certainly if a hernia is present. Postoperative management may include hemodialysis for 1 week or more.[347] Vaginal leakage of dialysate is rare but serious, because it can lead to recurrent peritonitis, often with fungus. This complication should be suspected in any woman with watery vaginal discharge that is positive for glucose. If the leak is through the fallopian tubes, then tubal ligation is corrective.[348]

The site of a subcutaneous dialysate leak can be located with scintigraphy, ultrasonography, or contrast imaging.[349,350] Surgical closure is recommended, hence the need for precise identification of the leak site. Although watchful waiting is tempting, the collective PD experience suggests that elective operative intervention is the best approach to these complications related to increased abdominal pressure.

Hydrothorax

Fluid migrates from the peritoneal to the pleural space in 0.6% to 5% of patients undergoing PD either via transdiaphragmatic lymphatics or defects in the tendinous portion of the diaphragm.[351] There is an increased incidence in women, patients with polycystic kidney disease or hernias, those prone to peritonitis, and children.[352] Right sided pleural effusions appear to be more common than left.[353] The heart or pericardium probably protects the tendinous portion of the left hemidiaphragm. The hydrothorax can occur abruptly and painfully following exercise or trauma and can be immediately life threatening. A more common presentation is that of gradual progression of orthopnea or dyspnea, usually without pain. One half of the cases present within the first month of PD, and only one fifth present 1 year or more after initiation.[353] Resolution (i.e., being able to continue PD) is more likely in those cases where the presentation is within 1 year of initiating PD.

The simultaneous measurement of the concentrations of albumin, glucose, and lactate dehydrogenase in peritoneal effluent, pleural fluid, and blood may be helpful diagnostically. Peritoneal scintigraphy with radiolabeled albumin is a useful diagnostic maneuver; methylene blue should be avoided because of the pain it causes. Although helpful in localizing the defect, these diagnostic maneuvers probably do not alter management.[351] If the origin of the hydrothorax is dialysate, therapy is indicated regardless of whether there is a distinct leak versus lymphatic transport.

Initial attempts at conservative management should be made by decreasing volumes (decrease fill, decrease UF), performing PD supine, and periods of an empty abdomen. If conservative management fails, video-assisted thoracoscopic surgery is the preferred therapeutic modality. Chemical pleurodesis with tetracycline, blood, N-CWS (Nocardia rubra cell wall skeleton), triamcinolone, OK-432, talc, or fibrin adhesive have each been successful; however, these procedures can be very painful and are associated with unpredictable results.[351,353a]

Hyperlipidemia

Compared to HD patients, PD patients demonstrate higher concentrations of total cholesterol, triglycerides, Lp(a), apo A-I, and apo B as well as lower apo A-I: B ratios, and high-density lipoprotein (HDL) cholesterol concentrations.[354] The cause of these abnormalities is multifactorial. Although total caloric intake is equal in PD and HD patients because of absorbed dextrose from peritoneal dialysate, oral caloric intake is actually less in PD patients.[355] This absorbed simple carbohydrate may account for 25% of total caloric intake. Patients who require frequent hypertonic exchanges do so because of increased fluid and/or food intake. Therefore, it is difficult to determine whether hyperlipidemia is secondary to diet or glucose-based dialysate. It should be noted, however, that use of icodextrin has now been shown to improve glucose control, total cholesterol, and LDL levels but not hypertriglyceridemia.[356,357] This supports a role for the caloric load from glucose in the pathogenesis of hyperlipidemia in PD patients. In addition to the above, there is loss into effluent dialysate of oncotic proteins (e.g., albumin) and liporegulatory molecules (e.g., HDL cholesterol, apoproteins).[358,359] This sets the stage for hyperlipidemia and atherosclerosis.

The treatment of hyperlipidemia in PD patients must include an attempt to decrease the use of the most hypertonic exchanges. This should be done in conjunction with dietary restriction of fluids, fats, and simple carbohydrates. Lipid-lowering drugs of several classes have been utilized successfully. The major U.S. experience with fibric acid derivatives is with gemfibrozil, which increases lipoprotein lipase activity, the catabolism of very-low-density lipoproteins (VLDL), and the concentration of HDL_2 and HDL_3.[360] The dose should be reduced by initiation with 300 mg once daily and titrated gradually upward. Gemfibrozil can cause myositis, which may be manifested by increased serum potassium and/or creatine kinase concentrations. Hydroxymethylglutaryl-CoA reductase inhibitors, predominantly used to treat hypercholesterolemia, are safe and effective in PD patients.[361] The recently concluded SHARP study included patients on PD and showed that both simvastatin and ezetimibe are safe in the PD population as well.[362]

Intra-abdominal Pathology in Peritoneal Dialysis Patients

Less than 6% of peritonitis episodes in PD patients are owing to intra-abdominal pathology (IAP) and, although polymicrobial peritonitis may be associated with IAP, most cases are not.[363] Peripheral leukocytosis, an increasing PD cell count on antibiotic therapy, or an expanding pneumoperitoneum are important clues to IAP.[183,363,364] Obvious signs of IAP, such as fecal or biliary material in the dialysate or diarrhea containing dialysate, are not commonly observed. Risk factors for the development of IAP include diverticulosis, constipation and its treatment, and unrepaired hernia.[262] Death from IAP is linked to bowel gangrene; malnutrition; comorbidities such as liver failure, shock, bacteremia, pneumonia,

and gastrointestinal or intracerebral hemorrhage; and delayed surgical intervention—therefore, by a broad consensus, early surgical intervention in suspected IAP is strongly recommended.[365,366]

In general, slowly resolving peritonitis warrants close follow-up. Clear dialysate while on antibiotics is not an absolute sign of a benign process. Generalized abdominal peritonitis can mask localized signs and symptoms of IAP. Surgical consultation is urgently needed in the following conditions:

- Localized abdominal pain and tenderness
- Dilated loops of bowel on abdominal radiograph
- Progressive increase in intraperitoneal free air with continued peritonitis
- Hemoperitoneum with measurable dialysate hematocrit

Those perioperative interventions that best allow continuation or quick return to PD postoperatively include:

1. Tight wound closure for prevention of dialysate leakage, possibly using nonresorbable sutures.
2. Drain removal before resuming PD to allow adequate dialysis.
3. Preoperative extensive PD to increase platelet function and allow a few days without PD postoperatively for healing.
4. Elective repair of abdominal wall hernias (see earlier Abdominal Hernias in Continuous Peritoneal Dialysis) both for patient comfort as well as prevention of bowel incarceration.
5. Avoidance of constipation, because impacted stool often accompanies diverticulitis or perforated bowel.
6. Optimization of nutrition to counter the marked protein loss through an inflamed peritoneum.
7. Avoidance of PD with transfer to HD if extensive bowel wall repairs are made. A low threshold for transition to HD is generally a prudent decision.
8. Omentectomy at surgery if the omentum appears threatening to catheter flow function.[367]

ENCAPSULATING PERITONEAL SCLEROSIS

Encapsulating peritoneal sclerosis (EPS) is a potentially devastating complication of peritoneal dialysis. EPS is rare, affecting fewer than 5% of peritoneal dialysis patients and is sometimes diagnosed after renal transplantation.[368,369] Patients with EPS present with anorexia, nausea, vomiting, protein-energy wasting, and intestinal obstruction.[370] A thick-walled membrane "cocoon" is present, entrapping loops of bowel. This gives rise to the classic "sandwich" appearance on abdominal ultrasonography.[371]

The etiology and pathogenesis of EPS are uncertain although time on dialysis is a risk factor.[372] Peritoneal irritants, recurrent peritonitis, long-term use of PD, acetate-containing dialysis, chlorhexidine, beta-blockers, and high transporter status have all been implicated in the pathogenesis.[373] Historically, mortality was severe—as high as 60% within 4 months of diagnosis. However, contemporary data from Australia and New Zealand has demonstrated a considerably lower mortality and suggests that many of the deaths may not be related to EPS.[374]

Treatment consists of corticosteroids, supportive care with parenteral nutrition, and, in extreme cases, surgical enterolysis. ACEI and tamoxifen are considered potential treatments for EPS due to antifibrotic properties but high quality clinical data is lacking.

INTRAPERITONEAL INSULIN

Shortly after the advent of CAPD it was suggested that the intraperitoneal administration of insulin could improve glycemic control.[375] Although easily utilized in CAPD, the use of intraperitoneal insulin in patients performing APD, an increasing segment of the overall PD population, is more complex. Coupled with the trend toward use of longer-acting insulin preparations, this has resulted in a substantial decrease in the utilization of this route for insulin delivery. Furthermore, intraperitoneal insulin has been linked to subcapsular hepatic steatosis.[376]

HYPERTENSION

Volume control and sodium removal by PD are related to numerous factors, including dialysate composition (osmolality created by dextrose and sodium concentrations), peritoneal permeability and UF capacity, splanchnic circulation, and residual renal function.[377] After many months of PD, the antihypertensive effect of PD may be due to other factors as well.[378]At this time, body weight may actually increase, although this could reflect the increased caloric intake from the transperitoneal absorption of dextrose. Because the peritoneal membrane is associated with different transport properties than HD membranes, the more efficient removal of pressor substances by PD is speculated to play a role in this late hypertension control.[378] These pressor compounds could include Na-K–ATPase inhibitors, norepinephrine, and endothelin. However, after a year or more of PD, hypertension is less effectively controlled than after PD initiation.[109] This may be related to the development of peritoneal sclerosis, progressive obesity, dialysis prescription nonadherence, improved appetite and well-being and dietary indiscretion, increased hematocrit, loss of residual renal function, or other as yet unidentified factors.

Recent studies have focused attention on the differences in blood pressure between patients performing CAPD or APD. Some single-center studies have reported that patients performing APD have higher blood pressure and left ventricular mass than do those on CAPD.[379–381] This is likely due to decreased sodium removal and ultrafiltration in APD patients due to the shortened dwell times and

high transport, respectively.[379] However, careful attention to APD prescription—limiting the number of cycles to 3 to 5 at night, and avoiding long dwells with glucose-based solutions by leaving the abdomen dry for part of the day, or use of a day exchange, or use of icodextrin—allows for equivalent control of blood pressure and hypervolemia as can be achieved with CAPD.[382,383]

TRANSPLANTATION

Peritoneal dialysis patients may differ from their HD counterparts in several aspects that could influence transplant outcomes. Compared to HD patients, PD patients demonstrate a more normal immune response as characterized by T4:T8 lymphocyte ratios, T cell counts, T cell stimulation, and cell-mediated immunity.[384] However, many observational studies have shown that both the incidence of delayed graft function and long-term transplant outcomes in patients who performed PD prior to transplantation are equivalent—if not superior to—those obtained in patients who performed HD.[385–389] A higher incidence of vascular graft thrombosis after performance of PD has been reported but this remains controversial.[387]

Another difference between PD and HD patients potentially influencing transplantation is that the control of anemia, with or without erythropoietin, is easier with PD.[390] Thus, PD patients are less likely to experience blood transfusions and subsequent enhanced graft tolerance. Furthermore, the decreased transfusion requirement of PD patients makes hepatitis less likely, which is important considering the adverse effects of viral hepatitis on graft survival and the potential need for antiviral therapy prior to transplantation.

When compared to HD patients, PD patients have better blood pressure control and preserved residual renal function, which may affect care in the immediate posttransplant period. Patients receiving intraperitoneal insulin must be converted back to subcutaneous insulin once PD is terminated.

Because of a low frequency of delayed graft function, and because of the location of a pancreatic allograft in adults or the renal allograft in children, it has become common to remove PD catheters at the time of transplantation. If desired, the PD catheter may be kept in place for up to 2 to 3 months after transplantation. In that case, frequent flushing is recommended to maintain catheter patency and to avoid unlubricated or unbuffered bowel contact which, especially in the patient on steroids, may result in abscess formation and potentially even erosion of the catheter through the bowel wall.

A tunnel infection or active peritonitis generally precludes transplantation at that time. A prudent policy is to observe the course of the peritonitis for at least 2 weeks following the discontinuation of antibiotics. If no relapse has occurred, the patient is then reactivated on the recipient list.

There is no difference in the frequency or types of nonperitonitis-related posttransplant infections in recipients previously dialyzed by PD or HD.[391,392] If the PD catheter has been left in place and peritonitis develops, its course is not different from that seen in PD patients who are not receiving immunosuppressive medications. It requires essentially the same treatment with parenteral or intraperitoneal antibiotics, with the exception that allograft function may necessitate larger or more frequent doses. Posttransplant exit site or tunnel infections probably warrant catheter removal, especially if the infection is in proximity to the graft incision.

Posttransplant ascites may develop in PD patients, even with functioning grafts.[393] It is probably related to a hyperemic peritoneum whose mesothelium has been altered by the previous presence of dialysate. It may take weeks, but this does subside spontaneously. The ascites should be drained only when dictated by patient comfort because the protein content is generally high and negative protein balance may ensue.

REFERENCES

1. Wang AY, Wang M, Woo J, et al. A novel association between residual renal function and left ventricular hypertrophy in peritoneal dialysis patients. Kidney Int. 2002;62:639–647. http://www.ncbi.nlm.nih.gov/pubmed/12110029
2. Misra M, Nolph KD, Khanna R, et al. Retrospective evaluation of renal kt/V(urea) at the initiation of long-term peritoneal dialysis at the University of Missouri: relationships to longitudinal nutritional status on peritoneal dialysis. ASAIO J. 2003;49:91–102. http://www.ncbi.nlm.nih.gov/pubmed/12558314
3. Stompor T, Sulowicz W, Anyszek T, et al. Dialysis adequacy, residual renal function and serum concentrations of selected low molecular weight proteins in patients undergoing continuous ambulatory peritoneal dialysis. Med Sci Monit, 2003;9:CR500–504,
4. Rottembourg J, Issad B, Gallego JL, et al. Evolution of residual renal function in patients undergoing maintenance haemodialysis or continuous ambulatory peritoneal dialysis. Proc Eur Dial Transplant Assoc, 1983;19:397–403. http://www.ncbi.nlm.nih.gov/pubmed/6878254
5. Cancarini GC, Brunori G, Camerini C, et al. Renal Function Recovery and Maintenance of Residual Diuresis in CAPD and Hemodialysis. Perit Dial Bull, 1986;6:77–79.
6. Lysaght MJ, Vonesh EF, Gotch F, et al. The influence of dialysis treatment modality on the decline of remaining renal function. ASAIO transactions / American Society for Artificial Internal Organs, 1991;37:598–604. http://www.ncbi.nlm.nih.gov/pubmed/1768496
7. Lang SM, Bergner A, Topfer M, et al. Preservation of residual renal function in dialysis patients: effects of dialysis-technique-related factors. Peritoneal dialysis international : journal of the International Society for Peritoneal Dialysis, 2001;21:52–57.
8. Jansen MA, Hart AA, Korevaar JC, et al. Predictors of the rate of decline of residual renal function in incident dialysis patients. Kidney Int. 2002;62:1046–1053. http://www.ncbi.nlm.nih.gov/pubmed/12164889
9. McKane W, Chandna SM, Tattersall JE, et al. Identical decline of residual renal function in high-flux biocompatible hemodialysis and CAPD. Kidney Int. 2002;61:256–265. http://www.ncbi.nlm.nih.gov/pubmed/11786108
10. Vonesh EF, et al. The differential impact of risk factors on mortality in hemodialysis and peritoneal dialysis. Kidney Int. 2004;66(6):2389–2401.
11. Huang CC, Cheng KF, Wu HD. Survival analysis: comparing peritoneal dialysis and hemodialysis in Taiwan. Perit Dial Int. 2008;28 Suppl 3:S15–20.
12. Sanabria M, et al. Dialysis outcomes in Colombia (DOC) study: a comparison of patient survival on peritoneal dialysis vs hemodialysis in Colombia. Kidney Int Suppl. 2008(108):S165–172.
13. Woywodt A, et al. In-center intermittent peritoneal dialysis: retrospective ten-year single-center experience with thirty consecutive patients. Perit Dial Int. 2008;28(5):518–526. http://www.ncbi.nlm.nih.gov/pubmed/18708546
14. Twardowski ZJ, et al. Chronic nightly tidal peritoneal dialysis. ASAIO Trans. 1990;36(3):M584–588.

15. Steinhauer HB, et al. Increased dialysis eff ciency in tidal peritoneal dialysis compared to intermittent peritoneal dialysis. Nephron. 1991;58(4):500–501. http://www.ncbi.nlm.nih.gov/pubmed/1922626

16. Vychytil A, et al. Tidal peritoneal dialysis for home-treated patients: should it be preferred? Am J Kidney Dis. 1999;33(2):334–343. http://www.ncbi.nlm.nih.gov/pubmed/10023647

17. Juergensen PH, et al. Tidal peritoneal dialysis: comparison of different tidal regimens and automated peritoneal dialysis. Kidney Int. 2000;57(6):2603–2607. http://www.ncbi.nlm.nih.gov/pubmed/10844630

18. Aasarod K, Wideroe TE, Flakne SC. A comparison of solute clearance and ultraf ltration volume in peritoneal dialysis with total or fractional (50%) intraperitoneal volume exchange with the same dialysate flow rate. Nephrol Dial Transplant. 1997;12(10):2128–2132. http://www.ncbi.nlm.nih.gov/pubmed/9351077

19. Juergensen PH, et al. Tidal peritoneal dialysis to achieve comfort in chronic peritoneal dialysis patients. Adv Perit Dial. 1999;15:125–126. http://www.ncbi.nlm.nih.gov/pubmed/10682086

20. Morbidity & mortality. Am J Kid Dis. 2011;57(1, Supplement 1):e77-e86.

21. Keshaviah P. Adequacy of CAPD: a quantitative approach. Kidney Int Suppl. 1992;38:S160–164.

22. Lowrie EG, et al. Effect of the hemodialysis prescription of patient morbidity: report from the National Cooperative Dialysis Study. N Engl J Med. 1981;305(20):1176–1181.

23. Gotch FA, Sargent JA. A mechanistic analysis of the National Cooperative Dialysis Study (NCDS). Kidney Int. 1985;28(3):526–534. http://www.ncbi.nlm.nih.gov/pubmed/3934452

24. Kim DJ, et al. Dissociation between clearances of small and middle molecules in incremental peritoneal dialysis. Perit Dial Int. 2001;21(5):462–466. http://www.ncbi.nlm.nih.gov/pubmed/11757829

25. Jager KJ, et al. Mortality and technique failure in patients starting chronic peritoneal dialysis: results of The Netherlands Cooperative Study on the Adequacy of Dialysis. NECOSAD Study Group. Kidney Int. 1999;55(4): 1476–1485. http://www.ncbi.nlm.nih.gov/pubmed/10201013

26. Rocco M, et al. Peritoneal dialysis adequacy and risk of death. Kidney Int. 2000;58(1):446–457.

27. Shemin D, et al. Residual renal function in a large cohort of peritoneal dialysis patients: change over time, impact on mortality and nutrition. Perit Dial Int. 2000;20(4):439–444. http://www.ncbi.nlm.nih.gov/pubmed/11007376

28. Szeto CC, et al. Importance of dialysis adequacy in mortality and morbidity of chinese CAPD patients. Kidney Int. 2000;58(1):400–407. http://www.ncbi.nlm.nih.gov/pubmed/10886588

29. Bargman JM, Thorpe KE, Churchill DN. Relative contribution of residual renal function and peritoneal clearance to adequacy of dialysis: a reanalysis of the CANUSA study. J Am Soc Nephrol. 2001;12(10):2158–2162. http://www.ncbi.nlm.nih.gov/pubmed/11562415

30. Szeto CC, et al. Impact of dialysis adequacy on the mortality and morbidity of anuric Chinese patients receiving continuous ambulatory peritoneal dialysis. J Am Soc Nephrol. 2001;12(2):355–360. http://www.ncbi.nlm.nih.gov/pubmed/11158226

31. Brown EA, et al. Survival of functionally anuric patients on automated peritoneal dialysis: the European APD Outcome Study. J Am Soc Nephrol. 2003;14(11):2948–2957. http://www.ncbi.nlm.nih.gov/pubmed/14569106

32. Szeto CC, et al. Independent effects of renal and peritoneal clearances on the mortality of peritoneal dialysis patients. Perit Dial Int. 2004;24(1):58–64. http://www.ncbi.nlm.nih.gov/pubmed/15104337

33. Jansen MA, et al. Predictors of survival in anuric peritoneal dialysis patients. Kidney Int. 2005;68(3):1199–1205.

34. Lo WK, et al. Minimal and optimal peritoneal Kt/V targets: results of an anuric peritoneal dialysis patient's survival analysis. Kidney Int. 2005;67(5): 2032–2038.

35. Fried L, et al. Association of Kt/V and creatinine clearance with outcomes in anuric peritoneal dialysis patients. Am J Kidney Dis. 2008;52(6):1122–1130.

36. Paniagua R, et al. Effects of increased peritoneal clearances on mortality rates in peritoneal dialysis: ADEMEX, a prospective, randomized, controlled trial. J Am Soc Nephrol. 2002;13(5):1307–1320.

37. Lo WK, et al. Effect of Kt/V on survival and clinical outcome in CAPD patients in a randomized prospective study. Kidney Int. 2003;64(2):649–656. http://www.ncbi.nlm.nih.gov/pubmed/12846762

38. Mak SK, et al. Randomized prospective study of the effect of increased dialytic dose on nutritional and clinical outcome in continuous ambulatory peritoneal dialysis patients. Am J Kidney Dis. 2000;36(1):105–114. http://www.ncbi.nlm.nih.gov/pubmed/10873879

39. Moreno F, et al. Quality of life in dialysis patients. A spanish multicentre study. Spanish Cooperative Renal Patients Quality of Life Study Group. Nephrol Dial Transplant. 1996;11 Suppl 2:125–129. http://www.ncbi.nlm.nih.gov/pubmed/8804012

40. Merkus MP, et al. Quality of life in patients on chronic dialysis: self-assessment 3 months after the start of treatment. The Necosad Study Group. Am J Kidney Dis. 1997;29(4):584–592. http://www.ncbi.nlm.nih.gov/pubmed/9100049

41. Paniagua R, et al. Health-related quality of life predicts outcomes but is not affected by peritoneal clearance: The ADEMEX trial. Kidney Int. 2005;67(3): 1093–1104.

42. Pollock CA, et al. Total-body nitrogen by neutron activation in maintenance dialysis. Am J Kidney Dis. 1990;16(1):38–45. http://www.ncbi.nlm.nih.gov/pubmed/2368704

43. Heimburger O, et al. Hand-grip muscle strength, lean body mass, and plasma proteins as markers of nutritional status in patients with chronic renal failure close to start of dialysis therapy. Am J Kidney Dis. 2000;36(6):1213–1225. http://www.ncbi.nlm.nih.gov/pubmed/11096047

44. Jacob V, et al. Nutritional prof le of continuous ambulatory peritoneal dialysis patients. Nephron. 1995;71(1):16–22.

45. Wang AY, et al. Evaluation of handgrip strength as a nutritional marker and prognostic indicator in peritoneal dialysis patients. Am J Clin Nutr. 2005; 81(1):79–86.

46. Sreedhara R, et al. Prealbumin is the best nutritional predictor of survival in hemodialysis and peritoneal dialysis. Am J Kidney Dis. 1996;28(6):937–942.

47. Avram MM, et al. Markers for survival in dialysis: a seven-year prospective study. Am J Kidney Dis. 1995;26(1):209–219. http://www.ncbi.nlm.nih.gov/pubmed/7611254

48. Blake PG, et al. Serum albumin in patients on continuous ambulatory peritoneal dialysis—predictors and correlations with outcomes. J Am Soc Nephrol. 1993;3(8):1501–1507. http://www.ncbi.nlm.nih.gov/pubmed/8490120

49. Chung SH, Lindholm B, Lee HB. Influence of initial nutritional status on continuous ambulatory peritoneal dialysis patient survival. Perit Dial Int. 2000;20(1):19–26. http://www.ncbi.nlm.nih.gov/pubmed/10716579

50. Mehrotra R, Kopple JD. Nutritional management of maintenance dialysis patients: why aren't we doing better? Annu Rev Nutr. 2001;21:343–379.

51. Harty J, et al. The influence of small solute clearance on dietary protein intake in continuous ambulatory peritoneal dialysis patients: a methodologic analysis based on cross-sectional and prospective studies. Am J Kidney Dis. 1996;28(4):553–560.

52. Uehlinger DE. Another look at the relationship between protein intake and dialysis dose. J Am Soc Nephrol. 1996;7(1):166–168. http://www.ncbi.nlm.nih.gov/pubmed/8808125

53. Wang AY, et al. Independent effects of residual renal function and dialysis adequacy on actual dietary protein, calorie, and other nutrient intake in patients on continuous ambulatory peritoneal dialysis. J Am Soc Nephrol. 2001;12(11):2450–2457. http://www.ncbi.nlm.nih.gov/pubmed/11675422

54. Bergstrom J, et al. Protein and energy intake, nitrogen balance and nitrogen losses in patients treated with continuous ambulatory peritoneal dialysis. Kidney Int. 1993;44(5):1048–1057. http://www.ncbi.nlm.nih.gov/pubmed/8264134

55. Davies SJ, et al. Analysis of the effects of increasing delivered dialysis treatment to malnourished peritoneal dialysis patients. Kidney Int. 2000;57(4): 743–1754.

56. Nancy G, Steven F, Robert IL. The effect of improved dialytic eff ciency on measures of appetite in peritoneal dialysis patients. J Renal Nutr. 1996;6(4): 217–221.

57. Anderstam B, et al. Middle-sized molecule fractions isolated from ure-mic ultraf ltrate and normal urine inhibit ingestive behavior in the rat. J Am Soc Nephrol. 1996;7(11):2453–2460.

58. Peritoneal Dialysis Adequacy Work Group. Clinical practice guidelines for peritoneal dialysis adequacy. Am J Kidney Dis. 2006;48 Suppl 1:S98–129.

59. Dombros N, et al. European best practice guidelines for peritoneal dialysis. Adequacy of peritoneal dialysis. Nephrol Dial Transplant. 2005;20 Suppl 9:ix24–ix27.

60. Blake PG, et al. Clinical practice guidelines and recommendations on peritoneal dialysis adequacy 2011. Perit Dial Int. 2011;31(2):218–239. http://www.ncbi.nlm.nih.gov/pubmed/21427259

61. Lo WK, et al. Guideline on targets for solute and fluid removal in adult patients on chronic peritoneal dialysis. Perit Dial Int. 2006;26(5):520–522. http://www.ncbi.nlm.nih.gov/pubmed/16973505

62. Johnson D, et al. The CARI guidelines. Dialysis adequacy (PD) guidelines. Nephrology (Carlton). 2005;10 Suppl 4:S81–107.

63. Adequacy of dialysis and nutrition in continuous peritoneal dialysis: association with clinical outcomes. Canada-USA (CANUSA) Peritoneal Dialysis Study Group. J Am Soc Nephrol. 1996;7(2):198–207.
http://www.ncbi.nlm.nih.gov/pubmed/8785388

64. Van Biesen W, et al. Personal dialysis capacity (PDC(TM)) test: a multicentre clinical study. Nephrol Dial Transplant. 2003;18(4):788–796.

65. Twardowski ZJ, et al. Peritoneal equilibration test. Perit Dial Int. 1987;7(3):138–148.

66. Fischbach M, et al. Determination of individual ultrafiltration time (APEX) and purification phosphate time by peritoneal equilibration test: application to individual peritoneal dialysis modality prescription in children. Perit Dial Int. 1996;16 Suppl 1:S557–560.

67. Pannekeet MM, et al. The standard peritoneal permeability analysis: a tool for the assessment of peritoneal permeability characteristics in CAPD patients. Kidney Int. 1995;48(3):866–875.
http://www.ncbi.nlm.nih.gov/pubmed/7474677

68. Rocco MV, Jordan JR, Burkart JM. Determination of peritoneal transport characteristics with 24–hour dialysate collections: dialysis adequacy and transport test. J Am Soc Nephrol. 1994;5(6):1333–1338.
http://www.ncbi.nlm.nih.gov/pubmed/7893998

69. Krediet RT, et al. Simple assessment of the efficacy of peritoneal transport in continuous ambulatory peritoneal dialysis patients. Blood Purif. 1986;4(4):194–203.

70. Vonesh EF, et al. Kinetic modeling as a prescription aid in peritoneal dialysis. Blood Purif. 1991;9(5–6):246–270.
http://www.ncbi.nlm.nih.gov/pubmed/1819315

71. Johnson DW, et al. A comparison of peritoneal equilibration tests performed 1 and 4 weeks after PD commencement. Perit Dial Int. 2004;24(5):460–465.
http://www.ncbi.nlm.nih.gov/pubmed/15490986

72. Mujais S, Vonesh E. Profiling of peritoneal ultrafiltration. Kidney Int Suppl. 2002(81):S17–22.

73. Twardowski ZJ, et al. Short peritoneal equilibration test: impact of preceding dwell time. Adv Perit Dial. 2003;19:53–58.
http://www.ncbi.nlm.nih.gov/pubmed/14763034

74. Figueiredo AE, Conti A, Poli de Figueiredo CE. Influence of the preceding exchange on peritoneal equilibration test results. Adv Perit Dial. 2002; 18:75–77.
http://www.ncbi.nlm.nih.gov/pubmed/12402592

75. Mujais S, et al. Evaluation and management of ultrafiltration problems in peritoneal dialysis. International Society for Peritoneal Dialysis Ad Hoc Committee on Ultrafiltration Management in Peritoneal Dialysis. Perit Dial Int. 2000;20 Suppl 4:S5–21.

76. Pride ET, et al. Comparison of a 2.5% and a 4.25% dextrose peritoneal equilibration test. Perit Dial Int. 2002;22(3):365–370.
http://www.ncbi.nlm.nih.gov/pubmed/12227395

77. Twardowski ZJ. Nightly peritoneal dialysis. Why, who, how, and when? ASAIO Trans. 1990;36(1):8–16.
http://www.ncbi.nlm.nih.gov/pubmed/2407280

78. Mehrotra R, et al. The outcomes of continuous ambulatory and automated peritoneal dialysis are similar. Kidney Int. 2009;76(1):97–107.
http://www.ncbi.nlm.nih.gov/pubmed/19340090

79. Badve SV, et al. Automated and continuous ambulatory peritoneal dialysis have similar outcomes. Kidney Int. 2008;73(4):480–488.

80. Churchill DN, et al. Increased peritoneal membrane transport is associated with decreased patient and technique survival for continuous peritoneal dialysis patients. The Canada-USA (CANUSA) Peritoneal Dialysis Study Group. J Am Soc Nephrol. 1998;9(7):1285–1292.
http://www.ncbi.nlm.nih.gov/pubmed/9644640

81. Rumpsfeld M, et al. Predictors of baseline peritoneal transport status in Australian and New Zealand peritoneal dialysis patients. Am J Kidney Dis. 2004;43(3):492–501.
http://www.ncbi.nlm.nih.gov/pubmed/14981608

82. Davenport A, Willicombe MK. Hydration status does not influence peritoneal equilibration test ultrafiltration volumes. Clin J Am Soc Nephrol. 2009;4(7):1207–1212.
http://www.ncbi.nlm.nih.gov/pubmed/19556380

83. Oh KH, et al. Baseline peritoneal solute transport rate is not associated with markers of systemic inflammation or comorbidity in incident Korean peritoneal dialysis patients. Nephrol Dial Transplant. 2008;23(7):2356–2364.

84. Burkart J, Jordan JR, Rocco M. Assessment of dialysis dose by measured clearance versus extrapolated data. Perit Dial Int. 1993;13(3):184–188.
http://www.ncbi.nlm.nih.gov/pubmed/8369346

85. Brimble KS, et al. Meta-analysis: peritoneal membrane transport, mortality, and technique failure in peritoneal dialysis. J Am Soc Nephrol. 2006;17(9):2591–2598.

86. Wiggins KJ, et al. High membrane transport status on peritoneal dialysis is not associated with reduced survival following transfer to haemodialysis. Nephrol Dial Transplant. 2007;22(10):3005–3012.

87. Watson PE, Watson ID, Batt RD. Total body water volumes for adult males and females estimated from simple anthropometric measurements. Am J Clin Nutr. 1980;33(1):27–39.
http://www.ncbi.nlm.nih.gov/pubmed/6986753

88. Hume R, Weyers E. Relationship between total body water and surface area in normal and obese subjects. J Clin Pathol. 1971;24(3):234–238.
http://www.ncbi.nlm.nih.gov/pubmed/5573437

89. Tzamaloukas AH, et al. The prescription of peritoneal dialysis. Semin Dial. 2008;21(3):250–257.
http://www.ncbi.nlm.nih.gov/pubmed/18248525

90. Borah MF, et al. Nitrogen balance during intermittent dialysis therapy of uremia. Kidney Int. 1978;14(5):491–500.

91. Randerson DC, Farrell PC. Amino acid and dietary status in long-term CAPD patients. In: Atkins RC, Thomson NM, Farrell PC, eds. Peritoneal Dialysis. Edinburgh: Churchill Livingstone; 1981.

92. Kjeldahl J. Neue methode zur bestimmung des stickoffs nin organischen Korpern. Z Anal Chem. 1983;22.

93. Keshaviah PR, Nolph KD. Protein catabolic rate calculations in CAPD patients. ASAIO Trans. 1991;37(3):M400–402.

94. Teehan BP, Schleifer CR, Sigler MH. A quantitative approach to the CAPD prescription. Perit Dial Bull. 1985;5:152–156.

95. Keshaviah PR, et al. Lean body mass estimation by creatinine kinetics. J Am Soc Nephrol. 1994;4(7):1475–1485.

96. Kotanko P, Levin NW, Zhu F. Current state of bioimpedance technologies in dialysis. Nephrol Dial Transplant. 2008;23(3):808–812.

97. Vonesh EF, Story KO, O'Neill WT. A multinational clinical validation study of PD ADEQUEST 2.0. PD ADEQUEST International Study Group. Perit Dial Int. 1999;19(6):556–571.

98. Gotch FA, Lipps BJ. PACK PD: a urea kinetic modeling computer program for peritoneal dialysis. Perit Dial Int. 1997;17 Suppl 2:S126–130.

99. Harty J, et al. Impact of increasing dialysis volume on adequacy targets: a prospective study. J Am Soc Nephrol. 1997;8(8):1304–1310.
http://www.ncbi.nlm.nih.gov/pubmed/9259358

100. Gao H, Lew SQ, Bosch JP. The effects of increasing exchange volume and frequency on peritoneal dialysis adequacy. Clin Nephrol. 1998;50(6):375–380.
http://www.ncbi.nlm.nih.gov/pubmed/9877111

101. Page DE. Comparing an additional hour of cycler therapy to an additional midday exchange to achieve adequacy targets. Adv Perit Dial. 2000;16:102–103.
http://www.ncbi.nlm.nih.gov/pubmed/11045271

102. Wolfson M, et al. A randomized controlled trial to evaluate the efficacy and safety of icodextrin in peritoneal dialysis. Am J Kidney Dis. 2002;40(5): 1055–1065.

103. Finkelstein F, et al. Superiority of icodextrin compared with 4.25% dextrose for peritoneal ultrafiltration. J Am Soc Nephrol. 2005;16(2):546–554.

104. Juergensen PH, et al. Increasing the dialysis volume and frequency in a fixed period of time in CPD patients: the effect on Kpt/V and creatinine clearance. Perit Dial Int. 2002;22(6):693–697.
http://www.ncbi.nlm.nih.gov/pubmed/12556071

105. Sarkar S, et al. Tolerance of large exchange volumes by peritoneal dialysis patients. Am J Kidney Dis. 1999;33(6):1136–1141.
http://www.ncbi.nlm.nih.gov/pubmed/10352203

106. Fukatsu A, et al. Clinical benefits and tolerability of increased fill volumes in Japanese peritoneal dialysis patients. Perit Dial Int. 2001;21(5):455–461.
http://www.ncbi.nlm.nih.gov/pubmed/11757828

107. Konings CJ, et al. Fluid status in CAPD patients is related to peritoneal transport and residual renal function: evidence from a longitudinal study. Nephrol Dial Transplant. 2003;18(4):797–803.

108. Szeto CC, et al. Independent effects of residual renal function and dialysis adequacy on nutritional status and patient outcome in continuous ambulatory peritoneal dialysis. Am J Kidney Dis. 1999;34(6):1056–1064.

109. Menon MK, et al. Long-term blood pressure control in a cohort of peritoneal dialysis patients and its association with residual renal function. Nephrol Dial Transplant. 2001;16(11):2207–2213.
http://www.ncbi.nlm.nih.gov/pubmed/11682669

110. Wang AY, et al. Inflammation, residual kidney function, and cardiac hy-pertrophy are interrelated and combine adversely to enhance mortality and cardiovascular death risk of peritoneal dialysis patients. J Am Soc Nephrol. 2004;15(8):2186–2194.
http://www.ncbi.nlm.nih.gov/pubmed/15284304

111. Wang AY, et al. Important differentiation of factors that predict outcome in peritoneal dialysis patients with different degrees of residual renal function. Nephrol Dial Transplant. 2005;20(2):396–403.
112. Caravaca F, Arrobas M, Dominguez C. Influence of residual renal function on dietary protein and caloric intake in patients on incremental peritoneal dialysis. Perit Dial Int. 1999;19(4):350–356.
http://www.ncbi.nlm.nih.gov/pubmed/10507816
113. Lopez-Menchero R, et al. Importance of residual renal function in continuous ambulatory peritoneal dialysis: its influence on different parameters of renal replacement treatment. Nephron. 1999;83(3):219–225.
114. van de Kerkhof J, et al. Nepsilon-(carboxymethyl)lysine, Nepsilon-(carboxyethyl)lysine and vascular cell adhesion molecule-1 (VCAM-1) in relation to peritoneal glucose prescription and residual renal function; a study in peritoneal dialysis patients. Nephrol Dial Transplant. 2004;19(4):910–916.
115. Bammens B, et al. Removal of middle molecules and protein-bound solutes by peritoneal dialysis and relation with uremic symptoms. Kidney Int. 2003;64(6):2238–2243.
http://www.ncbi.nlm.nih.gov/pubmed/14633148
116. Termorshuizen F, et al. The relative importance of residual renal function compared with peritoneal clearance for patient survival and quality of life: an analysis of the Netherlands Cooperative Study on the Adequacy of Dialysis (NECOSAD)-2. Am J Kidney Dis. 2003;41(6):1293–1302.
http://www.ncbi.nlm.nih.gov/pubmed/12776283
117. Han SH, et al. Reduced residual renal function is a risk of peritonitis in continuous ambulatory peritoneal dialysis patients. Nephrol Dial Transplant. 2007;22(9):2653–2658.
118. Liao CT, et al. Rate of decline of residual renal function is associated with all-cause mortality and technique failure in patients on long-term peritoneal dialysis. Nephrol Dial Transplant. 2009;24(9):2909–2914.
119. Fang W, Oreopoulos DG, Bargman JM. Use of ACE inhibitors or angiotensin receptor blockers and survival in patients on peritoneal dialysis. Nephrol Dial Transplant. 2008;23(11):3704–3710.
http://www.ncbi.nlm.nih.gov/pubmed/18567695
120. Li PK, et al. Effects of an angiotensin-converting enzyme inhibitor on residual renal function in patients receiving peritoneal dialysis. A randomized, controlled study. Ann Intern Med. 2003;139(2):105–112.
http://www.ncbi.nlm.nih.gov/pubmed/12859160
121. Suzuki H, et al. Effects of an angiotensin II receptor blocker, valsartan, on re-sidual renal function in patients on CAPD. Am J Kidney Dis. 2004;43(6):1056–1064.
http://www.ncbi.nlm.nih.gov/pubmed/15168386
122. Phakdeekitcharoen B, Leelasa-nguan P. Effects of an ACE inhibitor or angiotensin receptor blocker on potassium in CAPD patients. Am J Kidney Dis. 2004;44(4):738–746.
http://www.ncbi.nlm.nih.gov/pubmed/15384026
123. Williams JD, et al. The Euro-Balance Trial: the effect of a new biocompatible peritoneal dialysis fluid (balance) on the peritoneal membrane. Kidney Int. 2004;66(1):408–418.
124. Szeto CC, et al. Clinical biocompatibility of a neutral peritoneal dialysis solution with minimal glucose-degradation products—a 1–year randomized control trial. Nephrol Dial Transplant. 2007;22(2):552–559.
125. Fan SL, et al. Randomized controlled study of biocompatible peritoneal dialysis solutions: effect on residual renal function. Kidney Int. 2008;73(2):200–206.
http://www.ncbi.nlm.nih.gov/pubmed/17914351
126. Kim S, et al. Benefits of biocompatible PD fluid for preservation of residual renal function in incident CAPD patients: a 1-year study. Nephrol Dial Transplant. 2009;24(9):2899–2908.
http://www.ncbi.nlm.nih.gov/pubmed/19258384
126a. Johnson DW, Brown FG, Clarke M, et al. Effects of biocompatible versus standard fluid on peritoneal dialysis outcomes. J Am Soc Nephrol. 2012;23:1097–1107.
http://www.ncbi.nlm.nih.gov/pubmed/22440906
127. Nolph KD, et al. The kinetics of ultrafiltration during peritoneal dialysis: the role of lymphatics. Kidney Int. 1987;32(2):219–226.
128. Struijk DG, et al. Indirect measurement of lymphatic absorption in CAPD patients is not influenced by trapping. Kidney Int. 1992;41(6):1668–1675.
129. Imholz AL, et al. Residual volume measurements in CAPD patients with exogenous and endogenous solutes. Adv Perit Dial. 1992;8:33–38.
http://www.ncbi.nlm.nih.gov/pubmed/1475921
130. Koomen GC, et al. A fast reliable method for the measurement of intraperito-neal dextran 70, used to calculate lymphatic absorption. Adv Perit Dial. 1991;7:10–14.
http://www.ncbi.nlm.nih.gov/pubmed/1716991
131. Lam MF, et al. Retroperitoneal leakage as a cause of acute ultrafiltration failure: its associated risk factors in peritoneal dialysis. Perit Dial Int. 2009;29(5):542–547.
http://www.ncbi.nlm.nih.gov/pubmed/19776048
132. Heimburger O, et al. Peritoneal transport in CAPD patients with permanent loss of ultrafiltration capacity. Kidney Int. 1990;38(3):495–506.
http://www.ncbi.nlm.nih.gov/pubmed/2232493
133. Heimburger O, et al. A quantitative description of solute and fluid transport during peritoneal dialysis. Kidney Int. 1992;41(5):1320–1332.
http://www.ncbi.nlm.nih.gov/pubmed/1614047
134. Krediet RT, et al. Alterations in the peritoneal transport of water and solutes during peritonitis in continuous ambulatory peritoneal dialysis patients. Eur J Clin Invest. 1987;17(1):43–52.
135. Posthuma N, et al. Icodextrin use in CCPD patients during peritonitis: ultrafiltration and serum disaccharide concentrations. Nephrol Dial Transplant. 1998;13(9):2341–2344.
http://www.ncbi.nlm.nih.gov/pubmed/9761519
136. Krediet RT. The peritoneal membrane in chronic peritoneal dialysis. Kidney Int. 1999;55(1):341–356.
http://www.ncbi.nlm.nih.gov/pubmed/9893150
137. Williams JD, et al. Morphologic changes in the peritoneal membrane of patients with renal disease. J Am Soc Nephrol. 2002;13(2):470–479.
http://www.ncbi.nlm.nih.gov/pubmed/11805177
138. Davies SJ, et al. Peritoneal glucose exposure and changes in membrane solute transport with time on peritoneal dialysis. J Am Soc Nephrol. 2001;12(5):1046–1051.
139. Davies SJ, et al. Longitudinal membrane function in functionally anuric patients treated with APD: data from EAPOS on the effects of glucose and icodextrin prescription. Kidney Int. 2005;67(4):1609–1615.
140. Rodrigues A, et al. Peritoneal rest may successfully recover ultrafiltration in patients who develop peritoneal hyperpermeability with time on continuous ambulatory peritoneal dialysis. Adv Perit Dial. 2002;18:78–80.
http://www.ncbi.nlm.nih.gov/pubmed/12402593
141. Mistry CD, Gokal R, Peers E. A randomized multicenter clinical trial comparing isosmolar icodextrin with hyperosmolar glucose solutions in CAPD. MIDAS Study Group. Multicenter Investigation of Icodextrin in Ambulatory Peritoneal Dialysis. Kidney Int. 1994;46(2):496–503.
142. Davies SJ, et al. Icodextrin improves the fluid status of peritoneal dialysis patients: results of a double-blind randomized controlled trial. J Am Soc Nephrol. 2003;14(9):2338–2344.
http://www.ncbi.nlm.nih.gov/pubmed/12937311
143. Konings CJ, et al. Effect of icodextrin on volume status, blood pressure and echocardiographic parameters: a randomized study. Kidney Int. 2003;63(4):1556–1563.
http://www.ncbi.nlm.nih.gov/pubmed/12631373
144. Sav T, et al. Effects of twice-daily icodextrin administration on blood pressure and left ventricular mass in patients on continuous ambulatory peritoneal dialysis. Perit Dial Int. 2009;29(4):443–449.
http://www.ncbi.nlm.nih.gov/pubmed/19602610
145. Monquil MC, et al. Does impaired transcellular water transport contribute to net ultrafiltration failure during CAPD? Perit Dial Int. 1995;15(1):42–48.
http://www.ncbi.nlm.nih.gov/pubmed/7734560
146. Goffin E, et al. Expression of aquaporin-1 in a long-term peritoneal dialysis patient with impaired transcellular water transport. Am J Kidney Dis. 1999;33(2):383–388.
http://www.ncbi.nlm.nih.gov/pubmed/10023655
147. Han SH, et al. Mortality and technique failure in peritoneal dialysis patients using advanced peritoneal dialysis solutions. Am J Kidney Dis. 2009;54(4):711–720.
148. Dobos GJ, et al. Bicarbonate-based dialysis solution preserves granulocyte functions. Perit Dial Int. 1994;14(4):366–370.
http://www.ncbi.nlm.nih.gov/pubmed/7827187
149. Chaudhary K, Khanna R. Biocompatible peritoneal dialysis solutions: do we have one? Clin J Am Soc Nephrol. 2010;5(4):723–732.
http://www.ncbi.nlm.nih.gov/pubmed/20093342
150. Sundaram S, et al. Effect of two-chambered bicarbonate lactate-buffered peritoneal dialysis fluids on peripheral blood mononuclear cell and polymorphonuclear cell function in vitro. Am J Kidney Dis. 1997;30(5):680–689.
151. Mackenzie RK, et al. In vivo exposure to bicarbonate/lactate- and bicarbonate-buffered peritoneal dialysis fluids improves ex vivo peritoneal macrophage function. Am J Kidney Dis. 2000;35(1):112–121.
http://www.ncbi.nlm.nih.gov/pubmed/10620552
152. Pawlaczyk K, et al. Bicarbonate/lactate dialysis solution improves in vivo function of peritoneal host defense in rats. Perit Dial Int. 1999;19 Suppl 2:S370–377.

153. Tranaeus A. A long-term study of a bicarbonate/lactate-based peritoneal dialysis solution—clinical benefits. The Bicarbonate/Lactate Study Group. Perit Dial Int. 2000;20(5):516–523.
http://www.ncbi.nlm.nih.gov/pubmed/11117242
154. Wang T, et al. Effect of peritonitis on peritoneal transport characteristics: glucose solution versus polyglucose solution. Kidney Int. 2000;57(4):1704–1712.
http://www.ncbi.nlm.nih.gov/pubmed/10760106
155. Brulez HF, et al. In vitro compatibility of a 1.1% amino acid containing peritoneal dialysis fluid with phagocyte function. Adv Perit Dial. 1994;10:241–244.
http://www.ncbi.nlm.nih.gov/pubmed/7999837
156. Olszowska A, et al. Peritoneal transport in peritoneal dialysis patients using glucose-based and amino acid-based solutions. Perit Dial Int. 2007;27(5):544–553.
http://www.ncbi.nlm.nih.gov/pubmed/17704445
157. Tjiong HL, et al. Amino acid-based peritoneal dialysis solutions for malnutrition: new perspectives. Perit Dial Int. 2009;29(4):384–393.
http://www.ncbi.nlm.nih.gov/pubmed/19602603
158. Palmer RA, et al. Treatment of chronic renal failure by prolonged peritoneal dialysis. N Engl J Med. 1966;274(5):248–254.
http://www.ncbi.nlm.nih.gov/pubmed/5902218
159. Tenckhoff H, Schechter H. A bacteriologically safe peritoneal access device. Trans Am Soc Artif Intern Organs. 1968;14:181–187.
http://www.ncbi.nlm.nih.gov/pubmed/5701529
160. Gokal R, et al. Peritoneal catheters and exit-site practices toward optimum peritoneal access: 1998 update. (Official report from the International Society for Peritoneal Dialysis.) Perit Dial Int. 1998;18(1):11–33.
http://www.ncbi.nlm.nih.gov/pubmed/9527026
161. Strippoli GF, et al. Catheter type, placement and insertion techniques for preventing peritonitis in peritoneal dialysis patients. Cochrane Database Syst Rev. 2004(4):CD004680.
162. Twardowski ZJ, et al. The need for a "swan neck" permanently bent, arcuate peritoneal dialysis catheter. Perit Dial Int. 1985;5(4):219–223.
163. Lye WC, et al. A prospective randomized comparison of the Swan neck, coiled, and straight Tenckhoff catheters in patients on CAPD. Perit Dial Int. 1996;16(Suppl_1):S333–335.
164. Eklund BH, et al. Catheter configuration and outcome in patients on continuous ambulatory peritoneal dialysis: a prospective comparison of two catheters. Perit Dial Int. 1994;14(1):70–74.
165. Twardowski ZJ, et al. Six-year experience with Swan neck presternal peritoneal dialysis catheter. Perit Dial Int. 1998;18(6):598–602.
http://www.ncbi.nlm.nih.gov/pubmed/9932658
166. Bergamin B, et al. Finding the right position: a three-year, single-center experience with the "self-locating" catheter. Perit Dial Int. 2010;30(5):519–523.
http://www.ncbi.nlm.nih.gov/pubmed/20228177
167. Di Paolo N, Gaggiotti E. The self-locating peritoneal catheter. Int J Artif Organs. 2004;27(4):261–264.
http://www.ncbi.nlm.nih.gov/pubmed/15163060
168. Flanigan M, Gokal R. Peritoneal catheters and exit-site practices toward optimum peritoneal access: a review of current developments. Perit Dial Int. 2005;25(2):132–139.
http://www.ncbi.nlm.nih.gov/pubmed/15796138
169. Katyal A, Mahale A, Khanna R. Antibiotic prophylaxis before peritoneal dialysis catheter insertion. Adv Perit Dial. 2002;18:112–115.
http://www.ncbi.nlm.nih.gov/pubmed/12402600
170. Crabtree JH. Selected best demonstrated practices in peritoneal dialysis access. Kidney Int Suppl. 2006(103):S27–37.
171. Crabtree JH, Burchette RJ. Effective use of laparoscopy for long-term peritoneal dialysis access. Am J Surg. 2009;198(1):135–141.
http://www.ncbi.nlm.nih.gov/pubmed/19306986
172. Wright MJ, et al. Randomized prospective comparison of laparoscopic and open peritoneal dialysis catheter insertion. Perit Dial Int. 1999;19(4):372–375.
http://www.ncbi.nlm.nih.gov/pubmed/10507820
173. Rosenthal MA, et al. Comparison of outcomes of peritoneal dialysis catheters placed by the fluoroscopically guided percutaneous method versus directly visualized surgical method. J Vasc Interv Radiol. 2008;19(8):1202–1207.
174. Moncrief JW, et al. The Moncrief-Popovich catheter. A new peritoneal access technique for patients on peritoneal dialysis. ASAIO J. 1993;39(1):62–65.
http://www.ncbi.nlm.nih.gov/pubmed/8439683
174a. Elhassan E, McNair B, Quinn M, et al. Prolonged duration of peritoneal dialysis catheter embedment does not lower the catheter success rate. Perit Dial Int. 2011;31(5):558–564.
http://www.ncbi.nlm.nih.gov/pubmed/21632444
175. Park MS, et al. Effect of prolonged subcutaneous implantation of peritoneal catheter on peritonitis rate during CAPD: a prospective randomized study. Blood Purif. 1998;16(3):171–178.
http://www.ncbi.nlm.nih.gov/pubmed/9681160
176. Danielsson A, et al. A prospective randomized study of the effect of a subcutaneously "buried" peritoneal dialysis catheter technique versus standard technique on the incidence of peritonitis and exit-site infection. Perit Dial Int. 2002;22(2):211–219.
http://www.ncbi.nlm.nih.gov/pubmed/11990406
177. Brum S, et al. Moncrief-Popovich technique is an advantageous method of peritoneal dialysis catheter implantation. Nephrol Dial Transplant. 2010; 25(9):3070–3075.
http://www.ncbi.nlm.nih.gov/pubmed/20339096
178. Kubota M, et al. Implantation of presternal catheter using Moncrief technique: aiming for fewer catheter-related complications. Perit Dial Int. 2001;21 Suppl 3:S205–208.
179. Clark KR, et al. Surgical aspects of chronic peritoneal dialysis in the neonate and infant under 1 year of age. J Pediatr Surg. 1992;27(6):780–783.
http://www.ncbi.nlm.nih.gov/pubmed/1501047
180. Campos RP, Chula DC, Riella MC. Complications of the peritoneal access and their management. Contrib Nephrol. 2009;163:183–197.
http://www.ncbi.nlm.nih.gov/pubmed/19494613
181. Lye WC, et al. Breaking-in after the insertion of Tenckhoff catheters: a comparison of two techniques. Adv Perit Dial. 1993;9:236–239.
http://www.ncbi.nlm.nih.gov/pubmed/8105933
182. Bender FH, Bernardini J, Piraino B. Prevention of infectious complications in peritoneal dialysis: best demonstrated practices. Kidney Int Suppl. 2006(103):S44–54.
183. Robison RJ, et al. Surgical considerations of continuous ambulatory peritoneal dialysis. Surgery. 1984;96(4):723–730.
http://www.ncbi.nlm.nih.gov/pubmed/6385317
184. Swartz R, et al. The curled catheter: dependable device for percutaneous peritoneal access. Perit Dial Int. 1990;10(3):231–235.
http://www.ncbi.nlm.nih.gov/pubmed/2094463
185. Joffe P, Christensen AL, Jensen C. Peritoneal catheter tip location during non-complicated continuous ambulatory peritoneal dialysis. Perit Dial Int. 1991;11(3):261–264.
http://www.ncbi.nlm.nih.gov/pubmed/1912020
186. Zorzanello MM, Fleming WJ, Prowant BE. Use of tissue plasminogen activator in peritoneal dialysis catheters: a literature review and one center's experience. Nephrol Nurs J. 2004;31(5):534–537.
http://www.ncbi.nlm.nih.gov/pubmed/15518255
187. Wiegmann TB, et al. Effective use of streptokinase for peritoneal catheter failure. Am J Kidney Dis. 1985;6(2):119–123.
188. Nicholson ML, et al. The role of omentectomy in continuous ambulatory peritoneal dialysis. Perit Dial Int. 1991;11(4):330–332.
http://www.ncbi.nlm.nih.gov/pubmed/1751599
189. Leblanc M, Ouimet D, Pichette V. Dialysate leaks in peritoneal dialysis. Semin Dial. 2001;14(1):50–54.
http://www.ncbi.nlm.nih.gov/pubmed/11208040
190. Stegmayr B, et al. Absence of leakage by insertion of peritoneal dialysis catheter through the rectus muscle. Perit Dial Int. 1990;10(1):53–55.
http://www.ncbi.nlm.nih.gov/pubmed/2150764
191. Apostolidis NS, et al. The use of TWH catheters in CAPD patients: fourteen-year experience in technique, survival, and complication rates. Perit Dial Int. 1998;18(4):424–428.
http://www.ncbi.nlm.nih.gov/pubmed/10505566
192. Litherland J, et al. Computed tomographic peritoneography: CT manifestations in the investigation of leaks and abnormal collections in patients on CAPD. Nephrol Dial Transplant. 1994;9(10):1449–1452.
193. Holley JL, Bernardini J, Piraino B. Characteristics and outcome of peritoneal dialysate leaks and associated infections. Adv Perit Dial. 1993;9: 240–243.
http://www.ncbi.nlm.nih.gov/pubmed/8105934
194. Hirsch DJ, Jindal KK. Late leaks in peritoneal dialysis patients. Nephrol Dial Transplant. 1991;6(9):670–671.
http://www.ncbi.nlm.nih.gov/pubmed/1745393
195. Piraino B, et al. Peritoneal dialysis-related infections recommendations: 2005 update. Perit Dial Int. 2005;25(2):107–131.
http://www.ncbi.nlm.nih.gov/pubmed/15796137
196. Sewell DL, et al. Comparison of large volume culture to other methods for isolation of microorganisms from dialysate. Perit Dial Int. 1990;10(1):49–52.
http://www.ncbi.nlm.nih.gov/pubmed/2128189
197. Alfa MJ, et al. Improved detection of bacterial growth in continuous ambulatory peritoneal dialysis effluent by use of BacT/Alert FAN bottles. J Clin Microbiol. 1997;35(4):862–866.
198. Koopmans JG, et al. Impaired initial cell reaction in CAPD-related peritonitis. Perit Dial Int. 1996;16 Suppl 1:S362–367.
199. Gonthier D, et al. Erythema: does it indicate infection in a peritoneal catheter exit site? Adv Perit Dial. 1992;8:230–233.
http://www.ncbi.nlm.nih.gov/pubmed/1361794

200. Plum J, Sudkamp S, Grabensee B. Results of ultrasound-assisted diagnosis of tunnel infections in continuous ambulatory peritoneal dialysis. Am J Kidney Dis. 1994;23(1):99–104.
http://www.ncbi.nlm.nih.gov/pubmed/8285204
201. Gupta B, Bernardini J, Piraino B. Peritonitis associated with exit site and tunnel infections. Am J Kidney Dis. 1996;28(3):415–419.
http://www.ncbi.nlm.nih.gov/pubmed/8804241
202. Woodrow G, Turney JH, Brownjohn AM. Technique failure in peritoneal dialysis and its impact on patient survival. Perit Dial Int. 1997;17(4):360–364.
http://www.ncbi.nlm.nih.gov/pubmed/9284463
203. Piraino B, Bernardini J, Sorkin M. The influence of peritoneal catheter exit-site infections on peritonitis, tunnel infections, and catheter loss in patients on continuous ambulatory peritoneal dialysis. Am J Kidney Dis. 1986;8(6):436–440.
204. Fried L, et al. Hospitalization in peritoneal dialysis patients. Am J Kidney Dis. 1999;33(5):927–933.
http://www.ncbi.nlm.nih.gov/pubmed/10213651
205. Fried LF, et al. Peritonitis influences mortality in peritoneal dialysis patients. J Am Soc Nephrol. 1996;7(10):2176–2182.
http://www.ncbi.nlm.nih.gov/pubmed/8915978
206. Perez Fontan M, et al. Peritonitis-related mortality in patients undergoing chronic peritoneal dialysis. Perit Dial Int. 2005;25(3):274–284.
http://www.ncbi.nlm.nih.gov/pubmed/15981776
207. Holmes CJ. Peritoneal host defense mechanisms in peritoneal dialysis. Kidney Int Suppl. 1994;48:S58–70.
208. Betjes MG, et al. Analysis of the peritoneal cellular immune system during CAPD shortly before a clinical peritonitis. Nephrol Dial Transplant. 1994;9(6):684–692.
209. Ates K, et al. The longitudinal effect of a single peritonitis episode on peritoneal membrane transport in CAPD patients. Perit Dial Int. 2000;20(2):220–226.
http://www.ncbi.nlm.nih.gov/pubmed/10809247
210. Lai KN, et al. Changes of cytokine profiles during peritonitis in patients on continuous ambulatory peritoneal dialysis. Am J Kidney Dis. 2000;35(4):644–652.
211. Jorres A, et al. Impact of peritoneal dialysis solutions on peritoneal immune defense. Perit Dial Int. 1993;13 Suppl 2:S291–294.
212. Posthuma N, et al. Peritoneal defense using icodextrin or glucose for daytime dwell in CCPD patients. Perit Dial Int. 1999;19(4):334–342.
http://www.ncbi.nlm.nih.gov/pubmed/10507814
213. de Fijter CW, et al. Biocompatibility of a glucose-polymer-containing peritoneal dialysis fluid. Am J Kidney Dis. 1993;21(4):411–418.
http://www.ncbi.nlm.nih.gov/pubmed/8465822
214. Kim SG, et al. Could solutions low in glucose degradation products preserve residual renal function in incident peritoneal dialysis patients? A 1–year multicenter prospective randomized controlled trial (Balnet Study). Perit Dial Int. 2008;28 Suppl 3:S117–122.
215. Buoncristiani U. Continuous ambulatory peritoneal dialysis connection systems. Perit Dial Int. 1993;13 Suppl 2:S139–145.
216. Strippoli GF, et al. Catheter-related interventions to prevent peritonitis in peritoneal dialysis: a systematic review of randomized, controlled trials. J Am Soc Nephrol. 2004;15(10):2735–2746.
217. Monteon F, et al. Prevention of peritonitis with disconnect systems in CAPD: a randomized controlled trial. The Mexican Nephrology Collaborative Study Group. Kidney Int. 1998;54(6):2123–2128.
218. Kiernan L, et al. Comparison of continuous ambulatory peritoneal dialysis-related infections with different "Y-tubing" exchange systems. J Am Soc Nephrol. 1995;5(10):1835–1838.
http://www.ncbi.nlm.nih.gov/pubmed/7787152
219. Rabindranath KS, et al. Automated vs continuous ambulatory peritoneal dialysis: a systematic review of randomized controlled trials. Nephrol Dial Transplant. 2007;22(10):2991–2998.
http://www.ncbi.nlm.nih.gov/pubmed/17875571
220. de Fijter CW, et al. Clinical efficacy and morbidity associated with continuous cyclic compared with continuous ambulatory peritoneal dialysis. Ann Intern Med. 1994;120(4):264–271.
http://www.ncbi.nlm.nih.gov/pubmed/8291819
221. Oo TN, Roberts TL, Collins AJ. A comparison of peritonitis rates from the United States Renal Data System database: CAPD versus continuous cycling peritoneal dialysis patients. Am J Kidney Dis. 2005;45(2):372–380.
222. Ramalakshmi S, Bernardini J, Piraino B. Nightly intermittent peritoneal dialysis to initiate peritoneal dialysis. Adv Perit Dial. 2003;19:111–114.
http://www.ncbi.nlm.nih.gov/pubmed/14763045
223. Li PK, et al. Peritoneal dialysis-related infections recommendations: 2010 update. Perit Dial Int. 2010;30(4):393–423.
http://www.ncbi.nlm.nih.gov/pubmed/20628102
224. Bernardini J, et al. Randomized, double-blind trial of antibiotic exit site cream for prevention of exit site infection in peritoneal dialysis patients. J Am Soc Nephrol. 2005;16(2):539–545.
http://www.ncbi.nlm.nih.gov/pubmed/15625071
225. Holley JL, Bernardini J, Piraino B. Risk factors for tunnel infections in continuous peritoneal dialysis. Am J Kidney Dis. 1991;18(3):344–348.
http://www.ncbi.nlm.nih.gov/pubmed/1882826
226. Holley JL, et al. Ultrasound as a tool in the diagnosis and management of exit-site infections in patients undergoing continuous ambulatory peritoneal dialysis. Am J Kidney Dis. 1989;14(3):211–216.
227. Flanigan MJ, et al. Continuous ambulatory peritoneal dialysis catheter infections: diagnosis and management. Perit Dial Int. 1994;14(3):248–254.
http://www.ncbi.nlm.nih.gov/pubmed/7948237
228. Holley JL, Bernardini J, Piraino B. Infecting organisms in continuous ambulatory peritoneal dialysis patients on the Y-set. Am J Kidney Dis. 1994;23(4):569–573.
http://www.ncbi.nlm.nih.gov/pubmed/8154494
229. Kazmi HR, et al. Pseudomonas exit site infections in continuous ambulatory peritoneal dialysis patients. J Am Soc Nephrol. 1992;2(10):1498–1501.
230. Bernardini J, Piraino B, Sorkin M. Analysis of continuous ambulatory peritoneal dialysis-related Pseudomonas aeruginosa infections. Am J Med. 1987;83(5):829–832.
231. Abraham G, et al. Natural history of exit-site infection (ESI) in patients on con-tinuous ambulatory peritoneal dialysis (CAPD). Perit Dial Int. 1988;8(3):211–216.
232. Bayston R, et al. Recurrent infection and catheter loss in patients on continuous ambulatory peritoneal dialysis. Perit Dial Int. 1999;19(6):550–555.
http://www.ncbi.nlm.nih.gov/pubmed/10641776
233. Thodis E, et al. Decrease in Staphylococcus aureus exit-site infections and peritonitis in CAPD patients by local application of mupirocin ointment at the catheter exit site. Perit Dial Int. 1998;18(3):261–270.
http://www.ncbi.nlm.nih.gov/pubmed/9663889
234. Bernardini J, et al. A randomized trial of Staphylococcus aureus prophylaxis in peritoneal dialysis patients: mupirocin calcium ointment 2% applied to the exit site versus cyclic oral rifampin. Am J Kidney Dis. 1996;27(5):695–700.
http://www.ncbi.nlm.nih.gov/pubmed/8629630
235. Tacconelli E, et al. Mupirocin prophylaxis to prevent Staphylococcus aureus infection in patients undergoing dialysis: a meta-analysis. Clin Infect Dis. 2003;37(12):1629–1638.
236. Annigeri R, et al. Emergence of mupirocin-resistant Staphylococcus aureus in chronic peritoneal dialysis patients using mupirocin prophylaxis to prevent exit-site infection. Perit Dial Int. 2001;21(6):554–559.
http://www.ncbi.nlm.nih.gov/pubmed/11783763
237. Perez-Fontan M, et al. Mupirocin resistance after long-term use for Staphylococcus aureus colonization in patients undergoing chronic peritoneal dialysis. Am J Kidney Dis. 2002;39(2):337–341.
http://www.ncbi.nlm.nih.gov/pubmed/11840374
238. Montenegro J, et al. Exit-site care with ciprofloxacin otologic solution prevents polyurethane catheter infection in peritoneal dialysis patients. Perit Dial Int. 2000;20(2):209–214.
http://www.ncbi.nlm.nih.gov/pubmed/10809245
239. Mujais S, Story K. Peritoneal dialysis in the US: evaluation of outcomes in contemporary cohorts. Kidney Int Suppl. 2006(103):S21–26.
240. Mujais S. Microbiology and outcomes of peritonitis in North America. Kidney Int Suppl. 2006;(103):S55–62.
241. Tofte-Jensen P, et al. PD-related infections of standard and different disconnect systems. Adv Perit Dial. 1994;10:214–217.
http://www.ncbi.nlm.nih.gov/pubmed/7999831
242. Lupo A, et al. Long-term outcome in continuous ambulatory peritoneal dialysis: a 10-year-survey by the Italian Cooperative Peritoneal Dialysis Study Group. Am J Kidney Dis. 1994;24(5):826–837.
243. Szeto CC, et al. Change in bacterial aetiology of peritoneal dialysis-related peritonitis over 10 years: experience from a centre in South-East Asia. Clin Microbiol Infect. 2005;11(10):837–839.
244. Korbet SM, Vonesh EF, Firanek CA. A retrospective assessment of risk factors for peritonitis among an urban CAPD population. Perit Dial Int. 1993;13(2):126–131.
http://www.ncbi.nlm.nih.gov/pubmed/8494934
245. Farias MG, et al. Race and the risk of peritonitis: an analysis of factors associated with the initial episode. Kidney Int. 1994;46(5):1392–1396.
http://www.ncbi.nlm.nih.gov/pubmed/7853799
246. Lye WC, et al. A prospective study of peritoneal dialysis-related infections in CAPD patients with diabetes mellitus. Adv Perit Dial. 1993;9:195–197.
http://www.ncbi.nlm.nih.gov/pubmed/8105922

247. Viglino G, et al. Ten years experience of CAPD in diabetics: comparison of results with non-diabetics. Italian Cooperative Peritoneal Dialysis Study Group. Nephrol Dial Transplant. 1994;9(10):1443–1448.
248. Szeto CC, Kwan BC, Chow KM. Peritonitis risk for older patients on peritoneal dialysis. Perit Dial Int. 2008;28(5):457–460.
http://www.ncbi.nlm.nih.gov/pubmed/18708536
249. Yinnon AM, et al. Comparison of peritoneal fluid culture results from adults and children undergoing CAPD. Perit Dial Int. 1999;19(1):51–55.
http://www.ncbi.nlm.nih.gov/pubmed/10201341
250. Andrews PA, et al. Impaired outcome of continuous ambulatory peritoneal dialysis in immunosuppressed patients. Nephrol Dial Transplant. 1996;11(6):1104–1108.
251. Port FK, et al. Risk of peritonitis and technique failure by CAPD connection technique: a national study. Kidney Int. 1992;42(4):967–974.
252. Read RR, et al. Peritonitis in peritoneal dialysis: bacterial colonization by biofilm spread along the catheter surface. Kidney Int. 1989;35(2):614–621.
253. Eisenberg ES, et al. Colonization of skin and development of peritonitis due to coagulase-negative staphylococci in patients undergoing peritoneal dialysis. J Infect Dis. 1987;156(3):478–482.
http://www.ncbi.nlm.nih.gov/pubmed/3611833
254. Gorman SP, et al. Confocal laser scanning microscopy of peritoneal catheter surfaces. J Med Microbiol. 1993;38(6):411–417.
http://www.ncbi.nlm.nih.gov/pubmed/8510133
255. Beaman M, et al. Peritonitis caused by slime-producing coagulase negative staphylococci in continuous ambulatory peritoneal dialysis. Lancet. 1987;1(8523):42.
http://www.ncbi.nlm.nih.gov/pubmed/2879118
256. Swartz R, et al. Biofilm formation on peritoneal catheters does not require the presence of infection. ASAIO Trans. 1991;37(4):626–634.
257. Miller TE, Findon G. Touch contamination of connection devices in peritoneal dialysis—a quantitative microbiologic analysis. Perit Dial Int. 1997;17(6):560–567.
http://www.ncbi.nlm.nih.gov/pubmed/9655155
258. Szeto CC, et al. Enterobacteriaceae peritonitis complicating peritoneal dialysis: a review of 210 consecutive cases. Kidney Int. 2006;69(7):1245–1252.
259. Siva B, et al. Pseudomonas peritonitis in Australia: predictors, treatment, and outcomes in 191 cases. Clin J Am Soc Nephrol. 2009;4(5):957–964.
http://www.ncbi.nlm.nih.gov/pubmed/19406972
260. Troidle L, et al. Differing outcomes of gram-positive and gram-negative peritonitis. Am J Kidney Dis. 1998;32(4):623–628.
http://www.ncbi.nlm.nih.gov/pubmed/9774124
261. Wood CJ, et al. Campylobacter peritonitis in continuous ambulatory peritoneal dialysis: report of eight cases and a review of the literature. Am J Kidney Dis. 1992;19(3):257–263.
262. Singharetnam W, Holley JL. Acute treatment of constipation may lead to transmural migration of bacteria resulting in gram-negative, polymicrobial, or fungal peritonitis. Perit Dial Int. 1996;16(4):423–425.
http://www.ncbi.nlm.nih.gov/pubmed/8863339
263. Chuang YW, et al. Hypokalaemia: an independent risk factor of Enterobacteri-aceae peritonitis in CAPD patients. Nephrol Dial Transplant. 2009;24(5):1603–1608.
http://www.ncbi.nlm.nih.gov/pubmed/19103738
264. Harwell CM, et al. Abdominal catastrophe: visceral injury as a cause of peritonitis in patients treated by peritoneal dialysis. Perit Dial Int. 1997;17(6):586–594.
265. Burkart J, et al. Usefulness of peritoneal fluid amylase levels in the differential diagnosis of peritonitis in peritoneal dialysis patients. J Am Soc Nephrol. 1991;1(10):1186–1190.
266. Verger C, Danne O, Vuillemin F. Colonoscopy and continuous ambulatory peritoneal dialysis. Gastrointest Endosc. 1987;33(4):334–335.
http://www.ncbi.nlm.nih.gov/pubmed/3653661
267. Fried L, Bernardini J, Piraino B. Iatrogenic peritonitis: the need for prophylaxis. Perit Dial Int. 2000;20(3):343–345.
268. Troidle L, et al. Continuous peritoneal dialysis-associated peritonitis of nosocomial origin. Perit Dial Int. 1996;16(5):505–510.
269. Coward RA, et al. Peritonitis associated with vaginal leakage of dialysis fluid in continuous ambulatory peritoneal dialysis. Br Med J (Clin Res Ed). 1982;284(6328):1529.
http://www.ncbi.nlm.nih.gov/pubmed/6805592
270. Miles R, et al. Predictors and outcomes of fungal peritonitis in peritoneal dialysis patients. Kidney Int. 2009;76(6):622–628.
http://www.ncbi.nlm.nih.gov/pubmed/19516241
271. Prasad N, Gupta A. Fungal peritonitis in peritoneal dialysis patients. Perit Dial Int. 2005;25(3):207–222.
http://www.ncbi.nlm.nih.gov/pubmed/15981767
272. Goldie SJ, et al. Fungal peritonitis in a large chronic peritoneal dialysis population: a report of 55 episodes. Am J Kidney Dis. 1996;28(1):86–91.
273. Robitaille P, et al. Successful antifungal prophylaxis in chronic peritoneal dialysis: a pediatric experience. Perit Dial Int. 1995;15(1):77–79.
274. Williams PF, Moncrieff N, Marriott J. No benefit in using nystatin prophylaxis against fungal peritonitis in peritoneal dialysis patients. Perit Dial Int. 2000;20(3):352–353.
http://www.ncbi.nlm.nih.gov/pubmed/10898060
275. Thodis E, et al. Nystatin prophylaxis: its inability to prevent fungal peritonitis in patients on continuous ambulatory peritoneal dialysis. Perit Dial Int. 1998;18(6):583–589.
http://www.ncbi.nlm.nih.gov/pubmed/9932656
276. Lo WK, et al. A prospective randomized control study of oral nystatin prophylaxis for Candida peritonitis complicating continuous ambulatory peritoneal dialysis. Am J Kidney Dis. 1996;28(4):549–552.
277. Wadhwa NK, Suh H, Cabralda T. Antifungal prophylaxis for secondary fungal peritonitis in peritoneal dialysis patients. Adv Perit Dial. 1996;12:189–191.
http://www.ncbi.nlm.nih.gov/pubmed/8865899
278. Zaruba K, Peters J, Jungbluth H. Successful prophylaxis for fungal peritonitis in patients on continuous ambulatory peritoneal dialysis: six years' experience. Am J Kidney Dis. 1991;17(1):43–46.
http://www.ncbi.nlm.nih.gov/pubmed/1986569
279. Rocklin MA, Teitelbaum I. Noninfectious causes of cloudy peritoneal dialysate. Semin Dial. 2001;14(1):37–40.
280. Wang AY, Li PK, Lai KN. Comparison of intraperitoneal administration of two preparations of vancomycin in causing chemical peritonitis. Perit Dial Int. 1996; 16(2):172–174.
http://www.ncbi.nlm.nih.gov/pubmed/9147552
281. Fabris A, et al. Pharmacokinetics of antifungal agents. Perit Dial Int. 1993; 13 Suppl 2:S380–382.
282. Gokal R. Icodextrin-associated sterile peritonitis. Perit Dial Int. 2002;22(4):445–448.
http://www.ncbi.nlm.nih.gov/pubmed/12322814
283. Del Rosso G, et al. A new form of acute adverse reaction to icodextrin in a peritoneal dialysis patient. Nephrol Dial Transplant. 2000;15(6):927–928.
http://www.ncbi.nlm.nih.gov/pubmed/10831669
284. Martis L, et al. Aseptic peritonitis due to peptidoglycan contamination of pharmacopoeia standard dialysis solution. Lancet. 2005;365(9459):588–594.
http://www.ncbi.nlm.nih.gov/pubmed/15708102
285. Vychytil A, et al. New criteria for management of catheter infections in peritoneal dialysis patients using ultrasonography. J Am Soc Nephrol. 1998;9(2):290–296.
http://www.ncbi.nlm.nih.gov/pubmed/9527406
286. Crabtree JH, Burchette RJ. Surgical salvage of peritoneal dialysis catheters from chronic exit-site and tunnel infections. Am J Surg. 2005;190(1):4–8.
287. Szabo T, et al. Outcome of Pseudomonas aeruginosa exit-site and tunnel infections: a single center's experience. Adv Perit Dial. 1999;15:209–212.
http://www.ncbi.nlm.nih.gov/pubmed/10682104
288. Yinnon AM, Jain V, Magnussen CR. Group B Streptococcus (agalactiae) peritonitis and bacteremia associated with CAPD. Perit Dial Int. 1993;13(3):241.
http://www.ncbi.nlm.nih.gov/pubmed/8369359
289. Offer TP, et al. Group A streptococcal peritonitis associated with continuous ambulatory peritoneal dialysis. Am J Med. 1989;87(4):487.
290. Manley HJ, et al. Pharmacokinetics of intermittent intraperitoneal cefazolin in continuous ambulatory peritoneal dialysis patients. Perit Dial Int. 1999;19(1):65–70.
http://www.ncbi.nlm.nih.gov/pubmed/10201343
291. Grabe DW, et al. Pharmacokinetics of intermittent intraperitoneal ceftazidime. Am J Kidney Dis. 1999;33(1):111–117.
http://www.ncbi.nlm.nih.gov/pubmed/9915275
292. Shemin D, et al. Effect of aminoglycoside use on residual renal function in peritoneal dialysis patients. Am J Kidney Dis. 1999;34(1):14–20.
http://www.ncbi.nlm.nih.gov/pubmed/10401010
293. Lui SL, et al. Cefazolin plus netilmicin versus cefazolin plus ceftazidime for treating CAPD peritonitis: effect on residual renal function. Kidney Int. 2005;68(5):2375–2380.
294. Wiggins KJ, et al. Treatment of peritoneal dialysis-associated peritonitis: a systematic review of randomized controlled trials. Am J Kidney Dis. 2007;50(6):967–988.
295. Fontan MP, et al. Treatment of peritoneal dialysis-related peritonitis with ciprofloxacin monotherapy: clinical outcomes and bacterial susceptibility over two decades. Perit Dial Int. 2009;29(3):310–318.
296. Agraharkar M, et al. Use of cefazolin for peritonitis treatment in peritoneal dialysis patients. Am J Nephrol. 1999;19(5):555–558.
http://www.ncbi.nlm.nih.gov/pubmed/10575183

297. Ng R, et al. Vancomycin-resistant enterococcus infection is a rare com-plication in patients receiving PD on an outpatient basis. Perit Dial Int. 1999;19(3):273–274.
http://www.ncbi.nlm.nih.gov/pubmed/10433167
298. Vas S, Bargman J, Oreopoulos D. Treatment in PD patients of peritonitis caused by gram-positive organisms with single daily dose of antibiotics. Perit Dial Int. 1997;17(1):91–94.
http://www.ncbi.nlm.nih.gov/pubmed/9068032
299. Lye WC, Leong SO, Lee EJ. Methicillin-resistant Staphylococcus aureus nasal carriage and infections in CAPD. Kidney Int. 1993;43(6):1357–1362.
300. Smith TL, et al. Emergence of vancomycin resistance in Staphylococcus aureus. Glycopeptide-Intermediate Staphylococcus aureus Working Group. N Engl J Med. 1999;340(7):493–501.
http://www.ncbi.nlm.nih.gov/pubmed/10021469
301. Huen SC, et al. Successful use of intraperitoneal daptomycin in the treatment of vancomycin-resistant enterococcus peritonitis. Am J Kidney Dis. 2009;54(3):538–541.
302. Furgeson SB, Teitelbaum I. New treatment options and protocols for peritoneal dialysis-related peritonitis. Contrib Nephrol. 2009;163:169–176.
http://www.ncbi.nlm.nih.gov/pubmed/19494611
303. Servais A, et al. Rapid curbing of a vancomycin-resistant Enterococcus faecium outbreak in a nephrology department. Clin J Am Soc Nephrol. 2009;4(10):1559–1564.
http://www.ncbi.nlm.nih.gov/pubmed/19713290
304. Lye WC, et al. Once-daily intraperitoneal gentamicin is effective therapy for gram-negative CAPD peritonitis. Perit Dial Int. 1999;19(4):357–360.
http://www.ncbi.nlm.nih.gov/pubmed/10507817
305. Bunke M, Brier ME, Golper TA. Pseudomonas peritonitis in peritoneal dialysis patients: the Network #9 Peritonitis Study. Am J Kidney Dis. 1995;25(5):769–774.
306. Tzamaloukas AH, Murata GH, Fox L. Death associated with Pseudomonas peritonitis in malnourished elderly diabetics on CAPD. Perit Dial Int. 1993;13(3):241–242.
http://www.ncbi.nlm.nih.gov/pubmed/8369360
307. Szeto CC, et al. Clinical course of peritonitis due to Pseudomonas species complicating peritoneal dialysis: a review of 104 cases. Kidney Int. 2001;59(6):2309–2315.
308. Szeto CC, et al. Xanthomonas maltophilia peritonitis in uremic patients receiving continuous ambulatory peritoneal dialysis. Am J Kidney Dis. 1997;29(1):91–95.
309. Tzanetou K, et al. Stenotrophomonas maltophilia peritonitis in CAPD patients: susceptibility to antibiotics and treatment outcome: a report of f ve cases. Perit Dial Int. 2004;24(4):401–404.
http://www.ncbi.nlm.nih.gov/pubmed/15335157
310. Wang AY, et al. Factors predicting outcome of fungal peritonitis in peritoneal dialysis: analysis of a 9–year experience of fungal peritonitis in a single center. Am J Kidney Dis. 2000;36(6):1183–1192.
311. Wong PN, et al. Treatment of fungal peritonitis with a combination of intravenous amphotericin B and oral flucytosine, and delayed catheter replacement in continuous ambulatory peritoneal dialysis. Perit Dial Int. 2008;28(2):155–162.
312. Chan TM, et al. Treatment of fungal peritonitis complicating continuous ambulatory peritoneal dialysis with oral fluconazole: a series of 21 patients. Nephrol Dial Transplant. 1994;9(5):539–542.
http://www.ncbi.nlm.nih.gov/pubmed/8090334
313. Glancey GR, Cameron JS, Ogg CS. Peritoneal drainage: an important element in host defence against staphylococcal peritonitis in patients on CAPD. Nephrol Dial Transplant. 1992;7(7):627–631.
http://www.ncbi.nlm.nih.gov/pubmed/1323074
314. Usberti M, et al. Treatment of acute peritonitis by temporary discontinuation of dialysis and low doses of oral ciprofloxacin in patients on CAPD. Perit Dial Int. 1994;14(2):185–186.
http://www.ncbi.nlm.nih.gov/pubmed/8043679
315. Cairns HS, et al. Treatment of resistant CAPD peritonitis by temporary discontinuation of peritoneal dialysis. Clin Nephrol. 1989;32(1):27–30.
http://www.ncbi.nlm.nih.gov/pubmed/2489022
316. Kant KS, et al. Relapsing peritonitis in continuous ambulatory peritoneal dialysis (CAPD): treatment by interruption of CAPD and prolonged antibiotic therapy. Perit Dial Int. 1988;8(2):155–157.
http://www.ncbi.nlm.nih.gov/pubmed/15384799
317. Chow KM, et al. Predictive value of dialysate cell counts in peritonitis complicating peritoneal dialysis. Clin J Am Soc Nephrol. 2006;1(4):768–773.
http://www.ncbi.nlm.nih.gov/pubmed/17699285
318. Paterson AD, et al. Removal and replacement of Tenckhoff catheter at a single operation: successful treatment of resistant peritonitis in continuous ambulatory peritoneal dialysis. Lancet. 1986;2(8518):1245–1247.
319. Mitra A, Teitelbaum I. Is it safe to simultaneously remove and replace infected peritoneal dialysis catheters? Review of the literature and suggested guidelines. Adv Perit Dial. 2003;19:255–259.
http://www.ncbi.nlm.nih.gov/pubmed/14763074
320. Rutsky EA, et al. Acute pancreatitis in patients with end-stage renal disease without transplantation. Arch Intern Med, 1986;146(9):1741–1745.
http://www.ncbi.nlm.nih.gov/pubmed/3530164
321. Gupta A, et al. CAPD and pancreatitis: no connection. Perit Dial Int. 1992;12(3):309–316.
322. Caruana RJ, et al. Pancreatitis: an important cause of abdominal symptoms in patients on peritoneal dialysis. Am J Kidney Dis. 1986;7(2):135–140.
323. Quraishi ER, et al. Acute pancreatitis in patients on chronic peritoneal dialysis: an increased risk? Am J Gastroenterol.2005;100(10):2288–2293.
http://www.ncbi.nlm.nih.gov/pubmed/16181382
324. Singh S, Wadhwa N. Peritonitis, pancreatitis, and infected pseudocyst in a continuous ambulatory peritoneal dialysis patient. Am J Kidney Dis. 1987;9(1):84–86.
http://www.ncbi.nlm.nih.gov/pubmed/3812483
325. Flynn CT, Chandran PKG, Shadur CA. Recurrent pancreatitis in a patient on CAPD. Perit Dial Int. 1986;6(2):106.
326. Pannekeet MM, et al. Acute pancreatitis during CAPD in the Netherlands. Nephrol Dial Transplant. 1993;8(12):1376–1381.
327. Cheung CK, Khwaja A. Chylous ascites: an unusual complication of peritoneal dialysis. A case report and literature review. Perit Dial Int. 2008;28(3):229–231.
http://www.ncbi.nlm.nih.gov/pubmed/18474912
328. Rocklin MA, Quinn MJ, Teitelbaum I. Cloudy dialysate as a presenting feature of superior vena cava syndrome. Nephrol Dial Transplant. 2000;15(9):1455–1457.
329. Tsao YT, Chen WL. Calcium channel blocker-induced chylous ascites in peritoneal dialysis. Kidney Int. 2009;75(8):868.
http://www.ncbi.nlm.nih.gov/pubmed/19337227
330. Lew SQ. Hemoperitoneum: bloody peritoneal dialysate in ESRD patients receiving peritoneal dialysis. Perit Dial Int. 2007;27(3):226–233.
331. Tse KC, et al. Recurrent hemoperitoneum complicating continuous ambulatory peritoneal dialysis. Perit Dial Int. 2002;22(4):488–491.
http://www.ncbi.nlm.nih.gov/pubmed/12322820
332. Blumenkrantz MJ, et al. Retrograde menstruation in women undergoing chronic peritoneal dialysis. Obstet Gynecol. 1981;57(5):667–670.
http://www.ncbi.nlm.nih.gov/pubmed/7219918
333. Goodkin DA, Benning MG. An outpatient maneuver to treat bloody effluent during continuous ambulatory peritoneal dialysis (CAPD). Perit Dial Int. 1990;10(3):227–229.
http://www.ncbi.nlm.nih.gov/pubmed/2094462
334. Harnett JD, et al. Recurrent hemoperitoneum in women receiving continuous ambulatory peritoneal dialysis. Ann Intern Med. 1987;107(3):341–343.
http://www.ncbi.nlm.nih.gov/pubmed/3619223
335. Garcia-Urena MA, et al. Prevalence and management of hernias in peritoneal dialysis patients. Perit Dial Int. 2006;26(2):198–202.
http://www.ncbi.nlm.nih.gov/pubmed/16623425
336. Van Dijk CM, Ledesma SG, Teitelbaum I. Patient characteristics associated with defects of the peritoneal cavity boundary. Perit Dial Int. 2005;25(4):367–373.
http://www.ncbi.nlm.nih.gov/pubmed/16022094
337. O'Connor JP, et al. Abdominal hernias complicating continuous ambulatory peritoneal dialysis. Am J Nephrol. 1986;6(4):271–274.
338. Del Peso G, et al. Risk factors for abdominal wall complications in peritoneal dialysis patients. Perit Dial Int. 2003;23(3):249–254.
http://www.ncbi.nlm.nih.gov/pubmed/12938825
339. Digenis GE, et al. Abdominal hernias in patients undergoing continuous ambulatory peritoneal dialysis. Perit Dial Int. 1982;2(3):115–117.
340. Kopecky RT, et al. Complications of continuous ambulatory peritoneal dialysis: diagnostic value of peritoneal scintigraphy. Am J Kidney Dis. 1987;10(2):123–132.
341. Shah H, Chu M, Bargman JM. Perioperative management of periotoneal dialysis patients undergoing hernia surgery without the use of interim hemodialysis. Perit Dial Int. 2006;26(6):684–687.
http://www.ncbi.nlm.nih.gov/pubmed/17047236
342. Martinez-Mier G, et al. Abdominal wall hernias in end-stage renal disease patients on peritoneal dialysis. Perit Dial Int. 2008;28(4):391–396.
http://www.ncbi.nlm.nih.gov/pubmed/18556382
343. Crabtree JH. Hernia repair without delay in initiating or continuing peritoneal dialysis. Perit Dial Int. 2006;26(2):178–182.
http://www.ncbi.nlm.nih.gov/pubmed/16623420
344. Dejardin AS, Robert A, Goff n E. Intraperitoneal pressure in PD patients: relationship to intraperitoneal volume, body size and PD-related complications. Nephrol Dial Transplant. 2007;22(5):1437–1444.
http://www.ncbi.nlm.nih.gov/pubmed/17308323

345. Imvrios G, et al. Prosthetic mesh repair of multiple recurrent and large abdominal hernias in continuous ambulatory peritoneal dialysis patients. Perit Dial Int. 1994;14(4):338–343.
http://www.ncbi.nlm.nih.gov/pubmed/7827182
346. Orfei R, Seybold K, Blumberg A. Genital edema in patients undergoing continuous ambulatory peritoneal dialysis (CAPD). Perit Dial Int. 1984;4(4): 251–252.
347. Schleifer CR, et al. Management of hernias and Tenckhoff catheter complications in CAPD. Perit Dial Int. 1984;4(3):146–150.
348. Caporale N, Perez D, Alegre S. Vaginal leak of peritoneal dialysis liquid. Perit Dial Int. 1991;11(3):284–285.
http://www.ncbi.nlm.nih.gov/pubmed/1912027
349. Schultz SG, Harmon TM, Nachtnebel KL. Computerized tomographic scanning with intraperitoneal contrast enhancement in a CAPD patient with localized edema. Perit Dial Int. 1984;4(4):253–254.
350. Johnson BF, et al. A method for demonstrating subclinical inguinal herniae in patients undergoing peritoneal dialysis: the isotope "peritoneoscrotogram." Nephrol Dial Transplant. 1987;2(4):254–257.
351. Lew SQ. Hydrothorax: pleural effusion associated with peritoneal dialysis. Perit Dial Int. 2010;30(1):13–18.
http://www.ncbi.nlm.nih.gov/pubmed/20056973
352. Fletcher S, Turney JH, Brownjohn AM. Increased incidence of hydrothorax complicating peritoneal dialysis in patients with adult polycystic kidney disease. Nephrol Dial Transplant. 1994;9(7):832–833.
http://www.ncbi.nlm.nih.gov/pubmed/7970129
353. Nomoto Y, et al. Acute hydrothorax in continuous ambulatory peritoneal dialysis—a collaborative study of 161 centers. Am J Nephrol. 1989;9(5):363–367.
353a. Mutter D, et al. A recently described laparoscopic technique for the repair of hydrothorax appears promising. Perit Dial Int. 2011;31:692–694.
354. Kronenberg F, et al. Prevalence of dyslipidemic risk factors in hemodialysis and CAPD patients. Kidney Int Suppl. 2003(84):S113–116.
355. Grodstein GP, et al. Glucose absorption during continuous ambulatory peritoneal dialysis. Kidney Int. 1981;19(4):564–567.
http://www.ncbi.nlm.nih.gov/pubmed/7241890
356. Paniagua R, et al. Icodextrin improves metabolic and fluid management in high and high-average transport diabetic patients. Perit Dial Int. 2009;29(4):422–432.
http://www.ncbi.nlm.nih.gov/pubmed/19602608
357. Lin A, et al. Randomized controlled trial of icodextrin versus glucose containing peritoneal dialysis fluid. Clin J Am Soc Nephrol. 2009;4(11):1799–1804.
358. Steele J, et al. Lipids, lipoproteins and apolipoproteins A-I and B and apolipoprotein losses in continuous ambulatory peritoneal dialysis. Atherosclerosis. 1989;79(1):47–50.
359. Kagan A, et al. Kinetics of peritoneal protein loss during CAPD: I. Different characteristics for low and high molecular weight proteins. Kidney Int. 1990;37(3):971–979.
360. Chan MK. Gemfibrozil improves abnormalities of lipid metabolism in patients on continuous ambulatory peritoneal dialysis: the role of postheparin lipases in the metabolism of high-density lipoprotein subfractions. Metabolism. 1989;38(10):939–945.
361. Navaneethan SD, et al. HMG CoA reductase inhibitors (statins) for dialysis patients. Cochrane Database Syst Rev. 2009;(3):CD004289.
362. Sharp Collaborative Group Study of Heart and Renal Protection (SHARP): randomized trial to assess the effects of lowering low-density lipoprotein cholesterol among 9,438 patients with chronic kidney disease. Am Heart J. 2010;160(5):785–794.e10.
363. Tzamaloukas AH, et al. Peritonitis associated with intra-abdominal pathology in continuous ambulatory peritoneal dialysis patients. Perit Dial Int. 1993;13(Suppl 2):S335–337.
364. Wakeen MJ, Zimmerman SW, Bidwell D. Viscus perforation in peritoneal dialysis patients: diagnosis and outcome. Perit Dial Int. 1994;14(4):371–377.
http://www.ncbi.nlm.nih.gov/pubmed/7827188
365. Wellington JL, Rody K. Acute abdominal emergencies in patients on long-term ambulatory peritoneal dialysis. Can J Surg. 1993;36(6):522–524.
http://www.ncbi.nlm.nih.gov/pubmed/8258131
366. Spence PA, et al. Indications for operation when peritonitis occurs in patients on chronic ambulatory peritoneal dialysis. Surg Gynecol Obstet. 1985;161(5):450–452.
367. Fleisher AG, et al. Surgical complications of peritoneal dialysis catheters. Am J Surg. 1985;149(6):726–729.
http://www.ncbi.nlm.nih.gov/pubmed/4014548
368. Summers AM, et al. Single-center experience of encapsulating peritoneal sclerosis in patients on peritoneal dialysis for end-stage renal failure. Kidney Int. 2005;68(5):2381–2388.
http://www.ncbi.nlm.nih.gov/pubmed/16221244
369. Fieren MW, et al. Posttransplant encapsulating peritoneal sclerosis: a worrying new trend? Perit Dial Int. 2007;27(6):619–624.
370. Chin AI, Yeun JY. Encapsulating peritoneal sclerosis: an unpredictable and devastating complication of peritoneal dialysis. Am J Kidney Dis. 2006;47(4):697–712.
371. Campbell S, et al. Sclerosing peritonitis: identification of diagnostic, clinical, and radiological features. Am J Kidney Dis. 1994;24(5):819–825.
372. Brown MC, et al. Encapsulating peritoneal sclerosis in the new millennium: a national cohort study. Clin J Am Soc Nephrol. 2009;4(7):1222–1229.
http://www.ncbi.nlm.nih.gov/pubmed/19541815
373. Kawaguchi Y, et al. Encapsulating peritoneal sclerosis: definition, etiology, diagnosis, and treatment. International Society for Peritoneal Dialysis Ad Hoc Committee on Ultrafiltration Management in Peritoneal Dialysis. Perit Dial Int. 2000;20 Suppl 4:S43–55.
374. Johnson DW, et al. Encapsulating peritoneal sclerosis: incidence, predictors, and outcomes. Kidney Int. 2010;77(10):904–912.
http://www.ncbi.nlm.nih.gov/pubmed/20375981
375. Flynn CT, Nanson JA. Intraperitoneal insulin with CAPD - an artificial pancreas. Trans Am Soc Artif Intern Organs. 1979;25:114–117.
http://www.ncbi.nlm.nih.gov/pubmed/392876
376. Torun D, et al. Hepatic subcapsular steatosis as a complication associated with intraperitoneal insulin treatment in diabetic peritoneal dialysis patients. Perit Dial Int. 2005;25(6):596–600.
http://www.ncbi.nlm.nih.gov/pubmed/16411528
377. De Vecchi AF. Adequacy of fluid/sodium balance and blood pressure control. Perit Dial Int. 1994;14 Suppl 3:S110–116.
378. Saldanha LF, Weiler EW, Gonick HC. Effect of continuous ambulatory peritoneal dialysis on blood pressure control. Am J Kidney Dis. 1993;21(2):184–188.
http://www.ncbi.nlm.nih.gov/pubmed/8430680
379. Wang MC, et al. Blood pressure and left ventricular hypertrophy in patients on different peritoneal dialysis regimens. Perit Dial Int. 2001;21(1):36–42.
http://www.ncbi.nlm.nih.gov/pubmed/11280494
380. Ortega O, et al. Peritoneal sodium mass removal in continuous ambulatory peritoneal dialysis and automated peritoneal dialysis: influence on blood pressure control. Am J Nephrol. 2001;21(3):189–193.
381. Rodriguez-Carmona A, et al. Compared time profiles of ultrafiltration, sodium removal, and renal function in incident CAPD and automated peritoneal dialysis patients. Am J Kidney Dis. 2004;44(1):132–145.
382. Boudville NC, et al. Blood pressure, volume, and sodium control in an automated peritoneal dialysis population. Perit Dial Int. 2007;27(5):537–543.
http://www.ncbi.nlm.nih.gov/pubmed/17704444
383. Davison SN, et al. Comparison of volume overload with cycler-assisted versus continuous ambulatory peritoneal dialysis. Clin J Am Soc Nephrol. 2009;4(6):1044–1050.
http://www.ncbi.nlm.nih.gov/pubmed/19406971
384. Giacchino F, et al. Improved cell-mediated immunity in CAPD patients as compared to those on hemodialysis. Perit Dial Int. 1984;4(4):209–211.
385. Vanholder R, et al. Reduced incidence of acute renal graft failure in patients treated with peritoneal dialysis compared with hemodialysis. Am J Kidney Dis. 1999;33(5):934–940.
http://www.ncbi.nlm.nih.gov/pubmed/10213652
386. Bleyer AJ, et al. Dialysis modality and delayed graft function after cadaveric renal transplantation. J Am Soc Nephrol. 1999;10(1):154–159.
http://www.ncbi.nlm.nih.gov/pubmed/9890321
387. Vats AN et al. Pretransplant dialysis status and outcome of renal trans-plantation in North American children: a NAPRTCS Study. North American Pediatric Renal Transplant Cooperative Study. Transplantation. 2000;69(7): 1414–1419.
http://www.ncbi.nlm.nih.gov/pubmed/10798764
388. Chalem Y, et al. Access to, and outcome of, renal transplantation according to treatment modality of end-stage renal disease in France. Kidney Int. 2005;67(6):2448–2453.
http://www.ncbi.nlm.nih.gov/pubmed/15882291
389. Goldfarb-Rumyantzev AS, et al. The role of pretransplantation renal replacement therapy modality in kidney allograft and recipient survival. Am J Kidney Dis. 2005;46(3):537–549.
http://www.ncbi.nlm.nih.gov/pubmed/16129217
390. Besarab A, Golper TA. Response of continuous peritoneal dialysis patients to subcutaneous recombinant human erythropoietin differs from that of hemodialysis patients. ASAIO Trans. 1991;37(3):M395–396.
391. Maiorca R, et al. Kidney transplantation in peritoneal dialysis patients. Perit Dial Int. 1994;14 Suppl 3:S162–168.
392. Winchester JF, et al. Transplantation in peritoneal dialysis and hemodialysis. Kidney Int Suppl. 1993;40:S101–105.
393. Dutton S. Transient post-transplant ascites in CAPD patients. Perit Dial Int. 1983;3(3):164.