Therapy for AKI due to obstructive uropathy requires rapid diagnosis and intervention to relieve the obstructive process. Delayed interventions, especially in patients with complete obstruction, compromise recovery of kidney function. Upper urinary tract obstruction requires either retrograde ureteral stent placement or nephrostomy tube insertion when it is caused by severe retroperitoneal disease such as ureteral or bladder cancer. Relief of lower tract obstruction with a bladder catheter, a suprapubic tube (rarely), or a nephrostomy tube is the first step in treatment. Electrolyte and fluid management also are required to ensure patient safety in developing postobstructive diuresis. It is a phenomenon that occurs primarily in patients with bilateral, complete obstruction and is characterized by large urine volumes after relief of obstruction. Postobstructive diuresis is physiologic in that excess sodium and water are being excreted from the hypervolemic patient, but impaired tubular function (sodium and water) may lead to excessive diuresis and volume depletion. In this setting, judicious fluid repletion is required to avoid iatrogenic postobstructive diuresis as well as underresuscitation and hypotension.

## COMPLICATIONS OF AKI

Considering the normal functions of the kidneys, it is not surprising that a number of metabolic complications develop in the setting of AKI. Hyperkalemia is a potentially life-threatening complication that often requires urgent intervention. Hyperkalemia disturbs the magnitude of the action potential in response to a depolarizing stimulus. The electrocardiogram (ECG) is a better guide to therapy than a single measurement of potassium concentration. The sequential ECG changes observed in hyperkalemia are peaked T waves, PR prolongation, QRS widening, and a sine wave pattern. The presence of any of these ECG changes mandates prompt therapy.

Metabolic acidosis is common in AKI. However, it is usually well tolerated and does not require therapy unless arterial pH declines to less than 7.1. Hyperkalemia and severe metabolic acidosis not responsive to medical therapy are indications for initiation of RRT. Hypocalcemia is a common but asymptomatic finding and usually does not require therapy. Significant hyperphosphatemia may occur but often can be managed with oral phosphate binders. Anemia typically does not require treatment unless it is severe, is symptomatic, or contributes to cardiac dysfunction. Uremic manifestations of AKI are listed in E-Table 31-6. They may be subtle findings, or they may be obvious and life-threatening, requiring urgent RRT.

Importantly, infectious complications are the main cause of death because of the immune compromise, edema with end-organ dysfunction and skin breakdown, and numerous indwelling catheters in these patients.

## GENERAL MANAGEMENT OF AKI

Management of AKI begins with identification of the cause and pathogenesis of the inciting process. In addition, the complications associated with AKI need to be recognized and rapidly treated to avoid serious adverse events. Prerenal AKI requires optimization of renal perfusion by repletion of intravascular volume in those who are volume depleted and correction of heart failure, liver failure, and other "effective" causes of reduced intravascular volume. Intrinsic AKI requires directed therapy of the disturbed kidney compartment. Management of postrenal AKI mandates early intervention to relieve obstruction and preserve kidney function.

Most consequences of AKI are managed initially with conservative measures. These include interventions to correct hypovolemia or hypervolemia, improvement of hemodynamics, and correction of hyponatremia, hyperkalemia, metabolic acidosis, and hyperphosphatemia. Conversion of patients from oliguric to nonoliguric AKI makes management easier but does not improve outcomes in terms of morbidity or mortality. Manifestations of severe uremia and the other consequences of AKI, as listed in E-Table 31-6, may necessitate RRT if conservative measures are unsuccessful or incompletely reverse the complication.

Hospital-based RRT, which includes primarily acute hemodialysis and continuous renal replacement therapies (CRRTs), is required in certain patients with AKI. Continuous therapies, which can be employed only in the ICU, include continuous venovenous hemofiltration, hemodialysis, hemodiafiltration, slow low-efficiency dialysis, and extended daily dialysis. Emergent indications include severe hyperkalemia, uremic end-organ damage (e.g., pericarditis, seizure), refractory metabolic acidosis, and severe volume overload including pulmonary edema. Although the data do not support a cutoff BUN value to initiate RRT, it is sensible to initiate therapy before severe uremic complications develop. Intractable volume overload with anasarca complicated by skin breakdown is another potential indication. Acute hemodialysis is the modality most commonly employed to treat the consequences of AKI. However, critically ill patients who are hemodynamically unstable benefit most from continuous therapies. CRRT allows more precise control of volume, uremia, acid-base disturbances, and electrolyte disorders with less hemodynamic instability. CRRT also allows aggressive nutritional support. Peritoneal dialysis is rarely used for AKI but is a reasonable modality.

## OUTCOME AND PROGNOSIS OF AKI

Despite the significant advances in supportive care and RRT technology, acute and long-term complications, including mortality, remain common. The mortality associated with AKI in the hospital setting depends on the patient's severity of illness and burden of organ dysfunction. As the number of failed organs increases from 0 to 4, the mortality rate associated with AKI increases from less than 40% to more than 90%. Also, in-hospital mortality increases with AKI that develops in the medical or surgical ICU. Long-term outcomes for patients with AKI include increased risk for death (compared with hospitalized patients without AKI). Furthermore, patients with CKD who have a prehospitalization eGFR lower than 45 mL/min/1.73 $m^2$ who develop RRT-requiring AKI have a much higher mortality rate than patients with CKD not complicated by AKI. On the whole, all forms of AKI, including the RRT-requiring forms, appear to be associated with an increased risk for development of new CKD, progression of CKD, ESRD, and death.

*For a deeper discussion on this topic, please see Chapter 120, "Acute Kidney Injury," in* Goldman-Cecil Medicine, *25th Edition.*