The echogenicity of the kidneys is compared with that of liver parenchyma. Typically, the kidneys are less echogenic than the liver. Increased echogenicity of the kidneys suggests the presence of scarring and therefore CKD. Renal ultrasonography can also easily detect the presence of cysts in the kidneys and therefore is a useful technique to detect polycystic kidney disease. Pulsed Doppler imaging is often used to calculate the resistive index by estimating the systolic and diastolic Doppler velocities in the renal cortex. A resistive index greater than 0.8 suggests that interventional procedures to revascularize the kidney would be unlikely to benefit the patient in terms of improving blood pressure or protecting the long-term decline in kidney function. If the two kidneys differ in size by 1.5 cm, it suggests the presence of renovascular disease in an adult. In children, reflux nephropathy or congenital abnormalities are more common causes.

Computed tomography (CT) of the kidney is often helpful to evaluate complex cysts. In contrast to simple cysts, complex cysts are suspicious for the presence of malignancy, and CT can evaluate them better than ultrasonography. Likewise, CT is important for evaluating renal masses, stones, retroperitoneal conditions (e.g., hemorrhage, tumor, abscess), and renal vein thrombosis. In morbidly obese people, CT is often used to guide kidney biopsy. The use of contrast agents to assess vascular lesions of the kidney may be not be possible if kidney function is compromised due to fear of precipitating AKI. Limiting the volume of the contrast agent and volume repletion before radiocontrast administration may minimize renal injury.

Although intravenous pyelography can image the structures in the kidney, contrast CT has taken the place of classic intravenous pyelography in many centers because of the risk of inducing nephrotoxicity in patients with CKD. In contrast, retrograde pyelography is often used by urologists to define the site and nature of obstruction within the ureter and the pelvis. In addition, during the procedure, ureteric stones can be removed with the use of a basket device.

Magnetic resonance imaging (MRI) is useful for imaging of the vasculature and therefore for the diagnosis of renal vein thrombosis and renal artery stenosis. Gadolinium-based contrast agents are often used for MRI because of their paramagnetic properties. These agents should be avoided if the GFR is less than 30 mL/min/1.73 m^2, because in such patients they have been implicated in causing a disabling and untreatable condition called *nephrogenic systemic fibrosis*. Two other MRI contrast agents (one containing iron and another containing manganese) may be used in such patients but are approved by the U.S. Food and Drug Administration only for the evaluation of lesions in the liver. MRI cannot be performed in patients who have metallic implanted devices such as pacemakers, artificial joints, or aneurysmal clips.

After injection of a small amount of radioactive substance, radionuclide imaging can be performed to assess renal perfusion and function of the kidneys. One advantage of this technique is that it can assess kidney function and perfusion simultaneously for each kidney. It therefore allows diagnosis of renal artery stenosis, especially when it is performed before and after administration of angiotensin-converting enzyme (ACE) inhibitors.

Renal arteriography is the reference standard for the diagnosis of renal artery stenosis. It involves direct injection of a radiocontrast dye into the renal arteries. In patients with CKD, contrast injection can be limited and carbon dioxide can be injected to avoid nephrotoxicity. This technique is also useful for assessing vascular malformations in the kidney and for making a diagnosis of polyarteritis nodosa. In the latter condition, renal arteriography can detect the presence of microaneurysms.

APPROACH TO THE PATIENT WITH ACUTE KIDNEY INJURY

The approach to patients with AKI depends on four major factors: (1) the evaluation of risk or susceptibility to renal injury, (2) the nature of the AKI, (3) the severity of injury, and (4) the presence of distant organ effects or consequences. In all cases, it is important to evaluate and optimize intravascular volume early in the course, because this is a readily addressable factor that can prevent or minimize further injury.

The risk factors for AKI include, first and foremost, prior existence of CKD; CKD can easily be detected by a low estimated GFR or the presence of albuminuria. Other common risk factors for AKI include advanced age, diabetes mellitus, hypertension (especially when treated with inhibitors of the renin-angiotensin system), chronic liver disease or cirrhosis, and multiple myeloma.

AKI is a challenging medical problem, and a careful and stepwise approach to evaluation is essential. This approach is guided by knowledge of the causes of injury, which can be divided into five major groups: ischemia, toxins, obstruction, inflammation, and infection.

Ischemia can be caused by volume loss from the gastrointestinal system (vomiting or diarrhea), the skin (sweating, burns), or the kidneys (diuretics, Addison's disease, and solute diuresis). Comparing the body weight of the patient with those weights recorded in the medical record can be valuable. A substantial decrease in body weight may point toward volume depletion as a possible cause of AKI. Third-space fluid losses, as observed patients with ascites, pancreatitis, or ileus, can make the diagnosis of volume depletion challenging because such patients may not have an overall loss in body weight. Ischemia is a common cause of AKI due to poor perfusion associated with significant blood loss or sepsis or both. In the setting of ischemia, glomerular hypoperfusion is aggravated when patients are taking inhibitors of the renin-angiotensin system.

Nephrotoxins can be divided into two major groups: endogenous and exogenous. The endogenous toxins include paraproteins, myoglobin, hemoglobin, uric acid (e.g., in tumor lysis syndrome), and bile acids. Exogenous toxins include contrast dyes, aminoglycosides, chemotherapeutic agents such as cisplatin, and NSAIDs.

Inflammation can involve the glomerular, interstitial, and vascular compartments. Inflammation of these structures produces glomerulonephritis, interstitial nephritis, and vasculitis, respectively.

Infection is an important cause of injury to the nephron. Most often it happens in the intensive care unit, where early sepsis can manifest as a fall in urine output followed by an increase in serum creatinine, confirming AKI. The causes of AKI in the setting of sepsis are multifactorial and include ischemia, direct tubular dysfunction due to sepsis, and concomitant administration of drugs such as nephrotoxic antibiotics (commonly, high doses of vancomycin) and procedures (radiocontrast imaging), often performed