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Dilys A. Whyte and Richard N. Fine

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# Chronic Kidney Disease in Children

Dilys A. Whyte, MD,\*

Richard N. Fine, MD\*

Author Disclosure  
Drs Whyte and Fine have disclosed no financial relationships relevant to this article. This commentary does contain a discussion of an unapproved/investigative use of a commercial product/device.

**Objectives** After completing this article, readers should be able to:

1. Define chronic kidney disease (CKD).
2. Immunize a patient scheduled for renal transplantation.
3. Assess and manage growth and development in a patient who has CKD.
4. Discuss the risks and benefits of renal transplantation, including the advantages and disadvantages of living donor versus deceased donor transplantation.

## Introduction

Prior to 2002, the term chronic renal insufficiency was used to characterize patients who had progressive decline in renal function, defined as a glomerular filtration rate (GFR) of less than 75 mL/min per 1.73 m<sup>2</sup> body surface area. Chronic kidney disease (CKD) is the new term defined by the National Kidney Foundation Kidney Disease and Outcome Quality Initiative (KDOQI) Group to classify any patient who has kidney damage lasting for at least 3 months with or without a decreased GFR or any patient who has a GFR of less than 60 mL/min per 1.73 m<sup>2</sup> lasting for 3 months with or without kidney damage. The KDOQI Group also classified CKD into five stages:

- Stage 1: Kidney damage with a normal or increased GFR (>90 mL/min per 1.73 m<sup>2</sup>)
- Stage 2: Mild reduction in the GFR (60 to 89 mL/min per 1.73 m<sup>2</sup>)
- Stage 3: Moderate reduction in the GFR (30 to 59 mL/min per 1.73 m<sup>2</sup>)
- Stage 4: Severe reduction in the GFR (15 to 29 mL/min per 1.73 m<sup>2</sup>)
- Stage 5: Kidney failure (GFR <15 mL/min per 1.73 m<sup>2</sup> or dialysis)

GFR values for CKD staging are for children older than 2 years of age because the GFR values for younger children are low due to ongoing renal maturation. Children who have CKD may present to clinicians with a combination of problems involving growth, nutrition, electrolyte disturbances, renal osteodystrophy, anemia, immunizations, hypertension, and renal transplantation.

## Causes

CKD has a prevalence of 1.5 to 3.0 per 1,000,000 among children younger than the age of 16 years. The most common causes of CKD in children are urologic abnormalities (~30% to 33%) and glomerulopathies (~25% to 27%). These two abnormalities account for more than 50% of the reported causes of end-stage renal disease in children. The other major causes are hereditary nephropathies (~16%) and renal hypoplasia and dysplasia (~11%). Data from the North American Pediatric Renal Trial and Collaborative Studies 2006 demonstrate very similar information (Table 1). Histologic similarities among the various causes of CKD are many, and possible common mechanisms accounting for similarities include cell-specific damage, the role of growth factors, and the effects of metabolic factors. Ultimately, these mechanisms could lead to some degree of healing or to additional sclerosis (scarring).

## Growth Problems

Growth retardation is one of the major complications of a child who has CKD. The degree of growth failure has been correlated with age of onset of CKD. The cause of growth failure is believed to be multifactorial, including growth hormone (GH) and insulin-like growth factor-I (IGF-I) function, nutritional status, acid-base balance, and bone mineralization.

\*Stony Brook University, Stony Brook, NY.

**Table 1. Common Causes of Chronic Kidney Disease in Children**

Diagnosis	Incidence
Obstructive uropathy	22%
Aplasia/hypoplasia/dysplasia	18%
Glomerulonephritis	~10%
Focal glomerulosclerosis	~9%
Reflux nephropathy	8%
Data from the North American Pediatric Renal Trials and Collaborative Studies 2006.	

The age of CKD onset is correlated with the degree of growth retardation because children who have normal renal function achieve one third of their final adult height during the first 2 years after birth. Data support current treatments for achieving accelerated growth in infants and young children who have CKD, but most children still do not reach their genetic height potential despite optimal management.

GH is released from the anterior pituitary and is regulated by GH-releasing factor and somatostatin. The negative feedback mechanisms involve circulating GH and IGF-I concentrations. Most of IGF-I is bound to IGF-binding proteins, but it has been demonstrated that free IGF-I mediates many GH functions, including the stimulus for linear height of bone as well as some renal hemodynamic effects. GH has direct effects, not only on bone, but on other body tissues. When renal function is reduced, GH is increased because of decreased clearance by the kidneys despite the normal pulsatile release of the hormone, which continues despite the increased GH concentrations.

Resistance to GH and to IGF-I also is believed to lead to growth reduction. GH resistance most likely is due to several causes. In some studies, serum GH concentrations are increased in patients who have CKD, but the concentrations of GH receptors are reduced. Another cause may be the upregulation of intracellular inhibitors, labeled suppressors of cytokine signaling (SOCS). The SOCS proteins can alter the phosphorylation of GH receptors and may cause GH resistance.

Similarly, IGF-1 resistance probably is due to several causes. Total immunoreactive IGF concentrations in those who have CKD are normal, but the bioactive IGF is reduced by approximately 50%. IGF-binding proteins, of which six are now identified, increase with renal failure and most likely inhibit the actions of IGF-1 by binding with it, thereby preventing IGF-1 from binding to its

receptor. In addition, some postreceptor IGF-1 signaling defects in renal failure may add to IGF-1 resistance.

Other hormones that play a role in pubertal growth and development have been found to be reduced in children who have CKD, including luteinizing hormone, plasma testosterone, and free testosterone. These hormones play crucial roles in the pubertal development and pubertal growth spurt of children.

Treatment of growth failure initially involves resolving nutritional deficiencies and improving the acid-base balance of children who have CKD. Once these tasks are accomplished, affected children begin GH therapy if growth retardation persists. The use of GH has been demonstrated to produce some catch-up growth, and many patients who have CKD attain a final height considered normal for their age range. Most patients who have CKD grow when given the recommended starting dose of 0.05 mg/kg per day, administered subcutaneously daily. Whether patients in the pubertal age group require additional GH needs additional investigation.

## Nutrition

Children who have CKD have nutrition and protein deficiencies for several reasons, including anorexia, nausea and vomiting from the uremia, and an abnormal sense of taste. Young children, in particular, need sufficient caloric intake to grow. Protein intake should be optimized to allow for maintenance of nitrogen balance and preservation of lean body mass. Some patients may require supplemental nasogastric or gastrostomy tube feeding if they cannot maintain optimal height and weight gain by oral feeding. However, if protein intake is excessive, hyperfiltration may occur, leading to increased damage to the renal parenchyma. Micropuncture studies demonstrate an increase in the GFR (hyperfiltration) after an amino acid load due to reduction in afferent arteriolar resistance.

Prostaglandins, which can alter vascular tone and increase the GFR, recently have been implicated in the development of hyperfiltration because prostaglandin values have been noted to increase in response to an increased amino acid load. Protein restriction was believed to slow the progression of renal disease, but this effect has not been verified in children. Reducing protein intake to 0.8 to 1.1 g/kg per day has not been shown to affect linear growth negatively.

Because many vitamins are lost during dialysis, pediatric patients undergoing this therapy should supplement their diets with vitamins, especially folic acid, trace minerals, and B complexes.

Specialized formulas that have high energy contents

and lower electrolyte contents have been developed for infants and children who have CKD. These are reasonable formulas for an older child already on dialysis who may not be meeting his or her nutritional goal or who is experiencing poor weight gain or growth.

### Electrolyte Disorders

Metabolic acidosis develops in patients who have CKD because of abnormally decreased bicarbonate reabsorption of filtered bicarbonate, reduction of renal ammonia synthesis, decreased acidified tubular fluid, and decreased titratable acid excretion. Decline in GFR below 50% of normal is accompanied by a decline in bicarbonate reabsorption. Reduced bicarbonate reabsorption leads to systemic acidosis, which causes protein degradation and efflux of calcium from bone. Such factors play a role in the poor linear growth observed in children who have CKD.

Therapy should target maintaining a serum bicarbonate concentration of 20 to 22 mEq/L (20 to 22 mmol/L). Bicarbonate replacement consists of administering sodium bicarbonate supplements or phosphate binders. Most available binders have a base component such as calcium carbonate.

Hyperkalemia is a complication of CKD. In the healthy kidney, potassium reabsorption occurs in the proximal tubules and the loop of Henle, and secretion of up to 90% of the daily intake of potassium occurs in the distal tubules. As renal disease progresses, the distal tubules of the remaining nephrons continue to secrete potassium. Increased aldosterone also enhances potassium secretion by stimulating sodium-potassium exchange in the kidneys and the colon. However, hyperkalemia develops from an increase in dietary potassium that overwhelms the compensatory mechanisms or by use of medications that alter potassium secretion (spironolactone, amiloride, or angiotensin converting enzyme inhibitors). Table 2 lists approaches to treating hyperkalemia.

Hypokalemia also can occur in children who have CKD but tends to develop in patients who have tubular defects such as seen with Fanconi syndrome.

### Renal Osteodystrophy

Calcium, phosphorus, and magnesium balance are maintained by the kidney when people have normal renal function. In CKD, hypocalcemia and hyperphosphatemia occur. The normal kidney converts 25-dihydroxyvitamin D<sub>3</sub> into 1,25-dihydroxyvitamin D<sub>3</sub> when stimulated by hypocalcemia, parathyroid hormone (PTH) release, and decreased dietary intake of phosphate. PTH is degraded and removed by the kidney.

In renal disease, 1,25-dihydroxyvitamin D<sub>3</sub> production decreases, intestinal absorption of calcium decreases, and hypocalcemia develops. This series of events, in turn, causes an increase in PTH formation. However, PTH has little effect due to low vitamin D and high serum phosphate concentrations (phosphorus cannot be secreted by the diseased kidneys) as well as downregulation of PTH receptors. Abnormal bone mineralization with resultant fractures and osteitis fibrosa can occur (Figure).

Linear growth also can be affected by secondary hyperparathyroidism, with renal osteodystrophy possibly leading to alterations in the normal growth plate cartilage architecture due to abnormal bone mineralization and fibrosis of the endochondral bones.

For children who have CKD, bone pathology must be treated aggressively. Available forms of vitamin D supplementation are dihydrotachysterol (DHT), 25-hydroxyvitamin D<sub>3</sub> (calcifediol), 1-alpha-hydroxyvitamin D<sub>3</sub>, and 1,25-dihydroxyvitamin D<sub>3</sub> (calcitriol) (Table 3). Paricalcitol, a newer intravenous form of vitamin D, is administered to children who have CKD and are receiving hemodialysis. When choosing a vitamin D preparation, the age of the patient, the ability to swallow pills, and whether the patient is on hemodialysis or peritoneal

Table 2. Treatment of Hyperkalemia

Product	Dose	Potential Adverse Effects
Sodium bicarbonate	$[(0.6 \times \text{body weight}) \times (\text{bicarbonate desired} - \text{bicarbonate observed})] \div 2$ 0.5 to 1 mEq/kg IV over 1 hour	May cause hypocalcemia
Calcium gluconate (10%)	0.5 to 1 mL/kg IV over 5 to 15 min	Arrhythmia
Glucose and insulin	Glucose 0.5 g/kg with insulin 0.1 units/kg IV over 30 min	Hypoglycemia
Sodium polystyrene sulfonate	1 g/kg per dose PR or PO	May cause constipation/diarrhea
Beta agonists	5 to 10 mg aerosolized	Tachycardia, hypertension

IV=intravenously, PO=orally, PR=rectally



Figure. Deformity associated with renal osteodystrophy.

dialysis are important issues to consider. DHT can be formulated into a solution that is easy for infants to take orally, as can calcitriol, which also can be given to infants. Intravenous forms of vitamin D such as paricalcitol and doxercalciferol (doxercalciferol also has an oral formulation) usually are given to patients undergoing hemodialysis to ensure compliance and to reduce the number of necessary oral medications.

Vitamin D treatment typically is started once a child develops stage 3 CKD. Dosing is titrated according to the serum phosphorus concentration returning to normal values for age and the PTH values returning to the upper limits of normal for age.

Hyperphosphatemia is treated with administration of a phosphate binder with meals to facilitate bonding of phosphorus within the gastrointestinal tract, thereby increasing phosphate elimination. Commonly used binders are calcium-containing agents such as calcium carbonate and calcium acetate and other noncalcium-containing binders. Aluminum-containing phosphate binders should be avoided in children because of the risk of aluminum toxicity, which can occur in renal failure. Children who have CKD also should be following low-phosphate diets.

### Anemia

Anemia in CKD is caused by either an insufficient production of erythropoietin by the diseased kidneys or by iron deficiency. Anemia is defined as a reduction in red blood cell volume or hemoglobin concentration below the normal range for a healthy person. Morbidity, mortality, and quality of life data from the KDOQI guidelines suggest that maintaining the hematocrit in the range of 33% to 36% (0.33 to 0.36) and the hemoglobin at 11.0 to 12.0 g/dL (110.0 to 120.0 g/L) is important for children who have CKD.

Prior to the development of recombinant human erythropoietin, patients who had CKD had to undergo transfusions to increase their hematocrit values. Transfusions not only exposed patients to various infectious agents but exposed and sensitized them to human lymphocyte antigens, putting them at increased risk for rejection should they undergo renal transplantation. With improvement of anemia, children demonstrate improvement in cognitive development, cardiac function, and exercise tolerance, as well as decreased mortality.

As stated, anemia is caused by either an insufficient production of erythropoietin by the diseased kidneys or by iron deficiency. Due to decreased appetites, children who have CKD cannot increase their iron stores adequately through an oral diet. Oral iron therapy should be

Table 3. Vitamin D Analogs

Analog	Starting Dose
1,25-dihydroxyvitamin D <sub>3</sub> (calcitriol)	0.01 to 0.05 mcg/kg per day orally (<3 y of age) 0.25 to 0.75 mcg/day (>3 y of age) Can be titrated to maintain normal PTH concentrations
1,25-dihydroxyvitamin D <sub>3</sub> (alfacalcidol)	0.25 to 0.5 mcg/day orally Can be titrated to maintain normal PTH concentrations
Vitamin D <sub>2</sub> (dihydrotachysterol)	
Vitamin D <sub>2</sub> (doxercalciferol)	Oral and IV dosing available for adolescents and adults
Synthetic vitamin D analog (paricalcitol)	0.04 to 0.1 mcg/kg IV 3 times per week (>5 y of age)
IV=intravenous, PTH=parathyroid hormone	

administered at a dose of 2 to 3 mg/kg per day of elemental iron in two or three divided doses. Iron should be consumed on an empty stomach and not concomitantly with phosphate binders because iron binds to the phosphate binders.

Parenteral iron can be provided to those who continue to lose blood or who cannot tolerate oral iron. Parenteral iron can be administered easily to patients receiving hemodialysis because they already have vascular access. Intravenous iron also can be used for the peritoneal dialysis patient who is resistant to oral iron or is noncompliant in taking oral iron.

Erythropoietin can be administered subcutaneously to children who have CKD, including those undergoing peritoneal dialysis, or intravenously for those receiving hemodialysis. Erythropoietin can be given one, two, or three times per week. The initial dose ranges between 30 and 300 units/kg per week, with the usual maintenance dosage between 60 and 600 units/kg per week. The maintenance dose is determined and adjusted based on monthly hemoglobin values. A new form of erythropoietin, darbepoetin alfa, which has a longer half-life and requires dosing once every 2 weeks to once monthly, is being investigated for use in children.

## Hypertension

Hypertension is diagnosed in children who have CKD by finding an elevated blood pressure reading on three or more separate office visits at least 1 week apart. The diagnosis is based on the child's age, sex, and height percentile. Grades of hypertension are as follows, based on tables or graphs of normal values: (1)

1. Prehypertension: Average systolic or diastolic pressures are at the 90th percentile or greater but at or less than the 95th percentile for age, sex, and height
2. Stage I hypertension: Average systolic or diastolic pressure is at or greater than the 95th percentile for age, sex, and height
3. Stage II hypertension: Average systolic or diastolic pressure is more than 5 mm Hg higher than the 95th percentile
4. Hypertensive urgency and emergency: Average systolic or diastolic pressure is more than 5 mm Hg higher than the 95th percentile and clinical symptoms of headache, vomiting, seizures, or encephalopathy are present

In addition to determining the underlying cause of the hypertension, clinicians should monitor patients at least annually with echocardiography to assess function and left ventricular status. Medications are adjusted to improve cardiac function. Those children who fall into the hypertensive urgency and emergency category require intravenous

medications or a rapid-acting oral medication (nifedipine or minoxidil) to reduce blood pressure. (See Feld and Corey (1)) for information on antihypertensive medication choices.)

## Immunizations

Children who have CKD should be kept as medically stable as possible to prepare them for renal transplantation. Immunizations are important to reduce the risk of morbidity and mortality from vaccine-preventable infections. Although complete vaccination should be accomplished prior to renal transplantation, various transplant centers report that 20% to 30% of children do not complete their series of immunizations prior to transplantation. Because live vaccines are not recommended after transplantation, children who have received renal transplants and have not completed their live vaccine series prior to transplantation are at increased risk for developing varicella, measles, mumps, and rubella.

Unfortunately, vaccine immunogenicity in children who have CKD may be altered due to underlying conditions that may impair immune function. Dialysis (peritoneal and hemodialysis), nephrosis, and uremia affect the immune system adversely. Hepatitis B antibodies can be removed by dialysis, so children undergoing dialysis require frequent measurement of hepatitis B antibody titers.

The waiting time for a renal transplant can affect the vaccine schedule adversely. Prior to being placed on the deceased donor list, children may need to undergo an accelerated immunization schedule to provide some immunity before transplantation. For example, a child may receive the initial measles-mumps-rubella-varicella vaccine at 9 months of age prior to a planned transplantation scheduled in 2 to 3 months.

All children who have CKD should receive all routine immunizations, including those against diphtheria, tetanus, polio, *Haemophilus influenzae* type b, and measles-mumps-rubella. They also should receive influenza vaccine annually, and if the 7-antigen pneumococcal vaccine has not been administered prior to 2 years of age, it should be given to children 2 years of age or older who have CKD. Responsiveness to immunizations varies from child to child. Occasionally, booster vaccines are necessary, and measuring antibody titers should be routine. The meningococcal vaccine is recommended for adolescents who have CKD.

As stated, immune response to hepatitis B vaccine requires frequent evaluation, and children should receive a booster series if antibody titers become negative. Hepatitis B immunization is a challenge in children who have CKD because the individual may not follow the vaccination schedule, may have antibodies removed during di-

alysis, or may be taking medications that interfere with antibody response.

Contraindications to vaccinations are few. The immunization schedule can be delayed if the patient is acutely and severely ill, but vaccination should proceed once the patient's condition has improved. Live vaccines should not be given if the patient recently was given intravenous immune globulin or other antibody-containing products to help improve immune impairment.

Successful completion of pretransplantation immunization series in children who have CKD requires frequent communication among the pediatric nephrologist, pediatric transplantation team, and the primary general pediatrician.

## Renal Transplantation

Once a child progresses to end-stage renal disease, the ultimate treatment is renal transplantation. The donor kidney can come from a living related, living unrelated, or deceased donor. Children are given priority for a deceased donor kidney when placed on the United Network for Organ Sharing (UNOS) waiting list. UNOS, originally established in 1968 as a professional organization, formed a Kidney Center in 1977 that developed computer-based matching of organs to facilitate transplants. Today, the UNOS organ center is open 365 days per year, 24 hours per day to facilitate organ procurement and organ sharing across the United States.

Based on information from the North American Pediatric Renal Trials and Collaborative Studies, the 1-year survival rate among living donor recipients is 92% and the 5-year survival rate is 85%. The 1-year deceased donor recipient's survival rate is 84%, with the 5-year survival rate being 77%.

The current indication for transplantation in the pediatric population is end-stage renal disease, but many children receive preemptive kidney transplants without ever undergoing dialysis due to parental preference and on the basis of data demonstrating that preemptive renal transplant recipients do better than those who receive dialysis prior to transplantation. Most centers rarely perform transplants in infants younger than 6 months of age or those weighing less than 6 kg because of a suspected higher risk for graft failure due to infections, technical problems, and immunosuppressive pharmacokinetics. Most centers prefer the recipient to be older than 1 year of age and weigh at least 10 kg.

Contraindications to renal transplantation are few. One relative contraindication is human immunodeficiency virus (HIV) nephropathy because transplantation protocol requires further immunosuppression in an al-

ready immunodeficient individual. Currently, transplantation for children who have HIV nephropathy is being investigated. Other relative contraindications are preexisting malignancy, devastating neurologic diseases, and potential recurrence of the primary disorders, such as oxalosis. Despite these relative contraindications, transplantation may be offered, depending on the wishes of the family and the medical stability of the patient.

Posttransplantation medications aim to prevent graft rejection and include, but are not limited to, the calcineurin inhibitors cyclosporine and tacrolimus, mycophenolate mofetil, or steroids. New posttransplantation protocols seek to reduce the adverse effects of steroid therapy.

Immediately after transplantation, the major concerns for the recipient include the complications of rejection and infection. There is a potential for graft loss if these complications occur. Patients are followed closely by the transplant team in conjunction with their primary pediatric nephrologist. The immediate posttransplantation period is an important time for the general pediatrician to follow the patient and to communicate with the subspecialists. Should the transplant patient develop a fever (temperature  $\geq 100.4^{\circ}\text{F}$  [ $38.0^{\circ}\text{C}$ ]), the pediatrician should examine the patient; perform laboratory tests such as blood and urine cultures, complete blood count, and blood chemistries; and administer a broad-spectrum antibiotic intravenously while contacting the transplant/nephrology team.

Other long-term complications include noncompliance with taking the medications, which may lead to chronic rejection or graft loss from chronic rejection. Growth is another long-term complication about which the pediatrician should be concerned. If the recipient is not growing well, he or she should be re-evaluated for the use of GH.

## Summary

CKD affects multiple systems, including the endocrine (calcium-phosphorus metabolism, growth), hematologic, immune, and cardiovascular systems. Children who have CKD require close, coordinated medical attention by the primary care pediatrician, the pediatric nephrologist, and other pediatric subspecialists to ensure that they grow successfully into adulthood to the best of their potential.

**NOTE.** An article on acute renal failure was published in the September 2008 issue of *Pediatrics in Review*.

## Reference

1. Feld LG, Corey H. Hypertension in childhood. *Pediatr Rev.* 2007;28:283–298

## Suggested Reading

Fine RN, Whyte DA, Boydston I. Conservative management of chronic renal insufficiency. In: Avner ED, Harmon WE, Niaudet P, eds. *Pediatric Nephrology*. 5th ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2004:1291–1312

Mahan JD, Warady BA. Assessment and treatment of short stature in pediatric patients with chronic kidney disease: a consensus statement. *Pediatr Nephrol.* 2006;21:917–930

Verma A, Wade JJ. Immunization issues before and after solid organ transplantation in children. *Pediatr Transplantation.* 2006;10:536–548

Wong H, Mylrea K, Feber J, Drukker A, Filler G. Prevalence of complications in children with chronic kidney disease according to KDOQI. *Kidney Int.* 2006;70:585–590

## PIR Quiz

Quiz also available online at [www.pedsinreview.aappublications.org](http://www.pedsinreview.aappublications.org).

1. You are preparing to speak to a group of residents about end-stage renal disease. You intend to emphasize that the need for renal transplantation is *most* likely to occur among children who have:
  - A. Distal renal tubular acidosis.
  - B. Immunoglobulin A nephropathy.
  - C. Posterior urethral valves or other obstructive uropathy.
  - D. Poststreptococcal acute glomerulonephritis.
  - E. Reflux nephropathy.
2. In young children who have chronic kidney disease (CKD), growth failure *most* likely reflects:
  - A. Elevated concentrations of plasma luteinizing hormone.
  - B. Low concentrations of growth hormone.
  - C. Low concentrations of immunoreactive insulin-like growth factor-I.
  - D. Low concentrations of plasma testosterone.
  - E. Resistance to growth hormone.
3. Calcium and phosphorus metabolism are negatively affected by CKD, and resultant bone pathology must be treated aggressively. For optimal bone health, a 4-year-old child who has stage 3 CKD typically requires the oral administration of:
  - A. A calcium supplement and a phosphate binder.
  - B. A phosphate binder alone.
  - C. Vitamin D alone.
  - D. Vitamin D and a calcium supplement.
  - E. Vitamin D and a phosphate binder.
4. A 4-year-old boy who has CKD but is not yet receiving dialysis has a hemoglobin value of 8.1 g/dL (81.0 g/L), despite appropriate oral iron supplementation. At this point, the *most* appropriate intervention is:
  - A. Close observation.
  - B. Discontinuation of phosphate binder therapy.
  - C. Parenteral iron administration.
  - D. Periodic transfusions of packed red cells.
  - E. Weekly subcutaneous erythropoietin.
5. Currently, among children who have severe CKD, the *best* candidate for a preemptive renal transplant is a:
  - A. 3-month-old boy who has congenital nephrotic syndrome.
  - B. 12-month-old girl who has dysplastic kidneys and Werdnig-Hoffman disease.
  - C. 18-month-old boy who has posterior urethral valves.
  - D. 10-year-old boy who has oxalosis.
  - E. 10-year old girl who has human immunodeficiency virus nephropathy.

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