Anesthetic Concerns in Patients Presenting with Renal Failure

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KEYWORDS

• Renal function • Acute kidney injury
• Anesthesia • Renal failure

RENAL PHYSIOLOGY

The foremost function of the kidneys is to maintain fluid and electrolyte balance, by a tightly controlled system that is able to maintain homeostasis even in perilous metabolic situations. Other tasks include the excretion of metabolic waste products, control of vascular tone, and regulation of hematopoesis and bone metabolism.

The kidneys are the best-perfused organ per gram of tissue and receive 20% of the cardiac output. Global renal blood flow is autoregulated and is kept constant at a mean arterial pressure of 50 to 150 mm Hg in normotensive patients.[1] Blood flow to the glomerulus is regulated through the afferent and efferent sphincters, which adjust the glomerular filtration pressure. Depending on this filtration pressure a large amount of fluid (approximately 120 mL/min) is filtered into the capsular space of the Bowman capsule and then into the tubuli. Most of this glomerular filtrate is reabsorbed in the distal tubules of the inner medulla: active adenosine triphosphate (ATP) pumps move NaCl into the interstitium while water follows passively across an osmolar gradient. Urine and plasma osmolality are regulated by the feedback mechanism of the loop of Henle: increased interstitial NaCl concentrations (ie, as a result of hypovolemia) lead to an increased reabsorption of water and a decrease in urine output.

Renal blood flow is heterogenous. The renal cortex receives approximately 90% of renal blood flow, whereas the metabolically active renal medulla receives only about

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10%. Tissue P_{O_2} is 50 to 100 mmHg in the cortex, whereas it can be as low as 10 to 15 mmHg in the medullary thick ascending limb. The renal medulla extracts 79% of delivered oxygen compared with only 18% in the renal cortex, which renders the renal medulla extraordinarily sensitive to ischemia.

In response to hypotension, systemic activation of the sympathetic and adrenal systems leads to redistribution of renal blood flow within the kidneys preferentially toward the metabolically active medulla and inner cortex.[2] Initially there is a preservation of glomerular filtration rate (GFR) and renal function, but with prolonged or more severe ischemia, active NaCl pumps in the thick ascending limb break down and sodium reabsorption decreases. Chemoreceptors in the macula densa of the juxtaglomerular apparatus detect the increased intraluminal chloride concentration and release renin. Renin then causes constriction of the afferent arteriole and a dramatic decrease of GFR that leads to a further reduction of urine output and oliguric renal failure ensues. Without the feedback mechanism of the macula densa, GFR would remain high (120 mL/min), water could not be reabsorbed, and fatal dehydration would occur within hours.[3]

The kidneys are able to tolerate substantial insults while maintaining adequate function despite this theoretic vulnerability to ischemia. Multiple and severe insults are required to cause an injury severe enough to manifest a clinically relevant decrease of renal function (**Fig. 1**). The most common cause of perioperative renal injury is ischemia-reperfusion injury. Ischemia-reperfusion injury causes tubular necrosis and apoptosis, especially of the medullary thick ascending loop of Henle. During reperfusion there is an influx of proinflammatory cells, neutrophils, and macrophages, which

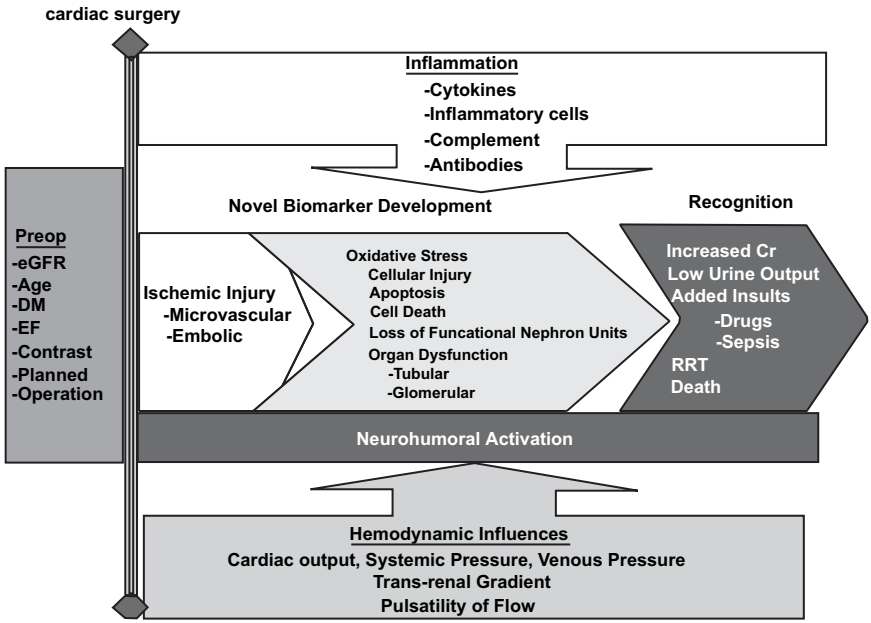

Fig. 1. Risk factors, mechanisms of injury and means of detection for AKI in relation to cardiac surgery. (*From* Bellomo R, Auriemma S, Fabbri A, et al. The pathophysiology of cardiac surgery-associated acute kidney injury (CSA-AKI). Int J Artif Organs 2008;31:167; with permission.)

release cytokines and radical oxygen species that further amplify necrosis and apoptosis.[4] In addition to direct injury, renal tubules become obstructed by cellular debris.[5]

Nephrotoxic insults caused by calcineurin inhibitors or aminoglycosides have a similar clinical presentation as ischemia-reperfusion injury even though the mechanism of injury is different. Calcineurin inhibitors cause profound afferent arteriolar vasoconstriction and decrease GFR,[6] whereas aminoglycosides cause tubular cell damage after reuptake into proximal tubular cells by megalin receptors.[7] Nonsteroidal antiinflammatory agents inhibit prostaglandin synthesis, which alone does not cause renal injury in normal subjects. In the setting of hypovolemia or in addition to other nephrotoxic insults, nonsteroidal antiinflammatory drugs may convert a small renal injury into overt renal failure as prostaglandin synthesis is essential to dilate the afferent arteriolar sphincter and maintain GFR.[8] Radiocontrast can cause renal injury, as it induces medullary vasoconstriction through activation of adenosin/endothelin receptors as well as by a direct cytotoxic effect of the high osmolality of radiocontrast.[9] The resultant clinical presentation is similar to ischemia-reperfusion injury, although usually a single insult with radiocontrast is not sufficient to induce clinically overt acute kidney injury (AKI).[10]

AKI

AKI often results from multiple insults and is frequently a consequence of a combination of prerenal azotemia and intrarenal acute tubular necrosis. In acute renal failure renal function deteriorates over hours or days. Primary renal diseases such as glomerulonephritis are rare in surgical populations and often associated with severe proteinuria and nephritic syndrome. Treatment of nephritic syndrome consists of replacement of protein loss and diuresis, steroids, and other immunosuppressive drugs that may reverse the symptoms.

Postrenal azotemia may be caused by renal calculi, tumors or even a blocked Foley catheter and rapid recovery of renal function will occur if the obstruction is removed or bypassed expeditiously. Iatrogenic injury of the ureter may occur during lower abdominal surgery, and the diagnosis of a dilated renal collecting system either by computed tomography scan or ultrasound should prompt rapid placement of either ureteral stents or nephrostomy tubes to relieve the pressure and avoid further, potentially irreversible injury to the kidney.

Reversible prerenal azotemia and acute tubular necrosis caused by medullary ischemia are two ends of a continuum. Prerenal azotemia is common and a physiologic response to hypovolemia. It increases tubular workload and decreases medullary blood supply. Any additional renal insult may result in sufficient medullary ischemia to cause acute renal failure. Urine output then decreases despite adequate intravascular filling, and waste products accumulate. The traditional division of prerenal versus intrarenal azotemia is therefore artificial, but may help guide treatment options, especially if further hydration may potentially reverse the condition. Once acute renal failure is established there is no intervention that has proven beneficial to expedite the recovery of renal function. In most cases renal function recovers spontaneously within days. However, it is essential to avoid further renal insults and support impaired physiologic systems to prevent progression to chronic renal failure (**Fig. 2**).

EPIDEMIOLOGY OF AKI

For many years the lack of a uniform definition and even a uniform term for renal injury has hampered clinical research of renal injury. The most commonly accepted term for

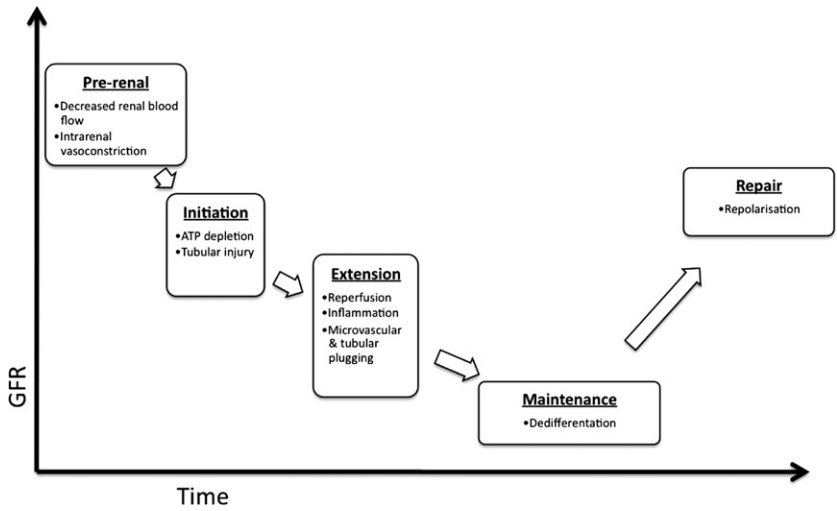

Fig. 2. Phases of AKI.

renal injury is acute kidney injury (AKI) as suggested by the American Society of Nephrology.[11] There is, however, no uniform definition of AKI. The Second International Consensus Conference of the Acute Dialysis Quality Initiative attempted to create a universally accepted definition of acute renal failure with the RIFLE criteria. The RIFLE criteria for AKI contain 5 categories (risk, injury, failure, loss, and end-stage kidney disease). The first 3 categories are defined by either percent change of serum creatinine or urine output criteria (**Fig. 3**).[12] The RIFLE criteria were initially widely recognized but later research showed that even small absolute changes of serum creatinine level affect morbidity and mortality.[13] The Acute Kidney Injury Network subsequently introduced a definition of AKI based on change of serum creatinine level of greater than 0.3 mg/dL within 48 hours after insult.[14]

Kheterpal and colleagues[15] recently reported that in a large national database 1% of all patients undergoing general surgery developed postoperative AKI. Patients developing AKI were more likely to be male, older, diabetic, and had more frequently a history of congestive heart failure, hypertension, ascites, or preoperative renal insufficiency. Emergency surgery doubled and intraperitoneal surgery more than tripled the risk for postoperative AKI. Patients who had AKI had a 3 times higher risk of postoperative morbidity and a fivefold increase in mortality.

AKI requiring renal replacement therapy (RRT) after cardiac surgery is rare (1%) but can be catastrophic and is associated with a mortality of greater than 60%. AKI based on the increase of serum creatinine level is more frequent but it is unclear if it is an independent contributor to mortality and morbidity or rather a reflection of more complex surgical procedures performed on complex medical patients.[16,17]

ASSESSMENT OF RENAL FUNCTION AND INJURY

Progress in renal protective strategies has been hampered by the lack of early sensitive markers of renal injury.[18] Direct measurements of GFR are cumbersome and rarely feasible. Substitute markers are required to estimate GFR but they have significant limitations.

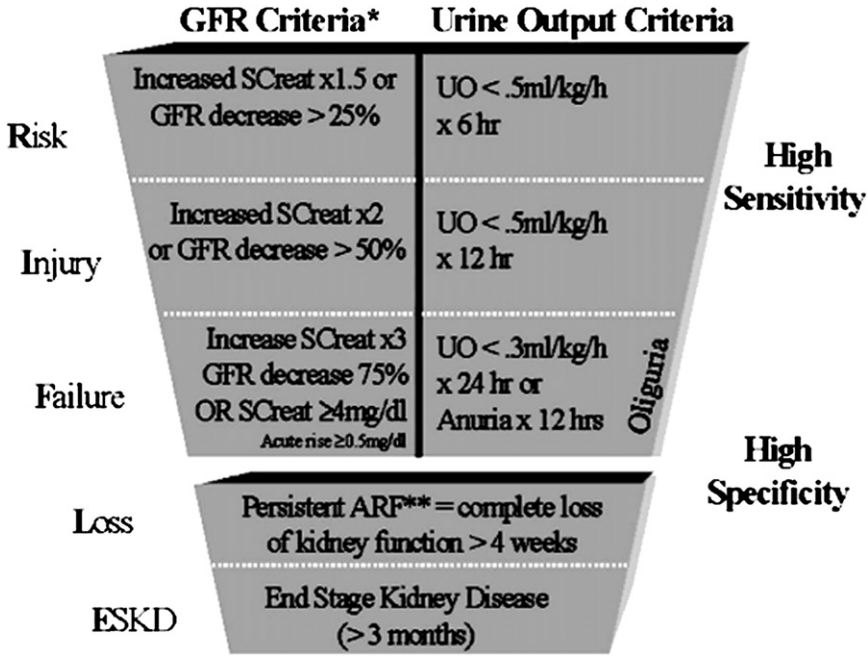

Fig. 3. Risk, injury, failure, loss of renal function, and end-stage kidney disease (RIFLE) criteria of AKI. (*From* Bellomo R, Ronco C, Kellum JA, et al. Acute renal failure – definition, outcome measures, animal models, fluid therapy and information technology needs: the Second International Consensus Conference of the Acute Dialysis Quality Initiative (ADQI) Group. Crit Care 2004;8:R206; with permission.)

Serum Creatinine

The most commonly used marker for renal function, serum creatinine level, is insensitive and slow to increase and, therefore, rarely detects renal injury rapidly enough for successful intervention. Small changes of serum creatinine level may represent large changes in GFR and there is not a linear relationship between creatinine and GFR. Serum creatinine requires time to accumulate and in the immediate perioperative period serum creatinine may even be decreased from preoperative levels because of dilution. Serum creatinine level is a marker of renal function and not injury.[19]

Urine Output

Urine output is not a reliable marker of renal function. Adequate urine output is usually associated with adequate renal function. Anuria is a sign of severe renal injury unless there is postrenal obstruction and requires immediate investigation. Low urine output may have various causes. Intra-abdominal surgery, especially laparoscopic, causes a decrease in renal blood flow and urine output that does not necessarily represent significant renal injury. Low urine output caused by hypovolemia may be secondary to easily reversible prerenal azotemia, which if left untreated can progress to acute renal injury.[20,21]

Fractional Excretion of Sodium

The fraction excretion of sodium (FeNa) measures the amount of filtered sodium that is excreted in the urine and is the most accurate test to aid the differential diagnosis of

prerenal azotemia and acute tubular necrosis[22] (**Table 1**): FeNa % = ($U_{Na} \times P_{Cr}$)/($P_{Na} \times U_{Cr}$). A FeNa less than 1% is consistent with prerenal azotemia and greater than 2% with tubular injury. Concomitant use of diuretics decreases the diagnostic power of FeNa. Similarly, the fractional excretion of urea can be measured and may be a better reflection of renal function in patients receiving diuretics.[23]

Novel Biomarkers

Several novel biomarkers of renal injury and function have been studied in recent years, some of which have shown promising results. Serum cystatin C is a protein produced by all nucleated cells that is freely filtrated by the kidneys and not reabsorbed. Serum cystatin C is independent of age, muscle mass, sex, or race and reflects GFR better than serum creatinine.[24] Further studies are required to validate serum cystatin C as the better marker of renal function. Urinary neutrophil gelatinase-associated lipocalin is a protein produced by tubular cells as a response to injury. It is easily detected in the urine within minutes after experimental renal injury and has been studied in a variety of clinical scenarios.[25–28] It is highly sensitive and specific for acute renal injury and only slightly increased with chronic renal insufficiency. In the future it may be an ideal end point for studies evaluating renal protective strategies but more studies are required before its clinical use is feasible.[18,29]

RENAL PROTECTION AND TREATMENT OF AKI

There is no proven prophylaxis or treatment of AKI despite countless studies. Many interventions that were deemed successful in preclinical or early, small clinical trials were shown to be ineffective in larger studies that reflected realistic clinical scenarios.

Fluids

Maintaining renal blood flow and GFR through adequate hydration will prevent further renal injury and preserve renal function. Hydration has been shown to be effective in the prevention of contrast-induced nephropathy and other clinical scenarios of renal injury and is probably the best strategy to prevent progression to frank renal failure.[30]

Dopamine

Multiple large randomized trials and meta-analyses have found no therapeutic or prophylactic effect of dopamine on AKI.[31–33] Dopamine may increase urine output and may also be beneficial as treatment of low cardiac output states and treatment of bradycardia, leading to improved renal perfusion.

Bicarbonate

The alkalinization of urine with sodium bicarbonate is effective in the treatment of pigment-induced nephropathy such as rhabdomyolysis. It increases the solubility of

Table 1
Prerenal versus renal azotemia

Test	Prerenal	Renal
Urine osmolarity (mOsm/L)	<400	250–300
Urine creatinine/plasma creatinine ratio	>40	<20
Urine osmolarity/plasma osmolarity	>1.5	<1.0
Urine sodium concentration (mEq/L)	<20	>40
Fractional excretion of sodium (%)	<1	>1.1

myoglobin and therefore prevents formation of tubular precipitates.[34] Nephrotoxic free radicals are preferentially formed in acidotic environments, for example, through the Haber-Weiss reaction. Treatment with sodium bicarbonate decreases the formation of free radicals in contrast-induced nephropathy as well. A randomized trial found that pretreatment of bicarbonate reduced the incidence of contrast-induced nephropathy from 13.7% to 1.7%.[35]

Loop Diuretics/Mannitol

The rationale for using diuretics in the treatment of AKI is to flush out casts of necrotic cells that may obstruct renal tubuli. Loop diuretics also increase renal blood flow through increased prostaglandin synthesis and decrease metabolic workload of the tubuli by decreasing active sodium reabsorption. However, most clinical studies and a meta-analysis of randomized controlled trials found no effect of loop diuretics in the treatment or prevention of AKI.[36] Treatment with diuretics can lead to hypovolemia and further exacerbate renal hypoperfusion; therefore, it is imperative to ensure normovolemia before their use.

Acetyl Cysteine

Acetyl cysteine is a free radical oxygen scavenger and modulates nitric oxide synthesis after oxidative cell stress. Multiple studies had promising results and reported the efficacy of acetyl cysteine in the prevention of contrast-induced nephropathy when given early before the insult. Other studies were equivocal. Recent meta-analysis showed no effect in the prophylaxis of contrast-induced nephropathy[37] or in the prevention of AKI after major surgery.[38] Further studies with better-defined end points are necessary to obtain a better understanding of the clinical efficacy of acetyl cysteine.

EFFECT OF ANESTHESIA AND SURGERY ON RENAL FUNCTION

Clinical studies have failed to ascertain the benefit of one anesthetic technique over another in a general surgery population.[39] Care should be taken to maintain normovolemia and normotension to avoid decreases in renal perfusion. Volatile anesthetics in general cause a decrease in GFR likely caused by a decrease in renal perfusion pressure either by decreasing systemic vascular resistance (eg, isoflurane or sevoflurane) or cardiac output (eg, halothane). This decrease in GFR is exacerbated by hypovolemia and the release of catecholamines and antidiuretic hormone as a response to painful stimulation during surgery.[40] Recent studies have also found an amelioration of renal injury by volatile anesthetics, likely caused by a reduction in inflammation.[41]

Sevoflurane has been implicated as a cause of renal injury through fluoride toxicity. High intrarenal fluoride concentrations impair the concentrating ability of the kidney and may theoretically lead to nonoliguric renal failure. However, studies have failed to show a relevant effect in clinical practice. Sevoflurane is considered safe even in patients with renal impairment as long as prolonged low-flow anesthesia is avoided.[42,43]

Positive-pressure ventilation used during general anesthesia can decrease cardiac output, renal blood flow, and GFR. Decreased cardiac output leads to a release of catecholamines, rennin, and angiotensin II with the activation of the sympathoadrenal system and resultant decrease in renal blood flow. Insufflation of the abdomen during laparoscopic surgery has a similar effect on renal blood flow and GFR. The increased intra-abdominal pressure during laparoscopic surgery is transmitted directly to the kidneys and results in a further reduction of renal blood flow.[44]

The use of regional anesthesia techniques that achieve a sympathetic block of levels T4 to T10 may be beneficial to patients with kidney disease or those at high risk for postoperative AKI, as the sympathetic blockade attenuates catecholamine-induced renal vasoconstriction and suppresses cortisol and epinephrine release.[45] Epidural anesthesia has no effect on renal blood flow in healthy volunteers as long as normotension and isovolemia are maintained[46] and may reduce the incidence of postoperative AKI.[47]

Aortic cross-clamping or occlusion of the inferior vena cava during liver transplantation can cause renal injury that frequently progresses to AKI and substantially increases mortality and morbidity.[48–50] Cardiopulmonary bypass impairs renal blood flow and renal perfusion and may cause renal injury that might not be apparent early after surgery, as serum creatinine is often diluted in the early postoperative period.[51] Avoiding cardiopulmonary bypass with the use of off-pump coronary bypass grafting (CABG) does not necessarily reduce the degree of renal injury: hypotension, microemboli, and renal hypoperfusion during off-pump CABG may cause renal injury comparable to cardiopulmonary bypass,[52] and the results of clinical trials have been equivocal.

Avoiding intraoperative renal insults and maintaining isovolemia, adequate cardiac output, and renal perfusion pressure are the best interventions to prevent postoperative AKI and are more important than the choice of a specific anesthetic technique.

PHARMACOLOGIC MANAGEMENT OF THE PATIENT WITH RENAL FAILURE

Many drugs commonly used during anesthesia are dependent to some degree on renal excretion for elimination, and this must be taken into consideration when planning an anesthetic for a patient with renal dysfunction. Patients with renal disease are sensitive to barbiturates and benzodiazepines secondary to decreased protein binding. Some narcotic agents including morphine and meperidine should be used judiciously if at all as they have active metabolites and may have prolonged activity in the setting of renal dysfunction. Fentanyl and hydromorphone are better choices.[53,54] Succinylcholine can be used if the patient's serum potassium level is normal, but is best avoided if the potassium level is unknown. Cisatracurium and atracurium are nondepolarizing muscle relaxants that do not rely on renal function for their elimination, but are metabolized by ester hydrolysis and Hoffmann elimination. Neuromuscular reversal agents rely on renal excretion and, therefore, their effects will be prolonged.[55] Many antimicrobial agents must be dosed according to renal function. Nonsteroidal antiinflammatory agents should be avoided in renal insufficiency or AKI as they may exacerbate renal injury.

COMPLICATIONS OF RENAL FAILURE AND ITS IMPLICATION FOR THE ANESTHESIOLOGIST

Patients with renal failure undergoing surgery are at a substantial risk for increased morbidity and mortality. Patients with renal failure often have other comorbidities, including hypertension, diabetes, peripheral vascular disease, and cardiac disease. Renal failure has various consequences on homeostasis that are not only restricted to water and electrolyte abnormalities, but affect many organ systems, making intraoperative management of these patients especially challenging.

These patients can be hemodynamically labile during surgery and anesthesia. For minor procedures such as an inguinal hernia repair or an arteriovenous fistula, routine anesthetic monitors are probably sufficient. For major surgery, the anesthesiologist should probably place an arterial line to allow for continuous blood pressure

monitoring to optimize patient care. Venous access can be challenging as well, as these patients often have vascular disease and have had a dialysis access procedure, which precludes the use of that arm for venous access. It is important to take your time and make sure that there is adequate venous access for the planned procedure. Central venous access to enable monitoring of central venous pressures may be beneficial to guide fluid management in patients who are oliguric or anuric during major surgery.

Patients with chronic renal failure undergoing elective surgery should receive dialysis treatment the day before planned surgery to optimize their electrolyte, metabolic, and volume status. It is appropriate to minimize intravenous fluid administration in this patient population for minor surgery. The importance of maintaining euvolemia during major surgery cannot be overstated to maintain adequate preload, thus avoiding hypotension and potential organ hypoperfusion.

Neurologic sequelae, including confusion, sedation, or obtundation, can result from uremic encephalopathy.[56] Intubation for airway protection may be necessary in extreme cases. Volume overload can to lead to pulmonary edema and hypoxia. Ventilatory support may be necessary until adequate fluid removal has been achieved with dialysis or diuresis. In addition, renal failure with a resultant metabolic acidosis may require hyperventilation as respiratory compensation that may not be sustainable in the spontaneously breathing patient. Mechanical ventilation may be required until the acidosis is corrected. Blood gas analysis at the point of care can alert the anesthesiologist to metabolic derangements and hypoxemia.

Anemia is frequent in patients with chronic renal failure and is caused in part by a decrease in erythropoietin production. Patients with renal dysfunction are at an increased risk for bleeding as they have altered platelet function and decreased levels of von Willebrand factor. Uremic coagulopathy is caused by impaired platelet aggregation and adhesiveness. Preoperative dialysis may be indicated if uremia is suspected. Alternatively, desmopressin, a vasopressin analog that releases von Willebrand factor and increases factor VII levels, can be administered preoperatively or intraoperatively to help correct uremic coagulopathy.[57]

Patients with renal failure can develop various metabolic derangements, including hyperkalemia, hypocalcemia, hyperphosphotemia, and metabolic acidosis. Frequent checking of arterial blood gases and electrolytes at the point of care, if possible, allows for early intervention and correction of derangements intraoperatively. Hyperkalemia results from the inability of the medullary tubuli to excrete potassium. If chronic it may be well tolerated but acute hyperkalemia warrants aggressive treatment. The anesthesiologist should be especially vigilant in monitoring the electrocardiogram for peaked T waves or a widening of the QRS complex. Hyperkalemia may worsen with the use of depolarizing muscle relaxants, ie, succinylcholine, and it should be avoided unless preoperative serum potassium levels are known.

Rapid transfusion of multiple units of packed red blood cells may increase potassium levels significantly. Metabolic acidosis, which often occurs in renal failure, worsens transfusion-induced hyperkalemia and may trigger arrhythmias and cardiac arrest.[58] The use of a cell saver device to prewash packed red blood cells or intraoperative continuous venovenous hemodialysis (CVVHD) should be considered in patients with restricted renal function who are likely to require many blood transfusions to prevent complications from hyperkalemia.

Treatment of hyperkalemia should be ideally aimed at the removal of excess potassium but temporizing interventions are warranted as well. Intravenous insulin moves extracellular potassium intracellularly by activating skeletal muscle Na-K ATP-dependent pumps. Usually 10 units of regular insulin intravenously are given together with

25 mL glucose 50% and glucose levels should be measured frequently afterwards. Intravenous calcium does not decrease plasma levels of potassium, but rather stabilizes the myocardium, preventing cardiac arrhythmias. Extravasation can cause severe skin necrosis and calcium should be given through a central venous catheter when possible. An increase in minute ventilation if the patient is mechanically ventilated results in an increase in plasma pH and a decrease in potassium levels. Treatment with sodium bicarbonate may increase plasma pH and drive potassium intracellularly; however, this effect is only a temporizing measure.

Treatment of hyperkalemia with loop diuretics increases potassium excretion. Cation exchangers such as sodium polystyrene sulfonate (Kayexalate) lowers potassium by binding intestinal potassium and excreting it in the stool. Sodium polystyrene sulfonate can be given orally or as a rectal enema. Treatment with sorbitol promotes osmotic diarrhea and amplifies the potassium-lowering effect but can lead to intestinal injury and colonic necrosis in patients with an ileus or after intestinal surgery and is not practical intraoperatively. If all of these interventions fail and hyperkalemia persists or becomes symptomatic, hemodialysis should be initiated as soon as possible.

Acidosis is common in acute renal failure and is often a result of the combination of impaired renal excretion of acid and an overproduction of lactic acid, for example secondary to septic shock. Hyperventilation of the mechanically ventilated patient helps to normalize pH but the spontaneously breathing patient is often unable to maintain an adequate minute ventilation to restore a normal pH. It is essential to recognize impending respiratory failure caused by increased breathing early and to intubate and mechanically ventilate the patient before cardiorespiratory collapse ensues. Severe metabolic acidosis at the end of surgery should preclude extubation until the metabolic derangements are corrected sufficiently to support spontaneous ventilation.[59]

Sodium bicarbonate is indicated in the mechanically ventilated patient when the pH decreases to less than 7.15.[60] At a pH of less than 7.15 most enzymatic and receptor-based systems fail to function properly (ie, catecholamine-based vasoconstriction). At this point treatment with sodium bicarbonate may restore the effectiveness of exogenous catecholamines and vascular tone. Sodium bicarbonate may also be indicated when bicarbonate loss and not an overproduction of acid is the cause for acidosis, as with the loss of bicarbonate through diarrhea or in renal tubular acidosis. Sodium bicarbonate should be given slowly to avoid overproduction of carbon dioxide. Carbon dioxide can easily traverse intracellularly, converting to carbonic acid and causing paradoxic intracellular acidosis.

Traditionally normal saline was the preferred intravenous fluid for patients with renal failure as it contains no potassium. However, recent studies reported that normal saline can cause a hyperchloremic metabolic acidosis that may increase the incidence of hyperkalemia more often than the use of a lactated Ringer solution (K 4 mEq/L).[61,62] The authors therefore recommend the use of lactated Ringer solution as the intravenous fluid in renal failure as long as hepatic function is sufficient to metabolize the lactate contained in the solution.

SPECIAL CONSIDERATIONS FOR PATIENTS WITH AKI

Perioperative renal failure is associated with a high morbidity and mortality. Preexisting renal insufficiency, advanced age, diabetes, and hypertension increase the risk of perioperative renal failure significantly. Assessment of the patient with acute renal failure presenting for surgery should include a thorough investigation of the cause. Subclinical sepsis may not be apparent except for an increase in serum creatinine level and white blood cell count but may unmask itself during anesthesia and surgery,

causing vasodilatation and hemodynamic instability. Cardiogenic shock as a result of myocardial ischemia or cardiac tamponade may also present as acute renal failure. If the cause of the AKI is unclear further investigation is warranted before all surgery, except for emergencies.

Septic shock causes renal failure, resulting from a decrease in systemic vascular resistance and resultant hypotension, essentially causing a maldistribution of blood flow away from the kidneys and other vital organs that results in the reduction of transglomerular perfusion pressure and GFR. In addition, renal hypoperfusion causes direct medullary ischemia and acute tubular necrosis. Further injury occurs as sepsis induces leukocytic infiltration and apoptosis of the kidneys.[63] Fluid administration and the maintenance of adequate cardiac output are key to supportive treatment. In addition, treatment with vasopressors is often required to maintain adequate perfusion to vital organs by normalizing the systemic vascular resistance.

Cardiogenic shock results in hypoperfusion of the kidneys, decreased oxygen delivery, and acute tubular necrosis.[64] Decreased urine output and volume overload as a consequence of AKI may further worsen cardiogenic shock by increasing filling pressures beyond the plateau of the Frank-Starling curve, which can further exacerbate pulmonary edema and hypoxia. Reducing ventricular filling pressures with either diuresis or fluid removal via dialysis can improve cardiac output. If no significant improvement is achieved with the normalization of filling pressures, inotropic support or placement of an intra-aortic balloon pump may be required to maintain adequate coronary perfusion.

Nephrotoxic renal injury requires judicious fluid administration to maintain renal perfusion and prevent further insults. It is critical to avoid further renal injury caused by hypovolemia or hypoperfusion to avoid frank acute kidney failure requiring RRT.

Uremic pericarditis occurs in 5% to 20% of all cases of untreated renal failure and is caused by a hemorrhagic pericardial effusion as a consequence of uremic coagulopathy or serous effusions. Patients in acute renal failure complaining of pleuritic chest pain or presenting with a pericardial rub on physical examination should undergo echocardiograhy to confirm the diagnosis. Uremic pericarditis usually responds to treatment of the underlying uremia but pericardial drainage may be necessary in tamponade or hemodynamic compromise.[65]

Patients with AKI undergoing major surgery require invasive monitoring with at a minimum an arterial line to allow for continuous blood pressure monitoring and frequent blood draws to follow electrolytes and arterial blood gases closely. A central venous catheter to enable monitoring of central venous pressure may be useful in guiding fluid management if the patient is oliguric or anuric. A pulmonary artery catheter or transesophageal echocardiography aids in the assessment of cardiac function and volume status more closely and may be useful in patients presenting with septic or cardiogenic shock. The goal of the anesthesiologist in treating the patient with AKI intraoperatively is to optimize volume and hemodynamic status, thus maintaining renal perfusion and preventing further renal injury. Nephrotoxic agents should be avoided as well.

RRT

There are 5 indications for RRT: volume overload, hyperkalemia, severe metabolic acidosis, symptomatic uremia, and intoxication of dialyzable substances. Patients with end-stage kidney disease require RRT or a renal transplant. There are 2 basic modes of RRT in patients with end-stage kidney disease: intermittent hemodialysis and peritoneal dialysis. The patient with chronic renal failure on hemodialysis should

undergo hemodialysis the day before elective surgery. If a patient with renal failure presents emergently for surgery and has an acute indication for dialysis but is hemodynamically unstable intraoperative continuous venovenous hemodialysis should be used if available.

The patient with acute renal failure who has not previously been dialyzed might require perioperative dialysis if any of the above indications are met. In addition, patients should undergo intraoperative dialysis if major blood loss with a large transfusion requirement is anticipated and the ensuing potassium load cannot be effectively managed through pharmacologic diuresis. Alternatively, cell saver can be used to wash packed red blood cells, effectively reducing the potassium load.

The mode of choice for intraoperative dialysis is continuous RRT unless the patient undergoes cardiopulmonary bypass and a dialysis membrane can be attached to the bypass circuit. Conventional hemodialysis is rarely feasible in the operating room as it may cause substantial hypotension. Any venovenous RRT requires the insertion of a large-bore, double-port venous catheter that allows flow rates of 150 to 300 mL/min.

Continuous RRT can be performed as either hemodialysis or hemofiltration. With continuous venovenous hemofiltration (CVVH) fluid is removed by creating a transmembrane pressure gradient. The amount of fluid removed depends on the blood flow and the surface area and water permeability of the filtration membrane. Solute removal is minimal and CVVH should be used intraoperatively only when volume overload is the sole indication for dialysis. During CVVHD solutes are removed by diffusion across a membrane against a concentration gradient, which allows for the effective removal of all dialyzable solutes and requires a blood flow rate of 150 to 300 mL/min and a dialysate flow rate of 2 to 6 L/min. Bicarbonate is preferable to acetate as a dialysate buffer as acetate can cause vasodilatation and hemodynamic instability.[66]

There is evidence that high-volume CVVHD is able to remove immunomodulatory substances such as tumor necrosis factors and endotoxins in septic shock that could decrease vasopressor requirements and improve outcome. Randomized controlled trials had conflicting results and further studies are required.[67,68]

SUMMARY

Patients presenting for surgery with renal insufficiency or failure present a significant challenge for the anesthesiologist. It is imperative that the anesthesiologist not only understands the management of these complex patients but also intervenes to prevent further renal injury during the perioperative period. Judicious fluid management, the maintenance of normovolemia, and avoidance of hypotension are priorities for the successful prevention of further renal injury. There may be instances when the use of CVVHD intraoperatively, if available, can be invaluable in the management of electrolyte and metabolic disturbances.

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