Chapter 39

Renal Disease

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Introduction

The kidneys have three primary functions: filtration, reabsorption, and secretion. To accomplish these functions, they receive about 25% of the cardiac output. The renal tubules and collecting ducts reabsorb up to 99% of filtered solutes, indicating that the total filtration volume is much greater than daily urine production. Neurohumoral substances and physiological factors that affect reabsorption of the filtered water and sodium include aldosterone, antidiuretic hormone (ADH), arterial blood pressure, atrial natriuretic factor, catecholamines, prostaglandins, renin-angiotensin, and stress.

Renal blood flow (RBF) is regulated by extrinsic nervous and hormonal control and by intrinsic autoregulation. Renal vasculature is highly innervated by sympathetic constrictor fibers originating in the spinal cord segments between T4 and L1. The kidneys lack sympathetic dilator fibers and parasympathetic innervation. Intrinsic autoregulation of RBF is demonstrated by a constant flow when mean arterial blood pressure ranges from 80 to 180 mm Hg. When the mean arterial blood pressure is between 80 and 180 mm Hg, the kidney can control blood flow by altering resistance in the glomerular afferent arterioles. Although the exact mechanism of renal autoregulation is not known, the significance of this phenomenon relates to protection of glomerular capillaries during hypertension and preservation of renal function during hypotension. However, even within the range of blood pressure described for function of renal autoregulation, extrinsic forces (e.g., neural, hormonal, and pharmacological) and intrinsic forces (e.g., renal insufficiency/failure) may cause alterations in RBF and glomerular filtration rate (GFR). Catecholamines are the major hormonal regulators of RBF. Epinephrine and norepinephrine cause dose-dependent changes in RBF and the GFR. Low doses increase arterial blood pressure and decrease RBF with no net change in the GFR. Higher doses cause decreased RBF and GFR. The renal vascular anatomy is unique in its distribution to cortical and medullary zones. Because of this vascular dichotomy, local tissue ischemia and hypoxia may occur even though total organ blood flow is normal. Oxygen delivery to the kidney is complex, and selective regional hypoxia is a possible source of renal injury during renal hypoperfusion. Experimental evidence indicates that the medullary thick ascending limb of Henle's loop, because of its high metabolic rate associated with active transport of electrolytes, is selectively vulnerable to hypoxic injury.[1]

Anesthetic Effects on Renal Function

Effects of anesthetics on RBF can be summarized with the following generalization: All anesthetics are likely to decrease the rate of glomerular filtration. Anesthetics may directly affect RBF, or they may indirectly alter renal function via changes in cardiovascular and/or neuroendocrine activity. Most anesthetics decrease the GFR as a consequence of decreased RBF (Table 39.1). Anesthetics that cause catecholamine release (e.g., ketamine, tiletamine, and nitrous oxide) have variable effects on RBF. Inhalation anesthetics tend to decrease RBF and GFR in a dose-dependent manner. Light planes of inhalation anesthesia preserve renal autoregulation of blood flow, whereas deep planes are associated with depression of autoregulation and decreases in RBF. Although isoflurane has little effect on RBF, it decreases GFR and urine output.[2] Nitrous oxide in combination with halothane does not appear to alter autoregulation of RBF.[3] Desflurane has no effect on RBF at concentrations up to twice the minimal alveolar concentration (MAC); however, it decreases renal vascular resistance at concentrations greater than 1.75 MAC.[4]

Thiobarbiturates increase systemic vascular resistance but decrease renal vascular resistance with no net change in RBF. In contrast, ketamine increases RBF and renal vascular resistance.[5] Most anesthetics cause less disruption of renal autoregulation of blood flow at lower doses (lighter anesthetic planes). Different responses to anesthetics may occur in controlled studies of RBF compared with clinical use of anesthetics. Renal responses to anesthetics also depend on the preexisting hydration status and quantity of perioperative fluids administered, as well as preexisting renal insufficiency/failure.

Due to systemic hypotension or renal vasoconstriction, renal ischemia may occur during anesthesia. Systemic hypotension may be caused by excessive depth of inhalation anesthesia, as all potent halogenated anesthetics cause peripheral vasodilation. Inhalant anesthetics also depress myocardial contractility and cardiac output in a dose-dependent manner. Hypotension may also be induced by phenothiazine or butyrophenone tranqui-

Table 39.1. Effects of anesthetics on renal blood flow (RBF) and glomerular filtration rate (GFR)[a].

Drug	RBF	GFR
Desflurane	No change	Decrease
Enflurane	Decrease	Decrease
Etomidate	No change	No change
Halothane	Slight decrease	Decrease
Isoflurane	Slight decrease	Decrease
Ketamine	Increase	Decrease or no change
Propofol	No change	No change
Sevoflurane	Slight decrease	Decrease
Thiopental	No change	No change or slight decrease

[a]Injectable anesthetics administered as a single intravenous bolus. Halogenated inhalants administered for maintenance of anesthesia.

lizers. Phenothiazine and butyrophenone tranquilizers block α-adrenoceptors and dopamine receptors. α-Adrenergic blockade may induce peripheral vasodilation and hypotension. Dopamine receptor blockade by acepromazine premedication may prevent dopamine-induced increases in RBF during surgery.

Intraoperative administration of epinephrine for hemostasis will increase renal vascular resistance and may reduce RBF significantly.[6] Renal failure may occur occasionally in animals that have been given epinephrine for hemostasis during otherwise uneventful anesthesia.[7] Following the initial ischemic insult, renal perfusion may remain altered because of other mechanisms. In experimental dogs injected intrarenally with norepinephrine, saline administration restored RBF but did not correct oliguria.[8] Necropsy of animals following acute renal failure may not detect renal damage because histological evidence may not be evident until 3 or 4 days after injury.[9]

Anesthesia and the stress associated with surgery cause release of aldosterone, vasopressin, renin, and catecholamines. Thus, RBF and GFR (and therefore urine production) are generally decreased with surgery in any patient. For most patients, the effects of inhaled anesthetics on renal function are reversed at the termination of anesthesia. Some patients, however, may not regain the ability to regulate urine production for several days.[10] Postanesthetic oliguria should be evaluated as soon as is feasible.

Some drugs used in the perianesthetic period have a significant effect on urine production. α_2-Adrenergic agonists can dramatically increase urinary output and reduce urinary osmolality.[11] Xylazine is believed to decrease ADH concentration in mares, accounting, in part, for increased urine production.[12] Detomidine-induced diuresis has also been reported in horses.[13] Because α_2-adrenergic agonists can induce diuresis, they should not be used in animals that have urethral obstruction. Reports on the effects of opioids on ADH secretion are confusing. The antidiuresis following morphine administration in animals has been attributed to increased release of ADH.[14] Others suggest this is a response to stress associated with surgical stimulation.[15] Opioids may cause urine retention when administered systemically or as an epidural injection.

Table 39.2. Potential nephrotoxins in the perianesthetic period.

Aminoglycoside antibiotics
Amphotericin B
Bilirubin
Free fluoride ion
Hemoglobin
Iodinated radiographic contrast agents
Methoxyflurane
Myoglobin
Nonsteroidal anti-inflammatory agents
Oxalate

Nephrotoxic drugs administered during anesthesia may cause oliguria (Table 39.2). Methoxyflurane is the only anesthetic known to cause nephrotoxicity as a consequence of biotransformation to oxalate and free fluoride ion.[16–18] Methoxyflurane anesthesia combined with flunixin meglumine precipitates renal failure in dogs.[19] Aminoglycoside antibiotics are potentially nephrotoxic and also enhance the renal toxicity of methoxyflurane.[20]

Effects of Renal Insufficiency on Anesthesia

Renal insufficiency/failure and renal azotemia in patients with renal insufficiency can alter the response to anesthetics. Azotemia may be associated with changes in the blood-brain barrier, leading to increased drug penetration into the central nervous system. Patients with renal insufficiency/failure may be acidotic, which will increase the fraction of unbound barbiturate and other injectable drugs in the plasma. Thus, lower doses of highly protein-bound injectable anesthetics may be required in acidotic patients.

Hyperkalemia may be present in animals with renal insufficiency/failure, obstructed urethra, or rupture of the urinary bladder. Acidosis may be associated with a concurrent increase in serum potassium. Patients in renal failure with hypocalcemia are at even greater risk, because hypocalcemia potentiates the myocardial toxicity of hyperkalemia. Further, administration of succinylcholine will transiently increase serum potassium concentration.[21] Succinylcholine-induced increases in potassium are potentially life threatening in animals with hyperkalemia. In contrast, elevation in serum potassium is not observed after administration of nondepolarizing neuromuscular blocking agents. It should be remembered that patients with hypermagnesemia associated with chronic renal failure may have prolonged recovery from nondepolarizing neuromuscular blocking agents.[22] In general, patients having a serum potassium concentration greater than 5.5 or 6.0 mEq/L should not be anesthetized until the hyperkalemia can be addressed. Electrocardiographic (ECG) abnormalities are commonly observed with potassium concentrations exceeding 7 mEq/L. The resting membrane potential of cardiac muscle depends on the permeability and extracellular concentra-

tion of potassium (Fig. 39.1). During hyperkalemia, the membrane's resting potential is raised (partially depolarized), and fewer sodium channels are available to participate in the action potential. As the serum potassium concentration increases, repolarization occurs more rapidly and automaticity, conductivity, contractility, and excitability are decreased. These changes produce the classic ECG appearance of a peaked T wave with a prolonged PR interval progressing to wide QRS complexes and loss of P waves. Mild chronic hyperkalemia may not require treatment prior to anesthesia. If treatment is instituted for chronic hyperkalemia, serum potassium should be lowered gradually to allow intracellular potassium time to reestablish physiological transmembrane concentration gradients. If hyperkalemia is acute or ECG abnormalities are noted, treatment should be initiated prior to induction of anesthesia. The most rapid treatment for the cardiac effects associated with hyperkalemia is 10% calcium chloride (0.1 mg/kg IV). Calcium will increase the membrane's threshold potential, resulting in increased myocardial conduction and contractility. Because increased serum potassium concentration causes the resting potential to be less negative (partially depolarized), the calcium ion–induced increase in threshold potential temporarily restores the normal gradient between resting and threshold potentials. It should be recognized that administration of calcium will not affect the serum potassium concentration, and its effects will therefore be short-lived. Regimens to decrease the serum potassium concentration by shifting potassium intracellularly include bicarbonate administration and combined infusion of glucose and insulin. Because acidemia favors extracellular movement of potassium and worsens hyperkalemia, intermittent positive pressure ventilation may be required to prevent anesthetic drug–induced hypercapnia and respiratory acidosis.

Patients with chronic renal failure may be anemic because of bone marrow suppression, chronic gastrointestinal tract blood loss, reduced red blood cell life span, and decreased erythropoietin production. In response to anemia, the cardiovascular system may become hyperdynamic in an attempt to maintain oxygen delivery. Chronic renal disease may be associated with hypertension and increased cardiac output but reduced cardiac reserve. Patients undergoing anesthesia should have a red blood cell transfusion if the hematocrit is less than 18% (cats) or 20% (dogs). Additionally, pulse oximetry can be used to rapidly detect hemoglobin desaturation and alert one to the potential for a decrease in tissue oxygen delivery.

In dogs and cats with mild renal insufficiency, a rapid intravenous induction of anesthesia may be accomplished with thiobarbiturates, propofol, etomidate, diazepam-ketamine, or diazepam-opioid combinations. Severely depressed patients can be mask induced with isoflurane or halothane. Anesthesia may be maintained with isoflurane or sevoflurane. The use of medications, including nonsteroidal anti-inflammatory drugs (NSAIDs), that are potentially nephrotoxic should be avoided (Table 39.2).

When renal function is questioned, the urinary bladder can be catheterized and urine production monitored via a closed, sterile urine-collection system. Urine production is an indirect measure of renal perfusion. Normal urine output for dogs is 0.5 to 1.0 mL $\cdot$ $kg^{-1} \cdot h^{-1}$. In normal horses, daily urine production has been

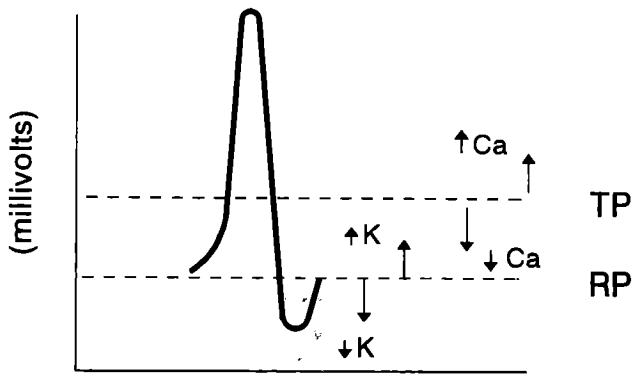

Fig. 39.1. Relationships between extracellular concentrations of potassium (K^+) and calcium (Ca^{2+}) and the resting potential (RP) and threshold potential (TP). An action potential is generated when there is sufficient depolarization to reach the TP. Increased extracellular potassium will result in raised (less negative) RP, whereas increased extracellular calcium will result in raised TP.

estimated to be about 15 L; horses that are denied feed and water for 24 h produce about 6 L.[23] Empty intravenous fluid bags can be saved for the purpose of collecting urine from catheterized patients.

If the urinary tract is not obstructed or the patient is anuric, intravenous fluids should be administered during anesthesia at the rate of 20 mL/kg for the first hour. A rate of 10 mL $\cdot$ $kg^{-1} \cdot h^{-1}$ is used thereafter. Lower fluid rates should be used if the patient has mild hypoproteinemia or cardiovascular disease. The choice of intravenous fluid is based on the animal's electrolyte and acid-base status. In general, animals with mild to moderate renal insufficiency/failure that are well prepared for surgery or anesthesia are given lactated Ringer's solution. Arterial blood pressure must be measured to detect systemic hypotension and decreased renal perfusion pressure. The mean arterial blood pressure should be maintained above 70 to 80 mm Hg. The central venous pressure (CVP) can be measured via a jugular catheter to evaluate the rate of intravenous fluid administration. Normal CVP should be between 3 and 5 cm of water. If the CVP rises more than 10 cm of water, the fluid administration should be slowed or stopped. If the CVP falls in response to the fluids being stopped, they may be resumed at a slower rate. An elevated CVP of more than 10 cm of water indicates inadequate myocardial function or volume overload. Myocardial function may be improved by infusion of dopamine (1 to 3 $\mu g \cdot kg^{-1} \cdot min^{-1}$). Low doses of dopamine will also improve RBF either through increased cardiac output, dopaminergic diuretic effects, or both. Doses of dopamine above approximately 10 $\mu g \cdot kg^{-1} \cdot min^{-1}$ may cause α-adrenergic renal vasoconstriction and decreased RBF. Improvement in renal perfusion in cats given dopamine is less well documented, but may involve α-adrenoceptor stimulation rather than dopamine receptor agonism.[24] Putative renal dopamine receptors (DA1) in cats appear to differ from those identified in kidneys of other species.[25] Other DA1 agonists, such as fenoldopam, may prove useful in improving RBF and diuresis in this species.

Tests of Renal Function

Measurements of the GFR and renal tubular function, such as urine specific gravity and blood urea nitrogen (BUN), are not specific for renal disease. Serum creatinine is a more specific indicator of the GFR than BUN because it is influenced by fewer extrarenal variables. It is important to keep in mind that greater than 75% nephron loss is necessary for most patients to become persistently azotemic. Thus, patients with mild renal insufficiency may not have elevated serum creatinine. Persistent proteinuria and/or cellular or granular cylinduria may indicate renal damage prior to the onset of renal azotemia.

In addition to BUN, serum creatinine concentrations, and urinalysis, patients with renal insufficiency should be evaluated to determine acid-base balance, electrolyte concentrations (especially potassium), exercise intolerance, hematocrit, hydration, and urine production.

Postoperative Oliguria

Postoperative oliguria (<0.5 mL · kg^{-1} · h^{-1}) should be investigated. If the animal does not have congestive heart failure or pulmonary edema, a fluid challenge of 5 mL/kg isotonic sodium chloride may be given. If urine production increases, the animal was hypovolemic and fluids should be continued. If not, dopamine may be infused at a rate of 1 to 3 μg · kg^{-1} · min^{-1}. Dopamine improves renal function when used at low doses by increasing RBF, GFR, urine output, and sodium excretion, and by decreasing renal vascular resistance.[26] At moderate doses (approximately 5 to 10 μg · kg^{-1} · min^{-1}), dopamine activates ß-adrenoceptors in addition to DA1 receptors, which may dilate renal arterial beds and increase cardiac output. As previously mentioned, dopamine doses above approximately 10 μg · kg^{-1} · min^{-1} can cause α-adrenoceptor stimulation, vasoconstriction, and decreased RBF even though systemic arterial blood pressure may increase. Dopamine may also be beneficial because it inhibits intrarenal norepinephrine release and has an antialdosterone effect.[1] Recall that, in patients medicated with acepromazine, low-dose dopamine may be ineffective at increasing RBF, because of dopaminergic receptor blockade.

The use of diuretics in the perioperative period is controversial. In a study of human patients, acute renal failure treated with diuretics such as mannitol and furosemide was not resolved.[27] In a dog model of uranyl nitrate–induced acute renal failure, a combination of furosemide and dopamine was effective in restoring RBF and creatinine clearance, whereas either drug alone was not.[28] In a more recent study, greater diuresis, natriuresis, and calciuresis and less kaliuresis occurred in normal dogs given a constant rate infusion of furosemide (0.66 mg/kg IV loading dose followed by 0.66 mg/kg/h) when compared with intermittent bolus administration.[29] Furosemide is used to promote diuresis in patients with pulmonary edema but should not be used when a patient is known to be hypovolemic. In hypovolemia, furosemide may increase nephrotoxicity of other drugs by increasing their contact time in the renal tubules.[30] Mannitol, an osmotic diuretic, can be given (0.25 to 0.5 g/kg slowly IV) to prevent pulmonary edema or hyponatremia if the kidneys do not respond to fluid administration and the patient becomes volume overloaded.

Urethral Obstruction

Patients with urethral obstruction may become hyperkalemic, azotemic, acidotic, and hyperphosphatemic. Cats may also develop hyperglycemia, hypocalcemia, and hyponatremia. Hyponatremia is associated with leakage of urine into the peritoneal cavity.[31] Any metabolic abnormalities should be evaluated and addressed prior to anesthesia, if possible. Hyperkalemia is the primary concern in most cases of urethral obstruction, and a preanesthetic electrocardiogram assessment is warranted. Treatment of hyperkalemia has been discussed in preceding paragraphs.

In small animals with urethral obstruction, fine-needle centesis of the urinary bladder may be performed prior to anesthesia, although bladder injury is a potential concern. Rupture of the urinary bladder during induction of anesthesia in a horse has been described.[32] Perineal urethrostomy may be performed in stallions with urethral blockage by using standing restraint and epidural anesthesia. If general anesthesia is required while the bladder is distended, every attempt should be made to assist the horse into sternal or lateral recumbency during induction.

Anesthesia may be induced by using injectable or inhalation anesthetics. In many animals, distension of the urinary bladder is associated with increased heart rate. Cats that are chamber induced should not be induced with halothane due to its arrhythmic effects. Chamber induction with isoflurane or sevoflurane is preferred for small animals. Intravenous ketamine with a benzodiazepine has been used in obstructed cats even though active metabolites of the drug are excreted by the kidney. The rationale is that, once the obstruction is relieved, excretion of the anesthetic will proceed normally. However, cats with a long-term urethral obstruction may develop metabolic disturbances and renal insufficiency such that elimination of drugs is slowed even after the obstruction has been removed. Thus, if dissociative anesthesia is used, low doses of ketamine (1 to 2 mg/kg IV) can be used in combination with diazepam (0.2 mg/kg IV). With low doses, anesthetic action will be reduced after redistribution of the drug into body tissues. More commonly, injectable anesthetic induction in cats with renal disease is performed by administering propofol (2 to 5 mg/kg IV) or etomidate (1.0 mg/kg IV) slowly to effect.

Ruptured Urinary Bladder

Rupture of the urinary bladder is a surgical emergency. Animals may become hyperkalemic, hyponatremic, hypochloremic, and acidotic after urinary bladder rupture.[33] Intravenous fluids, such as 0.9% sodium chloride, should be given to aid in correcting electrolyte imbalances. Potassium enters the abdominal cavity from the ruptured bladder and is reabsorbed into the circulation, causing an increased serum potassium concentration. An electrocardiogram should be evaluated prior to induction of anesthesia to determine whether cardiac arrhythmias or evidence of hyperkalemia are present. Acute hyperkalemia (serum $[K^+]$ > 5.5

mEq/L) should be treated prior to anesthesia. Anesthesia may be induced by face-mask administration of isoflurane or sevoflurane. Young foals (weighing < 200 kg) may be nasotracheally intubated while awake and then induced rapidly with an inhaled anesthetic. In larger foals, xylazine (1.1 mg/kg IV) or diazepam (0.1 mg/kg IV) in combination with ketamine (2.2 mg/kg IV) can be used for induction.[34] Isoflurane and sevoflurane are preferred over halothane because they possess less myocardial depressant action and potentiation of catecholamine-induced cardiac arrhythmias.[35] Administration of dextrose-containing solutions with or without insulin may be desired to counter hyperkalemia. Positive-pressure ventilation may be used to prevent anesthetic-associated hypercapnia and accompanying respiratory acidosis. Appropriate monitoring should be used, including end-tidal carbon dioxide, pulse oximetry, arterial blood-gas analysis, arterial blood pressure, and electrocardiogram assessment.

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