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“Decreased urine output and acute kidney injury in the PACU”

Kara Beth Chenitz, MD [Fellow] and

Department of Internal Medicine, Nephrology Division, Hospital of the University of Pennsylvania, 3400 Spruce Street, 1 Founders Building, Philadelphia, PA 19104, Telephone: (215) 662-2638, Kara.Chenitz@uphs.upenn.edu

Meghan B. Lane-Fall, MD [Clinical Associate and Post-doctoral Research Fellow]

Department of Anesthesiology and Critical Care, Hospital of the University of Pennsylvania, 3400 Spruce Street, 680 Dulles Building, Philadelphia, PA 19104, Telephone: (215) 573-7399, Facsimile: (215) 662-7106, Meghan.Lane-Fall@uphs.upenn.edu

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Introduction

Decreased urine output and acute kidney injury (AKI) are among the most important complications that may develop in the post-anesthesia care unit (PACU). Patients with postoperative AKI include those who develop incident kidney disease as well as those with progression of chronic kidney disease. Irrespective of the chronicity of renal impairment, development of AKI is associated with poor patient outcomes. Development of AKI is associated with longer hospital stays¹ and increased in-hospital and overall mortality (25–90%)^{2–4} when compared to patients that do not develop AKI. Due to the deleterious effects of declining renal function, there is an urgent need to prevent the development of AKI. Should AKI develop in the postoperative period, it is equally important to recognize it early and begin appropriate management. We begin this review by defining decreased urine output, oliguria and AKI. We then discuss the epidemiology and risk factors for development of AKI, followed by recommendations for the AKI prevention. Next, we summarize the pathophysiology and differential diagnosis of the most common etiologies of decreased urine output and AKI in the PACU. We conclude by presenting diagnostic strategies and management considerations.

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Correspondence to: Meghan B. Lane-Fall.

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Definitions: Decreased Urine Output, Oliguria, and Acute Kidney Injury

Decreased urine output and oliguria

Historically, oliguria has been defined as urine excretion less than 400 mL/day⁵. AKIN and RIFLE classifications of acute kidney injury (see Figure 1) define oliguria in progressive stages: $<0.5 \text{ mL/kg/hr} \times 6 \text{ hours}$, $<0.5 \text{ mL/kg/hr} \times 12 \text{ hours}$, and $<0.3 \text{ mL/kg/hr} \times 24 \text{ hours}$ ^{6, 7}. Technically then, at least six hours of decreased urine output are required for designation of oliguria according to AKIN and RIFLE criteria. Studies evaluating the sensitivity and specificity of urine output as a diagnostic and prognostic measure have yielded mixed results^{8, 9}. Nonetheless, it remains general consensus that $<0.5 \text{ mL/kg/hr}$ defines decreased urine output. In the proper clinical setting therefore, urine output less than 0.5 mL/kg/hr of any duration should prompt consideration and evaluation for causes of oliguria or AKI.

Acute kidney injury

Prior to 2004, there was no consensus definition of acute kidney injury. There were greater than 35 definitions in the literature, making evidence-based judgments about prevention and therapy difficult¹⁰. Thus, in 2004 the Acute Dialysis Quality Initiative examined existing data, and where evidence was lacking, sought expert opinion to form a consensus definition of what was then termed acute renal failure (ARF) – the RIFLE classification⁶.

RIFLE (see Table 1) is an acronym that represents 3 measures of renal dysfunction – Risk of renal dysfunction, Injury to the kidney, Failure of kidney function – and two outcome measures – Loss of kidney function and End stage renal disease (ESRD). Loss of kidney function is defined as ARF requiring renal replacement therapy (RRT) for over 4 weeks, and ESRD is defined as requiring renal replacement therapy for over 3 months. The classification scheme includes separate criteria for changes in serum creatinine and urine output, allowing designation of the severity of AKI on the basis of either measure. Whichever criterion leads to the more severe classification is used⁶. In 2006 the RIFLE criteria were validated in a retrospective study of more than 20,000 inpatients. Approximately 20% of the study patients were found to have some degree of renal impairment and there was an almost linear increase in hospital mortality from normal renal function to failure¹⁰.

Despite the apparent success of the RIFLE criteria, new data suggested that smaller changes in serum creatinine than previously cited in the RIFLE criteria may be associated with adverse outcomes^{7, 11}. Thus, experts in the field convened in 2007 to form the Acute Kidney Injury Network (AKIN). ARF was changed to AKI to represent the entire spectrum of renal impairment. The RIFLE criteria were modified to further refine the definition of AKI. New and broader criteria to diagnose AKI were adopted in efforts to increase clinical awareness and intervention if indicated⁷. Additionally, GFR was eliminated as a criterion for renal impairment because GFR, as calculated by the Modification of Diet in Renal Disease (MDRD) equation, has only been validated when renal function is in steady state, not when renal function is in flux as it is in AKI⁸. These changes resulted in a new classification scheme known as the AKIN classification (see Figure 1).

AKIN stages 1, 2, and 3 replace RIFLE's Risk, Injury, and Failure, and patients who require RRT are automatically classified as stage 3. Also, AKIN delineates additional criteria to classify decreased urine output. Urinary tract obstruction must be excluded and "adequate" resuscitation must be attempted before applying the diagnostic criteria for decreased urine output. Thus, with this new modification, transient changes in urine output can be excluded.

In studies comparing AKIN with RIFLE, the two classifications generally provided similar diagnostic and prognostic value. Despite the modification to RIFLE, AKIN has not been found to improve sensitivity and predictive ability over RIFLE^{8, 12, 13}. Furthermore, in the AKIN scheme, the diagnosis of AKI must be made within a 48-hour timeframe. Thus, if the serum creatinine rises gradually, such as 0.1 mg/dL per day, a slowly progressive AKI would be misclassified^{8, 14}. Given the variability in designation of acute kidney injury in the RIFLE and AKIN classifications, efforts are ongoing to reconcile the two schemes and create a single classification universally applicable to the diagnosis of AKI⁸.

Epidemiology and risk factors for development of postoperative AKI

The risk of developing AKI ranges from 2–5% in hospitalized patients^{1, 15, 16} and 1–25% in acutely ill patients, depending on the population being studied and the classification used to define AKI⁶. Post-surgical patients may carry even higher risk of decline in renal function than a general inpatient population. Investigators have found a 5–10% risk of developing AKI in mixed surgical populations^{17–19}. Various factors have been implicated in the risk of postoperative AKI (see Table 1). In a single-center study, Abelha et al evaluated patients without chronic kidney disease who underwent non-cardiac surgery. They found significant univariate associations between AKI and the following risk factors: age, emergency and high-risk surgery (intraoperative, intrathoracic, suprainguinal vascular procedures), ischemic heart disease, congestive heart failure, higher American Society of Anesthesiologists (ASA) physical status, and higher Revised Cardiac Risk Index (RCRI) score¹⁷. In a different study, perioperative AKI risk factors were examined in the context of cardiothoracic surgery. In a multivariable logistic regression model, age, smoking status, baseline serum creatinine, and diabetes mellitus were found to be preoperative risk factors. Intraoperative risk factors included use of inotropes, erythrocyte transfusion, aortic cross clamp time, urine output while on cardiopulmonary bypass (CPB), furosemide administration during CPB, and the need for a new CPB pump run. Postoperative AKI risk factors were erythrocyte transfusion and use of vasoconstrictors, inotropes, diuretics and antiarrhythmic drugs¹⁹.

Causes of postoperative decreased urine output and acute kidney injury

There are myriad causes of decreased urine output and AKI in the post-operative setting. These causes may be divided into those germane to all or most postoperative settings and those unique to specific surgical settings. We consider these two categories in turn.

Causes of decreased urine output and AKI in a general postoperative population

Three broad categories of decreased urine output/AKI etiologies describe the location of the defect in relation to the urogenital system (see Table 2).

Pre-renal—Pre-renal causes of decreased urine output and AKI include those etiologies that decrease perfusion to the afferent arteriole of the glomerulus. In the post-operative patient, hypotension and hypovolemia are the two most important causes of decreased renal perfusion. Either of these phenomena can be absolute or relative.

With regard to hypotension, a mean arterial pressure (MAP) of 60–65 mmHg is usually sufficient to maintain renal perfusion in a patient without pre-existing cardiovascular disease²⁰. Lower perfusion pressures may manifest as decreased urine output. Transient hypotension is common in the PACU. In the post-operative patient, euvoletic hypotension may be related to neuraxial analgesia²¹ or sedation. Cardiac ischemia or pump failure should be considered as causes of hypotension, especially in patients with cardiovascular disease. Hypotension accompanied by tachycardia should prompt evaluation for hypovolemia,

adrenal insufficiency and anaphylaxis. Hypotension with bradycardia may indicate beta-blockade, heart block, high neuraxial blockade or prolonged hypoxemia.

Relative hypotension may occur when patients with pre-existing hypertension exhibit blood pressure values significantly lower than their usual blood pressure. In this setting, maintaining MAP within 20% of baseline should be sufficient to maintain renal perfusion – this is within the range of blood pressure dipping that occurs in most people during sleep²².

Hypovolemia may also decrease renal perfusion. Marked hypovolemia is often associated with hypotension, but it is possible to lose in excess of 15% of blood volume before hypotension manifests²³. Causes of absolute hypovolemia include insufficient replacement of surgical blood loss and dehydration. Dehydration, in turn, may be related to osmotic diuresis (e.g. hyperglycemia or mannitol administration), loss of ascites, and pre-operative losses such as those caused by vomiting or bowel preparation. Conversely, hypovolemia relative to the kidney may occur in euvolemic or hypervolemic states when renal perfusion is diminished. An important cause of relative hypovolemia in the perioperative setting is intra-abdominal hypertension (IAH). IAH occurs when intra-abdominal pressure increases from a normal value of 5–7 mmHg to values in excess of 12 mmHg. IAH results from intra-abdominal fluid accumulation and can occur in the setting of edema or intra-abdominal hemorrhage. The elevated abdominal pressure compromises renal preload and afterload, predisposing to renal dysfunction. If intra-abdominal pressure exceeds 20–25 mmHg, frank abdominal compartment syndrome may result, with cardiopulmonary compromise and multi-organ dysfunction²⁴.

Renal—Acute tubular necrosis (ATN) is the most important intra-renal cause of AKI in the perioperative setting. ATN, historically characterized by histopathological findings, is now largely a clinical diagnosis²⁵. Evidence of renal dysfunction in the appropriate setting is usually sufficient to establish a presumptive diagnosis of ATN. Renal ischemia can cause endothelial and epithelial cell dysfunction that becomes apparent once blood flow is restored. Both apoptotic and immune mechanisms are implicated in the renal dysfunction that follows ischemia and reperfusion²⁶. In the perioperative setting, ischemia and reperfusion may occur with peri-procedural systemic hypotension, in the setting of cardiopulmonary bypass²⁷, or after aortic cross-clamping for vascular repair²⁸ or control of hemorrhage. Exposure to nephrotoxins, such as iodinated radiocontrast administered for endovascular procedures, is also associated with ATN²⁰.

Acute interstitial nephritis (AIN) is less commonly associated with decreased urine output and acute kidney injury in the immediate postoperative period. Non-steroidal anti-inflammatory drugs (NSAIDs), numerous antibiotics, and diuretics are among the drugs reported to cause drug-induced AIN²⁹.

Post-renal—Post-renal causes of decreased urine output and AKI are those causing physical obstruction of the urinary system distal to the renal pelvis. Obstruction may be internal (e.g. blood clots, urinary catheter debris) or external to the urethra or urinary catheter (e.g. prostatic hypertrophy, urinary catheter kinking). Functional obstruction may occur with conditions impairing bladder emptying, such as neurogenic bladder³⁰, bladder spasm or narcotic-related urinary retention.

Procedure-specific causes of post-operative decreased urine output and AKI

In addition to the causes listed above, it is important to consider how the technical aspects of specific surgical procedures may predispose an at-risk patient to the development of decreased urine output and/or AKI (see Table 3).

Urologic surgery—Patients who undergo intra-abdominal urologic surgery such as cystectomy and diversion are at risk for urologic complications related to manipulation of the ureters or anastomosis of the ureters to a urinary reservoir or conduit. These complications are rare in the immediate post-operative period; a retrospective secondary data analysis of 6577 patients in the United States undergoing radical cystectomy showed a urinary complication rate of 2.92%³¹. A more recent large single center prospective observational study showed a genitourinary (GU) complication rate less than 7% in the 30 days following surgery that increased over time. The types of GU complications encountered included acute renal failure or worsening of pre-existing renal insufficiency, urinary infections, ureteral stenosis and hydronephrosis³². When evaluating the urine output of a patient after cystectomy, it is important to note urine excretion may occur via an abdominal urostomy or continent cutaneous diversion³³. A urethral catheter in this setting may be left as a pelvic drain but will not drain urine. Ultimately, the patient's genitourinary anatomy should be clarified with the surgical team.

General and gynecologic surgery—Any intra-abdominal procedure in the lower abdomen or pelvis presents a risk for ureteral injury. Placement of ureteral stents for intra-operative ureteral identification is common, but does not eliminate the risk of ureteral injury³⁴. In a recent single center study of more than 5000 colorectal surgery patients, ureteral injury was noted in 14 patients. Half of the patients had ureteral stents placed. In two of the 14 patients, the presenting symptoms were anuria (one patient) and acute renal failure (one patient) on post-operative day 1³⁵.

Cardiac surgery—Despite the increasing use of fast-track anesthesia and extubation in the operating room for patients receiving coronary artery bypass grafting, most cardiac surgery patients are admitted post-operatively to an intensive care unit for close monitoring³⁶. If post-operative cardiac surgery patients are encountered in the PACU, they may be at risk for AKI as a result of ischemia-reperfusion injury or complement activation by the cardiopulmonary bypass circuit²⁷.

Vascular surgery—Open abdominal aortic aneurysm repair may be associated with ureteral injury, as discussed above for general and gynecologic surgery. Ischemia-reperfusion and dislodgement of atheromatous plaques by aortic cross-clamping are also important factors in AKI following aortic aneurysm repair²⁸. In contrast, endovascular stenting of aortic aneurysms is associated with decreased risk of AKI²⁸, but intra-operative contrast load or renal artery occlusion by the aortic stent are unique mechanisms leading to kidney injury³⁷.

Prevention of AKI

As mentioned earlier, it is preferable to avoid development of acute kidney injury in the postoperative period. There are several types of interventions that have been studied for their potential to help avoid development of postoperative AKI: monitoring, fluid therapy, vasopressor therapy, avoidance of intra-abdominal hypertension, avoidance of contrast-induced nephropathy, and pharmacologic interventions. We consider each of these types of interventions in turn.

Monitoring

As discussed above, postoperative AKI may be related to perioperative renal hypoperfusion and ischemic injury as a consequence of hemodynamic instability resulting from hypovolemia, hypotension, or decreased cardiac output. As such, it is reasonable to consider whether aggressive hemodynamic monitoring is beneficial. However, a preferred method of

hemodynamic monitoring to prevent development of AKI has yet to be determined³⁸. A randomized control trial evaluated the use pulmonary artery catheters in high-risk surgical patients, and found no benefit in prevention of AKI³⁹. Nevertheless, use of these catheters remains commonplace when cardiac output is uncertain³⁸. Less invasive cardiac monitoring techniques such as pulse pressure variation and use of esophageal Doppler probes are increasingly used for perioperative hemodynamic management^{38, 40}, but it is not clear whether use of these methods protects against development of postoperative AKI.

Fluid therapy

The administration of fluid to restore intravascular volume is a mainstay of therapy in preventing AKI, though the optimal amount of fluid therapy is unclear. Lopes et al demonstrated that intraoperative fluid boluses titrated in accordance with the variation in arterial pulse pressure improves postoperative outcomes⁴¹. However, Bouchard et al demonstrated that fluid accumulation in critically ill patients with acute illness is associated with increased mortality, and is not associated with recovery of renal function⁴². Furthermore, liberal fluid administration in patients undergoing bowel surgery has been associated with increased cardiopulmonary and tissue healing complications, arguing for restricted fluid therapy in this context⁴³.

There is also controversy surrounding fluid choice in volume resuscitation. Colloids have been shown to more effectively increase cardiac filling⁴⁴, but an overall mortality benefit of colloids in comparison to crystalloids has not been demonstrated [28]. Hydroxyethyl starch, a synthetic colloid, has been shown in multiple studies – including several randomized control trials – to be nephrotoxic⁴⁵. Use of blood as a resuscitation fluid is also not without risk. As mentioned above, erythrocyte infusion was shown to be independently associated with the development of AKI in cardiothoracic surgery¹⁹.

Fluid therapy in the setting of cardiopulmonary bypass has also been studied. Haase et al proposed that one of the potential etiologies of CPB-associated AKI is urinary acidity leading to toxicity from reactive oxygen species and complement activation. To answer this question, this group conducted a pilot study determining whether urinary alkalization is renoprotective in patients undergoing CPB. The study concluded that load and continuous administration of sodium bicarbonate in patients undergoing CPB reduces the risk of AKI. This finding has yet to be validated in larger trials⁴⁶.

Vasopressor therapy—In efforts to reduce the risk of AKI, it is necessary to maintain adequate renal perfusion pressures. Once intravascular volume has been restored with fluid administration, if the patient continues to have arterial hypotension, administration of vasoconstricting medication is indicated. The mainstay of clinical practice is to keep the mean arterial pressure above 60–65 mmHg, but patients with renovascular disease or long standing hypertension may require higher arterial pressures to maintain renal perfusion²⁰.

Avoidance or treatment of intra-abdominal hypertension

Another cause of decreased renal perfusion is intra-abdominal hypertension, discussed above as one of the causes of decreased urine output and acute kidney injury. Several conditions can cause elevated abdominal pressures, such as blood or ascites accumulation in the abdomen, distended bowel, or operative abdominal closure with edematous bowel, in the setting of a non-compliant abdominal wall⁴⁷. Abdominal pressure can be measured through bladder catheter transduction. Once abdominal hypertension is identified, procedural intervention to decrease abdominal pressures, such as therapeutic paracentesis or exploratory laparotomy, may prevent acute kidney injury²⁰.

Avoidance of contrast-induced nephropathy

Intravenous fluids given before, during and after the administration of iodinated contrast has been shown to reduce the risk of contrast induced nephropathy (CIN)⁴⁸. Thus, fluid administration is recommended during procedures involving the use of intravenous iodinated contrast agents, such as endovascular procedures. However, the optimal type of fluid – sodium bicarbonate, normal saline, or other isotonic crystalloid solutions – remains controversial. In patients undergoing coronary angiography, intravenous sodium bicarbonate was shown to reduce the risk of CIN as compared to normal saline, but there was no improvement in hospital length of stay or mortality⁴⁹. As intravenous fluids are beneficial in the prevention of CIN, diuretics and volume depletion pose potential risk and should be avoided²⁰. Studies evaluating N-acetylcysteine in the prevention of CIN yielded mixed results⁴⁹.

Pharmacologic interventions

Both dopamine and atrial natriuretic peptide (ANP) initially showed promise in the prevention of AKI due to their vasoactive effects leading to increased renal blood flow. Although evidence suggests ANP may reduce the necessity for RRT, neither dopamine nor ANP was associated with improved mortality. Likewise fenoldopam, a selective dopamine receptor agonist, initially showed potential for renoprotective benefit. In large studies, however, fenoldopam was not shown to be beneficial in prevention of AKI^{50–52}.

In summary, prevention of post-operative AKI should include optimization of renal perfusion by managing hemodynamics, intravascular volume, and avoiding increases in intra-abdominal pressure. Administering fluids in the setting of radiocontrast exposure may prevent development of AKI. Although not explored here, it is also prudent to avoid administration of nephrotoxins such as non-steroidal anti-inflammatory drugs (NSAIDs), aminoglycosides and tacrolimus, when possible, in patients who have risk factors for development of postoperative AKI.

Presentation of decreased urine output and acute kidney injury in the PACU

As discussed earlier, the term “oliguria” refers to decreased urine output that has persisted for at least six to twenty-four hours⁶. We use the term “decreased urine output” here to describe the decreases in urine output that are likely to be seen in the PACU patient, whose average length of stay is insufficient to allow diagnosis of oliguria. Urine output of less than 30 mL/hr (roughly 0.5 mL / kg / hour for a 70-kilogram patient) should be considered cause for concern.

Recognition of decreased urine output is much more straightforward than recognition of acute kidney injury in the PACU. For patients receiving postoperative laboratory studies, AKI may manifest as increased blood urea nitrogen (BUN) and creatinine, with or without decrease in urine output. According to RIFLE criteria, a 50% increase in creatinine places the patient at risk of acute kidney injury⁶. If preoperative creatinine values are not available, it is important to remember that serum creatinine must be interpreted with the patient’s age, sex and body weight in mind. Normal creatinine (corresponding to GFR > 100 mL/min) for a 20-year-old 70-kilogram man is roughly 1.0 mg/dL based on the MDRD equation; a normal value for a 90-year-old 50-kilogram woman is 0.6 mg/dL⁵³.

More rarely, patients with severe AKI in the PACU may exhibit signs of florid renal failure that usually constitute indications for urgent renal replacement therapy: volume overload leading to congestive heart failure and pulmonary edema, hyperkalemia and acidemia⁵⁴.

Postoperative patients are unlikely to acutely demonstrate other indications for urgent dialysis, including hypermagnesemia and symptomatic uremia.

Diagnosis of decreased urine output and acute kidney injury in the PACU

The evaluation of decreased urine output and AKI in the PACU should be guided by the patient's history and presentation. As mentioned earlier, hypotension and hypovolemia are important causes of AKI. It is important to approach diagnosis of decreased urine output and acute kidney injury in a stepwise fashion, considering the most common and most easily remedied causes first. We present an example of a diagnostic algorithm in Figure 2.

Management of decreased urine output and acute kidney injury in the PACU

Determining the etiology of AKI is the initial step in management⁵⁵. Once reversible causes of AKI such as obstruction, hypotension, hypovolemia and intra-abdominal hypertension are excluded or ameliorated, the fundamental strategy for renal protection in the early stages of AKI continues to be maintenance of adequate renal perfusion and minimization of nephrotoxins⁵². Renal perfusion may be maintained with fluid administration and/or vasoactive medication, but as discussed above, the amount of fluid or the selection of the type of fluid or vasoactive medication has not been established.

What is the role for pharmacologic treatment of postoperative AKI? The use of diuretics in the management of AKI in general is historically contested. Oliguria has been associated with increased mortality in patients with AKI⁵⁶; it also makes fluid balance difficult to achieve. Animal models support the use of diuretics in AKI. The kidney demands a certain amount of oxygen to reabsorb sodium. Since loop diuretics block sodium reabsorption, experimental evidence has suggested that they also increase oxygen generation in renal tissue thereby reducing the risk of AKI⁵⁷. Also in animal models, loop diuretics have been shown to improve renal blood flow by decreasing renal vascular resistance. However, in clinical trials, the use of diuretics in AKI failed to show any significant benefit in clinical outcome. Thus, diuretics may be useful in maintaining fluid balance, but they are not an effective treatment for AKI itself. Moreover, hypovolemia with diuretic use must be avoided so as not to compound renal injury⁵⁷. Other pharmacologic interventions such as dopamine or ANP have not been shown to improve outcomes in AKI⁵².

Once the diagnosis of renal failure has been made, and reversible causes treated, the management plan must then be focused on minimizing the complications associated with renal failure. Weight and fluid intake should be strictly recorded, serum chemistries should be drawn assess for metabolic disarray. If profound hyperkalemia, metabolic acidosis or volume overload is detected, renal replacement therapy may be indicated⁵⁸.

Summary

Decreased urine output (urine output less than 0.5 mL/kg/hour) is commonly encountered in the post-anesthesia care unit. Acute kidney injury in the PACU is rarer, but development of AKI is a very poor prognostic sign. Prevention of AKI is optimal, but once it has developed, rapid recognition and supportive treatment is important to optimize patient outcomes. The mainstay of supportive therapy for decreased urine output and AKI is restoration of intravascular volume and renal blood flow. Pharmacologic treatment and renal replacement therapy are usually not indicated for treatment of decreased urine output and acute kidney injury in the immediate postoperative setting.

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References

1. Chertow GM, Burdick E, Honour M, et al. Acute kidney injury, mortality, length of stay, and costs in hospitalized patients. *Journal of the American Society of Nephrology*. 2005; 16(11):3365–3370. [PubMed: 16177006]
2. Carmichael P, Carmichael AR. Acute renal failure in the surgical setting. *ANZ Journal of Surgery*. 2003; 73(3):144–153. [PubMed: 12608979]
3. Ishani A, NDCB, et al. The magnitude of acute serum creatinine increase after cardiac surgery and the risk of chronic kidney disease, progression of kidney disease, and death. *Archives of Internal Medicine*. 2011; 171(3):226–233. [PubMed: 21325112]
4. Levy EM, Viscoli CM, Horwitz RI. The effect of acute renal failure on mortality: A cohort analysis. *Journal of the American Medical Association*. 1996; 275(19):1489–1494. [PubMed: 8622223]
5. Klahr S, Miller SB. Acute oliguria. *New England Journal of Medicine*. 1998; 338(10):671–675. [PubMed: 9486997]
6. Bellomo R, Ronco C, Kellum JA, et al. Acute renal failure - definition, outcome measures, animal models, fluid therapy and information technology needs: the Second International Consensus Conference of the Acute Dialysis Quality Initiative (ADQI) Group. *Critical care (London, England)*. 2004; 8(4):R204–R212.
7. Mehta RL, Kellum JA, Shah SV, et al. Acute kidney injury network: Report of an initiative to improve outcomes in acute kidney injury. *Critical Care*. 2007; 11
8. Cruz DN, Ricci Z, Ronco C. Clinical review: RIFLE and AKIN--time for reappraisal. *Critical Care*. 2009; 13(3):211. [PubMed: 19638179]
9. Macedo E, Malhotra R, Bouchard J, et al. Oliguria is an early predictor of higher mortality in critically ill patients. *Kidney international*. 2011; 80(7):760–767. [PubMed: 21716258]
10. Uchino S, Bellomo R, Goldsmith D, et al. An assessment of the RIFLE criteria for acute renal failure in hospitalized patients. *Critical Care Medicine*. 2006; 34(7):1913–1917. [PubMed: 16715038]
11. Lassnigg A, Schmidlin D, Mouhieddine M, et al. Minimal changes of serum creatinine predict prognosis in patients after cardiothoracic surgery: a prospective cohort study. *Journal of the American Society of Nephrology*. 2004; 15(6):1597–1605. [PubMed: 15153571]
12. Bagshaw SM, George C, Bellomo R, et al. A comparison of the RIFLE and AKIN criteria for acute kidney injury in critically ill patients. *Nephrology Dialysis Transplantation*. 2008; 23(5):1569–1574.
13. Haase M, Bellomo R, Matalanis G, et al. A comparison of the RIFLE and Acute Kidney Injury Network classifications for cardiac surgery-associated acute kidney injury: a prospective cohort study. *Journal of Thoracic and Cardiovascular Surgery*. 2009; 138(6):1370–1376. [PubMed: 19733864]
14. Ostermann M. Acute kidney injury on admission to the intensive care unit: where to go from here? *Critical Care*. 2008; 12(6):189. [PubMed: 19090977]
15. Kheterpal S, Tremper KK, Englesbe MJ, et al. Predictors of postoperative acute renal failure after noncardiac surgery in patients with previously normal renal function. *Anesthesiology*. 2007; 107(6):892–902. [PubMed: 18043057]

16. Shusterman N, Strom BL, Murray TG, et al. Risk factors and outcome of hospital-acquired acute renal failure. Clinical epidemiologic study. *American Journal of Medicine*. 1987; 83(1):65–71. [PubMed: 3605183]
17. Abelha FJ, Botelho M, Fernandes V, et al. Determinants of postoperative acute kidney injury. *Critical Care*. 2009; 13(3)
18. Shaw A, Swaminathan M, Stafford-Smith M. Cardiac Surgery-Associated Acute Kidney Injury: Putting Together the Pieces of the Puzzle. *Nephron Physiology*. 2008; 109(4):p55–p60. [PubMed: 18802376]
19. Parolari A, Pesce LL, Pacini D, et al. Risk factors for perioperative acute kidney injury after adult cardiac surgery: role of perioperative management. *Annals of Thoracic Surgery*. 2012; 93(2):584–591. [PubMed: 22269725]
20. Venkataraman R, Kellum JA. Prevention of acute renal failure. *Chest*. 2007; 131(1):300–308. [PubMed: 17218591]
21. Bauer M, George JE Iii, Seif J, et al. Recent advances in epidural analgesia. *Anesthesiology Research and Practice*. 2012; 2012
22. Loreda JS, Nelesen R, Ancoli-Israel S, et al. Sleep quality and blood pressure dipping in normal adults. *Sleep*. 2004; 27(6):1097–1103. [PubMed: 15532203]
23. Gutierrez G, Reines HD, Wulf-Gutierrez ME. Clinical review: Hemorrhagic shock. *Critical Care*. 2004; 8(5):373–381. [PubMed: 15469601]
24. Ameloot K, Gillebert C, Desie N, et al. Hypoperfusion, Shock States, and Abdominal Compartment Syndrome (ACS). *Surgical Clinics of North America*. 2012; 92(2):207–220. [PubMed: 22414408]
25. Kellum JA. Acute kidney injury. *Critical Care Medicine*. 2008; 36(SUPPL. 4):S141–S145. [PubMed: 18382185]
26. Kinsey GR, Okusa MD. Pathogenesis of acute kidney injury: Foundation for clinical practice. *American Journal of Kidney Diseases*. 2011; 58(2):291–301. [PubMed: 21530035]
27. Diaz GC, Moitra V, Sladen RN. Hepatic and Renal Protection During Cardiac Surgery. *Anesthesiology Clinics*. 2008; 26(3):565–590. [PubMed: 18765223]
28. Wald R, Waikar SS, Liangos O, et al. Acute renal failure after endovascular vs open repair of abdominal aortic aneurysm. *Journal of Vascular Surgery*. 2006; 43(3):460–466. e462. [PubMed: 16520155]
29. Kodner CM, Kudrimoti A. Diagnosis and management of acute interstitial nephritis. *American Family Physician*. 2003; 67(12):2527–2534. 2539. [PubMed: 12825841]
30. Tseng TY, Stoller ML. Obstructive Uropathy. *Clinics in Geriatric Medicine*. 2009; 25(3):437–443. [PubMed: 19765491]
31. Konety BR, Allareddy V, Herr H. Complications after radical cystectomy: Analysis of populationbased data. *Urology*. 2006; 68(1):58–64. [PubMed: 16806414]
32. Shabsigh A, Korets R, Vora KC, et al. Defining Early Morbidity of Radical Cystectomy for Patients with Bladder Cancer Using a Standardized Reporting Methodology. *European Urology*. 2009; 55(1):164–176. [PubMed: 18675501]
33. Konety, BR.; Barbour, S.; Carroll, PR. Urinary Diversion and Bladder Substitution. In: Tanagho, EA.; McAninch, JW., editors. *Smith's General Urology*. 17 ed.. New York: McGraw-Hill; 2008.
34. Brandes S, Coburn M, Armenakas N, et al. Diagnosis and management of ureteric injury: An evidence-based analysis. *BJU International*. 2004; 94(3):277–289. [PubMed: 15291852]
35. Palaniappa NC, Telem DA, Ranasinghe NE, et al. Incidence of iatrogenic ureteral injury after laparoscopic colectomy. *Archives of Surgery*. 2012; 147(3):267–271. [PubMed: 22430909]
36. Van Mastrigt GAPG, Heijmans J, Severens JL, et al. Short-stay intensive care after coronary artery bypass surgery: Randomized clinical trial on safety and cost-effectiveness. *Critical Care Medicine*. 2006; 34(1):65–75. [PubMed: 16374158]
37. Maleux G, Koolen M, Heye S. Complications after endovascular aneurysm repair. *Seminars in Interventional Radiology*. 2009; 26(1):3–9. [PubMed: 21326525]
38. Schetz M, Bove T, Morelli A, et al. Prevention of cardiac surgery-associated acute kidney injury. *International Journal of Artificial Organs*. 2008; 31(2):179–189. [PubMed: 18311734]

39. Sandham JD, Hull RD, Brant RF, et al. A randomized, controlled trial of the use of pulmonary artery catheters in high-risk surgical patients. *New England Journal of Medicine*. 2003; 348(1):5–14. [PubMed: 12510037]
40. Marquez J, McCurry K, Severyn DA, et al. Ability of pulse power, esophageal Doppler, and arterial pulse pressure to estimate rapid changes in stroke volume in humans. *Critical Care Medicine*. 2008; 36(11):3001–3007. [PubMed: 18824912]
41. Lopes MR, Oliveira MA, Pereira VO, et al. Goal-directed fluid management based on pulse pressure variation monitoring during high-risk surgery: a pilot randomized controlled trial. *Critical Care*. 2007; 11(5):R100. [PubMed: 17822565]
42. Bouchard J, Soroko SB, Chertow GM, et al. Fluid accumulation, survival and recovery of kidney function in critically ill patients with acute kidney injury. *Kidney international*. 2009; 76(4):422–427. [PubMed: 19436332]
43. Brandstrup B, Tonnesen H, Beier-Holgersen R, et al. Effects of intravenous fluid restriction on postoperative complications: comparison of two perioperative fluid regimens: a randomized assessor-blinded multicenter trial. *Annals of surgery*. 2003; 238(5):641–648. [PubMed: 14578723]
44. Trof RJ, Sukul SP, Twisk JW, et al. Greater cardiac response of colloid than saline fluid loading in septic and non-septic critically ill patients with clinical hypovolaemia. *Intensive Care Med*. 2010; 36(4):697–701. [PubMed: 20165941]
45. Groeneveld AB, Navickis RJ, Wilkes MM. Update on the comparative safety of colloids: a systematic review of clinical studies. *Annals of surgery*. 2011; 253(3):470–483. [PubMed: 21217516]
46. Haase M, Haase-Fielitz A, Bellomo R, et al. Sodium bicarbonate to prevent increases in serum creatinine after cardiac surgery: a pilot double-blind, randomized controlled trial. *Critical Care Medicine*. 2009; 37(1):39–47. [PubMed: 19112278]
47. Malbrain ML, Chiumello D, Pelosi P, et al. Incidence and prognosis of intraabdominal hypertension in a mixed population of critically ill patients: a multiple-center epidemiological study. *Critical Care Medicine*. 2005; 33(2):315–322. [PubMed: 15699833]
48. Rudnick MR, Kesselheim A, Goldfarb S. Contrast-induced nephropathy: how it develops, how to prevent it. *Cleveland Clinic journal of medicine*. 2006; 73(1):75–80. 83–77. [PubMed: 16444919]
49. Navaneethan SD, Singh S, Appasamy S, et al. Sodium bicarbonate therapy for prevention of contrast-induced nephropathy: a systematic review and meta-analysis. *American Journal of Kidney Diseases*. 2009; 53(4):617–627. [PubMed: 19027212]
50. Nigwekar SU, Navaneethan SD, Parikh CR, et al. Atrial natriuretic peptide for management of acute kidney injury: a systematic review and meta-analysis. *Clinical Journal of the American Society of Nephrology*. 2009; 4(2):261–272. [PubMed: 19073785]
51. Friedrich JO, Adhikari N, Herridge MS, et al. Meta-analysis: low-dose dopamine increases urine output but does not prevent renal dysfunction or death. *Annals of Internal Medicine*. 2005; 142(7):510–524. [PubMed: 15809463]
52. Tolwani A, Paganini E, Joannidis M, et al. Treatment of patients with cardiac surgery associated acute kidney injury. *International Journal of Artificial Organs*. 2008; 31(2):190–196. [PubMed: 18311735]
53. Roth D, Rogers N, Greene T, et al. A More Accurate Method To Estimate Glomerular Filtration Rate from Serum Creatinine: A New Prediction Equation. *Annals of Internal Medicine*. 1999; 130(6):461–470. [PubMed: 10075613]
54. Gibney N, Hoste E, Burdmann EA, et al. Timing of initiation and discontinuation of renal replacement therapy in AKI: Unanswered key questions. *Clinical Journal of the American Society of Nephrology*. 2008; 3(3):876–880. [PubMed: 18322044]
55. Sykes E, Cosgrove JF. Acute renal failure and the critically ill surgical patient. *Annals of the Royal College of Surgeons of England*. 2007; 89(1):22–29. [PubMed: 17316516]
56. Bagshaw SM, Bellomo R, Kellum JA. Oliguria, volume overload, and loop diuretics. *Critical Care Medicine*. 2008; 36(4 Suppl):S172–S178. [PubMed: 18382190]
57. Nigwekar SU, Waikar SS. Diuretics in acute kidney injury. *Seminars in Nephrology*. 2011; 31(6):523–534. [PubMed: 22099509]

58. Palevsky PM. Indications and timing of renal replacement therapy in acute kidney injury. *Critical Care Medicine*. 2008; 36(4 Suppl):S224–S228. [PubMed: 18382198]

Key Points

Decreased urine output is a common occurrence in the post-anesthesia care unit (PACU).

Acute kidney injury (AKI) is uncommon in the PACU, but is associated with increased morbidity and mortality.

Maintaining adequate renal perfusion by ensuring euvolemia and normotension is key to preventing the development of AKI in the PACU.

Once decreased urine output or AKI develop, urinary obstruction should be ruled out and intravascular volume and blood pressure should be restored.

Intra-abdominal hypertension can compromise renal perfusion despite euvolemia and normotension, increasing the risk of postoperative AKI.

Synopsis

Decreased urine output and acute kidney injury (also known as acute renal failure) are among the most important complications that may develop in the post-anesthetic period. In this article, we present definitions of decreased urine output, oliguria, and acute kidney injury. We review the epidemiology, pathophysiology and prevention of postoperative acute kidney injury. Finally we offer approaches to diagnosis and management of the post-surgical patient with decreased urine output and/or acute kidney injury.

RIFLE class	Serum creatinine*	Urine output	AKIN class
Risk	1.5 x baseline	< 0.5 mL/kg/hr x 6 hours	Stage 1
Injury	2 x baseline	< 0.5 mL/kg/hr x 12 hours	Stage 2
Failure	3 x baseline or creatinine > 4 mg/dL or acute rise of ≥ 0.5 mg/dL	<0.3 mL/kg/hr x 24 or anuria x 12 hours	Stage 3 **
Loss [§]	*RIFLE criteria include changes in glomerular filtration rate (GFR): RISK: 25% reduction in GFR INJURY: 50% reduction in GFR FAILURE: 75% reduction in GFR **AKIN Stage 3 is automatically designated for any patients receiving renal replacement therapy. §Loss in the RIFLE criteria = persistent acute renal failure, i.e. complete loss of renal function for >4 weeks		
End Stage Renal Disease			

Increasing AKI severity

Figure 1.
Grading of acute kidney injury according to RIFLE and AKIN classifications (6,7)

Figure 2.
Diagnostic algorithm for decreased urine output and acute kidney injury in the PACU.

Table 1

Risk factors for development of postoperative AKI.

Preoperative factors	<ul style="list-style-type: none"> • Preoperative renal dysfunction • Increasing age • Heart disease (ischemic or congestive) • Smoking • Diabetes mellitus • ASA PS 4 or 5
Intraoperative factors	<ul style="list-style-type: none"> • Emergency surgery, or intraperitoneal, intrathoracic, suprainguinal vascular surgeries • Erythrocyte transfusion • Inotrope use • Aortic cross-clamp time • CPB: furosemide use, urine output, need for a new pump run
Postoperative factors	<ul style="list-style-type: none"> • Erythrocyte transfusion • Vasoconstrictor use • Diuretic use • Antiarrhythmic drug use

Modified from [17], [19]. ASA PS: American Society of Anesthesiologists Physical Status classification. CPB: cardiopulmonary bypass.

Table 2

Common causes of post-operative decreased urine output and acute kidney injury.

	Site of defect		
	Pre-renal	Renal	Post-renal
Differential diagnoses	<ul style="list-style-type: none"> • Hypotension <ul style="list-style-type: none"> ◦ Absolute ◦ Relative • Hypovolemia <ul style="list-style-type: none"> ◦ Absolute ◦ Relative (e.g. IAH) 	<ul style="list-style-type: none"> • Acute tubular necrosis <ul style="list-style-type: none"> ◦ Ischemia-reperfusion ◦ Radiocontrast • Acute interstitial nephritis 	<ul style="list-style-type: none"> • Urinary catheter obstruction <ul style="list-style-type: none"> ◦ Catheter kinking ◦ Debris • Prostatic hypertrophy • Bladder spasm • Urinary retention

IAH: Intra-abdominal hypertension.

Table 3

Selected procedure-specific causes of decreased urine output and AKI.

Surgical specialty	Example of procedure	Connection to AKI
Urology	Cystectomy and diversion	Ureteral damage, compromised ureteral anastomoses
General surgery and gynecology	Colectomy, hysterectomy	Ureteral damage
Cardiac surgery	Any procedure with CPB	Acute tubular necrosis from ischemia-reperfusion, non-pulsatile flow, complement activation
Vascular surgery	Endovascular aortic aneurysm repair	Renal ischemia from partial or complete stenting over the renal arteries, contrast-induced nephropathy