Peritoneal Dialysis

An Adjunct to Pediatric Postcardiotomy Fluid Management

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Patients requiring cardiopulmonary bypass for congenital heart surgery commonly exhibit impaired renal function and extravascular fluid retention. These conditions contribute to early postoperative fluid overload, which may result in significant morbidity and mortality. We examined the safety and efficacy of peritoneal dialysis in removing extravascular fluid from critically ill postcardiotomy patients. A retrospective case review from July of 1995 through April of 1996 was conducted. All patients undergoing peritoneal dialysis achieved a net negative fluid balance. Average urine output increased from 2.1 cc/kg/hr to 3.9 cc/kg/hr (P <0.01) during the pre-peritoneal dialysis to post-peritoneal dialysis period, and the mean number of inotropic agents decreased from 2.2 to 1.7 (P <0.05). Controlled comparison revealed that the peritoneal dialysis cohort more rapidly achieved a negative weight-adjusted fluid balance throughout the early postoperative course. The peritoneal dialysis group's illness severity decreased more rapidly within the 24-hour period after initiation of peritoneal dialysis than did that of the control cohort over the same period of time. No difference in postoperative morbidity or mortality existed between the study groups. Complications from the catheter placement were minimal, and no patient experienced peritonitis or metabolic or hemodynamic instability during peritoneal dialysis catheter placement, usage, or removal. Peritoneal dialysis is a safe and effective form of renal replacement therapy, even among critically ill pediatric postcardiotomy patients. Early postsurgical institution of peritoneal dialysis may hasten early postoperative recovery. We speculate that intraoperative catheter placement reduces the complication rate associated with this treatment modality. ***(Tex Heart Inst J 1997;24:269-77)***

Section editor:
Timothy F. Feltes, MD

Key words: *Cardiopulmonary bypass/adverse effects; heart defects, congenital/surgery; heart surgery/adverse effects; infant, newborn; peritoneal dialysis*

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Advances in intraoperative management of the neonate during the past 2 decades have radically changed the expected outcome for many forms of congenital heart disease. Lesions previously considered lethal are now effectively palliated or corrected in the newborn period. This improved operative survival has led to the training and practice of a new breed of cardiac intensivists, nurses, and healthcare providers with expertise in the management of the postcardiotomy infant. These multidisciplinary professionals have developed an understanding of the neonate's unique physiologic and pathologic responses to surgical stress. Moreover, they have learned to be innovative in the therapeutic management of these critically ill children in order to match the success of their surgical colleagues.

Edema of the lungs and systemic tissues after cardiopulmonary bypass (CPB) may impair pulmonary gas exchange, alter hemostasis, decrease ventricular filling, and delay cerebral metabolic recovery in the postoperative neonate.[1] Renal dysfunction following cardiac surgery may exacerbate these ill effects by reducing clearance of total body water and solute.[2-6] Therefore, successful management of total body fluid balance is critical in minimizing postcardiotomy morbidity.

Various forms of therapy have been developed to reverse tissue edema and enhance postoperative fluid and solute clearance. Conventional medical treatment has included fluid restriction and diuretic therapy, as well as inotropic support and afterload manipulation. When these measures fail, extracorporeal means of fluid removal such as hemofiltration or hemodialysis must be considered. However, these treatments are invasive, may be associated with hemodynamic instability, and typically have a significant lag time prior to initiation. We have found peritoneal dialysis (PD) to be a highly effective, relatively simple means of fluid management that can be rapidly employed with minimal morbidity, even in the most critically ill infants. In this report, we will explain the mechanisms of postop-

erative fluid retention in this select group of patients and describe our experience with the successful use of postcardiotomy PD.

Regulation of Transmembrane Fluid Filtration

Under normal circumstances, fluid is continuously filtered from the intravascular space to the tissue interstitium. From there, transudate is removed via the tissue lymphatic system and returned to the vascular compartment. The rate of transvascular fluid filtration is controlled by a balance of hydrostatic and oncotic forces in combination with the permeability and surface area properties of the microvascular bed. These forces are commonly referred to as the Starling forces of fluid balance and are related in the Starling Equation:

$$Q_f = K (P_{mv} - P_{int}) - \sigma (\pi_{mv} - \pi_{int})$$

where Q_f = rate of transvascular fluid filtration, K = hydraulic conductance constant, P_{mv} = microvascular hydrostatic pressure, P_{int} = interstitial hydrostatic pressure, σ = permeability constant, π_{mv} = microvascular oncotic pressure, and π_{int} = interstitial oncotic pressure.

The high sieving characteristics of the microvascular bed, coupled with a favorable balance between the hydrostatic and oncotic forces, usually keep Q_f at a rate compatible with the siphoning capacity of the lymphatic vascular bed (Q_l). Thus, under basal conditions, Q_f is less than or equal to Q_l. Tissue edema develops whenever Q_f is greater than Q_l. Such a situation may occur when 1) alteration of the Starling forces nets an increase in Q_f beyond the capacity of Q_l or 2) tissue Q_l is effectively reduced, causing interstitial fluid accumulation. In the postcardiotomy infant, both conditions may exist.

Hydrostatic Forces. Microvascular hydrostatic pressure (P_{mv}) is the principal force altering fluid filtration in the patient with heart failure. Increased P_{mv} in the pulmonary and systemic vascular beds results in pulmonary and dependent tissue edema in patients with congestive heart failure who have increased sodium and water resorption. P_{mv} may increase by virtue of increased pressure in either the arterial or venous circulatory limb, and there is an obligatory increase in P_{mv} when venous pressure increases. During heart failure, right and left ventricular filling pressures increase, resulting in increased pulmonary and systemic venous pressures. The rise in P_{mv} increases Q_f, causing passive congestion of the liver and spleen. Edema does not develop, however, until Q_f exceeds Q_l. An increase in the arterial pressure may or may not affect P_{mv}. Often, arterial pressure increases due to vasoconstriction, which typically occurs proximal to fluid filtering sites in the capillary bed. Following CPB, vasoconstriction probably does not lead to a significant direct increase in P_{mv}. However, P_{mv} may increase secondary to impaired ventricular emptying and venous congestion caused by increased cardiac filling pressure.

Another influence upon intrapulmonary P_{mv} in the postcardiotomy neonate is the relative immaturity of the pulmonary vascular bed. In the neonatal lamb, the pulmonary capillary bed is relatively nonrecruitable.[7] A nonrecruitable vascular bed has relatively fixed resistance. If a procedure is performed that increases pulmonary blood flow, P_{mv} will obligately increase (flow × resistance = pressure), thus raising Q_f.

Oncotic Forces. The balance of oncotic forces between the vascular compartment and the interstitium normally counters the effects of the hydrostatic pressure gradient in fluid transudation. The oncotic forces in the microvascular bed (π_{mv}) are typically high relative to those in the interstitium (π_{int}). This is because the microvascular bed is relatively impermeable to serum proteins that are concentrated in the intravascular space. This considerable sieving ability is reflected in a σ that approaches 1. In the newborn lamb, the pulmonary σ approximates 0.75 and changes very little with age.[8] If, however, the microvascular bed is injured, then σ may decrease drastically so that fluid and solute filter into the interstitial space and π_{mv} and π_{int} approximate one another. Under these conditions, Q_f becomes more dependent upon the hydrostatic pressure gradient. Microvascular injury may occur during CPB as a result of microembolization, which leads to endothelial dysfunction, or as a consequence of the inflammatory response, which gives rise to a capillary leak syndrome. With either form of vascular damage, serum proteins pour into the interstitium, raising π_{int}, which draws free water into the interstitial space. Until time for repair of the damaged microvascular bed has elapsed, the patient may be prone to increased fluid filtration and tissue edema. Conditions that decrease π_{mv}, such as anemia or hypoalbuminemia, may also contribute to increased fluid filtration in the postoperative patient.

Lymphatic Function. Because the lymphatic system drains into the systemic venous system, increased central venous pressure may impair lymphatic drainage. In the lamb, lung lymph flow varies inversely with superior vena caval pressure.[9] A decrease in Q_l by this mechanism is likely responsible for the pulmonary edema seen in neonates with cor pulmonale or in patients following a total cavopulmonary diversion procedure. The lymphatic capillary bed develops in parallel to the vascular capillary bed; consequently, as in the pulmonary vascular bed, the lymphatic bed may be incompletely developed. Lack of a recruitable lymphatic bed may limit Q_l. Finally, Q_l may also be decreased if lymphatic capillaries are obstructed, as may occur with extensive intrathoracic scarring.

Postoperative Fluid Retention

Maturational Influences. At birth and for several months thereafter, the kidneys function with very limited reserve. The glomerular filtration rate is reduced compared to that of older infants and children due to a restricted glomerular capillary surface area.[3,10-14] Renal vascular resistance is high, with correspondingly elevated circulating renin levels and decreased outer cortical blood flow.[15] There is also evidence that the neonatal kidney is more vulnerable to conditions of hemodynamic stress with loss of autoregulation leading to blood-pressure-dependent renal blood flow and ischemia-induced renal injury. All of these conditions render the neonate more prone to complications of ischemia than is the older infant or child.[3,6]

Hormonal responses to volume loading may also be limited in the newborn. Atrial natriuretic factor is produced in response to cardiac dilation that occurs with volume loading. It acts upon the kidney to promote diuresis and natriuresis. Atrial natriuretic factor production is attenuated in the newborn.[16]

Preoperative Conditions. Postoperative fluid balance may be affected by numerous preoperative conditions that have been shown to predispose the neonate to renal insufficiency.[6,17] At particular risk is the infant with a ductal-dependent cardiac lesion (e.g., severe coarctation) who first presents with cardiovascular collapse and severe renal ischemia. Additional factors, such as the presence of preoperative cyanosis or acidosis or the need for prostaglandin E_1 infusion, may further compromise the neonatal kidney.[1,3] Structural renal abnormalities are frequently encountered in association with cardiac defects (e.g., VACTERL syndrome*).[16]

Renal Ischemia and the Renin-Angiotensin System. Infants with congenital heart disease commonly manifest congestive heart failure associated with decreased renal plasma flow in the preoperative and early postoperative periods.[10,12] Renal ischemia triggers the release of renin from the juxtaglomerular cells of the kidney, which in turn triggers the production of angiotensin I. Angiotensin I is converted to angiotensin II, which potentiates sympathetic activity (increasing vascular resistance), diminishes the renal effects of atrial natriuretic factor, and promotes aldosterone release from the adrenal cortex. Aldosterone, in turn, increases sodium resorption and kaliuresis from the distal tubule. These processes favor postoperative fluid accumulation.

Renal Insufficiency and Renal Failure. The frequency of acute renal failure is higher in neonates than in older infants, following cardiac surgery.[5,18] Various reports place the incidence of overt renal insufficiency resulting in uremia at 2% to 9% of postcardiotomy cases, but the presence of a relative renal insufficiency early in the postoperative course that leads to fluid retention is considerably higher.[2-6,19,20] Cardiopulmonary bypass and associated reperfusion and rewarming of ischemic organs have long been known to trigger an inflammatory response, which can adversely affect renal function. Induction of this stress response, consisting of release of a cascade of metabolically and hormonally active substances, has been attributed to 1) blood contact with nonendothelialized surfaces of pump tubing and the oxygenator, 2) nonpulsatile CPB flow, 3) hemodilution, 4) hypothermia, 5) inadequate or reduced anesthetic depth, and 6) ischemia-induced endotoxin exposure that leads to systemic inflammation.[10,21-23] The CPB stress response process is thought to begin with oxygen free radical release from endothelial cells rendered dysfunctional by prolonged hypoxia and subsequent reperfusion.[21] This triggers endothelial cell adhesion molecule (selectin) expression, which in turn activates local serum complement (e.g., anaphylatoxins C3a, C5a, and $C5a_{des\ arg}$). Complement activation causes chemotactic activation and "trapping" of neutrophils within organs.[21,24-26] Cytokines (tumor necrosis factor [TNF] and interleukin-1, -6, and -8) are then produced by activated leukocytes, platelets, and endothelium, causing upregulation and expression of leukocyte adhesion molecules known as integrins.[22,26,27] Integrins bind to counterreceptors termed intercellular adhesion molecules on the surface of endothelial and tissue-specific cells (e.g., glomerular or renal tubular epithelium), which results in tighter leukocyte attachment and transmigration of neutrophils into the interstitial space. Consequently, neutrophils degranulate, releasing proteolytic enzymes and reactive oxygen radicals that mediate end-organ injury. Organ dysfunction may also result from the initiation of coagulation, from fibrinolytic and kallikrein cascades, and from the elaboration of substances such as catecholamines, cortisol, growth hormone, prostaglandins, leukotrienes, glucose, insulin, and beta endorphins, to name but a few.[10,25] The metabolic and inflammatory derangements caused by the stress response to CPB may persist for more than 24 hours after congenital heart surgery,[25] thereby producing vascular damage, extravascular fluid and solute accumulation, and renal injury. The stress response causes a decrease in glomerular filtration rate and renal blood flow via vasoconstriction and alteration of intrarenal flow patterns, despite maintenance of normal cardiac output rates on CPB.[2,3]

Capillary Leak Syndrome. The same inflammatory response that impairs renal function is thought to mediate the formation of generalized edema encoun-

* VACTERL is an abbreviation for vertebral, anal, cardiac, tracheal, esophageal, renal, and limb; it is used to designate a pattern of congenital anomalies.

tered in the postcardiotomy infant. This condition, known as capillary leak syndrome, results primarily from protein losses into the interstitium that raise extravascular oncotic pressure and increase net transcapillary fluid movement. The syndrome is characterized by severe edema of the peripheral tissues, along with development of pleural and pericardial effusions and ascites within the 1st few hours after surgery. It may be associated with multiorgan dysfunction and can be anticipated in the infant who has experienced a protracted CPB course, especially if circulatory arrest was prolonged. Capillary leak syndrome usually subsides within 3 to 4 days after surgery.[16]

Management of Fluid Balance in the Postcardiotomy Neonate

Nonperitoneal Dialysis Methods. Medical therapy for postcardiotomy fluid retention and renal insufficiency is directed toward the following goals: 1) improving renal perfusion (through afterload reduction and positive inotropic support), 2) promoting diuresis (via diuretic therapy), and 3) restricting fluid intake. When these measures fail, extracorporeal renal replacement therapy may be used. Hemofiltration uses convection across a semipermeable membrane to remove water and low-molecular-weight protein solutes from the circulating blood volume. Conventional hemofiltration entails diversion of blood from the vent of the arterial filter to the hemofilter (where ultrafiltrate is removed) and back to the venous reservoir. It is often used during the rewarming phase of CPB to remove free water and proinflammatory substances.[1,27,28] Modified hemofiltration is usually performed after separation from CPB and involves shunting blood from the arterial cannula to the hemofilter (excluding the gas exchanger in the main CPB circuit), with blood return to a vent inserted into the right atrium. Careful monitoring of the patient's volume status and filling pressures is required during this procedure, since hypotension may result.

Many investigators have demonstrated the benefits of perioperative hemofiltration.[29-32] Elliott,[29] using primarily the modified technique, described reduced CPB-associated water accumulation, blood loss, and the need for transfusion in pediatric patients receiving hemofiltration, compared with controls. He also noted improved immediate postbypass hemodynamics characterized by increased mean blood pressure, cardiac contractility, and cardiac index and by lowered pulmonary vascular resistance. Journois and associates[30] employed conventional hemofiltration of pediatric patients during CPB rewarming to demonstrate improvement in postoperative hemodynamics, early oxygenation, blood loss, and duration of mechanical ventilation. Despite reductions in the plasma concentrations of inflammatory mediators, no specific differences in renal function were detected as a result of the hemofiltration process.[29-31,33,34]

Hemodialysis involves both ultrafiltration and diffusion of solute across a semipermeable membrane. Fluid removal is determined by the hydrostatic pressure difference between the blood and dialysate compartments, whereas solute removal proceeds by diffusion down a concentration gradient to the point of equilibration between the patient's serum and the dialysate. This modality has been used for postcardiotomy renal insufficiency.[35] Its applicability has been limited by the need for large-bore intravenous (IV) access and a large extracorporeal blood volume, the risk of metabolic derangement arising from rapid changes in serum solute composition, and, perhaps most important, the risk of severe hemodynamic instability. This latter problem is caused by the intermittent nature of hemodialysis fluid removal. Such maneuvers are poorly tolerated in hemodynamically compromised postcardiotomy patients who cannot expeditiously and adequately refill the intravascular space with fluid from the interstitial space.[36]

In an attempt to achieve slow and continuous fluid removal from hypervolemic postcardiotomy patients with renal insufficiency, continuous arteriovenous hemofiltration (CAVH) and continuous venovenous hemofiltration (CVVH) have been employed. These treatment modalities remove ultrafiltrate at a rate that allows for vascular refilling from the extravascular space. The CAVH circuit draws from the arterial limb and drains to the venous limb of the patient's circulation. Continuous arteriovenous hemofiltration is driven by the patient's arteriovenous pressure gradient and is therefore both autoregulated by and dependent upon arterial blood pressure for ultrafiltrate formation. Hemodynamic instability during CAVH may decrease ultrafiltrate production. Continuous venovenous hemofiltration involves circulation of blood from the venous system, through a filter, and back to the venous pool. The process is driven by an external pump, rendering it independent of the patient's systemic blood pressure.

While CAVH and CVVH may be used effectively for postcardiotomy fluid management, several technical aspects of these processes hamper their widespread use. First, both forms of hemofiltration require central IV access, which may be difficult to obtain and maintain, especially in younger patients. This is particularly true for CAVH, which uses arterial access. Hemorrhage and limb ischemia have been reported with use of CAVH.[36,37] Second, systemic anticoagulation (usually in the form of continuous infusion heparin) with its attendant risks is necessary to maintain activated clotting times of 150 to 180 seconds. Third, extracorporeal hemofiltration

circuits often require a blood priming volume that exposes the patient to the risk of infection through blood product transfusion.[36]

Peritoneal Dialysis. The use of PD for cardiology patients originated in the 1950s as a treatment to alleviate the symptoms of adult congestive heart failure. However, as equipment and techniques were adapted to smaller patients, this alternative to hemodialysis became practical for use in children with congenital heart disease. In 1966, Nora and colleagues[38] described the use of a short course of PD to treat 7 patients aged 9 weeks to 28 months who were in severe congestive heart failure refractory to conventional medical therapy. All patients showed clinical improvement, with 50% more fluid withdrawn than was introduced.

Postcardiotomy use of PD in children became an accepted practice in the 1970s and the successful aspects of this therapeutic intervention have subsequently been characterized.[17] It has now been relatively well established that early intervention with PD in the course of postoperative renal dysfunction is warranted, both to prevent the negative organ system effects of capillary leak and resultant edema and to treat the subset of oligoanuric patients who may rapidly reverse their low cardiac output state.[3,4,17,19] It has also been noted in some series[3,4,39] that a time interval greater than 24 hours between termination of CPB and initiation of PD is associated with a better prognosis. This finding may simply reflect the likelihood that greater illness severity tracks with the requirement for earlier dialysis and poor prognosis. More controversial, however, is the notion that patients become resistant to dehydration by PD secondary to low cardiac output and reduced peritoneal blood circulation.[17] Several articles[3,19,40] have documented successful peritoneal ultrafiltrate removal from patients requiring significant inotropic support with evidence of low cardiac output. The issue of whether PD influences postoperative mortality remains unclear.[39,41]

How does PD compare with other forms of extracorporeal fluid and renal management in the postcardiotomy patient? In 1 series[37] of pediatric postcardiotomy patients with renal failure, the effectiveness of CAVH, CVVH, and PD was analyzed. The authors reported distinct advantages of CAVH and CVVH over PD, citing a greater net negative fluid balance, improved caloric intake after initiation of therapy, and more effective reduction of serum urea and creatinine levels. Complications related to PD included failure of dialysate drainage and peritonitis, while hemorrhage and hypothermia were encountered with CAVH/CVVH. Considerable mortality was observed among the 3 patient cohorts (58% to 67%), without significant differences in outcome related to the treatments. The investigators concluded that hemofiltration after congenital heart surgery offered significant advantages when compared with the more traditional approach of PD.[37]

Texas Children's Hospital Experience with Peritoneal Dialysis

Our recent experience with postcardiotomy PD at Texas Children's Hospital (TCH) has been substantial. We have employed this form of therapy in 44 (5%) children undergoing open heart repair since July of 1995. Conventional hemofiltration at the cessation of CPB was employed in the majority of patients to achieve a higher hematocrit and reduce extravascular fluid retention. However, hemofiltration did not obviate the need for subsequent postoperative renal replacement therapy in a small subset of patients.

It is our current practice at the time of cardiac surgery to place a soft silicone rubber uncuffed PD catheter in all neonates undergoing intracardiac repair. Patients thought to be at risk for development of right heart failure (e.g., pulmonary atresia/intact ventricular septum patients undergoing right ventricular overhaul), patients with increased pulmonary vascular resistance, and patients undergoing total cavopulmonary diversion are considered for PD catheter placement on a case-by-case basis. Candidates for PD catheter placement have comprised 23.7% of our surgical population.

In the early postoperative course, the PD catheter initially acts as a passive peritoneal drain. We have found this valuable in offsetting abdominal distention in many cases, thereby increasing the ease of postcardiotomy ventilation. Our indications for institution of PD include 1) oligoanuria (urine output less than 1 cc/kg/hr) unresponsive to intermittent or continuous diuretic infusion (in the setting of adequate ventricular filling pressure) for 4 hours, 2) fluid overload/anasarca with insufficient diuresis, and 3) low cardiac output with metabolic instability (e.g., acidosis, hyperkalemia, rapidly rising core temperature) and renal insufficiency. Once the decision to employ PD has been made, therapy can be rapidly initiated.

We use continuous manual PD using a standard Gesco setup with 60-minute exchanges. A dialysate dextrose concentration of 1.5% is used initially and is adjusted to achieve a hemodynamically tolerable negative fluid balance. Potassium is added to the dialysis fluid as needed to maintain serum potassium levels in the range of 3.5 to 4.5 meq/L. Antibiotics at doses sufficient to maintain appropriate serum levels supplement the dialysate in selected cases to treat systemic infections. Small PD dwell volumes (10 cc/kg) are cycled to avoid ventilatory compromise. Ultrafiltered fluid is occasionally replaced with colloid either to titrate the rate of net fluid removal or

to support the patient's arterial blood pressure. Because protein loss can occur during PD, daily serum albumin levels are monitored.

Our initial evaluation of patients receiving PD focused retrospectively on 19 infants who underwent congenital heart surgery during the 8-month period from July of 1995 to April of 1996 (Table I). Children undergoing the Norwood palliation of hypoplastic left heart syndrome represented the largest subgroup of treatment patients. The median patient age was 10 days; mean time from arrival in the cardiac intensive care unit to initiation of PD was 22 hours; and mean duration of PD was 50 hours. The total negative fluid balance from all sources averaged 106 cc/kg/day during dialysis, with PD ultrafiltration accounting for an average of 93 cc/kg/day (Table II). The average urine output increased from 2.1 cc/kg/hr to 3.9 cc/kg/hr (P <0.01) (Table III). The mean number of inotropic agents also decreased during the pre-PD to post-PD period from 2.2 to 1.7 (P<0.05). From this we concluded that PD could be effectively employed to achieve a negative fluid balance in hemodynamically unstable infants with postcardiotomy renal dysfunction. Our findings echoed those of Werner and colleagues,[19] who also demonstrated the effectiveness of postcardiotomy PD in infants with diminished cardiac function on inotropic therapy. We found that all 19 of our patients had a net negative fluid balance on PD, which challenges the findings of Fleming and associates,[37] who observed a net negative fluid balance in only 35% of PD patients.

TABLE I. Demographic Data from 19 Postcardiotomy Infants Undergoing Peritoneal Dialysis

Cardiac lesion (n=19)	
Hypoplastic left heart syndrome	5
Transposition of the great vessels	4
Tetralogy of Fallot	3
Total anomalous pulmonary venous return	2
Interrupted aortic arch	2
Anomalous left coronary artery	1
ASD/VSD	1
Truncus arteriosus	1
Age at start of PD (days), median (range)	10 (3–186)
Preoperative weight (kg), mean (range)	3.9 (2.7–6.8)
Time from surgery to start of PD (hr), mean (range)	22 (5–40)
Duration of PD (hr), mean (range)	50 (13–92)

ASD = atrial septal defect; PD = peritoneal dialysis; VSD = ventricular septal defect

TABLE II. Fluid Balance Data from 19 Postcardiotomy Infants Undergoing Peritoneal Dialysis

Total fluid in (cc/kg/day), mean (range)	93 (50–149)
Total fluid out (cc/kg/day), mean (range)	199 (120–421)
Via urine output	87 (46–165)
Via PD ultrafiltrate	93 (43–233)
Net negative fluid balance (cc/kg/day), mean (range)	106 (49–273)

PD = peritoneal dialysis

While we and others[3,17,19] had described the clinical benefits of PD for postcardiotomy fluid management, no studies known to us had established these benefits as distinct from the effects of early postoperative recuperation over time. Therefore, we sought to compare the effectiveness of PD in the removal of extravascular fluid from critically ill postcardiotomy patients less than 1 year of age who underwent either PD or conventional diuretic therapy without PD. In order to choose an appropriate control group of children with similar illness severity, patients were assigned an illness "severity score" at the end of the 1st postoperative day on the basis of degree of renal dysfunction, postoperative fluid balance, need for inotropic support and afterload reduction, and level of mechanical ventilatory support (Table IV). The PD (n=6) and control (n=7) groups were otherwise matched for age, weight, intraoperative PD catheter placement, and cardiopulmonary bypass/hemofiltration parameters. Scoring was repeated at 24-hour postoperative intervals. Only those patients whose initial severity scores reflected critical illness (severity score greater than 9) were selected for further investigation.

TABLE III. Pre- versus Postperitoneal Dialysis Clinical Parameters in 19 Postcardiotomy Infants Undergoing Peritoneal Dialysis

Parameter	Pre-PD	Post-PD	*P* value
Serum creatinine (mg/dL), mean	0.76	0.79	NS
Urine output (cc/kg/hr), mean	2.10	3.90	<0.01
Number of inotropic agents,* mean	2.15	1.65	<0.05

NS = not significant; PD = peritoneal dialysis

*Includes dopamine, dobutamine, epinephrine, norepinephrine, and milrinone

TABLE IV. Illness Severity Scoring System for Postcardiotomy Infants Undergoing Peritoneal Dialysis

	Severity Score			
	0	**1**	**2**	**3**
Dopamine (mcg/kg/min)	0	<5	5–9	>10
Dobutamine (mcg/kg/min)	0	<5	>5<10	>10
Milrinone (mcg/kg/min)	0	<0.3	0.3–0.5	>0.5
Epinephrine/Norepinephrine (mcg/kg/min)	0	0	0	>0
Nipride/Nitroglycerin (mcg/kg/min)	0	<0.5	0.5–1.5	>1.5
Urine output (cc/kg/hr)	>4	>2	1–2	<1
Postoperative fluid balance (cc)	<0	<300	300–500	>500
Mean airway pressure (cm H20)	0	<6	6–10	>10

The results of this controlled study demonstrated that the PD cohort achieved a negative weight-adjusted fluid balance more rapidly than did the control group throughout the early postoperative course (-2.1 cc/kg/hr versus -1.1 cc/kg/hr, respectively; P=0.03). In addition, the severity scores of infants receiving PD decreased 14% within 24 hours of initiation of therapy, compared with a 7% reduction in the control cohort over the same period of time (P not significant). No infectious, metabolic, or mechanical/traumatic morbidity resulted from PD catheter placement, use, or removal, and there was no significant difference in postoperative mortality between groups.

Thus we were able to document that PD is an effective and safe means of postoperative fluid management even in the most critically ill postcardiotomy infants. Our data would appear to dismiss the argument that PD is ineffective in patients with impaired cardiac output due to decreased mesenteric perfusion. When compared with conventional postsurgical management strategies, use of PD may hold additional advantages related to facilitation of early postoperative recovery. These effects have not been routinely evaluated, as some investigators have excluded patients who underwent fewer than 24 hours of renal replacement therapy.[37] This reporting bias could underestimate the utility of PD for those patients who we have observed to benefit greatly from a short course of dialysis.

We have noted an extremely low mortality rate among our series of PD patients. Of the 44 patients who underwent PD, 8 (18%) have died. This compares favorably with a recently published series[19] in which the mortality rate was 46.9%. Indications for PD and its frequency of use were comparable with those in our institution.

We have also observed a much lower complication rate for both the placement of PD catheters and institution of PD than reported in the literature.[19,37] Among all 209 patients who received PD catheters intraoperatively at TCH, only 9 (4.3%) have developed complications. These consisted of 6 patients who developed omental herniations upon catheter removal (requiring brief operative reductions), 2 with minor wound dehiscences at catheter insertion sites (treated with routine wound care), and 1 with small bowel obstruction necessitating laparotomy and adhesion takedown. The complication rate among patients undergoing PD (13.6%) was higher than that of the overall group of patients who received PD catheters. We did not observe peritonitis as a complication of PD or significant dialysate leakage around the catheter insertion site, 2 complications commonly cited in the literature. Intraoperative PD catheter placement may enable heightened sterility and improved localization of the instrument through placement under direct vision.

Although we have not formally evaluated postcardiotomy pulmonary function after extrarenal fluid removal, other investigators[36,42] have documented improved oxygenation indices and pulmonary mechanics after both CAVH and modified ultrafiltration. Furthermore, Werner and associates[19] have recently demonstrated modest improvements in the mean airway pressures and alveolar-arterial oxygen gradients of children receiving postcardiotomy PD, although this study was not controlled for the effect of time. These observations parallel our own experience of improved pulmonary mechanics after PD-mediated extravascular fluid removal.

The mechanisms by which postcardiotomy renal replacement therapies improve renal and cardiorespiratory function are certain to be multiple but most likely relate to extravascular fluid removal (i.e., prevention of generalized edema) and elimination of circulating inflammatory mediators. Several studies have documented the beneficial effects of reduction of total body water after CPB. Elliott,[29] using post-bypass ultrafiltration, demonstrated an increase in

myocardial contractility accounted for by a decrease in myocardial wall volume or myocardial edema. Others[29,31,43] have shown improvement in pulmonary mechanics via reductions in pulmonary edema (resulting in increased lung compliance, decreased pulmonary vascular resistance, and improved oxygenation) and improved hematologic status through hemoconcentration (manifest as a reduction in blood loss and the need for transfusion therapy).

The clinical consequences of inflammatory mediator removal after CPB remain controversial. Although the precise ability of PD to remove proinflammatory elements has yet to be adequately quantified, numerous investigations have shown convincingly that inflammatory mediators (e.g., TNFα, C3a, C5a, and interleukin-1β, -6, and -8)[28-30,33,44] and cardioinhibitory substances (e.g., myocardial depressant factor) are removed by hemofiltration.[43] These findings have been associated with diminished postbypass pulmonary injury and augmented myocardial function. However, a direct cause-and-effect relationship between removal of the measured substances and improvement in clinical endpoints has not been proved.[26] It may be true that those mediators removed by renal replacement therapy have already performed their deleterious functions at the local tissue/organ level before release into the circulation. Also unclear is the potentially beneficial role that may be played by extracted inflammatory elements with regard to wound healing, protection from ischemic injury, and inhibition of cytokine release.[26]

We conclude that PD is a safe and effective form of renal replacement therapy, even among critically ill pediatric postcardiotomy patients. Early postsurgical institution of PD may greatly facilitate fluid and electrolyte management and hasten early postoperative recovery. We believe that intraoperative catheter placement reduces the complication rate associated with this treatment modality.

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