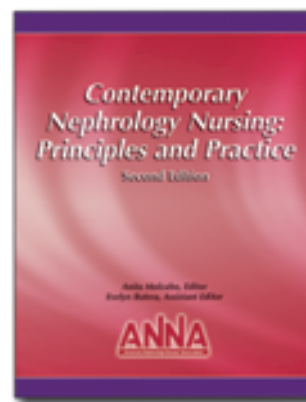


Editor's Note The revised *Contemporary Nephrology Nursing: Principles and Practice (2nd edition)* textbook has recently been published by ANNA. The Second Edition was edited by Anita Molzahn, PhD, RN, Editor, and Evelyn Butera, MSN, RN, CNN, Assistant Editor. The following is an excerpt of Chapter 20, "The Child With Kidney Disease," by Cyrena Gilman, MN, RN, CNN, and Annette Frauman, PhD, RN, FAAN.

The remainder of the chapter, which includes an overview of Pediatric Treatment Modalities, Specific Nursing Interventions, An Overview of Normal Life, and Information Programs Assisting the Family, may be found in the newly published *Contemporary Nephrology Nursing: Principles and Practice*.

For additional information on purchasing *Contemporary Nephrology Nursing: Principles and Practice (2nd edition)*, please refer to the order form immediately following this article.



The Child with Kidney Disease

Cyrena Gilman, MN, RN, CNN
Annette Frauman, PhD, RN, FAAN

This chapter will present an overview of care of the child with all stages of chronic kidney disease (CKD). No attempt will be made to repeat information that is identical to that for adult patients. The age range to be covered will include children from birth to 20 years. Although the American Academy of Pediatrics (AAP) defines the age range of pediatrics as birth to 21 years, much of the data, such as the United States Renal Data System (USRDS) related to children with end stage renal disease (ESRD), goes up to age 20. Pediatric nurses provide care to children and parents within the context of families. Therefore, additional emphasis will be given to families of children in this chapter.

Philosophy of Pediatric Renal Nursing

A philosophy about the care of children is an essential component of the pediatric nephrology nursing approach. The following philosophical assumptions guide this chapter.

Kidney disease, its symptoms, and the side effects of treatment present many impediments to attaining a normal lifestyle. Each of the treatment modalities commonly used for children with CKD imposes time constraints and limitations that detract from the ability to attend school and play as other healthy children do (Fielding & Brownbridge, 1999). Children with CKD and their families need as much normality in their lives as possible within these constraints and look to nurses for help with achieving as normal a lifestyle as possible.

Health care providers and family members should hold the same expectations for children with kidney disease as for healthy children of similar developmental levels. Providers should expect developmentally congruent behaviors from these children. Chronically ill children need to participate in their own treatment regimen, should not be indulged or treated specially based solely on having the illness, or be treated as a "poor thing" (Gilman & Frauman, 1979). Children who are frequently

referred to as a "poor thing," verbally or in other ways, will soon come to believe it. They will not progress developmentally, will not become adequately socialized, and will not be prepared to become functional adults, whether sick or well at that stage of life. In order to avoid becoming a "poor thing," children must be encouraged to be as independent as possible for their developmental levels.

Brief History of Pediatric Nephrology Care

At one time, conservative management was the only modality available to children with CKD, and most children with CKD died. Occasionally children were kept alive with intermittent peritoneal dialysis (IPD), but the widely varying serum chemistries and frequent admissions to the ICU for painfully invasive procedures effectively prevented any semblance of normality in their lives. If renal function did not return, the treatment was discontinued, and again, most children died.

In the mid-to-late 1960s, a few centers began to attempt hemodialysis (HD) of children, usually adolescents, as a last resort to preventing death. In 1971, Dr. Kjellstrand and colleagues at the University of Minnesota determined that 10% of circulating blood volume could be spared in an extracorporeal circuit. Based on that discovery, medical equipment manufacturers began producing smaller hemodialyzers and bloodlines to accommodate pediatric patients with chronic renal failure. However, with chronic HD, diets were severely restricted, external arteriovenous shunts were frequently problematic, and intradialytic complications and anemia were widespread.

The peritoneum was not employed for renal therapy until 1948-1949. In 1960, Dr. Harold McDonald at the University of Michigan developed procedures for inserting peritoneal catheters with a trocar and automated delivery systems for peritoneal dialysate, which helped further the care of younger patients (McBride, 1984). However, Moncrief and Popovich's development of continuous

Chapter 20

ambulatory peritoneal dialysis (CAPD) in 1976 resulted in the widespread successful use of PD for treating pediatric patients. It was first used on a child in Toronto in 1978.

A working kidney transplant has been the goal for pediatric patients with CKD for many years (Alexander, 1990). Improved surgical techniques and better immunosuppressive medications have aided achieving that goal. However, none of the recent improvements in transplantation have been specific to pediatrics.

Epidemiological Data

The Medicare chronic renal failure program recorded the admission to services of 1,349 children between the ages of birth and 19 (incident patients) in 2001. Incident rates per million population were: 10 for ages 0-4, 8 for ages 5-9, 15 for ages 10-14, and 28 for ages 15-19. Males have higher incidence rates among children in all four age groups (USRDS, 2003). Prevalence rates per million population were 27 for ages 0-4, 43 for ages 5-9, 84 for ages 10-14, and 157 for children ages 15-19.

Treatment modalities for children differ sharply from those of adults, with far greater use of peritoneal dialysis (PD) and transplant as first modality, compared with adults (USRDS, 2003). After 2 years of treatment, 31% are receiving HD, 15.2% CAPD or continuous cycling peritoneal dialysis (CCPD), and 44.9% have a functioning transplant (USRDS, 2003). Comparisons of cohorts from 1987-1991 and 1992-1996 show that overall incident survival has not changed. However, mortality analyses show a 4% fall in annual mortality rates for children age 0-9 and a much larger decline of 21% for those children age 10-19 (USRDS, 2003).

Etiologies of CKD in Children

The etiologies of CKD are quite different in children than in adults. For example, diabetes and hypertension, two major causes of kidney disease in adults, are rarely causes of CKD in children. Prevalence rates for children with diabetes as a primary cause of kidney failure are 3 per 10 million population (USRDS, 2003). In contrast, for all kidney failure patients, the rate was 3,119. Diabetes does occur in children, but the degree of damage necessary to result in CKD usually takes more than 20 years to evolve, by which time the child has become an adult (Mogyorosi & Ziyadeh, 2001).

Serious kidney disease in children may result in hypertension, but essential hypertension is rarely seen in children. The most frequent causes of CKD in children can be broken down into two major categories: congenital and acquired (USRDS, 2003). Males predominate among pediatric patients, especially among those with obstructive conditions and other congenital/hereditary conditions (USRDS, 2003). Most frequently, CKD that has its onset between birth and age 5 is due to congenital disease. That which occurs between ages 6 and 10 is most often a result of urological defects, which are also congenital, and after age 12, glomerulonephritis is the most frequent cause of CKD in children.

Congenital

Malformations of the urinary tract are extremely common, probably related to the complex embryology of the kidney and urinary tract. The human kidney is first detectable in the fifth gestational week. It is initially composed of a small epithelial bud surrounded by undifferentiated mesenchymal stem cells. The ureteric bud is a branch from the mesonephric or Wolffian duct and forms the calyces, collecting tubules, and ureters. Each newly formed collecting tubule has a metanephric tissue cap at the distal end. Cells within this cap form small vesicles that give rise to s-shaped tubules. Capillaries grow into one end of the S and become glomeruli. The tubules with their glomeruli form nephrons (Sadler, 2000). Similarly, the embryonic cloaca differentiates into a male or female perineum, urinary bladder, and urethra, also through a complex process (Stephens, Smith, & Hutson, 2002). These complex processes can result in abnormalities at any stage. While many malformations are non-pathogenic, such as a solitary kidney, others result in a complete lack of renal function or obstructions of the urinary tract causing renal damage in fetal life.

Obstructive uropathy may be associated with dysgenesis, or damage may occur because of back pressure of urine and/or resulting infections of the urinary tract. Obstructions may occur at any point along the urinary tract, from the meatus to the renal pelvis. These may be formed of abnormal structures, misplaced vessels, or valves where there should be none. Abnormal innervation to the ureters, such as that seen in prune-belly syndrome and other obstructive uropathy, slows or causes cessation of the normal peristalsis of the ureter, resulting in an obstruction just as an ileus, or nonfunctioning bowel, causes gastrointestinal (GI) obstruction. Other congenital syndromes cause renal failure through abnormalities of the glomerulus, including Alport's syndrome, nail-patella syndrome, Fabry disease, and Charcot-Marie-Tooth syndrome (Stephens et al., 2002). Inherited errors of metabolism, such as cystinosis and oxalosis, also can cause renal failure in children and infants. Autosomal recessive polycystic kidney disease (ARPKD) is a congenital form of polycystic disease that only occurs in children.

Acquired

Many cases of acute glomerulonephritis in children follow infection with an organism, including group A beta hemolytic streptococci, and viruses such as varicella, Epstein-Barr, rubeola, and cytomegalovirus. Often, the precipitating organism is not identified. Pathology of the glomerulus results from infiltration of the tissues with antibodies, resulting in enlargement of the glomeruli and obliteration of capillary lumens. The degree of luminal obliteration correlates with decreased glomerular filtration rates. In some children, especially those with initially severe disease, progression to renal failure occurs, though often slowly. Hemolytic uremic syndrome also causes acute renal failure in children with resulting damage that may or may not be permanent.

Other types of glomerulonephritis cause damage to the glomerulus and the basement membrane with progression to end stage kidney disease in some cases. Collagen vascular diseases, malignancies, and hemoglobinopathies may cause CKD in children just as they do in adults. Increased understanding of the interaction between genetic factors and external insults, such as infection or toxins, is adding to our understanding of the causes, prognosis, and treatment of glomerular disorders (Dell, McDonald, Watkins, & Avner, 2004).

Effects of Renal Failure on Children

Many of the effects of renal failure on children and adults are identical; others are markedly different due in part to children's unique metabolic state that results from growth, their height-to-weight ratio, and their higher percentage of body water.

Cardiovascular

The cardiovascular effects of renal failure in children are similar to those seen in adults, including hypertension, which can result in retinopathy and encephalopathy if unchecked; circulatory overload; edema; congestive heart failure; pericarditis; and arrhythmias. Hypertension in children with CKD is usually caused by extracellular volume expansion and is controlled by ultrafiltration (UF) (Solhaug, Adelman, & Chan, 1992). Children also may have marked left ventricular hypertrophy with considerable lateral displacement of the point of maximum impulse (PMI) (Morris, Skinner, Wren, Hunter, & Coulthard, 1993). Recent evidence has indicated that, like adults, children with CKD are at greatly increased risk of cardiovascular disease (Chavers & Schnaper, 2001; Parekh, Carroll, Wolfe, & Port, 2002). In a study of deaths among pediatric patients, Parekh et al. (2002) report that 25% were cardiac-related. Children with CKD are at increased risk of cardiovascular disease due to vascular calcifications, hypertension, and hyperlipidemia (Chavers & Schnaper, 2001).

Cutaneous

The cutaneous effects of CKD on children are quite obvious upon even cursory observation. The overall appearance is relatively pale or sallow in spite of receiving erythropoietin. Accumulation of urinary pigments in the skin may occur, just as they do in the adult. The skin is somewhat less likely to be dry or scaly in younger children with CKD, and itching is less frequently observed than in adults.

As in adults, when dialysis is discontinued or is not possible, urea may be deposited on the skin as sweat evaporates. The resulting crystal formations resemble frost and are thus called uremic frost. Bruising is sometimes noted in children with CKD. This is due to the capillary fragility resulting from CKD and is a sequela of most of the treatment modalities requiring heparinization.

Gastrointestinal (GI)

Children with CKD are quite likely to suffer frequently from nausea and anorexia, sometimes progressing to vomiting. The anorexia may be due in part to changes in taste, particularly a constant metallic aftertaste, but is generally not well understood. The problem can be profound and interfere seriously with the child's nutritional needs. In addition, children with CKD, because of their compromised immune state, are susceptible to infections of the GI tract as well as to GI bleeds. Because children who have an infection of the urinary tract often experience nausea and vomiting, in contrast to the symptoms of frequency, urgency, and pain commonly seen in adults, these symptoms should be evaluated in children with that fact in mind.

Genitourinary

Children with CKD secondary to congenital causes are much more likely to have high-output renal failure than are adults. The ability of these children with CKD to concentrate their urine is greatly impaired, and they may have a fixed urinary specific gravity with a total inability to respond to fluctuations in fluid status. In these children, fluid is removed by the kidney(s), but the urine specific gravity is extremely low, and other substances are poorly excreted at best. Serum blood urea nitrogen (BUN), creatinine, and electrolyte levels may resemble those of oliguric or anuric patients. Children, especially those with nephrotic syndrome, can have extremely high urine proteins to the point that the urine will gel if refrigerated.

Female adolescents with CKD may experience amenorrhea and anovulation even after the onset of puberty, which is often greatly delayed. Some pediatric nephrologists may prescribe hormonal contraceptives to adolescent females to prevent menses in an attempt to minimize additional blood loss. They may not have a corresponding loss of libido, however, and may have difficulty with vaginal lubrication and painful intercourse, just as adults with renal failure do. Similarly, males have delayed puberty and may suffer from impotence and sterility. However, the adolescent sex drive is powerful, and loss of libido and sterility should not be depended on to inhibit either male or female adolescents. Despite decreased likelihood of pregnancy, sexually active adolescents with CKD need to use birth control measures. Choosing an appropriate method may not be easy because of the need to balance safety and efficacy. Barrier methods (diaphragm, condom) are safest, but may not be well accepted by adolescents and are likely to be ineffective due to sporadic use. Intrauterine devices are contraindicated, and hormonal contraceptive methods may not be well tolerated. Additionally, because of their suppressed immune system, sexually transmitted diseases (STDs) are particularly hazardous for adolescents with CKD. They should be given information about preventive measures before they become sexually active. Hergenroeder and Brewer (2001) report that only 56% of pediatric nephrologists say they interview patients without parents present. The pediatric

nephrologists also stated that they ask only 53% of male patients and only 55% of female patients about intercourse.

Hematologic

Like adults, anemia is a problem for children with CKD, though this problem has been considerably diminished with the advent of recombinant erythropoietin. Darbepoetin has not yet been approved for use in the pediatric patient population, but the decreased frequency of dosing schedule is as attractive to children and their families as it is to adults.

Children who are posttransplantation and receiving azathioprine should be carefully monitored for its effects on bone marrow function, including diminished thrombocytes, erythrocytes, and leukocytes. Many centers are discovering that oral iron supplements do not adequately maintain hemoglobin in the optimal range, especially for children receiving HD. In that event, intravenous (IV) iron supplements may be required. Administration of IV iron causes a significant increase in serum ferritin (Warady, Schaefer, Alexander, Firanek, & Mujais, 2004). In fact, a maintenance IV iron protocol of 2 mg/kg/wk for pediatric HD patients reduced the dose of erythropoietin needed to maintain optimal blood hemoglobin levels. It was also more economical; improved efficacy of erythropoietin doses resulted in a 26% reduction in cost (Morgan, Gautam, & Geary, 2001).

Metabolic

Children with CKD are more likely than adults to experience metabolic acidosis. However, the other metabolic effects of CKD, including azotemia, hyperkalemia, sodium retention or wasting, hypermagnesemia, hyperuricemia, and lipid abnormalities, are similar.

Neuromuscular

Neuromuscular effects of CKD on children are also similar to those seen in adults. However, problems with growth and development are particularly problematic. Fatigue, muscle wasting, and lethargy greatly interfere with the developmental drive of children and affect their ability to perform new skills and tasks. Other neuromuscular effects are discussed in the section on growth and development.

Pulmonary

The pulmonary effects of CKD in children are similar to those in adults. Because of the child's smaller size, a relatively small amount of excess fluid may result in pulmonary edema. Children with active glomerular disease may have pulmonary edema secondary to fluid shifts. Additionally, the hypercoagulable state of active nephrotic syndrome can contribute to pulmonary thrombus formation. The respiratory status of infants or children with pulmonary compromise may be worsened by retained peritoneal dialysate, retained fluid post HD, or excessive intradialytic weight gains.

Children with renal damage in utero may not produce sufficient quantities of urine to produce adequate amniotic fluids (oligohydramnios), which results in pulmonary hypoplasia (Limwongse & Cassidy, 2004). These children may have compromised pulmonary function in addition to kidney disease after birth.

Skeletal

The effects of CKD on bone in children are devastating and include abnormalities of metabolism such as hypocalcemia, hyperphosphatemia, and hyperparathyroidism. Renal osteodystrophy is common along with pathologic fractures due to demineralized bone. Calcium salts may be deposited in soft tissue (around joints, in blood vessels, heart, lungs, and conjunctiva).

A particularly devastating effect of long-term renal failure and dialysis is the deposition of beta-2 microglobulin in the soft tissues and bones. As in adults, the longer one is on dialysis and exposed to elevated levels of beta-2 microglobulin, the more likely one is to have this type of amyloidosis. If a pediatric patient rejects several transplants at a young age and faces the remainder of life on some type of dialysis, development of beta-2 microglobulin bone disease is inevitable (McCarthy, Williams, & Johnson, 1994). McCarthy et al. (1994) found that CAPD patients are less likely to develop beta-2 microglobulin disease than are HD patients, and patients hemodialyzed against high-flux synthetic membranes have a lower incidence than those dialyzed against cellulose acetate membranes. The progression of amyloid deposition is delayed with synthetic membrane HD, but not prevented. The researchers found that most patients will begin to develop the syndrome after 4 or 5 years of therapy; when the number of "beta-2 microglobulin months" (the serum beta-2 microglobulin level times the number of months of dialysis) exceeds 1500, the disease will definitely be present. Only natural kidney function, whether from native kidneys or a transplant, can completely avoid this devastating problem.

Immune

Children with CKD, like adults, have long been noted to have decreased resistance with an increased incidence of bacterial, viral, and fungal infections. Uremic patients also have a much-higher incidence of malignancies than the normal population (Benfield & Michael, 1992). In a study of children receiving CAPD, significantly lower serum IgG levels were found than in the controls, but all levels were in the normal range. Additionally, these children had essentially normal responses to live virus vaccines for measles, mumps, and rubella (MMR) (Hisano et al., 1991).

Uremic patients have normal to slightly elevated granulocyte counts, but the migration and chemotactic function of these cells is usually slightly abnormal. Phagocytosis, on the other hand, is typically normal (Benfield & Michael, 1992).

Numerous investigators have found widely varying

results in studying the number and function of lymphocytes in uremic patients. Although normal levels of IgG, IgA, and IgM are found in uremic patients, seroconversion and antibody titer formation are usually impaired. Impaired T-cell function, increased suppressor cell activity, intrinsic monocyte/macrophage dysfunction, and abnormal production of interleukin-2 have been demonstrated in uremic patients but are difficult to reproduce in animals with experimentally-induced uremia. Uremic patients also have been noted to have thymic atrophy at autopsy (Benfield & Michael, 1992).

The vitamin and mineral deficits associated with the malnutrition present in most uremic patients have serious effects on the immune system. Some kidney diseases are treated with long-term immunosuppressive medication before end stage kidney disease is reached. Thus, the impaired immune system function found in uremic patients is most likely influenced by many factors, not the uremic condition alone (Benfield & Michael, 1992).

Children with CKD require immunizations to prevent communicable disease just as other children do. However, live virus vaccines should not be given to immunosuppressed children, whether pre or posttransplant, necessitating that the MMR vaccine and varicella vaccine be given during treatment for early stage kidney disease or while undergoing dialysis. A need for repeat doses of varicella vaccine (Furth et al., 2003) and pneumococcal vaccines (Fuchshuber et al., 1996) in order to produce immunity has been reported. Laube et al. (2002) describe a suggested protocol for children with CKD that resulted in no infections with the diseases for which the children were immunized except for varicella.

It is critical that all live virus vaccines be given before transplantation, as the immune-suppressed child's response to these will be altered. As children with kidney failure often do not have, or do not see, a primary care provider, the responsibility for ensuring that this is done often devolves upon the pediatric nephrology nurse.

Growth and Development

Children with CKD have the same developmental needs as healthy children. They may need additional assistance from parents, health care providers, educators, and others to achieve their maximum potential at each of the developmental stages. Certainly prevention of developmental problems is preferred to intervention after they have occurred. The focus needs to be positive: Enhance what the child can do, encourage progress, and de-emphasize deficits or handicaps.

Children are usually approached based on the age they appear to be. For the child with short stature or delayed puberty due to CKD, this is inappropriate. In order to help the child with CKD achieve as normal a life as possible, the pediatric nephrology nurse must first determine exactly what developmental level the child has attained. In determining this, the whole child must be observed. The child's behavior; relationships with family, friends, and peers; fine and gross motor coordination; and

coping mechanisms all help establish what his or her developmental levels in those domains are. In addition, the child's likes and dislikes, as well as abilities compared with other children of the same chronological age must be considered.

Children with CKD go through the same developmental stages that other children do. However, the rate at which they progress through the stages is often markedly delayed and not necessarily consistent across developmental domains. Generalized psychological assessment tools for younger children (birth to 6 years), such as the Denver Developmental Screening Test (DDST), the Goodenough Draw-A-Man test, and the Brazelton Scale for Infant Development, can help in establishing a developmental level. The Vineland Adaptive Behavior Scales can be used clinically to determine which social and adaptive skills the child possesses and, by implication, which ones need to be worked on next. It is an excellent idea to have testing conducted by a child psychologist using global measures such as the Bayley and the Weschler Intelligence Scale for Children (WISC). The evaluation should also include tests of neuropsychological function, such as Raven's Progressive Matrices; the Beery Developmental Test of Visuomotor Integration; and tests of vigilance, auditory memory, and reaction time, since these have been shown to be affected by CKD (Fennell et al., 1990). These tests will provide an in-depth examination of the child's developmental attainments, which can be used in patient education or in assisting the child and family to obtain appropriate school placement. If a clinical or developmental psychologist is not available on the team caring for the child, their services may sometimes be obtained through the school system, a nearby university's psychology department, or a child psychiatry facility. Children with CKD from birth are more likely to suffer from much more severe developmental disabilities than children who acquire the disease later (Crocker et al., 2002)

Physical growth and sexual maturation. Physical growth failure is perhaps the most pervasive developmental issue for children with CKD. Despite numerous studies, the mechanisms of growth failure are still not well understood (Watkins & Richards, 2004). Less than optimal nutrition; metabolic acidosis; fluid and electrolyte disturbances; disorders of calcium and phosphorus metabolism; medications used in treatment, especially corticosteroids; and decreased production and diminished ability to use hormones have all been implicated in growth failure (Mehls, Blum, Schaefer, Tonshoff, & Scharer, 1992). The anorexia common in children with renal insufficiency and failure undoubtedly contributes. Additionally, children with severe protein-wasting glomerular disease have a greater tendency toward malnutrition. Probably all of these factors and perhaps others are influential. How each or the combination results in growth failure is not well known.

In general, the earlier the onset of renal failure and the longer its duration, the more severe growth deficien-

cies will be. Therefore, children with congenital kidney disease will suffer greater growth retardation than those with acquired kidney disease. Height reductions are most profoundly affected in infancy and adolescence, the periods in which growth is most rapid. Once growth potential is lost in these time periods, it will probably never be regained (Mehls et al., 1992). Children with renal disease often have a relatively normal growth curve, but usually remain well below the 3rd percentile of normal parameters. Additionally, adult height is usually reduced by up to 2.5 standard deviations.

Puberty, with its rapid growth and maturation of secondary sex characteristics, is often delayed and its intensity diminished. In a 1990 study, Scharer noted that puberty was delayed in their subjects by an average of 2.5 years, and the growth attainment in the prepubertal growth spurt was significantly impaired.

Use of human growth hormone has become a standard treatment for children with CKD. Children with CKD have normal or elevated levels of the hormone but are apparently not able to use their natural supply. The administration of additional hormone does enhance growth as demonstrated by numerous studies (Benfield, Parker, Waldo, Overstreet, & Kohaut, 1993; Fine et al., 1994; Koch et al., 1989). The earlier growth failure is documented and treatment with growth hormone begun, the better the final height outcome (Fine, Sullivan, & Tejani, 2000).

In order for growth hormone to work most effectively, the pediatric patient's nutrition must be optimal. Anemia, metabolic acidosis, and renal osteodystrophy must be treated and corrected. The child must have remaining growth potential, which is shown by open epiphyses and a Tanner stage of less than 5. Ongoing monitoring of length or height, weight, growth velocity, bone age, and Tanner stage is essential during the administration of growth hormone therapy (Miller, Macdonald, Kolnacki, & Simek, 2004).

Cognitive development. The effects of CKD on cognitive development of infants have been the subject of a number of studies. These studies have primarily evaluated the efficacy of various treatment modalities in preventing detrimental effects of CKD on neurological development. Polinsky, Kaiser, and Stover (1987) concluded from a review of 15 of these studies that CKD most consistently affected gross motor and language development. Development of infants is improved after renal transplantation, with studies showing improvement on a variety of measures, including the DDST, the Bayley, and the Revised Yale Developmental Schedule. However, neonates with CKD continue to have developmental delays even after transplantation.

Studies of the effects of CKD on cognitive development in older children have used both global measures of intelligence (for example, Stanford-Binet, WISC-R) as well as specific neuropsychological measures such as tests of vigilance, auditory memory, and reaction time. Associations have been found between age at onset of CKD, length of time in renal failure, treatment method,

and a variety of measures of cognitive development (Fennell et al., 1990). Based on their body of work, Fennell and colleagues (1990) have advanced the hypothesis that renal failure affects cognitive development in two ways: one that influences the acquisition of new skills and is less likely to improve after successful transplantation, and another that affects attention, speed of processing, and modulation of responses. This latter effect seems to be dependent on abnormal blood chemistries and is, thus, more susceptible to improvement after successful transplantation (Fennell et al., 1990). A 2000 study (Brouhard et al.) that compared 62 children with ESRD with sibling controls found that the average IQ percentile rank for the patients was significantly lower than their siblings. Patients also tended to score lower on achievement tests in spelling, arithmetic, and reading. A review article published in 1994 (Frauman & Myers, 1994) contains numerous references and further information.

School attendance in children with CKD has received some attention, mostly in children who have received successful transplantation. Studies of these children are optimistic. In 1991, Morel and colleagues reported that of 9 transplant recipients of school age, all were attending; of 48 older than high school age, 93% had graduated, and 60% had attended college or technical training. A German study reported a high school completion rate of 14% (Rosenkranz, Bonzel, & Bulla, 1992), while a European Dialysis and Transplant Association (EDTA) study reports that of 617 adult subjects receiving renal replacement therapy as children, 41% did not complete school and only 58% were employed (Ehrich et al., 1992).

Psychosocial development. The effects of CKD on psychosocial development are not as devastating as once feared. Children with CKD seem to be quite resilient psychologically, especially those with successful renal transplants. Children treated at home with either HD or PD fare better than those on incenter treatment. Psychosocial adjustment is better for those with transplants than for those on either mode of dialysis. Most studies of children with CKD as they approach adulthood have found few problems except in the areas of growth failure and the formation and maintenance of intimate relationships (Beck, Nethercut, Crittenden, & Hewins, 1986; Morel et al., 1991).

In a 1996 study, Frauman, Gilman, and Carlson found that scores on the Vineland Adaptive Behavior Scales, a measure of psychosocial development, were more than two thirds of a standard deviation below the age standard mean scores. Percentile ranks for subscales for communication, daily living skills, and socialization were 19, 21, and 23 respectively. There was no significant difference between the group of subjects with a functioning transplant and those who had returned to dialysis following a transplant failure.

References

- Alexander, S.R. (1990). Controversies in pediatric renal transplantation. *American Kidney Fund Letter*, 7, 5-21.
- Andreoli, S.P., Langefeld, C.D., Stadler, S., Smith, P., Sears, A., & West, K.

- (1993). Risks of peritoneal membrane failure in children undergoing long-term peritoneal dialysis. *Pediatric Nephrology*, 7, 543-547.
- Beck, A.L., Nethercut, G.E., Crittenden, M.R., & Hewins, J. (1986). Visibility of handicap, self-concept, and social maturity among young adult survivors of end stage renal disease. *Developmental and Behavioral Pediatrics*, 7, 93-96.
- Bell, S.B. (1988). CAVH in pediatrics: Meeting the challenge. *ANNA Journal*, 15, 25-26.
- Benfield, M.R. (2003). Current status of kidney transplant: Update 2003. *Pediatric Clinics of North America*, 50, 1301-1343.
- Benfield, M.R., & Bunchman, T.E. (2004). Management of acute renal failure. In E.D. Avner, W.E. Harmon, & P. Niaudet (Eds.), *Pediatric nephrology* (5th ed.) (pp. 1253-1266). Philadelphia: Lippincott Williams and Wilkins.
- Benfield, M., & Michael, A.F. (1992). Immunology of uremia. In C.M. Edelmann (Ed.), *Pediatric kidney disease* (2nd ed.) (pp. 783-790). Boston: Little, Brown and Company.
- Benfield, M., Parker, K.L., Waldo, F.B., Overstreet, S.L., & Kohaut, E.C. (1993). Treatment of growth failure in children after renal transplantation. *Transplantation*, 55, 305-308.
- Betz, C.L., & Sowden, L.A. (2004). *Mosby's pediatric nursing reference* (5th ed.). St. Louis: Mosby.
- Brem, A.S., Lambert, C., Hill, C., Kitsen, J., & Shemin, D.G. (2001). Clinical morbidity in pediatric dialysis patients: Data from the network 1 clinical indicators project. *Pediatric Nephrology*, 16, 854-857.
- Chadha, V., Warady, B.A., Blowey, D.L., Simckes, A.M., & Alon, U.S. (2000). Tenckhoff catheters prove superior to cook catheters in pediatric acute peritoneal dialysis. *American Journal of Kidney Diseases*, 35, 1111-1116.
- Chavers, B., & Schnaper, H.W. (2001). Risk factors for cardiovascular disease in children on maintenance dialysis. *Advances in Renal Replacement Therapy*, 8, 180-190.
- Crocker, J.F.S., Acott, P.D., Carter, J.E.J., Lirenman, D.S., MacDonald, G.W., McAllister, M., Mc Donnell, M.C., Shea, S., & Bawden, H.N. (2002). Neuropsychological outcome in children with acquired or congenital renal disease. *Pediatric Nephrology*, 17, 908-912.
- Dell, K.M., McDonald, R.A., Watkins, S.L., & Avner, E.D. (2004). Polycystic kidney disease. In E.D. Avner, W.E. Harmon, & P. Niaudet (Eds.), *Pediatric nephrology* (5th ed.) (pp. 675-699). Philadelphia: Lippincott Williams and Wilkins.
- Dracopoulos, D.T., & Weatherly, J.B. (1983). Chronic renal failure: The effects on the entire family. *Issues in Comprehensive Pediatric Nursing*, 6, 141-146.
- Ehrich, J.H., Rizzoni, G., Broyer, M., Brunner, F.P., Brynger, H., Fassbinder, W., Geerlings, W., Selwood, N.H., Tufveson, G., & Wing, A.J. (1992). *Nephrology Dialysis and Transplantation*, 7, 579-586.
- Evans, J.H.C., Smye, S.W., & Brocklebank, J.T. (1992). Mathematical modeling of haemodialysis in children. *Pediatric Nephrology*, 6, 343-353.
- Fennell, R.S., Fennell, E.B., Carter, R.L., Mings, E.L., Klausner, A.B., & Hurst, J.R. (1990). A longitudinal study of the cognitive function of children with renal failure. *Child Nephrology and Urology*, 10, 199-204.
- Fielding, D., & Brownbridge, G. (1999). Factors related to psychosocial adjustment in children with end stage renal failure. *Pediatric Nephrology*, 13, 766-770.
- Fine, R.N., Koch, V.H., Boechat, M.I., Lippe, B.H., Nelson, P.A., Fine, S.E., & Sherman, B.M. (1994). Growth after recombinant human growth hormone treatment in children with chronic renal failure: Report of a multicenter randomized double-blind placebo controlled study. *Journal of Pediatrics*, 124, 374-382.
- Fine, R.N., Sullivan, E.K., & Tejani, A. (2000). The impact of recombinant human growth hormone treatment on final adult height. *Pediatric Nephrology*, 14, 679-681.
- Frankenfield, D.L., Neu, A.M., Warady, B.A., Watkins, S.L., Friedman, A.L., & Fivush, B.A. (2002). Adolescent hemodialysis: Results of the 2000 ESRD clinical performance measures project. *Pediatric Nephrology*, 17, 10-15.
- Frauman, A.C., & Gilman, C.M. (1985). "Normal life" - A goal for the child with chronic renal failure. *ANNA Journal*, 12, 192-195.
- Frauman, A.C., & Gilman, C.M. (1989). Creating a therapeutic environment in a pediatric renal unit. *ANNA Journal*, 16, 20-22, 26.
- Frauman, A.C., & Gilman, C.M. (1990). Care of the family of the child with end stage renal disease. *ANNA Journal*, 17, 383-386, 401.
- Frauman, A.C., Gilman, C.M., & Carlson, J.R. (1996). Rehabilitation and social and adaptive development of young renal transplant recipients. *ANNA Journal*, 23, 467-471, 484.
- Frauman, A.C., & Myers J.T. (1994). Cognitive, psychosocial, and physical development in infants and children with end stage renal disease. *Advances in Renal Replacement Therapy*, 1, 49-54.
- Fuchshuber, A., Kuhnemund, O., Keuth, B., Luticken, R., Michalk, D., & Auerfeld, U. (1996). Pneumococcal vaccine in children and young adults with chronic renal disease. *Nephrology Dialysis and Transplant*, 11, 468-473.
- Furth, S.L., Hogg, R.J., Tarver, J., Moulton, L.H., Chan, C., & Fivush, B.A. (2003). Varicella vaccination in children with chronic renal failure. *Pediatric Nephrology*, 18, 33-38.
- Gilman, C.M., & Frauman, A.C. (1979). Psychosocial care of the child in renal failure. *AANNT Journal*, 6, 143-148.
- Gilman, C.M., & Frauman, A.C. (1982). Dialysis desensitization: Preparation of the very young child for chronic hemodialysis. *Dialysis and Transplantation*, 11, 660-661.
- Gilman, C.M., & Frauman, A.C. (1987). Use of play with the child with chronic illness. *AANNT Journal*, 14, 259-261.
- Goldstein, S.L. (2001). Hemodialysis in the pediatric patient: State of the art. *Advances in Renal Replacement Therapy*, 8, 173-179.
- Goldstein, S.L., & Jabs, K. (2004). Hemodialysis. In E.D. Avner, W.E. Harmon, & P. Niaudet (Eds.), *Pediatric nephrology* (5th ed.) (pp. 1395-1410). Philadelphia: Lippincott Williams and Wilkins.
- Gruskin, A.B., Baluarte, H.J., & Dabbagh, S. (1992). Hemodialysis and peritoneal dialysis. In C.M. Edelmann (Ed.), *Pediatric kidney disease* (2nd ed.) (pp. 827-916). Boston: Little, Brown and Company.
- Gunn, V.L., & Nechyba, C. (2002). *The Harriet Lane handbook. A manual for pediatric house officers*. Philadelphia: Mosby.
- Hardgrove, C. (1980). Children respond to therapeutic art. *Hospitals*, 54, 67-69.
- Harmon, W.E., & Jabs, K. (1994). Hemodialysis. In M.A. Holliday, T.M. Barratt, & E.D. Avner (Eds.), *Pediatric nephrology* (3rd ed.) (pp. 1354-1372). Baltimore: Williams & Wilkins.
- Harvey, E.A. (2001). Peritoneal access in children. *Peritoneal Dialysis International*, 21, S218-S222.
- Hergenroeder, A.C., & Brewer, E.D. (2001). A survey of pediatric nephrologists on adolescent sexual health. *Pediatric Nephrology*, 16, 57-60.
- Hisano, S., Miyazaki, C., Hatae, K., Kaku, Y., Yamane, I., Ueda, K., & Okamura, S. (1991). Immune status of children on continuous ambulatory peritoneal dialysis. *Pediatric Nephrology*, 6, 179-181.
- Hislop, S., & Lansing, L. (1983). A comparison of pediatric home peritoneal dialysis modalities: The family point of view. *AANNT Journal*, 11, 22-23, 53.
- Hockenberry, M.J., & Wong, D.L. (2004). *Wong's clinical manual of pediatric nursing*. St. Louis: Mosby.
- Jain, S.R., Smith, L., Brewer, E.D., & Goldstein, S.L. (2001). Non-invasive intravascular monitoring in the pediatric hemodialysis population. *Pediatric Nephrology*, 16, 15-18.
- Johnson, K.B. (Ed.). (1993). *The Harriet Lane handbook: A manual for pediatric house officers* (13th ed.). St. Louis: Mosby-Year Book, Inc.
- Keen, M.L., & Gotch, F.A. (1991). Dialyzers and delivery systems. In M.G. Cogan & P. Schoenfeld (Eds.), *Introduction to dialysis* (2nd ed.) (pp. 1-44). New York: Churchill Livingstone.
- Kjellstrand, C.M., Shideman, J.R., Santiago, E.A., Mauer, S.M., Simmons, R.L., & Buselmeier, T.J. (1971). Technical advances in hemodialysis of very small pediatric patients. *Proceedings of the Dialysis and Transplant Forum*, 124-132.
- Koch, V.H., Lippe, B.M., Nelson, P.A., Boechat, M.I., Sherman, M.M., & Fine, R. (1989). Accelerated growth after recombinant human growth hormone treatment of children with chronic renal failure. *Journal of Pediatrics*, 115, 365-371.
- Laube, G.F., Berger, C., Goetschel, P., Leumann, E., Neuhaus, T.J. (2002). Immunizations in children with chronic renal failure. *Pediatric*

- Nephrology*, 17, 638-642.
- Leonard, M.B., Donaldson, L.A., Ho, M., & Geary, D.F. (2003). A prospective cohort study of incident maintenance dialysis in children: An NAPRTC study. *Kidney International*, 63, 744-755.
- Limwongse, C., & Cassidy, S.B. (2004). Syndromes and malformations of the urinary tract. In E.D. Avner, W.E. Harmon, & P. Niaudet (Eds.), *Pediatric nephrology* (5th ed.) (pp. 93-121). Philadelphia: Lippincott Williams and Wilkins.
- Macias, W.L., Mueller, B.A., Scarim, S.K., Robinson, M., & Rudy, D.W. (1991). Continuous venovenous hemofiltration: An alternative to continuous arteriovenous hemofiltration and hemodiafiltration in acute renal failure. *American Journal of Kidney Diseases*, 18, 451-458.
- McBride, P. (1984) The development of hemo- and peritoneal dialysis. In A.R. Nissenson, R.N. Fine, & D.E. Gentile (Eds.), *Clinical dialysis* (pp. 1-26). Norwalk, CT: Appleton-Century-Crofts.
- McCann, L. (Ed.). (2002). *Pocket guide to nutrition assessment of the patient with chronic kidney disease* (3rd ed.). [Brochure]. New York: Council on Renal Nutrition of the National Kidney Foundation.
- McCarthy, J.T., Williams, A.W., & Johnson, W.J. (1994). Serum beta-2 microglobulin concentration in dialysis patients: Importance of intrinsic renal function. *Journal of Laboratory and Clinical Medicine*, 123, 495-505.
- Mehls, O., Blum, W.F., Schaefer, F., Tonshoff, B., & Scharer, K. (1992). Growth failure in renal disease. *Bailliere's Clinical Endocrinology and Metabolism*, 6, 665-685.
- Miller, D., Macdonald, D., Kolnacki, K., & Simek, T. (2004). Challenges for nephrology nurses in the management of children with chronic kidney disease. *Nephrology Nursing Journal*, 31, 287-294.
- Mogyorosi, A.M. & Ziyadeh, F.N. (2001). Diabetic nephropathy. In S.G. Massry & R.J. Glassock (Eds.), *Massry & Glassock's textbook of nephrology* (4th ed.) (pp. 874-895). Philadelphia: Lippincott Williams & Wilkins.
- Morel, P., Almond, P.S., Matas, A.J., Gillingham, K.J., Chau, C., Brown, A., et al. (1991). Long-term quality of life after kidney transplantation in childhood. *Transplantation*, 52, 47-53.
- Morgan, H.E.G., Gautam, M., & Geary, D.F. (2001). Maintenance intravenous iron therapy in pediatric hemodialysis patients. *Pediatric Nephrology*, 16, 779-783.
- Moritz, M.L., Vats, A., & Ellis, D. (2003). Systemic anticoagulation and bleeding in children with hemodialysis catheters. *Pediatric Nephrology*, 18, 68-70.
- Nightingale, F. (1859). *Notes on nursing: What it is and what it is not*. London: Harrison Publishing.
- North American Pediatric Renal Transplant Cooperative Study (NAPRTCS). (2004). *2004 annual report*. Boston: Author.
- O'Grady, N.P., Alexander, M., Dellinger, E.P., Gerberding, J.L., Heard, S.O., Maki, D.G., et al. (2002). Guidelines for the prevention of intravascular catheter-related infections. *Morbidity and Mortality Weekly Report*, 51, 1-26.
- Ouwendyk, M., Leitch, R., & Freitas, T. (2001). Daily hemodialysis: A nursing perspective. *Advances in Renal Replacement Therapy*, 8, 257-267.
- Parekh, R.S., Carroll, C.E., Wolfe, R.A., & Port, F.K. (2002). Cardiovascular mortality in children and young adults with end-stage kidney disease. *The Journal of Pediatrics*, 141, 191-197.
- Polinsky, M.S., Kaiser, B.A., & Stover, J.B. (1987). Neurologic development of children with severe chronic renal failure from infancy. *Pediatric Nephrology*, 1, 157-165.
- Rigden, S. (1994). Planning therapy. In M.A. Holliday, T.M. Barratt, & E.D. Avner (Eds.), *Pediatric nephrology* (3rd ed.) (pp. 1419-1422). Baltimore: Williams & Wilkins.
- Rosenkranz, J., Bonzel, K.E., & Bulla, M. (1992). Psychosocial adaptation of children and adolescents with chronic renal failure. *Pediatric Nephrology*, 6, 459-463.
- Sadler, T. W. (2000). *Langman's medical embryology* (8th ed.). Philadelphia: Lippincott Williams & Wilkins.
- Sharma, A.K. (2001). Reassessing hemodialysis adequacy in children: The case for more. *Pediatric Nephrology*, 16, 383-390.
- Shemesh, D., Olsha, O., Mabjeesh, N.J., & Abramowitz, H.B. (2001). Dialysis access induced limb ischemia corrected using quantitative duplex ultrasound. *Pediatric Nephrology*, 16, 409-411.
- Shroff, R., Wright, E., Ledermann, S., Hutchinson, C., & Rees, L. (2003). Chronic hemodialysis in infants and children under 2 years of age. *Pediatric Nephrology*, 18, 378-383.
- Solhaug, M.J., Adelman, R.D., & Chan, J.C.M. (1992). Hypertension in the child with chronic renal insufficiency or undergoing dialysis. *Child Nephrology and Urology*, 12, 133-138.
- Soliday, E., Kool, E., & Lande, M.B. (2001). Family environment, child behavior, and medical indicators in children with kidney disease. *Child Psychiatry and Human Development*, 31, 279-295.
- Smith, J.M. & McDonald, R.A. (2000). Progress in renal transplantation for children. *Advances in Renal Replacement Therapy*, 7, 158-171.
- Stapleton, S., & Wright, J. (1992). Continuous arteriovenous hemofiltration: An alternative dialysis therapy in neonates. *Neonatal Network*, 11, 17-25.
- Stephens, F.D., Smith, E.D., & Hutson, J.M. (2002). *Congenital anomalies of the kidney, urinary and genital tracts* (2nd ed.). London: Martin Dunitz.
- Suddaby, E.C., Bell, S.B., & Murphy, K.J. (1990). Continuous hemofiltration in infants and children. *Pediatric Nursing*, 16, 79-82.
- Thompson, G.N., Butt, W.W., Shann, F.A., Kirby, D.M., Henning, R.D., Howells, D.W., & Osborne, A. (1991). Continuous venovenous hemofiltration in the management of acute decompensation in inborn errors of metabolism. *The Journal of Pediatrics*, 118, 879-884.
- The Renal Network. (2004). *Other renal links*. Retrieved from <http://therenalnetwork.org/RenalLinks/OtherRenalLinks.html>.
- United States Renal Data System (USRDS). (2003). *United States Renal Data System 2003 annual data report* (NIH Publication No. 93-3176). Bethesda, MD: U.S. Government Printing Office.
- Valeri, A., Radhakrishnan, J., Vernocchi, L., Carmichael, L.D., & Stern, L. (1993). The epidemiology of peritonitis in acute peritoneal dialysis: A comparison between open- and closed-drainage systems. *American Journal of Kidney Diseases*, 21, 300-309.
- Verina, E., Honda, M., Warady, B.A., & Piraino, B. (2000). Prevention of peritonitis in children on peritoneal dialysis. *Peritoneal Dialysis International*, 20, 625-630.
- Warady, B.A., Morganstern, B.Z., & Alexander, S.R. (2004). Peritoneal dialysis. In E.D. Avner, W.E. Harmon, & P. Niaudet (Eds.), *Pediatric nephrology* (5th ed.) (pp. 1375-1394). Philadelphia: Lippincott Williams and Wilkins.
- Warady, B.A., Schaefer, F., Alexander, S.R., Firaneck, C., & Mujais, S. (2004). *Care of the pediatric patient on peritoneal dialysis – Clinical process for optimal outcomes*. [Brochure]. Deerfield, IL: Baxter Healthcare Corporation.
- Watkins, S.L., & Richards, G.E. (2004). Evaluation of growth and development. In E.D. Avner, W.E. Harmon, & P. Niaudet (Eds.), *Pediatric nephrology* (5th ed.) (pp. 425-448). Philadelphia: Lippincott Williams and Wilkins.
- Watson, A.R. (1995). Strategies to support families of children with end stage renal failure. *Pediatric Nephrology*, 9, 628-631.
- Zobel, G., Stein, J.L., Kuttig, M., Beitzke, A., Metzler, H., & Rigler, B. (1991). Continuous extracorporeal fluid removal in children with low cardiac output after cardiac operations. *The Journal of Thoracic and Cardiovascular Surgery*, 101, 593-597.

Purchase your copy!

Editor's Note: To read the entire text from this chapter, you must purchase your copy of *Contemporary Nephrology Nursing: Principles and Practice*, 2nd Edition. An order form and table of contents may be found on pages 317-318 of the May/June issue of *Nephrology Nursing Journal*.