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### 11.1 Introduction

Pediatric end-stage renal disease (ESRD) is a rare medical disorder, but with an exponentially increasing prevalence over the past 20 years. While the preferred treatment for pediatric ESRD patients is renal transplantation, the majority of children receive dialysis prior to transplant. Peritoneal dialysis (PD) is the dialysis modality most commonly prescribed to pediatric patients with ESRD worldwide, in large part due its ease of administration to infants, children, and adolescents and the cost-effective nature of the therapy. It also still has a substantial role in the treatment of acute kidney injury (AKI) around the globe. The widespread usage of the therapy underlies the importance of medical providers caring for pediatric patients with kidney disorders to have an understanding of key aspects of the clinical application of PD. In turn, this chapter will provide an overview of PD usage in the pediatric patient, with an emphasis on catheter selection, prescription, and the diagnosis and management of PD-related complications.

### 11.2 History of Pediatric Peritoneal Dialysis

The use of the peritoneum for saline injections as fluid resuscitation in children was first described at the beginning of the twentieth century [1]. Approximately 40 years later, the first descriptions of attempts to use the peritoneum to treat

children with renal failure were published [2, 3]. Pediatric surgeons Swan and Gordon described what was considered the first successful demonstration of PD using continuous peritoneal lavage in three children with acute anuric kidney injury in 1949 [3]. The use of intermittent PD followed continuous peritoneal lavage and was soon found to be well tolerated and effective for children of all ages, in part because of the lack of need for the large extracorporeal blood circuits required of hemodialysis (HD). Subsequently, acute PD became the preferred renal replacement therapy (RRT) in children with AKI.

In the 1960s, Henry Tenckhoff developed the permanent indwelling peritoneal catheter and thereafter, the first home PD regimen was developed [4]. However, it was not until the description of continuous ambulatory peritoneal dialysis (CAPD) by Moncrief and Popovich in 1976 that PD flourished as a chronic dialytic therapy for pediatric patients [5]. CAPD was first used in a 3-year-old child in 1978 in Toronto and offered an RRT option for infants with ESRD, previously considered too small for chronic dialysis.

At the beginning of the 1980s, automated machines were developed for use with intermittent PD. Automated PD (APD) was first used in a child in 1981 by Price and Suki and became the preferred modality of pediatric programs in North America [6]. Before the 1980s, fewer than 100 pediatric patients were reported to have been treated with CAPD worldwide. Currently, the 2013 United States Renal Data System (USRDS) Annual Data Report (ADR) describes more than 900 patients <19 years on chronic PD and the International Pediatric Peritoneal Dialysis Network (IPPN) registry has voluntary enrollment of >2500 pediatric PD patients worldwide [7, 8].

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## 11.3 Principles of Peritoneal Dialysis

### 11.3.1 Physiologic and Anatomic Concepts of the Peritoneal Membrane in Children

Early studies by Putiloff more than 100 years ago revealed that the pediatric peritoneum possessed a greater surface area per scaled body weight when compared to adults [9]. Following this anatomic discovery, studies of the function of the peritoneum suggested that the pediatric peritoneal membrane was also more efficient than the adult peritoneal membrane related to the greater pediatric peritoneal surface area [10]. However, these early kinetic studies were flawed with inconsistencies in dialysis mechanics and research methods. Studies conducted over the past 25 years have instead established a similar functionality of the pediatric and adult peritoneum [11].

The peritoneal exchange process involves two transport mechanisms, diffusion and convection. Diffusion refers to the exchange of solute down a concentration gradient between two solutions separated by a semipermeable membrane. Convection refers to the movement of solute along with water across a semipermeable membrane down a pressure gradient led by ultrafiltration (UF). The recruitment of the functional peritoneum for these forms of transport in children, as in adults, is dependent upon several physiologic factors, most notably the peritoneal capillary microcirculation. It is currently proposed that the peritoneal membrane and the peritoneal microcirculation permit solute and water transport via a three-pore model [12]. The ultrasmall pores of the peritoneal capillary bed make up 1–2% of the total pore area and are involved with approximately 40% of the sodium-free water exchange. Small pores comprise 90% of the total pore area and participate primarily in low molecular weight compound exchange (i.e., urea) via diffusive transport as well as some convective transport. Finally, large pores comprise 5–7% of the total pore area and allow higher molecular weight compound (i.e., proteins, albumin) transport driven by solvent drag associated with convective forces [12].

### 11.3.2 Diffusive Transport

Studies of diffusive transport demonstrate that the rate of diffusive transfer is directly related to the functional membrane size, the dialysate concentration gradient, and a parameter known as the mass transfer area coefficient (MTAC). This parameter is essentially independent of dialysis mechanics and is an expression of the diffusive permeability of the functional peritoneal membrane in the absence of an osmotic gradient between blood and dialysate [13]. Calculation of the MTAC is quite rigorous, and the studies in which the MTAC

values were measured in pediatric patients have yielded mixed results. In the largest study, Warady et al. found that MTACs for potassium, glucose, and creatinine decreased inversely with increasing age, suggesting either an inverse relationship between age and the functional peritoneal surface area or an age-related inverse relationship with the peritoneal permeability [14]. It should be noted that although the age-related differences in the MTAC values obtained by Warady et al. were statistically significant, the differences were small and of arguable clinical importance.

In addition to the MTAC, the transmembrane concentration gradient, the peritoneal permeability, and any residual peritoneal volume also affect the rate of diffusive transport. The concentration gradient between blood and dialysate diminishes over time and is influenced by factors including cycle frequency and dialysate volume. The impact of dialysate volume rests on the principle of geometry of diffusion, which states that the larger the volume of dialysate, the longer the transmembrane concentration gradient will persist and thereby drive diffusion. In children, this principle has been a confounding variable in many early PD studies in which exchange volumes were scaled to body weight as opposed to body surface area (BSA). Given that infants have a greater ratio of BSA to body weight compared to adults, earlier studies in pediatric PD patients in which exchange volumes were scaled to body weight resulted in relatively small dialysate volumes in the youngest patients and thus an inaccurate interpretation of enhanced membrane transport capacity [10]. Finally, the presence of residual exchange volume from previous exchanges diminishes the concentration gradient and thus limits solute transport. Studies have shown that the residual volume can be substantial in children [14].

### 11.3.3 Convective Transport

Convection involves the transfer of dissolved solute across the peritoneal membrane in association with ultrafiltered water, a process also known as solvent drag. Determination of the exact fraction of solute transport which occurs due to convection is complex due to the relationship with transperitoneal UF and peritoneal membrane permeability. Transperitoneal UF is a time-dependent process, which occurs simultaneously with fluid absorption and can dilute the dialysate solute concentration, enhancing diffusive transport [15]. Therefore, mathematical models are often necessary to differentiate the amount of diffusive and convective solute transport that occurs during PD [16].

The membrane permeability is expressed as a sieving coefficient, which is the ratio of the dialysate concentration of solute and its plasma water concentration in the absence of diffusive transport. A study conducted by Pyle estimated that in a 4-h CAPD exchange with 4.25% dextrose dialy-

sis solution, the contribution of convection to urea removal was 12%, 45% for inulin, and 86% for protein [17]. Thus, in the context of solute removal, convection is thought to contribute to a lesser degree to removal of small solutes and is primarily responsible for most large solute removal [18].

### 11.3.4 Ultrafiltration

UF describes the movement of fluid across the peritoneal membrane. It is a complex process that is primarily driven by the balance between osmotic pressures created by dextrose within the dialysis solution, most notably, and the uptake of fluid into the peritoneal and lymphatic tissues. Early studies of UF in infants and younger children suggested that adequate UF was difficult to achieve because researchers noted a more rapid dissipation of the dialysate dextrose concentration in this population [19]. However, as noted above, earlier studies used exchange volumes scaled to body weight. Subsequent pediatric PD studies with exchange volumes scaled to BSA demonstrated an age-independent UF capacity [20].

### 11.3.5 Peritoneal Lymphatic Absorption

Peritoneal lymphatic absorption involves the movement of fluid into the peritoneal interstitium driven by hydraulic pressure. It has been estimated to account for nearly a 20% reduction of net UF. While there are limited studies on the contribution of lymphatic absorption to net UF in children receiving PD, studies by Schroder et al. and de Boer et al. found that net UF and lymphatic absorption were not age dependent [21, 22]. When lymphatic absorption rates were scaled to BSA in children, there were no differences when compared to adult reference values [23].

### 11.3.6 Peritoneal Dialysis for Acute Kidney Injury

PD was the first RRT used for the management of AKI in children. Although there have been many advancements in vascular access techniques as well as improvements in HD and continuous renal replacement therapies (CRRT) for the treatment of AKI, acute PD remains the modality of choice in many parts of the world, particularly in infants and small children [24]. Its advantages include its wide availability, the avoidance of the need for vascular access and large extracorporeal blood volume requirements, and its slow and well-tolerated solute and fluid removal. Furthermore, while there have been no randomized clinical trials comparing the different dialysis modalities for the treatment of pediatric AKI, observational studies have not demonstrated a difference in

mortality between children treated with PD and those treated with CRRT [25].

### 11.3.7 Indications for the Initiation of Acute Peritoneal Dialysis

In general, most of the indications for acute PD in the pediatric age group mirror those seen in adults. Although the majority of cases of AKI can be managed conservatively, severe metabolic disturbances, particularly hyperkalemia that is not responsive to medical management, mandate prompt initiation of dialysis. Additionally, there is accumulating evidence that fluid overload in the setting of AKI is associated with adverse outcomes, particularly in the pediatric population [26–30]. A multicenter, prospective study of children demonstrated that the percent fluid accumulation prior to starting RRT was significantly lower in survivors versus non-survivors [29].

### 11.3.8 Acute Peritoneal Dialysis Access

The two most commonly used accesses for acute PD are the percutaneously placed Cook catheter and the surgically placed Tenckhoff catheter. The Cook catheter offers the advantage of bedside placement by a nephrologist or intensivist via the Seldinger technique. Since only local anesthesia is required, it can be placed promptly, even in an unstable patient [31, 32]. However, its use is hampered by a very high rate of complications such as obstruction from omentum and leakage of dialysate from the catheter entry site on the abdominal wall. Chadha et al. [33] in a single-center retrospective study of infants and young children with AKI found that by day 6 of dialysis, only 46% of the Cook catheters were functioning without complications. In comparison, they found that over 90% of surgically placed Tenckhoff catheters were free of complications at the same time point. Thus, the authors suggested that if dialysis is expected to be required for more than 5 days, a Tenckhoff catheter should either be placed initially or elective replacement of the Cook catheter with a Tenckhoff catheter should be performed in a timely manner. More recently, a multipurpose percutaneous catheter (Cook Mac-Loc Multipurpose Drainage catheter) showed promising results in a small cohort of infants with AKI with a mean complication-free survival of approximately 11 days [32].

Current pediatric recommendations from the International Society of Peritoneal Dialysis (ISPD) reflect the published data and support a surgically placed Tenckhoff catheter as the “catheter of choice” for acute PD [34]. Additionally, the guidelines recommend that the catheter be inserted laparoscopically, given the reduced chance for leakage compared to placement by laparotomy. Methods to decrease the risk

of dialysate leakage are particularly important when dialysis is to be initiated emergently. There is preliminary evidence that the application of fibrin sealant (glue) at the peritoneum can be used to treat leaks that occur soon after the placement of a Tenckhoff catheter and the implementation of PD. Rusthoven et al. demonstrated in eight infants, ages 0.8–57 months that application of fibrin glue into the subcutaneous catheter tunnel through the exit site was able to successfully correct leaks that occurred within 48 h of initiating therapy while using a single-cuff Tenckhoff catheter [35]. Additionally, Sojo et al. demonstrated that application of fibrin glue to the peritoneal cuff suture at the time of implantation reduced the incidence of leakage in the early postoperative period [36]. Dialysate leakage only occurred in 9% of the fibrin glue group versus 57% of the control group.

### 11.3.9 Acute Peritoneal Dialysis Prescription

Much like the chronic PD prescription (see below), there are four main components of the acute PD prescription: fill volume, dialysate composition, dwell times, and total length of dialysis therapy. The initial fill volume of 10 ml/kg should be used for at least the first 24–48 h to minimize the risk of dialysate leakage through the catheter insertion site. The fill volume can be gradually increased to a target volume of 30–40 ml/kg (800–1100 ml/m<sup>2</sup> BSA) to achieve adequate fluid and solute removal goals [34]. Whether performing manual exchanges or automated exchanges, the initial dwell time should be at least 30–60 min, with gradual prolongation as the patient is stabilized and fluid and solute removal targets are achieved. Acute PD should be continuous for the first 1–3 days with gradual shortening of the total daily duration of dialysis as tolerated by the patient [34]. Given the use of rapid exchanges, often with higher dialysate dextrose concentrations, along with the continuous nature of the dialysis, close monitoring of electrolytes is mandatory. Patients undergoing acute PD are at risk for hypernatremia secondary to sodium sieving, a consequence of disproportionately greater water to sodium transport via aquaporin-mediated pores, and hyperglycemia secondary to substantial dextrose absorption [37].

### 11.3.10 Causes of End-Stage Renal Disease in Children

While a variety of disorders result in ESRD during childhood, approximately one half are congenital in origin and one half are acquired lesions. The largest source of data on primary diagnoses comes from the dialysis registry of the North American Pediatric Renal Trials and Collaborative Studies (NAPRTCS) [38]. The most common diagnoses in

**Table 11.1** Primary renal disorders [38]

	N	%
<i>All dialysis patients</i>	7039	100.0
<i>Primary diagnosis</i>		
FSGS	1016	14.4
A/hypo/dysplastic kidney	998	14.2
Obstructive uropathy	888	12.6
Reflux nephropathy	244	3.5
SLE nephritis	226	3.2
HUS	216	3.1
Chronic GN	214	3.0
Polycystic disease	201	2.9
Congenital nephrotic syndrome	182	2.6
Prune belly	144	2.0
Medullary cystic disease	140	2.0
Idiopathic crescentic GN	130	1.8
Familial nephritis	130	1.8
MPGN—Type I	116	1.6
Pyelo/interstitial nephritis	101	1.4
Cystinosis	99	1.4
Renal infarct	90	1.3
Berger's (IgA) nephritis	86	1.2
Henoch-Schönlein nephritis	67	1.0
MPGN—Type II	64	0.9
Wilms tumor	55	0.8
Wegener's granulomatosis	49	0.7
Drash syndrome	39	0.6
Other systemic immunologic disease	37	0.5
Oxalosis	32	0.5
Membranous nephropathy	29	0.4
Sickle cell nephropathy	21	0.3
Diabetic GN	10	0.1
Other	887	12.6
Unknown	528	7.5

*FSGS* focal segmental glomerulosclerosis, *SLE* systemic lupus erythematosus, *HUS* hemolytic uremic syndrome, *GN* glomerulonephritis, *MPGN* membranoproliferative glomerulonephritis, *IgA* immunoglobulin A

the registry's cohort of >7000 patients are focal segmental glomerulosclerosis, congenital aplasia/hypoplasia/dysplasia, and obstructive uropathy, accounting for 14.4, 14.2 and 12.6% of cases, respectively (Table 11.1) [38].

### 11.3.11 Indications and Contraindications for Chronic Peritoneal Dialysis

In many cases, the choice of PD as the initial dialytic modality is based on patient/family preference and center philosophy. In addition, however, an important aspect of the selection process for PD is a thorough evaluation of the family's social, psychological, and economic background so as to best determine the likely ability of the family to cope with the "burden of care" associated with the provision of home dialysis therapy on a daily basis [39]. An evaluation of the

parent or caregiver's educational background and learning capacity is also desirable so that a realistic assessment of their ability to process the information necessary to carry out home dialysis can be made. All that being said, the absolute indications for PD in children with ESRD are a very small patient, lack of a vascular access, and the presence of contraindications to anticoagulation [40–45].

With recognition that the performance of PD requires a patent abdominal cavity and a functioning peritoneal membrane, the only absolute contraindications to PD consists of the presence of one of the following: omphalocele, gastroschisis, bladder extrophy, diaphragmatic hernia, or an obliterated peritoneal cavity. While the lack of an appropriate caregiver for home therapy is a relative contraindication, the presence of a colostomy, gastrostomy, ureterostomy, and/or pyelostomy or a ventriculoperitoneal shunt does not preclude chronic PD [46].

### 11.3.12 Chronic Peritoneal Dialysis Usage

Data on the use of chronic PD by children with ESRD are derived from a number of different registries from around the globe. The NAPRTCS has demonstrated that chronic PD was the initial dialysis modality prescribed to 62.9% of more than 7000 patients, most commonly through the use of APD (see below) [1]. Noteworthy is the fact that 85% of the children <5 years were prescribed chronic peritoneal dialysis (CPD) versus 51% of those >12 years. The USRDS ADR provides data on all patients 0–19 years old and on dialysis, in contrast to only those cared for in pediatric centers, as reported by the NAPRTCS [8]. In turn, the ADR recently reported that only 28% of 1410 incident patients received PD, in contrast to the 49% who were prescribed HD. A decade-old compilation of data from 12 European registries revealed that 34% of the incident patients received PD (vs. 48% HD), whereas European data published in 2013 showed that 50% of the pediatric patients initiated therapy with PD and 34% with HD, with patient/family choice and patient size being the most influential factors regarding modality selection [47, 48].

## 11.4 Chronic Peritoneal Dialysis Prescription

### 11.4.1 Choice of Modality

In centers where APD is freely available and without financial constraints, this PD modality is generally preferred in the pediatric population. It is the modality used by 82.9% of the infant, child, and adolescent PD patients in the USA [49, 50]. The preference for APD is in large part because of the manual nature and daytime requirements of CAPD, in con-

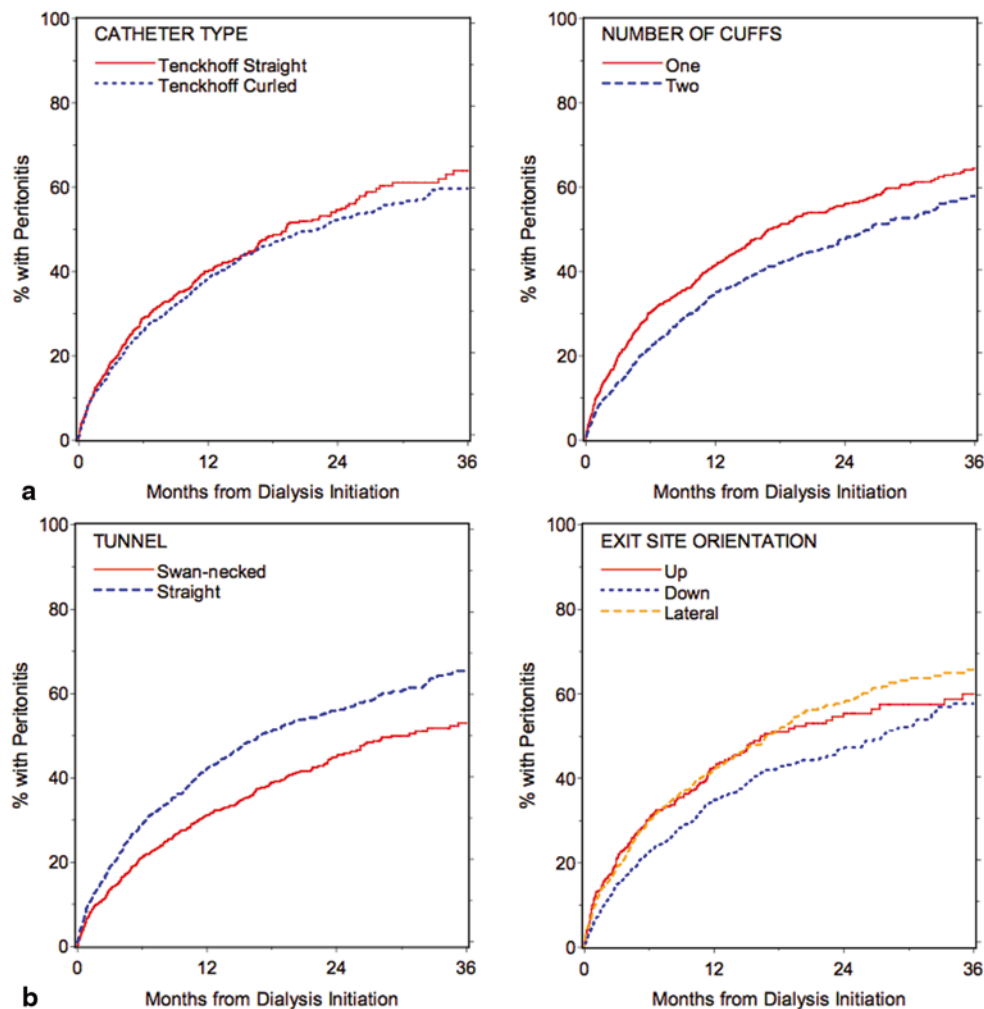
trast to the nocturnal provision of multiple exchanges with APD, as well as the greater ability to tailor the PD prescription to the patient's needs. The peritoneal membrane transport capacity can also influence the PD modality choice, with those individuals demonstrating high membrane transport capacity more likely to require either continuous cycling peritoneal dialysis (CCPD) in which the dialysate is left in the abdomen following nocturnal cycling or nocturnal intermittent PD (NIPD) in which the abdomen is left dry during the daytime to achieve adequate UF. Recent data from the IPPN have, in fact, revealed that 37.4% of the patients were prescribed CCPD, 37.5% NIPD, and 23.8% CAPD. Finally, tidal PD, an APD variant in which only a portion of each exchange is drained until completion of the entire dialysis session, has been used to alleviate “drain pain” in children on PD [51].

### 11.4.2 Chronic Peritoneal Dialysis Access

The most important consideration for the successful placement and long-term function of a Tenckhoff catheter in the pediatric patient with ESRD is the experience of the surgeon [52]. This can be particularly problematic at centers caring for a small number of patients, where the need to provide chronic dialysis to very young children may be a rare event. Because of the importance of the access and the desire for the outcome of placement to be complication free, surgical placement should ideally be limited to only a few surgeons per center and on rare occasion, it may be preferable to refer the patient to another, more experienced center for access placement, in a manner similar to what has been recommended for vascular access [53].

There are a variety of configurations of pediatric-sized Tenckhoff catheters available from several manufactures (Baxter, USA; Medionics, Canada; Covidien, USA). Current data from the IPPN show that ~70% and 30% of 2290 pediatric PD patients (median age 10.5 years) have curled versus straight catheters, respectively [54]. A majority of these catheters (86%) had two cuffs with a downward or lateral exit-site orientation. Although there are limited data available to permit determination of the “best” configuration, observational data from the NAPRTCS suggest that a dual-cuffed, swan-neck (allows for downward facing exit site) catheter is associated with a reduction in infectious complications compared with other catheter configurations (Fig. 11.1) [55]. This information is especially relevant in infants and young children because of their increased rates of peritonitis compared to older children [55]. The exit site should also be placed outside of the groin area and away from the diaper region and any potential gastrostomy site, with the superficial cuff located approximately 2 cm from the skin surface [56]. While the catheter must be immobilized to minimize the risk





**Fig. 11.1** Time to first peritonitis infection by peritoneal dialysis (PD) access characteristics [38]

of exit-site trauma, no sutures should be placed at the exit site as they increase the risk of bacterial colonization [57].

A somewhat controversial aspect of catheter placement is the decision whether or not to routinely perform an omentectomy. A survey of pediatric surgeons indicated that an omentectomy was performed routinely in 53% of the participating centers at the time of catheter placement [58]. The basis for its performance in children is that catheter obstruction (usually due to omental wrapping) is second only to peritonitis in terms of major catheter complications in this age group [59]. Ironically, most of the data in support of omentectomy come from the adult literature [60]. One retrospective study of children by Cribbs et al. demonstrated a decreased risk of early catheter failure with omentectomy, and Rinaldi et al. noted improved catheter survival with omentectomy, especially in children less than 2 years of age [61, 62]. Additionally, in a retrospective study of 92 pediatric patients (mean age 5 years), Conlin et al. demonstrated that the outflow obstruction rate was 5% in patients who received an omentectomy

versus 10% in patients who did not [63]. Finally, another single-center retrospective review of 207 patients (median age 10 years) revealed that failure to perform an omentectomy was associated with a higher rate of catheter failure [64].

One additional unique consideration for catheter placement in the pediatric age group is the timing and location of placement relative to the common need for gastrostomy tube (G-tube) placement in order to accommodate nutritional requirements (see below). As noted above, the catheter exit site should ideally be placed at a distance (often the contralateral side) from the site of a current or potential G-tube to decrease the risk of contamination and possible peritonitis. Likewise, it is recommended that when possible, the PD catheter should be placed either simultaneously or after placement of a G-tube to avoid contamination of the peritoneum from gastric contents [65]. When the catheter placement precedes G-tube placement, the latter procedure should take place under prophylactic antibiotic and antifungal therapy. Whereas percutaneous G-tube placement while on PD

should not be performed due to the high risk of infection and mechanical failure; placement via an open Stamm gastrostomy procedure is possible [66]. Conversely, PD catheter placement is possible in the setting of a well-established G-tube with no increased risk of bacterial or fungal peritonitis [67–69].

Ideally, the use of a PD catheter for chronic dialysis should be postponed until the exit site is completely healed with dressing changes avoided during the first postoperative week, unless they are required because of soiling or bleeding. Generally, a minimum of 2–3 weeks delay is preferred, although the exact timing will vary from patient to patient with complete healing taking up to 6 weeks in some patients [57].

A quality transformation effort, *Standardizing Care to Improve Outcomes in Pediatric End Stage Renal Disease* (SCOPE), is currently examining the impact of standardizing PD catheter care on infectious complications in 29 pediatric dialysis centers in the USA [70].

### 11.4.3 Peritoneal Dialysis Solutions

The peritoneal dialysis solutions (PDS) used are the same for children and adults. The composition of the PD solutions is aimed at promoting removal of water and solute waste products while maintaining electrolyte homeostasis and the long-term stability of the peritoneal membrane. Therefore, standard PD solutions contain an osmotic agent necessary to maintain a transmembrane osmotic gradient, a buffer to correct metabolic acidosis, magnesium, calcium, and electrolytes. However, over the past decade, we have come to better understand the harmful effects of prolonged exposure of the peritoneum to the high glucose, lactate, and osmolar concentrations found in many of the commercially available PD solutions. Glucose concentrations used in PD solutions are particularly nonphysiologic, and the glucose degradation products (GDPs) generated during the heat sterilization process are directly toxic to the peritoneal membrane and vasculature. These have been shown to induce production of and crosslink with advanced glycation end products (AGE), all of which can contribute to diabetiform vascular changes, ultrafiltration failure, and purification loss of the peritoneum [71].

The biocompatibility of PD solutions is of particular significance in the pediatric population who might require frequent, repeated, and a longer overall duration of exposure to PD solutions over a lifetime.

New PD solutions, which offer greater biocompatibility, are now available and offer lower GDP concentrations and are more pH neutral with a bicarbonate or bicarbonate/lactate buffer (Table 11.2). In children, the use of biocompatible PD solutions has been associated with equally good acidosis

control and better membrane preservation [72]. Additionally, the neutral pH of these PD solutions has been shown to induce less pain at peritoneal filling.

Icodextrin, an isosmotic glucose polymer, is also a commercially available alternative PD solution which offers a slower, sustained UF by means of colloid osmotic pressure [72, 73]. A 7.5% icodextrin solution produces sustained UF over a 12–14 h dwell similar to that obtained with a 3.86% glucose-containing solution [74]. The use of icodextrin in pediatric patients has been shown to significantly increase solute and water removal during long dwell periods and is generally used in instances of UF failure. Long-term experience with icodextrin is, however, limited in pediatrics, and the results in infants have been poorer than in older children [72, 75]. Its application should be generally limited to one exchange per day [73, 76].

### 11.4.4 Fill Volume

As mentioned previously, initial recommendations that fill volumes in children be prescribed per kilogram of body weight led to PD prescriptions with small, suboptimal fill volumes, particularly in infants and young children. The small fill volumes lead to premature loss of the osmotic gradient and impaired UF capacity [20]. Given the age-independent relationship between the peritoneal membrane surface area and BSA, it was subsequently determined that the fill volumes in children should be based on BSA rather than weight [77]. In turn, the KDOQI clinical practice guidelines recommend that for children >2 years of age, the fill volume should be 1100–1200 ml/m<sup>2</sup> BSA (Fig. 11.2) [78]. This volume can be increased to an upper limit of 1400 ml/m<sup>2</sup> as tolerated to achieve maximum recruitment of the peritoneal membrane vascular pore area [12]. In children <2 years of age, a lower fill volume of 600–800 ml/m<sup>2</sup> is recommended based more on tolerance [79]. Measurement of the intraperitoneal pressure (IPP) can be useful in determining the optimum PD volume to maintain a target IPP between 7 and 14 cm H<sub>2</sub>O [80]. A fill volume that is too large and generates an IPP of >18 cm H<sub>2</sub>O may contribute to complications such as abdominal pain, dyspnea, hydrothorax, hernia formation, GERD, and loss of UF due to increased lymphatic uptake.

### 11.4.5 Dwell Time

Determination of the length of each dialysis exchange, or dwell time, should also be selected based upon individual patient needs [12]. Long dwell times, as seen with CAPD, can be associated with insufficient UF, but are best for achieving phosphate purification. Most children, however, are treated

**Table 11.2** Characteristics of currently available peritoneal dialysis solutions (PDS) [12]. (Source: Used with permission from Fischbach [12])

	Manufacturer	Potential drawbacks	Potential benefits
Lactate buffered: Balance <sup>®</sup> , Gambrosol Tri <sup>®</sup>	Fresenius Gambro	More physiological pH, but not neutral. Local and systemic glucose exposure	Lower GDP levels More physiological pH (5.5–6.5) Improved-peritoneal membrane biocompatibility Preserved-membrane defense
Lactate/bicarbonate buffered: Physioneal <sup>®</sup>	Baxter	Local and systemic glucose exposure. Does not eliminate peritoneal lactate exposure	Lower GDP levels More physiological pH (7.4) Improved-peritoneal membrane biocompatibility Preserved-membrane defense Reduced-infusion pain
Bicarbonate buffered: BicaVera <sup>®</sup>	Fresenius	Local and systemic glucose exposure	Lower GDP levels More physiological pH(7.4) More peritoneal membrane biocompatibility Preserved-membrane defense Improved correction of acidosis
Lactate-buffered glucose containing Dianeal <sup>®</sup>	Baxter	Low pH (5.5) High GDP content Poor peritoneal membrane biocompatibility Infusion pain Local and systemic glucose exposure	Ease of manufacture; low cost
Icodextrin-containing; lactate buffered	Baxter	Hypersensitivity Low pH (5.5) Licensed for single daily use only Lactate containing	Sustained ultrafiltration Preservation of RRF Hypertonic glucose replacement Reduced hyperglycemia Improved short-term systemic hemodynamic profile Desirable effects on metabolic profile and body composition
Amino-acid containing: Nutrineal <sup>®</sup>	Baxter	Low pH (6.7) Licensed for single daily use only (avoid exacerbation of uremic symptoms and acidosis)	No GDPs Avoid systemic and peritoneal glucose exposure Peritoneal membrane protection Enhance nutrition

GDP glucose degradation product, RRF residual renal function

with APD, in which the dwell times can be manipulated to optimize both solute and fluid removal.

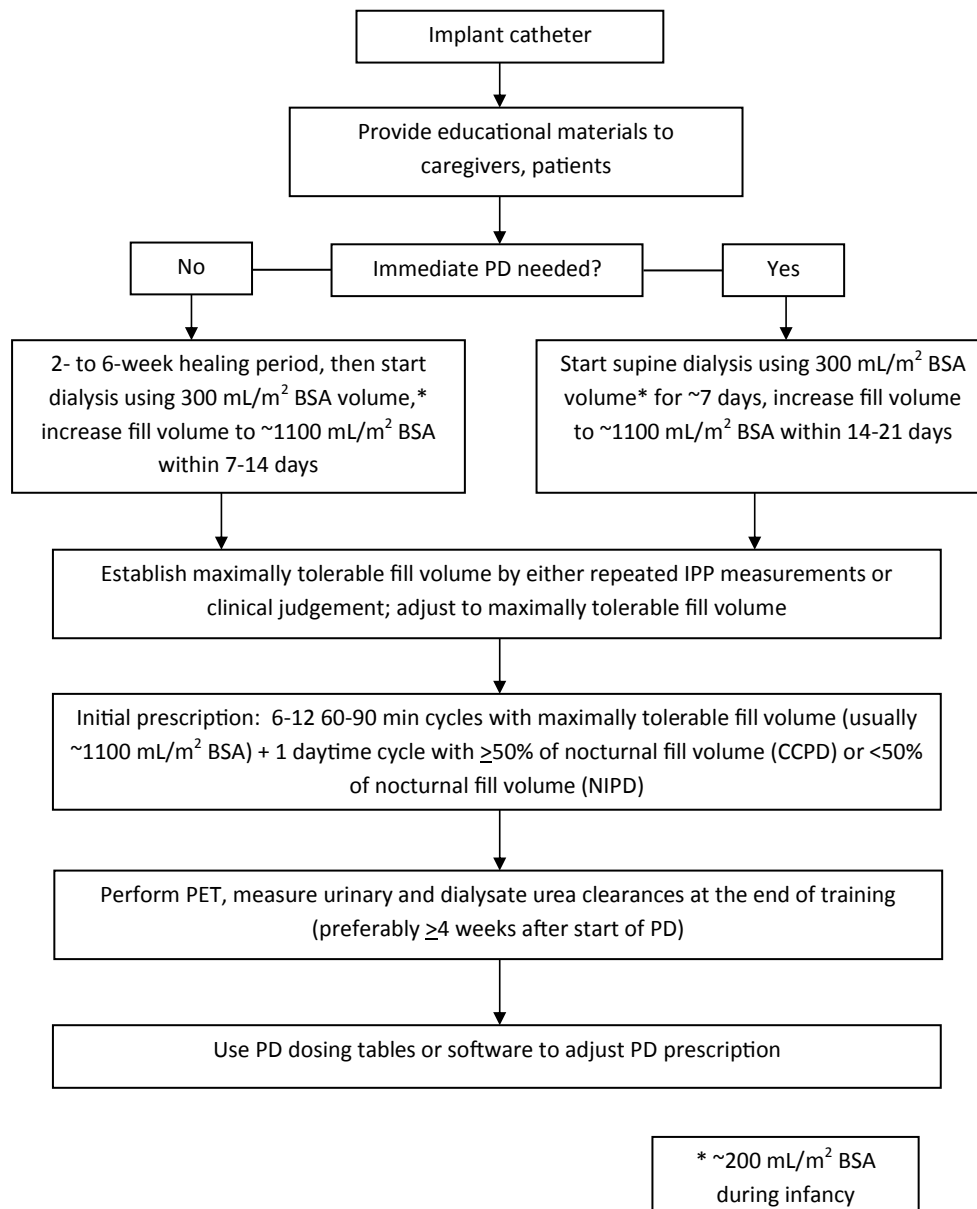
The majority of children on APD are prescribed an initial regimen of 6–12 exchanges over 8–10 h per night with a daytime fill volume for patients on CCPD consisting of approximately 50% of the nighttime volume [38, 81] (Fig. 11.2). Thus, the typical choice for an initial APD dwell time is approximately 1 h. However, the dwell time (as well as the fill volume) should be reevaluated periodically to ensure that the prescription is meeting the needs of the individual patient in terms of solute clearance and UF. The use of multiple short exchanges can also result in hyponatremia secondary to sodium sieving, as noted in the discussion of AKI management [37]. Additionally, short exchanges can contribute to poor phosphate clearance and the inherent increased risk for cardiovascular and metabolic bone disorders in children [12]. A clinically useful way to individualize dwell duration in pediatric patients on CPD is by determining the peritoneal

membrane transport capacity with the peritoneal equilibration test (PET) .

#### 11.4.6 Peritoneal Equilibration Test Evaluation in Children

PET was developed by Twardowski et al. to evaluate peritoneal membrane function in the clinical arena [82]. Reference curves were constructed for adults based upon the kinetics of solute equilibration of creatinine and glucose between dialysate and plasma (D:P ratio), which made possible the categorization of adult PD patients into those with high, high average, low average, and low peritoneal membrane solute transport rates. Thus, PET data provide information which can guide the application of the most appropriate dialysis prescription in terms of dwell time [81]. It is recommended that an initial PET evaluation should be conducted



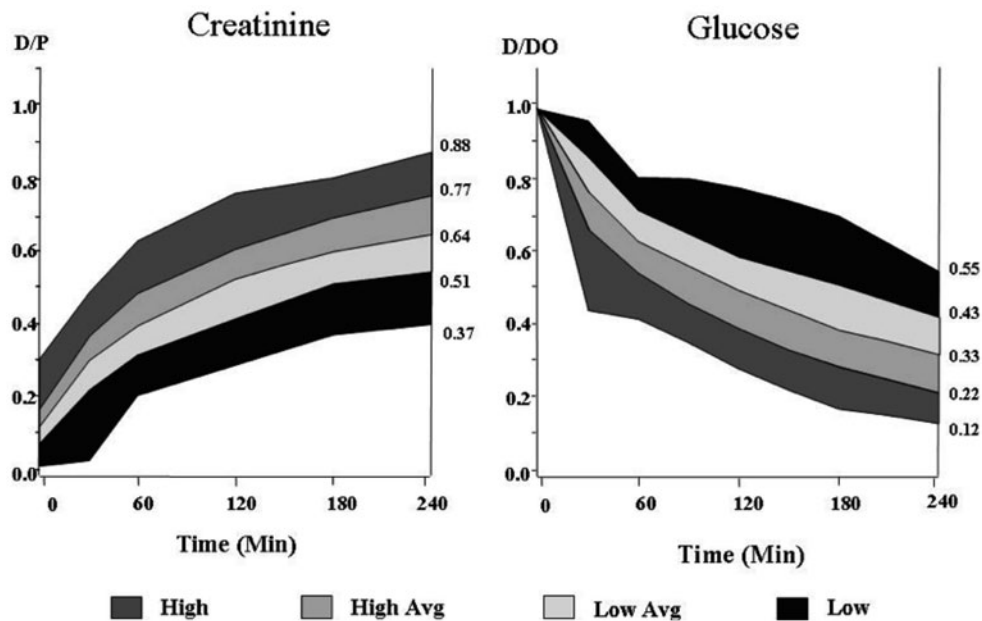


**Fig. 11.2** Algorithm for initiation of chronic peritoneal dialysis (PD). (Source: Used with permission from Warady [42])

4–8 weeks following the initiation of PD for most accurate results [81, 83, 84]. The PET should be repeated following clinical events known to alter peritoneal membrane transport (i.e., peritonitis), following the clinical demonstration of UF deterioration (i.e., worsening hypertension, increasing need for hypertonic dialysate, persistent fluid overload, erythropoietin-stimulating agents (ESA)-resistant anemia), or following worsening solute removal.

The PET for adults was designed to be performed during a 4-h dwell with a 2-L fill volume. However, in children, appreciation of the age-independent relationship between BSA and the peritoneal membrane surface area mandates use of a fill volume scaled to BSA when conducting studies of pediatric peritoneal transport kinetics. The Pediatric Peritoneal

Dialysis Study Consortium (PPDSC) and the Mid-European Pediatric Peritoneal Dialysis Study Group (MEPPS) have both conducted large multicenter trials using a 2.5% or 2.3–2.4% dextrose dialysis solution and a fill volume of 1000–1100 mL/m<sup>2</sup> BSA to develop reference kinetic data (i.e., D:P ratios and D:D0 ratios), which can be used to categorize a pediatric patient's peritoneal membrane transport characteristics and contribute to the prescription process (Fig. 11.3) [5, 85]. Commercially available modeling programs which use these data for PD prescription have been validated in children [86]. In infants <2 years of age, however, the fill volume used for the PET evaluation is typically the current clinically prescribed volume due to the infant's limited tolerance of high fill volumes. The 4-h PET procedure in children



**Fig. 11.3** Pediatric peritoneal equilibration test (PET) reference curves for creatinine and glucose

is described in Table 11.3. Finally, recent research suggests that a 2-h PET procedure in children performs as well as the 4-h procedure. The shortened version of the study is less labor intensive and less costly than the 4-h procedure [87, 88].

**Table 11.3** The peritoneal equilibration test (PET) procedure in children

Dwell period: 4 h
Fill volume: 1100 mL/m <sup>2</sup> BSA <sup>a</sup>
2.3–2.4% anhydrous glucose dialysis solution (Europe)
2.5% dextrose dialysis solution (North America, Japan)
Test exchange after prolonged (8 h) dwell, if possible as follows:
Drain the overnight dwell
Record the length of the dwell and the volume drained. Also note the dextrose concentration and volume infused
Infuse the calculated fill volume, note infusion time
Keep patient in supine position
Drain < 10% of dialysate solution into the drain bag at 0, 120, and 240 min
Invert bag for mixing and obtain sample. Reinfuse any remaining effluent
Obtain blood sample after 120 min
Measure creatinine and glucose in each sample
Calculate dialysate to plasma (D/P) creatinine and dialysate glucose to baseline dialysate glucose (D/DO) concentration ratios
Determine transporter state by comparing creatinine and glucose equilibration curves with pediatric reference percentiles

BSA body surface area

<sup>a</sup> In early infancy, volume may not be tolerable; in these cases, conduct PET with regular daily exchange volume for evaluation

#### 11.4.7 Peritoneal Dialysis Adequacy

PD adequacy in children should be characterized by a prescription that results in the achievement of optimal UF, sodium balance, and solute clearance so that the patient's clinical status is characterized by sufficient growth, blood pressure control, avoidance of hypo- or hypervolemia, and adequate psychomotor development. Care must always be taken to individualize therapy with these considerations in mind because of the absence of definitive data linking patient outcome to measures such as urea clearance in pediatrics [87].

Despite the appropriate emphasis on clinical parameters, small solute (urea) clearance has historically been used as a surrogate for PD adequacy. Urea removal scaled for the urea volume of distribution or  $Kt/V_{\text{urea}}$ , is this recommended measure of urea clearance and PD adequacy [89]. This measure includes evaluation of urea removal via residual renal function combined with urea removal via dialysis. Whereas data in adults support a target total (peritoneal and kidney)  $Kt/V_{\text{urea}}$  of at least 1.7/week, there is very little data correlating  $Kt/V_{\text{urea}}$  with outcomes in pediatrics [90, 91]. In turn, current KDOQI guidelines support the recommendation that the pediatric population should use clearance goals that meet or exceed current KDOQI adult standards, or a minimal delivered total  $Kt/V_{\text{urea}}$  of at least 1.8/week [81]. The total weekly  $Kt/V_{\text{urea}}$  is calculated as follows:

$$\text{Weekly } Kt/V_{\text{urea}} = \frac{(D_{\text{ur}} \cdot V_{\text{D}})(U_{\text{ur}} \cdot V_{\text{u}})}{P_{\text{ur}} \cdot V} \cdot 7$$

where  $D_{ur}$ ,  $U_{ur}$ , and  $P_{ur}$  are the dialysate, urinary, and plasma concentrations of urea,  $V_D$  and  $V_U$  are the 24-h dialysate and urine volumes, and  $V$  is the urea distribution volume. The ability to accurately estimate  $V$ , or the patient's total body water (TBW) volume in children can be accomplished by using validated gender specific formulas [92]. The formulae are as follows:

$$\text{Boys : TBW} = 0.10 \times (\text{HtWt})^{0.68} - 0.37 \times \text{weight}$$

$$\text{Girls : TBW} = 0.14 \times (\text{HtWt})^{0.65} - 0.35 \times \text{weight}$$

It is recommended that a 24-h collection of urine and dialysis fluid should be obtained within the first month after the initiation of dialysis for  $Kt/V_{urea}$  evaluation. Following this initial clearance, pediatric PD patients should reassess  $Kt/V_{urea}$  a minimum of twice yearly or following any change in clinical status that could alter dialysis performance and may mandate a modification of the dialysis prescription.

Finally, and as mentioned above, fluid removal is also an important measure of PD adequacy and should be optimized to prevent fluid overload. Overhydration represents an important clinical problem in pediatric PD patients because of its contribution to hypertension and an increased risk of adverse cardiovascular outcomes [93]. Data from the NAPRTCS have demonstrated that 57% of 4000 pediatric PD patients in the registry had hypertension. In another study, 68% of the pediatric patients on chronic PD were found to have left ventricular hypertrophy [94, 95]. Therefore, routine monitoring of volume status including repeated assessment of target dry weight and measurement of residual urine output are important components of PD adequacy evaluation. A modified PET using 4.25% dextrose dialysate can be used to evaluate UF kinetics in the patient with evidence of UF failure [90]. In patients experiencing decreased UF, therapeutic interventions may include use of a long daytime exchange

with icodextrin, an increase in the number of exchanges or an increased overall treatment time, and/or an increase in the dialysate glucose concentration [12]. Failure of these interventions to optimize fluid management may mandate transition to HD.

## 11.5 Infectious Complications of Peritoneal Dialysis

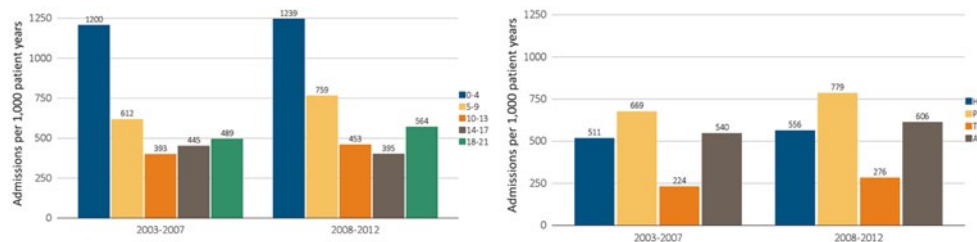
Records from the USRDS reveal that infection is the most common cause for hospitalization among children receiving PD with a hospitalization rate of >600 admissions per 1000 patient-years [8] (Fig. 11.4). Infection is also the most common reason for modality change for pediatric patients on PD [38].

### 11.5.1 Peritonitis

Peritonitis remains the most significant complication of chronic PD in the pediatric population, and one that can compromise the long-term viability of PD as a dialytic option. However, reductions in peritonitis rates have been reported in children in association with treatment of *Staphylococcus aureus* nasal carriage or application of topical antibiotics (e.g., mupirocin or gentamicin) at the catheter exit site, as well as with technical developments such as disconnect systems and the flush-before-fill technique [96–98]. The practice of prolonged training with an emphasis on hand hygiene has also proven beneficial [70].

#### 11.5.1.1 Incidence

Data from the NAPRTCS include information on 4248 episodes of peritonitis, which reflects an annualized peritonitis rate of 0.64 or 1 infection every 18.8 patient-months [38]. Similar to previous reports, the data reveal an inverse rela-



**Fig. 11.4** One-year adjusted rates of hospitalization for infection in pediatric patients (from day 90), by age and modality. (Source: From U.S. Renal Data System, USRDS [8]. The data reported have been supplied by the United States Renal Data System (USRDS). The interpretation

and reporting of these data are the responsibility of the author(s) and in no way should be seen as an official policy or interpretation of the US government. <http://www.usrds.org/faq.aspx>

**Table 11.4** Peritoneal dialysis (PD) peritonitis rates in pediatric patients [38]

	No. of episodes	Years of FU	Annualized rate		Expected months between infections	
			Rate	95 % CI	Months	95 % CI
Total	4248	6658	0.64	(0.62–0.66)	18.8	(18.3–19.4)
<i>Age</i>						
0–1 years	938	1193	0.79	(0.74–0.84)	15.3	(14.3–16.3)
2–5 years	552	821	0.67	(0.62–0.73)	17.9	(16.5–19.5)
6–12 years	1345	2145	0.63	(0.59–0.66)	19.1	(18.2–20.2)
>12 years	1413	2499	0.57	(0.54–0.59)	21.2	(20.2–22.4)
<i>Catheter</i>						
Straight	1180	1668	0.71	(0.67–0.75)	17.0	(16.0–18.0)
Curled	2697	4137	0.65	(0.63–0.68)	18.4	(17.7–19.1)
Presternal	225	420	0.54	(0.47–0.61)	22.4	(19.8–25.8)
<i>Cuff</i>						
One	2553	3440	0.74	(0.71–0.77)	16.2	(15.6–16.8)
Two	1620	2912	0.56	(0.53–0.58)	21.6	(20.6–22.7)
<i>Tunnel</i>						
Swan necked/curved	1161	2317	0.50	(0.47–0.53)	23.9	(22.6–25.4)
Straight	2995	4032	0.74	(0.72–0.77)	16.2	(15.6–16.8)
<i>Exit-site orientation</i>						
Up	702	850	0.83	(0.76–0.89)	14.5	(13.5–15.7)
Down	1181	2221	0.53	(0.50–0.56)	22.6	(21.4–23.9)
Lateral	1828	2466	0.74	(0.71–0.78)	16.2	(15.5–17.0)

CI confidence interval, FU follow up

tionship between the age of the patient and the peritonitis rate, with the highest rate (annualized rate: 0.79 or 1 infection every 15.3 months) seen in patients 0–1 year of age, in contrast to an annualized rate of 0.57 or 1 infection every 21.2 patient-months, in children more than 12 years of age (Table 11.4).

Noteworthy is a significant improvement in the overall annualized infection rate from 0.79 in 1992–1996 to 0.44 in recent years, likely related to the prophylactic measures described above, in addition to a greater use of PD catheters characterized by two cuffs and a downward pointed exit site and prophylactic antibiotic usage at the time of PD catheter placement and prior to invasive procedures, as described in the *Consensus Guidelines for the Prevention and Treatment of Catheter-Related Infections and Peritonitis in Pediatric Patients Receiving Peritoneal Dialysis* [57].

### 11.5.1.2 Presentation and Diagnosis

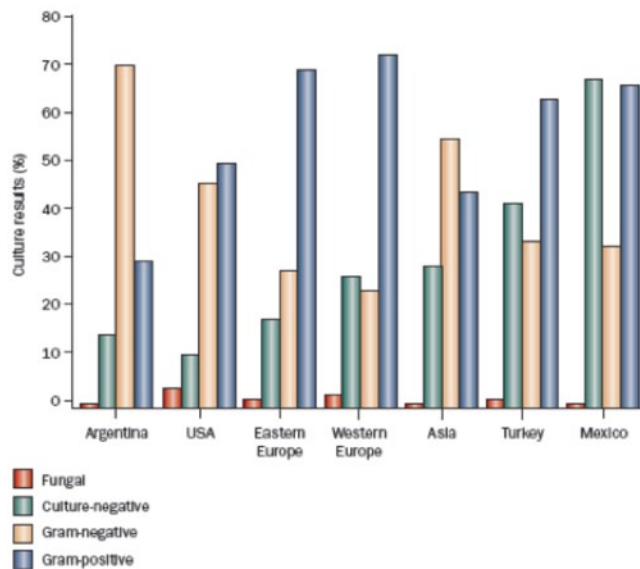
Peritonitis should be suspected in any patient with abdominal pain and/or cloudy PD effluent, accompanied by an effluent white blood cell (WBC) count  $>100/\text{mm}^3$  and at least 50% polymorphonuclear leukocytes. For patients on APD, the PD effluent WBC count should be obtained from a dwell instilled for at least 1–2 h. In those cases, the percentage of neutrophils may meet diagnostic criteria when the total WBC count does not, and still be indicative of peritonitis.

### 11.5.1.3 Microbiology

The successful prophylaxis of exposure to *S. aureus* has resulted in a decrease in the incidence of gram-positive peritonitis and an associated increase in the incidence of gram-negative infections. Data from the International Pediatric Peritoneal Dialysis Registry (IPPR) revealed that 44% of peritonitis episodes in children are secondary to gram-positive organisms, 25% to gram-negative organisms, 2% to fungi, and a remarkable 31% are culture negative [99]. Of the gram-positive organisms, coagulase-negative Staphylococci are most common. A significant worldwide variation in the microbiology of peritonitis and in the frequency of culture-negative infections was also evident in the IPPR analysis (Fig. 11.5).

### 11.5.1.4 Treatment

Empiric antibiotic treatment should be initiated as soon as the diagnosis of peritonitis is considered and an effluent sample is obtained for culture and Gram's stain using a standardized technique [57]. The antibiotic regimen used should provide coverage for both gram-positive and gram-negative organisms and should be given by the intraperitoneal route to ensure immediate bioavailability. The recently published pediatric peritonitis treatment guidelines propose empiric monotherapy with the fourth-generation cephalosporin cefepime where available, and in the absence of a history of methicillin-resistant *S. aureus* (Fig. 11.6). An alternative ap-



**Fig. 11.5** Distribution of peritonitis culture results according to geographical regions. (Source: Used with permission from Schaefer [125])

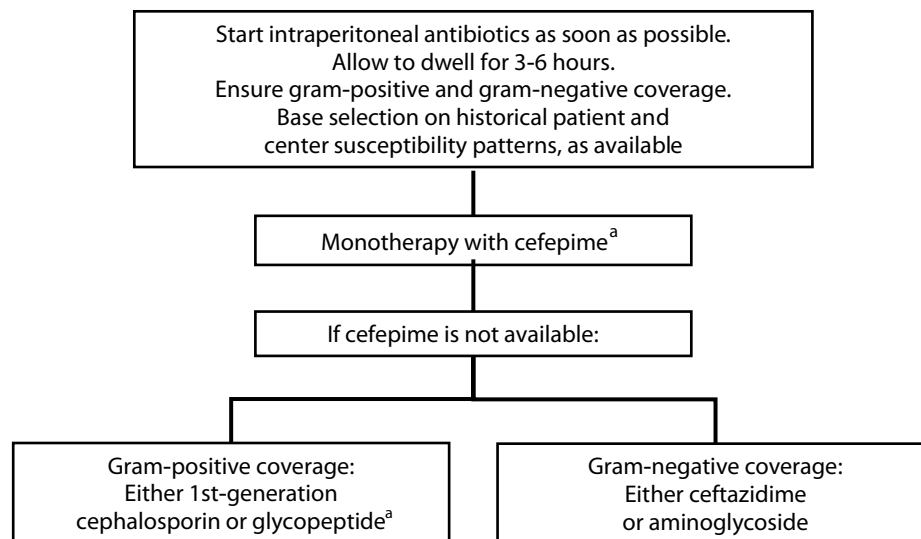
proach consists of the use of a first-generation cephalosporin or a glycopeptide (e.g., vancomycin) combined with ceftazidime or an aminoglycoside. In all cases, empiric therapy should be guided by the center-specific susceptibility pattern, and maintenance antibiotic therapy should be instituted once the antibiotic susceptibilities of the cultured organism have been determined (Table 11.5). In the IPPR analysis,

89% of the episodes were followed by full functional recovery, with only an 8.1% incidence of technique failure and <1% mortality rate [99].

#### 11.5.1.5 Exit-Site and Tunnel Infection

Exit-site and tunnel infections are significant causes of peritonitis and catheter failure [100]. Early efforts to reduce the incidence of these infections include the provision of intravenous prophylactic antibiotics (usually a first-generation cephalosporin) within 60 min *prior* to the incision for PD catheter placement, immobilization of the catheter without a suture following catheter placement to decrease the risk for exit-site trauma, and limited postoperative dressing changes [57, 101]. Subsequent measures should include delayed onset of dialysis (if possible) to decrease the risk of dialysate leakage, regular cleansing of the exit site with an antiseptic solution followed by the application of a topical antibiotic, proper hand hygiene, and regular exit-site and tunnel monitoring using a standardized scoring system to permit early detection of infection [57, 101, 102]. The combined use of topical mupirocin and sodium hypochlorite solution for exit-site care has been associated with reduced rates of catheter-related infections and prolonged catheter survival in children [103].

The diagnosis of an exit-site infection does not require a positive culture, as long as there is purulent discharge from the sinus tract or marked pericatheter swelling, redness, or tenderness. However, *S. aureus* does account for the major-



- a. If the center's rate of methicillin-resistant *Staphylococcus aureus* (MRSA) exceeds 10%, or if the patient has history of MRSA infection or colonization, glycopeptide (vancomycin or teicoplanin) should be added to cefepime or should replace the first-generative cephalosporin for gram-positive coverage.

Glycopeptide use can also be considered if the patient has a history of severe allergy to penicillins and cephalosporins.

**Fig. 11.6** Empiric therapy of peritonitis. (Source: Used with permission from Warady [57])



**Table 11.5** Antibiotic dosing recommendations for the treatment of peritonitis [57]. (Source: Used with permission from Warady [57])

Antibiotic type	Therapy type		
	Continuous <sup>a</sup>		Intermittent therapy <sup>a</sup>
	Loading dose	Maintenance dose	
<i>Aminoglycosides (IP)</i> <sup>b</sup>			
Gentamicin	8 mg/L	4 mg/L	
Netilmycin	8 mg/L	4 mg/L	Anuric: 0.6 mg/kg
Tobramycin	8 mg/L	4 mg/L	Non-anuric: 0.75 mg/kg
Amikacin	25 mg/L	12 mg/L	
<i>Cephalosporins (IP)</i>			
Cefazolin	500 mg/L	125 mg/L	20 mg/kg
Cefepime	500 mg/L	125 mg/L	15 mg/kg
Cefotaxime	500 mg/L	250 mg/L	30 mg/kg
Ceftazidime	500 mg/L	125 mg/L	20 mg/kg
<i>Glycopeptides (IP)</i> <sup>c</sup>			
Vancomycin	1000 mg/L	25 mg/L	30 mg/kg; repeat dosing: 15 mg/kg every 3–5 days
Teicoplanin <sup>d</sup>	400 mg/L	20 mg/L	15 mg/kg every 5–7 days
<i>Penicillins (IP)</i> <sup>b</sup>			
Ampicillin	–	125 mg/L	–
<i>Quinolones (IP)</i>			
Ciprofloxacin	50 mg/L	25 mg/L	–
<i>Others</i>			
Aztreonam (IP)	1000 mg/L	250 mg/L	–
Clindamycin (IP)	300 mg/L	150 mg/L	–
Imipenem-cilastin (IP)	250 mg/L	50 mg/L	–
Linezolid (PO)	< 5 Years: 30 mg/kg daily, divided into 3 doses 5–11 Years: 20 mg/kg daily, divided into 2 doses ≥ 12 Years: 600 mg/dose, twice daily		
Metronidazole (PO)	30 mg/kg daily, divided into 3 doses (maximum: 1.2 g daily)		
Rifampin (PO)	10–20 mg/kg daily, divided into 2 doses (maximum: 600 mg daily)		
<i>Antifungals</i>			
Fluconazole (IP, IV, or PO)	6–12 mg/kg every 24–48 h (maximum: 400 mg/daily)		
Caspofungin (IV only)	70 mg/m <sup>2</sup> on day 1 (maximum: 70 mg daily)	50 mg/m <sup>2</sup> daily (maximum: 50 mg daily)	

IP intraperitoneal, PO oral, IV intravenously

<sup>a</sup>For continuous therapy, the exchange with the loading dose should dwell for 3–6 h; all subsequent exchanges during the treatment course should contain the maintenance dose. For intermittent therapy, the dose should be applied once daily in the long-dwell, unless otherwise specified

<sup>b</sup>Aminoglycosides and penicillins should not be mixed in dialysis fluid because of the potential for inactivation

<sup>c</sup>In patients with residual renal function, glycopeptide elimination may be accelerated. If intermittent therapy is used in such a setting, the second dose should be time-based on a blood level obtained 2–4 days after the initial dose. Re-dosing should occur when the blood level is < 15 mg/L for vancomycin, or < 8 mg/L for teicoplanin. Intermittent therapy is not recommended for patients with residual renal function unless serum levels of the drug can be monitored in a timely manner

<sup>d</sup>Teicoplanin is not currently available in the United States

ity of infections, followed by *Enterococci*, *Pseudomonas*, *E. coli*, and *Klebsiella*. Antibiotic therapy is typically given by the oral route, should be based on the susceptibilities of the cultured organism and should be 2–4 weeks in duration and at least 7 days following resolution of the infection [57].

## 11.6 Noninfectious Complications of Peritoneal Dialysis

### 11.6.1 Sclerosing Encapsulating Peritonitis

Sclerosing encapsulating peritonitis (SEP) is a rare and extremely serious complication of PD characterized by the

presence of continuous, intermittent, or recurrent bowel obstruction associated with gross thickening of the peritoneum [104,105]. The cause of the disorder is likely multifactorial, but virtually all affected patients have received a prolonged course of PD, and most have evidence of high peritoneal permeability. The incidence in children has been documented to be 6.6% and 22% in those patients receiving PD for > 5 years and > 10 years, respectively [106]. The diagnosis is typically suspected based on clinical findings and confirmed by computed tomography (CT) or ultrasound. Treatment consists of cessation of PD and aggressive nutritional management in all, along with immunosuppressive therapy and surgery as deemed necessary [104, 107].

### 11.6.2 Hernia

The incidence of hernias in children receiving PD (8–57%) is inversely proportional to the patient's age, with the highest percentage noted in children <1 year of age [108]. The most common presentation is a painless swelling and 75% requires surgical correction followed by no/low volume dialysis for several days.

### 11.6.3 Hydrothorax

Hydrothorax, or the accumulation of dialysis fluid within the pleural space, occurs in 1.6–10% of patients. Contributing factors include increased IPP, a pleura-peritoneal pressure gradient, and congenital diaphragmatic defects. Whereas a presenting feature may consist of shortness of breath following the initiation of PD, the diagnosis may also be made when the displaced dialysate is evident on routine chest X-ray (usually right sided). Diagnostic techniques include scintigraphy or thoracentesis, with the detection of pleural fluid with a high dextrose concentration (>300 mg/dL) characteristic of dialysate consistent with the diagnosis. CT or magnetic resonance imaging (MRI) may be used to investigate a site of communication. Common approaches to management include permanent or temporary cessation of PD, decreased exchange volume, obliteration of the pleural space, or surgical repair of a diaphragmatic defect [109, 110].

## 11.7 Nutritional Management of Children on Peritoneal Dialysis

Malnutrition is a common complication in children who receive dialysis as a result of anorexia and poor intestinal absorption of nutrients [111]. Moreover, the protein needs of children with ESRD are increased when taking into account the protein losses that occur via the peritoneum. The KDOQI pediatric nutrition guidelines suggest following parameters of nutritional status and growth including dietary intake, length or height, height velocity, estimated dry weight, BMI, and head circumference based upon the child's age for PD patients [112]. Children on PD should receive at least 100% of the estimated energy requirements for normal age-dependent needs, with additional intake as need to address growth requirements [112]. The KDOQI guidelines also suggest that children on PD should receive a dietary protein intake of 100% of the daily recommended intake for ideal body weight, as well as additional protein intake to address protein losses via dialysis. Current recommendations for daily dietary protein intake are shown in Table 11.6.

Special attention must also be directed to the dietary management of sodium, potassium, and phosphorus. Infants, especially those with obstructive uropathy and poor renal tu-

**Table 11.6** Recommended daily protein intake (DPI) [112]. (Source: Used with permission from [112])

Age (months)	DPI (g/kg/day)
0–6	1.8
7–12	1.5
1–3	1.3
4–13	1.1
14–18	1.0

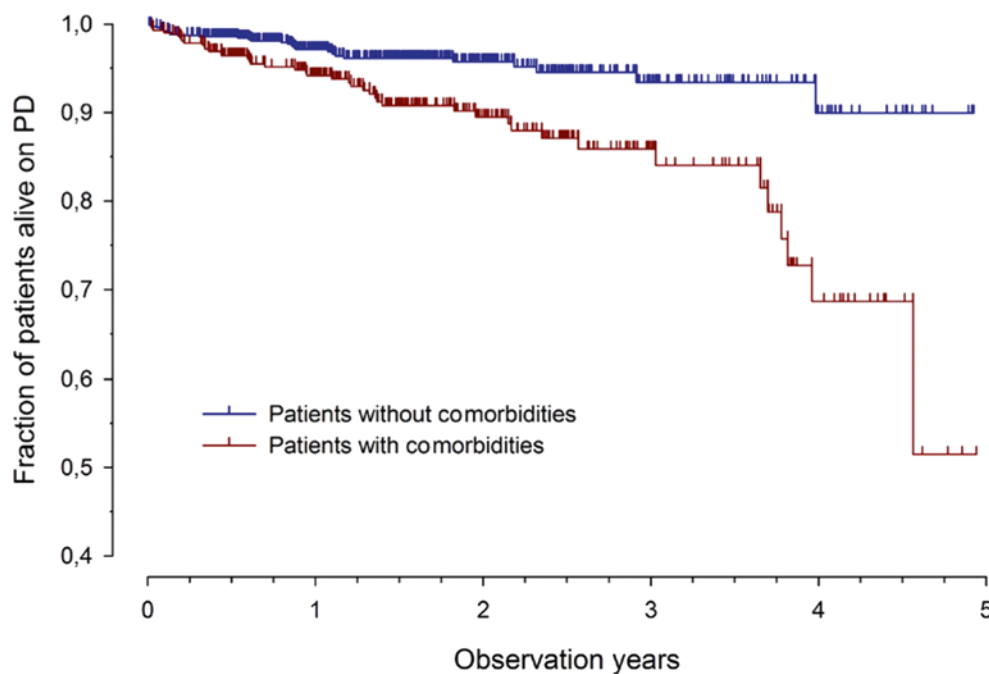
*DPI* daily protein intake

bular function, can have significant sodium losses via the dialysate and the native kidneys. Therefore, some infants require sodium supplementation to maintain total body sodium levels. A lack of supplementation can result in hyponatremia, severe central nervous system (CNS) manifestations, and poor growth [113]. Aggressive use of potassium-binding agents in infant formula can result in hypokalemia, a potential risk factor for peritonitis [114]. Finally, some infants on PD experience hypophosphatemia due to the use of low phosphorous infant formulas [115]. In those pediatric PD patients who experience hyperphosphatemia, management of dietary phosphorous intake is of critical importance because of the impact phosphorous has on bone turnover and linear growth, in addition to cardiovascular health.

## 11.8 Technique and Patient Survival

The need to terminate PD for reasons other than transplantation is most commonly the result of infectious complications. A NAPRTCS study found that 20% of patients transitioned from PD to HD over a 6-year period, the result of infection in 43% of the cases, followed by UF failure, patient/family choice and access failure as the most frequent reasons [116]. More recent data from the IPPN registry demonstrated similar findings with the following reasons for discontinuation of PD: kidney transplantation (60%), technique failure and switch to HD (20%), death (7%), and partial recovery of renal function (2%) [7].

Compared with adults, patient survival is excellent in children on PD, and there has been a steady improvement in mortality rates over the last 20 years, particularly in the youngest patients. Recent data from the USRDS based on children undergoing either chronic PD or HD have revealed mortality rates of 112.2 and 83.4 per 1000 person-years in those initiating dialysis in 1990–1994 and 2005–2010, respectively [117]. The highest mortality rates are seen in those patients who receive PD during the first year of life [118, 119]. Data from the Australia and New Zealand Dialysis and Transplant (ANZDATA) registry and the Italian dialysis registry are similar, but with more pronounced differences between various age groups [120, 121]. In addition to young age itself, an important predictor of mortality is the presence of nonrenal disease [122]. Data from the



**Fig. 11.7** Cumulative survival of pediatric peritoneal dialysis (PD) patients by absence or presence of at least one comorbidity. (Source: Used with permission from Neu [123])

IPPN have demonstrated that pediatric patients on chronic PD with a comorbidity (i.e., neurocognitive impairment or congenital heart disease) had a significantly lower survival rate compared with patients not having a comorbidity [123] (Fig. 11.7).

## References

- Blackfan K, Macey K. The intraperitoneal injection of saline solution. *Am J Dis Child*. 1918;15:19.
- Bloxsum A, Powell N. The treatment of acute temporary dysfunction of the kidneys by peritoneal irrigation. *Pediatrics*. 1948;1:52–7.
- Swan H, Gordon HH. Peritoneal lavage in the treatment of anuria in children. *Pediatrics*. 1949;4(5):586–95. (PubMed PMID: 15391042. Epub 1949/11/01).
- Tenckhoff H, Schechter H. A bacteriologically safe peritoneal access device. *Transac—Am Soc Artif Intern Organs*. 1968;14:181–7. (PubMed PMID: 5701529. Epub 1968/01/01).
- Popovich R, Moncrief J, Decherd J, et al. The definition of a novel portable/wearable equilibrium dialysis technique. *Transac—Am Soc Artif Intern Organs*. 1976;5.
- Price CG, Suki WN. Newer modifications of peritoneal dialysis: options in the treatment of patients with renal failure. *Am J Nephrol*. 1981;1(2):97–104. (PubMed PMID: 7349048. Epub 1981/01/01).
- Schaefer F, Borzych-Duzalka D, Azocar M, Munarriz RL, Sever L, Aksu N, et al. Impact of global economic disparities on practices and outcomes of chronic peritoneal dialysis in children: insights from the International Pediatric Peritoneal Dialysis Network Registry. *Perit Dial Int*. 2012;32(4):399–409. (PubMed PMID: 22859840. Pubmed Central PMCID: PMC3524840. Epub 2012/08/04).
- U.S. Renal Data System, USRDS. Annual data report: atlas of chronic kidney disease and end-stage renal disease in the United States. Bethesda: National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases; 2013.
- Putiloff P. Materials for the study of the laws of growth of the human body in relation to the surface areas of different systems: the trial on Russian subjects of planigraphic anatomy as a means for exact anthropometry; one of the problems of anthropology. Report of Dr. P. Putiloff at the meeting of the Siberian Branch of the Russian Geographic Society; 1884.
- Esperanca M, Collins D. Peritoneal dialysis efficiency in relation to body weight. *J Pediatr Surg*. 1966;1:162–9.
- Gruskin A, Lerner G, Fleischmann L. Developmental aspects of peritoneal dialysis kinetics. In: Fine R, editor. *Chronic ambulatory peritoneal dialysis and chronic cycling peritoneal dialysis in children topics in renal medicine*. Vol. 4. Boston: Martinus Nijhoff Publishing; 1987. pp. 33–45.
- Fischbach M, Warady BA. Peritoneal dialysis prescription in children: bedside principles for optimal practice. *Pediatr Nephrol* (Berlin, Germany). 2009;24(9):1633–42. (quiz 40, 42. PubMed PMID: 18807074. Pubmed Central PMCID: PMC2719743. Epub 2008/09/23).
- Nolph KD. Peritoneal anatomy and transport physiology. In: Drukker W, Parson FM, Maher JF, editors. *Replacement of renal function by dialysis*. Second edition. Boston. Martinus Nijhoff Publishing; 1983. p. 440.
- Warady BA, Alexander SR, Hossli S, Vonesh E, Geary D, Watkins S, et al. Peritoneal membrane transport function in children receiving long-term dialysis. *J Am Soc Nephrol*. 1996;7(11):2385–91. (PubMed PMID: 8959629. Epub 1996/11/01).
- Leypoldt JK. Solute transport across the peritoneal membrane. *J Am Soc Nephrol*. 2002;13(Suppl 1):S84–91. (PubMed PMID: 11792767. Epub 2002/01/17).
- Waniewski J. Mathematical models for peritoneal transport characteristics. *Perit Dial Int*. 1999;19(Suppl 2):S193–201. (PubMed PMID: 10406518. Epub 1999/07/16).
- Pyle W. *Mass transfer in peritoneal dialysis*. Austin: Univertisy of Texas; 1987.

18. Goldstein SL. Adequacy of dialysis in children: does small solute clearance really matter? *Pediatr Nephrol* (Berlin, Germany). 2004;19(1):1–5. (PubMed PMID: 14673636. Epub 2003/12/16).
19. Balfé J, Hanning R, Vigneus A. A comparison of peritoneal water and solute movement in younger and older children on CAPD. In: Fine R, Schaefer F, Mehls O, editors. *CAPD in children*. New York: Springer-Verlag; 1985. pp. 14–9.
20. Kohaut EC, Waldo FB, Benfield MR. The effect of changes in dialysate volume on glucose and urea equilibration. *Perit Dial Int*. 1994;14(3):236–9. (PubMed PMID: 7948234. Epub 1994/01/01).
21. Schroder CH, Reddingius RE, van Dreumel JA, Theeuwes AG, Monnens LA. Transcapillary ultrafiltration and lymphatic absorption during childhood continuous ambulatory peritoneal dialysis. *Nephrol Dial Transplant*. 1991;6(8):571–3. (PubMed PMID: 1956557. Epub 1991/01/01).
22. de Boer AW, van Schaijk TC, Willems HL, de Haan AF, Monnens LA, Schroder CH. Follow-up study of peritoneal fluid kinetics in infants and children on peritoneal dialysis. *Perit Dial Int*. 1999;19(6):572–7. (PubMed PMID: 10641778. Epub 2000/01/21).
23. Schaefer F, Fischbach M, Heckert KH et al. Hydrostatic intraperitoneal pressure in children on peritoneal dialysis. *Perit Dial Int*. 1996;16(S2):S79.
24. Phadke KD, Dinakar C. The challenges of treating children with renal failure in a developing country. *Perit Dial Int*. 2001;21(Suppl 3):S326–9. (PubMed PMID: 11887846. Epub 2002/03/13).
25. Bunchman TE, McBryde KD, Mottes TE, Gardner JJ, Maxvold NJ, Brophy PD. Pediatric acute renal failure: outcome by modality and disease. *Pediatr Nephrol* (Berlin, Germany). 2001;16(12):1067–71. (PubMed PMID: 11793102. Epub 2002/01/17).
26. Arikan AA, Zappitelli M, Goldstein SL, Naipaul A, Jefferson LS, Loftis LL. Fluid overload is associated with impaired oxygenation and morbidity in critically ill children. *Pediatr Crit Care Med*. 2011;13(3):253–8. (PubMed PMID: 21760565).
27. Goldstein SL. Overview of pediatric renal replacement therapy in acute kidney injury. *Semi Dial*. 2009;22(2):180–4. (PubMed PMID: 19426425. Epub 2009/05/12).
28. Goldstein SL, Devarajan P. Pediatrics: acute kidney injury leads to pediatric patient mortality. *Nat Rev Nephrol*. 2012;6(7):393–4. (PubMed PMID: 20585319).
29. Sutherland SM, Zappitelli M, Alexander SR, Chua AN, Brophy PD, Bunchman TE, et al. Fluid overload and mortality in children receiving continuous renal replacement therapy: the prospective pediatric continuous renal replacement therapy registry. *Am J Kidney Dis*. 2011;55(2):316–25. (PubMed PMID: 20042260).
30. Payen D, de Pont AC, Sakr Y, Spies C, Reinhart K, Vincent JL. A positive fluid balance is associated with a worse outcome in patients with acute renal failure. *Crit Care* (London, England). 2008;12(3):R74. (PubMed PMID: 18533029).
31. Bunchman TE. Acute peritoneal dialysis access in infant renal failure. *Perit Dial Int*. 1996;16(Suppl 1):S509–11. (PubMed PMID: 8728258).
32. Auron A, Warady BA, Simon S, Blowey DL, Srivastava T, Musharaf G, et al. Use of the multipurpose drainage catheter for the provision of acute peritoneal dialysis in infants and children. *Am J Kidney Dis*. 2007;49(5):650–5. (PubMed PMID: 17472847).
33. Chadha V, Warady BA, Blowey DL, Simckes AM, Alon US. Tenckhoff catheters prove superior to cook catheters in pediatric acute peritoneal dialysis. *Am J Kidney Dis*. 2000;35(6):1111–6. (PubMed PMID: 10845825).
34. Cullis B, Abdelraheem M, Abrahams G, Balbi A, Cruz DN, Frishberg Y, et al. Peritoneal dialysis for acute kidney injury. *Perit Dial Int*. 2014 (7–8);34(5):494–517. (PubMed PMID: 25074995. Pubmed Central PMCID: PMC4114667. Epub 2014/07/31).
35. Rusthoven E, van de Kar NA, Monnens LA, Schroder CH. Fibrin glue used successfully in peritoneal dialysis catheter leakage in children. *Perit Dial Int*. 2004;24(3):287–9. (PubMed PMID: 15185778. Epub 2004/06/10).
36. Sojo ET, Grosman MD, Monteverde ML, Bailez MM, Delgado N. Fibrin glue is useful in preventing early dialysate leakage in children on chronic peritoneal dialysis. *Perit Dial Int*. 2004;24(2):186–90. (PubMed PMID: 15119641. Epub 2004/05/04).
37. Rusthoven E, Krediet RT, Willems HL, Monnens LA, Schroder CH. Sodium sieving in children. *Perit Dial Int*. 2005;25(Suppl 3):S141–2. (PubMed PMID: 16048281. Epub 2005/07/29).
38. North American Pediatric Renal Trials and Collaborative Studies (NAPRTCS). Annual dialysis report. Rockville: Emmes Corporation; 2011.
39. Warady BA, Alexander SR, Watkins S, Kohaut E, Harmon WE. Optimal care of the pediatric end-stage renal disease patient on dialysis. *Am J Kidney Dis*. 1999;33(3):567–83. (PubMed PMID: 10070923. Epub 1999/03/10).
40. Alexander SR, Salusky IB, Warady BA, Watkins SL. Peritoneal dialysis workshop: pediatrics recommendations. *Perit Dial Int*. 1997;17(Suppl 3):S25–7. (PubMed PMID: 9304653. Epub 1997/01/01).
41. Salusky IB, Holloway M. Selection of peritoneal dialysis for pediatric patients. *Perit Dial Int*. 1997;17(Suppl 3):S35–7. (PubMed PMID: 9304656. Epub 1997/01/01).
42. Warady B, Schaefer F, Alexander S, Firaneck C, Mujais S. Care of the pediatric patient on peritoneal dialysis. Clinical process for optimal outcomes. McGaw Park: Baxter Healthcare; 2004. p. 89.
43. Al-Hermi BE, Al-Saran K, Secker D, Geary DF. Hemodialysis for end-stage renal disease in children weighing less than 10 kg. *Pediatr Nephrol* (Berlin, Germany). 1999;13(5):401–3. (PubMed PMID: 10412860. Epub 1999/07/21).
44. Kovalski Y, Cleper R, Krause I, Davidovits M. Hemodialysis in children weighing less than 15 kg: a single-center experience. *Pediatr Nephrol* (Berlin, Germany). 2007;22(12):2105–10. (PubMed PMID: 17940806. Epub 2007/10/18).
45. Eisenstein I, Tarabeih M, Magen D, Pollack S, Kassir I, Ofer A, et al. Low infection rates and prolonged survival times of hemodialysis catheters in infants and children. *Clin J Am Soc Nephrol: CJASN*. 2011;6(4):793–8. (PubMed PMID: 21127138. Pubmed Central PMCID: PMC3069371. Epub 2010/12/04).
46. Dolan NM, Borzych-Duzalka D, Suarez A, Principi I, Hernandez O, Al-Akash S, et al. Ventriculoperitoneal shunts in children on peritoneal dialysis: a survey of the International Pediatric Peritoneal Dialysis Network. *Pediatr Nephrol* (Berlin, Germany). 2013;28(2):315–9. (PubMed PMID: 22972407. Epub 2012/09/14).
47. Watson AR, Hayes WN, Vondrak K, Ariceta G, Schmitt CP, Ekim M, et al. Factors influencing choice of renal replacement therapy in European paediatric nephrology units. *Pediatr Nephrol* (Berlin, Germany). 2013;28(12):2361–8. (PubMed PMID: 23843162. Epub 2013/07/12).
48. Strazdins V, Watson AR, Harvey B. Renal replacement therapy for acute renal failure in children: European guidelines. *Pediatr Nephrol* (Berlin, Germany). 2004;19(2):199–207. (PubMed PMID: 14685840. Pubmed Central PMCID: PMC1766478. Epub 2003/12/20).
49. Verrina E, Cappelli V, Perfumo F. Selection of modalities, prescription, and technical issues in children on peritoneal dialysis. *Pediatr Nephrol* (Berlin, Germany). 2009;24(8):1453–64. (PubMed PMID: 18521632. Pubmed Central PMCID: PMC2697927. Epub 2008/06/04).
50. Collins AJ, Foley RN, Herzog C, Chavers B, Gilbertson D, Herzog C, et al. US Renal Data System 2012 Annual Data Report. *Am J Kidney Dis*. 2013;61(1 Suppl 1):A7, e1–476. (PubMed PMID: 23253259. Epub 2013/01/04).



51. Warady BA, Bohl V, Alon U, Hellerstein S. Symptomatic peritoneal calcification in a child: treatment with tidal peritoneal dialysis. *Perit Dial Int.* 1994;14(1):26–9. (PubMed PMID: 8312409. Epub 1994/01/01).
52. Watson AR, Gartland C. Guidelines by an Ad, Hoc European Committee for elective chronic peritoneal dialysis in pediatric patients. *Perit Dial Int.* 2001;21(3):240–4. (PubMed PMID: 11475338. Epub 2001/07/28).
53. NKF-K/DOQI. Clinical practice guidelines for vascular access update 2000. *Am J Kidney Dis.* 2001;37(1 Suppl 1):S137–81. (PubMed PMID: 11229969).
54. Borzych-Dazalka D, Patel H, Flynn J, White C, Hooman N, Brophy P, et al. Peritoneal dialysis access revision, management and outcome—findings from the International Pediatric Peritoneal Dialysis Network (IPPN). *Perit Dial Int.* 2014 2014; Abstracts from the Annual Dialysis Conference:S18.
55. Zappitelli M, Goldstein S, Symons J, Somers M, Baum M, Brophy P, et al. Protein and calorie prescription for children and young adults receiving continuous renal replacement therapy: a report from the prospective pediatric continuous renal replacement therapy registry group. *Crit Care Med.* 2008;36(12):3239–45.
56. Chadha V, Jones LL, Ramirez ZD, Warady BA. Chest wall peritoneal dialysis catheter placement in infants with a colostomy. *Adv Perit Dial.* 2000;16:318–20. (PubMed PMID: 11045319. Epub 2000/10/25).
57. Warady BA, Bakkaloglu S, Newland J, Cantwell M, Verrina E, Neu A, et al. Consensus guidelines for the prevention and treatment of catheter-related infections and peritonitis in pediatric patients receiving peritoneal dialysis: 2012 update. *Perit Dial Int.* 2012;32(Suppl 2):S32–86. (PubMed PMID: 22851742. Pubmed Central PMCID: PMC3524923. Epub 2012/08/08).
58. Washburn KK, Currier H, Salter KJ, Brandt ML. Surgical technique for peritoneal dialysis catheter placement in the pediatric patient: a North American survey. *Adv Perit Dial.* 2004;20:218–21. (PubMed PMID: 15384830. Epub 2004/09/24).
59. White CT, Gowrishankar M, Feber J, Yiu V. Clinical practice guidelines for pediatric peritoneal dialysis. *Pediatric nephrology* (Berlin, Germany). 2006;21(8):1059–66. (PubMed PMID: 16819641. Epub 2006/07/05).
60. Nicholson ML, Burton PR, Donnelly PK, Veitch PS, Walls J. The role of omentectomy in continuous ambulatory peritoneal dialysis. *Perit Dial Int.* 1991;11(4):330–2. (PubMed PMID: 1751599).
61. Cribbs RK, Greenbaum LA, Heiss KF. Risk factors for early peritoneal dialysis catheter failure in children. *J Pediatr Surg.* 2010;45(3):585–9. (PubMed PMID: 20223324. Epub 2010/03/13).
62. Rinaldi S, Sera F, Verrina E, Edefonti A, Gianoglio B, Perfumo F, et al. Chronic peritoneal dialysis catheters in children: a fifteen-year experience of the Italian Registry of Pediatric Chronic Peritoneal Dialysis. *Perit Dial Int.* 2004;24(5):481–6. (PubMed PMID: 15490990. Epub 2004/10/20).
63. Conlin MJ, Tank ES. Minimizing surgical problems of peritoneal dialysis in children. *J Urol.* 1995;154(2 Pt 2):917–9. (PubMed PMID: 7609212. Epub 1995/08/01).
64. Phan J, Stanford S, Zaritsky JJ, DeUgarte DA. Risk factors for morbidity and mortality in pediatric patients with peritoneal dialysis catheters. *J Pediatr Surg.* 2013;48(1):197–202. (PubMed PMID: 23331815. Epub 2013/01/22).
65. Rees L, Brandt ML. Tube feeding in children with chronic kidney disease: technical and practical issues. *Pediatr Nephrol* (Berlin, Germany). 2010;25(4):699–704. (PubMed PMID: 19949817).
66. von Schnakenburg C, Feneberg R, Plank C, Zimmering M, Arbeiter K, Bald M, et al. Percutaneous endoscopic gastrostomy in children on peritoneal dialysis. *Perit Dial Int.* 2006;26(1):69–77. (PubMed PMID: 16538878. Epub 2006/03/17).
67. Ledermann SE, Spitz L, Moloney J, Rees L, Trompeter RS. Gastrostomy feeding in infants and children on peritoneal dialysis. *Pediatr Nephrol* (Berlin, Germany). 2002;17(4):246–50. (PubMed PMID: 11956875).
68. Ramage IJ, Harvey E, Geary DF, Hebert D, Balfe JA, Balfe JW. Complications of gastrostomy feeding in children receiving peritoneal dialysis. *Pediatr Nephrol* (Berlin, Germany). 1999;13(3):249–52. (PubMed PMID: 10353416).
69. Warady BA, Bashir M, Donaldson LA. Fungal peritonitis in children receiving peritoneal dialysis: a report of the NAPRTCS. *Kidney Int.* 2000;58(1):384–9. (PubMed PMID: 10886585. Epub 2000/07/08).
70. Neu AM, Miller MR, Stuart J, Lawlor J, Richardson T, Martz K, et al. Design of the standardizing care to improve outcomes in pediatric end stage renal disease collaborative. *Pediatr Nephrol* (Berlin, Germany). 2014;29(9):1477–84. (PubMed PMID: 25055994. Epub 2014/07/25).
71. Schaefer B, Macher-Goeppinger S, Testa S, Holland-Cunz S, Querfeld U, Schaefer F, et al. An International Peritoneal Dialysis Biopsy Study in Children on peritoneal dialysis and healthy controls. *Perit Dial Int.* 2013;33(Suppl 1).
72. Canepa A, Verrina E, Perfumo F. Use of new peritoneal dialysis solutions in children. *Kidney Int Suppl.* 2008;(108):S137–44. (PubMed PMID: 18379537. Epub 2008/05/03).
73. Schroder CH. Optimal peritoneal dialysis: choice of volume and solution. *Nephrol Dial Transplant.* 2004;19(4):782–4. (PubMed PMID: 15031330. Epub 2004/03/20).
74. Rusthoven E, Krediet RT, Willems HL, Monnens LAH, Schröder CH. Peritoneal transport characteristics with glucose polymer-based dialysis fluid in children. *J Am Soc Nephrol.* 2004;15(11):2940–7.
75. Dart A, Feber J, Wong H, Filler G. Icodextrin re-absorption varies with age in children on automated peritoneal dialysis. *Pediatr Nephrol* (Berlin, Germany). 2005;20(5):683–5. (PubMed PMID: 15719251. Epub 2005/02/19).
76. McIntyre CW. Update on peritoneal dialysis solutions. *Kidney Int.* 2007;71(6):486–90. (PubMed PMID: 17299524. Epub 2007/02/15).
77. Durand PY. Optimization of fill volumes in automated peritoneal dialysis. *Perit Dial Int.* 2000;20(6):601–2. (PubMed PMID: 11216546. Epub 2001/02/24).
78. Fischbach M, Terzic J, Menouer S, Haraldsson B. Optimal volume prescription for children on peritoneal dialysis. *Perit Dial Int.* 2000;20(6):603–6. (PubMed PMID: 11216547. Epub 2001/02/24).
79. Schaefer F, Warady BA. Peritoneal dialysis in children with end-stage renal disease. *Nat Rev Nephrol.* 2011;7(11):659–68. (PubMed PMID: 21947118. Epub 2011/09/29).
80. Fischbach M, Terzic J, Laugel V, Escande B, Dangelser C, Helmstetter A. Measurement of hydrostatic intraperitoneal pressure: a useful tool for the improvement of dialysis dose prescription. *Pediatr Nephrol* (Berlin, Germany). 2003;18(10):976–80. (PubMed PMID: 12898379. Epub 2003/08/05).
81. KDOQI. Clinical practice guidelines and clinical practice recommendations for updates. Hemodialysis adequacy, peritoneal dialysis adequacy and vascular access. *Am J Kidney Dis.* 2006;28(Suppl 1):S1.
82. Twardowski ZJ, Nolph KD, Khanna R. Limitations of the peritoneal equilibration test. *Nephrol Dial Transplant.* 1995;10(11):2160–1. (PubMed PMID: 8643196. Epub 1995/11/01).
83. Johnson D, Mudge D, Blizzard S, Arndt M, O'Shea A, Watt R, et al. A comparison of peritoneal equilibration tests performed 1 and 4 weeks after PD commencement. *Perit Dial Int.* 2004;24(5):460–5.
84. Rocco MV, Jordan JR, Burkart JM. Changes in peritoneal transport during the first month of peritoneal dialysis. *Perit Dial Int.* 1995;15(1):12–7. (PubMed PMID: 7734554. Epub 1995/01/01).



85. Schaefer F, Klaus G, Mehls O. Peritoneal transport properties and dialysis dose affect growth and nutritional status in children on chronic peritoneal dialysis. Mid-European Pediatric Peritoneal Dialysis Study Group. *J Am Soc Nephrol*. 1999;10(8):1786–92. (PubMed PMID: 10446947. Epub 1999/08/14).
86. Verrina E, Amici G, Perfumo F, Trivelli A, Canepa A, Gusmano R. The use of the PD Adequest mathematical model in pediatric patients on chronic peritoneal dialysis. *Perit Dial Int*. 1998;18(3):322–8. (PubMed PMID: 9663898. Epub 1998/07/15).
87. Warady BA, Jennings J. The short PET in pediatrics. *Perit Dial Int*. 2007;27(4):441–5. (PubMed PMID: 17602153. Epub 2007/07/03).
88. Cano F, Sanchez L, Rebori A, Quiroz L, Delucchi A, Delgado I, et al. The short peritoneal equilibration test in pediatric peritoneal dialysis. *Pediatr Nephrol (Berlin, Germany)*. 2010;25(10):2159–64. (PubMed PMID: 20574772. Epub 2010/06/25).
89. Fadrowski JJ, Frankenfield D, Amaral S, Brady T, Gorman GH, Warady B, et al. Children on long-term dialysis in the United States: findings from the 2005 ESRD clinical performance measures project. *Am J Kidney Dis*. 2007;50(6):958–66. (PubMed PMID: 18037097. Epub 2007/11/27).
90. Paniagua R, Amato D, Correa-Rotter R, Ramos A, Vonesh EF, Mujais SK. Correlation between peritoneal equilibration test and dialysis adequacy and transport test, for peritoneal transport type characterization. Mexican Nephrology Collaborative Study Group. *Perit Dial Int*. 2000;20(1):53–9. (PubMed PMID: 10716584. Epub 2000/03/15).
91. Lo WK, Lui SL, Chan TM, Li FK, Lam MF, Tse KC, et al. Minimal and optimal peritoneal Kt/V targets: results of an anuric peritoneal dialysis patient's survival analysis. *Kidney Int*. 2005;67(5):2032–8. (PubMed PMID: 15840054. Epub 2005/04/21).
92. Morgenstern BZ, Mahoney DW, Warady BA. Estimating total body water in children on the basis of height and weight: a reevaluation of the formulas of Mellits and Cheek. *J Am Soc Nephrol*. 2002;13(7):1884–8. (PubMed PMID: 12089384. Epub 2002/06/29).
93. Bakkaloglu SA, Borzych D, Soo Ha I, Serdaroglu E, Buscher R, Salas P, et al. Cardiac geometry in children receiving chronic peritoneal dialysis: findings from the International Pediatric Peritoneal Dialysis Network (IPPN) registry. *Clin J Am Soc Nephrol: CJASN*. 2011;6(8):1926–33. (PubMed PMID: 21737855. Pubmed Central PMCID: PMC3359542. Epub 2011/07/09).
94. Mitsnefes MM, Daniels SR, Schwartz SM, Meyer RA, Khoury P, Strife CF. Severe left ventricular hypertrophy in pediatric dialysis: prevalence and predictors. *Pediatr Nephrol (Berlin, Germany)*. 2000;14(10–11):898–902. (PubMed PMID: 10975295. Epub 2000/09/07).
95. Mitsnefes M, Stablein D. Hypertension in pediatric patients on long-term dialysis: a report of the North American Pediatric Renal Transplant Cooperative Study (NAPRTCS). *Am J Kidney Dis*. 2005;45(2):309–15. (PubMed PMID: 15685509. Epub 2005/02/03).
96. Warady BA, Ellis EN, Fivush BA, Lum GM, Alexander SR, Brewer ED, et al. “Flush before fill” in children receiving automated peritoneal dialysis. *Perit Dial Int*. 2003;23(5):493–8. (PubMed PMID: 14604204. Epub 2003/11/08).
97. Strippoli GF, Tong A, Johnson D, Schena FP, Craig JC. Catheter-related interventions to prevent peritonitis in peritoneal dialysis: a systematic review of randomized, controlled trials. *J Am Soc Nephrol*. 2004;15(10):2735–46. (PubMed PMID: 15466279. Epub 2004/10/07).
98. Bakkaloglu SA. Prevention of peritonitis in children: emerging concepts. *Perit Dial Int*. 2009;29(Suppl 2):S186–9. (PubMed PMID: 19270214. Epub 2009/05/16).
99. Warady BA, Feneberg R, Verrina E, Flynn JT, Muller-Wiefel DE, Besbas N, et al. Peritonitis in children who receive long-term peritoneal dialysis: a prospective evaluation of therapeutic guidelines. *J Am Soc Nephrol*. 2007;18(7):2172–9. (PubMed PMID: 17582162. Epub 2007/06/22).
100. Chadha V, Schaefer F, Warady B. Peritonitis and exit-site infections. In: Warady B, Schaefer F, Alexander S, editors. *Pediatric dialysis*. New York: Springer; 2012. pp. 231–56.
101. Warady BA, Neu AM, Schaefer F. Optimal care of the infant, child, and adolescent on dialysis: 2014 update. *Am J Kidney Dis*. 2014;64(1):128–42. (PubMed PMID: 24717681. Epub 2014/04/11).
102. Bernardini J, Price V, Figueiredo A. Peritoneal dialysis patient training, 2006. *Perit Dial Int*. 2006;26(6):625–32. (PubMed PMID: 17047225. Epub 2006/10/19).
103. Chua AN, Goldstein SL, Bell D, Brewer ED. Topical mupirocin/sodium hypochlorite reduces peritonitis and exit-site infection rates in children. *Clin J Am Soc Nephrol: CJASN*. 2009;4(12):1939–43. (PubMed PMID: 19820132. Pubmed Central PMCID: PMC2798867. Epub 2009/10/13).
104. Honda M, Warady BA. Long-term peritoneal dialysis and encapsulating peritoneal sclerosis in children. *Pediatr Nephrol (Berlin, Germany)*. 2010;25(1):75–81. (PubMed PMID: 21476232. Pubmed Central PMCID: PMC2778779. Epub 2010/01/01Eng).
105. Vidal E, Edefonti A, Puteo F, Chimenz R, Gianoglio B, Lavoratti G, et al. Encapsulating peritoneal sclerosis in paediatric peritoneal dialysis patients: the experience of the Italian Registry of Pediatric Chronic Dialysis. *Nephrol Dial Transplant*. 2013;28(6):1603–9. (PubMed PMID: 23585587. Epub 2013/04/16).
106. Hoshii S, Honda M. High incidence of encapsulating peritoneal sclerosis in pediatric patients on peritoneal dialysis longer than 10 years. *Perit Dial Int*. 2002;22(6):730–1. (PubMed PMID: 12556080. Epub 2003/01/31).
107. Kawaguchi Y, Saito A, Kawanishi H, Nakayama M, Miyazaki M, Nakamoto H, et al. Recommendations on the management of encapsulating peritoneal sclerosis in Japan, 2005: diagnosis, predictive markers, treatment, and preventive measures. *Perit Dial Int*. 2005;25(Suppl 4):S83–95. (PubMed PMID: 16300277. Epub 2005/11/23).
108. Bakkaloglu A. Non-infectious complications of peritoneal dialysis in children. In: Warady BASF, Alexander S, editors. *Pediatric dialysis*. 2nd ed. New York: Springer; 2012. pp. 257–71.
109. Bakkaloglu SA, Ekim M, Tumer N, Gungor A, Yilmaz S. Pleurodesis treatment with tetracycline in peritoneal dialysis-complicated hydrothorax. *Pediatr Nephrol (Berlin, Germany)*. 1999;13(7):637–8. (PubMed PMID: 10507835. Epub 1999/10/03).
110. Szeto CC, Chow KM. Pathogenesis and management of hydrothorax complicating peritoneal dialysis. *Current Opin Pulm Med*. 2004;10(4):315–9. (PubMed PMID: 15220759. Epub 2004/06/29).
111. Canpolat N, Caliskan S, Sever L, Tasdemir M, Ekmekci OB, Pehlivan G, et al. Malnutrition and its association with inflammation and vascular disease in children on maintenance dialysis. *Pediatr Nephrol (Berlin, Germany)*. 2013;28(11):2149–56. (PubMed PMID: 23765444. Epub 2013/06/15).
112. KDOQI. Clinical practice guideline for nutrition in children with CKD. *Am J Kidney Dis*. 2009;53(Suppl 2):S1.
113. Paulson WD, Bock GH, Nelson AP, Moxey-Mims MM, Crim LM. Hyponatremia in the very young chronic peritoneal dialysis patient. *Am J Kidney Dis*. 1989;14(3):196–9. (PubMed PMID: 2773922. Epub 1989/09/01).
114. Chuang Y-W, Shu K-H, Yu T-M, Cheng C-H, Chen C-H. Hypokalaemia: an independent risk factor of enterobacteriaceae peritonitis in CAPD patients. *Nephrol Dial Transplant*. 2009;24(5):1603–8.
115. Roodhooft AM, Van Hoeck KJ, Van Acker KJ. Hypophosphatemia in infants on continuous ambulatory peritoneal dialysis. *Clin Nephrol*. 1990;34(3):131–5. (PubMed PMID: 2225564. Epub 1990/09/01Eng).

116. Leonard MB, Donaldson LA, Ho M, Geary DF. A prospective cohort study of incident maintenance dialysis in children: a NAPRTCS study. *Kidney Int.* 2003;63(2):744–55. (PubMed PMID: 12631143. Epub 2003/03/13).
117. Mitsnefes MM, Laskin BL, Dahhou M, Zhang X, Foster BJ. Mortality risk among children initially treated with dialysis for end-stage kidney disease, 1990–2010. *JAMA.* 2013;309(18):1921–9. (PubMed PMID: 23645144. Pubmed Central PMCID: PMC3712648. Epub 2013/05/07).
118. Coulthard MG, Crosier J. Outcome of reaching end stage renal failure in children under 2 years of age. *Arch Dis Child.* 2002;87(6):511–7. (PubMed PMID: 12456551Eng).
119. Shroff R, Rees L, Trompeter R, Hutchinson C, Ledermann S. Long-term outcome of chronic dialysis in children. *Pediatr Nephrol (Berlin, Germany).* 2006;21(2):257–64. (PubMed PMID: 16270221Eng).
120. McDonald SP, Craig JC. Long-term survival of children with end-stage renal disease. *N Engl J Med.* 2004;350(26):2654–62. (PubMed PMID: 15215481. Epub 2004/06/25).
121. Verrina E, Edefonti A, Gianoglio B, Rinaldi S, Sorino P, Zaccchello G, et al. A multicenter experience on patient and technique survival in children on chronic dialysis. *Pediatric Nephrol (Berlin, Germany).* 2004;19(1):82–90. (PubMed PMID: 14648343).
122. Wood EG, Hand M, Briscoe DM, Donaldson LA, Yiu V, Harley FL, et al. Risk factors for mortality in infants and young children on dialysis. *Am J Kidney Dis.* 2001;37(3):573–9. (PubMed PMID: 11228182Eng).
123. Neu AM, Sander A, Borzych-Duzalka D, Watson AR, Valles PG, Ha IS, et al. Comorbidities in chronic pediatric peritoneal dialysis patients: a report of the International Pediatric Peritoneal Dialysis Network. *Perit Dial Int.* 2012;32(4):410–8. (PubMed PMID: 22859841. Pubmed Central PMCID: PMC3524853. Epub 2012/08/04. Eng).
124. Warady BA. Peritoneal dialysis. In: Silverstein DM, Symons JM, Alon US, editors *Pediatric nephrology: a handbook for training healthcare providers.* Singapore: World Scientific Publishing (In Press.). pp. 551–84.
125. Schaefer F, Feneberg R, Aksu N, Donmez O, Sadikoglu B, Alexander SR, et al. Worldwide variation of dialysis-associated peritonitis in children. *Kidney Int.* 2007;72(11):1374–9.