



Preventing and Responding to Acute Kidney Injury

How to recognize the incremental changes in kidney function that adversely affect patient outcomes.

OVERVIEW: Acute kidney injury is an independent risk factor for both prolonged length of hospital stay and in-hospital mortality. Recent analysis shows that over the past decade the incidence of acute kidney injury requiring dialysis rose rapidly in the United States, with associated death more than doubling. In 2007, the Acute Kidney Injury Network proposed a new classification system for acute kidney injury, which recognized that incremental changes in kidney function may adversely affect outcomes. By identifying the signs and symptoms of acute kidney injury in its early stages, nurses may be able to help reduce the severity of injury and contribute to improved outcomes.

Keywords: acute kidney injury, acute renal failure, chronic kidney disease, creatinine, glomerular filtration rate, renal disease, urine output

Sally Jansen, a healthy, active 62-year-old, fell in her home after tripping on a throw rug. (This case is a composite based on my experience.) Ms. Jansen immediately realized that she'd "broken something" in her hip. She tried to drag herself toward the phone on the other side of the kitchen, but the excruciating pain caused her to pass out. When she regained consciousness, she called out for help, but there was no one to hear her. Because Ms. Jansen was taking warfarin (Coumadin and others) for atrial fibrillation, her left thigh swelled rapidly with an expanding hematoma. She controlled her hypertension with the diuretic furosemide (Lasix) and the angiotensin-converting enzyme (ACE) inhibitor lisinopril (Prinivil, Zestril) and had taken both that morning. By the time her daughter found her on the floor, 12 hours after the fall, Ms. Jansen was in excruciating pain and, having been unable to hold her urine, soaking wet.

On admission to the hospital, Ms. Jansen was markedly hypotensive and tachycardic secondary to blood loss and dehydration. Her hemoglobin concentration was 7.5 g/dL, requiring aggressive fluid resuscitation and three units of blood. Immediately following an open reduction internal fixation of the

hip, a 7.5 × 8 cm area of deep tissue injury over her sacrum opened to a stage IV pressure ulcer.

Ms. Jansen's history revealed her to be at high risk for acute kidney injury. If you were her nurse, would you be able to identify her risk factors? Would you know what steps to take to monitor her condition and prevent acute kidney failure?

ACUTE KIDNEY INJURY

An acute kidney injury is an abrupt change in kidney function signaled by a rise in serum creatinine and a reduction in urine output. This type of injury usually occurs within 48 hours of one or more precipitating events.

In 2007, the Acute Kidney Injury Network (AKIN) proposed that the term "acute kidney injury" be used to describe "the entire spectrum of acute renal failure."¹ The AKIN classification system, which recognized that incremental changes in kidney function may adversely affect outcomes, defined acute kidney injury as follows¹:

An abrupt (within 48 hours) reduction in kidney function currently defined as an absolute increase in serum creatinine of more than or

equal to 0.3 mg/dL . . . , a percentage increase in serum creatinine of more than or equal to 50% (1.5-fold from baseline), or a reduction in urine output (documented oliguria of less than 0.5 mL/kg per hour for more than six hours).

The AKIN categorized the severity of acute injury into three stages based on increase in serum creatinine and reduction in urine output.¹ Prior to the AKIN, the Acute Dialysis Quality Initiative classified kidney disease by criteria that progressed along a timeline

from observed risk through injury, failure, loss of function, and ultimately, end-stage renal disease (RIFLE).² (See Table 1.^{1,2}) Both sets of criteria are frequently used in research involving acute kidney injury.³⁻¹¹

Unlike acute kidney injury, chronic kidney disease progresses slowly and is staged based on glomerular filtration rate (GFR), with a normal or high GFR (at or above 90 mL/min/1.73 m²) signifying kidney damage, or stage 1, and a low GFR (below 15 mL/min/1.73 m²) signifying kidney failure, or stage 5.¹²

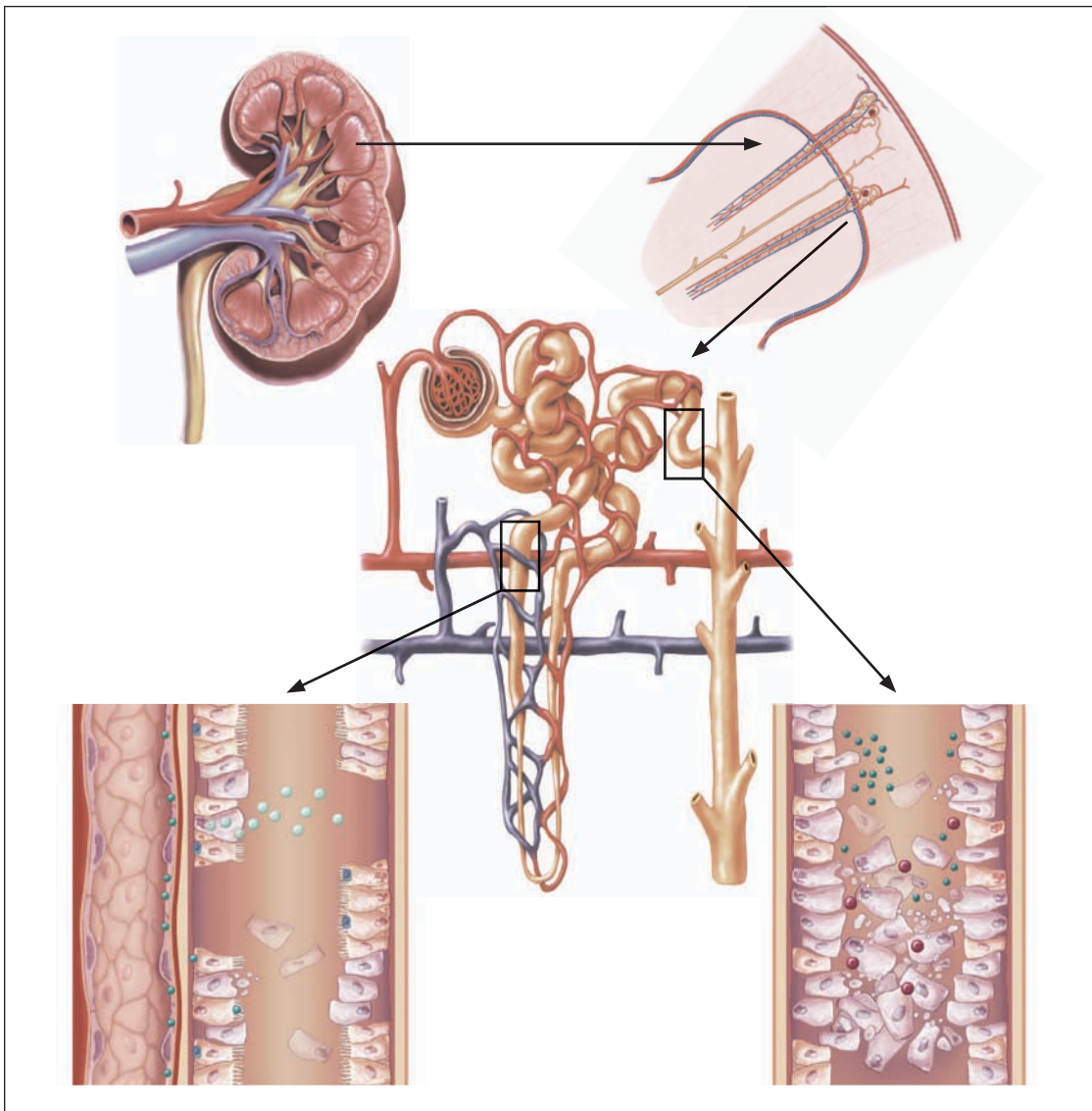


Figure 1. Local inflammation, direct toxicity to tubular cells, and hypoperfusion lead to vasoconstriction, loss of endothelial cells, and accumulation of inflammatory byproducts. Untreated, the result is acute kidney injury with mechanical obstruction in the renal tubules and ultimately irreversible cellular destruction. Illustration by Anne Rains.

Table 1. AKIN and RIFLE Criteria for Defining Acute Kidney Injury^{1,2}

AKIN Criteria	AKIN Stage	RIFLE Criteria	RIFLE Class
Serum creatinine: an increase of ≥ 0.3 mg/dL or to $\geq 150\%$ to 200% (≥ 1.5 - to- 2 -fold) above baseline, OR Urine output: < 0.5 mL/kg/hr for > 6 hr	1	Creatinine: an increase 1.5 times above baseline, OR GFR: a decrease $> 25\%$ from baseline, OR Urine output: < 0.5 mL/kg/hr for 6 hr	Risk
Serum creatinine: an increase to $> 200\%$ to 300% (> 2 - to- 3 -fold) above baseline, OR Urine output: < 0.5 mL/kg/hr for > 12 hr	2	Creatinine: an increase 2 times above baseline, OR GFR: a decrease $> 50\%$ from baseline, OR Urine output: < 0.5 mL/kg/hr for 12 hr	Injury
Serum creatinine: an increase to $> 300\%$ (> 3 -fold) above baseline or of ≥ 4 mg/dL with an acute increase of at least 0.5 mg/dL, OR Urine output: < 0.3 mL/kg/hr for 24 hr, or anuria for 12 hr	3	Creatinine: an increase 3 times above baseline, OR GFR: a decrease $> 75\%$ from baseline, OR Urine output: < 0.3 mL/kg/hr for 24 hr, or anuria for 12 hr	Failure
N/A	N/A	Persistent ARF: complete loss of renal function for > 4 wk	Loss
N/A	N/A	ESRD	ESRD

AKIN = Acute Kidney Injury Network; ARF = acute renal failure; ESRD = end-stage renal disease; GFR = glomerular filtration rate; N/A = not applicable; RIFLE = risk, injury, failure, loss, end-stage renal disease.

There are three major mechanisms of injury at work in acute kidney injury:

- hypoperfusion
- direct tissue injury
- hypersensitivity reactions that cause renal inflammation

A number of conditions, treatments, and events may trigger these mechanisms, putting patients at risk.

HYPOPERFUSION

Hypoperfusion from any cause, including hypovolemia (from blood loss, dehydration, or burns), vasoconstriction, and hypotension or shock, alters blood flow within the kidney, impeding function and such protective renal feedback mechanisms as autoregulation of GFR and blood flow. Risk factors include sepsis; cardiac surgery; hip fracture; and treatment with vasopressors, ACE inhibitors, or angiotensin receptor blockers (ARBs).

Hypotension and shock. Sustained hypotension occurs secondary to such factors as hemorrhagic, hypovolemic, cardiogenic, or septic shock. In all types of shock, oxygen and nutrient delivery to the tissues is compromised as cellular metabolic demands cannot be met. Following resuscitation efforts, dysfunction may be evident in multiple vital organs, including the kidney.¹³

Hypovolemia may be secondary to bleeding, burns, dehydration, or surgery in which fluid is lost. Shock may result not only from actual loss of body fluid, but also from relative hypovolemia secondary to vasodilation or from the loss of intravascular fluid into the interstitial tissues as occurs with inflammation. During hypovolemic shock, urine output is reduced to conserve water and sodium, and the sympathetic nervous system stimulates vasoconstriction to maintain homeostasis. Initially, the microcirculatory response (the constriction of arterioles, capillaries, and venules) is protective, but eventually tissue acidosis becomes severe and arteriolar smooth muscle begins to fail. In addition, impaired oxygen delivery to the tissues causes hypoxic vasodilation and the release of inflammation-inducing chemicals. If the shock syndrome progresses, the availability of oxygen and nutrients diminishes as inflammatory byproducts accumulate, ultimately leading to irreversible cellular destruction. Shock-related changes can be persistent for long periods of time, even after aggressive fluid resuscitation.¹³

Sepsis often precipitates acute kidney injury, especially in patients who are critically ill. Of the 270 patients with sepsis identified in a study by Shema and colleagues, nearly 52% developed acute kidney injury, compared with 5.5% of those who did not have sepsis.⁹

The sequence of kidney tissue destruction during the septic process has not been clearly defined, but it's known that microvascular perfusion is altered, impairing oxygen delivery to the tissues and contributing to cellular ischemia.¹⁴ With early identification, the vast majority of causative infections can be treated before sepsis develops.^{15,16}

Cardiac surgery. Renal hypoperfusion is thought to be a major factor in acute kidney injury related to cardiac surgery—both during and after surgery.^{17,18} Reported incidence of acute kidney injury with cardiac surgery varies with the defining criteria.

Using RIFLE criteria to categorize postsurgical kidney injury in 3,460 patients who underwent cardiac surgery in one of seven Canadian hospitals, researchers identified 24% of patients as being at risk, 7% as having kidney injury, and 3% as experiencing renal failure.¹⁹ A U.S. study that used RIFLE criteria to retrospectively categorize 1,358 patients who underwent cardiac surgery at two New York hospitals found that 40% of patients developed postoperative acute kidney injury.¹⁷

An Italian study that defined acute kidney injury as a 50% reduction in GFR analyzed 536 patients undergoing coronary artery bypass grafting and found that 49 (9%) developed postoperative acute kidney injury, including 23 (4%) who required dialysis.²⁰

The following have been identified as important independent risk factors for kidney failure following cardiac surgery:

- advanced age^{17,21}
- valve replacement or reconstructive surgery²¹
- combined valve and coronary artery bypass grafting^{17,21}
- longer duration of cardiopulmonary bypass²¹
- longer duration of aortic cross-clamp time²¹
- preoperative congestive heart failure, diabetes, or neurologic disease¹⁷

Hip fracture. In patients with traumatic hip injury, hypoperfusion may arise from hypovolemia (owing to hemorrhage or dehydration) as well as from the physiologic response to trauma and immobility. In a study of 90 patients over age 65 who were undergoing surgery for a femoral fracture, 24% developed acute kidney injury as defined by RIFLE criteria.²² In a study by Bennet and colleagues of 170 patients with hip fracture, 16% developed acute renal dysfunction, also using RIFLE criteria.²³ In a study by White and colleagues of 1,511 patients undergoing surgery for hip fracture, 69 of 730 patients (9%) who had normal renal function before surgery developed renal dysfunction after surgery (using reduced GFR to define dysfunction).²⁴ In this same study, only 364 of the 545 patients (67%) who had a reduced GFR upon admission had evidence of postoperative renal dysfunction. The study authors suggest that identifying preoperative abnormal kidney function can improve intraoperative fluid resuscitation in patients with

hypotension and hypovolemia secondary to hemorrhage or rhabdomyolysis.

For those who develop acute kidney injury following hip fracture, mortality rates are high. In the study by Bennet and colleagues, inpatient mortality in patients with postoperative renal dysfunction was 19%, compared with 0% in patients without.²³ Within 120 days following surgery, 41% of those with renal dysfunction had died, compared with 13% of those whose renal function was normal.²³

Perfusion-altering drugs. Treatment with vasopressors, ACE inhibitors, ARBs, or nonsteroidal anti-inflammatory drugs (NSAIDs) may, under certain conditions, impair renal perfusion.

Vasopressors, such as dopamine and norepinephrine, which are used in critical situations to maintain blood pressure, may alter blood flow to the kidneys by causing vasoconstriction. The risk of acute kidney injury increases when a patient receiving vasopressors receives inadequate fluid resuscitation.²⁵

At least initially, the kidney is somewhat protected by the renin–angiotensin–aldosterone system, which maintains intraglomerular pressure. Afferent arterioles that branch off the renal artery dilate to increase blood flow to the glomerulus, and efferent arterioles constrict. Some medications, however, may interfere with the renin–angiotensin system, including ACE inhibitors and ARBs, both of which are widely used to treat hypertension, especially in the presence of comorbid congestive heart failure.^{17,23,25}

The risk of acute kidney injury increases when a patient receiving vasopressors receives inadequate fluid resuscitation.

Two studies that sought to measure the impact of these drugs on kidney function in preoperative cardiac surgery patients yielded opposite results. In the first, a study of 536 patients who underwent coronary artery bypass grafting, 281 patients had been taking preoperative ACE inhibitors; of these, 6% developed an acute kidney injury (defined in this study as either postoperative renal failure requiring dialysis or a 50% or greater postoperative drop from baseline in GFR), compared with 12% of those who hadn't been receiving these drugs. The authors concluded that ACE inhibitors may have provided some kidney protection.²⁰ Although this study did not use RIFLE criteria, the change in GFR used to define acute kidney injury was consistent with that in the “injury” category in the RIFLE criteria; patients who fell into the “risk” category were not considered to have an acute kidney injury.

Conversely, the authors of a retrospective cohort study of 1,358 patients who underwent cardiac surgery found that the 52% of patients who were taking preoperative ACE inhibitors or ARBs were 28% more likely to develop postoperative acute kidney injury, as defined by the RIFLE criteria, than those who were not receiving these drugs.¹⁷ The authors recommend that both drugs be stopped before cardiac surgery. The conflicting results in these two studies illustrate the difficulties that emerge when attempting to compare studies in which different criteria were used to define acute kidney injury.

NSAIDs decrease prostaglandin-induced inflammation by inhibiting the cyclooxygenase (COX) enzymes COX-1 and COX-2 and thereby blocking prostaglandin synthesis. Unfortunately, in doing so, NSAIDs interfere with the protective role of prostaglandins in the kidney—regulating the release of renin, salt, and water, as well as balancing glomerular vascular tone (vasoconstriction or vasodilation).²⁶ When renal perfusion is compromised owing to hypovolemia, dehydration, hypotension, or blood loss, NSAIDs may exacerbate the problem.

DIRECT TISSUE INJURY

A number of substances may directly injure renal tissue, including nephrotoxic drugs, contrast media, and various products of disease.

Nephrotoxic drugs. Some renally excreted drugs directly result in acute tubular necrosis (see Table 2^{17,23,25-29}). Drugs that fall into this category include amphotericin B (an antifungal medication), aminoglycosides (including neomycin, gentamicin, tobramycin, and amikacin), and vancomycin.^{25,27} If an aminoglycoside or vancomycin is used, drug levels should be evaluated regularly, as recommended in the prescribing information. Damage caused by nephrotoxic drugs is typically dose dependent. Patients most likely to experience drug-induced acute kidney injury are elderly and those with preexisting kidney insufficiency.

Contrast media used for radiologic exams is also a well-known cause of acute kidney injury.^{25,30,31} Although the mechanism of injury is not well understood, it appears to involve direct toxicity to the tubular cells and a hemodynamically mediated effect that results in renal ischemia. Adequate hydration is essential in preventing and treating associated kidney injury. Bicarbonate solutions may also be helpful in treatment.³⁰

Disease products. In diseases such as sepsis, various toxins are released, including inflammatory cytokines, tumor necrosis factor, vasoactive substances, thrombogenic agents, and other biologically active mediators that may directly injure kidney cells.³² Tumor necrosis factor is known to be directly toxic to the kidney and also may increase the rate of apoptosis, the genetically directed, natural process of

preprogrammed cellular death that allows new cells to replace older ones.

Similarly, rhabdomyolysis, a life-threatening condition in which skeletal muscle tissue breaks down and releases the oxygen-carrying muscle protein, myoglobin, into the blood, may directly injure kidney cells. Myoglobin normally binds oxygen in order to provide an extra supply to muscle cells during exercise. When skeletal muscle is damaged and excess myoglobin is released into the circulation, it has a toxic effect on kidney cells and also accumulates in renal tubules, causing mechanical obstruction. Acute kidney injury, sometimes leading to acute kidney failure, may follow.²⁸

Rhabdomyolysis may result from a number of factors, including traumatic muscle injury; heat-related hyperthermia; high-voltage electrical or extensive third-degree burns; pressure injuries related to immobility; toxins (such as ethylene glycol, snake venom, black widow spider venom, or carbon monoxide); and a wide variety of prescription, over-the-counter, and illicit drugs, as shown in Table 2. When muscle injury is combined with extracellular volume depletion or dehydration, risk of rhabdomyolysis is elevated. In young athletes, rhabdomyolysis may be triggered by vigorous exercise combined with dehydration. In older patients, it may result from impaired mobility and subsequent pressure injuries combined with dehydration that results from an inability to replenish fluids.²⁸

HYPERSENSITIVITY REACTIONS

When drug-induced hypersensitivity reactions cause renal inflammation (acute interstitial nephritis), kidney dysfunction occurs within seven to 14 days of drug administration. This type of acute kidney injury is usually reversible, but recovery may take several months and, in rare cases, dialysis may be necessary. A wide range of drugs can induce a hypersensitivity reaction, as shown in Table 2. In the event that a patient has a hypersensitivity reaction (such as rash, hives, eosinophilia, or fever) to a medication or any substance, discontinue the drug or end patient contact with the antigen immediately and monitor the patient closely for any signs of acute allergic interstitial nephritis, which may not be evident for seven to 10 days after the initial reaction.

INCIDENCE AND POTENTIAL FOR RECOVERY

It's difficult to gauge the incidence of acute kidney injury in various patient populations because defining criteria differ among studies. Published incidence rates range from 22% to 57% in U.S. ICUs; from 34% to 36% in British, German, and Chinese ICUs; are lower than 10% among noncardiac surgical patients in a Portuguese postanesthesia care unit (PACU); and are 5% or lower among hospitalized patients in Israel (Table 3⁴⁻¹¹).⁴⁻¹¹

Table 2. Potentially Harmful Drugs in Patients at Risk for Acute Kidney Injury

Drug or Class	Mechanism of Injury
Amphotericin B ²⁵ Aminoglycosides, such as gentamicin, tobramycin, amikacin ²⁵ Vancomycin ^{25,27}	Direct tissue injury
NSAIDs, such as aspirin, ibuprofen, ketolorac, celecoxib ^{25,26}	Hypersensitivity inflammatory reaction and hemodynamically mediated injury
Antibiotics, such as penicillins, cephalosporins, rifampin, sulfonamides, ciprofloxacin ²⁵ Diuretics, including furosemide, bumetanide, thiazide-type diuretics ²⁵ 5-Aminosalicylates, such as mesalamine, sulfasalazine, basalazide, olsalazine ²⁵ Allopurinol ²⁵ Proton pump inhibitors, such as omeprazole, lansoprazole ²⁵ H ₂ -antagonists, such as cimetidine ²⁵ Protease inhibitors, such as indinavir ²⁵	Hypersensitivity inflammatory reaction
Vasopressors, such as dopamine, norepinephrine ²⁵ ACE inhibitors, such as benazepril, captopril, enalapril, fosinopril, lisinopril, quinapril, ramipril ^{17,23,25} ARBs, such as candesartan, irbesartan, valsartan, losartan ^{17,23,25}	Hemodynamically mediated injury
Anesthesia (because of related malignant hyperthermia) ²⁸ Antipsychotics, such as chlorpromazine (because of neuroleptic malignant syndrome) ²⁸ Cyclic antidepressants, such as amitriptyline, doxepin, imipramine, nortriptyline ²⁸ SSRI antidepressants, such as citalopram, sertraline, fluoxetine, paroxetine, escitalopram ²⁸ Corticosteroids, such as prednisone ²⁸ Fibric acid derivatives, such as bezafibrate, fenofibrate, gemfibrozil ²⁸ Statins, such as atorvastatin, fluvastatin, lovastatin, pravastatin, rosuvastatin, simvastatin ²⁸ Illicit drugs, including heroin, methadone, barbiturates, cocaine, amphetamines, Ecstasy, LSD, PCP, toluene (from glue sniffing) ²⁸ Synthetic amphetamine "party drugs" such as BZP, known as Nemesis and Herbal Ecstasy; MDMA, known as Ecstasy ²⁹	Rhabdomyolysis

ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocker; BZP = N-benzylpiperazine; LSD = lysergic acid diethylamide; MDMA = 3,4-methylenedioxymethamphetamine; NSAID = nonsteroidal antiinflammatory drug; PCP = phencyclidine; SSRI = selective serotonin reuptake inhibitor.

Following an acute kidney injury, full recovery of kidney function is not only possible, but probable. Of the 8,279 patients who developed an acute kidney injury while hospitalized in 191 U.S. Department of Veterans Affairs (VA) ICUs, 56.5% recovered full kidney function, including 66% of those with AKIN stage 1 disease and 30% of those with AKIN stage 2 disease.¹⁰ Among those whose

disease progressed to AKIN stage 3, only 7% fully recovered.

It's important to identify patients at risk for acute kidney injury and, if possible, intervene before injury occurs. Acute kidney injury is an independent risk factor for both prolonged length of hospital stay and in-hospital mortality.³³ In U.S. hospitals, in-hospital mortality among affected patients ranges from 13.8%

Table 3. Incidence of Acute Kidney Injury and Associated Mortality in Various Patient Populations

Patient Populations	Sample Size	Incidence, %	In-Hospital Mortality, % ^a
United States			
Patients in seven ICUs at a large U.S. medical center ⁷	14,524	57 ^b	16 vs. 6.7
ICU admissions in the MIMIC II database ^{6,c}	16,728	31 ^b	22 vs. 7
Patients with AMI in 56 U.S. medical centers ⁵	31,532	22.5; 26.6 in 2000; 19.7 in 2008 ^d	19.9 in 2000; 13.8 in 2008 (without AKI, NR)
Patients in 191 ICUs at U.S. Department of Veterans Affairs medical centers ¹⁰	325,395	22 ^b	NR
International			
Admissions to 22 ICUs in the United Kingdom and Germany ⁸	41,972	35.8 ^e	20.9, risk; 45.6, injury; 56.8, failure; 8.4, without AKI
Patients in five Chinese ICUs ¹¹	1,036	34.1 ^b	54.4 (without AKI, NR)
Noncardiothoracic surgical patients in a Portuguese PACU ⁴	1,200	9.6 ^b	26 vs. 3
Admissions to a large hospital in northern Israel ⁹	34,802	1–5.1 ^f	21.8 vs. 1.6

AKI = acute kidney injury; AMI = acute myocardial infarction; MIMIC II = Multiparameter Intelligent Monitoring in Intensive Care; NR = not reported; PACU = postanesthesia care unit.

^a In-hospital mortality is expressed as percent with AKI vs. percent without AKI unless otherwise indicated.

^b AKI was defined by Acute Kidney Injury Network criteria.

^c The MIMIC II database version 2.4 (a database of ICU admissions at Boston's Beth Israel Deaconess Medical Center) is available at <http://mimic.physionet.org>. In this study, researchers selected admissions that included at least two serum creatinine values.

^d AKI was defined as an absolute increase in serum creatinine of ≥ 0.3 mg/dL or a relative increase of $\geq 50\%$ during hospitalization.

^e AKI was defined by risk, injury, failure, loss, end-stage renal disease (RIFLE) criteria.

^f Using three sets of diagnostic criteria—*International Classification of Diseases, Ninth Revision (ICD-9)*, RIFLE, and an increase in serum creatinine of at least 0.5 mg/dL over baseline—AKI incidence was found to be 1%, 3.8%, and 5.1%, respectively.

to 22%, compared with from 6.7% to 7% among patients without acute kidney injury, as shown in Table 3.⁵⁻⁷ Recent analysis reveals that the incidence of acute kidney injury requiring dialysis rose rapidly over the past decade in the United States, and associated death more than doubled.³⁴

After hospital discharge, patients who developed acute kidney injury while hospitalized continue to be at elevated risk for death. Of 114 Portuguese PACU patients who developed an acute kidney injury and survived to discharge, 38% died within six months, compared with 10% of those who had not developed acute kidney injury.⁴

TREATING AN EVOLVING ACUTE KIDNEY INJURY

If a patient develops an acute kidney injury, the goal is to identify and, if possible, correct the underlying causes. For injury related to volume depletion, sepsis, or rhabdomyolysis, iv fluid administration is crucial in the early stages. For rhabdomyolysis, 10 to 15 mL/kg per hour (700 to 1,050 mL per hour for a 70-kg

person) may be needed to keep the kidney tubules clear of myoglobin.³⁵

Bear in mind that excessive fluid administration can also result in vital organ failure.^{35,36} Patients who receive excessive fluid administration during resuscitative efforts are less likely to survive than those who receive the appropriate amount of fluid. The heart and lungs are particularly susceptible to the adverse effects of excessive fluid loading, and adult respiratory distress syndrome secondary to increased interstitial pulmonary edema is a life-threatening complication.³⁶ When receiving fluid by iv bolus, patients' cardiopulmonary status can change very quickly. Such patients should be assessed every 15 to 30 minutes for signs and symptoms of fluid overload. Since few medical-surgical nursing units have staffing sufficient for adequate assessment, these patients should be cared for in a critical care unit with hemodynamic monitoring in place.

If nephrotoxic drugs are suspected, immediate discontinuation is necessary. If the cause is heart

failure, it's vital to treat the cardiac issues. It may be necessary to administer vasoactive drugs, such as norepinephrine, to maintain an adequate mean arterial pressure (MAP).³⁵

Badin and colleagues used the RIFLE definition to evaluate the relationship between MAP and development of acute kidney injury in 217 patients in early shock.³⁷ Hypotension was defined as a systolic arterial pressure below 90 mmHg or a MAP of less than 65 mmHg for more than 10 minutes. Acute kidney injury occurred frequently for all types of shock studied: for septic shock, 63 of 127 patients (50%); for hypovolemic shock, 17 of 42 patients (40%); for hemorrhagic shock, five of nine patients (56%); for cardiogenic shock, 8 of 18 patients (44%); and following cardiac arrest, 6 of 15 patients (40%). In septic shock, the authors found that maintaining a MAP between 72 and 82 mmHg helped to reduce the incidence of secondary acute kidney injury, although this was not the case for the other types of shock studied.

A retrospective study of 16,728 adults in ICUs used electronic data records to evaluate the relationship between acute kidney injury and MAP, with hypotension defined as a MAP of less than or equal to 80 mmHg.⁶ Using AKIN criteria, acute kidney injury was evident in 31% of records. Among patients with prolonged hypotension, 34% had an acute kidney injury. The lower the MAP, the greater the risk; for each 1 mmHg below 80, the risk of acute kidney injury increased by 3%. Duration of hypotension also played a role: for each hour of hypotension, the risk of kidney injury more than doubled with each 10 mmHg drop in MAP below 80.

NURSING INTERVENTIONS

Nurses can have a dramatic impact on both the avoidance and outcome of acute kidney injury by performing comprehensive nursing assessments, educating others on prevention, identifying early signs and symptoms of acute kidney injury, intervening when necessary, and advocating for their patients. The nursing interventions below may enable your patients to avoid acute kidney injury.

- Know the risk factors and diagnostic criteria for acute kidney injury.
- Perform a thorough nursing assessment on every patient admission (see *Assessing Patients' Risk of Acute Kidney Injury*).
- Talk to your patients about their medical and medication history, so you're able to identify factors that put them at risk.
- Teach patients about risk factors for acute kidney injury, including nephrotoxic medications, and emphasize that they should report any change in urination, urine appearance, and urine output after starting any new medication.
- Caution patients using NSAIDs to be mindful of potential adverse effects, such as hypersensitivity

and hemodynamically mediated acute kidney injury, and to drink plenty of water when taking these medications.

- Instruct patients to discontinue any medication to which they have a hypersensitivity reaction (such as rash, hives, facial swelling, or angioedema) and promptly notify the prescriber.
- A repeat serum creatinine level should be obtained seven to 10 days after any hypersensitivity reaction to assess for signs or symptoms of evolving acute interstitial nephritis, especially if the patient is taking a potentially nephrotoxic drug.
- Evaluate the patients' serum creatinine level before administering any potentially nephrotoxic drugs.
- Immediately communicate all information regarding risk of acute kidney injury to the primary care provider, especially if surgery is imminent.
- Teach patients—before surgery, after surgery, and at discharge—to report any reduction in urine output or voiding frequency, especially if they have any kidney-related risk factors because of surgery, injury, medications, or other factors.
- Clean all equipment used between patients, practice scrupulous hand hygiene, and instruct patients on infection prevention, because sepsis is a common risk factor for acute kidney injury.
- Following high-risk surgeries, use a catheter drainage bag with an hourly urine meter and report urine outputs of 0.5 mL/kg per hour or less.
- In postoperative patients, if urine output is less than 0.5 mL/kg per hour, assess vital signs and urine output every 15 to 30 minutes until vital signs are stable or the patient is transferred to a higher level of care.
- In postoperative patients, report any hypotension or tachycardia.
- Prioritize IV fluid administration for patients in the initial stages of an acute kidney injury.

FOLLOW-UP WITH MS. JANSEN

Upon Ms. Jansen's admission to the postoperative nursing unit, her nurse took a thorough history and recognized that the patient was at risk for acute kidney injury for several reasons. First, the ACE inhibitor lisinopril she was taking for her high blood pressure put her at risk for hypoperfusion secondary to a disruption of the renin-angiotensin-aldosterone system. Second, she was dehydrated from having been unable to drink fluids all day and having taken the diuretic furosemide that morning. She was also still seriously hypovolemic from blood loss, with her hemoglobin level remaining at 7.5 g/dL. Her large, pressure-related deep tissue injury, in conjunction with the earlier 12 hours of immobility, increased Ms. Jansen's risk of rhabdomyolysis, and the faint reddish color of her urine suggested that myoglobin was present. The nurse suggested

Assessing Patients' Risk of Acute Kidney Injury

- ⇒ Obtain a thorough medical and medication history.
- ⇒ Identify patients with known risks for acute kidney injury:
 - sepsis
 - blood loss
 - hypotension or shock
 - recent or current nephrotoxic drug use
 - hypersensitivity reaction
 - muscle damage or rhabdomyolysis
 - recent cardiac surgery
 - recent hip fracture
- ⇒ Assess:
 - neurologic status
 - vital signs
- ⇒ Increase frequency of vital sign assessment (as appropriate for the setting), based on nursing assessment findings and patient risk factors.^a
- ⇒ Evaluate serum creatinine on admission (baseline) and over time (request laboratory orders if not already available):
 - Acute Kidney Injury Network (AKIN) Stage 1: an increase of ≥ 0.3 mg/dL or to $\geq 150\%$ to 200% above baseline
 - AKIN Stage 2: an increase to $> 200\%$ to 300% above baseline
 - AKIN Stage 3: an increase to $> 300\%$ above baseline or of ≥ 4 mg/dL
- ⇒ Monitor intake and output.
- ⇒ Determine if urine output is adequate (at least 0.5 mL/kg/hr):
 - 150 lbs. = 34 mL/hr
 - 200 lbs. = 45 mL/hr
 - 250 lbs. = 57 mL/hr
 - 300 lbs. = 68 mL/hr
- ⇒ Assess urine output hourly for patients at high risk, using a urine meter for indwelling catheters:
 - AKIN Stage 1: < 0.5 mL/kg/hr for more than six hours
 - AKIN Stage 2: < 0.5 mL/kg/hr for more than 12 hours
 - AKIN Stage 3: < 0.3 mL/kg/hr for 24 hours, or anuria for 12 hours
- ⇒ During fluid resuscitation, assess patients at least hourly for signs and symptoms of fluid volume overload, including pulmonary crackles and periorbital or peripheral edema.
- ⇒ Apprise the nurse manager or nursing supervisor of the severity of the patient's condition to ensure that the level of care and observation is appropriate.^b

^a For patients at high risk in the immediate postoperative phase, assessment may be every 15 minutes using automated vital sign equipment. For home health patients, assessment may be one to three times a week.

^b Patients with new-onset, evolving acute kidney injury who require aggressive fluid resuscitation (those with hypotension, low urine output, and rising creatinine, for example) are best treated in a critical care environment where staffing is sufficient for the intensity of care required. For patients with end-stage renal disease or terminal comorbid conditions, however, such aggressive care may not be appropriate.

obtaining a urine myoglobin test, and the results confirmed that Ms. Jansen had rhabdomyolysis. The nurse apprised the orthopedic surgeon, anesthesiologist, and hospitalist of Ms. Jansen's risk factors and alerted them that myoglobin was present in her urine.

As soon as Ms. Jansen arrived in her postoperative room, the nurse performed a complete assessment, noting that Ms. Jansen's blood pressure was low at 90/58 mmHg. Based on the assessment findings, the nurse

- placed an automated blood pressure cuff, setting it to take measurements every 15 minutes and to sound an alarm at a systolic pressure of 98 mmHg or less or a heart rate of 100 beats per minute or higher.
- asked the patient care technician to empty the urine bag (which contained 2,500 mL upon Ms. Jansen's arrival from the PACU), replaced the regular bag with a urine meter, and instructed

the technician to measure and record output hourly.

- checked on Ms. Jansen every half hour to ensure that she was awake, alert, and oriented to person, place, and time.
- requested physician orders for a postoperative basic metabolic panel to be run immediately.

An hour after surgery, Ms. Jansen's blood pressure was dropping and her urine meter contained only 20 mL. Since she weighed 165 lbs., her hourly urine output should have been 37.5 mL. The nurse reviewed the laboratory results, noting that Ms. Jansen's serum creatinine had increased by 0.4 mg/dL over her preoperative level. Knowing that long durations of low urine output are associated with poor outcomes, the nurse immediately notified the surgeon and hospitalist and administered the prescribed fluid boluses without delay. After the second hour of low urine output, the nurse requested that Ms. Jansen be transferred to the ICU, where staffing would permit closer

observation. In the ICU, Ms. Jansen received another two units of blood and additional fluid boluses until her blood pressure, pulse rate, and central venous pressure stabilized.

Although Ms. Jansen's multiple risk factors caused her to develop acute kidney injury, timely, appropriate nursing and medical care prevented progression to chronic kidney failure. The fact that Ms. Jansen was a fairly healthy and active 62-year-old prior to the fall contributed to her positive outcome. Ms. Jansen required dialysis for three weeks before her renal function returned to the point that dialysis was no longer necessary. The acute kidney injury left her with mild, residual kidney insufficiency, but she was able to resume all of her previous activities after several months of recovery. ▼

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REFERENCES

- Mehta RL, et al. Acute Kidney Injury Network: report of an initiative to improve outcomes in acute kidney injury. *Crit Care* 2007;11(2):R31.
- Acute Dialysis Quality Initiative. Acute dialysis quality initiative: a process towards developing consensus and guidelines for diagnosis, prevention and treatment of acute renal failure in the critically ill. *ADQI Newsletter* 2002 Summer-Fall. http://www.adqi.net/ADQI_newsletter%20_fall_2002.pdf.
- Abelha FJ, et al. Determinants of postoperative acute kidney injury. *Crit Care* 2009;13(3):R79.
- Abelha FJ, et al. Outcome and quality of life of patients with acute kidney injury after major surgery. *Nefrologia* 2009;29(5):404-14.
- Amin AP, et al. Trends in the incidence of acute kidney injury in patients hospitalized with acute myocardial infarction. *Arch Intern Med* 2012;172(3):246-53.
- Lehman LW, et al. Hypotension as a risk factor for acute kidney injury in ICU patients. *Comput Cardiol (2010)* 2010;37:1095-8.
- Mandelbaum T, et al. Outcome of critically ill patients with acute kidney injury using the Acute Kidney Injury Network criteria. *Crit Care Med* 2011;39(12):2659-64.
- Ostermann M, Chang RW. Acute kidney injury in the intensive care unit according to RIFLE. *Crit Care Med* 2007;35(8):1837-43.
- Shema L, et al. Hospital-acquired acute kidney injury in Israel. *Isr Med Assoc J* 2009;11(5):269-74.
- Thakar CV, et al. Incidence and outcomes of acute kidney injury in intensive care units: a Veterans Administration study. *Crit Care Med* 2009;37(9):2552-8.
- Zhou J, et al. Risk factors for the prognosis of acute kidney injury under the Acute Kidney Injury Network definition: a retrospective, multicenter study in critically ill patients. *Nephrology (Carlton)* 2012;17(4):330-7.
- National Kidney Foundation. Kidney disease outcomes quality initiative (KDOQI) clinical practice guidelines for chronic kidney disease: part 4—definition and classification of stages of chronic kidney disease. Guideline 1: definition and stages of chronic kidney disease. New York City; National Kidney Foundation; 2002. KDOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification; http://www.kidney.org/professionals/KDOQI/guidelines_ckd/p4_class_g1.htm.
- Szopinski J, et al. Microcirculatory responses to hypovolemic shock. *J Trauma* 2011;71(6):1779-88.
- Doerschug KC, et al. Impairments in microvascular reactivity are related to organ failure in human sepsis. *Am J Physiol Heart Circ Physiol* 2007;293(2):H1065-H1071.
- Daniels KR, Frei CR. The United States' progress toward eliminating catheter-related bloodstream infections: incidence, mortality, and hospital length of stay from 1996 to 2008. *Am J Infect Control* 2013;41(2):118-21.
- Umscheid CA, et al. Estimating the proportion of healthcare-associated infections that are reasonably preventable and the related mortality and costs. *Infect Control Hosp Epidemiol* 2011;32(2):101-14.
- Arora P, et al. Preoperative use of angiotensin-converting enzyme inhibitors/angiotensin receptor blockers is associated with increased risk for acute kidney injury after cardiovascular surgery. *Clin J Am Soc Nephrol* 2008;3(5):1266-73.
- Elahi M, et al. Acute kidney injury following cardiac surgery: impact of early versus late haemofiltration on morbidity and mortality. *Eur J Cardiothorac Surg* 2009;35(5):854-63.
- Karkouti K, et al. Acute kidney injury after cardiac surgery: focus on modifiable risk factors. *Circulation* 2009;119(4):495-502.
- Benedetto U, et al. Preoperative angiotensin-converting enzyme inhibitors and acute kidney injury after coronary artery bypass grafting. *Ann Thorac Surg* 2008;86(4):1160-5.
- Sirvinkas E, et al. Cardiopulmonary bypass management and acute renal failure: risk factors and prognosis. *Perfusion* 2008;23(6):323-7.
- Azevedo VL, et al. Postoperative renal function evaluation, through RIFLE criteria, of elderly patients who underwent femur fracture surgery under spinal anesthesia. *Ren Fail* 2008;30(5):485-90.
- Bennet SJ, et al. Acute renal dysfunction following hip fracture. *Injury* 2010;41(4):335-8.
- White SM, et al. An analysis of renal dysfunction in 1511 patients with fractured neck of femur: the implications for peri-operative analgesia. *Anaesthesia* 2009;64(10):1061-5.
- Pannu N, Nadim MK. An overview of drug-induced acute kidney injury. *Crit Care Med* 2008;36(4 Suppl):S216-S223.
- Musu M, et al. Acute nephrotoxicity of NSAID from the foetus to the adult. *Eur Rev Med Pharmacol Sci* 2011;15(12):1461-72.
- Elyasi S, et al. Vancomycin-induced nephrotoxicity: mechanism, incidence, risk factors and special populations. A literature review. *Eur J Clin Pharmacol* 2012;68(9):1243-55.
- Khan FY. Rhabdomyolysis: a review of the literature. *Neth J Med* 2009;67(9):272-83.
- Berney-Meyer L, et al. Nephrotoxicity of recreational party drugs. *Nephrology (Carlton)* 2012;17(2):99-103.
- Roche AM, James MF. Colloids and crystalloids: does it matter to the kidney? *Curr Opin Crit Care* 2009;15(6):520-4.
- Venkataraman R. Can we prevent acute kidney injury? *Crit Care Med* 2008;36(4 Suppl):S166-S171.
- Wan L, et al. Pathophysiology of septic acute kidney injury: what do we really know? *Crit Care Med* 2008;36(4 Suppl):S198-S203.
- Barrantes F, et al. Acute kidney injury predicts outcomes of non-critically ill patients. *Mayo Clin Proc* 2009;84(5):410-6.
- Hsu RK, et al. Temporal changes in incidence of dialysis-requiring AKI. *J Am Soc Nephrol* 2013;24(1):37-42.
- Joslin J, Ostermann M. Care of the critically ill emergency department patient with acute kidney injury. *Emerg Med Int* 2012;2012:760623.
- Schrier RW. Fluid administration in critically ill patients with acute kidney injury. *Clin J Am Soc Nephrol* 2010;5(4):733-9.
- Badin J, et al. Relation between mean arterial pressure and renal function in the early phase of shock: a prospective, explorative cohort study. *Crit Care* 2011;15(3):R135.