Acute kidney injury: A nursing challenge

Rely on interprofessional collaboration, follow the evidence.

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CNE
1.5 contact hours

Learning Objectives

1. Explain causes and pathophysiology of acute kidney injury (AKI).
2. Describe pre-renal, intra-renal, and post-renal AKI and AKI staging.
3. Discuss nursing interventions in interdisciplinary collaborative management that can impact patient outcomes.

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AKI by the numbers

Worldwide, an estimated **1 in 5 adults** and **1 in 3 children** may experience AKI during hospitalization. The **prevalence** can be as high as **50%** among patients in the intensive care unit, with **mortality** rates as high as **80%** among those who require renal replacement therapy. AKI can **increase cost of care over two-fold**, depending on severity of disease, as a result of increased length of stay and costly interventions, such as dialysis.

Kay Dempsey, a 70-year-old woman, arrives by ambulance at the emergency department (ED) after being found lethargic, pale, and diaphoretic in the nursing home where she lives. Her medical history includes hypertension, hyperlipidemia, type 2 diabetes, heart failure (HF), and kidney stones. On admission, her vital signs are blood pressure (BP) 86/40 mmHg, heart rate (HR) 136 beats per minute (bpm), respiratory rate (RR) 30 breaths per minute, oxygen saturation 94% on 6 L of oxygen by nasal cannula, and temperature 101.4° F (38.5° C). In addition, she has +1 edema of her lower extremities.*

Abnormal findings from Ms. Dempsey's initial basic metabolic panel (BMP) include blood urea nitrogen (BUN) 35 mg/dL (normal: 8–23 mg/dL), serum creatinine (SCr) 1.6 mg/dL (normal: 0.6–1.2 mg/dL), potassium 5.2 mEq/L (normal: 3.5–5 mEq/L), and glucose 224 mg/dL (normal: 70–110 mg/dL).

Notable findings from the complete blood count (CBC) upon admission include white blood cells 24,000/mm^3 (normal: 4,500–11,000/mm^3), hemoglobin 10 g/dL (normal: 12–14 g/dL), and hematocrit 30% (normal: 36–44%). Arterial blood gasses show that Ms. Dempsey's are pH 7.30 (normal: 7.35–7.45), PaO2 116 mmHg (normal: 80–100 mmHg), PaCO2 30 mmHg (normal: 35–45 mmHg), and HCO3 16 mEq/L (normal: 22–26 mEq/L). Her serum lactate is 5.4 mmol/L (normal: 0.6–2.2 mmol/L).

In the ambulance, Ms. Dempsey received a 500 mL bolus of 0.9% normal saline without improvement in her vital signs. A physical exam in the ED reveals a weeping Stage 4 sacral pressure injury. Following sepsis protocol, the ED physician initiates 30 mL/kg of normal saline and consulted the intensivist.

Understanding kidney injury

The renal system comprises the kidneys, ureters, and urethra, which maintain homeostasis via filtration and excretion of waste products, participate in acid–base balance, produce renin for maintaining hemodynamics, and produce erythropoietin to stimulate red blood cell production. Insult to this system can result in electrolyte derangements, increased risk of stroke, long-term renal failure, or death. Kidney failure is broadly classified as either acute or chronic (chronic kidney disease [CKD]). This article focuses on acute kidney injury AKI. (See *AKI by the numbers*.)

AKI, a subset of acute kidney disease (AKD), is characterized by a sudden decrease in renal function, which develops over a few hours or several days. It can lead to significant increases in creatinine and reductions in urine output. Practice guidelines developed by the Kidney Disease Improving Global Outcomes (KDIGO) workgroup defines AKI as meeting any of the following criteria:

- Increase in SCr by 0.3 mg/dL or more within 48 hours
- Increase in SCr to 1.5 times baseline or more, which is known or presumed to have occurred within the past 7 days
- Urine output less than 0.5 mL/kg/hour for 6 hours

These criteria rely on monitoring the patient's creatinine and accurately measuring urine output of at-risk patients, including older adults, who have lower baseline renal function as measured by the glomerular filtration rate (GFR). Other risk factors include hypertension, diabetes, heart failure, cardiovascular disease, and liver disease. Use of nephrotoxic agents—such as antibiotics, non-steroidal anti-inflammatory drugs (NSAIDs), and radiocontrast dyes—also increase AKI risk.

Causes and pathophysiology

Susceptibility to AKI increases in the presence of combinations of exposures, risk factors, and comorbidities. For example, an older adult diagnosed with sepsis who has a history of diabetes and experiences volume depletion is at increased risk for AKI. When admitting at-risk patients, providers should conduct risk assessments that target interventions to address reversible causes of AKI. (See *AKI causes*.)

Intraoperative hypotension (mean BP less than 60 mmHg for at least 10 minutes) among noncardiac surgery patients is associated with an increased risk for postoperative AKI. In addition, AKI is more common among patients with a history of CKD, a body mass index >32 kg/m2, and a GFR <60 mL/minute.

The intensivist evaluates Ms. Dempsey in the ED, where a comprehensive physical exam, in-

AKI causes

Depending on the cause of acute kidney injury (AKI), the condition is categorized as pre-renal, intra-renal, or post-renal.

Causes	Pathophysiologic features	Care priorities

Pre-renal (decreased blood flow leads to a drop in GFR)
• Hypotension
• Hypovolemia
• Associated conditions
  ◦ Anaphylaxis
  ◦ Bowel obstruction
  ◦ Burns
  ◦ Cirrhosis
  ◦ Heart failure
  ◦ Hepatorenal syndrome
  ◦ Major surgery
  ◦ Overdiuresis
  ◦ Peritonitis
  ◦ Sepsis
  ◦ Shock
  ◦ Trauma
	• High BUN/creatinine ratio
	• Perfusion reduction (hypoperfusion of structurally normal kidneys)
		• Provide fluid resuscitation.
		• Establish hemodynamic stability.
		• Treat reversible causes.

Intra-renal (disease process or treatment damages the kidneys)
• Nephrotoxic drugs
  ◦ Antibiotics (e.g., aminoglycosides)
  ◦ Antiretrovirals (e.g., tenofovir)
  ◦ Chemotherapy (e.g., cyclosporin, methotrexate)
  ◦ Iodinated contrast media
  ◦ NSAIDs
• Toxins
  ◦ Myoglobin (e.g., rhabdomyolysis)
  ◦ Uric acid
• Associated conditions
  ◦ Glomerulonephritis
  ◦ Hemolytic uremic syndrome
  ◦ Infective endocarditis
  ◦ Organic solvent poisoning
  ◦ Renal artery stenosis
  ◦ Renal embolism
  ◦ Renal vasculitis
	• Insolubility of some drugs in the urine may lead to tubular precipitation as crystals.
	• Iodinated contrast causes renal vasoconstriction, leading to tubular epithelium injury and consequent loss of function, apoptosis, and necrosis.
	• Parenchymal damage results from structural injury to the nephron, which leads to inflammation and interstitial injury of the glomeruli, tubules, and surrounding tissues.
		• Adjust renal dose.
		• Eliminate nephrotoxic medications.
		• Treat reversible causes.

Post-renal (a process downstream of the kidney prevents urine drainage) • BPH • Obstruction of indwelling urinary catheter • Renal calculi • Tumor (bladder, prostate, or cervical cancer)	• Significant intrarenal or extrarenal urinary tract obstruction	• Perform urinary catheterization. • Prevent UTI. • Relieve obstruction. • Treat reversible causes.

BPH = benign prostatic hyperplasia, BUN = blood urea nitrogen, NSAIDs = nonsteroidal anti-inflammatory drugs, UTI = urinary tract infection

cluding a bedside ultrasound of various organs, is performed. Bibasilar mild consolidation are noted on the lung ultrasound. The abdominal and renal ultrasound rule out post-renal causes and bowel obstructions. The intensivist orders the patient transferred to the medical intensive care unit (MICU) for further evaluation and treatment.

The provider determines urosepsis as the most likely cause of Ms. Dempsey's AKI. He orders two sets of blood cultures, urine and sputum cultures, and a wound culture and sensitivity. After an infectious disease consult, the provider orders an empiric I.V. antibiotic regimen of piperacillin/tazobactam. (See Gerontologic considerations.*)*

Manifestations

Unlike myocardial infarction (MI) or stroke, which frequently have immediate signs such as chest pain or hemiparesis, respectively, AKI signs are nonspecific or occur later because of downstream effects that take time to manifest. These signs include fluid overload (which presents as edema, most noticeably in the lower extremities), electrolyte imbalances (such as hyperkalemia and hyperphosphatemia, which may cause nausea and vomiting), generalized weakness, and ECG abnormalities (such as peaked T waves, heart blocks, and ventricular tachycardia).

Urea accumulation in patients with AKI can lead to metabolic encephalopathy, which may manifest as altered mental status, seizures, and coma. In patients who exhibit these signs and symptoms as a direct result of AKI, mortality and morbidity rates are high. They'll require renal replacement therapy and multimodal symptom management.

On her first MICU day, Ms. Dempsey remains lethargic. She maintains adequate ventilation and continues to receive 4 L of oxygen via nasal cannula with 96% O_2 saturation. In anticipation of septic shock, the intensivist orders a multilumen central venous catheter inserted via the right internal jugular vein. Urinalysis indicates the presence of nitrites, leukocyte esterase, and large amounts of white blood cells and bacteria.

Repeat BUN and SCr are 38 mg/dL and 1.8 mg/dL, respectively, an increase from baseline. Ms. Dempsey's BMP is significant for increasing potassium (5.8 mEq/L) and phosphorus (5.3 mg/dL). A 12-lead ECG reveals peaked T-waves and a PR interval of 0.24 seconds (normal: 0.12 to 0.20 seconds). The provider orders 10 units of regular insulin I.V. push, 1 ampule of 50% dex-

trose I.V. push, and 10 g of oral sodium zirconium cyclosilicate. After 4 hours, Ms. Dempsey's potassium is 5.3 mEq/L, and a repeat ECG shows a PR interval of 0.18 seconds and peaked T waves with less amplitude. The ECG changes signify resolution of the first-degree AV block and a return to baseline of T-waves as the serum potassium moves closer to the normal range.

Diagnosis

The KDIGO workgroup identified three stages of AKI from Stage 1 (mild) to Stage 3 (the most severe). The AKI stage serves as a predictor of mortality, and the guidelines recommend staging patients according to the criteria with the highest stage. For example, if SCr is at Stage 3 and urine output is at Stage 2, the patient should be staged at Stage 3 based on SCr. (See *AKI staging*.)

Because renal and non-renal factors can influence SCr, staging criteria have limitations. For example, SCr measurements may be confounded by outside factors such as diuretic use, creatine supplementation, variable volumes of distribution in different patients, and altered creatinine production. Differentiating between chronic kidney injury and AKI without a baseline for the individual patient can prove difficult.

In addition to monitoring SCr, BUN, electrolytes, CBC, and urinalysis, providers may order imaging procedures based on the suspected cause of AKI. For example, the provider may order a kidney ultrasound to rule out urinary tract obstruction or structural abnormalities.

The provider orders a daily BMP and CBC but decides against any imaging procedures requiring an iodinated contrast to prevent exacerbating AKI with a nephrotoxic substance. The patient's central venous pressure ranges from 14 to 18 mmHg (normal: 8–12 mmHg). A chest x-ray indicates bilateral infiltrates, and an echocardiogram shows an ejection fraction of 30%.

Novel biomarkers

To aid CKD diagnosis, Inker and colleagues recommend using cystatin C levels to estimate GFR or in confirmatory testing for eGFR. Cystatin C is a protein produced by nucleated cells and eliminated by the kidneys. It's processed similarly to SCr, but is less dependent on muscle mass and isn't affected by race, which allows for more accurate GFR estimation. In critically ill patients, cystatin C has been shown to detect AKI 1 to 2 days before an increase in SCr. An increased cystatin C level indicates reduced GFR. Normal cystatin C values vary according to the lab and assay manufacturer, so note lab reference values.

Contrast-induced AKI

Providers make a diagnosis of contrast-induced AKI (CI-AKI) based on various values.

Gerontologic considerations

Physiologic renal changes associated with aging include the following:

- reduced kidney size and weight
- lower glomerular filtration rate
- glomerulosclerosis
- decreased sodium reabsorption and potassium excretion
- reduced ability to concentrate urine

With the overall reduction in physiologic reserves, older adults have a higher risk for acute kidney injury. Complications associated with kidney disease, such as cardiovascular disease, contribute to decreased quality of life, frailty, higher care costs, and increased mortality.

Therapeutic implications include ensuring the following:

- adequate hydration
- effective management of comorbidities such as diabetes and hypertension to reduce renal damage
- close monitoring of serum creatinine levels and urine output
- comprehensive geriatric assessment

AKI staging

The Kidney Disease Improving Global Outcomes workgroup identified three acute kidney injury (AKI) stages, with Stage 1 being mild and Stage 3 being severe. Staging is determined based on serum creatinine (SCr) level and urine output.

Stage	Serum creatinine	Urine output
1	1.5 to 1.9 times baseline OR ≥0.3 mg/dL increase	<0.5 mL/kg/hr for 6 to 12 hours
2	2.0 to 2.9 times baseline	<0.5 mL/kg/hr for ≥12 hours
3	3.0 times baseline OR Increase in SCr to ≥4.0 mg/dL OR Initiation of renal replacement therapy OR In patients <18 years, decrease in eGFR to <35 mL/min per 1.73 m^2	<0.3 mL/kg/hr for ≥24 hours OR Anuria for ≥12 hours

Source: Kidney Disease: Improving Global Outcomes kdigo.org/wp-content/uploads/2016/10/KDIGO-2012-AKI-Guideline-English.pdf

GFR estimation

Normal serum creatinine (SCr) is 0.6–1.2 mg/dL for men and 0.5–1.0 mg/dL for women; normal urine output ranges from 0.5 to 1 mL/kg/hour. Glomerular filtration rate (GFR) is the most-used kidney function index, with SCr and urine output changes serving as surrogates for GFR changes. In young, healthy adults, normal GFR is approximately 120 mL/min/1.73m^2; it decreases with age. Age, sex, and body size affect normal GFR readings. An abrupt decline in GFR is assessed from an increase in SCr or oliguria (abnormally low urine output).

To achieve an estimated GFR (eGFR) without the coefficient for race, most organizations use the 2021 CKD-EPI Creatinine Equation; some use eGFRcr-cys. Both equations omit race as a variable, but the 2021 CKD-EPI Creatinine Equation doesn't include cystatin C. eGFRcr-cys is more accurate and has smaller differences between Black and non-Black individuals than new equations without race that use creatinine or cystatin C alone. Individual organizations determine which eGFR equation the local laboratory uses. Clinicians also can access the National Kidney Foundation's online eGFR calculator.

For example, they'll diagnose CI-AKI if the patient experiences increased SCr of ×0.5 mg/dL or a 25% increase from baseline within 7 days after exposure to contrast medium, increased plasma creatinine level by at least 0.3 mg/dL over baseline within 48 hours after exposure, or urinary output of less than 0.5 mL per kg of body weight per hour that persists for at least 6 hours after exposure.

Collaborative management

AKI management requires interdisciplinary collaboration aimed at making renal dose adjustments, providing hemodynamic support, avoiding nephrotoxic medications, and preventing hyperglycemia. Nurses play a vital role in this care, as well as in managing continuous renal replacement therapy (CRRT).

Renal dose adjustment

Based on the severity of kidney dysfunction, many renally cleared medications require serum-level checks, as well as dosage and frequency reviews and adjustments based on creatinine clearance or eGFR. Nurses must monitor for signs of drug toxicity among patients with AKI, especially those prescribed medications with a narrow therapeutic range, such as digitalis and antibiotics. (See *GFR estimation*.)

Hemodynamic support

To prevent AKI resulting from hypotension and shock, hemodynamic monitoring can include checking cardiac filling pressures. Providers may order volume expansion with normal saline, particularly for patients at increased risk for CI-AKI. KIDGO recommends avoiding diuretics to treat AKI, except when managing volume overload. Hourly urine output monitoring is essential for critically ill patients. The theoretical advantage of urine output monitoring over SCr monitoring is the speed of the response. For example, if GFR suddenly falls to zero, an SCr increase wouldn't be detected for several hours; however, urine output would be affected immediately.

Patients with pre-renal AKI require adequate volume and perfusion pressure. If the patient is in shock, the provider may order vasopressors. Concern that normal saline may result in hyperchloremic acidosis and increase AKI risk (or worsen existing AKI), has resulted in use of balanced salt solutions (crystalloids with a chloride concentration closer to that in human plasma). Henninger and colleagues found that, among ICU patients with sepsis, the incidences of AKI didn't differ when using either lactated Ringer's solution or normal saline for fluid resuscitation. When initiating fluid resuscitation, nurses should consider the patient's comorbidities; contraindications for both normal saline and lactated Ringer's solutions exist but are outside the scope of this article.

Nephrotoxic medication avoidance

Nephrotoxic drugs play a role in 20% to 30% of AKI cases. On admission, nurses should obtain a full list of the patient's medications, including nonprescription drugs and herbal supplements. Prescribers should always check each prescription for renal adverse effects. For example, controversy exists around the use of vitamin C in the treatment of sepsis; evidence indicates that high doses of vitamin C can lead to AKI in these patients.

Providers should avoid procedures that require iodinated radiocontrast in patients at risk for AKI. Routine administration of acetylcysteine isn't recommended to prevent AKI or long-term adverse events after angiographic procedures. Nurses should monitor SCr and urine output for all patients who have a radiocontrast procedure. Periprocedural use of normal saline has been shown to prevent CI-AKI, although the optimal volume isn't known. The American College of Radiology guidelines on the administration of contrast media recommend isotonic saline at a rate of 100 mL per hour for 6 to 12 hours before angiography and 4 to 12 hours after angiography. After angiography, nurses should monitor patients for signs of pulmonary edema. All clinicians should know the eGFR thresholds below which antibiotic,

contrast medium, chemotherapy, and other agents shouldn't be used or their doses adjusted.

Hyperglycemia prevention

Acute or critical illness can cause stress hyperglycemia as a result of insulin resistance and anxiety response. Uncontrolled hyperglycemia is associated with increased incidences of AKI in patients with MI. The KDIGO workgroup recommends insulin therapy with a target plasma glucose of 110 to 149 mg/dL in critically ill patients. To avoid hypoglycemia, nurses should frequently monitor the blood glucose of patients at risk for AKI and those on insulin therapy. A study by Marenzi and colleagues has shown that in patients with diabetes experiencing an acute MI, AKI is better predicted by the combined evaluation of acute and chronic glycemic values. This means that clinicians also should check A1C levels.

On Day 2 in the MICU, Ms. Dempsey develops hypotension (BP 88–92/50–54 mmHg) with an HR of 100 to 120 bpm and sinus tachycardia, even after a 2-liter normal saline bolus. She's electively intubated, which reduces her BP to as low as 52/28 mmHg. The provider orders norepinephrine at 5 mcg/minute, which is titrated up to 20 mcg/minute. A mean arterial pressure of 62–68 mmHg is achieved. To facilitate ventilator adherence, the provider orders I.V. fentanyl and propofol.

The nurse inserts a nasogastric tube and an indwelling urinary catheter. Ms. Dempsey's hourly urine output is 10 to 15 mL/hour for the next 5 hours. A nutrition consult is requested to optimize the patient's nutrition while minimizing electrolyte imbalance. The consult results in Ms. Dempsey receiving a diet low in protein, sodium, potassium, and phosphorous. The patient's blood cultures grow Escherichia coli*, so the provider orders continuation of I.V. piperacillin/tazobactam and adds I.V. meropenem.*

For critically ill patients with AKI, nurses should conduct daily and periodic eGFR checks to monitor disease progression and treatment response. Even after an apparent resolution of AKI, long-term risks exist for CVD (especially HF) and CKD.

Continuous renal replacement therapy

If a patient with AKI becomes anuric and shows signs of a prolonged inability to clear electrolytes and waste (for example, refractory hyperkalemia, severe metabolic acidosis, and metabolic encephalopathy), they meet the criteria for renal replacement therapy, which may include intermittent hemodialysis (IHD) and CRRT. Providers may opt for CRRT over IHD for patients who are hypotensive and can't tolerate the large shifts in volume.

CRRT for patients with severe AKI due to sepsis has the added benefit of removing endotoxins and cytokines (such as interleukins) and tumor necrosis factor alpha, which have a direct correlation with mortality. The electrolyte composition of the therapy fluid is tailored to the patient's electrolyte panel, so nurses should draw serial metabolic panels. The nurse's role also includes maintaining asepsis to prevent central line-associated bloodstream infection.

Over the next 12 hours, Ms. Dempsey's metabolic acidosis worsens, her serum lactate increases to 10.2 mmol/L, she has persistent hyperkalemia up to 6.0 mEq/L, and she's unresponsive to medical treatment. The patient's hemodynamic instability prompts the provider to order CRRT, which Ms. Dempsey tolerates well. After 12 hours of uninterrupted therapy, her vasopressor requirements decrease, and her metabolic acidosis improves—serum lactate falls to 5.8 mmol/L and potassium to 5.4 mEq/L; however, urine output remains <10 mL/hour.

After 2 weeks in the MICU, Ms. Dempsey recovers from sepsis and the provider orders extubation. She completes 48 hours of CCRT and is weaned off of norepinephrine. Her serum lactate drops to 2.0 mmol/L, and her potassium returns to normal. At the time of transfer to a stepdown unit, Ms. Dempsey's urine output is 0.5 mL/kg/hour and her SCr is 1.3 mg/dL.

Apply the best evidence

AKI is common in hospitalized patients, especially those who are critically ill. Nurses play an integral role in applying the best evidence in the screening, management, and prevention of AKI. AN

*Name is fictitious.

Access references at myamericannurse.com/?p=353920.

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POST-TEST • Acute kidney injury: A nursing challenge

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Please mark the correct answer online.

1. Which of the following is correct about acute kidney (AKI) incidence and cost of care?

a. Worldwide, one in ten adults may experience AKI during hospitalization.
b. Worldwide, one in four children may experience AKI during hospitalization.
c. The prevalence of AKI can be as high as 25% among patients in intensive care units.
d. AKI can increase the cost of care over two-fold.

2. Which of the following meets the definition for AKI in the practice guidelines developed by the Kidney Disease Improving Global Outcomes (KDIGO) workgroup? (Select all that apply.)

a. Increase in serum creatinine by 0.1 mg/dL or more within 48 hours
b. Increase in serum creatinine by 0.3 mg/dL or more within 48 hours
c. Increase in serum creatinine to 1.0 times baseline or more, which is known or presumed to have occurred within the past 7 days
d. Increase in serum creatinine to 1.5 times baseline or more, which is known or presumed to have occurred within the past 7 days
e. Urine output <0.5 mL/kg/hour for 6 hours

3. Intra-renal acute kidney injury can be caused by which of the following?

a. Hypovolemia
b. Renal calculi
c. Nephrotoxic drugs
d. Urinary catheter obstruction

4. AKI staging is based on which of the following measures? (Select all that apply.)

a. Serum creatinine
b. Glomerular filtration rate
c. Blood urea nitrogen
d. Urine output
e. Urinalysis
f. Renal ultrasound

5. Which of the following is not considered a physiologic renal change associated with aging?

a. Reduced kidney size and weight
b. Lower glomerular filtration rate
c. Glomerulosclerosis
d. Increased sodium reabsorption and potassium excretion
e. Reduced ability to concentrate urine

6. Intraoperative hypotension among noncardiac surgery patients is associated with an increased risk for postoperative AKI. Which of the following is the correct definition of intraoperative hypotension?

a. Mean BP less than 60 mmHg for at least 10 minutes
b. Mean BP less than 80 mmHg for at least 10 minutes
c. Mean BP less than 60 mmHg for at least 5 minutes
d. Mean BP less than 80 mmHg for at least 5 minutes

7. AKI signs include which of the following? (Select all that apply.)

a. Fluid overload
b. Electrolyte imbalances
c. Generalized weakness
d. ECG abnormalities (such as peaked T waves, heart blocks, and ventricular tachycardia).

8. The KIDGO workgroup recommends avoiding diuretics to treat AKI, except when managing what condition?

a. Metabolic encephalopathy
b. Hypovolemia
c. Volume overload
d. Post-renal AKI

9. Which of the following is correct about iodinated radiocontrast in patients at risk for AKI? (Select all that apply.)

a. Nurses should monitor SCr and urine output for all patients who have a radiocontrast procedure.
b. Routine administration of acetylcysteine is recommended.
c. Normal saline infusion pre- and postprocedure is recommended.
d. All clinicians should know the eGFR threshold below which contrast medium shouldn't be used or should be adjusted.

10. Which of the following is correct about continuous renal replacement therapy (CRRT)? (Select all that apply.)

a. CRRT may be preferred over hemodialysis for patients who can't tolerate large shifts in volume.
b. CRRT removes endotoxins and cytokines, which can reduce mortality in sepsis patients.
c. The electrolyte composition is tailored to the patient's electrolyte panel.
d. CRRT removes tumor necrosis factor alpha.