Acute Renal Failure in Children

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

1. Define acute renal failure (ARF).
2. Differentiate the three forms of ARF.
3. Initiate treatment, including stabilization, of a patient who has ARF.
4. Discuss the various medications necessary for treating a patient who has ARF.

Introduction

Acute renal failure (ARF) is defined as an acute decline in renal function characterized by an increase in blood urea nitrogen (BUN) and serum creatinine values, often accompanied by hyperkalemia, metabolic acidosis, and hypertension. Significant morbidity and mortality can accompany ARF. Patients who have ARF recover their renal function either partially or completely or they develop end-stage renal disease. They also may develop associated multiorgan disease.

ARF is divided into three forms: prerenal failure (most common), intrinsic renal failure, and postrenal failure. Treatment ranges from conservative medical management to dialysis or renal transplantation, depending on the severity of kidney disease and degree of renal function recovery. Worldwide, most cases of ARF in children are due to hemolytic-uremic syndrome or volume depletion.

Prerenal Failure

Causes

Prerenal failure refers to hypoperfusion of the kidneys. There are a variety of causes for such hypoperfusion, the most common of which is hypovolemia due to gastrointestinal (GI) diseases, congenital heart disease, and sepsis (Table 1).

Pathophysiology

Prerenal ARF may result from a sudden decline in renal perfusion due to a sudden decline in intravascular volume. Decreased perfusion can lead to ischemic or toxic injury to the renal cells, with a subsequent decrease in the glomerular filtration rate (GFR). To compensate, the body tries to re-establish renal perfusion and restore intravascular volume in several ways. Afferent arterioles attempt to maintain renal blood flow by relaxing vascular tone, thereby decreasing renal vascular resistance. Decreased renal perfusion also stimulates increased catecholamine and vasopressin secretion and activation of the renin-angiotensin system that, in turn, causes vasoconstriction.

Finally, with renal hypoperfusion, vasodilatory prostaglandins, such as prostacyclin, are generated, which help maintain renal perfusion by mediating vasodilation of the microvasculature. Therefore, administration of aspirin or other nonsteroidal anti-inflammatory drugs (NSAIDs) to comfort the patient during a period of renal hypoperfusion actually may worsen ARF because these medications can inhibit the compensatory mechanisms of prostaglandins.

When renal perfusion pressure is low, as in renal artery stenosis, intraglomerular pressure is improved by the release of angiotensin II, increasing efferent arteriolar resistance. Nitric oxide, a potent vasodilator, and kallikrein-kinin system activation also play roles in compensatory regulatory mechanisms in ARF. These roles still are being studied and need to be clarified because their timing can affect ARF status.

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Table 1. Causes of Prerenal Acute Renal Failure

Extracellular Fluid Volume Deficits

- Gastrointestinal losses
  - Vomiting
  - Diarrhea
  - Decreased oral intake of fluids
  - Loss of fluids via intestinal stoma
  - Nasogastric loss of fluids
- Increased urinary loss of fluids
  - Osmotic diuresis (mannitol, glucosuria)
  - Diabetes insipidus (central, nephrogenic)
  - Loss of urinary concentrating ability
  - Renal insufficiency
  - "Medullary washout"
  - Diuretic use
  - Adrenal insufficiency
- Blood losses
  - Hemorrhage
- Redistribution of extracellular fluid
  - Hypoalbuminemia
  - Nephrotic syndrome
  - Liver disease
- Vasodilation
  - Sepsis
  - Anaphylaxis
- Skin losses of fluids
  - Excessive sweating
  - Cystic fibrosis
  - Inflammatory skin disease
  - Burns
  - "Third space" fluid loss
  - Edema (any cause)
  - Intestinal injury
  - Peritonitis
  - Pancreatitis

Cardiac Dysfunction

- Congenital heart disease
- Cardiomyopathy
- Arrhythmia
- Acquired valvular disease
- Tamponade

Prerenal failure also occurs as a result of volume contraction or decreased effective blood volume. Causes of volume contraction include: dehydration from gastroenteritis; urinary losses from salt-losing renal or adrenal diseases, central or nephrogenic diabetes insipidus, or diuretic use; hemorrhage; and disease states that cause "third-spacing" of fluids, such as nephrotic syndrome, sepsis, pancreatitis, and capillary leak syndromes. Decreased effective blood volume occurs with diseases that cause cardiac dysfunction such as congestive heart failure, cardiac tamponade, and arrhythmias.

Infants are at increased risk for excessive volume losses because of the kidney's inability to concentrate urine maximally and to conserve salt and water adequately. Furthermore, because an infant has a large body surface in relation to its body mass, insensible water loss through the skin can add significantly to volume loss, especially during febrile illnesses.

Oliguria or anuria may develop in the patient presenting with prerenal ARF because renin-angiotensin II-aldosterone system activation leads to release of antidiuretic hormone in an attempt to enhance sodium and water reabsorption.

Other intrinsic vasoactive substances that play roles during acute prerenal failure are: adenosine, which is a potent vasoconstrictor within the renal vasculature, but a vasodilator in the peripheral vasculature; nitric oxide, which is a vasodilator; and endothelin, which also is a potent vasoconstrictor. The therapeutic role of these specific agents or other agents that affect their concentrations in the treatment of ARF is being studied.

Diagnosis

In prerenal failure, clinical history should reveal causes of volume depletion, such as dehydration due to vomiting or gastroenteritis, hemorrhage, cardiac failure, or third-space fluid losses. Laboratory findings indicative of prerenal failure include decreased urine output, normal urinary sediments, increased urine osmolality (>400.0 mOsm in the older child and >350.0 mOsm in the neonate), low urinary sodium (<10.0 mEq/L [10.0 mmol/L]), low fractional excretion of sodium ($<1\%$ in the older child and $<2.5\%$ in the newborn), and an increased BUN-to-creatinine ratio. Renal ultrasonography and renal scan findings should be normal.

Intrinsic Renal Disease
Causes

Renal or intrinsic renal failure describes parenchymal injury due to vascular spasm, intravascular coagulation, and microvascular injury. The most common causes of intrinsic renal failure include acute tubular necrosis, interstitial nephritis, hemolytic-uremic syndrome, glomerulonephritis, and nephrotoxic drugs (Table 2).

Pathophysiology

Parenchymal injury to the kidney from either an ischemic or toxic insult can result in cellular dysfunction with cell breakdown and necrosis. The medullary thick ascending limb of the loop of Henle (mTAL) is very vulnerable to

Table 2. Causes of Renal and Postrenal Acute Renal Failure

Renal

- Acute Glomerulonephritis
  - Postinfectious
  - Immune-mediated
    - Systemic lupus erythematosus
    - Membranoproliferative glomerulonephritis
  - Rapidly progressive/crescentic
    - Idiopathic/pauci-immune
    - Antiglomerular basement membrane (Goodpasture syndrome)
    - Systemic lupus erythematosus
- Intrarenal Vascular Disease
  - Hemolytic-uremic syndrome
  - Vasculitis
    - Polyarteritis nodosa
    - Hypersensitivity vasculitis
    - Henoch-Schönlein purpura
    - Renal venous thrombosis
- Acute Interstitial Nephritis
  - Infectious
  - Pyelonephritis
  - Allergic/drug-induced
  - Infiltrative
- Tubular
  - Acute tubular necrosis
    - Hypoxia/anoxia
    - Ischemia
    - Nephrotoxin
  - Intratubular obstruction
    - Pigment nephropathy
    - Hemoglobin, myoglobin
    - Uric acid nephropathy
  - Oxalosis

Postrenal

- Congenital
  - Bladder outlet obstruction-posterior urethral valves
  - Upper tract obstruction bilaterally or in single kidney
  - Ureteropelvic obstruction
- Acquired (rare)
  - Urolithiasis
  - Clots
  - Tumors

Figure. "Backleak" of tubular fluid into the circulation through the injured tubules.

blocking tubule fluid flow. This tubular obstruction, in turn, causes a "backleak" of tubular fluid into the circulation through the injured tubules (Figure).

Diagnosis

Clinical history may reveal dehydration, hypoxic-ischemic events, toxic ingestion, NSAID or other nephrotoxic medication use, signs and symptoms of sepsis, gross hematuria, or trauma. With intrinsic renal failure, the patient's decreased urine output can be described as oliguria (<0.5 mL/kg per hour in a child or <1 mL/kg per hour in an infant) or as anuria (no urine output). Laboratory examination of the urine sediment may demonstrate red blood cell casts, granular casts, and red blood cells, findings seen in glomerulonephritis. When evaluating for possible glomerulonephritis, other appropriate biochemical studies include streptococcal antibodies, hepatitis B and C panels, and complement studies.

A low complement C3 value may indicate an underlying diagnosis of systemic lupus erythematosus or membranoproliferative or poststreptococcal glomerulonephritis. Streptococcal antibodies, including the anti-streptolysin O titer, anti-DNAase B titer, and group A antibody to *Streptococcus pyogenes* titer, should be obtained, especially if hematuria or proteinuria are present and if the patient has a history of impetigo or an upper respiratory tract infection 10 to 14 days prior to presentation. Obtaining hepatitis B_s and C antigen and antibodies also should be considered to determine other possible causes of hematuria or proteinuria. For a patient in whom there is a high suspicion for glomerulonephritis, a biopsy may be warranted if the patient has, in addition to gross hematuria and proteinuria, rapidly rising BUN and creatinine serum values.

Examination of the urine in cases of intrinsic renal failure demonstrates a low urine osmolality (<350.0

hypoxia due to the low oxygen tension in the medullary area and the high rate of oxygen consumption by the mTAL. The straight segment of the proximal tubule also is vulnerable to ischemia due to its high energy need to perform phosphorylation for solute transport. Cellular and brush-border debris from cellular necrosis collect within the hairpin turn in the loop of Henle, thereby
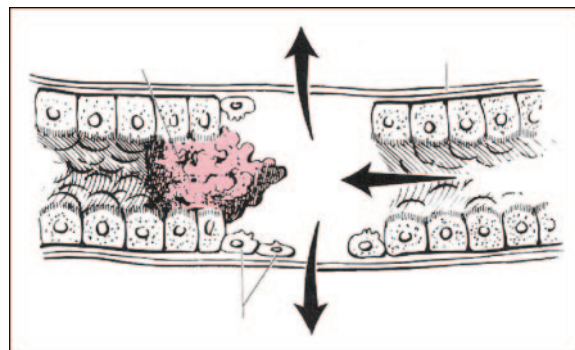
Table 3. **Urine Indices for Acute Renal Failure***

	Prerenal Failure	Renal Failure	Tubular Function
U_{Na^+} (mmol/L)	<20	>40	Sodium retention
U_{Osm} (mOsm/L)	>500	<350 to 450	Urine concentration
$U_{Cr} \div P_{Cr}$	>40	<20	Urine concentration
$FE_{UricAcid} = U_{UricAcid} \div P_{UricAcid} : U_{Cr} \div P_{Cr}$	<12%	>20%	Proximal tubular resorption
$FE_{Na^+} = (U_{Na^+} \div U_{Cr} : P_{Na^+} \div P_{Cr}) \times 100$	<1%	>3%	Sodium retention and urine concentration

*These indices have been validated only in the absence of diuretics. Low FE_{Na} despite prerenal or acute renal failure in acute glomerulonephritis, vasculitis, some cases of acute interstitial nephritis, transition of prerenal to renal failure, "acute on chronic" renal failure, myo- or hemoglobinuric renal failure. Cr=creatinine, FE=fractional excretion, Osm=osmolality, P=plasma, U=urine

mOsm) and a high urinary fractional excretion of sodium (>2% in the older child and >2.5 to 3% in the newborn) (Table 3).

Renal scans can be helpful in diagnosis because they can demonstrate the extent of kidney function. Technetium-99-diethylenetriaminepenta-acetic acid (^{99m}Tc-DTPA) or technetium-99-mercaptotriglyclglycine (^{99m}Tc-MAG-3) scans can delineate areas of normal or low perfusion associated with poor renal function and areas of significant parenchymal damage by demonstrating a delay in the accumulation of the radioisotope and an absence of isotopic excretion into the collecting system.

If necessary, a renal biopsy is the next step in determining the cause of intrinsic renal failure. General indicators for renal biopsy include rapidly increasing serum creatinine concentration, the need to establish a diagnosis of acute versus chronic glomerulonephritis, positive serology for systemic diseases such as membranoproliferative glomerulonephritis or systemic lupus erythematosus, and azotemia with urinary findings of hematuria or proteinuria. A biopsy may demonstrate an active lesion in which immunosuppressive medications, such as steroids, may help to reverse the disease process and recover renal function.

Postrenal Failure

Causes
Postrenal failure results from obstruction to urinary flow. Causes of obstruction include renal calculi, bladder outlet obstruction, and internal or external ureteral compression (Table 2).

Pathophysiology
Obstruction of the ureter, bladder, or urethra can cause an increase in fluid pressure proximal to the obstruction. This increase in pressure, in turn, causes renal damage, resulting in decreased renal function.

Diagnosis
Clinical history may reveal signs or symptoms of an obstruction, such as gross hematuria and colicky pain, as seen in a patient who has renal stones. A history of prenatal ultrasonography demonstrating bilateral hydronephrosis and hydroureters suggests the presence of posterior urethral valves. The physical examination may reveal a palpable flank mass, as seen in a patient who has ureteropelvic obstruction. Urine output and urinary sediment findings may be variable. Patients who have obstructive renal failure frequently show a dilated renal pelvis on renal ultrasonography. In obstruction, a radioisotope scan shows isotope collection within the kidney or at any level of the ureter or bladder, with delayed or absent excretion of the isotope.

Management of ARF
Medical management of ARF includes maintaining renal perfusion and fluid and electrolyte balance, controlling blood pressure, treating anemia, providing adequate nutrition, adjusting medications for the degree of renal impairment, and initiating renal replacement therapy (dialysis) when indicated.

Whether a patient should be cared for on the wards or in an intensive care setting depends on his or her clinical presentation. The patient who clearly has cardiopulmonary collapse, who requires close monitoring of vital signs, or who requires dialysis should be admitted to the intensive care unit.

Vasoactive Agents
Vasoactive agents frequently are administered to improve a patient's blood pressure and ensure adequate perfusion of the kidneys. Maintaining adequate perfusion may require close monitoring of central venous pressure, especially if the patient requires admission to the intensive care unit. Although low-dose dopamine can improve renal blood flow by causing vasodilation, it is debated in

Table 4. **Electrolyte Abnormalities Seen in Acute Renal Failure**

Signs/Symptoms	Management	Comment
Metabolic Acidosis	Sodium bicarbonate if pH <7.2 or HCO_3 <12.0 mEq/L (12.0 mmol/L) [0.6×BW(body weight)×HCO_3 desired−HCO_3 observed]÷2 OR 0.5 to 1.0 mEq/kg IV over 1 h	May cause hypocalcemia
Hyperkalemia	IV sodium bicarbonate 1 mEq/L over 10 to 30 min	May cause hypernatremia, hypocalcemia
	Calcium gluconate (10%) IV 0.5 to 1.0 mL/kg over 5 to 15 min	Arrhythmias, hypercalcemia, bradycardia
	Glucose 0.5 g/kg with insulin 0.1 U/kg IV over 30 min	Hypoglycemia
	Beta agonists 5 to 10 mg via nebulization	Tachycardia, hypertension
	Sodium polystyrene sulfonate 1 g/kg PR or PO	Hypernatremia, constipation
Hyperphosphatemia/Hypocalcemia	Calcium carbonate PO 45 to 65 mg/kg per day If tetany, IV calcium gluconate (10%) 0.5 to 1 mL/kg (up to 10 mL)	

HCO_3=bicarbonate, IV=intravenous, PO=oral, PR=per rectum

the literature whether "renal dosing" of dopamine (0.5 to 3.0 mcg/kg per minute) is beneficial.

Animal studies show that atrial natriuretic peptide (ANP) increases GFR by dilating afferent arterioles while constricting efferent arterioles. ANP has been shown to be of some benefit to adult patients who have oliguric renal failure. Additional studies are warranted on the use of ANP for patients who have ARF, especially in the pediatric population.

Fluids

Fluid management depends on the patient's hemodynamic status and urinary output. The patient who presents with oliguria and hemodynamic instability should be given a fluid bolus of 20 mL/kg of an isotonic solution such as normal saline, packed red blood cells, or even albumin, although albumin's effectiveness is debated in the literature. These fluids should be administered as rapidly as possible, especially if the patient presents with sepsis. Repeat boluses may be given if the child remains hemodynamically unstable, as indicated by persistent low blood pressure or increased heart rate, decreased capillary refill, or no urinary output. A repeat bolus can be administered within minutes if the patient is in shock or until clinical improvement is evidenced.

Some nephrologists administer furosemide or mannitol to promote urination in an attempt to ease fluid management of ARF and increase the ability to give more nutrition. Both mannitol and furosemide increase urinary flow rate and decrease intratubular obstruction; both also may limit oxygen consumption in damaged

cells. Results of investigatory studies suggest that administration of furosemide or mannitol alone does not change the need for renal replacement therapies such as dialysis. Both furosemide and mannitol cause significant diuresis. Furosemide promotes excretion of sodium and potassium by inhibiting the sodium-potassium-chloride cotransporter in the thick ascending limb of Henle, thus allowing more water in the tubules. Mannitol is an osmotic diuretic. Prior to administering furosemide or mannitol, intravascular volume should be restored and the fractional excretion of sodium determined to identify the type of renal failure (Table 3). The patient's hemodynamic status should be assessed carefully before furosemide or mannitol administration.

Once intravascular volume is re-established, fluid intake should be restricted to 400 mL/m² per day (5% dextrose in water) plus urinary output and extrarenal losses, if the child has a urinary output of at least 1 mL/kg per hour, has pulmonary edema, has third-spacing of fluids, or meets the criteria for having ARF. This formula accounts for insensible water loss from the respiratory and GI tracts as well as from excretory losses. Final fluid adjustments depend on daily weights and close monitoring of the patient's intake and output.

Electrolytes

Frequent electrolyte abnormalities seen in ARF that must be corrected include hyponatremia, hyperkalemia, acidosis, and hypocalcemia (Table 4).

Hyponatremia (sodium concentrations <130.0 mEq/L [130.0 mmol/L]) usually is associated with hyponatre-

mic dehydration. If the serum sodium concentration is greater than 120.0 mEq/L (120.0 mmol/L), fluid restriction or water removal with dialysis should be considered, and sodium should be corrected to at least 125.0 mEq/L (125.0 mmol/L) by using the following calculation:

$$(125\text{-plasma sodium}) (\text{Wt in kilograms})(0.6)=\text{mEq Na}$$

This amount of sodium should be delivered slowly over several hours. If the patient is asymptomatic and the sodium value is less than 120.0 mEq/L (120.0 mmol/L), rapid correction to 125.0 mEq/L (125.0 mmol/L) should be considered because of the increased risk for seizures. Rapid correction versus slow correction is based on duration of hyponatremia as well as the patient's clinical appearance. If the patient is symptomatic and having seizures, 3% sodium chloride should be used for rapid correction of the sodium deficit to 125.0 mEq/L (125.0 mmol/L). Two equations can be used for administering 3% sodium chloride:

   a) 10 to 12 mL/kg of 3% NaCl infused over 1 hour
   b) Amount of 3% NaCl (mL)=[XmEq/L×body weight (kg)×0.6 L/kg]÷[0.513 mEq Na/mL 3% NaCl]

where X=(125 mEq/L−actual serum sodium) to raise serum sodium to 125.0 mEq/L (125.0 mmol/L) rapidly.

Hyperkalemia is a life-threatening abnormality in ARF that must be treated aggressively and quickly. Hyperkalemia is defined as mild-to-moderate when the potassium concentration is between 6.0 and 7.0 mEq/L (6.0 and 7.0 mmol/L) and severe when the concentration is greater than 7.0 mEq/L (7.0 mmol/L) with any electrocardiographic changes. Hyperkalemia develops because of a decrease in renal function, abnormal tubular potassium secretion, potassium shift out of cells into the extracellular space due to acidosis, or breakdown of tubular cells. If hyperkalemia is suspected, the patient should undergo electrocardiography to look for tall, peaked T waves.

Treatment of hyperkalemia should be initiated in patients whose potassium values are 6.0 mEq/L (6.0 mmol/L) or higher as well as in those who have peaked T waves, weakness, paresthesias, or tetany. Treatment of hyperkalemia includes infusion of 10% calcium gluconate (10 to 15 mL/kg) through a central venous line to stabilize the membrane potential; infusion of bicarbonate to shift the potassium intracellularly; infusion of insulin (0.1 to 0.5 U/kg) plus 10% to 25% glucose to allow for uptake of potassium from the extracellular space to the intracellular space; or administration

of sodium polystyrene sulfonate (1 g/kg rectally or orally) to exchange sodium for potassium in the colonic mucosa. Once administered, the resin takes a few hours to be effective.

If these attempts do not correct the patient's hyperkalemia, dialysis should be initiated, especially if anuria is present. Once the decision to begin dialysis has been made, the previously noted measures should be continued until dialysis is available. Once dialysis has begun, all electrolytes should be monitored to assess rebound after the initial and subsequent potassium concentrations are lowered.

Acidosis in the acute setting can be corrected by administering sodium bicarbonate intravenously if the acidosis is severe (bicarbonate, <8.0 to 10.0 mEq/L [8.0 to 10.0 mmol/L]). Calculation for the amount of bicarbonate to administer to correct the acidosis is:

$$\text{mEq of bicarbonate}=(\text{desired-observed bicarbonate})\times\text{kg}\times0.5$$

The correction should be undertaken with care because this is only a temporizing measure, and overcorrecting the acidosis may cause hypocalcemia.

Hypocalcemia results from several causes, including hyperphosphatemia, abnormal GI uptake, and skeletal resistance to parathyroid hormone. Hypocalcemia can be treated with intravenous calcium gluconate in acute severe situations, such as when the patient has tetany or cardiac arrhythmias. Oral calcium carbonate also is effective. Care should be taken when giving these products in the presence of hyperphosphatemia. Oral phosphate binders, such as calcium carbonate and calcium acetate, and noncalcium phosphate binders can be administered to decrease phosphorus concentrations. Vitamin D products can be provided to prevent secondary hyperparathyroidism that can occur in patients whose ARF is prolonged (Table 5).

Anemia
Children who have ARF do not need transfusions unless there is active bleeding, hemodynamic instability, or a hematocrit value below 25% (0.25).

Hypertension
Hypertension in ARF usually is due to volume overload or changes in vascular tone. If volume overload is present, diuresis with furosemide or dialysis should be initiated to return the patient to hemodynamic stability. If increased vascular tone is the cause of hypertension, intravenous antihypertensive treatment may be neces-

Table 5. **Vitamin D Dosing for Acute Renal Failure**

Vitamin D Analogs	Generic Name	Starting Dosage
1,25–dihydroxyvitamin D_3	Calcitriol	0.01 to 0.05 mcg/kg per day orally (<3 y of age) 0.25 mcg to 0.75 mcg per day (>3 y of age) Can be titrated to maintain normal PTH concentrations
Vitamin D_2	Dihydrotachysterol	
Vitamin D_2	Doxercalciferol	Oral and intravenous dosing available for adolescents and adults
Synthetic vitamin D analog	Paricalcitol	0.04 to 0.1 mcg/kg intravenously 3 times per week (>5 y of age)
1,25–dihydroxyvitamin D_3	Alfacalcidol	0.25 to 0.5 mcg/day orally and can be titrated to maintain normal PTH concentrations

PTH=parathyroid hormone

sary. Intravenous antihypertensive medications should be used if the patient cannot take oral medications (ie, intubation) or if severe hypertension exists (systolic or diastolic blood pressures at least at the 99th percentile for age, sex, and height). Sodium nitroprusside is an effective antihypertensive drug, but its use requires monitoring of thiocyanate concentrations because the kidney excretes this byproduct of metabolized nitroprusside. Intravenous labetalol, nicardipine, enalaprilat, and diazoxide have been administered to patients who have ARF to manage hypertensive crises. In cases where hypertension is not as severe, short-acting nifedipine can be administered (Table 6).

Nutrition

Proper nutrition is a major issue in patients who have ARF. Because these patients can be in a catabolic state, malnutrition is common. Infants who have ARF can be given a formula low in phosphorus. Older children can be fed formulas that provide proteins of high biologic value. If the patient cannot be fed enterally, supplemental intravenous alimentation should begin. Caloric intake should be aimed at providing greater than 70% of calories as carbohydrates (as dextrose up to 25%) and less than 20% as lipids, with biologic value proteins up to 0.5 to 2 g/kg per day.

Table 6. **Antihypertensive Medications**

Medications	Dose	Class	Comment
Nifedipine	0.25 to 1 mg/kg per dose PO/sublingual (maximum of 10 mg/dose or 3 mg /kg per day)	Calcium channel blocker	May cause reflex tachycardia; may increase cyclosporine concentration
Diazoxide	2 to 5 mg/kg per dose IV	Vasodilator	Use with caution; may cause rapid hypotension, hyperglycemia, sodium or water retention
Labetalol	0.2 to 1 mg/kg per dose IV 0.25 to 3 mg/kg per hour IV	Alpha and beta blocker	Contraindicated in patients who have asthma; may worsen heart failure
Hydralazine	0.1 to 0.5 mg/kg IV (drip 0.75 to 5 mcg/kg per minute)	Vasodilator	Tachycardia is seen frequently; fluid retention; flushing
Sodium nitroprusside	0.5 to 10 mcg/kg per minute IV	Vasodilator	Thiocyanate toxicity; rapid hypotension; may increase intracranial pressure and cerebral blood flow
Enalaprilat	5 to 10 mcg/kg per dose IV every 8 to 24 h	Angiotensin converting enzyme inhibitor	May cause angioedema of head or neck, hyperkalemia, renal dysfunction or failure, severe hypotension; use with extreme caution in renal failure

IV=intravenous, PO=oral

Medications

The kidneys clear many medications. If the patient who has ARF already is taking medications that are excreted by the kidney, the dosing of these drugs should be evaluated to determine if the dosages or intervals between doses should be adjusted to account for the degree of renal impairment.

Renal Replacement Therapy

When conservative medical management is unsuccessful in restoring renal function, dialysis is the therapy of choice. Indications for initiating dialysis include congestive heart failure, anemia, hyperkalemia, severe acidosis, pericarditis, and inadequate nutrition. For the acute presentation that requires dialysis, continuous venovenous (CVVH) or continuous arteriovenous hemofiltration (CAVH), acute hemodialysis, or acute peritoneal dialysis are appropriate forms of emergent dialysis for the pediatric patient. CVVH or CAVH allows fluid removal in the face of very low blood pressures. Also, neither CVVH nor CAVH interferes with providing adequate nutrition, enterally or parentally, for the patient in the intensive care unit.

Prognosis

Recovery from ARF may take days to weeks, a period that requires frequent, careful patient assessment. If renal failure continues for several weeks, transition to chronic care may be necessary. Over the last 15 to 20 years, improvement in the care of very ill children has led to a better prognosis for those who have ARF. Prognosis depends on several factors, including the need for dialysis, the time between onset of illness and presentation to medical care, and the underlying disease. Patients who present at a younger age and have multisystem organ failure appear to have worse prognoses. Therefore, early identification of patients who have ARF and early intervention are necessary to improve the current 10% to 60% mortality rates associated with these risk factors.

NOTE. An article on chronic renal failure will be published in the October 2008 issue of *Pediatrics in Review*.

Suggested Reading

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Goldstein SL. Pediatric acute kidney injury: it's time for real progress. *Pediatr Nephrol*. 2006;21:891–895

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PIR Quiz

Quiz also available online at www.pedsinreview.aappublications.org.

1. A previously well 2-year-old child has been ill for the past 4 days and has not urinated in the past 20 hours. There is no evidence of congestive heart failure on physical examination. The diagnosis of prerenal failure is strongly supported by:

   A. A history of recurrent colicky abdominal pain.
   B. A serum potassium concentration of 6.7 mEq/L (6.7 mmol/L).
   C. A urine sodium concentration of 9.0 mEq/L (9.0 mmol/L).
   D. Presacral and periorbital edema on examination.
   E. The presence of red blood cell casts in the urine.

2. A previously well 2-year-old child has been ill for the past 4 days and has not urinated in the past 20 hours. The diagnosis of intrinsic renal failure is strongly supported by:

   A. A history of recurrent colicky abdominal pain.
   B. A normal urinary sediment.
   C. A serum potassium concentration of 6.7 mEq/L (6.7 mmol/L).
   D. A urinary fractional sodium excretion of 3.5%.
   E. The absence of presacral or periorbital edema on examination.

3. A previously well 7–year–old boy who has been vomiting for the past 4 days presents with tachycardia, tachypnea, a blood pressure of 110/65 mm Hg, periorbital puffiness, and mild pitting edema of the lower extremities. His urine has been brown, and he has urinated only twice in the past 48 hours. While awaiting the results of appropriate laboratory studies, the most important *initial* step is an immediate infusion of:

   A. Diazoxide.
   B. Dopamine.
   C. Furosemide.
   D. Mannitol.
   E. Normal saline.

4. The initial venipuncture for the previously cited 7–year–old boy reveals a serum potassium value of 6.9 mEq/L (6.9 mmol/L). Electrocardiography reveals tall, peaked T waves. To relieve the hyperkalemia most quickly, the *most* appropriate next step is:

   A. Confirmation of hyperkalemia before starting treatment.
   B. Continuous arteriovenous hemofiltration.
   C. Infusion of insulin in 10% glucose.
   D. Oral administration of sodium polystyrene sulfonate.
   E. Rectal administration of sodium polystyrene sulfonate.

5. The initial venipuncture for the previously cited 7–year–old boy also reveals a serum sodium concentration of 126.0 mEq/L (126.0 mmol/L). After initial fluid resuscitation, the repeat value is 129.0 mEq/L (129.0 mmol/L). At this time, the *most* appropriate intervention is:

   A. Fluid restriction.
   B. Observation.
   C. Prophylactic administration of an anticonvulsant.
   D. Rapid correction of serum sodium concentration with 3% sodium chloride.
   E. Repeat normal saline boluses until sodium values are normal.

Clarification

In response to a reader's question, the authors of "Vomiting in Children: Reassurance, Red Flag, or Referral?" (*Pediatr Rev.* 2008;29:183–192) have provided an expanded explanation of the role of antihistamines in treating diarrhea. This explanation may be viewed by going to: http://pedsinreview.aappublications.org/cgi/eletters/29/6/183.

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